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IMPLEMENTING AN INPATIENT CHAIN OF SURVIVAL AT AN ACADEMIC MEDICAL CENTER

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Introduction: Publication of “To Err Is Human” by the Institute of Medicine in 2000 has brought attention to the number of preventable deaths resulting from errors in the United States healthcare system. Patients often have signs of physiological deterioration for several hours before “unexpected” cardiopulmonary arrest occurs. In certain situations, even when recognition of deterioration is prompt, intervention may be delayed due to multiple barriers. We have initiated a quality improvement project in the form of an “inpatient chain of survival” as an effort to reduce the incidence of out of Intensive Care Unit (ICU) cardiopulmonary arrest.

Methods: This quality improvement project was performed on a high-acuity telemetry/stepdown unit at Banner University Medical Center – Tucson, a 479 bed academic medical center in Tucson, Arizona. During 2014, there were 12 episodes of cardiopulmonary arrest (CPA) on this 30-bed unit. In order to improve patient safety, we developed an “inpatient chain of survival.” The links in this chain are education regarding the major patterns of pre-arrest physiology; more rigorous monitoring and risk stratification; a structured response to deteriorating patients; and mandatory audit and review of all episodes of CPA as potentially preventable adverse events. Major interventions included recurring staff education and coaching related to detecting pre-arrest patterns; afternoon nurse safety rounds led by experienced charge nurses; mandatory calling criteria for the rapid response team; development of an order set for RRT responses; and multidisciplinary review of all 2014 CPA events. Education and chart reviews began in late 2014 and early 2015, and the nurse safety rounds, mandatory RRT calling, and order sets were implemented on June 1, 2015

Results: We noticed that prior to 50% of the 12 CPA events in 2014, signs of deterioration, including oxygen desaturation and change in mental status were present. 67% of the events happened during the day shift (0700-1900). As expected, after the initiation of the initiative, there is an increase in rapid response activation. From June 1st 2015 to date (October 31<sup>st</sup> 2015), no CPA events have occurred on the unit. The first phase of this quality improvement project will be continued for 6 months, complete analysis of RRT calls and outcomes will be presented at the meeting.

Conclusion: The encouraging initial result of the chain of survival demonstrates that a structured protocol is effective in a reduction in ‘unexpected’ cardiopulmonary arrests. But most importantly, the change in culture through increased awareness of patient safety and interdisciplinary collaboration are the main contributors to the success of this protocol.

## 5-COMPONENT BUNDLE FOR CLOSTRIDIUM DIFFICILE PREVENTION

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**Background:** Clostridium difficile infection (CDI) is a common cause of harm in hospitals. At our institution, the estimated cost of harm in 2013 was \$1.4 million. Risk factors for CDI include antibiotic use, gastric acid suppression, increasing age, hospitalization, serious co-morbidities, and prior CDI. In the first 3 months of 2014, hospital-onset (HO) CDI incidence increased, while other hospital acquired conditions decreased. Our community 9-bed medical-surgical ICU adopted a CDI prevention bundle with the goal of a 25% reduction in HO-CDI over a 6 month period and sustain it throughout the fiscal year.

**Methods:** The CDI bundle included 5 components: (1) environmental cleaning surveillance with; (2) improving staff awareness of CDI rates and adenosine triphosphate (ATP) swabbing results; (3) hand hygiene compliance above 90%; (4) antibiotic stewardship; and (5) proton pump inhibitor (PPI) stewardship. We conducted environmental surveillance by measuring ATP relative light units (RLU), with RLU below 45 considered clean of various high-touch items in ICU rooms. Clinical pharmacists conducted antibiotic and PPI stewardship through in-service training of providers and system wide emphasis on documentation of antibiotic stop dates. For hand hygiene, we posted reminder signs and covertly monitored hand washing. The intervention began in April 2014. We defined HO-CDI per standard definitions and calculated rates per 10,000 patient-days, and a rate-ratio between pre-intervention (Apr 2013-Mar 2014) and post-intervention (Apr 2014-Mar 2015) periods.

**Results:** Environmental cleaning surveillance was done on 35 items; results ranged from a bedside table at 9 RLU to a patient remote at 910 RLU. Hand hygiene compliance was maintained at 95%. The amount of commonly prescribed antibiotics decreased from 369.9 to 262.4 per 100 patient-days (29.2%) and total antibiotic doses decreased from 1805 to 1527 (15.7%). PPI use decreased from 79.9 to 62 per 100 patient-days (22.4%). HO-CDI decreased from 8 cases (40.0 cases/10,000 patient-days) pre-intervention to 2 cases (9.0 cases/10,000) post-intervention (rate ratio 0.22, 95% CI 0.03-0.97;  $p = 0.04$ ). After the intervention there were no cases of HO-CDI for 9 continuous months.

**Discussion:** Implementation of a 5-component bundle successfully decreased HO-CDI by nearly 80% over a subsequent 12-month period. Both antibiotic and PPI usage decreased substantially. The environmental cleaning surveillance helped increase staff awareness of which items were less frequently cleaned. Overall, this team-based, multidisciplinary bundle is an effective way to prevent HO-CDI and currently is being implemented throughout our intensive care units.

AGE-RELATED DIFFERENCES IN PARACRINE-MEDIATED ANGIOGENESIS

First Author: Tamara Ashvetiya Authors: Chao-Wei Hwang, Robert Weiss, Gary Gerstenblith, Peter Johnston

**BACKGROUND:** Growing evidence suggests that beneficial effects of stem cell therapy are mediated to a large extent by paracrine action. The elderly stand most to benefit from such therapy, but little is known about age-related differences in paracrine-mediated tissue regeneration. We compared angiogenesis stimulated by young and old mesenchymal stem cells (MSCs) contained in semi-permeable “bioreactor” tubes (500 kDa molecular weight cut-off) that allow free exchange of paracrine factors (PFs), but not cells.

**METHODS:** MSCs isolated from young (6 wk) and old (18-24 mo) C57BL mice were grown in bioreactors at normoxia or at hypoxia (5% O<sub>2</sub>). Bioreactors were exposed to human vascular endothelial cells (HUVECs); resultant tubule formation (TF) was imaged and normalized to controls. Media was collected from outside bioreactors at 7 days and VEGF ELISA was performed.

**RESULTS:** At normoxia PFs from young MSCs induce greater TF than old MSCs (p=0.006). After exposure to hypoxia, more TF is induced by old MSCs, but not young, and there is no longer a difference between young and old (p=0.49). At normoxia, paired bioreactors containing 100Kyoung/100Kold MSCs induce equivalent TF to 200K young MSCs (p=0.73), and significantly more than 200K old MSCs (p=0.03). Young MSCs produce more VEGF than old at both normoxia (p=0.004) and hypoxia (p=0.004), though only the young augment VEGF production with hypoxia exposure (p=0.02).

**CONCLUSIONS:** Young MSCs show superior paracrine-mediated angiogenesis, as measured by HUVEC TF, compared to old MSCs at normoxia. Exposure to hypoxia improves relative PF-mediated angiogenesis by old cells compared to young. Exposure to young MSCs “rejuvenates” old MSCs as measured by HUVEC TF. These observations may provide a means to improve PF-mediated regenerative effects of aged stem cells, and suggest that allogeneic young cells may be ideally suited for stem cell therapy in older individuals. VEGF production and in vitro angiogenesis are not directly correlated, suggesting that other PFs also contribute to angiogenesis. The mechanisms involved in this process deserve further study.

	HUVEC Assay (TF±stand. dev.)	VEGF ELISA Assay (pg/ml)
200K Young/Normoxia	2.84±0.97(N=14)	37.2±8.9(N=6)
200K Old/Normoxia	1.86±0.71(N=14)	15.0±11.3(N=6)
200K Young/Hypoxia	2.90±0.91(N=18)	100.3±46.7(N=6)
200K Old/Hypoxia	2.69±1.0(N=22)	9.81±4.9(N=6)
100KYoung/100K Old Normoxia	2.71±0.67(N=6)	NA.



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CARDIAC CT IS A FEASIBLE SCREENING STRATEGY FOR CORONARY ARTERY DISEASE (CAD) IN LONG TERM ALLOGENEIC STEM CELL TRANSPLANT (ALLO-SCT) SURVIVORS

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Significant increases in CAD risk and cardiovascular events have been described in long-term allo-SCT survivors compared to age and gender-matched population controls. Since pharmacologic interventions positively influence the evolution of CAD and subsequent cardiovascular events, an effective screening strategy is essential. However, optimum screening strategy in this unique population is undefined.

We conducted a prospective non-randomized study using cardiac CT to evaluate Agatston coronary calcium scoring by CT with concomitant coronary CT angiography for screening asymptomatic allo-SCT survivors. 67 subjects (40 males; 27 females) with a median age of 49 years (range 24 - 76) at transplant and a median follow up interval of 10.5 years (range: 3-22) were studied. Angiography was excluded in 7 subjects with renal dysfunction. CAD was defined as presence of lesions on CT angiography and/or calcium score > 0 (for subjects without angiography). 10-year Framingham scores were also calculated: 3 were classified as high risk, 5 were intermediate and 59 were low risk.

CAD was detected in 30 of 67 (44.78%) subjects. Characteristics of coronary plaques were: 29.3% calcified, 42.7% mixed calcified / non-calcified, and 28% non-calcified. Coronary lesions were mostly non-obstructive (89.3%), but obstructive lesions were seen in 10.7%. Lesion distributions by arterial territory were: left main 11.4%, left anterior descending 35.8%, left circumflex 21.4% and right coronary artery 31.4%.

Radiation exposure during the procedure was negligible, at a median of 0.72 mSv (interquartile range: 0.95) for the coronary calcium score and 0.97 mSv (interquartile range: 1.26) for the coronary CT angiogram. There were no adverse events.

Calcium score was able to detect CAD in 2 of 7 subjects who could not have angiograms because of renal dysfunction. Current (2010 AHA/ACC) guidelines suggest a role for coronary calcium scoring for screening asymptomatic non-transplant individuals with intermediate Framingham risk. However, cardiac CT detected CAD in 24 of 59 (40.7%) low Framingham risk survivors. Coronary calcium scoring alone may be adequate for screening and avoids the use of IV contrast.

Accelerated CAD was detected in 45% of our alloSCT survivors by cardiac CT screening. Coronary calcium score with or without CT angiogram is a safe, feasible and sensitive screening technique for CAD. It is informative even in asymptomatic, low-risk survivors and is far more sensitive than the Framingham risk score.

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LONG TERM MORTALITY ASSOCIATED WITH CORONARY ARTERY CALCIUM

First Author: Patrick Moon, MD, Member Second Author: Joshua D. Mitchell, MD, FACP, Last (Senior) Author: Todd C. Villines, MD, FACP

**Introduction:** Coronary artery calcium scores (CAC) have been linked to cardiovascular morbidity and mortality; guidelines suggest their use in helping stratify intermediate risk patients. Additional studies with extended follow-up are needed to better determine the prognostic value of coronary artery calcium. We sought to verify the prognostic value of both normal and positive CAC scores in a large single centered cohort with over ten year follow-up.

**Methods:** We retrospectively studied patients referred for CAC scoring from Walter Reed National Military Medical Center from 1997 to 2009. The military data repository (MDR) was searched to determine incidence of myocardial infarction (MI), stroke, revascularization and death. We searched for diagnosis codes from records in the MDR from October 1, 1988 until December 31, 2014. Patients were excluded if they could not be definitively matched to a record in the MDR or if they had pre-existing MI, stroke, or revascularization prior to their coronary artery calcium score. Kaplan Meier and Cox Regression were used for time to event analysis. Statistics were computed using SAS 9.3.

**Results:** During the study period there were a total of 36,532 CAC scans. Thirty patients were removed since they could not be positively matched in the MDR and 297 were removed to presence of MI, stroke, or revascularization prior to their CAC scan. Of the 36,532 scans there were 30,976 unique patients of whom 36% had a positive CAC score (n=11215). Mean follow up was over 11.7 years. Patients were grouped into scores of 0, 1-100, 101-400 and over 400. Survival decreased with increasing CAC score ( $p < 0.0001$ ). Focusing on mild disease, there were still more deaths in patients with a CAC score of 1-100 (266 of 6955 patients) compared to a CAC score of 0 (352 of 19,761 patients) (HR 2.07,  $p < 0.0001$ ). There was no difference in mortality in patients with a CAC score of 1-10 (46 of 2300 patients) compared to a CAC score of 0 (352 of 19,761) ( $p = 0.62$ ).

**Conclusion:** In patients without preexisting MI, stroke, or revascularization increasing CAC scores were associated with decreased survival. A mildly positive CAC score (1-10) showed no difference in survival compared to a CAC score of 0.

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IPODS TRIAL: INTERVENTION TO PROMOTE OSTEOPOROSIS SCREENING WITH DXA SCAN

First Author: Sonam Kiwalkar, MD, Tina Brar, MD, Suzana John, MD, Michael Henderson, MD.

**Introduction:** Osteoporosis is a silent disease until it is complicated by fractures. The 2014 National Osteoporosis Foundation reported, annually 2 million fractures are attributed to osteoporosis causing more than 432,000 hospital admissions, 2.5 million medical office visits and 180,000 nursing home admissions. Having found low rates of screening in our clinic, we designed a trial to improve provider awareness and screening for osteoporosis using DXA scan.

**Methods:** Cohort, retrospective and prospective study, to analyze pre and post intervention data on ordering of DXA scans in females > 65 years, between November 2013 to March 2015. The study took place in primary care, resident run outpatient clinics of Rochester General Hospital, NY. All post-menopausal women > 65 years who were scheduled for routine visits in outpatient clinics of Rochester General Hospital were included in the trial. Extensive chart review was done by investigators every day from November 2014 to March 2015 for prospective arm. All post-menopausal women >65 years who attended clinic from November 2013 to October 2014 were included in retrospective arm. The intervention included a one-time 10 minute educational lecture to educate providers on risk factors, diagnosis and treatment of osteoporosis during in October 2014, with staff message reminders sent by investigators every 5 weeks, in prospective arm. Primary analyses compared rates of ordering DXA scan before and after implementation of educational session. Rate of completion of DXA scan in the population where it was ordered was computed and patient non-compliance was also explored. Secondary analyses looked at number of patients receiving appropriate treatment as per DXA scan results, as well as calcium and vitamin D supplementation. Barriers to ordering a screening DXA by providers were also computed by provider based survey.

**Results:** The primary analyses included rate of screening in eligible women before the intervention, which was 15.6%. After the intervention, it increased to 49.8%, which was statistically significant (p-value: 0.0001). The rate of DXA completion before intervention was 11%, and after intervention was 23.6% (p-value: 0.0001). Patient non-compliance was 29.7% vs. 44.4% (p=0.01) before and after the intervention respectively. Per the survey, most providers felt that time constraint was an important barrier in limiting ordering of DXA scans.

**Conclusions:** The IPODS trial intervention of education and periodic reminders resulted in a significant increase in DXA scan ordering. Further studies are required to assess the lasting effect of the intervention and to analyze barriers faced by non-complaint patients. This is being addressed in IPODS phase 2 trial.

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ELIGIBILITY FOR PCSK9 THERAPY IN CHOLESTEROL CENTER PATIENTS WITH INITIAL LDL CHOLESTEROL = 130 BUT

First Author: Kevin S Lee, MD Co Authors: Marloe Prince MD, Vybhav Jetty MD, Parth Shah MD, Michael Goldenberg, Ashwin Kumar, Charles J. Glueck MD, Ping Wang PhD

**Background:** By lowering LDL cholesterol (LDLC) ~60% beyond statins, PCSK9s have the potential to profoundly improve primary and secondary prevention of atherosclerotic coronary artery (CAD), peripheral (PAD), and carotid artery disease,

**Purpose:** In 189 patients referred to a regional Cholesterol Center for diagnosis and treatment of hypercholesterolemia, who had initial LDL cholesterol (LDLC) = 130 but <160 mg/dl, our specific aim was to determine how many would be eligible for PCSK9 therapy by extant preferred commercial insurance criteria.

**Methods:** Current preferred commercial insurance criterion for PCSK9 therapy consisted of =1 of the following 3 conditions:

- 1) Heterozygous familial hypercholesterolemia (*previous* LDLC >190 mg/dl and Tendon Xanthomas),
- 2) Atherosclerotic cerebral-cardio-peripheral vascular disease
- 3) *Failure to tolerate 2 or more statins*

**Results:** At entry, in the 189 patients, mean  $\pm$  SD and median LDLC were 144  $\pm$  9 mg/dl and 143 mg/dl respectively. Of the 189 patients (96 female and 93 male with median age 53) 16 (8%) were diagnosed as having heterozygous FH, with median LDLC of 139 mg/dl, and 32 (17%) had sustained a cerebral-cardio- peripheral vascular event with median LDLC on treatment of 146 mg/dl. Of the 189 patients, in 44 (23%) the maximum tolerated statin dose was zero (*complete* statin intolerant), with median LDLC of 145 mg/dl. Of the 44 statin intolerant patients, 18 (41%) had either HeFH or an atherosclerotic event, and 26 (59%) had neither positive. Altogether 73 of 189 (39% of patients with entry LDLC = 130 but < 160 were eligible for PCSK9 therapy from commercial carriers.

**Conclusion:** Of 189 patients referred to a regional Cholesterol diagnosis and treatment center with initial LDLC =130 but <160 mg/dl, 73 (39%) met current commercial insurance carrier preferred criteria for PCSK9 drug coverage.

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PSYCHIATRIC CONDITIONS AS A PREDICTOR OF OUTCOMES IN PATIENTS HOSPITALIZED WITH CONGESTIVE HEART FAILURE

First Author: Toni Anne A. De Venecia, MD Marvin Lu MD Mary Ziccardi Rodriguez MD Vincent Figueredo MD

Background: The high cost of caring for patients with congestive heart failure (CHF) results primarily from frequent hospital readmissions for CHF exacerbations. Proven determinants of heart failure readmission rates are non-compliance with medications and diet, lack of discharge planning or follow-up and failed social support system. Only depression has been identified to affect readmission and outcomes of heart failure patients. This study aims to determine the prevalence and relationship of common psychiatric conditions to outcomes of patients hospitalized with CHF.

Methods: This is a single center retrospective study of 298 patients admitted for CHF to Einstein Medical Center Philadelphia admitted from June 2010 to June 2011. Admission variables, including marital status, living conditions, illicit drug use, compliance with diet and medications, acute kidney injury and psychiatric conditions, were obtained. Primary care and cardiologist follow up on discharge, were also obtained. Readmission by 30 days and death by 1 year were determined using retrospective chart review and SSDI, respectively.

Results: Of 298 patients, 28 patients (11%) were known to have a psychiatric disorder. Seventeen patients were diagnosed with depression, two with bipolar disease, and nine with schizophrenia. Ten of these patients were readmitted within 30 days for CHF exacerbation. In univariate analysis, patients with psychiatric conditions had significant 30 day readmissions with an odds ratio of 5.2 (1.1-7.1;  $p < 0.05$ ). Other factors of significance were lack of cardiologists follow up, non-compliance with medications and living status. In multivariate analysis, after controlling for significant variables, having a psychiatric diagnosis was still significantly predictive of 30 day readmission with an odds ratio of 2.8 (CI 1.1-7.0;  $p < 0.05$ ). No association was found with one year mortality.

Conclusion: CHF patients known to have psychiatric disorder get readmitted more often within 30 days after discharge. Ensuring psychiatric follow up on discharge should be considered and can potentially improve HF care and related clinical outcomes.

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NATURAL FREQUENCY VERSUS ODDS BASED STATISTICAL METHODS IN UNDERSTANDING THE MAMMOGRAPHY AND BREAST CANCER DEBATE

First Author: Bhaskar Ganguly, MD Mahesh Krishnamurthy, MD David Livert, PhD

**Introduction:** Breast cancer screening with mammography continues to be a hotly debated issue among health care providers as well as the general American population- so much so that even the politicians have their own opinions on this. One factor that confuses the matter is the lack of understanding of statistics by health care providers. This study was performed to assess physician understanding of biostatistics and if understanding changed when presented with odds based statistics versus natural frequency.

**Method:** The research design was a cross-sectional study. The study was conducted in 2 phases. In the first phase, 24 residents and 11 attending physicians were asked to answer the odds based statistics question:

*Estimate the probability that a woman with a positive mammogram has breast cancer with the probability that a woman in her age group has breast cancer is 0.8 percent and if a woman has breast cancer the probability is 90 percent that she has a positive mammogram. But even if a woman does not have breast cancer the probability is 7 percent she will still have a positive mammogram.*

In the second phase, 8 residents and 5 attending physicians were asked to answer the question revised in terms of natural frequency:

*Estimate the probability that a woman has breast cancer with a positive mammogram, when you know that 10 out of every 1,000 women have breast cancer, and of those 10 women 9 will test positive on mammography, and of the remaining 990 women without cancer about 89 will nevertheless test positive.*

**Results:** The results showed that when the question was rephrased in natural frequency terms the correct answer from residents increased from 32% to 63% while for attending physicians the number of correct answers increased from 18% to 20%.

**Conclusion:** The results of the study showed that physicians might not have a good understanding of statistical data. As the principal providers of information regarding breast cancer screening to patients, this highlights an area that promotes generalized misunderstanding. Our study showed that there was improvement in understanding when the data was rephrased in natural frequency terms among residents, however not among attending physicians. A solution would be to change the presentation of statistical data to natural frequency. In addition, exposure to natural frequency needs to begin early in the medical career for this understanding to carry over to post residency careers.

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CHANGE IN PRESCRIPTION HABITS AFTER FEDERAL RESCHEDULING OF HYDROCODONE COMBINATION PRODUCTS

First Author: Susan Seago, MD Second Author: Adam Hayek, DO Last Author: Megan Greene Newman, MD

**INTRODUCTION:** According to the most recent data released by the Centers for Disease Control, forty-six Americans die every day due to narcotic prescription drug overdose. Nationally, health care providers wrote two hundred and fifty-nine million prescriptions for narcotic analgesics in 2012, or roughly one bottle of narcotics per US adult. In an attempt to reduce misuse, the Drug Enforcement Administration changed the schedule of hydrocodone combination products from schedule three to schedule two on October 6, 2014. These medications now require a triplicate prescription, which has increased restrictions on prescribing and dispensing practices. Although many praised the increased regulation, our project sought to determine whether the new legislation is effective in decreasing total narcotics prescribed or if it simply changed the type of pain medication prescribed.

**METHODS:** Fourteen Baylor Scott & White pharmacies encompassing a two-hundred-mile radius of central Texas were queried for prescription information on hydrocodone/acetaminophen, morphine, codeine/acetaminophen, and tramadol. Pharmaceutical data from July 2014 through September 2014 before the rescheduling was then compared to data from November 2014 through January 2015 to evaluate trends in prescription drug usage.

**RESULTS:** Analysis revealed a 42% percent reduction in hydrocodone/acetaminophen 5/325mg, 14% reduction in hydrocodone/acetaminophen 10/325mg and 7% reduction in oral morphine sulfate after federal rescheduling of hydrocodone combination productions. During the same period, however, there was a 583% increase in codeine/acetaminophen 30/325mg, 827% increase in codeine/acetaminophen 60/325mg, and 9% increase in tramadol 50mg. When all narcotic prescriptions were converted to morphine equivalents, there was only a 3% reduction in total amount of pain medications prescribed after rescheduling.

**CONCLUSIONS:** While the rescheduling of hydrocodone combination products resulted in a reduced number of prescriptions for both the 5/325mg and 10/325mg formulation, this was offset by a dramatic increase in alternate narcotic analgesics such as codeine/acetaminophen and tramadol which do not require a triplicate prescription. Additionally, there was no significant reduction in total pain medication prescribed after converting all agents to morphine equivalents. Our study illustrates an important and evolving trend trend in narcotic prescription habits after federal rescheduling of hydrocodone combination products and highlights the need for further research on effective means for controlling prescription drug misuse in the United States.

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A NEW RISK FACTOR TO CONSIDER: PATIENTS WITH SEVERE PULMONARY HYPERTENSION WHO UNDERGO TRANSCATHETER AORTIC VALVE REPLACEMENT HAVE INCREASED RISK OF MORTALITY

Sridhar Reddy MD Sarika Ballari MBBS Jagdesh Kandala MD, MPH Ranjith Shetty MD Kapil Lotun MD

**Introduction:** The prevalence of pulmonary hypertension (PH) in patients with aortic stenosis (AS) is starting to be recognized more commonly, with estimates that 65% of patients with severe symptomatic AS also have PH. Most studies to date assessed PH severity using noninvasive measurements of pulmonary artery systolic pressure, which correlates only modestly with invasive measurements. Using the gold standard of right heart catheterization, we looked to differentiate patients with baseline PH into mild, moderate and severe and assess the impact these varying severities have on clinical outcomes after transcatheter aortic valve replacement (TAVR).

**Methods:** We performed a retrospective analysis of patients from 2012-2014 who underwent TAVR at our institution and were then followed for 1 year in an ambulatory clinic setting. Prior to the procedure, patients underwent right heart catheterization in order to assess pulmonary artery systolic pressures (PAPs) and were differentiated into mild, moderate and severe PH. Primary end-point was clinical outcomes measured as all cause death at hospital discharge and at one year. Secondary end-point was hospital length of stay.  $P < 0.05$  was considered statistically significant.

**Results:** A total of 73 patients were found to have PH. Mild PH was present in 19(26%), moderate PH in 45(61.6%), and severe PH was present in 9(12%). Among these, a total of 6(8.2%) died at 30-day follow up. At 30-day follow up, all-cause mortality was 0 in the mild PH group, 3 (6.6% in moderate pulmonary hypertension, and 3(33.3%,  $p=0.009$ ) in severe PH.

After mean follow up duration of  $6.2 \pm 5.6$  months, a higher mortality rate of 66.6%( $n=6$ ) was noted in severe PH group when compared to 15.5%( $n=7$ ) in the moderate PH group, and 15.7%( $n=3$ ) in mild PH ( $p=0.002$ ). Kaplan-Meier survival analysis demonstrated worse event free survival in patients with severe pulmonary hypertension. In a multivariate Cox proportional hazards model, severe pulmonary hypertension was an independent predictor of all cause mortality with a hazard ratio of 3.4( $p=0.035$ ).

**Conclusions:** The major findings of this study were that the mortality rate of patients with severe PH was significantly higher than mortality rates of patients with mild and moderate PH. Based on these findings, we can conclude that severe PH is an independent predictor of all cause mortality in patients undergoing TAVR. Our results are unique in that we used invasive hemodynamic measurements to show that patients with severe PH are predisposed to worse outcomes than patients with mild and moderate PH. This study suggests that the stratification of PH according to severity is useful for risk stratification of patients with severe AS being considered for TAVR. This can have important implications for treatment decisions amongst physicians and patients.

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CLOSTRIDIUM DIFFICILE INFECTIONS IN HOSPITAL & COMMUNITY SETTINGS

First Authors: Heather N Bitar, DO, Amanda Frugoli, DO, and Shadi Sharif, DO Additional Authors: Christine Ouellette, RN and Henry Oster, MD

Background: Clostridium difficile infections (CDI) are on the rise and are costing the healthcare system 4.8 billion dollars per year. The purpose of this study was to investigate whether there is a significant difference in mortality based on source of infection, seasonality, and age of subjects given the recent rise in CDI rates in our community.

Methods: This retrospective study analyzed three populations: Community-acquired CDI, Hospital-acquired CDI, and Post-acute care facility residents with CDI. Adults with a primary or secondary diagnosis of CDI were included. Any subject under the age of 18 or pregnant were excluded. Effect of age, seasonality, and source of infection were studied using logistic regression. Ribotyping using agar dilution method was performed on randomly selected samples to survey for hyper virulent strains. The main outcome was all cause mortality.

Results: A total of 451 cases of CDI were identified at Community Memorial Hospital from April 2009 through December 2014. This number equates to 5.8 CDI cases per 1000 admissions consistent with national data of 5.9 per 1000 cases. There was no difference in mortality with seasonality or source of infection. The most significant pattern identified was the relationship between mortality and age, which demonstrated a 6.8% increase risk of death for every year after age of 59 ( $p = 0.00138$ ; OR = 1.068). A sub-population ribotyping analysis revealed that half had a hyper virulent strain with 014/020 more prevalent than 027, but the population was too small for additional inferences.

Conclusion: There was no significant difference in all-cause mortality based on source of infection or seasonality. The strongest association was between age and mortality with a 6.8% increase in relative risk of death with each additional year after age 59. Further studies are needed to investigate other variables contributing to the local rates of infection.

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OPIOID PRESCRIBING DISTRIBUTION: NOT JUST A FEW BAD APPLES

Jonathan H Chen Keith Humphreys Nigam H Shah Anna Lembke

**Background:** Prior research has suggested that the opioid overdose epidemic is largely driven by small groups of prolific prescribers. For example, the California Workers' Compensation Institute found that just 10% of their prescribers accounted for ~80% of their opioid prescriptions. This implies that monitoring, education and intervention efforts should be directed at this small group of high-volume prescribers. Medicare data can address whether this reflects general practice patterns across the nation.

**Methods:** We examined individual prescriber-level data from the 2013 Medicare Part D (prescription drug coverage) claims dataset created by the Centers for Medicare and Medicaid Services. Part D covers ~68% of the ~50 million people on Medicare, the federal insurance program for Americans who have certain disabilities or are 65 or older.

The data represents the counts for 1.2 billion prescription claims organized by drug name and individual prescriber National Provider Identifier (NPI) number (N=808,020). Each NPI includes location and specialty of practice.

We calculated the cumulative percentage of claims for schedule II opioids from the top 10% of individual prescribers (sorted by number of claims) relative to the total claims from all prescribers. We repeated this for all drugs and for each state.

**Results:** Specialties with the most total opioid claims include Family Practice (15.3 million), Internal Medicine (12.8 million), Nurse Practitioners (4.1 million), and Physician Assistants (3.1 million). Specialties with the most opioid claims per provider include Interventional Pain (1,124), Pain Management (921), and Anesthesia (484).

The top 10% of Medicare opioid prescribers account for 57% of opioid claims, less than the proportion of Medicare prescriptions for all drugs (63%) or for the California Workers' Compensation prescriptions for opioids (79%). Minimal regional variation is observed, with per state values ranging from 56.6% to 57.7%.

**Comment:** The data represent a comprehensive national population, though they lack information on providers' complete practices, patient factors, and medication dosing. With those cautions, an important finding is evident.

Contrary to research highlighting a small subset of high-volume prescribers accounting for a disproportionately large percentage of opioid prescribing, Medicare opioid prescribing is distributed across many prescribers and is, if anything, less skewed than all drug prescribing. The trends hold up across state lines, with negligible geographic variability.

Although some skewing is expected for social phenomena under the Pareto "80/20" Rule, as of 2013, opioid prescribing appears to be a widespread practice relatively indifferent to individual doctors, specialty, or region. High-volume prescribers are not responsible for the high national volume of opioid prescriptions. Efforts to promote safer opioid prescribing habits must address a broad swath of prescribers to be effective.

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CHAPTER WINNING ABSTRACT

## PREVALENCE OF HIGH NORMAL OR HIGH SERUM CALCIUM IN SECONDARY HYPERPARATHYROIDISM OF CKD

Lakshmi Ganesan<sup>2</sup>, Kevin Codorniz<sup>1, 2</sup>, and James McMillan<sup>1</sup> VA Loma Linda Healthcare System<sup>1</sup>, Loma Linda University Medical

**Introduction:** Chronic kidney disease (CKD) affects over 10% of the global population. These patients deal with the long-term consequences of renal failure, including dysfunction of the calcium-phosphate-parathyroid hormone (PTH) axis.<sup>1</sup> To control secondary hyperparathyroidism and prevent complications, current Kidney Disease Outcomes Quality Initiative (KDOQI) guidelines recommend administration of active vitamin D to patients with CKD stage 3 and 4 who have PTH concentration above target range for their stage of kidney disease, normal serum Vitamin D concentration, and serum calcium corrected for albumin  $>9.5\text{mg/dL}$ , and furthermore whether secondary hyperparathyroidism sufficiently explains their underlying disease. The purpose of our study was to determine the prevalence of corrected serum calcium  $>9.5\text{mg/dL}$  in a CKD population.

**Methods:** A retrospective, cross-sectional study was designed to determine the prevalence of high or high normal serum calcium in a subpopulation of patients with high PTH and CKD stages 3 and 4. A computerized list of patients at the Veteran Administration Loma Linda Healthcare System was generated with laboratory records between 04/2010 to 07/2010, showing eGFR  $15\text{ml/min}/1.73\text{m}^2$  and serum PTH  $>70\text{pg/mL}$ . Patients were excluded if they were on dialysis, had AKI, malabsorption, or if they were being treated with bisphosphonates, sorafenib/sunitinib, or calcitriol. This population was divided into high ( $>9.5\text{mg/dL}$ ) or low-normal ( $7.0$ ). 188 patients were excluded. Of the remaining patients (133), 81 patients had concomitant corrected hypercalcemia ( $9.5\text{mg/dL}$  or higher) resulting in a prevalence of 60.9% (81 of 133).

**Conclusions:** Secondary hyperparathyroidism in CKD can lead to significant long-term consequences if left untreated, including osteodystrophy, or vascular calcifications.<sup>1,3</sup> Current guidelines are unclear regarding treatment for patients with secondary hyperparathyroidism and corrected calcium  $>9.5\text{mg/dL}$ . Furthermore the pathophysiology of this finding is unclear. One of the possible explanations includes concomitant primary and secondary hyperparathyroidism. These findings could also signify a yet undescribed mechanism of secondary hyperparathyroidism. Our study showed that in a VA population, 60.9% of patients with CKD stage 3 or 4 and secondary hyperparathyroidism have a calcium  $>9.5\text{mg/dL}$  and may fall outside of current guidelines for treatment. This highlights the need for further study into this population of patients. We plan to continue examining the data, including patients who progress to hypercalcemia once Vitamin D and PTH goals are obtained.

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RACIAL/ETHNIC DIFFERENCES IN FALL PREVALENCE AMONG OLDER WOMEN

First Author: Yifan Geng, MD Second Author: Joan C Lo, MD Third Author: Nancy Gordon

**INTRODUCTION** Falls are the leading cause of fracture in older persons, and it is well known that risk of falls differs by age and other health factors. Fracture risk also increases with age and clinical risk factors, in addition to differences by race/ethnicity (higher for whites, lower for Asians). However, few studies have examined falls risk by race/ethnicity, particularly within contemporary U.S. populations with high representation of Asians. This study examines racial/ethnic predictors of prevalent falls among older women within a large northern California healthcare delivery system.

**METHODS** This cross-sectional study was conducted using data from women ages 65-90 years old who completed a Kaiser Permanente Northern California (KPNC) Member Health Survey (MHS) in 2008 or 2011. Self-reported information pertaining to age, race/ethnicity, health status, comorbidity (history of diabetes, stroke, arthritis), mobility limitations, and frequency of falls (1+ and 2+) within the past 12 months were examined. Prevalent fall rates for blacks, Hispanics and Asians were compared to white non-Hispanics (whiteNH), and the association of race/ethnicity and risk of falls was examined using multivariable logistic regression analyses (with odds ratio, OR and 95% confidence interval, CI), controlling for age, comorbidities, health status, and mobility limitations. Analyses used pooled respondent data weighted to the age-gender composition of the KPNC membership in 2011.

**RESULTS** There were 6338 women who completed the 2008 or 2011 survey, of whom 74.9% were whiteNH, 6.8% Hispanic, 7.3% black and 11% Asian (46% Chinese, 46% Filipino). Unadjusted prevalence of 1+ fall the past year was 28.4% for whiteNH, 27.7% for Hispanic, 23.3% for black, and 20.4% for Asian women. Statistically significant associations were found between fall risk and age (OR 1.04, CI 1.02-1.05), presence of diabetes (OR 1.24, CI 1.04-1.48), prior stroke (OR 1.66, CI 1.22-2.26), arthritis (OR 1.65, CI 1.43-1.90), self-reported poor health (OR 2.83, CI 1.94-4.14) and mobility limitation (OR 3.07, CI 2.58-3.66). After adjusting for age, comorbidity, poor health and mobility limitation, Asian (OR 0.65, CI 0.51-0.83) and black (OR 0.75, CI 0.57-0.99) women had a significantly lower odds of having had 1+ fall in the past year compared to whiteNH women. Asian women were similarly less likely than whiteNHs to have 2+ falls (adjusted OR 0.61, CI 0.43-0.87).

**DISCUSSION** Among older women, the adjusted odds of having a fall in the past year were one-fourth and one third lower for blacks and Asians, respectively, when compared to whiteNH women. This may contribute to the lower hip fracture rates observed in U.S. Asian and black women. While limitations of this study include the exclusion of non-English speaking patients and recall bias, future studies should examine disparities in fall risk and other clinical factors contributing to racial/ethnic differences in hip fracture incidence and outcome.

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IDENTIFY OPPORTUNITY TO IMPROVE CLINIC PERFORMANCE AND REDUCE PATIENT SERVICE TIME  
IN A COUNTY PRIMARY CARE CLINIC: A QUALITY IMPROVEMENT PROJECT

Tony Hung, MD, MBA Anna Shvartsur Steven Hadawar John Sy, MD Richard Tennant, MD

**Objective:** Identify opportunity to improve the clinic performance and reduce patient service time in a Los Angeles county hospital primary care clinic

**Study Design:** Operational process analysis

**Methods:**

1. Map out the critical processes for a clinic visit
2. Identify the key sources of arrival and service variability at these processes
3. Examine the patient flows through these processes to determine the process times
4. Calculate the capacity and utilization and identify potential bottlenecks
5. Identify how best to improve the clinic performance and reduce patient service time by reducing variability, improving capacity, and reducing utilization at each process

**Results:** A total of 64 patient visits were compiled for analysis. Four critical clinic processes were identified in the sequence: 1. Check-in 2. Vitals 3. Physician 4. Dispo. Total service time for each clinic visit averaged 88.73 minutes, of which 58.64 minutes contributed to wait time. An estimated 24.50 minutes of wait time were behind-the-scenes (unmeasured) processes, resulting in a total clinic process time of 54.59 minutes. Process time of Physician averaged 34.15 minutes, followed by Dispo (12.89 minutes), Vitals (5.33 minutes), and Check-in (2.22 minutes). Wait time was highest between Vitals and Physician (24.59 minutes), followed by wait time between Check-in and Vitals (17.61 minutes), and least between Physician and Dispo (16.44 minutes). Physician process was identified as the bottleneck, with the lowest capacity (10.54 patient - served per hour) and the highest utilization (75.88%). Significant wait time totaling 35 minutes was due to variability occurred prior to the Physician process. Variability included arrival of patient time, interruptions of LVNs for translation, complexity of patients, room assignment and availability, printer malfunctioning, and multiple order forms. Wait time after Physician process was primarily due to behind-the-scenes processes (13.43 minutes). Behind-the-scenes processes included writing orders, printing prescriptions, processing orders, scheduling and documentation.

**Conclusions:** Opportunities to reduce variability, improve capacity, and reduce utilization are identified. System-wide proposal to target the reduction of variability include enforcing on-time appointment policy, establishing volunteer translators program, improving allocation of room within the clinic, scheduling regular check-up for printers and simplifying ordering forms. Establishing a dedicated health care coordination program is proposed to offload the bottleneck to target behind-the-scenes processes by assisting with writing orders, printing prescriptions, processing orders and reviewing treatment plan. Potential of a total 48.43 minutes of service time can be reduced to improve performance in the clinic.

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CHAPTER WINNING ABSTRACT

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IMPROVING DOOR-TO-NEEDLE (DTN) TIME FOR TISSUE PLASMINOGEN ACTIVATOR (TPA) ADMINISTRATION FOR ACUTE ISCHEMIC STROKE IN COMMUNITY-BASED HOSPITAL BY INTERNAL MEDICINE RESIDENCY PROGRAM

First Author: Reza Khosravani Goshtaseb, MD Second Author : Syung Min Jung, M.D

**Background:** In January 2013, the Internal Medicine (IM) residency program at San Joaquin General Hospital launched a 'stroke team' using tele-neurology service to provide timely tPA administration for patients with acute ischemic stroke. In patients with acute ischemic stroke, DTN time less than 60 minutes for tPA administration and early endovascular intervention have shown better clinical outcomes.

**Purpose:** Quality improvement project was initiated to shorten DTN time for tPA administration.

**Project Description:** We formed a multidisciplinary team comprised of internal medicine residents and attendings, emergency department (ED) physicians and nurses, emergency medical services (EMS) staff, radiology staff and the tele-neurology service. The team met monthly to review all the stroke alert cases and implemented Plan, Do, Study, Act (PDSA) model for any areas for improvement and ongoing monitoring.

**Project Details:** The main areas that were identified for improvement included: 1) Collaboration with EMS to improve pre-notification, 2) Implementation of our 'stroke card' for EMS, 3) Direct transfer of patients from ambulance to CT, 4) Improving tele-neurologist response time, 5) Capturing all stroke signs for walk-in patients, and 6) Real-time feedback with the multidisciplinary team members. Serial meetings and educational sessions took place with our county-wide EMS to improve the pre-notification rate. A 'stroke card' was created and introduced to the EMS as a more effective communication tool. Multiple educational sessions were held with the ED and EMS to take patients directly to the CT from the ambulance. Serial teleconferences were held with our tele-neurology service to shorten their video response time. Hospital-wide nursing education sessions were done for early recognition of stroke symptoms. We also initiated real-time feedback within our multidisciplinary team via our secure text system and email.

**Results:** Data was gathered from 63 with a diagnosis of acute ischemic stroke from January 2013 to August 2015. Our EMS pre-notification rate improved from 60% in 2014 to 77% in 2015. Now nearly 100% of our pre-notified stroke patients are taken to the CT directly from the ambulance. The revised tele-neurology triage system has shortened video response time significantly for potential tPA cases. Overall, the percentage of tPA administration within 60 minutes of arrival improved from 53% in 2013 and 63% in 2014 to 93% in 2015.

**Conclusion:** We improved our tPA DTN time remarkably via this multidisciplinary approach. As a result of this project, there also has been valuable time saved in the transfer of our 'drip and ship' patients to the endovascular centers. The IM residency program in our community-based hospital, which does not have a full-time neurologist, was nonetheless able to significantly improve the quality of stroke care through the concerted, systematic, and collaborative efforts.

MEDICATION REFILL MANAGEMENT AND EDUCATION IN AN INTERNAL MEDICINE RESIDENT CLINIC

Stephanie Kim MD MPH, Joan Lo MD, Leslea Brickner MD FACP, Mary Patton MD FACP

**INTRODUCTION:** Medication refills are an important part of primary care practice and directly affect patient outcomes and satisfaction. Recent data show that medications are prescribed at 75% of all office visits nationwide, with more than two-thirds of prescriptions representing refills. Consistent prescribing practices and timely refills are important aspects of resident education in prescription management, especially with remote electronic refills. Inherent challenges of handling refills in residency programs include more frequent provider turnover and multiple provider coverage. We evaluated the impact of an educational intervention on residents' understanding of and comfort level with the medication refill process.

**METHODS:** This project was conducted in the Kaiser Permanente Oakland Internal Medicine Resident Clinic over a 5-month period (January-May 2015), where residents rotate one-half day per week and care for 100-150 panel patients. The educational intervention comprised of a reference card provided to residents detailing recommended follow-up (labs and appointments) for 8 commonly refilled medication classes, accompanied by a brief pre-clinic tutorial facilitated by an attending physician. Residents were also asked to complete pre- and post-intervention surveys assessing (1) knowledge of program-specific refilling practices (understanding of pharmacy symbols to differentiate high and low priority refill requests and coordination of care with clinical pharmacists); (2) behaviors that include chart review of recent labs and appointments upon receiving a refill request; and (3) comfort level regarding the medication refill process and follow-up. The post-intervention survey also included a question evaluating the helpfulness of the intervention.

**RESULTS:** Among 32 internal medicine residents, 29 (90.6%) participated in the intervention (27.6% PGY-1, 37.9% PGY-2, 34.5% PGY-3). Pre- and post-intervention surveys were completed by 23 (71.9%) and 27 (84.4%) residents, respectively. At baseline, there was variable understanding of pharmacy refill request symbols (representing high and low priority refill requests), which improved by 27% post-intervention. Most residents indicated they reviewed recent patient labs (95.7%) and appointments (91.3%), with little change observed post-intervention (100% and 88.9%, respectively). Resident comfort level with lab monitoring when refilling medications improved across all PGY levels, from 52.2% to 81.5%, demonstrating a 29.3% increase overall and 52.9% increase for PGY-1 residents. The intervention was well-received and resident comments were uniformly positive.

**CONCLUSION:** While there was initial variability in resident knowledge of refill request symbols and comfort level with lab monitoring when refilling medications, overall improvement was seen post-intervention, particularly for interns. These findings demonstrate the utility of a targeted educational intervention to improve resident management of prescription refills, especially early in training. Our intervention was easy to implement and may be a useful approach adaptable to other residency programs. Future directions include implementing an annual intervention program with evaluation for sustained effects on resident knowledge, behavior and comfort level, as well as patient-centered outcomes.

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“SMOKE AND BUBBLES”: CAN ULTRASOUND FINDINGS GUIDE CARDIAC RESUSCITATION?

Pamela Lam, DO Kim Phan, DO Bruce Kimura, MD

Although commonly used in emergency medicine to diagnose the etiology of hypotension or dyspnea, few data exists on the integration of bedside ultrasound techniques into resuscitation of cardiac arrest. During a “code blue,” the effectiveness of ACLS is mainly evaluated through assessment of vital signs during pulse and rhythm checks every 2 minutes, as per protocol. Due to time and space constraints imposed by a code situation, the use of bedside ultrasound is challenging but could provide diagnostic and prognostic information to guide resuscitation.

We describe a case of a pulseless electrical activity (PEA) arrest in which a pocket-sized ultrasound device was used during ACLS-guided resuscitation. A 66 year-old gentlemen with severe aortic stenosis and left ventricular dysfunction suffered a PEA arrest while sedated for a transesophageal echocardiogram. Initial images during an agonal rhythm were best afforded by the subcostal window, which is usually performed to detect tamponade and assess ventricular function. In our case, an echogenic or “smoke-filled” blood pool was seen in an acontractile heart. In animal models of cardiac arrest, blood pool echogenicity was seen to increase in acoustic intensity during the progression to asystole, and in small clinical studies, such “smoke” was associated with adverse outcomes. With the initiation of chest compressions in the patient, subcostal imaging became technically difficult and imaging was only feasible during pulse and rhythm checks. During the first pulse check, asystole was confirmed and the previous sign of smoke had cleared, suggesting that stasis had been improved through effective chest compressions. Later when PEA developed, imaging demonstrated echogenic “bubbles” within the right heart, the inferior vena cava, and hepatic veins, most easily attributed to the multiple intravenous injections given during the code. The bubble finding suggested patency of the antecubital intravenous access and rapid drug delivery to the right heart. However, the presence of bubbles in the inferior vena cava could represent reflux into the abdominal venous system during chest compressions, resulting in only partial dosing efficacy. The patient had return of spontaneous circulation after 8 minutes and was transferred to the ICU. Despite a trial of hypothermia, the patient never regained consciousness and died 5 days later.

In critically-unstable patients, studies support ultrasound be employed for rapid diagnosis but have not described utility once ACLS has begun. The case presented demonstrated novel ultrasound findings obtained by a pocket-sized device that can potentially assist any resuscitative effort by providing initial prognostication and allowing monitoring of the efficacy of chest compressions and the delivery of intravenous therapies. Future studies are needed to address the value of incorporating portable ultrasound techniques into ACLS to assess fundamental goals common to all resuscitative efforts.

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PATIENT-CONTROLLED ANALGESIA IN CANCER-RELATED PAIN: CLINICAL PREDICTORS OF PATIENT OUTCOMES

First Author: Emily Martin, MD Additional Authors: James Murphy, MD; Madison Sharp; Carolyn Revta; Eric Roeland, MD; Heidi Yeung, MD

**Introduction:** Patient-controlled analgesia (PCA) is widely used in the inpatient setting for uncontrolled pain and is well-studied in the post-operative setting. Data regarding its use for patients admitted with cancer-related pain, however, is limited.

**Research Objectives:** The purpose of this study is to define the patterns of PCA use and related outcomes in a retrospective cohort of patients admitted to the hospital with cancer-related pain.

**Methods:** We identified 90 patients admitted to a single academic center with a cancer diagnosis who received PCA for non-surgical, cancer-related pain and who survived to discharge between January 2013 and January 2014. Data including patient demographics, type of cancer and pain, time course from admission to PCA initiation to PCA discontinuation to discharge, opioid-specific adverse events, and 30-day readmission rates for pain were collected. Univariable and multivariable linear regression models were used to look for any association between patient and clinical variables with PCA duration. Logistic regression models were used to evaluate the relationship between patient and clinical variables and 30-day readmission rates.

**Results:** The median length of hospitalization was 10.15 days with a median PCA duration of 4.40 days. Hematologic tumors were associated with longer PCA use ( $p=0.0001$ ), as was younger age ( $p=0.032$ ). There was a borderline significant trend towards decreased 30-day readmission rates with longer PCA use ( $p=0.054$ ). No correlation was found between 30-day readmission and any covariate studied including palliative care consult, sex, pain type, cancer type (solid vs. hematologic), age, or time from discontinuation of PCA to discharge.

**Conclusion:** This study found that in patients admitted with cancer-related pain, there is longer PCA use in younger patients and in patients with hematologic malignancies, with a trend towards decreased 30-day readmission rates in those with longer PCA use.

**Implications for Research, Policy or Practice:** Future research is needed to identify subsets of oncology patients that will benefit the most from PCA use.

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LUNG HYPERINFLATION IS ASSOCIATED WITH PULMONARY EXACERBATIONS IN ADULTS WITH CYSTIC FIBROSIS

Kosal Seng, Lynn Fukushima, Adupa Rao, Joseph Milic-Emili, Ahmet Baydur

**Background:** Forced expiratory volume 1 second (FEV<sub>1</sub>) has traditionally been used as a readily available marker of health in adult cystic fibrosis (CF). However, due to the obstructive nature of this disease, it is possible that lung hyperinflation could be more closely related to disease severity than is FEV<sub>1</sub>. The purpose of this study was to determine if hyperinflation is more closely associated with quality of life, functional status, and pulmonary exacerbations than FEV<sub>1</sub> in patients with CF.

**Methods:** Sixty-eight adult patients with CF were evaluated in this retrospective study. We used IC and functional residual capacity (FRC) and their ratios to total lung capacity (TLC) as measures of lung hyperinflation. We used bivariate correlations and backwards regression analysis to assess possible associations between FEV<sub>1</sub>, lung hyperinflation, and measures of disease severity including questionnaire based quality of life, pulmonary exacerbation frequency, and mortality. The respiratory component (CFQ-R) of the Cystic Fibrosis Questionnaire **was** used as a measure of quality of life.

**Results:** Both FEV<sub>1</sub> and IC were negatively correlated with pulmonary exacerbations over a 3 year period (p=0.002, p<0.001 respectively), while FRC/TLC correlated positively with exacerbations (p=0.004). Backwards regression analysis showed that among pulmonary function variables, IC had the strongest relationship with exacerbations over 3 years ( $r^2 = 0.331$ , p<0.001). A lower CFQ-R score was associated with greater mortality (p=0.007). However, no statistically significant relationships were found between lung function and mortality.

**Conclusions:** FEV<sub>1</sub> and lung hyperinflation - as measured by IC and FRC/TLC - are both associated with pulmonary exacerbation frequency. This suggests that chronic dynamic hyperinflation contributes significantly to disease severity in adult cystic fibrosis.

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I-PASS- IMPROVING RESIDENT HANDOFFS – A QUALITY IMPROVEMENT INTERVENTION AT A COMMUNITY-BASED INTERNAL MEDICINE RESIDENCY PROGRAM

Sneha Sundaram, Aaron Losey, Carie Chin-Garcia, Ingeborg Schafhalter-Zoppoth

**Introduction:** Effective communication is a key component to patient safety. Educational interventions can improve handoffs. The most well studied handoff bundle, I-PASS (illness severity, patient summary, action items, situation awareness and contingency planning, synthesis by receiver), has been associated with reductions in medical errors and adverse events. Using a quality improvement PDSA (plan-do-study-act) framework, we piloted the I-PASS system in our residency program.

**Methods:** In the PLAN phase, the current state of night handoffs was assessed over five evenings. Key components of verbal and written handoffs (I-PASS items, code status, surrogate decision maker, medications to avoid) and qualitative perceptions on safety and quality of handoffs were collected. We created tools to implement I-PASS, evaluated obstacles, and formulated our hypothesis: our interventions would increase adherence to critical handoff components to at least 50%. The DO phase was an I-PASS educational initiative introducing EMR smart phrases to standardized inclusion of critical elements in written handoffs. The STUDY phase one month later included handoffs observation for five nights with subsequent morning interviews. Critical components of handoffs before and after I-PASS were compared. In the ACT phase, we evaluated I-PASS robustness and obtained feedback via survey.

**Results:** In the PLAN phase, 200 verbal and 229 written handoffs were observed, compared to 235 verbal and 247 written handoffs in the STUDY phase. For verbal handoffs, I-PASS increased mentioning of illness severity (16.5% vs 44.7%,  $p < 0.0001$ ) and synthesis by the receiver (3.5% vs 31%,  $p < 0.0001$ ). Amount of time required to hand off each patient did not increase significantly (1.2min vs 1.3min,  $p = 0.546$ ). For written handoffs, inclusion of the following parameters significantly increased: illness severity (3.1% vs 49%,  $p < 0.0001$ ), action list (54.6% vs 83.4%,  $p < 0.0001$ ), and medications to avoid (17.9% vs 31.5%,  $p = 0.0007$ ). There was no significant difference in the number of patients for which the intern reported needing better sign out or confidence in taking care of patients. Education on I-PASS was not thought to change the subjective quality of handoffs. The majority of residents reported using I-PASS for verbal sign out and smart-phrases for written sign out immediately post-intervention (73% and 69% respectively) and 10 months later (77% and 92%).

**Conclusions:** In this quality improvement project, use of the I-PASS handoff bundle was associated with increased inclusion of key components of verbal and written handoffs without prolonging handoff time. I-PASS is now used as the established handoff tool in our residency program. Ten months later, use of I-PASS is sustained. Currently, the effect on medical errors and adverse events is unknown. Future projects are planned to further improve patient handoffs.

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PREVENTION OF REPERFUSION-INDUCED CARDIAC INJURY IN ACUTE MYOCARDIAL INFARCTION THROUGH REDUCTION OF OXIDATIVE STRESS

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**Introduction:** Acute Myocardial Infarction (AMI) is the first cause of death worldwide. Its treatment consists in the recovery of coronary flow by Percutaneous Angioplasty (PCA). The sudden arrival of oxygen to the previously ischemic myocardial tissue and the iron mobilization produces Reactive Oxygen Species (ROS), causing reperfusion-induced cardiac injury. Antioxidants such as ascorbic acid (AA), N-acetyl cysteine (NAC) and deferoxamine (DFO) may prevent reperfusion-induced cardiac injury.

**Methods:** Randomized, placebo-controlled clinical trial in patients with AMI treated with PCA, which were administered high doses of AA (320 mmol/L) or placebo intravenously previous to myocardial reperfusion. In Langendorff model we made a model of myocardial ischemia-reperfusion with crescent concentrations of AA, NAC and/or DFO.

**Results:** There was a significant increase of ejection fraction between 2 cardiac MRI in the group that received vitamin C, compared to placebo ( $p < 0.05$ ). The heart perfused with NAC-DFO and AA-NAC demonstrated to have the highest pressure at 30 and 40 minutes of reperfusion, respectively.

**Conclusion:** High doses of antioxidants administered previous to myocardial reperfusion induced by PCA, may attenuate reperfusion-induced cardiac injury.

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RISK FACTORS ASSOCIATED WITH SEVERE HYPOGLYCEMIA IN HOSPITALIZED PATIENTS AT BANNER UNIVERSITY MEDICAL CENTER – PHOENIX

MJ del Rosario, ER Maharam, C O'Malley, V Chockalingam

**Introduction:** Hypoglycemia is a common (25%) occurrence in patients admitted to the hospital who have diabetes. Hypoglycemia is associated adverse outcomes, including increased healthcare cost, morbidity and mortality. A direct cost of \$68 and total cost of \$2638 per case of hypoglycemia in the hospital has been demonstrated in recent cost-analyses. With mortality and cost-benefit, there is incentive in identifying and ultimately decreasing events.

Our facility in particular, Banner University Medical Center – Phoenix (BUMC-P), ranks among the bottom quartile of hospitals according to recent reports released by Society of Hospital Medicine. Improving this rate could convey both benefits in medical outcomes as well as decreased costs for our facility. Previous studies have identified various risk factors associated with hypoglycemic events including age, renal impairment, large daily insulin dosing and type of insulin regimen, but the population at BUMC-P is unique and warrants independent study. Identifying and educating clinicians regarding these risks factors may assist in decreasing our facility hypoglycemia rate to or below the national average.

**Methods:** Our group has conducted a small retrospective review of inpatient hypoglycemic cases (defined as blood sugar < 70). Cases (n=55) of hypoglycemia are flagged and evaluated by physicians specializing in internal medicine and endocrinology. Reviewers indexed suspected risk factors including: age, degree of hypoglycemia, weight, body mass index, presence of diabetes diagnosis including type, liver disease, sepsis, renal impairment, prior hypoglycemic events, intravenous fluids, glycemic control regimen and dosing, diet, whether insulin dosing was changed in the day preceding and how, notes, and an assessment of cause (clinician judgment). Our control group consisted of age, sex-matched hospitalized patients with diabetes that did not experience a hypoglycemic event.

**Results:** Final statistical analysis (using multivariate statistical analysis including chi-square testing) revealed a significant ( $p < 0.001$ ) proportion of studied events occur (56%) during the hours between 0200 and 0800. In this sample, patients who experienced hypoglycemia weigh significantly less than control samples without hypoglycemia (71.65 vs. 95.5). Additionally, the basal insulin dose per kilogram was statistically different between groups (0.39 u/kg vs. 0.21 u/kg).

**Conclusions:** While further data collection and analysis is ongoing, our data shows that a disproportionate of events occurred during early morning hours, in patients with lower body weight and those who received larger doses per kg of basal insulin. These findings suggest an over-reliance of basal insulin to control inpatient blood glucose. Strategies to curb inappropriate use of basal insulin may reduce the incidence of hypoglycemia in our institution.

THE NICOTINIC ACID ANALOG, ACIPIMOX, INCREASES EXERCISE CAPACITY IN OVERWEIGHT OR OBESE SUBJECTS WITH TYPE 2 DIABETES

First Author: Lauren N Dise, MD PhD Additional Authors: Leah Herlache MS, Katie Rogers MS, Jane E.B. Reusch MD, Judy G Regensteiner PhD, and Irene E. Schauer MD PhD

Obesity and type 2 diabetes (T2D) are associated with insulin resistance, elevated free fatty acid (FFA) levels, mitochondrial dysfunction, and decreased exercise capacity ( $VO_2$ peak). Acipimox, a nicotinic acid analog and NAD<sup>+</sup> precursor, has been shown to decrease FFA levels, acutely improve insulin sensitivity and, recently, to improve mitochondrial function. We hypothesized that acipimox would improve exercise capacity in T2D. Overweight or obese subjects with T2D (n=6, age  $54.9 \pm 7.8$  years, BMI  $31.2 \pm 5.4$  kg/m<sup>2</sup>) were enrolled in a double-blind, random-order, placebo-controlled, crossover study of 7-9 days of treatment with acipimox/placebo. Acipimox treatment reduced fasting baseline  $VO_2$  ( $3.08 \pm 0.58$  vs  $3.49 \pm 0.48$ , p=0.034), increased peak  $VO_2$  during maximal exercise testing ( $19.7 \pm 3.1$  vs  $18.2 \pm 2.6$  ml/kg/min, p=0.006), and increased work load capacity at anaerobic threshold ( $75.8 \pm 22.9$  vs  $65.7 \pm 26.3$  watts, p=0.034). There was no significant difference in steady state  $VO_2$  during exercise at 85% of baseline anaerobic threshold ( $11.2 \pm 2.1$  vs  $11.4 \pm 1.9$  ml/kg/min, p=0.237, n=6). As the tachyphylaxis to FFA-lowering occurred earlier than previously reported, acipimox did not consistently reduce FFA levels ( $764.2 \pm 500.3$  vs  $605.8 \pm 113.2$  uEq/L, p=0.448) or improve insulin sensitivity by hyperinsulinemic euglycemic clamp (M-value:  $6.00 \pm 1.52$  vs  $6.04 \pm 0.69$  mg glucose/kg lean body mass/min/uIU/mL insulin x 100, p=0.940). Triglyceride levels were reduced by acipimox ( $99 \pm 33$  vs  $134 \pm 43$  mg/dl, p=0.02). These results suggest that acipimox improves exercise capacity, and that the improvement is not acutely dependent on FFA levels or insulin sensitivity. Increased  $VO_2$ peak may occur through improved mitochondrial function independent of FFA levels and may therefore be unaffected by tachyphylaxis to FFA suppression and potentially clinically significant.

CRITICAL HYPOGLYCEMIA IN HOSPITALIZED PATIENTS WITH DIABETES MELLITUS: ROOT CAUSE ANALYSIS AND INTERVENTIONS TOWARD STANDARDIZED REPORTING AND MANAGEMENT.

**All Authors:** Katherine Araque, MD, Deepak K. Kadayakkara, MD, Nino Gigauri, MD, Diane Sheehan, APRN, Sachin Majumdar, MD, Kasia Lipska, MD, Gregory Buller, MD, Clare Flannery, MD

**Introduction:** Critical hypoglycemia is associated with increased mortality in hospitalized patients and one year after discharge. We performed a retrospective chart review and root cause analysis of hypoglycemia events (HGE) in our hospital. We identified system based contributions to the event rate, and initiated interventions to improve the system.

**Methods:** Pre-intervention phase: A retrospective, observational study of HGE, defined as glucose less than 50 mg/dl, occurring in patients with diabetes mellitus (DM) admitted to two inpatient wards, from August through October 2014. Data were collected on patient demographics, HGE risk factors, post-event treatment, MD notification, and insulin dose changes. Calculated measures included time to recovery from HGE (defined as BG  $\geq$ 100 mg/dl) and time from HGE to next finger stick. Descriptive statistical analysis and root cause analysis were performed. Intervention phase: Interventions included education to health care providers at all levels, modification of electronic medical record order sets and creation of protocol for critical hypoglycemia and standardization of treatments Post-intervention phase: Repeat observational study of HGE in same two inpatient wards, from July to December 2015.

**Results:** In pre-intervention phase, 35 HGE occurred in 22 patients over 3 months. Standardized treatment was received only in 34% of HGE. Median time to recovery after HGE was  $225 \pm 46$  min, and from HGE to next finger stick was  $76 \pm 14$  min. Incidence of HGE was 7.3% of the total inpatient admissions in the two wards. 37% of events were recurrent hypoglycemia. 37.1% of the events had no treatment documented. When treatment was documented, treatments were not standardized. MD notification occurred only in 51.4% of the HGE. During the first three months post-intervention, 30 HGE occurred in 20 patients. 100% of the events were treated, and 90% of the patients were treated per protocol with either glucose gel or 50% dextrose injection. MD notification occurred for 77% of the HGE. In this initial dataset, median time to recovery is  $87 \pm 30$  min, and mean time from HGE to next finger stick was  $26 \pm 11$  min.

**Discussion:** In the pre-intervention phase, we identified the burden of HGE by sampling two hospital wards with the highest incidence of HGE over 3 months. In a preliminary data collection post-intervention, there is notable improvement in the targeted responses. Data collection continues for another 3 months, prior to final analysis. With this analysis, we anticipated a refinement of the interventions. The overall goal of this study and intervention is to improve patient safety by improving the treatment of critical hypoglycemia and reducing recurrent events.

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## THE PRIVACY CONCERNS OF ONCOLOGY PATIENTS IN USING TELEMEDICINE

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**Background and Objective:** Over the past decade, telemedicine has gained more attention for delivering care for patients in the rural areas, with comorbid conditions, or in need of more intense monitoring. Cancer patients undergoing active treatment may develop symptoms that traditional healthcare delivery may fail to fully address. Although telemedicine seems promising in the oncology setting, less is known about patients' concerns and attitude toward using telemedicine.

Our objective is to determine the level of privacy concern by cancer patients, and its correlation with patients' socio-demographics and attitudes toward telemedicine in the healthcare setting.

**Methods:** This is a cross sectional study conducted at Memorial Sloan Kettering Cancer Center, NY, US. After literature review, we have developed a **modified Technology Acceptance Model** questionnaire for cancer patients. The questionnaire has 4 domains of perception of social benefit, healthcare benefit, risks and barriers, and cues to action, with total of 21 items. Patients were asked to state their level of agreement with each item on a 5-point Likert scale. For the purpose of this study, answers were dichotomized into strong agreement and less than strong agreement for items on social, healthcare benefit, and cues to action domains. Answers were dichotomized into strong disagreement and less than strong disagreement for items pertaining to the risks and barriers domain. An additional item assesses patients' prior experience with videoconferencing applications. Chi Square analysis was used to assess the correlation between privacy concern and other items.

**Results:** Out of 191 cancer patients, only 47 (24.6%) were in strong disagreement that using videoconferencing applications may violate their privacy. Those with strong disagreement were more likely to agree to videoconferencing than those with less than strong disagreement (61.7% vs. 37.5%,  $P=0.004$ ). The concern over privacy had statistically significant correlation with female gender ( $P=0.04$ ), & lower income ( $P=0.012$ ). Prior experience with these applications had had negative correlation with privacy concerns ( $P<0.001$ ). Patients with positive attitude toward social benefit of these applications were less likely to have concern over privacy. More patients with privacy concerns thought that these applications are difficult to use, that they may make mistakes when using these applications, and that they would require assistance in using these apps as well as tech support. More patients without privacy concerns had somebody at home, or someone among their friends, or family members, using these apps. Also, they were more likely to think many people in general, and many people of their age, are using these apps. Those without privacy concern were also more likely to have positive attitude toward the potential healthcare benefit of these apps.

**Conclusion:** Despite the promise of telemedicine in the oncology setting, a significant majority of patients have some level of concern over their privacy when using these applications. This correlation was predominantly demonstrated in females of lower income status. Future studies in this setting should address the roots of patients' concern over privacy, in order to successfully implement a telemedicine program in the oncology setting.

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CHAPTER WINNING ABSTRACT

Pamela Bailey, DO Mark LaSorda, DO Laura Quint, MBBS Loretta Consiglio-Ward, MSN, RN Jhenette Lauder, MD Leah Milton, PharmD Bridget Ryan, RN LeeAnn Tanaka, DO Anthony Tascone, MD

**Introduction:** As medical providers, we have stood at the bedside wondering if what we were doing is the *right* thing for our patients. However, due to reasons like awkwardness, reticence to initiate the conversation and fear of offending patients, physicians hesitate to have discussions regarding what patients want at the end of life.

Within our electronic medical record (EMR), caregivers are meant to document a patient's wishes regarding their care in a 'Goals of Care' (GoC) note. We surveyed hospital staff's perceptions of the intended meaning of 'goals' and planned an intervention to increase GoC documentation to 25% on one inpatient unit, as an attempt to improve patient satisfaction.

**Methods:** A multidisciplinary group developed a survey that was sent electronically to physicians, nurses, social workers, case managers, and physical therapists across the hospital. We developed a short intervention to include GoC on a nursing rounding checklist to improve documentation of GoC conversations. The intervention required a patient's nurse to assess whether the patient's goals were addressed, and if not, to query the physician regarding GoC conversations and their documentation. After the two week period of having the nurses make this assessment, we did an analysis via the EMR whether patients had a 'GoC' document recorded.

**Results:** Of 292 survey respondents, 209 (77%) accepted as part of their job responsibilities to discuss goals of care, while 26 (10%) responded 'no' and 36 (13%) were 'not sure.' The vast majority (245; 91%) felt that physicians should be responsible for conducting and documenting GoC discussions, while majorities also replied nurses (163; 61%) and social workers (159; 59%). Other responses included clergy and physical therapy. The average provider comfort level in discussing GoC with patients/caregivers was 3.68, between 'comfortable' and 'fairly comfortable.' A minority (114; 43%) knew where to document GoC discussions in the EMR.

We placed a 'GoC addressed?' on the nursing checklist on one inpatient unit, and the GoC documentation increased from 1% to 22% of all admissions to that unit over a two week period.

**Discussion:** In 2014, the Institute of Medicine's report *Dying in America* made the recommendations that clinician-initiated conversations regarding advance care planning need to be integrated into the plan of care, and that clinicians must have meaningful dialogue regarding patients' values, care goals, and preferences related to serious illness.

As medical care moves towards a patient centered model, goals of care discussions will become even more vital for increasing patient satisfaction. Appropriate documentation of the patient's goals can improve overall communication between all team members and eliminate excess cost from unwanted interventions.

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PROVIDER PREFERENCES IN THE MANAGEMENT OF OSTEOARTHRITIS IN PATIENTS WITH CHRONIC KIDNEY DISEASE.

First Author: Alyssa Mathew, DO Second Author: Amit Patel, MD Third Author: Edward Ewen, MD

**Background:** Osteoarthritis (OA) is common, affecting over 27 million individuals aged 25 and over in the United States, accounting for over 25% of arthritis-related ambulatory office visits to healthcare providers annually. The American College of Rheumatology (ACR) currently recommends acetaminophen and NSAIDs as initial pharmacological treatment. Due to predilection of disease to an older population and increasing lifespan of adults, treatment options become limited because of multiple comorbidities. One major obstacle includes use of NSAIDs in patients at risk for developing chronic kidney disease (CKD). We examined provider preference in management of patients with osteoarthritis in the presence of CKD and other comorbidities with the hypothesis that as glomerular filtration rate (GFR) declines the use of NSAIDs will also decline.

**Methods:** We performed a retrospective cohort analysis of all patients age 18 and older seen in a 5 year period in 19 primary care practices. All demographic information, clinical observations, and office visit activity were obtained from a shared electronic health record. OA was defined as an active diagnosis of osteoarthritis on problem list. CKD was defined by calculated GFR using the CKD-Epi equation. OA patients managed outside primary care setting were excluded from analysis. NSAIDs were defined as both topical and oral agents. Outcomes measures included the correlation of CKD stage with management of OA with NSAIDs compared to opioids. Outcomes and description variables were compared using Chi-square tests in the matched population as appropriate.

**Results:** Of 5316 eligible patients with both OA and at least CKD stage 3, 68.1% were female with average age of 67.1 +/-11.9 years. Nearly one-fifth (18.5%) of the population had CKD stage 3 or greater. Patients with a CKD stage of 3b or greater were significantly less likely to be on NSAIDs (74.4% vs. 25.6%,  $p < 0.001$ ). Statistically significant increase in the use of opioids occurred as GFR decreased, again most notably from CKD stage 3b through CKD stage 5.

**Conclusion:** There is a strong direct correlation between estimated GFR and NSAID prescription by primary providers, however, it first appears in relatively advanced renal disease. Interestingly there was just as strong of an indirect correlation between GFR and prescription of opioids. Limitations of this study included our inability to determine the reason for these prescriptions, and possible documentation errors. We did not investigate the use of topical NSAIDs or physical therapy due to data ascertainment limitations. Future directions will include evaluation of osteoarthritis management in light of other co-morbidities including diabetes and hypertension, as these diseases are known to accelerate renal dysfunction.

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ADDRESSING OBESITY IN A PRIMARY CARE RESIDENT TEACHING PRACTICE

N. Pearl Philip MD, Ed Ewen M

**Introduction:** The public health challenge posed by obesity is costly: 46% increase in inpatient costs, 27% increase in outpatient costs, and 80% increase in spending on prescription drugs, amounting to \$147 billion to nearly \$210 billion a year according to current estimates. Despite these facts, physicians often do not engage obese patients in discussions about their weight. This retrospective cross-sectional study aims to characterize prevalence and impact of obesity in a primary care resident teaching practice, and to determine the extent to which recognition of obesity as a medical problem is associated with weight loss.

**Methods:** Using data extracted from the outpatient electronic medical record (EMR), we identified the prevalence of obesity defined as BMI >30 in a primary care resident teaching practice. We calculated BMI based on most recent recorded weight and height. We compared documentation of obesity on a patient's problem list to prevalence of obesity based on BMI, and reviewed charts to determine whether weight loss discussions had occurred. We used Chi-square analysis to determine the association of obesity (as defined by patient BMI) with co-morbidities, and Mann-Whitney U Test to evaluate the frequency of weight loss discussion during the office visit within the past year among patients who did or did not carry a listed diagnosis of obesity. For each of these groups, we randomly selected 100 patients. Paired t-test was used to evaluate the change in BMI over the past year within each group.

**Results:** Of 3,111 active patients in the last year, 1525 (49%) had BMI =30. Among all patients with BMI >30, only 29% had obesity documented as a problem within the EMR. BMI =30 was significantly associated ( $p<0.05$ ) with the following co-morbidities: type II diabetes, hypertension, stroke, asthma, dyslipidemia, low back pain, osteoarthritis, musculoskeletal injury, fibromyalgia, gout, non-gout crystal arthropathy, obstructive sleep apnea, and behavioral health disorders. Physicians discussed weight loss during 41% of visits when obesity was documented on patients' problem lists compared to during 23% of visits in obese patients who did not have obesity documented ( $p=.0003$ ). However, neither group demonstrated significant weight loss ( $p<0.05$ ).

**Conclusion:** Although the prevalence of obesity in this outpatient practice surpassed the national average obesity rate of 34.9% in 2014, and despite many co-morbidities, resident physicians are not adequately recognizing or addressing the disease of obesity. Even though documentation of obesity was associated with increased discussion of weight loss, a significant difference in BMI did not result. These findings suggest that much still needs to be done to improve obesity interventions in this setting, possibly including more effective resident training as well as the development of effective obesity treatments.

## 5-COMPONENT BUNDLE FOR CLOSTRIDIUM DIFFICILE PREVENTION

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**Background:** Clostridium difficile infection (CDI) is a common cause of harm in hospitals. At our institution, the estimated cost of harm in 2013 was \$1.4 million. Risk factors for CDI include antibiotic use, gastric acid suppression, increasing age, hospitalization, serious co-morbidities, and prior CDI. In the first 3 months of 2014, hospital-onset (HO) CDI incidence increased, while other hospital acquired conditions decreased. Our community 9-bed medical-surgical ICU adopted a CDI prevention bundle with the goal of a 25% reduction in HO-CDI over a 6 month period and sustain it throughout the fiscal year.

**Methods:** The CDI bundle included 5 components: (1) environmental cleaning surveillance with; (2) improving staff awareness of CDI rates and adenosine triphosphate (ATP) swabbing results; (3) hand hygiene compliance above 90%; (4) antibiotic stewardship; and (5) proton pump inhibitor (PPI) stewardship. We conducted environmental surveillance by measuring ATP relative light units (RLU), with RLU below 45 considered clean of various high-touch items in ICU rooms. Clinical pharmacists conducted antibiotic and PPI stewardship through in-service training of providers and system wide emphasis on documentation of antibiotic stop dates. For hand hygiene, we posted reminder signs and covertly monitored hand washing. The intervention began in April 2014. We defined HO-CDI per standard definitions and calculated rates per 10,000 patient-days, and a rate-ratio between pre-intervention (Apr 2013-Mar 2014) and post-intervention (Apr 2014-Mar 2015) periods.

**Results:** Environmental cleaning surveillance was done on 35 items; results ranged from a bedside table at 9 RLU to a patient remote at 910 RLU. Hand hygiene compliance was maintained at 95%. The amount of commonly prescribed antibiotics decreased from 369.9 to 262.4 per 100 patient-days (29.2%) and total antibiotic doses decreased from 1805 to 1527 (15.7%). PPI use decreased from 79.9 to 62 per 100 patient-days (22.4%). HO-CDI decreased from 8 cases (40.0 cases/10,000 patient-days) pre-intervention to 2 cases (9.0 cases/10,000) post-intervention (rate ratio 0.22, 95% CI 0.03-0.97;  $p = 0.04$ ). After the intervention there were no cases of HO-CDI for 9 continuous months.

**Discussion:** Implementation of a 5-component bundle successfully decreased HO-CDI by nearly 80% over a subsequent 12-month period. Both antibiotic and PPI usage decreased substantially. The environmental cleaning surveillance helped increase staff awareness of which items were less frequently cleaned. Overall, this team-based, multidisciplinary bundle is an effective way to prevent HO-CDI and currently is being implemented throughout our intensive care units.

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TRENDS OF SEVERITY AND OUTCOMES OF PATIENTS WITH INVASIVE PNEUMOCOCCAL PNEUMONIA  
IN RELATION TO SEASONALITY IN NORTHERN AND SOUTHERN HEMISPHERES

First Author: Ahmed Babiker, MBBS Siddartha Bhandary, MD MPH Jennifer Fuh, MS Jose Bordon, MD PhD.

**Background:** Despite efficacious antibiotics and conjugate vaccines, pneumococcal pneumonia remains a major cause of morbidity and mortality. Many pathogens have exhibited predictable seasonality. Although incidence of invasive pneumococcal disease has demonstrated a seasonal course in temperate climates, with infection incidence peaking in winter months, the forces which drive this seasonality remain poorly understood. Understanding the trends of invasive pneumococcal pneumonia can aid prevention of pneumococcal pneumonia mortality, which has been reported as upwards to 60% in susceptible populations. In this study, we investigated the trends and outcomes of patients with invasive pneumococcal pneumonia in relation to seasonality in the northern and southern hemispheres.

**Methods:** We analyzed data from the Community Acquired Pneumonia Organization (CAPO) database, which has collected multinational cases of confirmed community-acquired pneumonia in adults since 2001. Inclusion criteria for our analysis were patients with valid hospital identification, time to clinical stability greater than 0 days, and blood cultures positive for *Streptococcus pneumoniae*. Exclusion criteria included residence in tropical regions and positive cultures for *Streptococcus pneumoniae* in sites other than the blood. Patients who met the inclusion criteria were categorized as having invasive pneumococcal pneumonia. Prevalence by season was analyzed by chi square test. Mortality by season was analyzed by multivariable logistic regression, adjusting for pneumonia severity index score, need for ICU admission, history of chronic obstructive pulmonary disease, and pneumococcal bacteremia. Time to clinical stability and length of hospital stay were analyzed using Kaplan Meier survival curves. P-values less than 0.05 were considered to indicate statistical significance.

**Results:** Of 4,507 cases of pneumococcal pneumonia, 425 cases met the criteria for invasive pneumococcal pneumonia for analysis. Winter, spring, summer, and fall accounted for 36%, 29%, 9%, and 26% of the cases, respectively. There was a significant decrease in the incidence of invasive pneumococcal pneumonia during the summer ( $p < 0.001$ ). Of the 425 cases, 317 (75%) occurred in the northern hemispheres and 108 (25%) in the southern hemisphere. There was no significant difference in mortality, time to clinical stability, or length of hospital stay between these two groups.

**Conclusion:** *Streptococcus pneumoniae* is the most common etiological agent of pneumonia and remains a major cause of mortality worldwide. In our study, prevalence of pneumococcal pneumonia exhibited a marked seasonality in both southern and northern hemispheres. However, we found no association between clinical outcome and this seasonality.

UTILITY OF GLASGOW-BLATCHFORD SCORE IN PATIENTS WITH UPPER GASTROINTESTINAL BLEEDING PRESENTING AT THE EMERGENCY DEPARTMENT IN A REFERENCE HOSPITAL IN PANAMA

**All Authors:** Calixto Duarte-Chang; Susan Beitia; Enrique Adames

**INTRODUCTION:** Upper gastrointestinal bleeding (UGIB) is a potentially life-threatening condition and remains a significant cause of hospital admission worldwide. In our area, all patients with UGIB, both high risk and low risk according to Glasgow-Blatchford Score (GBS) are admitted. The use of risk scoring systems like GBS may be useful to distinguish high risk patients, who may need clinical intervention and hospitalization, from low risk patients, in which management as outpatients can be considered. Thus, the aim of this study consisted in identifying the utility of GBS score in stratifying high risk patients with UGIB from low risk managed in the emergency department of a tertiary referral hospital.

**METHODOLOGY:** A prospective study involving 97 patients admitted to the emergency department with suspected UGIB was carried out between September 2015 and December 2015. We prospectively analyzed the medical records of all adults  $\geq 18$  years patients in which upper endoscopy was performed within the first 24-48 hours. They were stratified according to GBS score at low risk (GBS = 2) and high risk (GBS = 3). Sensitivity, specificity, positive predictive value (PPV) and negative (NPV) of GBS score was analyzed based on the requirement for endoscopic, radiological treatment (arterial embolization), transfusion of blood products or surgery, as gold standard to classify patients at high risk.

**RESULTS:** A total of 97 patients were included in the study. Of these, 71 (73%) patients were stratified as high risk. The mean age were  $58,7 \pm 2,1$  years and  $47,5 \pm 3,5$  for high risk and low risk respectively. Nonvariceal UGIB was presented in 18 (19%) of the patients. 60 (61%) of patients stratify as high risk presented melena as the initial manifestation compared with patients stratify as low risk ( $p=0,005$ ). Furthermore, high GBS score, need of blood transfusión and endoscopic treatment, and lower hemoglobin value were more frequent in the high risk group compared with low risk group ( $p < 0,05$ ). The most common endoscopic findings consisted of gastric ulcer (26,8%) followed by duodenal ulcer (19,6%). No mortality resulted during the study period. Sensitivity of 98% and a specificity of 17% was observed using using a cutoff of = 2 with a PPV of 74% and NPV of 80%. However, a cutoff = 8, resulted in 96.42% sensitivity and 56,52% specificity, a PPV and NPV was 84% and 87% respectively. The area under the ROC curve was 0.76 (CI 95%, 0,63-0,90)

**CONCLUSION:** This study shows that a cutoff of GBS = 3 has a sensitivity of 99% for stratifying UGIB as high risk, with a relatively low specificity of 23%. In fact, it ranges between 13 and 68% according to recent studies. However, a cutoff of GBS = 8, resulted inn 97% sensitivity with higher specificity (62%), along with a NPV of 89%. In this sense, a value of GBS  $<8$  compared with GBS = 2 would identify patients more precisely who could benefit from outpatient management. In conclusion, in our study, the GBS score had a significant utility for stratifying patients at high and low risk of complications. A cutoff value for GBS = 8 stratified patients at low risk of complications more precisely than GBS = 2.

## TOPICAL MITOMYCIN C APPLICATION CAN IMPROVE THE OUTCOME OF RECURRENT BENIGN ESOPHAGEAL

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**Background** Recurrent benign esophageal strictures represent a subset of complex esophageal strictures. Application of Mitomycin C (MMC) was shown recently in small case series to improve the outcome of complex benign strictures; however these studies were mainly limited to pediatric populations. The aim of this study is to investigate and update our outcome of topical MMC application for recurrent benign esophageal strictures which failed prior standard dilation.

**Methods** Prospectively collected data was abstracted by retrospective chart review of all patients who underwent topical MMC application for recurrent benign esophageal strictures between 2008 and 2015. All patients underwent balloon dilation followed by topical MMC application over 2-3 minutes (0.4mg/ml). The primary endpoint was to demonstrate an improvement in the periodic dilation index (PDI) and dysphagia score. The secondary endpoint was to determine complication rates.

**Results** 12 patients (mean age 70 years, SD 11.6; 42% male; ASA 2-3 100%) with 5 anastomotic (41%), 2 radiation-induced (17%), 2 combined anastomotic and radiation-induced (17%), 2 caustic (17%), and 1 peptic (8%) stricture were included. Strictures had a mean length of 13mm (SD 6.7) and mean diameter of 8.1mm (SD 2.6). Prior to topical MMC application, all patients required an average of 8.5 dilations (SD 5) over 6.7 months (SD 3.7). Additionally, patients had a median dysphagia score of 3, SD 1 (Table 1). At the index dilation with MMC application, strictures were dilated to a mean diameter of 14.1mm (SD 2.2). One patient had concomitant stent placement and 6 patients underwent additional needle knife incision prior to balloon dilation. During follow up (10 months, SD 8.7) 10 patients developed recurrent stricture verified by EGD, however PDI improved from 1.5 (SD 0.9) to PDI 0.3 (SD 0.3) following MMC application ( $p=0.022$ ). In addition to dilation patients with recurrent strictures underwent repeat MMC application or incisional therapy or stent placement (Table 2). The dysphagia score decreased from a median of 3 to 2 ( $p=0.046$ ). One patient developed vocal cord paralysis following MMC application; however it was considered unrelated to treatment. No other complications were recorded.

**Conclusion** MMC application in conjunction with dilation decreases the frequency of esophageal dilations in patients with highly recurrent benign esophageal strictures, although strictures continue to form. Not all patients benefit from MMC treatment, as 2 patients required esophagectomy.

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WORKPLACE HEALTH/FITNESS PROGRAMS AS AN INTERVENTION AT THE LEVEL OF THE WORKPLACE FOR THE OBESITY EPIDEMIC

First Author: Ekaterina Kostioukhina, MD

**Introduction:** Obesity and its correlated diseases are a growing problem in America. As much as 36% of the US population is obese. In Palm Beach County the obesity rate was measured to be 19.9% of the population which is an increase from 14.5% in 2007. Currently, there are arduous efforts at different levels of care to improve the treatment of this disease and to slow down and reverse the growing epidemic. In this project, we describe one approach for weight loss and wellness management at the level of the workplace of a city hospital. The Total Transformation Challenge (TTC) program was designed by a professional trainer and was contracted by a hospital to serve as the health/fitness program for its employees since 2007. It offers a comprehensive approach to health/fitness with different strategies for motivation and adherence to the plan. Marketing strategies were used to recruit members and motivational techniques such as competition.

**Methods:** The study population consists of employees of a city hospital who voluntarily joined the program. Since 2007, 3,130 employees enrolled in the program, 23.6% (N=739) employees completed the program. Informed consent was obtained at the time of enrolment. A variety of measurable markers of success and diversity of available activities were used to retain participants in the 12 week program. Collected data included demographic characteristics, vital signs, total cholesterol level and hemoglobin A1C. Measurements were obtained at the start of the program and were reassessed weekly and at the completion of the program. The cost per person of this intervention is approximately 300\$ per participant with a success rate of 46% as defined by completion of 12 week program of active participation in addition to three pre-identified goals set individually. In this study, we described the program, the cost analysis and the effects on measurable markers such as: success rate, weight loss achieved, changes in hemoglobin A1C, changes in lipid level and changes in blood pressure levels. Results: Total weight loss among all participants was 5,523 lbs. The results showed a reduction in cholesterol levels in 27.2% of participants who completed the program, reduction in hemoglobin A1C in 16%, reduction in blood pressure levels in 23.5% and increased ability to perform tasks with increased muscle strength in 100%. The results achieved and data obtained are preliminarily, and more detailed statistical analysis will be released later. However, these results suggest that this method can be used as one of the many approaches and in combination with strategies at other different levels in fighting the obesity epidemic. Our sample size is small and the need for larger studies with more focused metabolic panels is mandatory.

**Conclusion:** This approach to weight loss/health maintenance is an effective program for the improvement of numerous health factors in a population of hospital employees and can be further considered as one useful intervention for the obesity epidemic and its correlated diseases at the level of large institutions that have a moderate population of employees.

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IMPROVING INFLUENZA VACCINATION RATE WITH HEALTH MAINTENANCE TABLES

First Author: Sue-Wei Luu, MD Second Author: Maryam Sattari, MD, MS

**Introduction:** More than 200,000 people in the United States are hospitalized each year for illnesses associated with seasonal influenza virus infections. Influenza vaccination is associated with decreased mortality, hospitalization rates, and medical costs, yet only 43.6% of adults older than 18 reported flu vaccination during the 2014-2015 influenza season. Effective interventions are needed to increase flu vaccination coverage. To improve flu vaccination rates, we created and implemented a tool referred to as the health maintenance table (HMT) for use in our electronic medical record (EMR). The goal of this study was to evaluate the impact of this tool on rate of influenza vaccination.

**Methods:** We performed a retrospective review of medical charts for patients who visited one of our Internal Medicine clinics during the month of August 2014 and extracted the following information for 238 consecutive patients: (a) whether HMT was included in the clinical encounter documentation, (b) patient's age at the time of clinic visit, (c) patient's gender, (d) whether the patient was eligible for flu vaccination, and (e) whether flu vaccination was given. We used SPSS software version 16 (SPSS, Chicago, IL) to determine the rate of influenza vaccination in HMT and non-HMT users.

**Results:** We considered a patient eligible for flu vaccination if he/she was 18 years of age or older and there was not any documentation of anaphylactic reaction after previous flu vaccine or to a vaccine component, moderate or severe acute illness, fever, or history of Guillain-Barré syndrome within 6 weeks of previous influenza vaccination. Of the 238 patients included in this study, 162 were female and 76 were male. The average age of patients was 54.73 (range 20-90). All the patients were eligible for influenza vaccination and 112 (47.1%) received the vaccination. Vaccination rate was 41.1% (67/163) for HMT non-users and 60.0% (45/75) for HMT users. The difference in vaccination rates between the two groups was statistically significant ( $p = 0.007$ ). We did not find statistically significant associations between flu vaccination and patients' gender or the provider level (faculty versus housestaff). However, there was a statistically significant difference in vaccination rates among different patient age groups, with older patients more likely to be vaccinated.

**Conclusion:** Association of increased influenza vaccination rates with HMT use suggests that routine implementation of HMT might be a cost-effective method for increasing vaccination rates and therefore, decreasing hospitalizations, morbidity and mortality, and medical costs due to influenza virus infections. The other advantages of the HMT are that it is simple, intuitive, and medical provider-friendly and can be incorporated into paper-based medical charts as well as EMR. Future research is warranted to elucidate the effectiveness of HMT implementation in different healthcare settings as well as its downstream effects on patient outcomes.

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## COMPUTER TRAINING IMPROVES FUNCTION FOR ELDERLY VETERANS WHO ARE VISUALLY IMPAIRED

First Author: Diana Marian, MD,

Background: A major concern is the disproportionate number of elderly individuals who are visually impaired in the U.S. and the number is expected to grow significantly as the baby boom generation continues to age. With the rapid advancements in communication technology and the need to access computers to continue an independent and productive life, our elderly, visually impaired population is at greater risk of social isolation, functional decline and generally poor outcomes. The challenge is to identify quick, effective, low-cost solutions to prevent or delay the negative outcomes. There is limited data as to the effectiveness of teaching computer skills to visually impaired older adults. WPB VAMC is one of only 13 programs in the U.S. currently providing this training to visually impaired veterans.

Methods: Our program consists of average 4 weeks of training in the following computer skills: Access Technology, Document Management, Printer, Scanner, Navigation of other drives, E-mail, Internet Updates/Maintenance. The effectiveness is measured by points of improvement in functional independence measure (FIM) before and after completing the program. The FIM ranges from 1 (total assistance) to 7 (complete independence). We have reviewed and reported data of veterans 70 years and older, who completed the program from 2013 to 2015.

Results: 104 veterans completed the program. The average FIM prior to the training was 1.30 and upon completing the program, the average FIM was 5.02. Overall 94.18% demonstrated an average improvement of 3.7 points in each computer skill. The computer skills with greatest and least FIM improvement were Scanner and updates/maintenance with an improvement of 4.26 and 2.9 points respectively.

Conclusions: This unique program of teaching computer access technology to veterans who are visually impaired, demonstrates its effectiveness in improving functional independence as measured by the FIM. This program therefore has the potential to enable older adults with visual impairment to improve communication and contact with their families, preserve their cognitive function and maintain their self-esteem and independence.

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MICRORNA EXPRESSION AND ALTERATION IN THE DEVELOPMENT OF COLON CANCER

First Author: Sajiv Sethi, MD Additional authors: Yiwei Li, Dejuan Kong and Fazlul H Sarkar

**Introduction:** MicroRNAs (miRNAs) are tiny molecules involved in post-transcriptional regulation of genes. Their altered expression is critically involved in tumor growth, migration, invasion, angiogenesis, drug resistance, and metastasis. It is known that some miRNA like miR-Let-7a and miR-155 play a role in cancer metastasis; however, their roles in colon cancer are not completely understood. We set out to determine the role of various miRNAs in angiogenesis, cellular proliferation and metastasis and to identify a method to regulate miRNA expression for early tumor detection, patient prognosis and treatment.

**Methods:** We extracted RNA from the conditioned medium of a known cancer cell line (C4-2B) with skeletal metastatic potential. At the same time, we cultured mature and immature osteoblastic cells (FOB1.19). We initially measured the relative expression of miRNA's such as 7a, 7c, 200b, 155 and 181a in both cancer cells as well as osteoblasts. We took this further and incubated the osteoblastic cell medium with C4-2B cells to evaluate the ability to alter miRNA expression.

**Results:** Expression of let-7a and let-7c was low in cancer cells and high in osteoblast cells. Conversely, miR155 and 181a were highly expressed in cancer cells and decreased in osteoblast cells, confirming the tumor suppressive and oncogenic nature of these biomarkers respectively. Additionally, we found that we could attenuate the oncogenic miR155 levels in cells by incubating the cancer cells in the medium collected from mature osteoblast cells. We were also able to successfully increase the expression of tumor suppressive miR 7a, and 7c 200b in the cancer cells.

**Conclusions:** We were able to successfully illustrate the varying levels of miRNAs and classify them based upon their role in cellular development. Our data demonstrates that when cancer cells are grown in the presence of mature osteoblasts, the high level of let-7 in mature osteoblasts could attenuate the progressive behavior of cancer cells. Moreover, mature osteoblasts could attenuate the expression of oncogenic miR-155 in the cells. These results suggest the oncogenic nature of miR-155 and anti-tumor activity of mature osteoblast. Therefore, mature osteoblasts in bone microenvironment could inhibit proliferation and bone metastasis through the regulation of let-7 family and miR-155.

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CHAPTER WINNING ABSTRACT

Prakhar Vijayvargiya, MD Diana Marian, MD Larry Bush, MD, FACP

Mandatory pneumococcal immunization became a required quality core measure for all hospitalized patients in 2012. In the attempt to meet 100% compliance with this Joint Commission on Accreditation of Health Care Organizations/Centers for Medicare and Medicaid Services mandate, many patients may receive vaccination even though they do not meet any of the Advisory Committee on Immunization Practice recommended indications, whereas others may be immunized a greater number of times than warranted.

This is a retrospective review of the hospital record of 500 consecutive patients admitted to a large urban medical center between April 2014 and July 2014 who received the pneumococcal polysaccharide vaccine (PPSV23) as per the hospital's standing order immunization protocol.

During the period studied, 28% of those patients who were vaccinated with PPSV23 (138/500) were done so not in accordance with Advisory Committee on Immunization Practice recommendations. The majority of the almost one third of nonindicated immunizations were given to patients younger than 65 years, while the remainder occurred in persons aged older than 65 years who had already been vaccinated once after this birthdate. 14% of those vaccinated were unaware of their previous vaccination status.

In the interest of improving and standardizing the quality of care, hospital policies created to satisfy mandated core measures may promulgate unintentional consequences, some of which could potentially be harmful. This study demonstrated that an effort to achieve 100% compliance with the pneumococcal immunization "all-or-none" expectation led to evidence-based immunization guideline recommendations not being correctly adhered to.

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IMPROVING THE CARE OF A CHRONIC DISEASE: DIABETES SPECIFIC QUALITY MEASURES IN A RESIDENT CONTINUITY CLINIC

Anastasia-Stefania Alexopoulos MBBS, Bilal Ahmad MD, Tyler Peck MD, Nurcan Ilksoy MD, Racha Halawi MD, MS

**Introduction:** Diabetes mellitus is among the most common chronic diseases managed in the primary care setting. Diabetic retinopathy and foot complications are significant causes of morbidity, and current guidelines recommend annual screening for early identification and treatment. We aimed to increase the percentage of yearly eye and comprehensive foot exams of patients who follow in a large public teaching hospital by increasing the number of referrals placed in a resident primary care clinic.

**Methods:** This work is part of the yearly performance improvement projects conducted by the internal medicine residency program. A total of 402 patient charts were electronically reviewed starting December 2014 until April 2015. Chart reviewing was conducted by the residents who were members of the clinic of interest throughout the duration of the study. Three tests of change were implemented. The first test of change was piloted in January and consisted of a "dotphrase" incorporated in clinic notes to serve as a reminder to order referrals. The second test of change was conducted in February whereby a "patient card" was handed to diabetic patients at intake with the purpose of reminding patients and providers of those who require screening. The third test of change was conducted in March and involved distributing pocket "resident cards" along with a one-on-one resident education on screening guidelines. A descriptive analysis of the collected data was subsequently performed using the Microsoft Excel statistical package.

**Results:** Baseline data collected in December 2014 showed that 62% of our patients had received eye exams and 39% received comprehensive foot exams in the preceding year. Of those who did not have referrals available, 17% were given ophthalmology referrals and 17% were given podiatry referrals by the resident providers during their visit in December. With the implementation of the tests of change, a steady rise was noted in the percent referrals placed, peaking in March 2015 and reaching 57% and 53% for ophthalmology and podiatry, respectively. Data sampled in April 2015 revealed a slight decline in the percentage of eye referrals to 48% and a larger decline in the podiatry referrals to 24%. The number of patients receiving an annual eye and foot exam had not increased throughout the duration of the project.

**Conclusion:** Our results suggest that a multi-disciplinary approach to education of diabetes management can be successful in increasing referral rates for appropriate screening. In addition, all efforts and planned interventions will need to be sustained given the decline noted in referral rates once the tests of change were concluded. We postulate that a longer observation period is required to start noting an improvement in actual screening rates given the many scheduling limitations in our large public hospital.

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IMPROVING COMMUNICATION SKILLS OF INTERNAL MEDICINE RESIDENTS USING FAMILY MEETING SIMULATION EXERCISES IN THE MEDICAL INTENSIVE CARE UNIT

Travis Watai, MD, Brent Matsuda, MD, Gehan Devendra, MD, Cody Takenaka, MD, Kamal Masaki, MD, and Reid Ikeda, MD. Department of Medicine, John A. Burns School of Medicine, Honolulu, Hawaii.

**BACKGROUND:** Internal Medicine residents rotating through the Medical Intensive Care Unit (MICU) discuss the clinical status and prognosis of critically-ill patients during family meetings, often without formal training. The role for a curriculum focused on enhancing delivery of distressing news to families in the MICU has not been well-defined in previous studies. Our objective was to evaluate the impact of a curriculum designed to improve the communication skills and comfort level of Internal Medicine residents conducting family meetings in the MICU.

**METHODS:** Second and third year Internal Medicine residents rotating through the MICU (n=16) received a 30 minute didactic lecture designed to improve communication during family meetings with content specific to the SPIKES protocol (a six-step method for disclosing unfavorable medical information to patients). Pre and post-lecture Mini-Clinical Evaluation Exercise (Mini-CEX) simulations involving two different enacted scenarios were utilized to evaluate resident interpersonal and communication skills. Each Mini-CEX was graded simultaneously by two MICU attendings using a modified SPIKES evaluation tool. Residents answered a brief questionnaire to measure the educational value of the overall experience, as well as their comfort level with leading end-of-life discussions before the didactic session and after the post-lecture Mini-CEX.

**RESULTS:** Resident performance significantly improved after the lecture (mean pre-lecture score = 23.3 versus post-lecture score 44.9,  $p < 0.0001$ ). There was a high level of correlation between the Mini-CEX scores of the two MICU attendings (pre-lecture scores correlation coefficient=0.92,  $p < 0.0001$ ; post-lecture scores correlation coefficient=0.77,  $p = 0.0004$ ). We did not find significant differences in scores by gender, post-graduate level, or US graduate versus international medical graduate. The self-assessed comfort level scores of residents delivering bad news showed significant improvement (mean pre-curriculum score = 3.0 versus post-curriculum score = 3.4,  $p = 0.004$ ). This curriculum was very well received, with the majority of residents rating both the lecture and the Mini-CEXs as “very helpful.”

**CONCLUSIONS:** Establishing rapport with family members during difficult clinical situations is imperative to quality patient care. This study revealed that a focused curriculum including a brief lecture and Mini-CEXs given to Internal Medicine residents rotating in the MICU was highly effective in improving communication skills and resident comfort level during end-of-life discussions. This simple educational intervention emphasizes the value of a formal curriculum to enhance resident performance and confidence in delivering bad news.

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THE ROLE AND PROGNOSTIC SIGNIFICANCE OF ABSOLUTE NEUTROPHIL COUNT IN PATIENTS WITH HEART FAILURE WITH PRESERVED EJECTION FRACTION

First Author: Abdalla Hassan, MD Dana Villines Shafaq Mahmoud, MD Lloyd W. Klein, MD, FSCAI

**Background:** The role of inflammation and neutrophil/lymphocyte ratio has been defined in systolic heart failure (HF) and coronary artery disease, but it is uncertain if such a relationship exists in HF with preserved ejection fraction (HFpEF) patients (pts). We sought to identify the prognostic impact of Absolute Neutrophil Count (ANC) in HFpEF in the absence of coronary artery disease history (CAD Hx).

**Methods:** In this retrospective cohort, the institutional HF data base was queried to identify HFpEF pts diagnosed in 2006 (registry initiation date) based on symptoms, BNP, and Echocardiogram with no CAD Hx. Measured outcomes were cardiovascular disease (CVD) mortality and number of HF re-admissions (poor outcome if > 2 HF re-admissions).

**Results:** We identified 125 eligible pts. Mean age was  $70.8 \pm 11.7$  years. Women constituted 56.6%. Mean follow up was for  $8.75 \pm 0.17$  years. The CVD mortality rate was 7.1%. Poor outcome was identified in 52.6%. Mean ANC was  $6.3 \pm 3.2$ , and in multivariate regression analysis, including baseline demographic, clinical, and biochemical covariates, ANC remained significantly associated with poor outcome and an independent predictor of mortality (OR 1.14, 95% CI 1.02-1.29,  $P=0.04$  after adjustment for age, sex, hypertension, and other risk factors).

**Conclusion:** In HFpEF pts, ANC is a significant predictor of poor outcome as well as mortality in the absence of coronary artery disease. Accordingly, ANC can be utilized as one of the non-invasive prognostic markers in HFpEF pts.

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INDICATIONS AND EFFICACY OF GAMMA KNIFE STEREOTACTIC RADIOSURGERY FOR RECURRENT GLIOBLASTOMA: TWO DECADES OF INSTITUTIONAL EXPERIENCE

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**Introduction:** The role of stereotactic radiosurgery (SRS) for recurrent glioblastoma, along with the radionecrosis risk in this setting remain unclear. Larger studies would help inform proper indications, efficacy and anticipated complications.

**Methods:** We retrospectively analyzed recurrent glioblastoma patients who underwent Gamma Knife SRS between 1991-2013. We used the partitioning deletion/substitution/addition (partDSA) decision tree algorithm to identify potential predictor covariate cut points followed by Kaplan-Meier and proportional hazards modeling to identify factors associated with post-SRS and post-diagnosis survival.

**Results:** 174 glioblastoma patients with median age of 54.1 years underwent SRS a median of 8.7 months after initial diagnosis. 75% had one treatment target (range=1-6) and median target volume and prescription were 7.0 cm<sup>3</sup> (range 0.3-39.0) and 16.0 Gy (range 10-22), respectively. Median overall survival was 10.6 months (range 1.4-157.6 months) following SRS and 19.1 months after diagnosis (range 2.3-206.2 months). Kaplan-Meier and multivariate modeling revealed that younger age at SRS, higher prescription dose and longer interval between original surgery and SRS are significantly associated with improved post-SRS survival. 46 patients (26%) underwent salvage craniotomy a mean of 6.6 months after SRS with 63% showing radionecrosis or mixed tumor and necrosis versus 35% showing purely recurrent tumor. The necrosis/mixed group had lower mean isodose prescription compared to the tumor group (16.2 vs. 17.8 Gy, p=0.0031) and larger mean total treatment volume (10.0 vs. 5.4 cm<sup>3</sup>, p=0.0091).

**Conclusions:** Gamma Knife may benefit a subset of focally recurrent glioblastoma patients, particularly those who are younger with smaller recurrences. Higher prescriptions are associated with improved post-SRS survival and do not seem to have greater risk of symptomatic treatment effect.

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## DETECTION OF COLORECTAL CANCER BY SCREENING METHODS VERSUS DIAGNOSTIC COLONOSCOPY IN UNDERSERVED POPULATION

First Author: Rohit Kumar, MD Jibran Ahmed, MD Romy Thekkekara, MD Ena Mahapatra, MD

**Introduction:** The colorectal cancer (CRC) is the third most common type of cancer in the United States with reported age-adjusted incidence of 51.6 per 100,000 persons. Increase in screening rates has significantly lowered both incidence and mortality rates of CRC. Overall screening rate in United States, approximately 65%, has been stable since 2010 but the rate is significantly lower in underserved population. The objective of this retrospective study is to determine the demographic and the stage at which CRC is detected by colonoscopy performed as part of routine screening versus diagnostic work up at a tertiary safety net hospital for underserved population.

**Methods:** This is a retrospective study reviewing medical records of all patients between ages 50 and 75 years diagnosed with CRC from January/2009 to December/2010. Patients were classified in following categories: 1) Screening done with colonoscopy, FOBT/FIT or flexible sigmoidoscopy, 2) Diagnostic colonoscopy for symptomatic patients, 3) Interval CRC after initial negative screening. Exclusion criteria were index colonoscopy at outside center, more than average risk or recurrent disease. Patients' demographic characteristics, insurance status and CRC stage at detection was also reviewed.

**Results:** In this study 225 patients' charts were reviewed out of which 168 were eligible for the analysis. The median age at detection was 60 (range 50-75) years with 36% female and 60% African-American patients. The insurance status was Medicare in 24%, Medicaid in 27% and 29% of patients did not have any insurance. CRC was detected by screening in only 8% of patients (category 1) while 85% and 7% were in diagnostic (category 2) and interval CRC (category 3), respectively. Colonoscopy was the most common method (64%) for routine screening (Category 1) but in patients who were diagnosed with interval CRC (category 3), FOBT/FIT was the most common prior screening method. The median CRC stage at detection in category 1, 2 and 3 was stage I, III and II, respectively

**Discussion:** The effectiveness of screening in reducing the incidence and mortality of CRC is well established. In our institution, 34% healthy individuals are up-to-date with routine CRC screening which is much below the goal, laid by Healthy people 2020, of 70.5%. Several interventions to overcome multiple barriers tailored to needs of this specific population are required. The implementation of Affordable Care Act is expected to improve the CRC screening rates.

ACCURACY OF EUS-FNA TO DIAGNOSE SOLID PANCREATIC LESIONS IN CHRONIC PANCREATIC VS NORMAL PANCREATIC PARENCHYMA: A META-ANALYSIS AND SYSTEMATIC REVIEW

First Author: Pankaj Malhotra, MD Second Author: Matthew L. Bechtold, MD Third Author: David Forcione, MD Fourth Author: Srinivas R. Puli, MD

Since its initiation, endoscopic ultrasound-guided fine needle aspiration (EUS-FNA) has become the primary modality by which tissue diagnosis of suspicious pancreatic lesions is performed in patients with normal pancreatic anatomy. However, patients with chronic pancreatitis (CP) often possess structural alterations that complicate diagnosis of masses suspicious for malignancy. Because there is no universally accepted data assessing the utility of EUS-FNA among this patient population, we performed a systematic review and meta-analysis to evaluate the efficacy of EUS-FNA in determining the etiology of pancreatic masses in patients with CP compared to their normal counterparts.

Data obtained from EUS-FNA studies with a criterion standard (established by surgery or appropriate follow-up) were selected. Articles were searched in Medline, Pubmed, Ovid journals, Cumulative index for nursing & allied health literature, INTERNATIONAL PHARMACEUTICAL ABSTRACTS, old Medline, Medline nonindexed citations, and Cochrane Central Register of Controlled Trials & Database of Systematic Reviews. Pooling was conducted by both Mantel-Haenszel method (fixed effect model) and by the DerSimonian Laird method (random effects model). The heterogeneity of studies was tested using Cochran's Q test based upon inverse variance weights.

Preliminary data retrieval pinpointed 3610 reference articles, of which 360 relevant articles were selected for review. Data was extracted from 3 (N=204) studies for EUS-FNA in the chronic pancreatitis arm, and 41 studies (N=4766) for EUS-FNA in the normal pancreatic parenchyma arm. Pooled sensitivity of EUS-FNA for pancreatic masses in patients with chronic pancreatitis was 63.4% (95% CI 46.9-77.9), while that for patients with normal pancreatic parenchyma was 86.8% (95% CI 85.5-87.9). Pooled specificity of EUS-FNA for pancreatic masses in patients with chronic pancreatitis was 100% (95% CI 97.8-100), whereas that for patients with normal pancreatic parenchyma was 95.8% (95% CI 94.6-96.7). The positive likelihood ratio of EUS-FNA in patients with chronic pancreatitis was 53.9 (95% CI 10.2-283.5), in comparison to 15.2 (95% CI 8.5-27.3) for patients without chronic pancreatitis. The negative likelihood ratio of EUS-FNA in patients with chronic pancreatitis was 0.42 (95% CI 0.14-1.25), in comparison to 0.17 (95% CI 0.13-0.21) for patients with normal pancreases. The diagnostic odds ratio for EUS-FNA in detecting the etiology of pancreatic masses in patients with chronic pancreatitis is 138.37 (95% CI 23.36-819.81). The diagnostic odds ratio for patients with normal pancreatic parenchyma is 105.9 (64.0 - 175.3).

The main finding of our study is that although EUS-FNA is less sensitive in patients with chronic pancreatitis when compared to their normal counterparts (63.4% vs. 87.5%), it essentially retains its specificity (100.0% vs. 95.7%). Consequently, different diagnostic maneuvers can be employed to increase this procedure's efficacy, including multiple FNA passes. Although we believe that EUS-FNA remains viable in patients with chronic pancreatitis, further refinement in EUS technologies is needed to improve this modality.

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EFFICACY OF PREOPERATIVE BILIARY DRAINAGE IN MALIGNANT OBSTRUCTIVE JAUNDICE: A META-ANALYSIS AND SYSTEMATIC REVIEW.

Harsha Moole, MD; Srinivas R. Puli, MD

**Background:** In patients requiring surgical resection for malignant biliary jaundice, it is unclear if preoperative biliary drainage would improve mortality and morbidity in these patients by restoration of biliary flow prior to operation. This is a meta-analysis to pool the evidence and assess the utility of preoperative biliary drainage in patients with malignant obstructive jaundice.

**Aim:** Primary outcomes are mortality benefit and major adverse events comparison in patients with malignant obstructive jaundice undergoing direct surgery versus preoperative biliary drainage.

**Methods:**

**Study Selection Criteria:** Studies using preoperative biliary drainage in patients with malignant obstructive jaundice.

**Data collection & extraction:** Articles were searched in Medline, Pubmed, Ovid journals, CINAH, International pharmaceutical abstracts, old Medline, Medline non-indexed citations, and Cochrane Central Register of Controlled Trials & Database of Systematic Reviews. Two authors independently searched and extracted data. Any differences were resolved by mutual agreement.

**Statistical Method:** Pooled proportions were calculated using both Mantel-Haenszel method (fixed effects model) and DerSimonian Laird method (random effects model). The heterogeneity among studies was tested using Cochran's Q test based upon inverse variance weights.

**Results:** Initial search identified 2230 reference articles, of which 204 were selected and reviewed. 26 studies (N=3532) for preoperative biliary drainage in malignant obstructive jaundice which met the inclusion criteria were included in this analysis. Odds ratio for mortality in pre-operative biliary drainage (PBD) group versus direct surgery (DS) group was 0.96 (95%CI = 0.71 to 1.29). Pooled fixed effect size of number of major adverse effects in the PBD group was 10.40 (95%CI = 9.96 to 10.83) compared to 15.56 (95%CI = 15.06 to 16.05) in the DS group. The pooled effects estimated by fixed and random effect models were similar. The p for chi-squared heterogeneity for all the pooled accuracy estimates was > 0.10.

**Conclusions:** In patients with malignant biliary jaundice requiring surgery, preoperative biliary drainage group had significantly less major adverse effects than direct surgery group. The mortality rates in both the groups were comparable. Hence, patients might benefit from biliary drainage prior to surgical resection.

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QI PROJECT: IMPROVEMENT OF SMOKING CESSATION INTERVENTION IN RESIDENT RUN GENERAL MEDICINE CLINIC.

First Author: Chan Yeu Pu, MD Karmapath Aryal, MD

Introduction Tobacco use is a leading cause of premature morbidity and mortality worldwide. Current prevalence of smoking in USA is estimated to 17.3% and in our clinic is 16.4%. One in five deaths every year in US is secondary to cigarette smoking. Life expectancy for smokers is at least 10 years less than the non-smokers.

The purpose of our project was to find the status of cigarette smoking screening by resident physicians among the clinic patients and determine the interventions provided to help active smokers quit smoking. The next step was to try to improve the screening as well as the intervention rates. The clinic has morning and afternoon shifts with 6 groups of residents working in each shift.

Methodology: We attended the huddles (which include all the residents working in that shift) just prior to clinic to provide verbal reminders for smoking screening and intervention for the positive ones, for 3 consecutive days for 2 weeks (6 days). We used printouts of "5 A Guide" with verbal reminders to help residents understand the steps of smoking cessation better. We also sent email reminders once to all the residents prior to verbal reminder. We collected data from 3 weeks prior to and 3 weeks after the intervention dates and compared them to see effectiveness of our intervention

Results and discussion: We reviewed 357 patient charts which comprise of 191 pre-intervention, 166 post-intervention. The mean age of patients was  $56 \pm 12$ . There were 56(15.7%) smokers, smoking screening was 62%. There were no significant difference between rate of screening before and after intervention (61.3% vs 62.7%,  $p=0.78$ ). Overall rate of provider intervention was 30%. There was no referral to smoking cessation clinic done. Interview of residents pertaining to smoking cessation clinic found that none of residents knew of its existence. Information obtained via chart review and resident interview was used to formulate the next round intervention. Residents are given smoking intervention performance feedback individually via email. Smoking intervention module will be added to annual general medicine clinic conference. All residents will be assigned to smoking cessation clinic during their ambulatory rotation.

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SERUM PROCALCITONIN VALUES WITHIN 24 HOURS OF ADMISSION CAN PREDICT SEVERITY OF ACUTE PANCREATITIS: A META ANALYSIS

First Author: Karthik Rangunathan, MD Other authors: Xavier Pereira MD, Vamsi Emani MD

**Introduction:** Severe acute pancreatitis develops in about 10-15% patients with acute pancreatitis and is associated with increased morbidity and mortality. Several laboratory markers have been tested to predict severe acute pancreatitis such as C-reactive protein, blood urea nitrogen, creatinine but none of these have been shown to be effective in predicting within 24 hours. Procalcitonin(PCT) is an acute phase reactant and has been studied as a marker in pancreatitis but mixed results have been reported in the literature. We aimed to do meta analysis to calculate the cumulative sensitivity, specificity, likelihood ratios and diagnostic odds ratio for PCT as a marker.

**Methods:** MEDLINE, EMBASE, and SCOPUS were searched to identify potential articles published from 1946 to October 2014. MeSH headings included in the search included severe acute pancreatitis, procalcitonin. All references from the identified articles were further searched to find more studies. Only studies in adult population and studies in English language were included. Studies that reported the predictive value of PCT measured within 24 hours of admission as a marker of severe acute pancreatitis as opposed to mild pancreatitis were only included. We collected sufficient data to construct a 2 x2 contingency table for true- positive, true- negative, false-positive and false negative results. DerSimonian-Laird random-effects model was used to estimate the pooled sensitivity, specificity, and likelihood ratio, and a diagnostic odds ratio. Analyses were performed using Meta-Disc Version 1.4.

**Results:** A total of 470 articles were identified from the search strategy and from articles identified through references. Of these 9 studies involving 594 patients were included in the final analysis based on our inclusion criteria. Pooled sensitivity and specificity of PCT as a marker of severe acute pancreatitis was 54% (95% CI 47 to 60) and 81% (95%CI 67-91). The combined positive likelihood ratio was 3.41 (95% CI 1.57 - 7.40) and negative likelihood ratio was 0.59 (95% CI 0.41 - 0.84). The pooled odd ratio was 6.51 (95% CI 2.18 - 19.47).

**Conclusion:** PCT when measured within the first 24 hours can predict severe acute pancreatitis with an odds ratio of 6.51. With high specificity, positive and negative likelihood ratio, high PCT can be used as a reliable marker in acute pancreatitis. PCT can be routinely measured in all patients getting admitted to the hospital for severe acute pancreatitis and proper triaging of these patients and early aggressive management may potentially reduce morbidity and mortality associated with severe acute pancreatitis

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DOES THE ETIOLOGY OF ACUTE PANCREATITIS MATTER? ANALYSIS OF A LARGE DATABASE

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**Background & Aim:** Acute pancreatitis is one of the most common gastrointestinal diagnoses of hospital discharge in the US. An episode can be triggered by multiple factors but usually has a mild course. Only between 10-20% of the episodes can be severe. We investigate the association between the most common etiologies of acute pancreatitis and systemic inflammatory response syndrome (SIRS), acute kidney injury (AKI), and pancreatic necrosis.

**Methods:** We performed a retrospective cohort study of consecutive patients admitted for acute pancreatitis at a large public hospital during two years, 01/2013 through 12/2014. We identified acute pancreatitis by ICD9 code or lipase = 3 times the normal upper limit. Two physicians reviewed each case to identify only those cases that were a patient's first documented episode of acute pancreatitis, and exclude other causes of hyperlipasemia. We excluded patients who were transferred to our center. Etiology of pancreatitis was categorized as follows: Gallstone disease, Alcoholic and Hypertriglyceridemia. We defined AKI as a serum creatinine increase >0.3 mg/dL within 48h, >50% increase within 7 days, or an elevated Scr that normalized by hospital discharge. We define SIRS as per the current 2 out of 4 criteria. Pancreatic necrosis was counted if it was discovered on abdominal CT scan with contrast during the first 96 hrs of admission. We constructed multivariable logistic regression models using STATA software version 13.

**Results:** We analyzed 460 patients according to the most common etiologies of acute pancreatitis. There were significant differences between the groups. Incidence according to etiology was: Gallstone pancreatitis 144 (33%), Alcoholic 142 (31%) and Hypertriglyceridemia 30 (7%) unknown 144 (29%). Hypertriglyceridemia pancreatitis was associated with higher incidence of SIRS on admission (OR 2.95 p<0.001; CI: 1.3-6.6), Persistent SIRS at 48h (OR 3.75 p<0.001; CI: 1.6-8.7) and AKI (OR 2.73 p<0.05; CI: 1-7.2) but interestingly none of these cases had pancreatic necrosis. Gallstone pancreatitis was protective against AKI (OR 0.27 p< 0.05; CI: 0.09-0.7). Alcoholic pancreatitis was associated with higher incidence of pancreatic necrosis (OR 3.0 p< 0.05; CI: 1-8.8).

**Limitations:** Retrospective study without complete comorbidity adjustment.

**Strength:** Large sample size of patients with a first episode of acute pancreatitis. Absence of transferred patients.

**Conclusion:** Among patients with acute pancreatitis, depending on the etiology of the episode the incidence of organ failure and pancreatic necrosis vary. Hypertriglyceridemia have higher rates of Persistent SIRS, hence higher risk for multi-organ failure as evidenced by higher rates of AKI in our cohort. Alcoholic acute pancreatitis has higher rates of pancreatic necrosis. Hypertriglyceridemia, gallstone and alcoholic acute pancreatitis should be managed as different entities. These findings have important clinical implications that warrant further investigations.

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HEPATITIS A AND B VACCINATION IN PATIENTS WITH CHRONIC LIVER DISEASE AND CIRRHOSIS

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**Introduction:** According to the Adult Committee on Immunization Practices (ACIP), patients with chronic liver disease and cirrhosis (CLD) should be vaccinated for Hepatitis A and B. It was hypothesized that patients with CLD at the continuity clinic for the St. Vincent Internal Medicine Residency were not routinely being offered these vaccinations. This prospective, quality improvement project, which was deemed exempt from IRB approval, determined the baseline Hepatitis A and B vaccination offers and administration rates in our CLD patients. Potential root causes for gaps in care were evaluated, and a process map was designed to implement countermeasures to identified process deficiencies.

**Methods:** The clinic was queried for patients seen between July 1, 2014 and January 8, 2016 with a diagnosis of chronic liver disease. A chart review was performed to confirm the presence of CLD and whether Hepatitis A and B vaccinations were offered. If either vaccine was given, it was determined whether the vaccination series was appropriately completed. For Hepatitis A, this is defined as 2 vaccines at 0 and 6 months. For Hepatitis B and Twinrix, a combination Hepatitis A and B vaccine, this would be defined as 3 vaccines at 0, 1, and 6 months. From this, a baseline Hepatitis A and B vaccination rate for CLD patients was established. The vaccination process was reviewed with staff physicians, residents, pharmacy, and nursing staff. During the process mapping, potential barriers to adequate vaccination were determined to better understand the need to institute measures to improve the vaccination rates

**Results:** Of the 36 patients identified, only 2% of eligible patients were current on their vaccinations. Patient follow-up and identification of appropriate candidates were key barriers to care. Letters were used to counter these measures and at one month post-intervention, the percentage of patients current with their vaccine schedules had risen to 25%. This included 5 patients (14%) who had immediate follow-up or initial vaccination, and 2 patients (5%) who completed their series. At a 6-month follow-up, a total of 43 patients were identified. Of those, 10 patients (23%) were appropriately vaccinated with another 4 patients (9%) that had completed their vaccination schedule. The interventions will be ongoing on a quarterly basis with outcomes assessed longitudinally.

**Discussion:** Patients with advanced liver disease are at a substantially higher risk of infection and death. Through disease progression, most immunizations lose effectiveness and superimposition of acute hepatitis A and B has been associated with an accelerated progression of liver disease. Since 1981 and 1995, hepatitis A and B have become vaccine-preventable diseases. This study identified some barriers to implementation of these recommendations and clinic inefficiencies. We will continue to approach these issues over the next several months through a multidisciplinary team involving nursing, residents, and attending physicians.

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## EPIDURAL CORTICOSTEROIDS FOR SPINAL STENOSIS: A LIVING SYSTEMATIC REVIEW

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**Introduction:** Spinal stenosis is a common condition often treated by epidural steroid injections. However, treatment may cause serious adverse effects such as vertebral fracture and infection. Trials assessing epidural steroid injections provide conflicting results. The most recent Cochrane review did not pool the existing trials due to heterogeneity and did not include more recent trials and a larger trial. Our objective was to build upon the work started by the prior review.

**Methods:** We conducted a living systematic review based on the Cochrane review by Ammendolia. We used collaborative, open-source, and cloud-based methods to build upon an existing but outdated review. We formally assessed trials with the Cochrane Risk of Bias Tool and the Summary of Findings Table as developed by the Grade Working Group. The review was conducted according to PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) guidelines adapted to living reviews. We sought trials that addressed the use of epidural steroid injections for the treatment of spinal stenosis and met criteria for inclusion developed by the American College of Physicians Journal Club. We started by including the same trials in the two most recent reviews. We then searched the Web of Science for trials that cited the Cochrane review. Next we searched for newer trials in the Cochrane Central Register of Controlled Trials and ClinicalTrials.gov. Lastly, we sought conceptually similar citations using PubMed's Find Related Data portal.

**Results:** We identified 3 trials more recent than the prior review. This resulted in 622 patients from 5 trials. All studies compared epidural injections of steroids with anesthetics versus anesthetics alone. The studies reported follow-up ranging from 1 day to 21 months after injections. As a group, the trials were at low risk of bias. We found that epidural injections of corticosteroids plus anesthetics are not effective at any point during follow-up when compared to epidural anesthetics alone. There was no heterogeneity of results. Secondary analysis of the trial by Fukusaki showed that epidural anesthetics alone versus epidural saline reduced pain at one week (statically significant) and maybe at one month (statically insignificant). The quality of evidence for benefit from epidural anesthetics is low due to being from a single small trial, not clearly blinded, leading to imprecision of results and the small study effect or publication bias.

**Conclusion:** We found no benefit from epidural steroid injections and possible short-term benefit from anesthetics alone based on low quality of evidence from a single small trial. This analysis questions the use of epidural steroids in the treatment of spinal stenosis. The possibility of benefit from anesthetics alone warrants further research.

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## INCREASING EVIDENCED-BASED MANAGEMENT OF INCIDENTALLY FOUND SOLITARY PULMONARY NODULES

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**Introduction:** Over 150,000 incidental solitary pulmonary nodules (SPNs) are found on radiologic imaging every year. The American College of Chest Physicians (ACCP) has specific guidelines for management of SPNs based on their CT appearance. Adherence to ACCP guidelines requires utilization of the Fleischner Society Guidelines (FSGs) for nodules < 8 mm and the ability to calculate malignancy probability for nodules > 8 mm. The primary goal of this project is to evaluate Wichita Radiology Group's (WRG) baseline adherence to the FSGs in their radiology reports. A secondary goal is to evaluate the reporting frequency of information necessary to calculate SPN malignancy probability according to the Mayo Clinic model. A recommendation on how to improve adherence to guidelines will be given.

**Methods:** The principles of Six Sigma were used and a DMAIC cycle was implemented. This report summarizes the diagnostic journey (define, measure, and analyze) and suggests a process improvement intervention (improve). A chart review from 12/03/14 to 4/1/14 searched for SPNs incidentally found on chest CT by WRG. The reports were assessed for nonconformance with FSGs and for inclusion of factors necessary to calculate SPN probability of malignancy. Based on the chart analysis a laminated reference card was created to be placed in radiology reading rooms. The card contains the FSGs and factors necessary to calculate probability of malignancy.

**Results:** A total of 37 incidentally found SPNs were found during the chart review. Seventy percent of reports with nodules < 8 mm (N = 23) were adherent with FSGs. Of the nonadherent reports, 71.4% gave a recommendation not consistent with FSGs and 28.6% gave no recommendation. Review of the literature suggests national adherence rates vary from 27.0% to 60.8%, however, can be as high as 82.8% with use of laminated reference cards. Reporting of information necessary to calculate SPN probability of malignancy varied by factor: 100% included age and location, 89.2% included nodule diameter, 27.0% included patient smoking history, 10.8% included absence or presence of nodule spiculation, and 0% included absence or presence of extrathoracic cancer.

**Conclusions:** WRG's adherence to FSGs is above average when compared to other reports in the literature, however, their ability to report factors required to calculate probability of malignancy according to the Mayo Clinic model could be improved in the following areas: nodule diameter, patient smoking history, absence or presence of nodule spiculation, and absence or presence of extrathoracic cancer. A reference card placed in radiology reading rooms could serve as a novel intervention to increase adherence to FSGs and reporting of information necessary calculate SPN probability of malignancy.

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TEMPLATE MANIFESTO CREATING A STANDARDIZED DISCHARGE SUMMARY TEMPLATE FOR SAFE TRANSITION OF CARE

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**Introduction:** The process of transitioning from the hospital to home requires careful transfer of information usually in the form of a discharge summary to ensure patient safety and prevent re-admissions. It is evident through numerous studies that discharge summaries often lack pertinent medical information. An electronic discharge summary template was created at Wesley Medical Center in accordance with the Joint Commission Mandate and Society of Hospital Medicine to ensure the information is complete when transferred.

**Methods:** A retrospective cohort study evaluated discharge summaries of patients who presented for hospital follow-up clinic appointments. The discharge summary template was implemented in July 2014 at Wesley Medical Center. Charts were reviewed from July 2014 to March 2015 for patients 18 years and older discharged from community hospitals in Wichita who followed up at KU Resident/Faculty Clinics. Their discharge summaries were evaluated for completeness. The primary outcome was a complete discharge summary, defined as including 1) important labs/imaging from the hospitalization, 2) discharge diagnosis, and 3) hospital course. Secondary outcomes were inclusion of pending labs, documentation of patient education/summary, anticipated problems/interventions and follow up information. The association with use of template and completeness of summary was measured controlling for hospital and type of hospitalist service. The Fisher's exact test and Poisson regression analysis were used to test uni-variable and multi variable associations.

**Results:** Of the 101 patient charts reviewed, discharge summaries which used the electronic template were complete using the aforementioned criteria 100% of the time compared to 50% or less if template not utilized. Additional pertinent information such as pending labs, anticipated problems and follow-up information was present almost 100% if electronic template was utilized compared to 30% or less if template not utilized.

**Conclusions:** Creating a standardized electronic template which requires the hospitalist to provide specific information about patient's hospital course will minimize incomplete documentation as to facilitate patient safety and a smooth transition of care.

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A RETROSPECTIVE ANALYSIS OF THE IMPACT OF WEIGHT LOSS ON RENAL FUNCTION

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Approximately 66% of Americans are overweight with approximately half classified as obese. Obesity is associated with increased morbidity and mortality and is an independent risk factor for the progression of chronic kidney disease. Weight loss is correlated with improved renal function and reductions in obesity related glomerulonephropathy. This study investigated the effect of a medically supervised weight loss program on renal function among program participants at baseline and following 12 weeks of therapy. This study was a retrospective analysis of adult patients voluntarily enrolled in a physician-directed community-based weight management program from 2009 to 2014. Patients consumed at least 800 kilocalories per day, attended weekly behavioral education classes, and expended approximately 300 kilocalories per day in physical activity. The primary outcome of improved renal function was assessed by using statistical analysis to compare weight loss and GFR. Secondary outcomes included changes in fasting blood sugar, HbA1c, total cholesterol, LDL, HDL, triglycerides, systolic blood pressure, and the number of diabetic and anti-hypertensive medications. A total of 71 patients with an average weight of 286 pounds, BMI of 53, and baseline GFR of 29 (stage 4 chronic kidney disease) were included. Following 12 weeks of therapy, 80% of participants improved in stage, 19% remained within the same stage, and 1% progressed to a higher stage ( $p=0.017$ ). Analysis revealed a positive correlation of 0.29 between weight loss and increased GFR ( $p=0.029$ ). Approximately 64% of patients required fewer anti-hypertensive medications and 83% of patients required fewer diabetic medications. Organized weight loss programs are a viable treatment modality for the prevention of co-morbid disease progression. This study indicated a positive correlation between weight loss and improved renal function, as evidenced by increased GFR, with the majority of participants exhibiting an improvement in chronic kidney disease stage. Analysis of secondary outcomes resulted in improvements in HbA1c, blood pressure, LDL, total cholesterol, and triglycerides. The majority of patients within the study required fewer diabetic and anti-hypertensive medications following weight loss. When controlling for both diabetes and hypertension, the effect of improved renal function with weight loss persisted.

COMPARING R<sub>2</sub>CHADS<sub>2</sub> AND CHA<sub>2</sub>DS<sub>2</sub>VASC SCORES IN STROKE PATIENTS WITH NON-VALVULAR ATRIAL FIBRILLATION AND RENAL FAILURE

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**Introduction:** Atrial fibrillation (AF) affects approximately 2.2 million patients in the US. It is the most common rhythm disorder among patients hospitalized with a primary diagnosis of an arrhythmia. The median age of patients developing AF is 75 years; approximately 70% of patients are between the ages of 65 and 85 years, and 84% are older than 65 years. In the Framingham study, after adjustment for age and other risk factors, AF developed in men at 1.5 times the rate in women (1). The CHA<sub>2</sub>DS<sub>2</sub>-VASC and R<sub>2</sub>CHADS<sub>2</sub> are the stroke risk assessment tool scores for patients with atrial fibrillation (3). Even though renal failure is independently associated with stroke (2), it was not included in the CHA<sub>2</sub>DS<sub>2</sub>-VASC scoring system, which is used for anticoagulation recommendation in non-valvular AF patients as endorsed by ACC/AHA. Our study retrospectively compared the R<sub>2</sub>CHADS<sub>2</sub> to the CHA<sub>2</sub>DS<sub>2</sub>-VASC risk stratification scores in stroke patients with non-valvular AF and renal failure.

**Methods:** A sample of 171 adult patients admitted over two years from one Midwestern hospital with a diagnosis of atrial fibrillation and stroke were reviewed. Data variables included: name, age, medical record number, sex, race, admitting physician specialty, type of admission, length of stay, admitting diagnosis, renal function and any previously documented CHA<sub>2</sub>DS<sub>2</sub>-VASC scores. If the CHA<sub>2</sub>DS<sub>2</sub>-VASC and R<sub>2</sub>CHADS<sub>2</sub> scores were not documented, they were calculated based on information within the medical record. GFR was calculated using the Chronic Kidney Disease Epidemiology Collaboration (CKD – EPI) formula.

**Results:** The sample of subjects tended to be white (88%) and female (60%). The mean age was 78. The median CHA<sub>2</sub>DS<sub>2</sub>-VASC score was 6 (range 2-9). The median R<sub>2</sub>CHADS<sub>2</sub> score was 4 (range 2-8). The average GFR was 69.77 (range 6-108). A weak, but significant, correlation was found between renal function and CHA<sub>2</sub>DS<sub>2</sub>-VASC score ( $r = -0.263$ ;  $p = 0.0005$ ). A stronger and significant correlation was revealed between the R<sub>2</sub>CHADS<sub>2</sub> and GFR ( $r = -0.70$ ;  $p < 0.00001$ ). CHA<sub>2</sub>DS<sub>2</sub>-VASC and R<sub>2</sub>CHADS<sub>2</sub> scores also were significantly correlated ( $r = 0.627$ ;  $p < 0.00001$ ).

**Discussion:** The risk of stroke in patients with impaired renal function is high. Although CHA<sub>2</sub>DS<sub>2</sub>-VASC and R<sub>2</sub>CHADS<sub>2</sub> are significantly correlated to each other, using R<sub>2</sub>CHADS<sub>2</sub> would be beneficial to assess stroke risk in patients with decreased renal function and non-valvular atrial fibrillation.

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INFLAMMASOME ACTIVATION DUE TO VINYL CHLORIDE METABOLITE EXPOSURE IN NAFLD CAUSED BY HIGH FAT DIET IN MICE.

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**Background.** Vinyl chloride (VC), a ubiquitous environmental contaminant, ranks 4<sup>th</sup> on the ATSDR Hazardous Substances Priority List. A major paradigm shift in environmental research is to assess the impact of underlying disorders that may modify risk. Indeed, studies by our group and others suggest that obesity increases susceptibility to environmental hepatotoxicants (e.g., industrial solvents). NAFLD, the hepatic manifestation of metabolic complications due to obesity, is rapidly emerging as the leading cause of liver disease with its entire spectrum from steatosis to steatohepatitis and cirrhosis and may increase the sensitivity to other insults. Recent studies demonstrate a critical role of the inflammasome in macrophage activation during NAFLD. Inflammasome activation is induced by pathogen-associated molecular patterns (“PAMPs”), such as LPS, as well as by molecules released from dead or dying cells (damage-associated molecular patterns; “DAMPs”). Previously we have shown that VC metabolite chloroethanol (ClEtOH) exacerbated injury and inflammation leading to necrotic cell death in an experimental model of high-fat diet (HFD) induced NAFLD. The purpose of the current study was to investigate the interaction between NAFLD and VC metabolites in the context of inflammasome activation in an experimental model of HFD-induced obesity.

**Methods.** Mice, fed a HFD (42% milk fat) or low fat control diet (LFD; 13% milk fat) for 10 weeks, were administered a bolus dose of ClEtOH or vehicle. Animals were sacrificed 0-24 hours after ClEtOH exposure. Plasma and tissue samples were harvested for determination of liver injury and inflammasome activation.

**Results.** In LFD-fed control mice, ClEtOH caused no detectable liver damage, as determined by plasma parameters (AST and ALT) and histologic indices of damage. In HFD-fed mice, ClEtOH increased HFD-induced liver damage, steatosis, hepatocyte ballooning, infiltrating inflammatory cells, hepatic expression of proinflammatory cytokines and markers of endoplasmic reticulum (ER) stress. VC-metabolite induced cell death favors necrosis due to mitochondrial dysfunction and ATP depletion in this model. Moreover, in animals on a HFD, ClEtOH exacerbated expression of key markers involved in inflammasome activation, such as NLRP3 and IL-1 $\beta$ .

**Conclusions.** Taken together, chloroethanol (as a surrogate VC exposure) can exacerbate liver injury and inflammasome activation in HFD-induced NAFLD. This serves as proof-of-concept that VC hepatotoxicity may be altered by risk-modifying factors such as diet-induced obesity and NAFLD. These data implicate exposure to VC as a risk factor in the development of liver disease in susceptible populations.

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IMPACT OF PULMONARY FUNCTION TESTS ON OUTCOMES IN PATIENTS AFTER LEFT VENTRICULAR ASSIST DEVICE IMPLANTATION

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**Background:** Pulmonary function tests (PFTs) are often part of the pre-implantation assessment for Left Ventricular Assist Device (LVAD) placement. However, limited data exists on the impact of individual components of PFTs on outcomes after LVAD implantation. We hypothesized that components of pre-implantation PFTs may act as a useful tool for prognostication of these patients.

**Methods:** Patients at our center who received LVADs, survived until discharge and underwent PFTs prior to implantation were included in the study. Demographics, baseline clinical characteristics and PFTs were obtained. Patients were followed up until October 30th 2014 or death. PFT parameters were reported as percentages of predicted values. Death from any cause was the primary outcome.

**Results:** 99 patients, who underwent successful LVAD placement and had pre-implantation PFTs during the study period formed the study group. Mean follow-up duration was  $1.6 \pm 0.9$  years. 22 deaths occurred during follow-up. Baseline clinical characteristics, including laboratory parameters were similar between survivors and non-survivors. Pre-operative mean FEV1 ( $54 \pm 15\%$  vs  $62 \pm 18\%$ ,  $P < 0.05$ ), mean forced vital capacity (FVC,  $57 \pm 12\%$  vs  $68 \pm 18\%$ ,  $P < 0.05$ ) and mean diffusing capacity of the lung for carbon monoxide (DLCO,  $43 \pm 11\%$  vs  $55\% \pm 17\%$ ,  $P < 0.01$ ) were significantly higher in survivors. On multivariate analysis a DLCO of less than 50% of predicted was an independent predictor of mortality (OR, 6.4; 95% CI, 2.03 – 20.11;  $P < 0.001$ )

**Conclusion:** Our findings suggest that pre-LVAD DLCO is an independent predictor of mortality and may serve as an important prognostication tool for patients undergoing LVAD implantation.

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CREATING A PATIENT SAFETY CULTURE ONE INCIDENT REPORT AT A TIME

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**Introduction:** Preventable medical errors are the third leading cause of death in the United States behind heart disease and cancer. In order to decrease the number of medical errors, it is vital to establish a culture of patient safety and encourage incident reporting. Physicians, particularly resident physicians, have a historically low rate of reporting incidents.

**Methods:** We completed a resident-led educational initiative to increase rates of incident reporting among Internal Medicine resident physicians at our institution. During week one of their four-week rotation, residents received individual education about incident reports and completed a pre-education survey about their existing knowledge of incident reports and any barriers to reporting. The education included what an incident report is, when it is appropriate to complete an incident report, demonstration of how to file a report electronically, and what happens to reports that are filed. At the time of education, residents also received a pocket-card of instructions of how to file an incident report. After education was completed, weekly "Patient Safety Rounds" were conducted to inquire about any patient safety events that occurred and reinforce the education they had received. At the end of the rotation, the residents filled out a post-education survey again assessing their knowledge of incident reports and any barriers to reporting. The number of incident reports filed by resident physicians was compared pre and post-intervention.

**Results:** A total of 57 residents were included in the intervention over 4 months. Before intervention, 89% knew what an incident report was and 19% knew how to file an incident report. A total of 49 residents completed the post-intervention survey. After education, 100% ( $p = 0.9$ ) of residents knew what an incident report was and 100% ( $p < 0.005$ ) knew how to file an incident report.

Four months prior to intervention, there were a total of 3 incident reports entered by resident physicians. This made up 1.6% of all incident reports during that time period. Four months post-intervention, there were a total of 45 incidents reported by resident-physicians, comprising 16.8% of total incidents reported at our institution ( $p < 0.01$ ).

Most commonly cited barriers pre-intervention were lack of knowledge of how to file an incident report (68%) and lack of knowledge about what needs to be reported (58%). Post-intervention, the most common barriers reported were lack of time (37%) and unclear outcomes of what happens when incident reports are filed (18%).

**Conclusions:** Education of incident reporting, along with reinforcement and promotion of filing incident reports through "Patient Safety Rounds" increased rates of resident reporting.

PRACTICE PATTERN TREATING OLDER WOMEN WITH EARLY STAGE BREAST CANCER AT JOHNS HOPKINS

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**Background:** The National Comprehensive Cancer Network (NCCN) Guidelines incorporated omission of radiation therapy (RT) after breast-conservation surgery in woman age  $\geq 70$  years with stage I, estrogen receptor positive breast cancer who plan to receive endocrine therapy (ET). This guideline change was based on The Cancer and Leukemia Group B C9343 trial. A follow up study demonstrated that there is a wide variation in implementing this change across 13 different NCCN institutions. We evaluated the practice pattern at Johns Hopkins, and sought to construct an internal guideline.

**Methods:** We identified women treated at our institution from 2009-2013 age  $\geq 70$  years at the time of diagnosis and met the C9343 inclusion criteria. RT omission rate was calculated for each year. We explored associations between RT omission and year, age, tumor size, race, nodal status and tumor type with t tests and Fisher's exact tests.

**Results:** A total 544 women age  $\geq 70$  years sought treatment at our institution, and 98 (18%) were candidates for RT omission based on the NCCN guidelines. Mean age was 76.2 years (Range 70-95). Overall RT omission rate was 36/98 (37%), but varied greatly by year (Range 8-56%,  $p=0.03$ ). This variation in omission rate was still present after excluding women who did not tolerate ET (Range 9-67%,  $p=0.02$ ). Older age was associated with higher RT omission rate (mean age 78.7 vs. 74.8,  $p=0.002$ ). Women who did not undergo nodal evaluation had higher RT omission rate (68%) than women who had nodal evaluation (29%) even when the evaluated node(s) were negative ( $p=0.003$ ). The RT omission rate did not vary by race (Caucasians: 24/69, 35%, Non-Caucasians: 11/27, 40%,  $p=0.64$ ), tumor type (ductal: 27/72, 38%, non-ductal: 9/26, 35%,  $p>0.99$ ), or tumor size ( $<1$ cm: 17/37, 46%; 1-1.4cm: 10/34, 29%; 1.5-2cm: 8/25, 32%,  $p=0.35$ ).

**Conclusions:** The implementation of the NCCN guideline, which was based on category I evidence, is not consistent at our institution. Our results suggest that other tools should be used to apply the guidelines more consistently. To achieve this, we have developed a Quality Improvement Protocol that incorporates life expectancy estimate and a brief geriatric assessment to the treatment of all woman age  $\geq 70$  years at our breast cancer clinics.

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AN EVALUATION OF KIDNEY FUNCTION AFTER ISLET TRANSPLANT GRAFT FAILURE

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Purpose of study: Previous studies evaluating kidney function on islet transplant (ITx) recipients with a functional graft have reported contrasting results. Given that the failure of graft function is not an uncommon event in ITx patients, it is important to assess safety, and long term complications on this population. Kidney function is of particular interest since these subjects had a combination exposure to nephrotoxic drugs and long term diabetes. To address this issue the present study aims to evaluate the kidney function in a group of ITx recipients with graft failure (GF) up to 10 years after its occurrence.

Methods: A prospective analysis in 12 participants using demographic, anthropometrical, laboratory data, immunosuppressive and anti-hypertensive therapy. Kidney function assessed by CKD-EPI (Chronic Kidney Disease Epidemiology Collaboration) calculated estimated glomerular filtration rate (eGFR); iohexol clearance GFR and urinary albumin creatinine ratio (UACR) on spot urine samples at study time points up to 10 years post GF at last follow up. Data was compared to collected data from before islet transplant and during immunosuppressant therapy (IS).

Results: Mean age: 49.5±9.3 years; diabetes duration: 34.3±13.6 years; 41.6% males (n=6). Average time to GF: 2.7±1.6 years and study follow up: 10.6±2.2 years, mean eGFR at study enrollment after GF was 89.2 ± 22 ml/min/1.73m<sup>2</sup>. The mean eGFR was not different between study time points (P=0.91), even when divided by time to exposure to immunosuppression (P=0.14). Mean eGFR rate of decline from before ITx to GF was -1.97 ± 0.08 mL/min/1.73m<sup>2</sup>/year, while after GF was -0.06±0.5mL/min/1.73m<sup>2</sup>/year P=0.62. Microalbuminuria prevalence was 16% at baseline; 42% at graft failure, 8.3% at last study follow up (P=0.01). Albuminuria correlations were observed for triglycerides (R=0.38;P=0.01), BMI (R=-0.349;P=0.02) and HbA1c (R=0.43;P=0.01).

Conclusions: ITx recipients maintained stable renal function during post-GF follow up, despite exposure to immunosuppressants for approximately 3 years and worsening of glucose control after GF; a selection bias for ITx patients may account for the stability of renal function. The discontinuation of immunosuppressive therapy might be responsible for the regression of microalbuminuria. The rate of decline in eGFR after GF is lower than before transplant although did not reach statistical significance. Comparisons with a type one diabetes control group would provide further evidence of the lack of impact on kidney function by IS on those with good kidney reserve.

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ELEVATED LIPOPROTEIN(A) IS ASSOCIATED WITH INCREASED RISK OF CARDIOVASCULAR EVENTS IN MEN – A META-ANALYSIS OF PROSPECTIVE STUDIES FOR AWARENESS OF NEED TO STANDARDIZE SCREENING

First Author: Ammu T Susheela, MBBS Ali Jannati, Darshan K Lakshminarayan, Suresh Devabhaktuni, Abdulhamied Alfaddagh, Kiran Singh, C. Micheal Gibson

**Introduction:** We conducted a meta-analysis to investigate the association between high levels of Lipoprotein(a) and risk of cardiovascular events among men.

**Methods:** A comprehensive and systematic review was conducted in PubMed, EMBASE and Cochrane databases to search for observational studies reported from January 1997 to October 2015 that investigated the association between higher levels of Lipoprotein(a) and risk of cardiovascular events among men. Specifically, we searched for studies that included phrases such as “Lipoprotein(a)”, “cardiovascular events”, and “myocardial infarction”. Data extracted from eligible studies were selected for further analysis. Studies that included stroke among cardiovascular events were excluded from analysis. We extracted the raw data including the number of individuals with higher and lower Lipoprotein(a) levels among those with and without cardiovascular events.

**Results:** A total of 4 articles including a total of 6,804 subjects were selected. We used  $\geq 0.259$  mmol/L ( $\geq 10$  mg/dL) as a cutoff value for higher Lipoprotein(a) levels. Out of the 4 articles, 3 of them reported data that allowed for calculating the association between higher Lipoprotein(a) levels and cardiovascular events among men, while the fourth study combined data from both men and women. Considering all four studies, there was no significant association between higher Lipoprotein(a) levels and risk of cardiovascular events (pooled OR = 1.11, 95% confidence interval, 0.73-1.67). However, considering the three remaining studies that provided separate data for men, there was a statistically significant association between higher Lipoprotein(a) levels and risk of cardiovascular events among men, pooled OR = 1.33, 95% confidence interval, 1.08-1.65.

**Discussion:** The meta-analysis shows that there is a significant association between higher Lipoprotein(a) levels and risk of cardiovascular events among men. Evidence regarding such association among women remains inconclusive and needs to be further investigated.

ELEVATED SERUM BETA-HYDROXYBUTYRATE LEVELS (B-HB) IN PATIENTS WITH TYPE 2 DIABETES MELLITUS USING A SODIUM-GLUCOSE COTRANSPORTER 2 (SGLT-2) INHIBITOR

First Author: Simant Singh Thapa, MD, Nitin Trivedi, MD, Abdulkadir Omer, MD, Department of Medicine, Saint Vincent Hospital, Worcester, MA

**Introduction:** In May 2015 the U.S Food and Drug Administration (FDA) issued a drug safety warning indicating that SGLT2 inhibitor class medicines may lead to euglycemic diabetic ketoacidosis (eDKA) with potentially severe consequences. The symptoms and signs of eDKA may be elusive until the clinical status becomes advanced. We have investigated serum B-hb levels as an early parameter of eDKA development in patients with type 2 diabetes mellitus (T2DM) using SGLT-2 inhibitors.

**Methods:** Between June 2015 and October 2015 we measured serum glycosylated hemoglobin (HbA1c), serum creatinine, anion gap and body mass index (BMI) in 15 patients with T2DM before and after SGLT-2 inhibitor use. We measured serum B-hb after SGLT-2 inhibitor use to investigate the association of elevated B-hb levels and the above parameters.

**Results:** 80% of the patients were male, average age  $56 \pm 5$  years and duration of diabetes of  $8 \pm 5$  years. The duration of SGLT-2 inhibitor use was  $160 \pm 79$  days. SGLT-2 inhibitors were used together with metformin in 100 % of the cases, 60% together with metformin and insulin, 13% together with metformin, insulin and a GLP-1 receptor agonist and 6% together with metformin, insulin, GLP-1 receptor agonist and sulfonylurea. With SGLT-2 use, HbA1c levels improved from  $8.1 \pm 1.3$  % to  $7.1 \pm 1.3$  % ( $p=0.003$ ), while BMI decreased from  $33 \pm 4.5$  (kg/m<sup>2</sup>) to  $31 \pm 4.1$  to ( $p<0.001$ ). Serum B-hb level was elevated ( $>2.8$  mg/dL) in 20% of patients ( $n=3$ ,  $26.6 \pm 18.5$  mg/dL, two patients on canagliflozin, one patient on empagliflozin, all male). Serum creatinine levels did not change. The serum anion gap was elevated in 60 % of the patients ( $15.6 \pm 2.52$  mEq/L); however, anion gaps did not differ between patients with elevated and normal B-hb levels. There was no difference in terms of patient age, duration of diabetes, duration of treatment, serum creatinine levels, change in BMI, or change in HbA1c between patients with elevated and normal B-hb levels.

**Conclusion:** SGLT-2 inhibitor class antidiabetic medication use improves blood glucose and body weight control in patients with T2DM. Our small pilot study showed elevated B-hb levels in 20 % of the patients treated with an SGLT-2 inhibitor together with other anti-diabetic agents. The clinical significance of this finding is unclear. Larger randomized controlled trials are needed to understand the relevance of an elevated serum B-hb level in early detection of eDKA in patient using SGLT-2 inhibitors.

FLUOROQUINOLONE RESISTANCE IN ESCHERICHIA COLI URINARY TRACT INFECTIONS AMONG PATIENTS WITH RHEUMATOID ARTHRITIS: DOES USE OF HYDROXICHLOROQUINE MATTER?

First Author: Karen E. Férrez, MD Y. Atisha-Fregoso, A. Ponce de León-Garduño, S. Ponce de León-Rosales

**Introduction:** Rheumatoid arthritis (RA) is a chronic autoimmune disease that requires chronic immunosuppressive treatment. Urinary tract infections (UTI) are common among patients with RA. It has been suggested that hydroxichloroquine (HCQ), a drug used in the treatment of RA, may have antimicrobial activity and induce selective resistance to fluoroquinolones in bacterial species. However, this has not been well established.

**Objective:** To evaluate the association between use of HCQ and development of fluoroquinolone resistance in *Escherichia coli* among patients with RA and UTI.

**Methods:** Retrospective case-control study. We evaluated 225 patients with RA and UTI. Cases were defined as patients receiving HCQ, and controls as those receiving a different treatment. Demographics and clinical features were assessed, and laboratory data was obtained from urine cultures and antibiotic susceptibility tests. Univariate and multivariate analyses were performed.

**Results:** There were 112 subjects in the HCQ group and 113 controls. The mean±SD age in the HCQ group and controls was 51 ± 15.66, and 60 ± 15.35 years, respectively. Duration of RA was 12± 8.67 and 17± 9.48 years in the HCQ and control groups, respectively. Overall, 61% of patients received methotrexate, 50% HCQ, and 29% prednisone. Class I/II functional status was observed in 63% of patients, and class III/IV in 37%. Resistance to ciprofloxacin was found in 102 (45%) patients overall, and it was less common among the HCQ group compared with controls, although the difference was not statistically significant (43% vs. 48%,  $p$ -value = 0.458). The factors associated with resistance to ciprofloxacin in univariate analysis were: UTI within the last six months, previous antibiotic use, diabetes mellitus, and RA functional status. In logistic regression analysis, RA functional status class and previous antibiotic use were the only significant independent predictors of fluoroquinolone resistance.

**Conclusions:** Resistance to ciprofloxacin is common among patients with RA and UTI. We found no evidence that the use of HCQ is associated with fluoroquinolone resistance in patients with RA and *Escherichia coli* UTI. RA functional status class III/IV was a significant independent predictor of fluoroquinolone resistance.

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ESTIMATION OF TEN-YEAR RISK FOR CARDIOVASCULAR DISEASE AND AGREEMENT BETWEEN RISK SCORES IN A LATIN AMERICAN POPULATION

First Author: Alain Sanchez Rodriguez MD, Coauthors: Katya Estefanía Bozada Gutiérrez, Kevin Asiel Ricaño Santos, Aaron Humberto Cruz-del Castillo, Joaquín Gerardo Joya Galeana MD

**Introduction:** Literature that outlines cardiovascular risk prediction in Latin American (LA) populations is limited, there is uncertainty about the reliability and grade of agreement between the available risk scores in this geographical area, yet cardiovascular diseases (CVD) are increasing in rate, morbidity and mortality in LA and are now recognized as the leading causes of death in Mexico.

**Objective:** to assess the cardiovascular 10-years risk and to estimate the grade of agreement between two CVD risk scores in asymptomatic subjects.

**Methods:** From January 2013 to February 2015 we performed a cross-sectional study which included asymptomatic subjects from two centers in the southeastern Mexican state of Veracruz. The 10 – year risk predictive equations for cardiovascular disease applied were the Framingham risk score (FRS) and the American College of Cardiology/American Heart Association risk score (ACC/AHA 2013). Agreement assessment was performed for both predictive models with Kappa index and intraclass correlation coefficient (ICC).

**Results:** Three hundred six patients with a mean age of 52 years (SD  $\pm$  10.8), 69.25% male, Body Mass Index (BMI) 30 m/kg<sup>2</sup> (SD  $\pm$  5.14). The estimated 10-year risk with the FRS equation were for Coronary Heart disease (CHD) = 8.8% (95% CI = 8.08 - 9.67%); for Myocardial Infarction (MI) 4.3% (CI 95% 3.7-4.9%); stroke risk = 1.7% (CI 95%= 1.54 - 2.0%); cardiovascular disease (CVD) 12.4% (CI 95% = 11.2 - 13.6%); Death from CHD 1.8 % (CI 95% 1.54-2.15); death from CVD 2.6 (IC 95%=2.20-3.16); AHA/ACC 2013 10-year risk of CVD or stroke of 8.06% (CI95% = 6.94 - 9.1%). According to the standard cut-off points for high risk patients of the FRS score for CVD (>20%), and the AHA/ACC 2013 (>7.5%) the prevalence of high risk patients were 20.92 (CI 95% 16.4 - 25.9%) and 33.66% (CI 95% = 28-39%), respectively, and the overall agreement between these risk scores for CVD was 87%, with a Kappa index of 0.68 (CI 95% = 0.59 – 0.77, p<0.05). The estimated ICC for absolute agreement was 0.82 (CI 95%=0.31-0.9, p<0.05).

**Conclusion:** we found a high prevalence of high risk patients with the equations applied. There is acceptable agreement between the AHA/ACC 2013 and FRS for CVD score. The AHA/ACC 2013 score classified a highest proportion of individuals at high risk for CVD predicted at 10 years.

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DEMOGRAPHIC FACTORS FOR 30 DAY READMISSION FOLLOWING MYOCARDIAL INFARCTION:  
ANALYSIS OF NATIONWIDE INPATIENT SAMPLE DATA.

First Author: Yashwant Agrawal, MD Final Author: Vishal Gupta, MD MPH

Introduction: Acute myocardial infarction (AMI) is the most common cause of mortality in the USA as per the data released by the CDC. Short term hospital readmissions account for a significant portion of health care budget and much of the focus recently has been on reducing 30 day readmission rate. This study was done to determine demographic parameters associated with 30 day readmission rate after index AMI.

Methods: Nationwide Inpatient Sample (NIS) data was used to extract data of patients discharged after AMI during index admission for years 2009-2013. Patients were identified by Clinical Classification Software (CCS) code 100, corresponding to ICD 9 procedure codes of 4100 - 4109, 41000 - 41002, 41010 - 41012, 41020 - 41022, 41030 - 41032, 41040 - 41042, 41050 - 41052, 41060 - 41062, 41070 - 41072, 41080 - 41082, 41090 - 41092. NIS represents 20% of all hospital data in the US. Patients readmitted within 30 days were identified and demographic parameters associated with high readmission rate identified.

Results: We identified a total of 2,371,867 hospitalizations with AMI during this 5-year period where 15.86% patients were readmitted within 30 days after index hospitalizations. Elder patients with age greater than 65 years (19%), female gender (18%), Medicare and Medicaid insurances (19% each), low median income for zip code (17%) and metropolitan areas (16%) were associated with high 30 day readmission rate. The most common identified cause of readmission from 2009-2011 was coronary atherosclerosis (15%) and from 2012-2013 the cause identified was congestive heart failure (13.5%).

Conclusions: This first national database study identifies the demographic variables associated with high 30 day readmission rate. Strategies to reduce morbidity and healthcare cost should be targeted more on these high risk groups and the identified causes with increased readmission rate. Further studies might be needed to supplement the results.

A QI STRATEGY TO OPTIMIZE THE USE OF THE ASCVD CALCULATOR FOR PRIMARY PREVENTION OF ATHEROSCLEROTIC CARDIOVASCULAR DISEASE (ASCVD), IN AN INTERNAL MEDICINE RESIDENCY PRACTICE (IMRP).

First Author: Amina Chaudhary, MD Neha Jain, MD Kamran Mushtaq, MD Shiny Varghese, MD Oyebimpe Adekolujo, MD Ruaa Al-Ward, MD Shagufta Ali, MD Susan Smith, MD

**Introduction:** Cardiovascular disease (CVD) remains the leading cause of morbidity and mortality in the US with an escalating incidence. Previous guidelines for prevention and management of CVD focused on target low density lipoprotein (LDL) with lifestyle modification and statin therapy which has been proven to reduce mortality and is the cornerstone of primary and secondary prevention.

The American College of Cardiology/American Heart Association (ACC/AHA) Task force released its 2013 Guidelines emphasizing the calculation of the 10-year risk of developing ASCVD using the Pooled Cohort Equation (accounting for age, gender, race, total cholesterol, high density lipoprotein, systolic blood pressure, treatment for hypertension, diabetes mellitus (DM), and smoking status). The new guidelines recommend lipid-lowering therapy for primary prevention of ASCVD in individuals with: 1) LDL-C = 190mg/dl; 2) age 40-75 years with DM; and 3) individuals 40-75 years without DM or ASCVD, but with an estimated 10-year ASCVD risk of = 7.5%.

The aim of this QI project was to increase ASCVD risk calculator use as a decision-aid to initiate statin therapy for primary prevention of CAD in patients at an IMRP.

**Methods:** We conducted a practice survey regarding use of the Pooled Cohort Equation. We collected baseline chart data using our inclusion criteria.

After randomization we reviewed 73 charts. We collected data on demographics, statin use, co-morbidities and documentation of ASCVD risk score. Statistical analysis used SPSS. We met with stakeholders. CQI interventions were applied.

**Results:** Chart Review Data: 70% were females, 15% had h/o ASCVD, 34% had h/o DM. Of non-DM, non-CAD, 41% were on statin therapy but only 2% of those had a documented ASCVD risk score. Three months after CQI intervention, chart review showed that ASCVD calculator use by physicians had increased to 8%.

**Discussion:** Based on the disappointing results we postulated some responsible factors: limited time allotted per patient (ASCVD risk calculation requires approximately three minutes), lack of guideline awareness, continuing use of outdated guidelines which target LDL only.

**Conclusion:** Our QI project revealed that our physicians are not following the latest guidelines. We need to emphasize the importance of using the ASCVD calculator, particularly when seeing a patient without traditional risk factors.

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TRENDS OF ACUTE LOWER-EXTREMITY DEEP VEIN THROMBOSIS EMERGENCY VISITS AND HOSPITALIZATION FROM 2006-2012: REVIEW OF NATIONAL INPATIENT SAMPLE DATA.

First Author: Hardik Chhatrala, MD, MPH; Sandeep Patri MD; Yashwant Agrawal MD

**INTRODUCTION:** Traditionally, patient with acute DVT have been managed with intravenous unfractionated heparin and simultaneous oral anticoagulation. Medical management for the deep vein thrombosis (DVT) has made tremendous progress since early 2000s with increasing use of subcutaneous low molecular heparin weight (LMWH) and simultaneous oral anticoagulation. However, the burden of lower-extremity DVT on US hospitals and its consequences are still less well-studied. This study attempts to determine the trends of lower-extremity DVT burden in the USA healthcare system from 2006-2012.

**Methods:** We queried Nationwide Emergency Department (ED) Sample data for all the emergency room visits with first listed diagnosis of acute lower-extremity DVT using International Classification Code 9 codes (453.40-453.42). Data was extracted for the years 2006-2012. Rate of ED visits, and admission rate to hospitals during ED was calculated.

**Results:** We identified total of 1,050,032 ED visits with first listed diagnosis of lower-extremity DVT from 2006-12 with a steady rise in ER visits from 38.8 per 100,000 in 2006 to 56.1 per 100,000 persons in 2012. Hospital admission rates during ED visits declined steadily from 63.6% to 56.5% ( $p < 0.05$ ).

**DISCUSSION:** Our study reveals that although the rate of ER visits for acute lower extremity DVT has steadily risen from 2006-12, there has been a notable decrease in the rate of hospitalization from the same. Rising ED visits can point towards rising burden of lower-extremity DVT, increased vigilance towards diagnosis and/or its documentation. Rise in DVT risk factors like obesity certainly contributes to rising DVT burden. Declining hospital admission rates can indicate that with the advent of LMWH, health care professionals are becoming increasingly comfortable with the idea of outpatient management of acute lower-extremity DVT. It will be worthwhile looking into patient demographics as well as trends of other DVT risk factors like malignancy, recent surgery/immobilization, hormone replacement therapy, smoking, etc. in these patients to further explain DVT trends in the population. With increasing use of novel oral anticoagulants like Xa inhibitors and direct thrombin inhibitors as monotherapy for acute DVT, we can expect a further decline in the rate of hospital admits from acute DVT.

CHAPTER WINNING ABSTRACT

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**Background:** Hypertension (HPT) is an important risk factor for heart disease worldwide. It is established that hypertension results in abnormal oxygenation response to stress. Additionally, abnormal resting cardiac energetics (PCr/ATP, measured by <sup>31</sup>P Phosphorus MR Spectroscopy, <sup>31</sup>P MRS) has been reported. It is not known if this energetic profile worsens with the increasing energy demand of exercise, and if so, if the blunted oxygen supply response may contribute to this. We hypothesized that cardiac energetics are abnormal at rest and are further impaired during acute exercise in HPT, and that this impairment is related to abnormal oxygenation during stress.

**Methods:** Cardiac <sup>31</sup>P MRS was performed in 17 hypertensive and 20 age, gender matched normals at rest and exercise. BOLD (blood oxygen dependent imaging) and perfusion were acquired at stress (i.v. adenosine,) and rest. Signal intensity change (SI%) and myocardial perfusion reserve index (MPRI) were measured.

**Results:** In normals, there was no change in PCr/ATP during exercise (rest: 2.16±0.08, exercise: 2.15±0.06, P=0.97). Resting PCr/ATP was reduced in HPT (1.63±0.07, P=0.001), and during exercise, there was a further reduction in PCr/ATP (1.49±0.07, P=0.03). There was a significantly reduced BOLD SI% response in HPT (BOLD SI%: 10±2%; normal 20±0.01% P=0.004). MPRI was also significantly reduced in HPT. BOLD SI% and MPRI correlates, (R=0.25, P=0.006). In HPT, there was a significant correlation between exercise PCr/ATP and BOLD SI% (R=0.51, P=0.04)

**Conclusion:** In the hypertensive heart, myocardial tissue hypoxia during stress may play a significant pathophysiological role by inducing adverse metabolic changes in the myocardium.

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NANOPARTICLE ENCAPSULATED PNEUMOCOCCAL SURFACE ADHESION A SUBUNIT (PSAA)  
CONFERRED BOTH SYSTEMIC AND MUCOSAL IMMUNITY

First Author: Daniel E Ezekwudo, MD,Ph,D; Dilip Devineni Ph.D; Ravi Palaniappan Ph.D, Martin Tobi, M.B.,ChB.

**Introduction:** According to World Health Organization, 15% of all death in children under the age of 5 is caused by pneumonia. In USA, about 3-5% of patients (adults and children) die of pneumonia each year. Current pneumococcal vaccine is limited in its efficacy against the strains since polysaccharide vaccines induce a B-cell dependent immune response thus do not protect the host against pneumococcal infection. This is partly owed to the inability of these vaccines to target the mucosa which represents the route of bacterial invasion. In our study, we designed nanoparticles-encapsulated pneumonia subunit antigen which showed great potential in eliciting both systemic and mucosal immunity in BALB/c mice.

**Method:** PsaA antigen containing polycaprolactone polymer/maltodextrin (PCL/MD) nanoparticles were formulated via nanoprecipitation. Using standardized protein quantification assay, the amount of encapsulated pneumococcal antigen was determined. Chemical linkages between the polymers and polymer-protein were examined via spectroscopy. BALB/c mice were used in the study and consist of twenty mice divided in four groups of five. The following regimen was administered; group 1: antigen loaded nanoparticles via oral route; group 2: placebo nanoparticles via peritoneal route; group 3: antigen loaded nanoparticles via peritoneal route. Following treatments, all groups were challenged with a pneumonia bacterial strain (EF3030); both systemic and mucosal immune responses were evaluated and compared to placebo group. Control or naïve mice were administered with the pneumonia challenge strain on day fourteen along with other groups.

**Results:** Study showed elevated serum antibody levels of IgG2a and IgA in the treated mice (group 1) Furthermore, cytokine responses upon *ex-vivo* stimulation of cells from lymphoid organs with PsaA antigen were evaluated and revealed high levels of IL-2, TNF-alpha and IFN-gamma. These results correlate with the PsaA signature serum antibody response of IgG2a. Specifically, elevated serum antibody levels of IgG2a and IgA were detected in group which was orally administered with antigen entrapped nanoparticles upon challenge with a pneumonia strain (EF3030). Bromodeoxyuridine lymphocyte proliferation assay revealed the dual effect of systemic and mucosal targeting in group 1 as evidenced by elevated OD (optical density) of the mucosal lymphocytes compared to other treatment groups. Taken together, it was concluded that the orally delivered nanoparticles appeared to target and induce much higher mucosal and systemic immune response than other treatment groups.

**Summary:** Our study showed that orally delivered PsaA antigens were able to induce local mucosal immune response as indicated by elevated IgA levels and PsaA specific IgG2a levels in the serum. Thus, apart from the inherent advantage of patient compliance, oral delivery of vaccines results in the manifestation of an immune response from both systemic and mucosal lymphoid organs. This formulation design can be extended to other vaccines where both mucosal and systemic immune responses is desired.

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THE BATTLE BETWEEN PAPER AND ELECTRONIC HEALTH RECORDS CONTINUES: A QUALITY IMPROVEMENT PROJECT COMPARING ACCURACY OF PHYSICAL EXAMINATION DOCUMENTATION BETWEEN PAPER AND ELECTRONIC CHARTS

Noora Kazanji, DO Siddhartha Yadav, MD Narayan KC, MD Sudarshan Paudel, MD John Falatko, DO Michael A. Barnes, MD

**Introduction:** The United States health system is in transition from paper to electronic health records (EHR). However, this transition has not been without limitations. There have been multiple reports of inaccuracies in EHR due to the use of pre-specified templates, smart phrases and copying and pasting from prior notes. This study aims to look into one aspect of physician documentation: accuracy of physical examination findings of inpatient hospital notes.

**Materials and Methods:** Five diagnoses (permanent atrial fibrillation, stroke, severe aortic stenosis, amputation and intubation) in which certain examination findings are invariably expected to be present were selected. All patients admitted with these diagnoses between August 2011 and July 2013 were identified. 250 patients were randomly selected in each arm. The accuracy of these diagnoses, baseline characteristics, and secondary outcomes were collected on these patients. All dictated progress notes were excluded.

**Results:** Accuracy was not statistically different between electronic and paper charts. Electronic charts, however, are more likely to have inaccurate documentation rather than to not mention an expected physical examination finding ( $p < 0.05$ ). Paper charts, on the other hand, are more likely to leave out expected physical examination findings rather than to inaccurately document ( $p < 0.05$ ). Electronic charts were also more likely to have more words and systems documented.

**Conclusion:** Overall, accuracy was not different between the two groups. Electronic charts were more likely to have inaccuracies and paper charts were more likely to have omissions ( $p < 0.05$ ). Further studies are needed to tease out the underlying factors driving these differences.

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FACTORS AFFECTING 30 DAY READMISSION RATES AFTER PULMONARY EMBOLISM

First Author: Sandeep Patri, MD Second Author: Monoj Konda, MD Final Author: Yashwant Agrawal, MD

Introduction: Pulmonary embolism (PE) is a serious medical condition associated with major morbidity, mortality and economic burden. Preventable hospital readmissions are a major financial problem for the healthcare system and identifying patient groups at risk of readmission will help address the issue. This retrospective study was performed to determine demographic parameters and major diagnosis related groups associated with 30 day readmission rates after an index PE.

Methods: Nationwide Inpatient Sample (NIS) data was used to extract data of patients discharged after PE during index admission for years 2009-2013. Patients were identified by Diagnosis Related Group (DRG) number 175 and 176 which represents PE with major complications and comorbidities (MCC) and PE without MCC. NIS represents 20% of all hospital data in US. Patients readmitted within 30 days were identified and demographic parameters associated with high readmission rate identified.

Results: We identified a total of 650,819 hospitalizations with PE as the index stay DRG of which 13.44% were readmitted within 30 days after index hospitalizations. Older patients with age greater than 65 years (14.05%), female gender (13.87%), Medicaid insurances (20.49%), low median income for zip code (15.1%) and metropolitan areas (13.71%) were associated with higher 30 day readmission rate. The most common identified cause of readmission in PE with MCC from 2009-2010 was heart failure and shock (4.25%) and from 2011-2013 was severe sepsis (4.8%). The most common identified cause of readmission in PE without MCC from 2009-2013 was PE without MCC itself (10.24%).

Conclusions: This study shows the significant burden of readmissions after pulmonary embolism in a large national database and identifies demographic variables and causes associated with high 30 day readmission rates. Measures such as ensuring good continuity of care transition etc. should be concentrated on the above identified demographic patient groups and also on preventing the various identified aetiologies for readmissions. More prospective studies are warranted to verify the above findings.

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THE EFFECT OF IMPLEMENTING GENE EXPRESSION CLASSIFIER ON OUTCOMES OF THYROID NODULES WITH INDETERMINATE CYTOLOGY

First Author: Jithma Prasad Abeykoon, MD Other authors: Luke Mueller, MD Jonas Paludo, MD Ashish Chintakuntlawar, MD, PhD Rami Mortada, MD

**Introduction:** Thyroid nodule cytology is classified into six categories under the Bethesda Classification System. Two of these categories, atypia of undetermined significance (AUS) and suspicious for follicular neoplasm (SFN), are labeled as 'indeterminate' diagnosis due to inability to clearly distinguish benign from malignant cytology. Afirma Gene Expression Classifier (AGEC) may help to determine the malignant potential of thyroid nodule among patients with 'indeterminate' cytology. This study aimed to assess whether the implementation of AGEC was associated with decreased proportion of surgical recommendations and lower financial cost.

**Methods:** A retrospective cohort study included data from electronic medical records at the University of Kansas School of Medicine-Wichita Endocrine Clinic about patients who underwent thyroid nodule fine-needle aspiration between 2004 and 2014. Data were analyzed using the SAS software for Windows version 9.3 (Cary, NC). Descriptive statistics were presented as frequencies and proportions for categorical variables. Chi-square analysis was conducted to analyze the proportion of surgical recommendations and surgical cytopathologic category of the biopsy results in pre and post implementation of the AGEC.

**Results:** A total of 299 consecutive patients' charts were screened. Sixty-one (20%) patients had an 'indeterminate' diagnosis and underwent further analysis. Out of those 61 patients, 27 (44%) patients underwent evaluation in our institution before AGEC was implemented and 34 (56%) patients underwent evaluation after implementation. There was no significant difference in the rate of 'indeterminate' cytology reported before and after AGEC implementation, 44% vs. 56% respectively ( $p=0.204$ ). However, surgical recommendation for patients with 'indeterminate' finding decreased from 81.5% to 50% ( $p=0.011$ ), before and after AGEC implementation, respectively. This potentially may also have decreased the rate of morbidity and complications linked to thyroid surgery. Cytopathologic diagnosis in patients who underwent surgery before the use of AGEC showed only 20% as malignant compared to 86% of malignant cytopathology in patients who underwent surgery after evaluation by AGEC ( $p=0.0004$ ). Primary cost-benefit estimate showed implementing AGEC has saved \$722/patient in medical evaluation and treatment of a thyroid nodule rendered with 'indeterminate' finding on cytology.

**Conclusion:** AGEC implementation appears to decrease the number of unnecessary surgeries, correctly identify patients who actually need surgical intervention, reduce potential surgical complications and lower financial burden for patients with 'indeterminate' diagnosis of thyroid nodules.

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THE IMPACT OF SMOKING CESSATION ON GLYCEMIC CONTROL IN TYPE II DIABETES

First Author: Joel D Beachey, MD James R Beal, PhD Eric L. Johnson, MD

*Background:* Cigarette smoking is detrimental to glycemic control in diabetes, leading to insulin resistance and increased fasting glucose. Diabetic smokers have greater risk for microvascular and macrovascular complications in addition to increased mortality in comparison to non-smokers. While there is increasing evidence supporting the deleterious nature of smoking in diabetic patients, few studies have investigated whether the harmful effects can be reversed upon smoking cessation.

*Methods:* The medical records of 192 type II diabetic patients with a documented history of at least one pack-year of smoking were retrospectively reviewed. Patients were categorized within either a "cessation group" or "current smoker group." Patients in the cessation group had a documented quit-date during the study period and at least a one-year period of continuous abstinence. Variables recorded in both groups included weight, blood pressure, and hemoglobin A1C. In the cessation group, these variables were recorded six months prior to and one year following smoking cessation. Variables before and after smoking cessation were compared. Data following smoking cessation was also compared to data of current smokers.

*Results:* 75 patients were included in the cessation group, and 117 patients were included in the current smokers group. There was no difference in gender, age, weight, years with diabetes, or smoking pack-year history between the groups. Subjects who quit smoking during the study period had significantly lower mean HbA1c after cessation ( $6.8\% \pm 0.72$  vs  $8.0\% \pm 1.4$ ,  $p < 0.001$ ). Blood pressure also decreased significantly, both systolic ( $129.6 \pm 15.2$  mmHg vs  $124.6 \pm 12.8$  mmHg,  $p = 0.001$ ) and diastolic ( $78.9 \pm 9.6$  mmHg vs  $72.7 \pm 9.7$  mmHg,  $p < 0.001$ ). Patients who quit smoking trended towards weight gain; however, this was not statistically significant ( $91.8 \pm 15.8$  kg vs  $93.8 \pm 15.4$ ,  $p = 0.028$ ). Average hemoglobin A1C in patients who quit smoking was significantly lower in comparison to patients in the current smokers group ( $6.8\% \pm 0.72$  vs  $7.9\% \pm 1.1$ ,  $p < 0.001$ ). BMI was not significantly different between current smokers ( $31.3 \pm 4.7$ ) compared to those who quit ( $31.7 \pm 4.6$ ) ( $p = 0.288$ ).

*Conclusions:* These findings suggest that smoking cessation may significantly improve glycemic control and blood pressure in patients with type II diabetes, despite a trend towards weight gain. Patients who quit smoking had a 15% reduction in hemoglobin A1C. In comparison to current smokers, patients who quit had significantly lower hemoglobin A1C and were more likely to be within goal range. Thus, smoking cessation should be a primary treatment goal in patients with type II diabetes.

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POINT OF CARE RISK STRATIFICATION TOOL FOR ESOPHAGITIS AND BARRETT'S ESOPHAGUS

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Introduction: Screening for Barrett's esophagus (BE), has historically focused on the presence of individual risk factors, particularly chronic gastroesophageal reflux (GERD) symptoms. Society guidelines recommend screening in subjects with multiple risk factors by assuming a positive correlation between the prevalence of BE and the number of risk factors. Though this risk stratification paradigm is not well investigated. This study aimed to assess whether the number of risk factors can serve as a clinical tool to predict the likelihood of erosive esophagitis (EE) and BE.

DESIGN AND METHODS: Olmsted county, MN residents aged 50 or older were recruited for a randomized, population-based study investigating community-based endoscopic screening methods. Anthropometric measurements and validated GI symptom questionnaire responses were obtained. From this comprehensive, prospective data set, the number of risk factors was assessed for each subject. Eight equally-weighted factors were included: male sex, Caucasian race, age > 75 years, GERD, ever tobacco use, alcohol abuse, family history of BE or esophageal cancer and central obesity (waist-to-hip ratio (WHR) for males >0.90 and females >0.85). Esophageal injury was defined as presence of BE and/or esophagitis LA criteria grade B, C, or D. The risk of esophageal injury was analyzed for three groups (0-2, 3-4, or 5-8 factors present) using logistic regression. Multivariable model predicting the presence of EE/BE was developed and receiver operating characteristic (ROC) curve analysis reported.

RESULTS: Of the 205 residents who underwent endoscopic assessment, 38 (18.5%) subjects were found to have esophageal injury or metaplasia (esophagitis (33 total, 29 B, 4 C, 0 D) / BE (16)). 46% were male with a mean (SD) age of 70 (10) years. 33% of subjects had GERD symptoms, and 98% were of Caucasian race. The rate of EE/BE in the group with 0-2 risk factors was 6.1%, while the rate in group 3-4 and group 5-8 was significantly greater at 20% and 30%, respectively ( $p < 0.05$ ). The odds of EE/BE was 3.7 times more (95% CI 1.5, 13.0.;  $p < 0.05$ ) for group 3-4 compared to group 0-2. Presence of 5-8 factors had an odds of 5.7 (95% CI 1.5, 22.5;  $p < 0.05$ ) compared to group 0-2. ROC curve analysis was 0.71.

CONCLUSION: Esophagitis and BE are prevalent in the population. These results confirm the additive nature of risk factors, with risk appearing to increase substantially with 3 or more risk factors and likely continuing to rise as the number of factors increase. This clinical risk stratification tool is based on eight factors which can be easily assessed and obtained by clinicians at point of care; thus increasing the utility of this tool. The multivariate model had a comparable predictive value (AUC of 0.71) to other tools, including the well-known M-BERET prediction model (AUC 0.72).

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POST-INTERVENTION ASSESSMENT OF HEPATITIS C BIRTH COHORT SCREENING

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**Introduction:** Hepatitis C virus (HCV) is the most common chronic bloodborne pathogen in the United States and is a leading cause of complications from chronic liver disease. According to the US Preventative Services Task Force (USPSTF), about three-fourths of individuals in the US living with HCV infection were born between 1945 and 1965 (the “Baby Boomer” birth cohort). Only 25-50% of patients with chronic HCV infection are aware of their infection. The Centers for Disease Control and Prevention (as of August 2012) and the USPSTF (as of June 2013) recommend a one-time screening for HCV infection in all individuals in the “Baby Boomer” birth cohort. In August 2014 charts from five primary care sites in the HealthPartners network were reviewed to evaluate compliance with the updated screening recommendations. Overall only 14% of patients in the recommended age cohort had a known HCV status. Based on this data HealthPartners adopted an electronic medical record (EMR) health maintenance flag for those patients meeting screening criteria in March of 2015.

**Methods:** Five primary care sites in the HealthPartners system were included: Inver Grove Heights Clinic, Midway Clinic, Stillwater Medical Group, University Avenue Clinic and Woodbury Clinic. Data was pulled for a five month period pre-intervention and the same five month period post-intervention (March through July 2014, March through July 2015). Patients born between 1945-1965, seen for routine health maintenance (V70.0 - Routine general medical examination at a health care facility) by Internal Medicine or Family Medicine providers, were included.

**Results:** Overall compliance with one-time screening recommendations increased significantly from 8.1% pre-intervention to 37.9% post-intervention (two sided z-test,  $\alpha < 0.05$ ,  $p < 0.0001$ ). However, pre-intervention compliance with one time HCV screening was lower than expected based on our pilot study (8.1% vs 14%). All five clinics included had increased compliance rates between 2014 and 2015. There was a general trend of increased compliance as time progressed in 2015. Although rates increased significantly after the intervention, >60% of patients in the age cohort remained with unknown HCV statuses.

**Conclusion:** There was a significant increase in compliance with recommendations for one-time HCV screening in the baby boomer age cohort in selected clinics which correlated with the implementation of an EMR flag. Room for improvement remains as > 60% of eligible patients have unknown HCV statuses. Further, increased rates of testing in 2015 nearly doubled the number of patients with positive screens. Additional study is needed to determine if the improvement in screening is limited to these clinics or is reflected in the entire HealthPartners system. Further study is also warranted to determine clinical outcomes of those patients who screened positive.

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INCREASING HIGH-VALUE VENOUS THROMBOEMBOLISM PROPHYLAXIS: A WIN-WIN SITUATION

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Venous thromboembolism (VTE) is a preventable and potentially fatal condition for which hospitalized medical patients are at risk. Compared with the more traditional option of unfractionated heparin, low molecular weight heparin (LMWH) has been shown to be superior for VTE prophylaxis with regard to both efficacy and bleeding risk, and to have a lower risk of heparin-induced thrombocytopenia. Our institution uses the LMWH enoxaparin, which also provides the advantage of once daily dosing and thus fewer injections for patients. There is also less cost associated with once daily dosing, in terms of nursing time expenditure and physical resources necessary to provide the injection itself. Despite the benefits of LMWH, baseline data at our institution revealed that only approximately 1 in 10 hospitalized patients received enoxaparin for VTE prophylaxis. Therefore, a quality improvement (QI) project was initiated to increase the use of LMWH for VTE prophylaxis among medical patients hospitalized on four inpatient general medical resident teaching services at Mayo Clinic Hospital, Saint Mary's Campus during April through August, 2015. To improve the utilization of LMWH for VTE prophylaxis, an interprofessional QI team was formed, consisting of resident and attending physicians with nursing and pharmacy leadership. A systems analysis was performed, with stakeholder interviews revealing prescriber knowledge regarding the use of LMWH for VTE prophylaxis as the largest barrier to its utilization. Several Plan-Do-Study-Act (PDSA) cycles were performed, with interventions including peer-to-peer education, pharmacist-to-resident education, educational posters, educational emails, and performance feedback. Process measure evaluation revealed that while peer-to-peer education and feedback resulted in some improvement in resident knowledge, pharmacist education lead to an even greater improvement. In an effort to promote sustainability, a recurring pharmacist-led educational session has now been incorporated into the monthly unit orientation for new residents rotating on the inpatient general medicine services. Data analysis demonstrated a significant increase in LMWH use among hospitalized medical patients from 10.5% in April to 71.2% in August ( $p < 0.00001$ ). Sustained over a one month period, this increase would conserve over 450 injections and nearly 80 total hours of nursing time. In this manner, usage of LMWH for VTE prophylaxis rather than unfractionated heparin improves the quality, patient satisfaction, cost and efficiency of healthcare, and is a 'win' for patients, nursing staff, and providers.

## IDENTIFICATION OF AN 'EVOLVING' PATTERN TO PREDICT EARLY PROGRESSION OF SMOLDERING MYELOMA TO ACTIVE DISEASE

Praful Ravi, Jeremy Larsen and S. Vincent Rajkumar

**Introduction:** Smoldering multiple myeloma (SMM) is an asymptomatic plasma cell dyscrasia that is found in 15% of newly-diagnosed patients with multiple myeloma (MM), a disease which accounts for nearly 2% of cancer deaths in the United States. The prognosis of SMM varies considerably and there is an urgent need to identify patients at risk of early progression to MM to enable prompt therapeutic intervention. We sought to identify factors associated with progression of SMM, and specifically to recognize predictors of progression to MM within 2years, a risk level deemed sufficient by the International Myeloma Working Group to merit consideration for early therapy.

**Methods:** Patients with SMM, who did not receive myeloma-specific therapy before SMM diagnosis or before confirmed progression to MM between 1995 and 2010 were selected from an IRB-approved database. Baseline and follow-up levels of hemoglobin (Hb), involved immunoglobulin (Ig), serum monoclonal protein (MP), and percentage clonal bone marrow plasma cells (BMPC) were extracted from the electronic medical record. Change in MP, involved Ig, and Hb was defined as “evolving” when  $\geq 10\%$  increase was observed in the first 6months of diagnosis (if MP was  $\geq 3\text{g/dL}$ ) or progressive annual increase in MP in the first 3years after diagnosis (if MP was  $< 3\text{g/dL}$ ), increase in involved Ig of  $\geq 10\%$  in the first year of diagnosis, and decrease in Hb of  $\geq 0.5\text{g/dL}$  in the first year after diagnosis. Statistical analysis was performed using SPSS v.20.

**Results:** 110 patients were eligible for this analysis; median age at SMM diagnosis was 64years and median follow-up was 9.9years. 70 patients (63.6%) progressed to MM, with a median time to progression (mTTP) of 3.9years (95% CI 2.1-5.7). Clinical manifestations at the time of progression were predominantly skeletal lesions (41%), anemia (34%) and renal complications (16%). Median overall survival was 8.7years (6.8-10.6).

On univariate analysis, mTTP was significantly shorter in individuals with evolving MP and/or Ig (n=55, 1.6yrs vs. 12.5yrs), evolving Hb (n=32, 0.9yrs vs. 6.5yrs), and those in whom BMPC was  $\geq 20\%$  at SMM diagnosis (n=43, 1.3yrs vs. 6.5yrs, all  $p < 0.001$ ). All three variables were associated with higher risk of progression to MM on multivariate analysis – evolving MP and/or Ig: HR=4.48 (2.19-9.16),  $p < 0.001$ ; evolving Hb: HR=2.10 (1.06-4.14),  $p = 0.033$ ; BMPC  $\geq 20\%$ : HR=2.30 (1.24-4.28),  $p = 0.009$ .

24 patients (21.8%) had BMPC  $\geq 20\%$  at diagnosis of SMM along with either an evolving pattern of MP and/or Ig or Hb (or both). In these individuals, mTTP was 0.9yrs (0.7-1.2yrs) and 20 (83.3%) progressed to MM within 2years of diagnosis.

**Conclusion:** BMPC  $\geq 20\%$ , and evolving changes in Hb and MP and/or Ig were independent predictors of progression from SMM to MM. Individuals with BMPC  $\geq 20\%$  at SMM diagnosis and either evolving changes in MP and/or Ig or Hb had  $> 80\%$  risk of progression within 2years. While external and prospective validation is required, these preliminary data identify a subset of patients with SMM who would be candidates for early therapy.

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CONTEXT-SENSITIVE CLINICAL DECISION SUPPORT THROUGH INFOBUTTONS IN ELECTRONIC HEALTH RECORDS: A SYSTEMATIC REVIEW

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**Introduction:** Health care providers need timely information to answer questions as they arise in practice. Context-sensitive clinical decision support tools, commonly referred to as infobuttons, are being integrated as part of the "meaningful use" initiative and are now required for certification in electronic health record (EHR) systems in the USA. We conducted a systematic review of infobuttons to determine overall usage, clinical impact in terms of patient outcomes and clinician behaviors, as well as cost of development.

**Methods:** We followed PRISMA standards for systematic reviews. We defined infobuttons as knowledge retrieval tools embedded in an EHR that automatically link to knowledge resources tailored to the specific clinical context (patient diagnosis, medication, lab result, user type, etc). We searched MEDLINE, EMBASE, CINAHL, the Cochrane Registry, Scopus, Web of Science, and ProQuest Dissertations, from their inception to July, 2015. We included studies on infobuttons in a clinical setting that reported comparative outcomes, usage frequency, or clinical impact. We abstracted information on population, setting, study design, duration, quality, sample size and quantitative outcome results. Variability in the context of original articles precluded meta-analytic pooling, so we used narrative synthesis to report our findings.

**Results:** From 598 potentially eligible articles, 22 met inclusion criteria. Two healthcare systems accounted for 82% of the articles. All articles dealt with locally-developed EHR systems. Infobutton overall usage ranged from 0.1-10.6 uses/month/potential user with substantial variation between institutions. Usage seems to have gradually increased over time by 6 to 37% among 3 institutions in one year. In four studies comparing infobuttons with other online resources, infobuttons were used once for every three to seven uses of other online resources. In the four articles evaluating clinical impact, users perceived that infobuttons favorably influenced clinical decisions in 15 to 44% of infobutton uses. User satisfaction was reported for 8 articles, and was universally favorable in terms of use, navigation and appearance. Two articles reported the cost of infobutton software development to range between 10-20 days.

**Conclusion:** Context-sensitive decision support in EHR through infobuttons has the potential to impact clinical work, but research evaluating patient and cost outcomes, and benefits in comparison with other means of access to knowledge resources or cost are lacking. No research has been conducted in the setting of recently-available commercial EHR systems. We also need further research to guide optimal implementation and to identify mechanisms to promote usage uptake.

## QUANTIFYING HEALTH CARE DISPARITIES FOR SCREENABLE CANCERS IN THE MISSISSIPPI DELTA

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**Introduction:** How can we measure disparities in health care? One metric of performance is comparing a disease's mortality-to-incidence ratio (MIR) across different health care systems. Differences in the MIR between regions may reflect an area harboring more virulent disease and/or disparities in health systems (e.g., access to screening, diagnostic services, treatment, and follow-up). Relative poverty and rurality may influence disparities between health systems. The Mississippi Delta is a rural, impoverished area of in which each county in the core region has a poverty rate > 30%. The purpose of this study is to compare the MIR for three screenable malignancies – breast, colorectal, and cervical cancer – between core Mississippi Delta counties and the state's non-Delta counties.

**Methods:** County-level data were obtained from the online Mississippi Cancer Registry (<http://www.cancer-rates.info/ms/index.php>) for the 2003-2012 breast, colorectal, and cervical cancer age-adjusted rates of incidence and mortality. The MIR was calculated by dividing the age-adjusted mortality rate by the age-adjusted incidence rate for the specified time period. Core Delta and non-Delta counties (excluding buffer counties as defined by the Mississippi Cancer Registry) were compared. To determine if rurality could account for detected differences, the MIR of Delta counties was also compared to only those non-Delta Mississippi counties classified as "rural" by the Mississippi Cancer Registry. All comparisons involved a two-sided student T-test and assumed unequal variance between the two samples.

**Results:** For 2003 - 2012:

- The cervical cancer MIR was not statistically significantly different between the Delta (average MIR of all counties in region = 0.49) and non-Delta (MIR = 0.36,  $p = 0.24$ ) counties in Mississippi.
- The breast cancer MIR was statistically significantly different between the Delta (MIR = 0.31) and non-Delta (MIR = 0.21,  $p = 0.03$ ) counties in Mississippi.
- The colorectal cancer MIR was statistically significantly different between the Delta (MIR = 0.46) and non-Delta (MIR = 0.21,  $p = 0.01$ ) counties in Mississippi.

When Mississippi Delta counties were compared to only those Mississippi non-Delta counties classified as rural, the MIR was no longer statistically different for breast cancer ( $p = 0.09$ ) but remained so for colorectal cancer ( $p = 0.02$ ).

**Conclusion:** In Mississippi from 2003 to 2012, the breast and colorectal cancer MIR was statistically different between Delta and non-Delta counties. When comparing Delta counties to rural non-Delta counties in the state, only the colorectal cancer MIR remained statistically different. Further investigation is needed into the biology and health care management of colorectal cancer in the Mississippi Delta to assess what may account for this difference.

## EVALUATING PERCEPTIONS OF AND BARRIERS TO SUCCESSFUL INPATIENT GLYCEMIC CONTROL AMONG RESIDENT PHYSICIANS

William B. Horton, MD S. Calvin Thigpen, MD

**Introduction** The link between uncontrolled hyperglycemia and increased patient morbidity, mortality, and length of hospital stay is well-established.<sup>1</sup> Before educational interventions and policies aimed at improving inpatient glycemic control can be established, institutions should gain a better understanding of how practitioners view inpatient glycemic control. We developed a questionnaire and surveyed resident physicians to examine their perceptions of and barriers to successful inpatient glycemic control.

**Methods** We designed a questionnaire and Institutional Review Board (IRB) approval was granted. We then administered the questionnaire to Internal Medicine and Medicine-Pediatric resident physicians at the University of Mississippi Medical Center to determine their viewpoints regarding the importance of inpatient glycemic control, knowledge of inpatient glycemic target values, and problems encountered when trying to manage hyperglycemia in hospitalized patients.

**Results** Of 87 eligible resident physicians, 73 (83.9%) completed the questionnaire (84.9% Internal Medicine residents; 34.2% first-year residents; 28.8% second-year residents; 28.8% third-year residents; 8.2% fourth-year residents). Most residents (72.2%) agreed that they felt comfortable treating and managing inpatient hyperglycemia and most (56.2%) also agreed that they had received adequate education and preparation; however, only a slim majority (51.4%) could identify appropriate inpatient random glucose target values in non-critically ill patients. Only 50% could correctly identify glycemic targets in critically ill patients and only 41.7% of respondents knew appropriate preprandial glucose targets in non-critically ill patients. Lack of knowledge of basal plus bolus insulin regimens and lack of discussion about glucose management on teaching rounds were both barriers to successful inpatient glycemic control for 51.4% of respondents. When asked to identify the single greatest barrier to successful inpatient glycemic control, lack of education (25.4%), nursing issues such as withholding insulin (22.4%), and system issues such as dietary components and fingerstick glucose/insulin timing (22.4%) were the most commonly cited answers. Fear of hypoglycemia was only the sixth most common response (4.5%).

**Discussion** Most residents reported feeling comfortable managing inpatient hyperglycemia but had trouble identifying appropriate inpatient glycemic targets from the most recent consensus guidelines.<sup>1</sup> Lack of education and nursing/system issues were the most commonly cited barriers to ideal inpatient glycemic management. Limitations of this study include the wording of the questions and Likert scale answers both being subject to interpretation of the respondent and respondents being from one academic medical center. Future interventions should focus on resident education along with improving system environments to aid in successful inpatient glycemic control.

### References

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IMPROVING COMPLIANCE OF GUIDELINE RECOMMENDED SCREENING MEASURES OF HEALTH MAINTENANCE IN TMC-HH CLINICS: A QI PROJECT

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**Introduction:** The electronic medical record system utilized by Truman Medical Center has a built-in Adult Health Maintenance (AHM) tab that was found to be underutilized. Based on guidelines by the United States Preventive Services Task Force (USPSTF), the tab can guide physicians practicing preventive medicine in primary care. The full utilization of this tool may increase the compliance with implementation and documentation of guideline based preventive practices in the ambulatory setting.

**Methods:** Using the primary care panel of Cohort B Internal Medicine residents, pre-intervention data pertaining to Tdap documentation via the AHM tab was collected for the weeks of 09/15/14, 10/20/14 and 11/24/2014. This cohort then received an interactive presentation on 12/30/2014 that focused on utilization and features of the AHM tab. Post-intervention Tdap documentation data for the weeks of 2/2/2015, 3/9/2015, and 4/17/2015 was collected and compared to pre-intervention data. Future phases of this project include educating the remainder of Internal Medicine resident cohorts as well as incoming interns using the same interactive educational tool. The project can also expand to include TMC LW and HH clinics.

**Results:** 10 residents participated in this QI project. The rate of documenting Tdap vaccine administration increased from 8.96% to 24.48% ( $P < 0.0001$ ). The rate of documenting declining of Tdap vaccination increased from 1.40% to 5.73% ( $P < 0.0001$ ). Further, the rate of documentation of vaccine-not-indicated increased from 0.56% to 5.21% ( $P < 0.0001$ ) after the intervention.

**Conclusion:** Education about the availability and features of the EMR-based AHM tab was an effective intervention that improved adherence to the utilization of the tab to implement guideline-based preventive practices in primary care as well as allow more consistent documentation of preventive care. The cohort implemented a plan-do-assess-study quality improvement cycle to identify potential challenges that were reassessed in anticipation for expansion of this project to future phases.

## OUTCOMES OF ACUTE MYELOID LEUKEMIA INDUCTION WITH ADDITION OF CLADRIBINE

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**Introduction:** A standard regimen for acute myeloid leukemia (AML) includes seven days of cytarabine and three days of an anthracycline (7+3). At Saint Louis University (SLU), our preferred regimen is cytarabine 200mg/m<sup>2</sup> by continuous infusion, idarubicin 12mg/m<sup>2</sup> daily, and five days of cladribine 5mg/m<sup>2</sup> daily (IAC or '7+3+5'). This regimen was derived from a Polish Adult Leukemia Group (PALG) study that included patients 60 or younger. The impact of IAC on older patients is important to review considering cladribine adverse effects and outcomes in this population have not been previously reported.

**Methods:** Retrospective analysis of patients who started IAC from July 2012 to August 2014 at SLU with follow-up until September 2015. Mortality, disease response, and adverse events were analyzed, with stratification by age and NCCN risk classification.

**Results:** Of 44 patients identified, 27/44 (61%) were 60 or older, 6/44 (14%) had abbreviated therapy (missing or reduced doses). Thirty-eight of 44 (86%) patients survived to hospital discharge, and 5 deaths occurred before day 28. Additionally, 5/6 of these early deaths occurred in patients aged 60 or older. NCCN stratification showed 7/44 (16%) favorable; 17/44 (39%) intermediate; and 20/44 (46%) poor risk cases. Complete response (CR) occurred in 68% after first induction while 7% required a second round, for a total CR rate of 75%. One-year overall survival was 26/44 (59%). Significantly more deaths (15/18 or 83%) occurred among patients  $\geq$ 60 years old (OR 5.8, 95% CI 1.4-25), and among higher NCCN risk classes. No deaths occurred in favorable risk group, but 7/44 (41%) and 11/44 (55%) occurred in intermediate and high-risk strata, respectively (p=0.039 by Chi-Square test).

**Adverse events:** Diarrhea occurred in 34/44 (77%) patients, 26 (59%) developed rash, and 14/44 (32%) experienced mucositis/stomatitis. Intensive care unit admission occurred in 14/44 patients (32%). Fever was documented in 43/44 (98%) patients, of which 23/44 patients (52%) had a documented infection. For patients surviving to discharge (38/44), median hospitalization was 32 days, neutrophil recovery (ANC>500) occurred at a median of 28 days, and platelet recovery (>50,000) at 29 days.

**Conclusions:** Addition of cladribine to cytarabine and idarubicin is an effective induction regimen for AML with similar rates of CR and treatment-related mortality compared to historical studies. In patients 60 or older, this regimen deserves further study and could be an effective option in those with favorable prognosis and limited co-morbidities. Continued survival analyses and investigation of adverse events will help define the role of this regimen in induction chemotherapy for AML.

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TEMPORAL AND REGIONAL TRENDS IN ACUTE PANCREATITIS: DOES MORTALITY OR SEVERITY VARY WITH DAY OF ADMISSION?

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**Introduction:** Hospitalization on a weekend has been shown to adversely affect clinical outcomes in a wide array of medical conditions, perhaps as a result of reduced level of services or quality of care. We hypothesized that acute pancreatitis (AP) patients may also be subject to this “weekend effect”. Our objective was to determine whether weekend hospitalizations for AP are associated with greater severity and higher mortality compared to weekday admissions.

**Methods:** A retrospective observational analysis was conducted using the Nationwide Inpatient Sample, 2005-2011 including adults (age = 18) with a primary ICD9 code for AP. Demographics, length of stay (LOS), etiology, hospital size, volume, teaching status and region were assessed. Admission day was dichotomized as weekday (Monday-Friday) and weekend (Saturday-Sunday). A comorbidity index was used for risk adjustment. Severe AP was defined as admission resulting in death, endotracheal intubation, respiratory failure, or renal failure. The primary outcome was to compare mortality rates in AP between weekend and weekday admissions. Secondary outcomes included comparing rates of severe AP, complications, LOS, and to analyze differences in mortality and incidence of severe AP with regard to hospital size, hospital teaching status, and region in the US.

**Results:** Of a total of 1,851,254 cases of AP identified, 479,628 (25.9%) occurred on a weekend. The overall mortality rate (1.00% vs 1.03%,  $P=0.024$ ) and LOS (4.0 vs 4.0 days) did not differ between weekend vs. weekday admissions; there was a minimal observed difference in severe AP rates (8.6% vs 8.4%,  $P<0.001$ ). Respiratory failure was more common on the weekend (2.1% vs. 1.9%,  $P<0.001$ ). Rates of renal failure, surgical intervention, and other complications were equivalent. Mortality rates were slightly higher in teaching vs. non-teaching (1.0% vs. 0.9%), urban vs. rural hospitals (1.1% vs. 0.9%) and large hospitals vs. medium or small (1.1% vs. 0.9% vs. 0.8)  $P<0.001$ . No seasonal or regional differences in AP severity or mortality were found.

**Conclusions:** Mortality rates for AP did not differ between weekend and weekday admissions. This is likely due to improvements in diagnosis and treatment, aggressive fluid hydration, widespread implementation of standardized prognostic scoring systems and treatment protocols - which may overcome the effect of reduced staffing on weekends. Higher mortality and greater severity were noted in large, urban and teaching hospitals, a result of confounding by the higher proportion of severe AP cases seen at such institutions. Also respiratory failure occurred more often on weekends. This may be due to a lower threshold for intubation and mechanical ventilation for critically ill patients during the weekend.

## UNILATERAL CONDUCTION HEARING LOSS DUE TO CENTRAL VENOUS OCCLUSION

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**Abstract** Central venous stenosis is a well known complication in patients with vascular access for hemodialysis. We report 2 cases of patients on hemodialysis with arteriovenous fistulas developing reversible unilateral conductive hearing loss secondary to critical stenosis of Central Veins draining the arterio-venous dialysis access. A proposed mechanism for the patient's reversible unilateral hearing loss is pterygoid venous plexus congestion leading to decreased Eustachian tube patency. Endovascular therapy was conducted to treat the stenosis and both patients hearing loss was returned to near normal after successful central venous angioplasty.

**Introduction** End stage renal disease is prevalent worldwide. As such, the advent of hemodialysis has become one of the more prominent medical advances of the century. There were 3.2 million patients on hemodialysis for ESRD at the end of 2013 [7]. This number is rapidly growing, and along with this rapid growth come a multitude of complications. In recent years the field of vascular access for patients requiring hemodialysis has had numerous advances in arteriovenous fistulas and grafts. However, despite advances, there are still many complications and unforeseen pathologies.

In current literature, central vein disease (CVD) is defined as greater than 50% narrowing of the thoracic central veins. These veins include the superior vena cava (SVC), brachiocephalic (BCV) and subclavian (SCV) [5]. The incidence of CVD has been reported to be as high as 23% in the total dialysis population and 41% in those with access-related complaints [6]. Many fistulas do not sustain dialysis due to poor maturation, thrombosis, or critical stenosis [3,4]. Central Venous stenosis and occlusion are a result of high vascular wall shear stress due to the presence of AVF or AVG upstream and vascular compression or distortion frequently seen in aging and due to previously placed central venous catheters [1]. Once critical stenosis has developed patients are at risk for central venous occlusion, which can result in face, neck, or arm swelling, along with other severe disabling complications [2,11].

After careful literature review, there is a paucity of data regarding the development of reversible unilateral conductive hearing loss secondary to critical stenosis of venous access. This disabling symptom of central venous occlusion has gone under recognized. We two case reports of reversible unilateral conduction hearing loss due to central venous occlusion. Both of these patients were on hemodialysis and hearing returned to near normal after central venous angioplasty.

**Case Report:** *Case #1:* Our first case is of a 66 year old male with ESRD on hemodialysis for the past 8 years. His vascular access is a left upper arm brachiocephalic arteriovenous fistula. He presented to our vascular access center with left arm swelling. He also complains of hearing loss from his left ear off and on for the past five months. On further inspection, we noticed a wick of cotton in the patients left ear that he places before sleeping because he hears a continuous, loud streaming noise. Physical examination showed a left upper arm Brachial Cephalic fistula which was hyperpulsatile on palpation and does not collapse on raising the arm. The patient was taken for an angiogram of his AV fistula, which showed a 90% stenosis of the left innominate vein. He underwent successful venous angioplasty with a 14mm x 40mm atlas venous angioplasty balloon to 10% residual. Patient had

significant improvement in left arm swelling, and most noticeably had significant improvement in hearing immediately following angioplasty in the recovery area.

*Case #2:* Our second case is of a 71 year old female with past medical history of hypertension, diabetes mellitus type II, and ESRD on hemodialysis for the past 2 years. Her vascular access is a left upper arm brachiocephalic arteriovenous fistula. She presented to our vascular access center with left arm and left sided facial swelling. She also complained of gradual left sided hearing loss for the past 2 months. Physical Examination revealed a left upper arm Brachial Cephalic fistula which was hyperpulsatile on palpation and did not collapse upon raising the arm. The patient was taken for an angiogram of her AV fistula, which showed a 80% stenosis of the left innominate vein. She underwent angioplasty with a 14mm x 40mm atlas venous angioplasty balloon to 20% residual. Immediately post procedure she had significant improvement in her hearing. She has since returned to our access center three times with similar complaints of arm swelling and hearing loss both of which resolve after central venous angioplasty.

**Discussion** Unilateral reversible conductive hearing loss is an uncommon but under recognized consequence of central venous occlusion. The exact mechanism of deafness, although not known, is likely due to swelling and congestion of the eustachian tubes [ET].

The Eustachian tube is a narrow tube that connects the middle ear to the back of the nose. Blockage of the Eustachian tube isolates the middle ear space from the outside environment. The lining of the middle ear absorbs the trapped air and creates a negative pressure that pulls the eardrum inward. When it becomes stretched inward, patients often experience pain, pressure, and hearing loss. Long-term blockage of the Eustachian tube leads to the accumulation of fluid in the middle ear space that further increases the pressure and hearing loss [10].

There are several muscles that allow for opening of the eustachian tube. The principal and perhaps only dilator of the tube is the tensor veli palatini (TVP)[9]. The TVP functions to tighten the anterior part of the soft palate and assist in opening the pharyngotympanic tube. Studies have shown that abnormal insertion of the TVP into the cartilage of the Eustachian tube was found to cause a functional obstruction that led to an increase in incidence of otitis media. This anatomical relationship between the TVP and the Eustachian tube is well described in literature.

Concurrently, the pterygoid plexus is the main venous drainage for the eustachian tube [9]. The proposed mechanism for our patient's reversible unilateral hearing loss is congestion of the pterygoid venous plexus (PVP) leading to decreased Eustachian tube patency. Oshima et. al [8] study used MRI to evaluate the anatomical relationship of the muscles of the neck and the PVP before and after neck compression. The results showed that the lateral pterygoid muscle became enlarged after neck compression. Simultaneously, the volume of the PVP observed between the medial pterygoid muscle and tensor veli palatini muscle was increased. The increased volume of the PVP led to protrusion of the ET anterior wall to the luminal side, and thus decreased ET patency [8]. Given this anatomical relationship, we postulate that in our patients, central vein occlusion led to PVP congestion, which in turn led to compression of the ET.

The loss of ET patency is what likely contributed to our patients' unilateral hearing loss. After adequate venous outflow was restored with angioplasty our patients' hearing was restored. These symptoms can be quite disabling to patients and significantly affect their quality of life. Timely recognition of the cause and treatment can reverse deafness, improve patient's quality of life, and prevent disability from becoming permanent.

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PR SEGMENT DEPRESSION: A PREDICTOR OF MALIGNANT PERICARDIAL DISEASE

First Author: Monica Ahluwalia, MD Second Author: Rupal O'Quinn, MD Third Author: Jackson Liang, DO Fourth Author: Bonnie Ky, MD Fifth Author: David Callans, MD Sixth Author: John Kucharczuk, MD Seventh Author: Joseph Carver, MD

**Introduction:** Electrocardiographic changes may manifest in patients with pericardial effusions. PR segment changes are frequently overlooked, but when present, can provide diagnostic significance. The diagnostic value of PR segment changes in determining benign versus malignant pericardial disease in cancer patients with pericardial effusions has not been investigated. We aimed to determine the relationship between PR segment changes and malignant pericardial disease in cancer patients presenting with pericardial effusions.

**Methods:** Consecutive patients with active malignancy who underwent surgical subxiphoid pericardial window by a single thoracic surgeon between 2011 and 2014 were included in this study. Pre- and post-operative electrocardiograms (ECGs) were reviewed and "significant PR changes" were defined as  $\geq 0.5\text{mV}$  PR-segment depression or elevation. Pericardial fluid cytology, flow cytometry and tissue biopsy were evaluated. Baseline characteristics and co-morbidities were compared between cancer patients with benign and malignant pericardial effusions.

**Results:** A total of 26 patients with active malignancy and pericardial effusion who underwent pericardial window over the study period were included. Eighteen (69%) patients had isoelectric PR segments, of whom none (0%) had evidence of malignant pericardial disease (100% negative predictive value). Eight (31%) patients had significant ECG findings (PR segment depression in leads I, II and/or aVF as well as PR elevation in aVR/V1), all 8 (100%) of whom had pathologically confirmed malignant pericardial disease (100% positive predictive value). PR segment changes in all 8 patients persisted (up to 11 months) on post-operative serial ECGs. The PR segment changes had no relationship to heart rate or the time of atrial-ventricular conduction.

**Conclusions:** In patients with active cancer presenting with pericardial effusion, the presence of PR segment changes is highly predictive of active malignant pericardial disease. When present, PR changes typically persist on serial ECGs even after pericardial window.

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## MET CALLS....HOW MUCH CAN WE AVOID?

First Author: Bishoy Hanna, MD Additional Authors: Aileen Hocbo MD, Dominick Zampino DO, FACP

Introduction: MET calls are medical urgencies requiring prompt attention by the medical team. MET calls are associated with up to 20% in-hospital mortality.

Methods: We retrospectively reviewed a pilot sample of MET calls in a community teaching hospital over a period of two months. We examined all non-code blue MET calls to investigate whether or not they could have been anticipated or avoided.

Results: We reviewed a total of 93 MET calls over a 2 months period. Clinical scenario and severity of illness clearly indicated the potential for clinical deterioration in 54 (58%) of the cases. 38(40.9%) MET calls were deemed avoidable while 39 (42.0%) MET calls were clearly unavoidable. The remaining 17 were not clearly discernable to fit under either category. The two most common causes of avoidable MET calls were unclear goals of care (23.7 %) and errors in medication reconciliation (23.7 %). Other avoidable scenarios included: under-resuscitation, documentation and/or communication errors, improper use of precautions, delay of definitive treatment by a specialist and missed correct diagnosis.

Discussion: Clarifying realistic goals of care, particularly in terminally ill patients, is of paramount importance. Such clarification can frequently circumvent the utilization of unnecessary interventions, tests, and improve patients' end of life experience. Palliative care consultation with continued follow-up can capture optimal opportunities to approach patients and families and clarify goals of care. Medication errors were responsible for the avoidable MET calls included narcotics overuse and clinical institute withdrawal assessment (CIWA) protocol underutilization.

Conclusion: Avoidable MET calls may represent as high as 40% of all non-code blue MET calls. Early involvement of the palliative care team in the treatment of terminally ill patients is a crucial component of their management. Careful and complete medication reconciliation is likely to decrease medication errors and minimize avoidable MET calls.

## HEPATITIS B CAN REPLICATE IN PLACENTAL CELLS LEADING TO TRANSPLENTAL TRANSMISSION OF HBV TO THE BABIES

First Author: Chandana Pande, MBBS Puja Sakhuja, Sheetalnath, Ekta Gupta, Ashish Kumar, Chinmay K Mukhopadhyay, Shiv Kumar Sarin

**Introduction:** During hepatitis B virus (HBV) entry into a cell, the hepatitis B surface antigen remains outside; hence demonstration of surface antigen inside the cell by immunohistochemistry (IHC) is a reliable method of establishing HBV replication inside the cell. HBV replication has been demonstrated in a variety of extra-hepatic tissues and cell types. Whether HBV replicates in placenta and its possible pathogenetic role in transplacental transmission of HBV has not been fully explored. We aimed to document HBV replication in placenta by demonstration of surface antigen by IHC and to correlate this finding with HBV transmission in the neonates of mothers having chronic hepatitis B.

**Patients and Methods:** Pregnant mothers with chronic hepatitis B, not on antiviral therapy, were enrolled. During delivery their placenta and cord blood from the baby were collected. The babies received HBV vaccine and HBIG soon after the delivery. Hepatitis B surface antigen was studied in the placental cells by IHC. Cord blood was studied for HBsAg and HBV DNA.

**Results:** Thirty-seven mothers were studied. The median HBV DNA level of these mothers was  $4 \times 10^4$  IU/mL (range 9 to  $3 \times 10^8$  IU/mL). Twelve (32%) of these women were HBeAg positive and rest HBeAg negative. IHC was positive for surface antigen (Figure) in 11 of 37 (30%) placenta while it was negative in 26 (70%). Of the 11 mothers in whom IHC was positive 7 (63%) babies were positive for HBsAg in their cord blood. Of the 26 mothers in whom IHC was negative, significantly less number of babies were positive for HBsAg in the cord blood (7/26 [27%];  $p < 0.05$ ). The median HBV DNA level in babies positive for HBsAg was  $5 \times 10^4$  IU/mL (range 81 to  $1 \times 10^8$  IU/mL).

**Conclusion:** Our study shows HBV replication in the placental cells leading to high transmission rate of HBV to the babies. Further studies would be needed demonstrating various replicative intermediates in placental cells to delineate the life-cycle of HBV in the placental cells. Figure: Focal cytoplasmic positivity (arrows) for HBsAg in syncytiotrophoblasts.

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MEASURE OF APPROPRIATENESS IN THE PLACEMENT OF INTRAVENACAVAL FILTERS AMONG GUIDELINES FROM MAJOR MEDICAL SOCIETIES

First Author: Braghadheeswar Thyagarajan, MD, Shil Patel DO, Natalie Swergold MD, Sayee Sundar Alagusundaramoorthy MD, Isha Verma MD, Thomas Baker, Margaret Eng MD

Introduction: 1% of hospital admissions in the United States is for venous thromboembolism. There is a large increase in the number of Intravenacaval (IVC) filters inserted each year. With healthcare focusing towards a movement of quality care along with value based purchasing, it has created significant interest among physicians in measuring and defining appropriateness in the treatment of medical conditions. We compared the indications for which the IVC filter was placed in our hospital with the current guidelines by three of the major medical societies, American Heart Association (AHA), American College of Chest Physicians (ACCP) and Society for Interventional Radiology (SIR) to compare the measure of appropriateness in the placement of IVC filters among the guidelines

Methods: We conducted a retrospective review of charts of all patients who underwent placement of an IVC filter in our hospital from January, 2010 to January, 2015. The ICD 9 code for the placement of IVC filter was used to identify these charts. Each patient chart was reviewed for patient demographics, indication, complications and follow-up. The indications were compared with the guidelines as defined by AHA, ACCP and SIR

Results: During the 5 years, 592 patients underwent the procedures which included 233 men and 359 women who had a mean age of 67.2 +/- 17.4. 75.8% filters were inserted by vascular surgery and 24.2% filters were inserted by interventional radiology. 1.8% of the patients had some form of complication either during insertion or retrieval. On comparing the indications, we had 35.5%(AHA), 30.9%(ACCP), 35.5%(SIR) which were appropriate, 4.4%(AHA), 4.4%(ACCP), 47.4%(SIR) which were relatively appropriate and 60.1%(AHA), 64.7%(ACCP), 17.1%(SIR) which were not appropriate respectively. Prophylaxis for bariatric surgery (14.4%) and high risk for fall (11%) were some of the commonly used indications not defined clearly as per the current guidelines. Chi-square test was used to compare the guidelines which showed there was no statistical difference between AHA and ACCP ( $p = 0.243$ ), while statistical difference was present between AHA and SIR ( $p < 0.001$ ) and ACCP and SIR ( $p < 0.001$ ).

Conclusion: This study demonstrates that there are significant differences between the guidelines from major medical societies for the placement of IVC filter which leads to compelling discrepancies amongst physicians in their decision for the placement of IVC filters. Amendments should be considered among the major medical societies for more uniformity among their guidelines. Further studies are required to validate commonly used indications such as high risk for fall and bariatric surgery which are currently not appropriate under the current guidelines

## THE ROLE OF ALCOHOL ABUSE AND TOBACCO USE IN THE INCIDENCE OF EARLY ACUTE CORONARY SYNDROME

First Author: Hassan Alkhawam MD Raef Madanieh MD, Mariya Fabisevich MD, Robert Sogomonian MD, James Nguyen MD, Mohammed El-Hunjul MD, Sumair Ahmed MD, Timothy J Vittorio MD

**Objective:** To investigate the synergistic role of alcohol abuse/dependence and tobacco use in the early incidence of acute coronary syndrome (ACS).

**Methods:** A retrospective chart analyses of 8,076 patients diagnosed with ACS between 2000 to 2014, defined by ICD-9 codes for acute MI, alcohol abuse/dependence and tobacco use. Average age of ACS was calculated for the general population. Patients were then divided into 4 subgroups based on alcohol abuse/dependence and tobacco use status as follows: non-alcoholic non-smokers, non-alcoholic smokers, alcoholic non-smokers and alcoholic smokers.

**Results:** The mean age of our 8,076 ACS patients' population was ~59.5 (95% CI 59.2-59.8). Patients with history of alcohol abuse/dependence appeared to develop ACS ~8.7 years younger than their non-alcoholic counterparts [Alcohol abuse groups: mean age 55.1 years old and 95% CI 52-58 versus Non-alcoholic group: mean age 63.8 and 95% CI 63.6-63.9,  $p < 0.0001$ ]. When tobacco use is incorporated as a risk factor, those with both alcohol abuse/dependence and tobacco use [Alcohol Smoker group: mean age 51.1, 95% CI: 48-54.2] seemed to develop ACS ~5 years earlier than those with history of either Smoker-Non-alcoholic [Mean age: 56.3, 95% CI: 55-55.7,  $p = 0.002$ ] or Alcoholic Non-smoker [Mean age: 56.1, 95% CI: 54.6-57.6,  $p < 0.0001$ ], and ~20 years earlier when compared to those with neither alcohol abuse/dependence nor tobacco use [mean age: 71.3, 95% CI: 71-71.6,  $p < 0.0001$ ].

The mortality rate of ACS in alcoholic abuse/dependence group was 9.1% versus 5.7% in non-alcoholic abuse patients (Odd Ratio (OR): 1.7,  $p = 0.1$ ). 30-days readmission in the alcoholic abuse/Dependence group was 18.6% versus 11.24% in non-alcoholics-abuse (OR: 1.8,  $p = 0.03$ ). Length of hospitalization was higher in ACS alcoholics patients' ~9.3 days versus ~5.2 days in non-alcoholics patients ( $p < 0.001$ ).

**Conclusion:** Alcohol abuse/dependence appears to be a risk factor for earlier ACS. In our population, the average age of ACS incidence in alcoholic patients was significantly earlier than non-alcoholic patients. Furthermore, alcoholic patients who also used tobacco developed ACS at an even younger age when compared to those who had history of either alcohol abuse/dependence or tobacco use alone, suggesting a possible synergistic effect of these two risk factors in developing early ACS. Healthcare intervention in this population through screening, counseling and education regarding alcohol abuse/dependence and smoking cessation is warranted to reduce early ACS morbidity and mortality.



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EFFECTS OF HUMAN IMMUNODEFICIENCY VIRUS (HIV) INFECTION IN EARLY INCIDENCE OF SYMPTOMATIC CORONARY ARTERY DISEASE

First Author: Hassan Alkhawam, MD, Amar Ashraf, MD, Denise Torres, MD, Joseph R. Masci, MD

**Background** Effective anti-retroviral therapy of HIV infection in resource-rich settings dramatically reduced the AIDS-related events. Now, non-AIDS-related events are the major causes of morbidity and mortality in HIV-infected individuals. In particular, cardiovascular disease (CVD) has emerged as an important cause of death in HIV-infected patients.

Data on the effect of CD4 count and HIV- viral load on cardiovascular disease is not consistent. Most studies suggest that lower CD4 count and higher HIV- viral load are associated with greater cardiovascular risk. In this study, we evaluated the early incidence of symptomatic coronary artery disease (CAD) in HIV-infected patients and correlated that to multiple variables- CD4 counts, viral load, age, sex and anti-retroviral medications.

**Method** A retrospective analysis of 9,320 patients who were admitted to hospital from 2006 to 2014 for chest pain and underwent coronary angiography. One hundred and five patients had documented HIV-Infection. We assessed the degree of CAD with coronary angiography as obstructive CAD (left main stenosis of >50% or any stenosis of >70%), non-obstructive CAD (>1 stenosis >20% but no stenosis >70%) and normal coronaries (no stenosis >20%).

**Result** Out of 9,320 patient with chest pain, 105 (1.1%) patients had documented HIV infection. Of 105 patients with documented HIV, 75 (71.5%) found to have CAD based on coronary angiography finding.

Patients with HIV-infection had more prevalence of CAD than non-HIV patient (OR: 2.2, 95% CI: 1.2-3.8,  $p=0.007$ ). The average age of CAD in HIV-infected patients was 54 versus 63.3 years old in non-HIV ( $p<0.001$ ). Out of 75 patients with CAD, 15 (20%) presented with STEMI, 33 (44%) with NSTEMI and 27 (36%) with stable angina.

Sub-group analysis revealed, the HIV-infected patients had more non-obstructive CAD when compared with non HIV-infected with CAD (OR: 2, 95% CI: 1.1-3.7,  $p=0.04$ ) and had more obstructive CAD but did not achieve statistical significance ( $p=0.1$ ).

Among HIV patients, 76% were males versus 24% females with HIV and CAD. The average CD4 counts of HIV-infected patients with CAD were 504 versus 327 in HIV-infected without CAD ( $p=0.01$ ). Furthermore, 52% of HIV patients with CAD had detectable viral load versus 27% of HIV-infected patient without CAD (OR: 3, 95% CI: 1.17- 7,  $p=0.02$ ). Of 75 patients with HIV-infection and CAD, 65 (87%) were on ART versus 12 out of 30 (40%) HIV-infected patients without the evidence of CAD (OR: 9.7, 95% CI: 3.6-26,  $p<0.0001$ )

**Conclusion** Our study confirmed the higher prevalence of CAD in HIV-infected patients. However, our data does not support the notion that higher CD4 count and antiretroviral therapy associated with lower risk of CAD.

However, we found detectable viral load associated with higher incidence of CAD. Further study needed to understand the effect of HIV-infection and antiretroviral treatment on CVD risk.

RELATIONSHIP BETWEEN HIATAL HERNIAS AND GERD IN PATIENTS ON PPI THERAPY

First Author: Sayf Bala, MD second Author:Sama Alchalabi, MD Third Author:Amine Hila, MD

**Background:** 24-hour Multichannel intraluminal impedance and pH (MII-pH) esophageal monitoring allows detection of Gastro-esophageal reflux (GER). MII-PH allows identification of acid and non-acid reflux. Hiatal hernia (HH) is more common in Western countries. Complications of HH are frequent and are usually related to reflux.

**Aim:** To evaluate the effect of the presence and size of HH on the GER characteristics assessed by MII-pH.

**Method:** Retrospective review of 119 consecutive adult patients on proton pump inhibitor (PPI) therapy who underwent an upper endoscopy and MII-pH studies (81 Females, mean age =52 years) between 2009 and 2014. Presence and size of HH was identified endoscopically. The group was divided based on the presence of HH and then subdivided based on the size of HH: small HH =2-3cm, medium HH=4-5cm and large HH =6cm. MII-pH reports were reviewed to assess GER characteristics.

**Results:** 63/119 (52%) patients had HH. There was no statistically significant difference between the presence and size of HH and GER characteristics (table)

	Small HH N=50 (79%)	Medium HH N=9 (14%)	Large HH N=4 (0.06%)	No HH N=56 (48%)	P -value <0.05  Kruskal-Wallis test  (size of HH vs no HH)	Hiatal hernia N= 63 (52%)	P value <0.05  Mann Whitney test (HH vs no HH)	
Mean age±SD (years)	57.00±15.81	55.00±15.28	58.00±10.17	51.09±17.62		51.7±15.62		
Mean values	Upright distal esophageal %time pH<4	6.792	5.762	1.750	4.973	0.7114	6.579	0.90
	Supine distal esophageal %time pH<4	9.430	11.95	9.250	8.871	0.4462	9.949	0.51
	Total distal esophageal %time pH<4	7.910	11.47	11.58	6.709	0.1438	8.644	0.70
	Upright gastric % time pH<4	40.19	40.35	53.55	37.65	0.9001	40.22	0.44
	Supine gastric % time pH<4	47.71	50.15	61.83	43.3	0.6291	48.2	0.43

Total gastric % time pH<4	42.20	43.78	55.93	41.2	0.7694	42.5	0.81
Number of acid reflux episodes	5.480	5.615	7.750	6.268	0.3143	5.508	0.58
Number of weakly acid reflux episodes	9.920	27.77	64.25	12.36	0.5313	13.60	0.68
Number of non-acid reflux episodes	1.120	0.07692	0.0	0.4286	0.7406	0.9048	0.63
number of Total reflux episodes	15.16	33.46	72.00	18.73	0.2604	18.94	0.4

**Conclusion:** In patients on PPI therapy, HH does not seem to increase GERD as measured by MII-pH.

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PREVALENCE OF MALIGNANCY IN A VETERANS COHORT OF PATIENTS WITH OBSTRUCTIVE SLEEP APNEA

First Author: Rami El-Yousef, MD Adrian Cargill, MD; Miriam Cohen NP; Michael Cutaia, MD; Mohammad Al-Ajam, MD

Sleep disordered breathing has been established as an independent risk factor for daytime sleepiness and risk of accidents as well as adverse cardiovascular outcomes. Recent reports about association between sleep disordered breathing and the incidence of cancer as well as worsened mortality rates in cancer patients emerged.

Campos-Rodriguez et al reported a statistically significant higher cancer incidence in patients who experienced the most severe intermittent hypoxia spending 12% of their total sleep time (TST) with oxyhemoglobin saturation <90% (TS90%) compared to patients with the least severe intermittent hypoxia TS90%<1.2% of TST . The association in this cohort of 4910 patients was most significant for men under 65 with an adjusted hazard ratio of cancer incidence of 2.95. Nieto et al, reported a relative hazard of death due to cancer among patients with severe obstructive sleep apnea to be 4.8 times compared to those with no respiratory disordered breathing.

Kendzerska et al, reported a 5.1% cancer prevalence at time of diagnosis of obstructive sleep apnea in 10149 patients. After adjusting for age sex BMI and smoking, they found no association either with cancer prevalence or cancer incidence with either apnea hypopnea index or intermittent hypoxia. The authors attributed this to a much younger study population and to a lower BMI leading to a lower pretest probability of cancer compared to other cohorts

**Methods:** We reviewed a cohort of 251 US veterans who had an in lab polysomnograms (n=103) as well as portable home PSG (n=148) at the Brooklyn veterans affairs hospital between January 1/2012 and December 31, 2012. Intermittent hypoxia related to obstructive sleep apnea and any tissue proved malignancy between 1990 up to 6 months after sleep study date was recorded. Apnea hypopnea index was not investigated given the new rules for reporting Respiratory event index for home sleep apnea testing initiated in July 2015.

**Results:** The 40 patients who spent more than 12% of their total sleep time with oxyhemoglobin saturation <90% (TS90%) had a 1.7 fold increase in cancer prevalence compared to the 138 patients who spent only 1.2% of their TST with SpO2<90%. However this difference was not statistically significant. This is in line with the study of Kendzerska et al from Canada. However, we did have a statistical difference in mean age between the 2 groups. Patients who desaturated higher were older. Unlike the Kendzerska's paper older age did not translate to higher prevalence. Our findings and those of the Canadian group are contrasting with the Spanish group and Wisconsin cohort.

A large prospective randomized trial that is properly powered is needed to answer the question of whether intermittent hypoxia of sleep is associated with higher carcinogenesis, whether CPAP therapy ameliorates this or not.

ACTIVATION OF N-METHYL-D-ASPARTATE RECEPTOR DOWNREGULATES INFLAMMASOME ACTIVITY AND LIVER INFLAMMATION VIA A  $\beta$ -ARRESTIN-2 PATHWAY

AHMAD FAROOQ, ANAUM MAQSOOD , AHSAN FAROOQ, RAFAZ HOQUE , WAJAHAT MEHAL

**ABSTRACT BODY:** Background: TLR4 and NLRP3 inflammasome activation are responsible for many inflammatory liver disease but little is known about their regulation. The NMDA receptor is known to be present on macrophages and its role in immune regulation has not been investigated. We used the NMDA ligand aspartic acid (AA) to test the role of NMDA activation in liver inflammation. Aims: To test if AA can modulate TLR4 and NLRP3 inflammasome signaling and liver injury via its known NMDA receptor.

**Methods:** The NLRP3 inflammasome was activated by LPS and ATP in primary mouse macrophages, Kupffer cells and human peripheral monocytes in the presence and absence of AA and production of pro-IL1 beta and IL-1 beta assayed. NMDA receptor and beta-arrestin 2 dependence of AA effects was examined in the RAW 264.7 cells using siRNA knockdown. AA was supplemented in vivo in the presence or absence of beta-arrestin 2 knockdown in the LPS/d-Gal hepatitis and acetaminophen hepatotoxicity. Liver tissue was examined for injury and inflammation by histological grading and serum transaminases.

**Results:** AA suppresses in vitro TLR4 and NLRP3 inflammasome dependent inflammation in human peripheral monocytes, mouse peritoneal macrophages and Kupffer cells as assessed by levels of pro-IL1 beta and IL-1 beta. AA immunosuppressive effects require NMDA and beta-arrestin 2. In vivo AA supplementation decreases liver inflammation and injury in the LPS/d-GalN hepatitis (hemorrhage 1.03 +/- 0.3 versus 3.89 +/- 0.2, ALT 744 +/- 406 versus 12560 +/- 5295,  $P < 0.05$ ), and acetaminophen hepatotoxicity (necrosis 0.1 +/- 0.1 versus 1.4 +/- 0.1, hemorrhage 1.77 +/- 0.2 versus 2.5 +/- 0.6, liver transcript for pro-IL-1 beta and Nlrp3 caspase 1 and serum IL-1 beta release,  $P < 0.01$ ). AA induced in vivo protection is dependent on NMDA and beta-arrestin 2.

**Conclusions:** Aspartic Acid acts through NMDA and beta-arrestin 2 to suppress TLR4 and NLRP3 mediated pro-inflammatory signaling and hepatitis. Aspartic acid has potential as a therapeutic agent in the treatment of acute liver failure.

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DECREASED EJECTION FRACTION AND AORTIC VALVE AREA ARE ASSOCIATED WITH INCREASED RIGHT VENTRICULAR OUTFLOW TRACT ELLIPTICITY IN PATIENTS WITH AORTIC STENOSIS

First Author: Affan Haleem, M.D. Second Author: Anjili Srivastava, D.O.

**Background:** Cardiac Computed Tomography (CT) demonstrates that right ventricular outflow tract (RVOT) and left ventricular outflow tract (LVOT) are not perfectly circular. However, it is unclear as to which factors increase the degree of ellipticity of these orifices. RVOT and LVOT diameters are utilized by echocardiography for calculation of pulmonic and aortic valve areas, respectively. Recognizing factors that increase ellipticity can help identify patients in whom pulmonic and aortic valve areas will be underestimated on echocardiography as well as contribute to our understanding of the pathophysiology behind outflow tract distortion.

**Methods:** We retrospectively reviewed cardiac computed tomography (CCT) images obtained from 65 consecutive patients at a single academic medical center being evaluated for transcatheter aortic valve replacement (TAVR). Direct RVOT planimetry was performed on the CCT images to obtain short diameter and long diameter dimension. The ellipticity index of the RVOT was defined as  $1 - (\text{short diameter} / \text{long diameter})$ . We used paired linear regression analysis to look for patient characteristics that are associated with increased RVOT ellipticity Index.

**Results:** The mean age and RVOT ellipticity index of the study's cohort is 82.7 and 0.15. RVOT ellipticity index was negatively correlated with left ventricular ejection fraction ( $r = -0.3$ ,  $p = 0.01$ ) and aortic valve area ( $r = -0.28$ ,  $p < 0.05$ ). It did not significantly correlate with age, BMI, LVOT ellipticity index, aortic valve mean gradient, peak gradient or baseline creatinine. 24% of RVOT ellipticity index variability was explained by left ventricular ejection fraction.

**Discussion:** Valvular ellipticity has significant clinical implications in patients undergoing trans catheter valvular replacement. However, the pathophysiology behind increased valvular ellipticity is unclear. This is the first time a clinical characteristic has been shown to be associated with increasing RVOT ellipticity in aortic valve stenosis patients. Increased afterload from decreased LVEF and decreased Aortic Valve Area may lead to chronically increased strain on the RVOT resulting in increased distortion of shape. Replication of these results in patients with congenital heart disease may reveal Aortic valve stenosis and LVEF as therapeutic targets for reduction of pulmonic valve regurgitation.

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UTILITY OF THE APACHE II SCORE IN TRIAGING PATIENTS TO THE ICU

First Author: Thaofiq Ijaiya, MD, Amulya Abburi MD; Alex Seidenschwarz MD; Jonathan Ang MD; Prasanta Basak MD; Stephen Jesmajian MD.

**INTRODUCTION:** Intensive care unit (ICU) beds are a scarce resource accounting for 8% of acute care beds in the United States. Consideration of alternative treatment locations has been suggested during times of extreme ICU bed shortage. The identification of patients who are more likely to benefit from ICU care is challenging. We investigate the use of APACHE II score as a risk stratification tool to guide admission triage decisions by comparing mortality rates and length of stay between direct ICU admissions and transfers from medical floors.

**METHODS:** We conducted a retrospective chart review of patients admitted to the medical ICU from January 2014 to December 2014. Admission APACHE II score, transfer time, length of stay and in-hospital mortality were also recorded. Patients transferred after one week and transfers unrelated to initial diagnosis were excluded. All variables were assessed with frequency distributions, and evaluated for association with outcome variables using t-tests or non-parametric equivalent (continuous variables) or chi square tests (categorical variables).

**RESULTS:** A total of 215 patients were included in the study. The mortality rate was 26% in the direct ICU admission group (n=41) and 30.5% in those that were transferred from the medical floors (n=18) (p=0.53). Among the patients in the transfer group, there was a trend towards increase mortality rate with time to transfer. When the groups were stratified by APACHE score, the transfer group in the APACHE range 15 to 19 and also 20 to 24 demonstrated higher mortality rates than would be expected from the APACHE II score prognosis index (p=0.06) and in comparison to direct ICU admissions (p=0.01).

There was a trend towards a longer length of ICU stay in patients transferred to the ICU and this was observed with APACHE II score group stratification.

**CONCLUSION:** Our findings demonstrate that the highest mortality discrepancy between patients admitted directly to the ICU versus those transferred from the medical floors was in the APACHE score range 15-24. This reflects the need to assess more closely those who are not "too well or too sick" and prioritize ICU admissions accordingly. We also demonstrated a correlation between delay in ICU transfer and mortality rate. This indicates a need for reevaluation of initial triage of patient early in the admission course.

Further studies are necessary to explore other factors and tools to effectively triage patients particularly during periods of ICU bed shortage.

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INVESTIGATION INTO ONE-SIZE-FITS-ALL DIURETIC STRATEGY FOR HEART FAILURE EXACERBATION ADMISSIONS

First Author: Hirotaka Kato, MB, Perry Fisher, MD, and Dahlia Rizk, DO

**Introduction:** Diuretic therapy is the mainstay of treatment for heart failure exacerbations but its optimal dosing strategy remains unclear. In 2011, the DOSE trial exhibited a trend toward improvement in patients' global symptom assessments when utilizing a high-dose diuretic strategy, which used nearly 80mg intravenous furosemide three times daily in the first 72 hours on average. To improve heart failure outcomes and test the applicability of this trial to our population, we implemented the initial dose of 80mg intravenous furosemide three times daily for patients admitted through our emergency department. We hypothesized that standardizing high dose furosemide for all patient populations may lead to some negative inpatient outcomes.

**Methods:** Consecutive 334 patients admitted for acute systolic or diastolic heart failure were identified in an urban, academic medical center and reviewed from July 2014 to June 2015. Total diuretic dose was defined as total diuretics administered in the first 72 hours after hospitalization. Multivariable regression models with stepwise selection method were used to assess the statistical association between variables and inpatient outcomes including length of stay, worsening renal function, 30-day readmission rate, and mortality.

**Results:** Higher total furosemide dose in first 72 hours was significantly associated with longer length of stay (Coefficient 0.0032,  $p=0.000$ ), higher reduction in GFR (Coefficient  $-0.00085$ ,  $p=0.000$ ), higher grade of worsening renal function (OR 1.0013, CI 1.0006 – 1.0019,  $p=0.000$ ). Importantly, there was no association between total furosemide dose and 30-day readmission rate or inpatient mortality. Higher reduction in GFR was significantly associated with longer length of stay (Coefficient  $-11.2$ ,  $p=0.000$ ). History of stroke and history of heart failure admission in past twelve months were significantly associated with higher 30-day readmission rate (OR 2.4, CI 1.15 – 5.09,  $p=0.019$  and OR 2.4, CI 1.32 – 4.53,  $p=0.004$ , respectively).

**Conclusion:** In patients admitted for acute heart failure exacerbation, higher diuretic dose in first 72 hours was associated with longer length of stay and worsening renal function in the setting of attempting to achieve the standardized dose of 80mg three times daily. Even though a high dose diuretic strategy is advantageous for reducing symptoms early per the DOSE trial, our data suggest that physicians should use clinical judgment, and that other variables may play a role in applying a high dose strategy to the appropriate patients.

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DISCONTINUATION OF EVEROLIMUS DUE TO UNRELATED ADVERSE EVENTS IN CANCER PATIENTS

Sherise C. Rogers, MD, MPH, Shenhong Wu, MD, PhD

**Introduction:** The mTOR (mammalian target of rapamycin) inhibitor, everolimus which is used widely as a chemotherapeutic agent in patients with solid tumors, can be discontinued during therapy due to serious adverse events. Because these adverse events may not be related to the drug itself, and can be caused by confounding factors such as underlying malignancies, comorbidities, and concurrent medications, it is often a challenge to differentiate adverse events related and unrelated to everolimus therapy. In order to assess the scope of this issue, a meta-analysis of randomized controlled trials was performed to examine the risk of everolimus discontinuation due to unrelated adverse events.

**Materials and Methods:** A PubMed search was performed to identify all randomized controlled clinical trials in which everolimus was compared to placebo with or without concurrent anti-neoplastic drugs in cancer patients. Eligible clinical trials reported a discontinuation rate due to adverse events for the everolimus group (related and unrelated to everolimus) and the placebo control group (unrelated to everolimus). A random- or fixed effects model was used to determine summary incidences, relative risks and 95% confidence intervals.

**Results:** A total of 15 RCTs were eligible for analysis, which included a total of 5,631 patients (everolimus: n=3366, control: n=2265). The summary incidence of everolimus discontinuation due to adverse effects was 12.3% (95% CI: 9.5-15.8%). In placebo controls, the summary incidence for discontinuation due to adverse events was 4.7% (95% CI: 3.1-7.0%). In comparison with everolimus, placebo controls were associated with discontinuation due to adverse events at a relative risk of 0.385 (95% CI: 0.275-0.539, p=0.006). Subgroup analysis showed the relative risk of discontinuation due to adverse events varied significantly with cancer types (P<0.001), with the lowest seen in renal cell cancer (RR=0.115, 95% CI: 0.028-0.46), and the highest seen in angiomyolipoma associated with tuberous sclerosis (RR=4.0, 95% CI: 0.754-21.2). Risk of discontinuation due to adverse events did not vary significantly by everolimus dose (p= 0.841) or when in combination with other agents (p= 0.494)

**Conclusion:** There is a substantial risk of everolimus discontinuation due to unrelated adverse events in cancer treatment.

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DOES MAMMOGRAPHIC DENSITY MODIFY THE CHARACTERISTICS OF THE BREAST CANCER ?

Aye M Soe,MD Yu Y Thar,MD Sonal Bordia, MD Maxim Shulimovich, MD Shalom Buchbinder, MD Aung M Tun,MD Lisset Rodriguez,MD Elizabeth Guevara,MD

**Introduction:** Increased mammographic breast density (MD) has been shown to be an independent risk factor for breast cancer (1, 2). Women with dense breast as determined by a diagnostic mammogram are at 1.8 to 6 times greater risk of breast cancer than those of the same age without increased breast density (3). We investigated whether there is any association of the mammographic density and the estrogen receptor ( ER ) or progesterone receptor ( PR ) status of the invasive breast cancer and ductal carcinoma in situ (DCIS). We also evaluated whether the association between MD and breast cancer is modified by body mass index (BMI), menopausal status and race.

**Methods:** We identified 144 patients with invasive breast cancer and 70 patients with DCIS diagnosed between 2012-2014 in our community hospital. We conducted the imaging reviews to identify mammographic density (MD) and chart reviews to obtain the tumor characteristics of those patients. Mammographic density was determined by breast imaging-reporting and data system (BI-RADS) classification. Multivariate analysis was performed to assess the association of BMI, menopausal status, race and ER or PR status of the breast cancer and MD.

**Results:** We observed a positive association between positive ER or PR status and high MD ( MD >50% ) in both invasive breast cancer and DCIS ( OR 4.73, 95 % CI 1.1-20.31 ; p value : 0.04 ). Among the patients with invasive breast cancer, BMI is inversely associated with high MD (odd ratio (OR ): 0.49; 95% CI : 0.32-0.7; p= 0.0003). In contrast, there is no association between BMI and MD in DCIS ( OR 0.78; 95 % CI 0.48-1.16 , p value : 0.25 ).There was trend towards a positive association between premenopausal status and high MD in both invasive breast cancer and DCIS but not statistically significant ( OR 2.69 , 95% CI 1.55-6.84; p: 0.08 ). Among the patients with both invasive breast cancers and DCIS, the inverse association has been observed between African American women and high MD although it was not statistically significant (OR 0.42; 95 % CI 0.14-1.22, p value: 0.11).

**Conclusion:** High MD may be associated with increased occurrence of ER or PR positive breast cancers. There is also a possible inverse correlation between MD and BMI among patients with invasive breast cancer. Further prospective studies are warranted to evaluate this possible associations.

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HIGH CREATININE IS ASSOCIATED WITH INCREASING LVOT ELLIPTICITY IN PATIENTS WITH AORTIC VALVE STENOSIS

First Author: Affan Haleem, M.D. Second Author: Anjili Srivastava, D.O. Third Author: Alimul Islam Fourth Author: Kevin Marzo, M.D. Fifth Author: Juan Gaztanaga, M.D. Sixth Author/Principal Investigator: Beevash Ray, M.D.

**Background** Cardiac computed tomography (CCT) demonstrates that right ventricular outflow tract (RVOT) and left ventricular outflow tract (LVOT) are not perfectly circular. However, it is unclear as to which factors increase the degree of ellipticity of these orifices. RVOT and LVOT diameters are utilized by echocardiography for calculation of pulmonic and aortic valve areas, respectively. Recognizing factors that increase ellipticity can help identify patients in whom pulmonic and aortic valve areas (AVA) will be underestimated on echocardiography.

**Methods** We retrospectively reviewed cardiac computed tomography images obtained from 70 consecutive patients at a single academic medical center being evaluated for transcatheter aortic valve replacement (TAVR). Direct LVOT planimetry was performed on the CCT images to obtain short diameter and long diameter dimension. The ellipticity index of the LVOT and RVOT was defined as  $1 - (\text{short diameter} / \text{long diameter})$ . We used paired linear regression analysis to look for patient characteristics that are associated with increased RVOT or LVOT ellipticity index.

**Results** The mean age and valve area of the study's cohort is 82.5 years and 0.72cm<sup>2</sup>, respectively. LVOT ellipticity index did not significantly correlate with patient's age, BMI, aortic valve mean gradient, RVOT ellipticity index or aortic valve peak gradient. However, patient's baseline creatinine positively correlated with LVOT ellipticity index ( $r=0.26$ ,  $p<0.05$ ) with nearly 23% of variability explained by creatinine level alone.

**Discussion** Valvular ellipticity has significant clinical implications in patients undergoing valvular replacement. It has already been established highly elliptical LVOTs can cause an underestimation of the AVA by echocardiography. This is the first time a clinical characteristic has been shown to be associated with increasing LVOT ellipticity in aortic valve stenosis patients. The pathophysiology of high baseline creatinine causing increased LVOT ellipticity remains unclear. However, chronic kidney disease, as suggested by increased baseline creatinine has been demonstrated to increase afterload, increase calcification and cause left ventricular hypertrophy. Future studies are needed to determine whether the increased ellipticity of high creatinine patients cause a clinically significant underestimation of AVA by echocardiography.

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SCREENING COLONOSCOPY IN REFUGEES: IS IT NECESSARY? – COMPARISON OF POLYP PREVALENCE AND CHARACTERISTICS IN REFUGEE POPULATION TO THE AMERICAN POPULATION

First Author: V Thoguluva Chandrasekar, MBBS Divey Manocha, MD Ganesh Aswath, MD Peter Cronkright, MD Savio John, MD

**Introduction:** Colonoscopy is one of the most preferred methods of colorectal cancer screening with the benefit of being both a diagnostic and therapeutic tool. United States is a land of increasing immigrant population with people from different regions of the world settling down as first generation refugees. The burden of colorectal cancer in this population is not completely defined. Whether the adoption of the present Colorectal Cancer (CRC) screening guidelines to this population leads to early cancer diagnosis and prevention remains unproven.

**Objective:** To compare the polyp burden and clinical characteristics of refugee and American population, predominantly from Nepal and Somalia, who undergo screening colonoscopy.

**Materials and methods:** A single center IRB approved retrospective study of refugee population (mostly Nepalese and Somali), who underwent colonoscopy at 50 years or older for CRC screening. All patients who had an alternate indication for colonoscopy other than CRC screening, previous colonoscopies, personal history of polyps and history of colon cancer were excluded. For every case, two age and gender matched controls from local American population were randomly assigned. Univariate and multivariate analysis was performed to evaluate demographic and clinical characteristics, adequacy of preparation, polyps and adenoma detection rate between the two groups.

**Results:** Prevalence of obesity (27.3 % vs 43.4 %) and family history of colorectal malignancy (0 % vs 16.4 %) was significantly lower among the refugee population. Adequacy of colon preparation was comparable between the two groups (58.7% vs 68%). The incidence of diverticulosis was significantly lower in the refugees (4.1% vs 34.4%). The prevalence of all polyps (29.8% vs 37.7%) and adenomas were comparable in both study groups (20.7% vs 29.1 %). The average size of the largest polyp resected was significantly smaller in the refugees when compared to the controls (1.40 mm vs 2.32 mm). The distribution of polyps in the distal colon was less frequent in the refugee population (14.5% vs 22%).

**Conclusion:** This study demonstrates that the adenoma burden was similar in the first generation refugee population compared to the American population. This study also suggests that refugees need to be screened as per the present CRC screening guidelines followed in the US for general population. Larger prospective studies are needed to determine whether detection of adenomas in the refugee population provides the same protective benefit against colon cancer, as shown in the American population.

**Primary funding source:** None

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## LEAD EXTRACTION AT DUKE HOSPITAL: INDICATIONS, OUTCOMES AND ADVERSE EVENTS

**All Authors:** Sean D. Pokorney, Robert K. Lewis, Alexander Christian, Ruth Ann Greenfield, James P. Daubert, Jonathan Piccini

**Introduction:** Cardiac implantable electronic device (CIED) are increasingly utilized. Few reports explore characteristics and outcomes of extraction for infection versus non-infection indications. Furthermore, although ICD leads are widely considered to be more difficult to extract than pacemaker leads, there are few direct comparisons.

**Methods/Results:** We performed a retrospective cohort analysis of 368 patients undergoing lead extraction (dwell time >1 yr) between 2005-2012 at Duke University Hospital. We compared patient characteristics/outcomes based upon indications for extraction (3 groups): no infection (NI, n=153), pocket infection (PI, n=126), and bacteremia and endocarditis (BE, n=86) Patients with infections were older with a median age of 57, 71, and 63 years for NI, PI, and BE respectively ( $p<0.0001$ ). Comorbid illness was much more common in PI and most prevalent in those with BE, including diabetes, hypertension, chronic kidney disease, and end-stage renal disease (Figure 1). There was a trend to longer implant times in the NI group: median 4.2, 5.7, and 4.5 years, for NI, PI, and BE respectively,  $p=0.07$ ). Extractions for infectious etiologies had a higher extraction-related MAE relative to NI (BE=5%, PI=9%, and NI= 2.0%,  $p=0.034$ ). In-hospital mortality was statistically similar between the categorizations but tended to be higher in the infectious groups (Figure 1). A second analysis was performed based on lead type: there were 136 (37%) pacing lead extractions and 232 (63%) ICD lead extractions. Pacing leads had a longer dwell time (6.14 yrs [IQR 1.2-10.9] versus 4.4 yrs [IQR 1.1-6.4],  $p<0.001$ ) and higher median LVEF (55% [IQR 35-55] vs. 30% [IQR 20-40],  $p<0.001$ ) compared with ICD lead patients. There were similar rates of all-cause MAE during index hospitalization (5.1% vs. 5.6%), death (2.2% vs 3.2%) and clinical success (97.0% vs. 97.0%,  $p=0.55$ ) in pacemaker and ICD extractions, respectively (Figure 2).

**Conclusions:** Multiple factors influence lead extraction outcomes. Patients who present for lead extraction due to local or systemic infection have higher comorbid illness and experience more adverse events. Patients with infection represent a particularly high-risk group. Despite much longer dwell times, major adverse events were similar in pacing lead cases compared with ICD extractions.

EFFICACY AND SAFETY OF APIXABAN COMPARED WITH WARFARIN IN PATIENTS WITH PERIPHERAL ARTERY DISEASE AND ATRIAL FIBRILLATION: INSIGHTS FROM THE ARISTOTLE TRIAL

First Author: Peter Hu, MD Authors: Renato D. Lopes, MD, PhD, Susanna Stevens, MS, Lars Wallentin, MD, PhD, Laine Thomas, PhD, John H. Alexander, MD, MHS, Michael Hanna, MD, Basil S. Lewis, MD, Freek Verheugt, MD, PhD, Chris B. Granger, MD, W. Schuyler Jo

Background: Vascular disease has been incorporated in the CHA2DS2-VASc risk tool to predict the risk of stroke/systemic embolism in patients with non-valvular atrial fibrillation.

Objectives: This post-hoc analysis of the ARISTOTLE trial aimed to determine the absolute rates of stroke/systemic embolism and bleeding associated with peripheral artery disease (PAD) as well as the efficacy and safety of apixaban vs warfarin in patients with and without PAD.

Methods: ARISTOTLE randomized patients with AF and risk of stroke to apixaban and warfarin for the prevention of stroke/systemic embolism.

Results: A total of 884 (4.9%) patients had PAD at baseline as defined by site investigators on the case report form. Patients with versus without PAD had non-significantly higher rates of stroke/systemic embolism [HR 1.32, 95% CI 0.93-1.88, p=0.12] and major or clinically relevant non-major (CRNM) bleeding [HR 1.12, 95% CI 0.90-1.39, p=0.31]. Those with PAD had an increased risk of all-cause death [HR 1.36, 95% CI 1.11-1.67, p=0.003] and CV death [HR 1.44, 95% CI 1.08-1.90, p=0.01] when compared with those without PAD. The effect of apixaban vs warfarin for the prevention of stroke/systemic embolism was similar in patients with PAD [HR 0.66, 95% CI 0.33- 1.31] and without PAD [HR 0.80, 95% CI 0.66-0.96, interaction p=0.61]. Patients with PAD appeared to have less reduction in major or CRNM bleeding with apixaban compared with warfarin [HR 1.04, 95% CI 0.69-1.57] versus those without PAD [HR 0.66, 95% CI 0.59-0.73, interaction p=0.03].

Conclusion: Patients with PAD in ARISTOTLE had non-significantly higher risk of stroke/systemic embolism when compared with patients without PAD. The benefits of apixaban versus warfarin on stroke and all-cause death are similar in patients with and without PAD. However, the reduction in bleeding with apixaban seemed to be greater in patients without.

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## COMPLIANCE WITH GUIDELINES FOR GASTROINTESTINAL BLEEDING IN CIRRHOTIC PATIENTS IN A COMMUNITY HOSPITAL

First Author: Ryan Jordan, DO, Poorvi King, MD, Lucas Faulkenberry, MD, Frederick Opper, MD, Paul Hayashi, MD

**INTRODUCTION:** Upper gastrointestinal (GI) bleeding due to esophageal varices is a common complication in cirrhotics who present to emergency departments. The American Association for the Study of Liver Disease has published guidelines on the management of gastroesophageal variceal bleeding, but studies have shown that adherence to the guideline-based therapies is low. For patients with known cirrhosis with a suspected upper GI bleed, guidelines recommend administration of a proton pump inhibitor (PPI), octreotide, and antibiotics. This preliminary study sought to assess a community hospital's compliance with these guidelines in the emergency department and the inpatient wards.

**METHODS:** IRB approval was obtained and a retrospective study was conducted at New Hanover Regional Medical Center (NHRMC), a large community-based hospital. All patients from October 1, 2013 (date of EMR implementation) to March 31, 2015 with a diagnosis of cirrhosis and esophageal variceal bleed were identified. Data collected included demographics, administration of antibiotics, proton pump inhibitors and octreotide, total units of packed red blood cells per patient, mean last hemoglobin prior to transfusion, length of hospital stay, length of ICU stay, inpatient mortality, and need for repeat esophagogastroduodenoscopy (EGD) due to re-bleed.

**RESULTS:** Within the 15 month study period, 63 encounters involving 51 patients met inclusion criteria. Only 13% of patients received antibiotics in the emergency department, while 68% received a PPI and 38% were given octreotide. These numbers increased to 48% for antibiotics, 100% for PPI, and 87% for octreotide once the patient was admitted to the hospital. Only 12.7% of patients received all 3 recommended therapies in the emergency department, while 57% received all 3 treatments once admitted to the hospital. Patients who received all 3 treatments were more likely to be alive at discharge than patients who did not, but this difference was not statistically significant ( $p=0.1093$ ). Nine patients (14%) were transfused at a hemoglobin higher than 8 g/dL, which is also not in compliance with published guidelines.

**DISCUSSION:** Guideline-based therapies for GI bleeding in patients with cirrhosis have been shown to reduce mortality in larger studies, though adherence to these guidelines is low. Our study, in a large community hospital, supports these findings. PPIs are the most commonly-administered therapy, while antibiotics were rarely administered in the emergency room and administered in less than half of patients who were admitted to the hospital. This study provides preliminary data and impetus for a second study, which involves creating order sets for cirrhotic and gastrointestinal bleed patients that will allow emergency department and admitting providers to easily order these therapies and possibly improve outcomes.

## CONSCIENTIOUS ORDERING

**All Authors:** First Author: Samaya Qureshi, MD Second Author: Ryan Brown, MD Third Author: Alejandro Paya, DO Fourth Author: Emily Mullen, MD

Rising costs continue to be a concern in healthcare and reduction of waste is a growing focus among physicians. The American College of Physicians High Value Care initiative helps physicians provide the best possible patient care while reducing unnecessary healthcare costs. One example of healthcare cost burden is unnecessary labs drawn during patient hospitalizations. This area is analyzed in the literature by exploring interventions to decrease unnecessary lab ordering. According to the Choosing Wisely campaign, an initiative of the ABIM Foundation, repetitive CBC and chemistry testing in the face of clinical and lab stability is listed as one of the five things physicians and patients should question. Additionally, the ACGME requests residency programs to educate residents in the science of Quality Improvement (QI) and High Value Care. With this background, Cone Health Internal Medicine Residency Program residents developed a project utilizing the Six Sigma DMAIC (Define, Measure, Analyze, Improve, and Control) process improvement methodology aimed at decreasing unnecessary laboratory test ordering.

The interdisciplinary project team included 11 Internal Medicine residents in different years of training, a faculty mentor, a QI facilitator, and other hospital employees. The aim of the project was to decrease the number of morning labs ordered per patient-day by 10% on the Internal Medicine Residency Program inpatient teams. Interventions included discussing project goals with the teams and posting a list of inclusion and exclusion criteria for ordering morning labs above each computer in the resident workroom. Residents were also encouraged to discuss ordering morning labs on patients after rounds daily. Baseline data from December 2013 regarding the quantity of morning labs and venipunctures ordered was compared to the data from January 2015 after implementing the above interventions. Primary outcome was number of labs per patient-day and secondary outcomes were percentage of patients with no morning labs, number of venipunctures per patient-day, and cost of savings in US dollars per month.

The original project goal of decreasing morning labs per patient-day by 10% was met and exceeded with a total 33% reduction ( $p < 0.05$ ). Venipunctures per patient-day decreased by 28% and patient encounters with no morning labs increased by 104% ( $p < 0.05$ ). The projected cost savings for January 2015 were approximately \$2,380 and \$28,560 over the course of a year. Through education and communication interventions, we showed significant reductions in lab ordering and health care cost and waste and hopefully improved physician practice and patient satisfaction. As a result, this project effectively involved medical residents in QI and High Value Care initiatives. Future goals include expanding this project to the Hospitalist services and sustaining our gains in high value care within the residency program.

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IMPROVING HCV SCREENING RATES IN A COMBINED INTERNAL MEDICINE AND PEDIATRICS CLINIC

**All Authors:** Nicholas Turner MD and Jane Trinh MD

**Introduction:** Hepatitis C (HCV) is a viral liver disease that can result in cirrhosis, hepatocellular carcinoma, liver transplantation or even death. The CDC estimates that 2.7-3.9 million Americans are living with hepatitis C infection, yet the majority are unaware at least in part because early infection is often asymptomatic. Even when screening is performed according to traditional risk factors, 31-47% of cases of hepatitis C may be missed. With the advent of targeted antiviral therapies, sustained virologic response rates can exceed 90%. Starting in 2013, hepatitis C screening was recommended by both the CDC and the USPSTF for all those born between 1945 and 1965. We designed a quality improvement project aimed at improving birth-cohort based HCV screening rates in an academic primary care clinic.

**Methods:** Baseline HCV screening rates were assessed from our EMR over a 3 month time period. During the "Plan, Do, Study, Act" (PDSA) cycles that followed, we implemented a series of interventions to clinic practices aimed at improving hepatitis C screening rates. Interventions were designed to increase provider awareness of HCV screening guidelines (surveys and individualized feedback), increase documentation of HCV screening or discussion (initially by incorporating an HCV reminder into note templates, later by petitioning for an update of our EMR's health maintenance tool) and increase provider comfort with discussing HCV testing with patients (creation of information packets to guide discussion). After each PDSA cycle, HCV screening rates were re-calculated using lists of patients seen for annual or new patient visits. We also examined aggregate data from the past 2 years of annual visits to identify and social or demographic factors which might predict missed HCV screening.

**Results:** At baseline, HCV screening rates were 23% and survey data indicated 75% of providers did not feel fully comfortable explaining the rationale for screening to patients. Over the next several PDSA cycles, clinic-wide screening rates exceeded 90%. Individual physician improvement ranged from 0% to 100%. After assessing for differences in rates of HCV screening according to gender, race, median income and socioeconomic status/educational background, we noted that screening was more likely to be missed among males ( $p=0.01$ ) and Caucasians ( $p=0.0004$ ). Screening rates also varied according to socioeconomic standing, but not in any consistent or linear pattern.

**Conclusions:** Quality improvement interventions can dramatically improve HCV screening rates in an academic primary care clinic. While further research is indicated to confirm our preliminary findings related to demographic risks for missed screening, our data at least suggest that Caucasian males may be more likely to be missed by providers for HCV screening or alternatively may be declining screening more often than other demographics.

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A SAFER SIGN-OUT: IMPLEMENTING A STANDARDIZED SIGN-OUT PRACTICE

**All Authors:** Lucy S. Witt MD, MPH, Deb Bynum MD, Kathryn El-Amraoui MD, Robert Rayson MD.

**Background:** In 2007 the Joint Commission declared that “communication failures are the most common root cause of sentinel events in US hospitals”(1). The Accreditation Council for Graduate Medical Education (ACGME) noted that poor handover practices contributed to many errors made by medical residents (2). These findings have led to the development of standardized handoff practices in residencies and hospitals, the intent of which is to improve resident communication and decrease medical errors in the setting of new ACGME work hour restrictions.

**Methods:** The University of North Carolina (UNC) is an academic, university-based program. During an internal morbidity and mortality conference, incomplete sign-out was identified as one of many possible root causes of an adverse patient outcome. In response to this, a standardized sign-out method termed “SITuP to sign-out” was developed. Current second and third year residents were surveyed prior to a lecture detailing safe handoff strategies and introducing the newly developed UNC method to standardize resident sign-out. Interns were surveyed prior to an educational conference that similarly introduced them to the UNC “SITuP to sign-out” method. Repeat surveys were conducted two months after implementation to assess adherence to and perceptions of the new sign-out method. Surveys were completed online and were anonymous.

**Results:** Following the implementation of SITuP, second and third year residents described sign-out as both more standardized and slightly safer. Fewer residents described receiving or providing incomplete sign-out after education on safe sign-out and implementation of SITuP. Adherence amongst upper level residents was more modest. Only 28% of second and third-year residents attested to using SITuP most of the time for verbal sign-out. For written sign-out, however, 78% of upper level residents described using SITuP most or all of the time.

Interns showed a significant improvement in their perception of personal understanding of safe and effective sign-out. Prior to receiving education regarding safe and effective sign-out 68% of interns believed they understood what appropriate sign-out included. This improved to 90% following the teaching session and implementation of SITuP. Uptake was also high amongst interns with 86% using SITuP most or all of the time. Interns were also better able to identify aspects of safe sign-out following education and implementation of SITuP.

**Discussion:** The SITuP method and associated educational lectures improved standardization of the Internal Medicine sign-out practices. Changes in knowledge and adherence to the method were more pronounced for interns than for second and third year residents. Further studies will work to assess on-going compliance and satisfaction with this method. Similarly future investigations could work to describe the effect on patient safety with the implementation of this handoff method.

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EPIDEMIOLOGY OF COMMUNITY-ONSET VERSUS NOSOCOMIAL FUNGEMIA- A COMPARATIVE STUDY

First Author: Kateryna Bertuch, MD, Francis Edeani, MD Stephen Blatt, MD, Yury Gonzales, MD, Jenni Steinbrunner, BS

Background: Previous studies have shown that invasive candidiasis is more prevalent in ICU patients, however, our clinical observation indicates a possible shift from the ICU to other health care settings and that the incidence of community-onset fungemia is on the rise. Although *C. albicans* is the most common cause of fungemia, there has been increased isolation of non-albicans species of candida, which have been reported to be more resistant to fluconazole. This study aims to compare risk factors, resistance patterns, species distribution, and outcomes between community-onset and nosocomial fungemia.

Methods: We performed a retrospective review of charts of all patients with at least one positive fungal blood culture between July 2010 and June 2014. Patients were categorized as community-onset fungemia defined as a positive fungal blood culture obtained within 48 hours of admission or nosocomial fungemia defined as a positive fungal blood culture obtained 48 hours or more after admission.

Results: A total of 124 patients with fungemia were included in the analysis. 36.3% (45) were defined as community-onset and 63.7% (79) were defined as nosocomial fungemia. Female patients were more likely to have community-onset fungemia compared with male patients ( $p=0.02$ ). Community-onset fungemia cases were less likely to have exposure to the following risk factors: invasive procedure ( $p<0.001$ ), intensive care unit ( $p<0.001$ ), antimicrobial use ( $p=0.02$ ), surgery ( $p=0.003$ ), and total parenteral nutrition ( $p=0.04$ ). Additionally, nosocomial fungemia was associated with older age ( $p=0.02$ ) and a longer hospital stay ( $p=0.02$ ). The mortality rate in patients with nosocomial fungemia (15%) was higher when compared with patients with community-onset fungemia (5%), however this difference was not statistically significant. There was no significant difference between the two groups in terms of antifungal resistance patterns and fungal species distribution.

Conclusion: Patients with community-onset fungemia were less likely to have the traditional risk factors for fungemia, had better prognosis, shorter length of hospital stay and better mortality rates. Fungemia should therefore be considered in patients with community-onset sepsis even in the absence of traditional risk factors for fungemia.

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ASSOCIATIONS WITH RESIDENT PHYSICIANS' EARLY ADOPTION OF ELECTRONIC CIGARETTES FOR SMOKING CESSATION

Eric Egnot MD, Kim Jordan MD, FACP, John O. Elliott PhD, MPH

**Purpose** The electronic cigarette is the latest trend in nicotine delivery with a dramatic increase in use since patented in 2004. Information continues to emerge on its role as a smoking cessation aid, but little is known about resident physician attitudes towards the device in clinical practice.

**Method** In 2015, an electronic survey was administered to resident physicians in one healthcare system in Columbus, Ohio. The survey included questions about personal smoking exposure, knowledge, beliefs, attitudes about electronic cigarettes and early adoption of electronic cigarettes with patients. Data was dichotomized based on a stages of change model that assessed resident physician adoption of electronic cigarettes for therapeutic use. Data was analyzed via chi-square tests and logistic regression using Odds Ratios (ORs) and 95% Confidence Intervals.

**Results** Of 338 residents, 142 (42%) responded. Of all residents, 25 (17.7%) reported that they have been recommending electronic cigarettes to their patients for the past 6 months or longer. In the multivariate model, residents = PGY3 (OR = 3.68, 95%CI: 1.20-11.29), peer-reviewed article exposure (OR = 6.65, 95%CI: 1.56-28.38) and the view that addictive potential is definitely or somewhat less than traditional cigarettes (OR = 5.05, 95%CI: 1.48-17.24) were associated with recommendation of electronic cigarettes.

**Conclusions** Few residents report recommending electronic cigarettes to patients who smoke. These residents consider the electronic cigarette less addicting than traditional cigarettes, supporting harm reduction strategies over strict abstinence. Residents require further evidence-based education on efficacy and long-term safety of electronic cigarettes before recommending to their patients.

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EVALUATION FOR EXTRANEOUS PULMONARY EMBOLISM CT IN THE EMERGENCY DEPARTMENT  
(EXPECTED)

Arjan Flora, MD; Andy Jedrzejczyk, MD; Maher Abdo; Ashley Kanjira, MD, Michael C. Plewa, MD

**Background/Purpose:** To evaluate the prevalence of extraneous CT angiograms (CTAs) to rule out pulmonary embolism (PE) in our emergency department (ED) that could have been avoided by meeting pulmonary embolism rule-out criteria (PERC) or were low risk by Wells or revised Geneva scores with negative D-dimer (DD) or no DD obtained.

**Methods:** Trained investigators retrospectively reviewed all adult cases of CTA for PE performed at a teaching hospital ED over a 6 month period in 2013. Wells, Geneva scores and PERC were calculated. Data and kappa scores are expressed with 95% confidence intervals (CI).

**Results:** Acute PE was diagnosed in 32 (8.8%; 6-12%) of 362 CTA cases. DD was not obtained in 96 (61.5%; 54-69%) of 156 low risk cases by either Wel's or revised Geneva scores. Low risk Wells and revised Geneva scores were made in 28% (24-33%) and 25% (21-30%) of cases, respectively, with PE diagnosed in 3.9% (2-10%) and 4.4% (2-11%), respectively. Extraneous CTA were identified by PERC in 11.7% (9-15%); low risk Wells score in 16.9% (13-21%), with no DD in 59 and negative DD in 2; and low risk Geneva score in 16.3% (13-20%), with no DD in 57, negative DD in 2. Agreement between Wells and revised Geneva score was low, with  $k=0.160$  (0.052-0.269).

**Conclusions:** We have identified opportunity for improvement in following published guidelines by measuring D-dimer for low risk cases and potentially avoiding as many as 1 of 6 CTA for PE orders. There are fewer extraneous CTAs in this cohort than reported in recent literature. Wells and revised Geneva scores identify more extraneous CTA for PE than PERC, however agreement is poor. We suggest evaluation of a pre-test calculator prompt within the electronic medical record prior to ordering a CTA as a potential tool to improve performance in diagnosing PE.

## FACTORS ASSOCIATED WITH RESIDENT ADHERENCE TO RECOMMENDED LUNG CANCER SCREENING GUIDELINES

First Author: Julie S Han, MD Second Authors: Kiet Ma DO, John O. Elliott PhD, MPH, Kim Jordan MD, FACP

**Background:** In December 2013, the US Preventative Services Task Force (USPSTF) recommended lung cancer screening with yearly low-dose computed tomography (LDCT) in adults aged 55-80 years with a 30 pack-year smoking history who currently smoke or smoked within 15 years. Screening is not recommended for patients with limited life expectancy, unwillingness to undergo curative surgery, or quit smoking for more than 15 years. Studies evaluating clinical guideline adherence have noted significant healthcare variation secondary to patient type, length of clinical experience, or perceived barriers. Limited studies have evaluated factors affecting resident physician guideline adherence.

**Study Objectives:** Utilize established health behavioral models to measure resident physicians' intentions to adhere to lung cancer screening recommendations.

**Methods:** Internal Medicine (n=36) and Family Medicine (n=18) residents from Riverside Methodist Hospital completed a voluntary survey on lung cancer screening guidelines. Prior to survey completion, residents were lectured on screening recommendations by the radiology department and received an educational pamphlet on LDCT screening. The survey was conceptualized using the theory of planned behavior (TPB) which posits that behavioral intention is predicted by Attitudes (favorable views of screening), Subjective Norms (professional expectations), and Perceived Behavioral Control (confidence in recommending screening and cost barriers). The Precaution Adoption Process Model (PAPM) was used to evaluate residents' self-reported referrals for screening in the last 6 months or since the 2013 USPSTF guidelines. Descriptive statistics and a series of regression models were conducted.

**Results:** Survey response rate was 94%: 70.6% (n=36) Internal Medicine and 29.4% (n=15) Family Medicine. In the unadjusted linear model ( $R^2=0.54$ ,  $p<0.001$ ), Attitudes (Beta=0.61,  $p<0.001$ ) and Subjective Norms (Beta=0.27,  $p=0.02$ ) were associated with Behavioral Intention. Associations remained ( $R^2=0.60$ ,  $p<0.001$ ) when controlling for demographic characteristics, Attitudes (Beta=0.58,  $p<0.001$ ) and Subjective Norms (Beta=0.26,  $p=0.026$ ). Based on the PAPM, 9.8% never heard/never thought about lung CT screening, 13.7% were undecided, 3.9% decided against screening, while 37% decided to screen in the next 6 months and 35.3% reported institution of screening in the past 6 months or since guideline release. In the unadjusted logistic model (Pseudo  $R^2=0.21$ ,  $p=0.015$ ), Subjective Norms (OR=1.57,  $p=0.046$ ) were associated with self-reported patient referrals for screening. This association remained (Pseudo  $R^2=0.32$ ,  $p=0.015$ ) when controlling for demographic characteristics (OR=1.66,  $p=0.036$ ). In all analyses, confidence in recommending screening, feeling in control of screening, and cost barriers were not predictive of screening intention or self-reported screening behavior.

**Conclusions:** Our findings suggest that focusing on positive attitudes towards screening and professional expectations may be useful when planning communication strategies and educational methods targeting adoption of evidence-based guidelines, hopefully reducing healthcare variations.



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RADIATION TREATMENT INCREASES PHOSPHATIDYLSERINE EXTERNALIZATION ON GLIOBLASTOMA CELLS – INDICATES POTENTIAL TARGET FOR THERAPY

Nida Hussain, MD Harold Davis, PhD Subrahmanya D. Vallabhapurapu Victor M. Blanco Zhengtao Chu, MD Xiaoyang Qi, PhD

**INTRODUCTION:** Glioblastoma multiforme (GBM) is the most aggressive form of primary brain tumor with a median survival of <15 months from diagnosis. Cancer-selective nanotherapeutic agents such as SapC-DOPS, which target phosphatidylserine (PS) on the cell surface, offer an alternative paradigm for treatment of intracranial neoplasms due to their ability to transverse the blood brain barrier without altering it. We have previously shown a strong association between surface PS levels and SapC-DOPS-induced killing of cancer cells. We hypothesized that radiation treatment would increase PS externalization in GBM cells which may enhance GBM cytotoxicity when combined with SapC-DOPS.

**METHODS:** Cell Surface PS Analysis: Human derived GBM cells (U87EGFR-Luc) were exposed to 0, 2, 6, 10 or 16 Gy then 24 hours later, the cells were stained with Annexin V-FITC and evaluated for cell surface PS using flow cytometry. Immunofluorescent Staining: Human GBM cells (U87EGFR-Luc) were injected intracranially into nude mice and allowed to grow until ~3 mm<sup>3</sup>. Microscope slide sections from these brains were probed with fluorescently labeled SapC-DOPS. The following day, slides were imaged using fluorescence microscopy.

**RESULTS:** Our study showed that fluorescently labeled SapC-DOPS selectively targeted GBM cells but did not stain normal brain tissue. In addition, we demonstrated that GBM cell lines exhibited a positive correlation between surface exposed PS levels and radiation responses. A significant increase in PS externalization was demonstrated by U87EGFR-Luc cells following ionizing radiation therapy at 6, 10 and 16 Gy ( $p = 0.009, 0.004$  and  $0.001$ ); respectively, compared to non-irradiated cells.

**CONCLUSIONS:** We have demonstrated that SapC-DOPS effectively and specifically targets cancer cells that express high PS on their surface and have shown that radiation increases PS expression on GBM cells. These studies provide a basis for combination therapies where increasing extracellular PS via radiation therapy, coupled with SapC-DOPS may enhance cancer cytotoxicity.

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IMPACT OF DEPRESSION THERAPY ON GLYCEMIC CONTROL IN HIGH RISK PATIENTS

**All Authors:** Tejasvi Pasupneti MD MPH, Michael Oravec MPH

**Introduction:** The management of diabetes mellitus (DM) patients with depression is often more challenging than in those without depression. Studies have consistently shown that comorbid depression results in worse glucose control, poor self-care habits, increased medication non-adherence and high rates of end organ complications. Despite current standards of care for managing depression in diabetics, there is little consensus about which depression therapies achieves optimal diabetic outcomes in different populations within a primary care setting. Furthermore, evidence on the benefits of psychosocial therapy and antidepressants directly on glycemic control is mixed. The purpose of this quality improvement project is to assess the impact of different depression treatment strategies on glycemic control in poorly controlled diabetic patients, by the internal medicine residents in a continuity clinic.

**Methods:** We conducted a retrospective observational study of patients with DM and concurrent depression at Summa Health System Internal Medicine Center between January 2010 and June 2014. Only patients with uncontrolled DM (defined as HbA1c  $\geq 7\%$ ) that screened positive for depression were included. Depression therapies were defined as pharmacotherapy only, psychosocial therapy only, combined and none. The primary outcome for the study was time to reach glycemic control. Existing quality assurance databases were used to extract information about therapy types, co-morbidities, and adherence. Patients were followed until either they reached HbA1c goal or until the end of the study period. Multivariate Cox regression was used to model time to reach target HbA1c. **Results:** A total of 419 patients with uncontrolled DM had a positive depression screen at baseline and were included in the analysis. Of these, 141 patients (33.7%) reached goal for HbA1c, with a mean follow up time of 20.0 a14.9 months. Average HbA1C at the start of the study period was 9.77 a 2.14. It was found that patients receiving psychosocial therapy were more likely to achieve the HbA1c goal faster than those receiving no therapy (HR 1.80, 95% CI 1.04, 3.11,  $p=0.037$ ). Receiving pharmacotherapy or a combination of pharmacopsychotherapy were not significant predictors for HbA1c goal compared to no therapy. In addition, patients with at least one outpatient visit every three months were more likely to achieve a faster glycemic control than those without the visits (HR 2.11, 95% CI 1.23, 3.63;  $p=0.007$ ).

**Conclusion:** Among depressed patients with uncontrolled diabetes, only individuals who were receiving psychosocial interventions reached HbA1C goal at a significantly faster rate. These quality assurance findings highlight the importance of psychosocial therapy in high risk patients. The results also provide residents with feedback on how crucial their referral patterns are with respect to clinical outcomes within the outpatient setting. Future plans include drafting quality improvement strategies related to better diabetic follow-up and tailoring depression therapy interventions.

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## GENDER LINKED DIFFERENCES IN HUMAN PLATELET DELTA GRANULES

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**Introduction** Cardiovascular disease is responsible for 1 in 4 female deaths in United States. United States Preventive Service Task Force (USPSTF) recommends aspirin in women between 55-79 years for primary prevention of cardiovascular disease. Multiple studies suggest that compared to men, women do not attain equal therapeutic benefit from anti-platelet medications like aspirin. This observation of aspirin resistance is likely due to underlying gender specific differences in platelet physiology. Platelet delta granules store molecules such as ADP and serotonin that are required for platelet adhesive function. We hypothesized that women have a greater number of platelet delta granules compared to men which can potentially explain the high degree of aspirin resistance in women.

**Methods** We performed a retrospective chart review of men and women of age between 18-65 years who have been tested for suspected platelet delta granule deficiency by electron microscopy (EM) at the University of Toledo Medical Center. We excluded patients who received a confirmed diagnosis of platelet delta granule deficiency based on the EM report. In men and women without platelet delta granule deficiency, we counted the mean number of delta granules and performed one tailed student t test to establish whether women have increased platelet delta granules compared to men

**Results** We examined number of platelet delta granules in 33 women and 10 men. The average age of study population was 35.2 years in men and 40.0 years in women. Study group was 84 percent white and 16 percent black in men and 75 percent white and 25 percent black in women. We observed that women had 5.0+/-0.18 (mean+/- standard error of mean) number of delta granules per platelet compared to 3.8+/-0.2 delta granules per platelet in men. One tailed student's t test showed that the difference in mean number of platelet delta granules among the two groups was statistically significant ( $p < 0.05$ ).

**Conclusion** Our study showed that women have a greater number of platelet delta granules and this observation can potentially explain the increased platelet reactivity and resistance to aspirin seen in women. The major limitation of this study is that we examined a small group of patients and further analysis in general population will provide us with a wider understanding of issue. The clinical importance of the study lies in including platelet granule testing as one of the tools to predict an individual's aspirin response, thereby tailoring the use of anti-platelet treatment with increased efficacy and safety.

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A PILOT STUDY EXAMINING PATIENT'S UNDERSTANDING OF DISCHARGE INSTRUCTIONS:  
STANDARD VERSUS A REDESIGN

First Author: Lakshmi Rangaswamy, DO Katherine Kendjorsky DO, John O. Elliott PhD, MPH, Kim Jordan MD, FACP

Background: The transition from hospital to home can be a time of confusion and misunderstanding for patients and family members. Part of this discharge period includes giving patients discharge instructions, which include medication changes, follow-up appointments, home care instructions, and other valuable information that patients can utilize after discharge home. Patients who clearly comprehend their post-discharge instructions are 30% less likely to be readmitted to the hospital or visit the ED than patients who misunderstand these instructions. A quality improvement project was designed to evaluate patient preference between the current discharge instructions at our institution and a redesign that focused on streamlining the document format based on health literacy concepts.

Study Aims: We hypothesized that a patient-focused redesign of the current discharge instructions would improve patient understanding. We also hypothesized patients would be able to find specific information easier on the redesigned discharge summary compared to the current hospital format.

Methods: After obtaining informed consent, an IRB-approved survey was administered to patients who were literate, English-speaking, over the age of 18 and with normal cognition on general medicine services. Assessment questions (5 point Likert scale: 0="Cannot find" to 4="Very easy to find," score range 0-16) focused on: 1. Whom to call with questions after discharge, 2. Ease of finding follow up instructions, 3. Ease in identifying medication instructions, and 4. Ease in identifying the reason for taking the medications. Follow up questions asked patients to verbally identify and state which version of the discharge instructions they used to find the information on: 1. The dosage and how many times a day to take medications and 2. Why they were taking the medications (an element not included in the current discharge instructions at our institution). Data was analyzed using descriptive statistics. Differences between the patient's paired ratings of the discharge summaries were examined via paired t-tests, Wilcoxon signed rank tests and 95% confidence intervals.

Results: Twenty patients were enrolled, mean age (standard deviation) 50.7 (14.0), 60% were female, 80% were Caucasian and 85% reported at least a high school diploma or some college education. The overall evaluation score results reveal patients preferred the redesigned discharge instruction format over the standard format: 14.6 (95%CI:13.8-15.3) vs 10.0 (95%CI:8.9-11.0),  $p < 0.001$ , large effect size  $d = 2.42$ . Ease of finding information was most noted on whom to call after discharge ( $p < 0.001$ ), identification of dosages/instructions ( $p = 0.013$ ) and reason for taking the medications ( $p < 0.001$ ). Ninety percent of patients correctly identified medication doses/instructions and 80% properly identified the reason for taking these medications on the redesigned format.

Conclusions: Our findings suggest that patients found critical information easier to find on our redesigned discharge instructions. We hope to incorporate these findings into future discharge instructions to improve patient care.

## PREVALENCE OF HEART FAILURE IN RURAL COMMUNITIES OF PAKISTAN

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**Objectives:** To determine the prevalence of heart failure (HF) and its causes in rural population of Pakistan.

**Methods:** Sample of 2000 subjects, age = 30 years, was randomly selected using stratified sampling technique from 22 villages of Rahim Yar Khan, Pakistan. Predesigned questionnaire was used for interview. Physical examination, fasting blood glucose, electrocardiography (EKG) was performed in all subjects. Echocardiography (ECHO) was done only on clinically suspected cases. Overall prevalence was defined on ECHO evidence of systolic/diastolic dysfunction and any of the three; current symptoms pertaining to HF, past history of HF and ECG abnormalities.

**Results:** Prevalence of HF was 3.4%. Prevalence based on each criterion including current symptoms, past history, ECG and echocardiography was 24.4%, 6.3%, 1.7% and 1.8% respectively. LV diastolic dysfunction was 1.6% and systolic LV dysfunction was 0.4%. Common associations of HF were obesity (40.6%), hypertension (39.1%), coronary heart disease (27.5%) and diabetes 8.7%. Hypertension, coronary heart disease (CHD), illiteracy and smoking in women were found to be strongly associated with HF.

**Conclusion:** This study found a high prevalence of HF in rural communities of Pakistan. Hypertension and CHD were dominant causes. Preventive strategies should be implemented to control these risk factors.

Table I: Distribution of Causes in Heart Failure

Causes of HF	% age	P. value
Obesity/Overweight	40.6%	0.4
Hypertension	39.1%	0.007
HDL-C	30.4%	0.5
Coronary Heart Disease	27.5%	0.01
Triglyceride	11.6%	0.12
Diabetes mellitus	8.7%	0.5
Total Cholesterol	2.9%	0.7
Valvular Heart Disease	2.5%	0.04
LDL-C	1.4%	0.9
Cardiomyopathy	0.8%	0.03

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QUANTIFYING VENTRICULAR ECTOPIC BEATS: ASSESSING THE ABILITY OF STANDARD 12-LEAD EKG TO PREDICT PVC BURDEN ON AMBULATORY HOLTER MONITORING

Noelle C. Garster, MD, MS and Charles A. Henrikson, MD, FHRS

**Introduction:** Ventricular ectopic beats (PVCs) are a common cause of palpitations and are often captured on standard 12-lead electrocardiogram (EKG). Determining the correlation between PVC quantity captured on EKG and PVC quantity captured on Holter monitoring would help define the utility of an EKG as a proxy measure for PVC burden on ambulatory monitoring.

**Methods:** We performed a single center, retrospective chart review of patients that completed both EKG and 24 or 48 hour ambulatory Holter monitoring at Oregon Health and Sciences University Hospital between 01/01/2011 and 12/31/2013. Primary outcome measures included quantity of PVCs on EKG, mean quantity of PVCs on Holter, and percentage of total beats on Holter monitor recorded as PVCs.

**Results:** A total of 1,240 patients were included in the analysis. Of these, 1128 patients had 0 PVCs on EKG and 112 patients had = 1 PVC on EKG (38 of these had > 3). Mean number of PVCs on Holter monitoring was 221 vs 10,564 for 24 hour Holter and 1432 vs 21,994 for 48 hour Holter respectively for these two groups. This difference was statistically significant. Percentage of total beats on Holter monitor recorded as PVCs were 1.40%, 6.00%, and 18.05% for patients with 0, 1 to 3, and > 3 PVCs on EKG respectively. 92.7% of patients with 0 PVCs on EKG had less than 1% PVC burden on Holter compared to 18.4% of patients with >3 PVCs on EKG. 63% of patients with >3 PVCs on EKG had 13% or greater PVC burden on Holter. Moderate to strong correlation was observed between PVC quantity on EKG and PVC quantity on Holter ( $r=0.63$ ).

**Conclusion:** The findings of our study demonstrate correlation between PVC quantity on EKG and PVC quantity on Holter monitoring. This study also confirms that EKG can be used as a proxy for quantifying mean PVC burden. This information can be utilized for quick bedside estimation of overall PVC burden if a patient's EKG is available. Furthermore these findings represent an opportunity for cost-savings by reducing redundant evaluations and also may potentially result in reduced time-to-referral for patients with symptomatic ventricular ectopy.

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CLEAR AS BLOOD: SELF-REFLECTION TO ENHANCE SHARED-DECISION-MAKING IN PROSTATE  
CANCER SCREENING DISCUSSIONS

Jia Luo, MD Maria Peila, MD, MSc Katherine Iossi, MD Carol Sprague, MD

**INTRODUCTION:** The average health literacy in the United States is at an eighth grade reading level. Low health literacy negatively impacts patient care. At the same time, studies have shown that physicians use up to two medical jargon words a minute; half of these are left unexplained. Awareness of jargon use can help improve communication, but helpful interventions are not known. Here we present an exercise in self-reflection demonstrating improved resident communication over four weeks.

**METHODS:** To evaluate medical jargon use, residents were asked to pair up and record a simulated patient-physician shared-decision-making roleplay. Prostate cancer screening was chosen, as it requires residents to engage in a more complex conversation than simply recommending a test. After recording their discussion, residents were asked to listen to and transcribe their conversation and identify words they would consider “medical jargon.” They were also supplied with a “clear communication strategies” handout and asked to reflect on utilizing these recommendations in future discussions. The roleplay was repeated four weeks later. Flesch-Kincaid, the most commonly used calculator for literacy, was used to calculate grade level of the transcription. Results are expressed in mean (standard deviation) format. Wilcoxon rank-sum 2-tailed tests were used to compare paired results.

**RESULTS:** Twenty-two residents completed the roleplay scenarios. Over 50 unique jargon terms were identified, including: *biopsy, symptom, false positive* and *screening*. All residents used more unique jargon words than they self-identified. Unique jargon use decreased from 6.4 (SD=2.4) to 4.0 (SD=1.6) words per discussion after four weeks ( $p=0.0003$ ). After the four weeks, residents explained the definition of jargon terms more frequently 14% vs 31% (SD=19% and SD=23%, respectively;  $p=0.001$ ), and asked more clarifying questions 1.2 vs 1.9 questions (SD=1.0 and 1.1, respectively;  $p=0.015$ ). Grade level analysis showed improvement in grade level at which information was conveyed from 8.5 (SD=2.0) to 7.2 (SD=1.9) ( $p=0.0012$ ). The four residents who did not identify any jargon did not improve with this intervention. Several residents commented that the exercise made them want to change their approach to patient communication.

**CONCLUSIONS:** These results confirm other studies that residents use significant amounts of jargon communicating with patients. Residents in our study identified fewer jargon terms in their own conversations than were present, including several who did not identify any. This suggests overestimation of communication clarity and highlights the need for communication training in residency. A short intervention using self-reflection to raise awareness of medical jargon use may improve resident-patient communication in future encounters. During follow up, residents were able to tailor their language to on average less than an 8<sup>th</sup> grade level. Future studies will include more residents to identify the ideal curriculum for long-term awareness of health literacy and clear communication that can be sustained past residency.

LOWER CIRCULATING LEVELS OF TUMOR NECROSIS FACTOR RELATED APOPTOSIS-INDUCING LIGAND (TRAIL) ARE ASSOCIATED WITH INCREASED SUB-CLINICAL CORONARY ATHEROSCLEROSIS IN SMOKERS

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**Introduction:** Cardiovascular disease is the leading cause of death worldwide. The basis of cardiovascular events, such as myocardial infarction, stroke, and cardiovascular death is atherosclerosis. The pathophysiological mechanisms that promote the development of vascular disease are inflammatory processes leading to cell death. Apoptosis is essential to the pathogenesis of cardiovascular disease particularly acute coronary syndrome. Tumor necrosis factor (TNF)-related apoptosis-inducing ligand (TRAIL) is a protein ligand that induces apoptosis. TRAIL and its receptors are expressed in the cardiovascular system and soluble TRAIL is measurable in human blood. There is significant *in vitro* evidence demonstrating anti-atherogenic effect of circulating TRAIL. Also, decreased circulating TRAIL levels have been reported in patients with acute myocardial infarction and in those undergoing coronary catheterization due to suspected coronary atherosclerosis. However, it remains unknown if TRAIL levels are associated with sub-clinical coronary atherosclerosis.

**Methods:** The study cohort included 461 current and former smokers enrolled in the Pittsburgh Specialized Center for Clinically Oriented Research in COPD (SCCOR) study. Major inclusion criteria were age between 40-80 years, and >10 pack year smoking history. Serum TRAIL levels were measured by electrochemiluminescence immunoassay, according to the manufacturer's protocol (Meso Scale Discovery, Gaithersburg, Maryland). Coronary atherosclerosis was assessed by a validated visual coronary artery calcium scoring system using non-EKG gated chest CT scans (Weston score). Ordinal logistic regression models were used to identify significant associations between categories of coronary artery calcium (CAC) scores (0, 1-3, 4-8, and 9-12) and TRAIL levels, and to adjust for cardiovascular risk factors.

**Results:** The mean age of the 461 participants was  $65.7 \pm 6.3$  years, 52.2% were male, and the mean pack years of smoking was  $55.0 \pm 30.8$  years. In multivariate analyses, each standard deviation (SD) decrease in TRAIL level was associated with 1.22-fold increase in the odds of having calcium scores in one higher category ( $p=0.04$ ) after adjusting for age, gender, race, body mass index, hypertension, diabetes, hyperlipidemia, pack-years of smoking and current smoking status.

Each SD decrease in TRAIL level was associated with increased risk of mortality (HR=1.37, 95% CI 1.06-1.69,  $p = 0.01$ ) independent of age, gender, BMI, pack years of smoking, smoking status, percentage predicted forced expiratory volume (FEV1) and FEV1/forced vital capacity (FVC) ratio.

**Conclusion:** Our results expand on current data linking decreased TRAIL levels with increased atherosclerosis by demonstrating a novel association between lower circulating TRAIL levels and increased risk of subclinical coronary atherosclerosis and mortality. Further investigation of the atheroprotective effects of TRAIL is warranted.

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TRAINING RESIDENT PHYSICIANS BY IMPLEMENTING TRANSITION OF CARE CURRICULUM AND ROTATION DECREASES THE GAP IN PHYSICIAN TO PHYSICIAN COMMUNICATION: A STUDY OF CARE TRANSITIONS

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Introduction: Improper care transitions account for more than 12 billion dollars in estimated added health care costs. Lack of communication between hospitalist and primary care physician (PCP) from admission through discharge leads to unnecessary duplications and potential areas for medical error. In a prospective observational study ( phase 1 ) performed at our institution to identify gaps in care transitions, only 37% of primary care physician's (PCP's) were contacted by hospitalists upon discharge. Measures to improve transitions of patient care were studied and implemented. The objective of this study ( phase 2 ) was to evaluate the effect of a resident transition of care champion (TOCC) on metrics of transition of care (TOC).

Methods: We conducted a prospective observational study of 306 patients admitted to the inpatient service of two acute care hospitals, one with TOCC group and the other without (control group ) over 16 weeks period. Primary study end points were: a) PCP contacted upon discharge, b) follow up appointment made upon discharge. Test hypothesis was "The presence of TOCC is associated with a higher occurrence of primary end points". Demographic data, admitting diagnosis and events during hospitalization were recorded. Data were analyzed using SPSS 20.0.

Results: Out of 306 patients enrolled, 156 were in TOCC institution and 150 were in control institution. Occurrence of primary endpoints was significantly higher in TOCC institution compared to control [PCP contact upon discharge 89.7 % vs 65.1 %,  $p= 0.001$ ) and follow up appointment (82 % vs 67.1 %,  $p= 0.003$ )]. Presence of TOCC at the institution was a significant independent predictor of the primary end point (OR = 4.2, 95% CI [2.1 - 8.3],  $p = 0.0001$ ).

Conclusion: Our study shows that appointment of TOCC, significantly, increases TOC as measured by direct physician to physician communication. Implementing protocol based checklist and trigger for communication may further improve TOC between inpatient team and outpatient providers.

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MED W(REC)K: SHUFFLING TO THE DIAGNOSIS

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Case Presentation: Mrs. C, a recently widowed 71-year-old female with a history of coronary artery disease, was admitted for uncontrolled hypertension and acute tubular necrosis. Other history and physical examination were unremarkable. She improved and was discharged on new blood pressure medications amlodipine (Norvasc) 10mg twice daily (with two refills), metoprolol and doxazosin. Over the next 3 months she experienced low energy, slow movements and a "stoic" facial expression. She was hospitalized once and seen by her family physician twice during this time frame. Her symptoms were mistaken for depression and Mrs. C was prescribed citalopram. Soon after, she was hospitalized for lightheadedness and poor ambulation. She was noted to have shuffling steps, a blank face, low voice, and bradykinesia. CT head was normal. Admission medication reconciliation (MED REC) showed that Mrs. C was taking metoprolol, doxazosin, alprazolam, citalopram, and Navane (thiothixene) 10mg twice daily. Upon review of her pill bottles, it was found that her pharmacy had accidentally dispensed Navane (an anti-psychotic) instead of Norvasc. A diagnosis of thiothixene-related drug-induced parkinsonism was made. Thiothixene was stopped and Mrs. C improved.

Discussion: This was mainly a pharmacist error, possibly related to under-staffing and corporate pressure to fill prescriptions within minutes. However, the dose and use of Navane was overlooked by multiple physicians for months. Navane was continued on MED REC during her second hospitalization, even though the indication was unknown. A thorough MED REC and review of indications is an important part of patient safety and is directly related to patient harm. It is proposed that preventable medication errors impact more than 7 million patients and cost nearly \$21 billion annually in all care settings. Another study revealed that upon hospital discharge, 30% of patients have at least 1 medication discrepancy. Decreasing use of pill bottle reviews, suboptimal patient education, and poor discharge communication are all safety risks.

Corrective Actions: Our institution subsequently decided to import external pharmacy records, and implemented a two-step independent medication review on both admission and discharge. This institution does not routinely give medication refills. For purposes of accountability and team effort, we releases weekly reports naming physicians who have not completed a MED REC. Physicians have been educated to write an indication on all prescriptions.

Conclusion: A MED REC offers a chance to review indications, interactions and provide patient education, all of which lead to patient empowerment. When unusual symptoms or poor treatment response occur, a review of medications must be performed.v

## A SHOCK TEAM IMPROVES SURVIVAL IN CARDIOGENIC SHOCK BY DECREASING TIME TO INTERVENTION

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**Purpose:** The aim of the study was to determine if a *Shock Team* improves outcomes in cardiogenic shock (CS). CS is a state of end-organ hypo-perfusion due to cardiac failure. A *Shock Team* was formed at our institution to rapidly assess patients in CS and intervene if appropriate. The team consists of a cardiothoracic surgeon, interventional cardiologist and heart failure physician. When CS is identified, a *Shock Code* is activated and a multidisciplinary team assemble at bedside.

**Method:** We performed a retrospective analysis on 14 *Shock Codes* between 2014-2015. Baseline characteristics, hospital course and hemodynamic data was reviewed.

**Results:** CS was confirmed in 13 patients. The average cardiac index was 1.67 L/min/m<sup>2</sup>. CS occurred due to ST elevation myocardial infarctions (n=7), acute decompensated heart failure (ADHF) (n=5) and LAD dissection (n=1). Six shock codes occurred after cardiac arrest. Seven patients had a right heart catheterization (RHC) within 90 minutes of the shock code, median 24 minutes. All patients had a RHC.

8 patients received mechanical circulatory support in a median of 67 minutes including veno-arterial extracorporeal membrane oxygenation (n=3), intra aortic balloon pump (n=3) and tandem heart (n=2). Patients who received a RHC within 90 minutes and subsequent interventions including temporary mechanical circulatory support and/or inotropes had a 71% 30-day survival versus 16 % for those with RHC at > 90 minutes. (p = 0.064).

46% of all patients survived and were alive at 8 months. Eight patients had an evaluation for advanced therapies; heart transplantation and ventricular assist devices (VAD). One patient received a heart transplant.

**Conclusion:** A *Shock Team* can potentially improve short and long term survival in cardiogenic shock patients by decreasing time to intervention and initiating evaluation for advanced therapies.

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USING INSERTABLE CARDIAC MONITORS TO RISK STRATIFY PATIENTS AND MANAGE ANTICOAGULATION IN ATRIAL FIBRILLATION

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Background: Oral anticoagulants (OACs) have an established role in the prevention of thromboembolic stroke in atrial fibrillation (AF). However in real world clinical practice settings, we find many such patients are at high risk of bleeding and/or insisting on withdrawal of OAC. Recent data has shown that the risk of stroke diminishes as the AF burden decreases. We aimed to ascertain whether an insertable cardiac monitor (ICM)-guided rhythm control strategy might obviate long-term use of OACs in AF patients at high bleeding risk.

Methods: We implanted ICMs in 96 AF patients with high risk of stroke [CHADS<sub>2</sub>=2, CHA<sub>2</sub>DS<sub>2</sub>-VASc score =2] and bleeding [HAS-BLED score =3] after restoration of normal sinus rhythm (NSR) for continuous rhythm monitoring and optimization of antiarrhythmic drugs (AADs). Monthly AF burden was followed and patients were risk stratified into: (i) Group A (always NSR/low AF burden, <1%), (ii) Group B (variable AF burden), and (iii) Group C (high AF burden, always AF). If patients maintained NSR/AF burden<1% for =3 consecutive months, they were offered the option to stop OAC after counseling of risks and benefits. Strict monthly ICM checks would continue. If AF burden rose to >1%, OAC would be restarted while AADs were titrated and risk factors controlled. All patients (age 73.3±11.7 years; 53% male) were followed clinically and with ICM monitoring for 22.04±11.24 months for outcomes including stroke, bleeding, and AAD's adverse effects.

Results: Patients in Group A (n=60), B (n=24), and C (n=12) had similar CHADS<sub>2</sub> (2.10±0.66, 2.05±0.51 and 2.14±0.38, respectively), CHA<sub>2</sub>DS<sub>2</sub>-VASc (3.05±1.01, 2.85±0.99, and 2.43±0.53, respectively) and HAS-BLED (3.02±1.01, 3.40±0.68, and 3.00±0.58, respectively) scores (p>0.05). At study end, 85% patients (n=82) maintained NSR with low risk of stroke, therefore eligible to discontinue OAC. Of those, 72% patients (n=59) discontinued OACs with no subsequent stroke. 39% (n=37) patients remained on OAC, and 11% patients on OAC (n=4) suffered severe bleeding requiring hospitalization, while those not on OAC suffered no such bleeding events. (p=0.02)

Conclusion: In AF patients with high bleeding risk, ICM-guided rhythm control with AADs and assessment of AF burden may allow safe discontinuation of OACs. This reduces patient morbidity and mortality by significantly decreasing major bleeding "events" requiring hospitalization. The ICM guided dose reduction in AADs also decreases the risk for developing significant adverse drug reactions and interactions.

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FACTORS PREDICTING IMPROVED GLYCEMIC CONTROL IN RESPONSE TO BEHAVIORAL SUPPORT INTERVENTIONS FOR POORLY CONTROLLED TYPE 2 DIABETES

Fuller WS, Long JS

Background: Management of type 2 diabetes is complicated by socioeconomic status, duration and severity of diabetes, and medical comorbidities including mental health issues. Prior studies have associated many of these factors with poor glycemic control. However, these factors have not previously been analyzed with regard to how they may influence the efficacy of behavioral support interventions.

Methods: 280 diabetic patients with their last two glycosylated hemoglobin (HbA1c) values > 8% were recruited from outpatient medical practices. Participants were randomized to four arms: usual care, peer mentoring, financial incentives, or a combination of mentoring and incentives. A baseline questionnaire asked about demographics, comorbidities, depression (via PHQ-2), medication adherence and diabetes-related attitudes. HbA1c was measured at baseline, at the conclusion of the 6 month intervention period, and at 12 month follow up. We performed multiple logistic regression to determine which baseline characteristics were predictive of a reduction in HbA1c of at least 1%.

Results: Participants were 70% female and 83% African American. The mean age was 53.1 years (SD 8.6). The mean baseline HbA1c was 9.6 (SD 1.8). At 6 months, odds of having a decrease in HbA1c of at least 1% were 1.6 (95% CI 1.3-2.0) for each 1% increase in baseline HbA1c and 4.0 (95% CI 1.1-15.1) for those with health literacy of 7th-8th grade level as compared to = 9th grade. These associations persisted at 12 month follow up. At 12 months, a baseline PHQ-2 score < 4, as compared to = 4, was predictive of lower odds of HbA1c reduction = 1% (0.3: 95% CI 0.1-0.8). Also predictive of a = 1% drop in HbA1c at 12 months were reporting less diabetes-related distress (OR 2.9, 95% CI 1.2-6.8) and self-identifying more barriers to medication adherence (OR 1.12, 95% CI 1.0-1.2).

Conclusions: Higher baseline score on the PHQ-2 depression screening questionnaire, lower health literacy, lower diabetes-related distress, and perceiving more barriers to medication adherence were predictive of improved responses to behavioral support intervention even after adjusting for baseline HbA1c. Patients with these features represent vulnerable populations in which diabetes control has historically been poor. Behavioral support interventions may have increased efficacy in these patients.

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INCREASED RISK OF OBSTRUCTIVE SLEEP APNEA IN ASTHMATICS ON REGULAR PARTICLE SIZE COMPARED TO EXTRA-FINE PARTICLE SIZE INHALED CORTICOSTEROIDS

First Author: Maria Paula Henao, MD Last Author: Timothy J. Craig, DO

**Introduction:** In asthmatics, increased severity of asthma has been shown to correlate with increased risk of obstructive sleep apnea (OSA). The etiology of this association remains unclear. Inhaled corticosteroids (ICS) have been shown to have a dose-dependent risk of localized side effects including oral candidiasis, dysphonia, and hypertrophy of the tongue. Additionally, inhaled corticosteroids formulated with extra-fine particle size, such as in beclomethasone, have been found to result in improved lung distribution with less localized side effects. In this study, the risk of developing OSA was compared in asthmatics using extra-fine and regular particle size inhalers.

**Methods:** Data from 9,105 asthmatics on ICS were obtained using Informatics for Integrating Biology and the Bedside (i2b2) at the Hershey Medical Center. For this study, we limited ICS to beclomethasone, ciclesonide, mometasone, and fluticasone. Beclomethasone and ciclesonide were categorized as extra-fine particle inhalers, defined by a mass median aerodynamic diameter of less than or equal to 1.1  $\mu\text{m}$ . Mometasone and fluticasone were classified as regular particle size, which was defined as having mass median aerodynamic diameter greater than 1.1. Statistical analysis was performed, including logistic regression.

**Results:** Obstructive sleep apnea was diagnosed in 14% of the patients in the extra-fine ICS group ( $n=2,157$ ) compared to 17% of patients in the regular ICS group ( $n=6,948$ ). In asthmatics of all ages ( $n=9,105$ ; range 0-90, mean 43), users of regular ICS were found to have greater odds (OR 1.58, 95%CI 1.14-2.18) of having a diagnosis of OSA when compared to users of extra-fine ICS. However, when limiting the analysis to patients aged 18 and older ( $n=6,992$ ), users of regular ICS did not have a statistically significant increased risk of a diagnosis of OSA (OR 1.19, 95%CI 0.95-1.49). Adult males, however, did have a significantly increased risk (OR 1.69, 95%CI 1.49-1.91). In the pediatric population (ages less than 18,  $n=2004$ ), use of regular ICS had a statistically increased risk of a diagnosis of OSA compared to users of extra-fine ICS (OR 1.58, 95%CI 1.14-2.18). There were no identifiable significant changes in OSA risk when stratifying by race.

**Discussion:** In this retrospective study, our data suggest that greater particle size of ICS may lead to an increased risk of developing OSA, although further prospective studies are necessary. Given the association between OSA and asthma, physicians should be aware of this possible side effect of ICS and the potentially better outcomes when using ICS with extra-fine particle size.

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INCIDENCE AND FOLLOW-UP OF PREECLAMPSIA, AN IDENTIFIED CARDIOVASCULAR RISK FACTOR, IN AN INTEGRATED HEALTH CARE NETWORK

Karlee Hoffman, Megan Kamath, Indu Poornima

**Background:** Pre-eclampsia, defined as new-onset hypertension and proteinuria after 20 weeks of gestation, occurs in approximately 3-5% of pregnancies. Studies have shown a correlation between pre-eclampsia and the development of cardiovascular disease later in life, and the American Heart Association has recognized pre-eclampsia as a risk factor for cardiovascular disease. We sought to define the incidence of preeclampsia within our integrated health network and the incidence of hypertension and diabetes on follow-up.

**Methods:** We identified women with a billing diagnosis of preeclampsia in our system between the years of 2009-2015. We retrospectively collected demographic, obstetrical and cardiovascular risk factor data by chart review on women with preeclampsia in calendar year 2012.

**Results:** The average incidence of preeclampsia was 10.4% (2661/25058) from 2009-2015 and in 2012 was 10% (n=329). Mean age was 30±6 years, 75% were Caucasian, 60% had 2 or less pregnancies, and 56% had a Cesarean section. In 3 year follow-up, 66% had follow-up with either gynecologist or primary care physician. In the same time period, 23% (75/329) were diagnosed with hypertension and 21% (68/329) were diagnosed with diabetes. In comparison to women without preeclampsia who delivered in our network, the incidence of diabetes was significantly higher (21% vs 11%, p=0.005).

**Conclusions:** Less than 70% of women diagnosed with preeclampsia during pregnancy are seen in routine follow-up and among them hypertension is common and a higher incidence of diabetes was noted compared to non-preeclamptic women. More rigorous follow up of this population is required to improve cardiovascular outcomes.

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## TYPE 2 DIABETES MELLITUS: A MULTIMODAL APPROACH TOWARDS IMPROVING PATIENT OUTCOMES

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Jefferson Hospital Ambulatory Practice is an Internal Medicine resident clinic that sees mostly Medicaid/Medicare patients. 20% of our patients have diabetes. Over the last two years, our group has devised a Quality Improvement project targeting our diabetic population. Nationally, mean diabetic A1c has declined from 7.6% in 2002 to 7.2% by 2010. However, 23% of diabetics in the US remain at high risk for complications with an A1c > 8%.

The aim of this study was to achieve a 15% increase in the number of diabetic patients with A1c < 8% from September 2014 through April 2015. Patient data sheets were generated monthly to identify the target population. The intervention consisted of implementing three systems, which utilized a multitude of resources within the clinic. On a monthly basis, residents performed phone calls to all identified target patients, and office staff mailed a standardized letter with appropriate lab slips. Second, in-office phlebotomy was established to facilitate completion of lab tests. Lastly, an in-house doctor of pharmacy visited the clinic twice weekly for diabetic education and co-management; the target patient list was provided to office staff and all patients were scheduled for individual pharmacy appointments.

With the implementation of the three system changes detailed above, we achieved an 8% increase in diabetics with A1c < 8% from September 2014 through April 2015. As of April 2015, 71% of diabetics in our practice have an A1c < 8%.

The primary aim was not achieved, but the outcome measure nonetheless yielded significant, positive results. It is expected that this trend will continue with further interventions that focus on the coordinated care needs of our patient population. Our recent practice changes provide novel solutions to delivering high-value, high-quality care in a resident-run clinic that serves a diverse, underserved population. Future interventions may include instituting checklists, designating a quality improvement team to oversee the diabetes patient registry, and coordinating pre-visit planning. Challenges such as patient continuity and compliance will persist. Nonetheless, it is essential that the approach to outpatient medicine continue to evolve while always considering the medical and environmental complexity of the patient population.

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HOW DO WE MAKE HEALTH POLICY FOR LOW-INCOME POPULATIONS? AN ANALYSIS OF PUBLIC INPUT AND MEDICAID WAIVERS UNDER THE AFFORDABLE CARE ACT

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**Introduction:** State Medicaid programs comprise the largest single source of health coverage in the United States, covering 68 million people. Since the Supreme Court ruled that the Affordable Care Act's (ACA) Medicaid expansion was optional for states, several states have received federal approval to expand coverage to low-income adults via Section 1115 waivers. These waivers allow states to modify coverage by using private health insurance plans, requiring cost-sharing, and/or limiting benefits. Although Section 1115 waivers have existed for decades, the ACA implemented a new process under which the public can submit comments on pending Medicaid waiver applications. No prior research has examined this comment process. This study sought to understand the role of the public comment process on Medicaid waiver approval across five states (AR, IN, IA, MI, PA).

**Methods:** Directed content analysis methods were used to identify and code themes in a total of 291 unique public comments. A 15-item coding instrument was created that included codes for type of commenter, content related to the provisions of waiver applications, and whether each comment supported or opposed the waiver application. The two coders then independently coded a random sample of 40 comments not included in pilot testing. Inter-rater reliability ranged from moderate to near perfect ( $0.50 \leq K \leq 0.95$ ). The remaining 251 unique comment letters were divided and independently coded by the two coders.

**Results:** Our final analytic sample included 291 unique comment letters (AR 4, IA 19, IN 103, MI 7, PA 158). The majority of comment letters (64%) were from private citizens; 9% were from health care providers; 5% were from non-health industry groups; 22% were from consumer or political advocacy groups. The most frequently mentioned concerns about the waivers were limits on coverage (53% of comments), cost-sharing (45%), and effects on vulnerable populations (41%). Nearly 21% of letters unequivocally supported the waiver application, 26% supported the application with changes, and 54% opposed the waiver application. Private citizens and consumer advocacy groups more often opposed the waiver applications (68% and 35%, respectively) than health care provider or industry groups (27% and 0%, respectively).

**Conclusion:** The new Medicaid section 1115 waiver comment process resulted in a robust participation. Citizens provided the majority of comments, focusing on benefit limits especially for vulnerable populations. Health care providers tended to align with citizen concerns, while advocacy groups tended to ask for specific policy changes to the waivers. Non-health industry groups overwhelmingly supported the waiver applications. This high level of participation might be explained by the fact that the waiver comment process was new, freely available online, and pertained to the highly salient issue of Medicaid expansion.

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DEPENDABILITY OF DIASTOLIC DYSFUNCTION ON AGE, BMI AND DIASTOLIC BLOOD PRESSURE

First Author: Safi U Khan, MBBS Zachary Wolfe, MD Issa Makki, MD

**Introduction:** Factors which predict the presence of diastolic dysfunction are not well defined. The aim of this study was to evaluate predictors of diastolic dysfunction in patients with hypertension. The study also aimed at evaluating independent predictors of diastolic dysfunction, calibrated by E/A ratio.

**Methods:** 75 patients with diastolic dysfunction (43 men; age [mean (SD)] 54 (18) years) were evaluated in a cross-sectional study conducted from July, 2011 to March, 2012 in the Cardiology Division, Nishtar Hospital, Pakistan. Echocardiography and anthropometric measurements were performed in the morning after a 12-hour overnight fast. Diastolic dysfunction was defined according to a modified algorithm by Khouri et al, including all I-III grades of severity with the following parameters [mean (SD)]: E/A [1.35 (0.63)], deceleration time (DT) [207 (51) ms] and E/E' [8.9 (3.06)]. Patients were classified into hypertensives (BP = 140/90 mmHg) and normotensives. SPSS version 20 (SPSS, Chicago, IL, USA) was used for analyses with P =0.05 as level of significance.

**Results:** Analysis revealed that old age [64 (13) years], higher BMI [33 (18) kg/m<sup>2</sup>], and increased EDV (end-diastolic volume) [115 (36) ml] contributed to diastolic dysfunction in hypertensives. Pearson's correlation between E/A ratio and anthropometric, blood pressure parameters, and cardiac variables was assessed. The variables showing statistical significance were then incorporated into multiple regression analysis to assess for independent predictors of E/A. Multiple regression modeling showed that 49% variation in E/A is contributed by age alone. BMI, EDV, LVID and DBP contribute further by 17%.

**Conclusion:** Older age and higher BMI contribute to diastolic dysfunction with preserved EF in patients with hypertension. Age, BMI, EDV and DBP are independent predictors of diastolic dysfunction in general.

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PATIENT PERSPECTIVES ON REASONS FOR FAILURE TO INITIATE ART IN MOZAMBIQUE:  
COMBATING STIGMA WITH COMPASSIONATE COUNSELING.

Anita Lyons, Laurina Moiane, Elpidio Demetria, Peter Veldkamp, Ramakrishna Prasad

**Background.** While antiretroviral treatment (ART) is now available to millions of patients in Africa, attrition is high at every level of the care cascade, from testing to ART initiation. At the Sao Lucas Health Center in Beira, Mozambique only 55% of patients who are eligible to start antiretroviral therapy (ART) actually initiate treatment within six months of diagnosis

**Objective.** This study sought to gather patient perspectives on failure to initiate ART at an urban health center in Mozambique.

**Methods.** HIV-positive patients  $\geq 18$  years of age linked to care at the Sao Lucas Health Center in Beira, Mozambique were asked three open-ended questions: (1) Why do so many patients who are eligible not start ART? (2) How can health professionals encourage patients to start ART? (3) How can we improve care? Survey answers were recorded in Portuguese, translated, and analyzed using qualitative methods to identify themes.

**Results.** Forty-nine participants had median age of 42 years, were 59% female and 78% currently on ART. Shame, fear, and denial were the most commonly cited reasons for failure to start medications. Participants described compassionate counseling and home visits as ways to encourage patients to follow-through with treatment. Practical suggestions for improvement in care included providing patients with food, opening the clinic earlier, and shortening waiting lines.

**Conclusions.** Shame and fear remain important barriers to care of HIV-positive individuals in Mozambique. Streamlining the healthcare experience and emphasizing a compassionate approach may benefit patients in the pre-ART period.

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TREATMENT OF ADENOVIRUS INDUCED GRAFT DYSFUNCTION IN RENAL TRANSPLANT RECIPIENTS

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Background: Fever, hematuria, flank tenderness and leukocytosis in renal transplant patients generally result in an extensive infectious disease work up. These clinical findings can be the presentation of adenoviral nephritis. At this time, there are no standardized treatment guidelines for this infection.

Purpose: A 75 year old female, 365 days post renal transplant was evaluated for hematuria, fever, and acute kidney injury. Initial work up including cystoscopy, urine cultures, and tests for BK virus, CMV, and EBV were negative. Renal biopsy was done and histopathological findings were suggestive of graft rejection. However, her serum and urine PCR were positive for adenovirus too. In the clinical setting, it was deduced that Adenoviral nephritis had mimicked graft rejection. Immunosuppression therapy was reduced and IVIG was administered. Fevers abated and her kidney function returned to baseline. We did a retrospective literature review to understand various treatment regimens for adenovirus infections in renal transplant patients.

Results: : Literature analysis revealed 51 adenovirus nephritis cases presenting with acute kidney injury (n=48), hematuria (n=44), fever (n=39) and dysuria (n=27) as the 4 most common signs and symptoms. Creatinine at baseline ( $1.45 \pm 0.66$ ), presentation (Cpres  $3.00 \pm 1.35$ ) and resolution (Cres  $1.49 \pm 0.58$ ) was reported in 41 of the cases. The mean follow up duration was 426 days. Treatment regimens were varied and included combinations of reduced immunosuppression (n=27), pulse dose steroids (n=17), IVIG (n=15; Cpres=2.22) and antivirals (n=14; Cpres=2.25), IVIG+antivirals (n=9) and IVIG without antivirals (n=6). There was no significant difference, as demonstrated by graft recovery and change of creatinine from baseline, in outcomes with the different regimens.

Conclusions: Adenovirus infections should be considered in patients with similar clinical presentations. Our review showed that therapy for adenovirus graft infection needs to be more streamlined. In the reported cases thus far, variations of using IVIG and antiviral medications did not change the outcomes. Reduction in immunosuppression has been associated favourable outcomes and should be the initial step for therapy.

## CAN MONOCYTOSIS ACT AS AN INDEPENDENT VARIABLE FOR PREDICTING DEEP VEIN THROMBOSIS?

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**Introduction:** Deep venous thrombosis (DVT) is a manifestation of venous thromboembolism (VTE). VTE is a major health care problem resulting in significant mortality, morbidity, and expenditure of resources. Approximately 1 percent of hospital admissions in the US are for VTE. It has been estimated that there are 900,000 cases of pulmonary emboli (PE) and DVT per year resulting in 60,000 to 300,000 deaths. About two-thirds of VTE cases are associated with a hospitalization within the prior 90 days, emphasizing the importance of medical illness, major surgery, or immobilization as risk factors. VTE contributes to significant morbidity and mortality both in the community and in hospital.

**Methods:** Retrospective study conducted in a tertiary Hospital from the Metropolitan city of San Juan, Puerto Rico. Patient's selection included those from January 2010 to December 2015. A total of 46 records were obtained from the hospital database following the established inclusion and exclusion criteria. For the control group a total of 42 record were selected. Patients included in this study were admitted with the diagnosis of deep venous thrombosis identified either by lower leg Doppler or computer tomography. Data collected was tabulated and entered in an Excel spreadsheet for further analysis. Statistical method used was pearson chi square test and fisher exact test. Sensitivity, specificity and positive predictive value were measured.

**Results:** A total of 88 records were evaluated. These records were distributed as follows; 46 patients with deep vein thrombosis and 42 patients for the control group. In the association of monocytosis with DVT the  $p$ -value measured was less than 0.001, with an Odd Ratio (OR) 9.35 and Interval Confidence (IC) 95% (3.2-27.3). The  $p$ -value for eosinophilia with DVT was 0.092, for males with DVT the  $p$ -value was 0.35 and age related groups with DVT  $p$ -value was 0.720. Sensitivity of monocytosis was 67.3%, specificity 80%, positive predictive value (PPV) 79.49% and negative predictive value (NPV) 63.9%.

**Conclusion:** This study revealed the association between monocytosis and DVT, thus patients with monocytosis are more likely to have or develop DVT. Our results indicated that elevated monocytes have a sensitivity of 67%, specificity of 81% and a positive predictive value of 78% identifying DVT. This evidence is consistent with previous studies establishing that monocytes could have an important role with the coagulation cascade activation and the formation of DVT. The association of monocytosis with DVT can be used in the future as a significant tool in those patients with suspected DVT. Adding monocytosis as a reference lab value in the pre-test probability for DVT to the current guideline established by the American College of Physicians and the American Academy of Family Physicians could be useful to increase the diagnostic yield of DVT

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RISK FACTORS FOR READMISSION IN PATIENTS ADMITTED WITH FEBRILE NEUTROPENIA

Justin Van Klein, Rebecca M. Slotkin, Andrew Gillis-Smith, John L. Reagan

Background: Febrile Neutropenia (FN) is a serious condition where a patient has a very low absolute neutrophil count (ANC) and at fever, which may indicate a serious infection. These patients are admitted to the hospital frequently for antibiotics and observation. These hospitalizations have been shown to be associated with higher costs than these patient's non-FN counterparts. The purpose of this study is to identify specific risk factors that might indicate that a patient is at higher risk for 30 day readmission.

Methods: Patients were selected via database quest for patients admitted with  $ANC < 1500$  and fever ( $T > 100.3$ ) on admission. Those admitted for FN and survived until discharge were studied retrospectively. Data was collected for these patients and they were divided into two groups; those with a 30 day readmission and those without. These findings were then analyzed for statistically significant difference.

Results: 141 patients met study criteria, 51 of which had a 30 day readmission (rate = 36%). Significant factors increasing risk for readmission were presence of a hematologic malignancy, diabetes, multiple prior admissions, longer initial inpatient stay, lack of inpatient antibiotics and administration of chemotherapy in the prior 6 months. [Complete dataset available on poster, available by request]

Conclusions: Findings of multiple risk factors for readmission allow clinicians to early identify FN patients at high risk. This may allow clinicians to implement strategies to prevent readmission, reducing cost and increasing quality of life of these patients. Surprisingly, many factors such as MASCC score, degree of neutropenia and outpatient follow up plans did not appear to be significant, though larger study populations may be required to detect this.

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CHAPTER WINNING ABSTRACT MORBIDITY IN THE AGING HIV-INFECTED AND HIV-UNINFECTED POPULATION

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Fourth Author: Philip Keiser, MD

**Introduction:** Increased life expectancy in HIV-infected individuals due to antiretroviral therapy exposes patients to increased risk of age-related morbidities; however, there is limited data on morbidity in aging HIV-infected individuals. We studied reasons for hospital admission in HIV-infected and HIV-uninfected individuals over 10 years to determine if there were associations between HIV status, diagnoses, and age.

**Discussion:** The INGENIX database of United Healthcare was queried for all hospital admissions in patients from the years 2000 to 2010. ICD-9 codes were used to determine the discharge diagnosis and HIV status. Patients were classified into age groups as follows: <35, 35-50, 51-65, >65. Reasons for admissions were compared for each group using a Pearson Chi-Squared test.

**Discussion (cont'd.):** There were 1,219,895 hospital admissions over the examined period. HIV-infected patients were more likely to be admitted for arthritis (1.3% vs. 0.8%,  $P<0.01$ ), cancer (2.6% vs. 1.5%,  $P<0.01$ ), and CKD (3.3% vs. 0.8%,  $P<0.01$ ) under age 35; cancer (5.4% vs. 3.4%,  $P<0.01$ ) and CKD (5.1% vs. 1.1%,  $P<0.01$ ) from age 36-50; and CKD from age 51-65 (7.6% vs. 1.4%,  $P<0.01$ ) and over age 65 (9.4% vs. 1.9%,  $P<0.01$ ) compared to HIV-uninfected patients in the same age groups.

**Conclusion:** HIV-infected individuals are less likely to have chronic illness related to aging than HIV-uninfected individuals over time, but CKD remains a significant problem, especially in older HIV-infected patients.

CAP ASSISTED COLONOSCOPY ENHANCES QUALITY BASED COMPETENCY IN COLONOSCOPY AMONG TRAINEES: A RANDOMIZED CONTROLLED TRIAL

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**Background and Aims:** Competency assessment in colonoscopy trainees have traditionally been informal and subjective. Recent validated assessment metrics such as the Assessment of Competency in Endoscopy (ACE) tool have revealed that the minimum procedure threshold to reach competency may be higher than once assumed. Cap assisted colonoscopy (CAC), in which a flexible transparent cap is attached to the end of the endoscope therefore improving visualization, may be a practical method to improve quality based competency measures in trainees. However, evidence to support this practice is lacking. We aim to compare quality based competency measures in CAC versus standard non cap colonoscopy (SC) among trainees with no prior experience in a randomized controlled trial.

**Methods:** This study was conducted at a single safety net university teaching hospital in the United States. All colonoscopies performed by three gastroenterology fellows without prior colonoscopy experience in the first three months of training were eligible for enrollment. Patients were excluded if they were of age < 18 or > 90, pregnant, had history of colon resection, diverticulitis within 1 month, current symptoms of colonic obstruction, severe hematochezia, referral for endoscopic mucosal resection, or required an unsedated procedure. Patients were randomized to either CAC or standard non cap colonoscopy (SC) in a 1:1 fashion via a computer randomization algorithm.

Patient demographics, outcome parameters, and ACE tool assessment were recorded for every procedure. All colonoscopies were performed under the supervision of board certified attending gastroenterologists.

Primary outcome was cecal intubation time (CIT). Secondary outcomes were independent cecal intubation rate (ICIR), polyp detection rate (PDR), adenoma detection rate (ADR), and ACE tool scores.

**Results:** A total of 203 colonoscopies were enrolled, 101 in CAC and 102 in SC. Baseline characteristics between groups were similar. CAC resulted in was significantly faster CIT at  $13.7 \pm 0.74$  mins compared with  $16.5 \pm 0.88$  mins in SC group ( $p = 0.02$ ). In multivariable regression modeling, this association remained significant after adjustment for accumulated colonoscopy experience. ICIR was also improved at 79.2% in CAC compared to 66.7% in SC ( $p = 0.04$ ). PDR was not significantly different at 52.5% in CAC and 52.9% in SC. ADR was not significantly different at 37.6% in CAC and 43.1% in SC ( $p = 0.42$ ). Distribution of overall motor and cognitive scores on ACE tool assessment were also significantly different between groups.

**Conclusions:** CAC resulted in significant improvement in CIT, ICIR, and overall ACE assessment scores when compared to SC in colonoscopy trainees without prior experience in the first three months of training.

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SHORT TERM APPLICATION OF TOCILIZUMAB DURING MYOCARDIAL INFARCTION (STAT-MI)

Christopher D. Smith, MD, Charles Haller, MD, Bryan C. Ramsey, MD, Matthew B. Carroll, MD

**Background:** Acute myocardial infarction (MI) occurs when blood supply falls below critical levels and normal cellular maintenance mechanisms are overwhelmed. Interleukin-6 is a known pro-inflammatory cytokine released in high levels during MI and is associated with poor outcomes. Tocilizumab is a humanized monoclonal antibody against the IL-6 receptor currently FDA approved for treatment of rheumatoid arthritis.

**Methods:** In a randomized, double-blinded, placebo controlled trial we assigned patients admitted to a single academic community hospital for acute MI to receive a single dose of 162mg subcutaneous tocilizumab or placebo injection in addition to standard of care medications and interventions. Demographic, co-morbidity, medical therapy data was collected as was pre and post injection routine labs and CRP levels. Patient pharmacologic and interventional treatments during their hospitalization were recorded and 30 day follow up major adverse cardiac event (primary endpoint), readmission and CRP data (secondary endpoints) was collected via phone interview, chart review and reach out to community medical centers to obtain further data.

**Results:** Early data analysis was triggered after a safety event in which two patients in the tocilizumab arm with ejection fractions below 45% were re-admitted for recurrent MI. Initial data from the first 16 subjects revealed no statistical significant difference in baseline demographics, comorbid conditions, medical therapies. There was no significant difference between study arms in medical or interventional therapies administered during hospitalization. Additionally no statistically significant difference was noted between study arms in either primary or secondary endpoints, namely the rate of major adverse cardiac event occurrence, readmission rates and the pre, post or 30 day follow up CRP levels .

**Conclusion:** At this time, with a modest number of subjects enrolled, no significant difference in MACE occurrence, CRP levels or QTc measurements have been noted, however single dose TCZ appears to be a safe adjunct therapy in acute MI. Recruitment is ongoing with planned repeat safety and efficacy analyses per the protocol.

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## IMPLEMENTATION OF A RAPID RESPONSE SYSTEM: EVALUATING THE EFFECT ON ACTIVATION AND CODE BLUE RATES

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**Introduction:** Critical deterioration in patients is often preceded by measurable signs of physiological decline during the hours prior to the event. The introduction of a Rapid Response System (RRS) has been shown to decrease mortality and cardiopulmonary arrests outside of the ICU. The purpose of this study was to evaluate the impact of a formal RRS training program with mandatory activation criteria on activation rates, call characteristics, and code blue events.

**Discussion:** A quasi-experimental pre-test, post-test design was used to assess outcomes following implementation of a formal RRS at a large military medical center. RRS implementation consisted of a mandatory hour-long lecture, computer-based training, and a marketing campaign with signs/placards available on all medical and surgical wards. The Rapid Response Team (RRT) consisted of a critical care nurse and respiratory therapist with the addition of the patient's primary nurse and responsible provider. Variables included the rate of RRS calls, reason for call, time and day of call, code blue events outside the ICU/ED/OR, and the final disposition of the call. As part of the training, an emphasis was placed on the mandatory RRS notification for any abnormal parameter. Data was gathered retrospectively (January-August 2014) and prospectively (September-December 2014) with respect to the intervention. After the RRS training intervention, the average number of calls per month rose from 39 (17 per 1000 discharges) to 123 (58 per 1000 discharges),  $p < 0.001$ . The mean number of code blue events decreased from 1.5 codes per month to zero per month,  $p < 0.001$ . Prior to the intervention, 45% of RRS calls were transferred to a higher level of care versus 34% after the intervention,  $p < 0.003$ . The most common reasons for RRS activation were tachycardia (27%), hypotension (23%), and staff concern (15%). There was no statistically significant difference for RRS activation regarding the day of the week, time of call, or ward location.

**Conclusion:** Our project demonstrated that a standardized RRS training program increases adherence to the RRS call parameters. Following our intervention, the rate of RRS activations significantly increased with a subsequent significant decline in code blue events and the number of transfers to the ICU. Our project demonstrated that strict adherence to a RRS reduced pulseless cardiac arrests outside the ICU, which has been shown to result in better patient outcomes.

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OBESITY AS INDEPENDENT RISK FACTOR FOR INCREASED MORTALITY IN ADVANCED CARDIAC LIFE SUPPORT (ACLS)

First Author: CAPT Arthur Holtzclaw, MD Contributing Authors: CPT Zorana Mrsic, MD CPT Roland Green, MD CPT James Coyle, DO LTC Christopher Colombo, MD

**Introduction:** The obesity epidemic continues to worsen with over 35% of adult Americans considered obese. Unsurprisingly, one study has shown increased mortality among morbidly obese patients undergoing ACLS for in-hospital cardiac arrest compared to normal weight controls. However, this study occurred prior to the 2010 ACLS guideline update emphasizing quality CPR. We have conducted a pilot study in preparation for a multicenter trial to replicate the above data and evaluate potential etiologies including decreased quality of chest compressions and increased thoracic impedance leading to less effective defibrillation.

**Methods:** A retrospective chart review was conducted to evaluate the effect of a patient's BMI on the outcomes of at our facility from 2009 to 2012. All patients between ages 18 and 89 who underwent ACLS were included with exclusion criteria of pregnancy, amputation, out-of-hospital arrest or lack of documentation. Data was compiled to include patient demographics and comorbidities as well as initial rhythm and outcome of ACLS to include ROSC, survival to 24 hours and survival to discharge. Patients were grouped by BMI with calculation of odds ratios with 95% confidence intervals to evaluate for differences in mortality. Results: 193 episodes of ACLS were identified with 82 excluded. Overall ROSC was 65% with a 24 hour survival of 52% and discharge survival of 37%. Obese patients were found to have a decreased survival that did not reach statistical significance: Odds ratio (OR) for ROSC 0.89 (95% CI: 0.34-2.34), 24 hour survival OR 0.94 (95% CI: 0.37-2.37), discharge survival OR 0.79 (95% CI: 0.30-2.08).

**Conclusions:** This study shows overall increased survival among all groups compared to prior studies but similar trend towards increased mortality among the obese. Using this data to generate a power analysis, we plan to expand this trial to multiple centers to evaluate for changes in mortality pre- and post-ACLS update as well as evaluating possible causes by analyzing objective data for CPR quality and average joules per defibrillation now available in code databases.

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CHAPTER WINNING ABSTRACT

Dr Benjamin Morang DO, Dr Craig Ainsworth MD, Dr Eugen Shippey MD

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**Introduction:** Critical Care bed space is a finite resource nationwide. William Beaumont Army Medical Center is only able to staff 12 intensive care unit (ICU) beds that are shared by medical, surgical and cardiac intensive care patients. Our MEDCEN serves an active duty population greater than 20,000 and intentional and unintentional drug overdoses consistently come into our emergency department (ED). It is currently our hospital policy to admit drug overdoses to the ICU for monitoring, despite initial presentation, vitals and Glasgow Coma Score. The argument for ICU level care is that we will be able to detect early the development of respiratory decompensation, worsening mental status, or life-threatening arrhythmias.

**Methods:** In a retrospective, performance improvement project, we looked at 21 months of admissions at our facility from January 2014 to October 2015, of patients who were admitted with a diagnosis of drug overdose to determine whether the patients required critical care interventions. We defined critical care interventions as the need for intubation for airway protection or hypoxia, need for vasopressor support, need for intravenous bicarbonate therapy for QTc prolongation or other hourly monitoring not offered on our medical floors. Our aim was to determine if disposition from the emergency department to the MICU is required in all patients who present with an overdose of medication.

**Results:** We reviewed the records of 60 admissions to the ICU with multiple etiologies of overdose at admission including acetaminophen, anti-depressants, anxiolytics, sleep aids, and acute alcohol intoxication. We found that if the patients were hemodynamically stable and did not require any of the critical care interventions listed above in the ED, that they did not go on to need any critical care interventions after admission to the ICU. Fifteen of the 60 patients required intubation in the ED, one patient required IV bicarbonate therapy for QTc prolongation and one was started on IV n-acetyl cysteine. None of the patients admitted for monitoring went on to develop complications of their drug overdose that required critical care interventions.

**Conclusion:** This PI project demonstrates that although there is concern for further worsening of mental status to the point of needing intubation, development of arrhythmias, metabolic derangement, or hemodynamic instability in those with overdose, if these patients are initially in stable condition on presentation to the ED, it is not likely that they will require critical care interventions. If our patients did not require intubation or other interventions in the ED, they did not go on to develop a need for intubation or any other critical care intervention after admission to the ICU. Patients who report drug overdose in the ED who are clinically stable may be observed on a medical ward with a sitter and physicians can safely be better stewards of critical care resources.

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IMPLEMENTATION OF ANAL CANCER SCREENING WITH HIGH RESOLUTION ANOSCOPY IN HIV-POSITIVE MEN AT A U.S. DEPARTMENT OF DEFENSE INFECTIOUS DISEASES CLINIC

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**Introduction:** The incidence of human papillomavirus (HPV) related anal cancer in HIV-positive patients appears to have increased despite the introduction of antiretroviral therapy (ART). Reported rates of anal cancer in HIV-positive men who have sex with men (MSM) are surpassing cervical cancer rates; recent estimates at 138/100,000 men per year. Anal HPV infection prevalence has been estimated as high as 57% in HIV-negative MSM and 88% in HIV-positive MSM populations. Similar to cervical cancer screening, there may be clinical and cost effective benefits for routine surveillance of anal dysplasia in the HIV positive population, yet there are no national recommendations. We present data from the only Department of Defense Infectious Diseases (ID) anal cancer screening program.

**Methods:** Naval Medical Center San Diego ID clinic provides care to 600 HIV-positive men. In 2012, we began screening with anal Papanicolaou (Pap) smear and referred any abnormal results for high resolution anoscopy (HRA), performed by an ID attending. Anal Paps were repeated at the time of HRA. Using paper and electronic clinic records, as well as information gathered from pre-procedural social history questionnaires, data were compiled for each patient.

**Results:** To date, 94 patients have been evaluated with HRA. Average age was 42 years, duration of HIV infection 10 years, and CD4 nadir 301 cells/ $\mu$ L. Average CD4 nadirs were 392 cells/ $\mu$ L, 299 cells/ $\mu$ L, 310 cells/ $\mu$ L, 188 cells/ $\mu$ L for those with no dysplasia, ASCUS, LGSIL, and HGSIL, respectively. Of those with low grade cytology (ASCUS or LGSIL), 27% had high grade pathology (AIN II or III); negative predictive value (NPPV) for AIN II or III was [x]%. Of those with high grade cytology (HGSIL), 100% had AIN II/III or one clinically early case of anal squamous cell carcinoma.

**Conclusion:** In 24 months, we've demonstrated the potential need of anal dysplasia surveillance in an HIV-infected population. Additional data is needed in order to link observation and intervention to clinical outcomes, which will help clarify the natural history of dysplasia and guide appropriate follow-up. Furthermore, our experience provides a model to be considered by other ID and HIV clinics interested in beginning anal dysplasia programs.

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## LEADERSHIP TRAINING IN GRADUATE MEDICAL EDUCATION: A SYSTEMATIC REVIEW

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**Introduction:** Leadership is needed more than ever to help navigate the uncertainties and manage change within the rapidly evolving healthcare system. Residency is designed to lay the groundwork required for young physicians to practice safely and independently as the future generation of health care providers. Housestaff are asked to ascend to new leadership roles that change every year, which provides a unique opportunity to develop and hone leadership skills while in training. Despite these expectations and opportunities, leadership skills among residents are often left to the individual's innate abilities, with little formal instruction or training. Unfortunately, this means many residents leave training ill-prepared for leadership positions or disenfranchised with the thought of assuming a formal leadership role. The published literature regarding leadership curricula within graduate medical education (GME) programs was reviewed to better understand how GME programs are preparing residents to lead during residency and in their post-residency careers.

**Methods:** A systematic review of leadership curricula was conducted using the Preferred Reporting Items for Systematic Reviews and Meta-Analysis guidelines. A systematic search of PubMed/MEDLINE, ERIC, and EMBASE databases through August 2014 was conducted by a clinical librarian. A total of 3,413 abstracts were reviewed by two independent reviewers for relevance to the proposed question. Those studies included were retrieved for full-text analysis and the excluded abstracts were coded by the reason for exclusion. Discrepancies from abstract review were resolved by an independent third-party reviewer. References of articles selected for full-text review were hand searched for additional articles not found in the initial database search. MedEdPortal was searched and an additional two papers were identified. Forty-eight papers were included in the review.

**Results:** Primary care specialties were included in the majority of leadership curricula (58%) while subspecialty and surgical services were included in less than a third of studies. The majority of programs were open to all residents while 17% focused on chief residents alone. Projects, mentors, and coaches were components of 44%, 27%, and 29% of curricula respectively. The majority of the curricula (46%) were longitudinal throughout training. The most frequently used pedagogical methods were lectures, small-group activities, and cases (85%, 69%, and 46%). Top content selections included teamwork, conflict resolution and giving feedback, but a wide range of topics have been taught.

**Conclusions:** Published examples of leadership curricula within GME are rare and heterogeneous in their content and effectiveness. A summary of best practices was created from the data and serves as a framework from which future curricula can be designed, implemented, and assessed. Recommendations for required elements of reporting leadership curricula are made in hopes of allowing better sharing of ideas facilitating replication for programs aiming to train their residents in leadership.

## WHAT IS THE IMPACT OF A COMMUNITY HEALTH TEAM ON DIABETES MANAGEMENT IN A RESIDENT CLINIC?

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**Introduction:** It is well known that diabetes mellitus (DM) is a common chronic disease affecting approximately 23.6 million people in the United States<sup>1</sup>. Patients with DM must be involved in disease self-management for optimal outcomes, although this can be a challenging task. Community health teams (CHT) have been organized to assist primary care health providers with chronic disease management. These teams provide multifaceted support and resources for individuals managing chronic illnesses. The Burlington Service Area Health Team (BSAHT) is one such CHT. BSAHT serves 20,000 primary care patients in Chittenden County, Vermont, including patients in the University of Vermont (UVM) Internal Medicine Resident Clinic. Utilizing motivational interviewing techniques and local community resources, RNs, social workers and personal trainers of the BSAHT can individualize the approach to DM management. Studies have reported a reduction in hemoglobin A1c values, a common biomarker of diabetic control, through the use of CHTs 2,3,4,5. To our knowledge, the role of CHTs in conjunction with an Internal Medicine Resident Clinic has never been reviewed to date.

The objective of this study was to see if there was a change in hemoglobin A1c values of patients referred to BSAHT by resident physicians in the UVM Internal Medicine Resident Clinic.

**Methodology:** In this retrospective observational design, patients were referred by a UVM internal medicine resident physician to a BSAHT from January 2009 to January 2013. A referral was made for nutrition help, diabetic education, medication management, exercise advice, or connection to healthy living workshops.

Hemoglobin A1c values pre- and post-referral were reviewed. The data was then further differentiated into those who completed the program and those who did not. Data was analyzed using Paired Samples Test and Repeated Measures Analysis of Variance.

**Results:** From January 2009 - January 2013, 320 referrals were made from the residency clinic to BSAHT. Of those, 277 patients had hemoglobin A1cs pre and post referral. There was a decrease in mean hemoglobin A1c from pre-referral and post-completion of the program for the BSAHT graduates, but this was not statistically significant. Similar results were seen for those who were referred, but did not complete the program. However, for all those referred to the program, (graduates + non-graduates) there was a statistical difference ( $p=0.044$ ) where the average hemoglobin A1c before referral was 7.35 and following referral was 7.19.

**Conclusion:** This retrospective observational study analyzed the Burlington Service Area Health Team's impact on hemoglobin A1c values of patients with diabetes mellitus from the University of Vermont Internal Medicine Residency Clinic. This study suggests that a multifaceted approach to diabetes mellitus management utilizing community health teams may lead to lower hemoglobin A1cs. Although the optimal use of CHTs in a resident clinic is unknown, further studies looking at the involvement of CHTs in resident clinics may be worth exploring given the limitation of healthcare resources.

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## EVALUATING IMPACT OF RESIDENT FEEDBACK ON DIABETIC PATIENT OUTCOMES

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**Introduction:** Residents are often unaware of their performance in clinic as compared to that of their peers. The intent of this project was to improve resident compliance with best care practices for diabetic patients using a novel feedback system to gauge performance in clinic. Per American Diabetes Association guidelines, diabetics should be screened annually for kidney disease with a urine microalbumin/creatinine ratio. Routine screening is integral in preventing and/or treating diabetic nephropathy, which is the leading cause of kidney failure in the United States. Diabetics with proteinuria benefit from close medical follow up to ensure tight blood pressure and glucose control.

**Methods:** The study was conducted at an urban academic ambulatory center. The study population consisted of members of a categorical internal medicine residency program, with a 4+1 ambulatory model. Our sample was comprised of 19 residents with an even distribution among PGY-1, PGY-2, and PGY-3. A total of 249 patients were evaluated.

First, we reviewed the charts of patients seen in clinic by our 19 residents over the previous two ambulatory weeks. Patients with “diabetes mellitus” listed as a medical problem by ICD code were identified. Then, we surveyed these patient’s records for a urine microalbumin/creatinine checked within the past year.

Subsequently, the residents were presented with individual and group compliance rates, and the recommendations for screening were reviewed through a short teaching module. The data was communicated via PowerPoint presentation at a weekly didactics session. Residents were notified that their performance would be reviewed again after another two ambulatory weeks. We hypothesized that there would be a significant increase in compliance with screening after administration of the feedback and education intervention.

**Results:** Pre-intervention data analysis showed that 63% of the 99 diabetic patients seen in clinic over the course of two ambulatory weeks had a screening urine microalbumin/creatinine within the last year. During the two ambulatory weeks post-intervention, there was a 74% screening rate for 150 diabetic patients seen. Nine of 19 residents had screening rates of greater than 80%. The P-value was 0.06 for pre- and post-intervention comparison.

**Conclusions:** While subjective feedback for residents from attending physicians is well-documented and frequently provided, objective feedback via chart audit is a less common component of residency programs. Though our initial data was not significant, it suggests a positive clinician behavioral shift in response to receiving data about patient management performance. A major limitation of this study is the small sample size. A potential confounding bias is that post-intervention data was collected later in the year, when residents were likely more experienced. In the future, we plan to expand this intervention to a larger sample of residents. We also anticipate monitoring appropriate initiation of ace inhibitors for patients with microalbuminuria.

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EFFECTS OF PROLASTIN C (PLASMA-DERIVED ALPHA-1 ANTITRYPSIN) ON THE ACUTE INFLAMMATORY RESPONSE IN PATIENTS WITH ST-SEGMENT ELEVATION MYOCARDIAL INFARCTION (FROM THE VCU-ALPHA 1-RT PILOT STUDY).

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Alpha-1 antitrypsin (AAT) has broad anti-inflammatory and immunomodulating properties in addition to inhibiting serine proteases. Administration of human plasma-derived AAT is protective in models of acute myocardial infarction in mice. The objective of this study was to determine the safety and tolerability of human plasma-derived AAT and its effects on the acute inflammatory response in non-AAT deficient patients with ST-segment elevation myocardial infarction (STEMI). Ten patients with acute STEMI were enrolled in an open-label, single-arm treatment study of AAT at 60 mg/kg infused intravenously within 12 hours of admission and following standard of care treatment. C-reactive protein (CRP) and plasma AAT levels were determined at admission, 72 hours, and 14 days, and patients were followed clinically for 12 weeks for the occurrence of new onset heart failure, recurrent myocardial infarction, or death. Twenty patients with STEMI enrolled in previous randomized trials with identical inclusion and/or exclusion criteria, but who received placebo, served as historical controls. Prolastin C was well tolerated and there were no in-hospital adverse events. Compared with historical controls, the area under the curve of CRP levels was significantly lower 14 days after admission in the Prolastin C group (75.9 [31.4 to 147.8] vs 205.6 [78.8 to 410.9] mg/l,  $p = 0.048$ ), primarily due to a significant blunting of the increase occurring between admission and 72 hours (delta CRP +1.7 [0.2 to 9.4] vs +21.1 [3.1 to 38.0] mg/l,  $p = 0.007$ ). Plasma AAT levels increased from admission (149 [116 to 189]) to 203 ([185 to 225] mg/dl) to 72 hours ( $p = 0.005$ ). In conclusion, a single administration of Prolastin C in patients with STEMI is well tolerated and is associated with a blunted acute inflammatory response.

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ACCURACY AND THE PROPER USE OF THE POINT-OF-CARE ANALYSIS IN HEMOGLOBIN A1C MEASUREMENT

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Serum hemoglobin A1C level is often used as a surrogate marker for the average serum glucose of an individual over the previous three months. At George Washington Medical Faculty Associates (MFA) Hemoglobin A1C can be measured either by sending venous blood for laboratory analysis or by analyzing capillary blood using the point-of-care (POC) instruments in the office. Some clinicians use POC values alone in glucose monitoring while others prefer to confirm the value with the additional laboratory testing. Thus, it is important to compare A1C measurements obtained by the laboratory method to POC analysis in order to determine the accuracy of the latter. Using the MFA electronic medical record system 115 patients were identified to have both POC and laboratory A1C values ordered on the same day, between August 2013 and August 2014. The differences between the two A1C values were analyzed and the average difference and coefficient of determination ( $r^2$ ) was calculated. We also computed the sensitivity and specificity of POC method in diagnosing diabetes while using laboratory analysis as the gold standard. Impact of ordering both A1C measurements on medical management was also evaluated by reviewing clinic notes post testing. A mean linear regression slope of 1.06 with  $r^2$  of 0.9 was obtained by comparing laboratory and POC values. In addition, 94 measurements (84%) had laboratory measurements greater than POC. The differences between the two measurements ranged from 0 to 3.2 with an average difference of 0.2% of A1C. POC method has a sensitivity of 88.6% and specificity of 96.3% in diagnosing diabetes. We also found that only in 4% of cases medical management was changed based on the difference between POC and laboratory values. In conclusion, the difference in A1C measurements between POC and laboratory methods is arguably insignificant in making clinical decisions during routine outpatient management of diabetes.

ANA-NEGATIVE RENAL-LIMITED LUPUS NEPHRITIS IS ASSOCIATED WITH POOR RENAL OUTCOMES

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**Background:** In 2012, the Systemic Lupus International Collaborating Clinics proposed biopsy-proven lupus nephritis (LN) plus positive ANA or ds-DNA are sufficient for a diagnosis of SLE. We hypothesize that this definition is too narrow, and renal-limited lupus may occur in serologically negative patients. ANA-negative lupus (ANL) was first described in 1976 by Koller in 5 patients with clinical SLE. No one has contrasted the renal outcomes in these patients with those of conventional seropositive LN patients, and it remains unclear whether these two groups are clinically distinct. To this end, we compare the entity of ANL and conventional LN with regards to pathology and renal outcome.

**Design:** This retrospective cohort study compares renal outcomes in 13 ANL patients vs. 44 control ANA-positive lupus (APL) patients. The definition of LN includes 'full house' staining and/or EM findings of LN. Other inclusionary criteria are ANA negativity and at least 6 months of follow-up. Poor renal outcome is defined as doubling of creatinine, progression to dialysis or renal transplantation. Exclusionary criteria include positive serology for HIV, Hepatitis B or Hepatitis C. Control cases are time-matched with identical IF and EM criteria, but are ANA-positive.

**Results:** Seven ANL patients were ANA and ds-DNA negative. Eleven ANL patients had 'full house' staining, none had tubular deposits, 6 had subepithelial, subendothelial and mesangial deposits, and none had tubuloreticular inclusions. In the study group, three patients (23 percent; 95% confidence interval 0.001 to 0.459) had poor renal outcomes vs. twelve patients (27 percent; 95% confidence interval 0.131 to 0.401) in the control group, creating a relative risk of 0.85 (two-tailed z-test  $p = 0.67$ ). No significant difference in renal outcomes was seen between these two groups.

**Conclusion:** ANA is an imperfect screening test for renal-limited lupus, as some patients with biopsy proven LN have negative ANA and ds-DNA serology. Further, the renal outcomes in ANL are equivalent to those in conventional LN, supporting lupus-specific treatment of renal-limited lupus. Pathologists should therefore have a lower threshold for diagnosing LN in patients with limited serology. Additionally, clinicians may misdiagnose renal-limited lupus by adhering strictly to the ACR or SLICC criteria. These criteria require revision to improve recognition of ANL patients.

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SAFETY AND EFFICACY OF FECAL MICROBIOTA TRANSPLANTATION IN SEVERE AND COMPLICATED CLOSTRIDIUM DIFFICILE INFECTIONS

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**Background:** CLOSTRIDIUM DIFFICILE infection (CDI) is a leading cause of healthcare-associated infections with a mortality rate of 10%. Surgery is the standard treatment for fulminant CDI but is associated with high mortality rates, ranging from 25% to 80%. Fecal microbiota transplantation (FMT) is a less invasive, understudied treatment modality shown to successfully treat recurrent CDI. However, limited data exist on the effect of FMT in severe and complicated CDI. The aim of this study is to evaluate the efficacy and safety of FMT in this population.

**Methods:** A retrospective study was conducted of all hospitalized patients with severe and complicated CDI who received FMT at the University of Virginia between July 2014 and July 2015. Pre-FMT and post-FMT data were collected. Study outcomes included adverse effects associated with FMT, improvement/resolution in symptoms after FMT, improvement of pseudomembranes, CDI recurrence, mortality, primary cure (no spontaneous CDI recurrence), secondary cure (no CDI recurrence after repeat FMT or CDI antibiotics), total length of stay (LOS), and intensive care unit (ICU) stay.

**Results:** A total of six patients with severe and complicated CDI underwent FMT (mean WBC 21.4 k/uL, 83% ICU care, 50% ileus, 67% vasopressor support, mean Charlson comorbidity index 4). All patients had bowel wall thickening by computed tomography scan. One FMT was performed in 17% of patients, two FMTs in 33%, three FMTs in 33%, and four FMTs in 17% (following a protocol, similar to that described by Fischer 2015, to determine the need to repeat FMT). The four patients who received FMT via colonoscopy/sigmoidoscopy had pseudomembranes, which improved with each subsequent FMT. All patients had immediate improvement of symptoms following the first FMT (abdominal pain improved in 80% and resolved in 20%; diarrhea improved in 50% and resolved in 17%). After second FMT, all symptoms resolved except for chronic diarrhea in one patient. CDI recurred in three patients (50%), but one recurrence was antibiotic-associated; all three resolved after repeat FMT or CDI antibiotic treatment. There were no adverse effects or deaths associated with FMT. Mean LOS was 22 days, mean LOS after FMT was 10 days, and mean ICU stay was 6 days (all prior to FMT).

**Conclusion:** FMT was efficacious and safe in this cohort of patients with severe and complicated CDI as shown by improvement/resolution of symptoms with each subsequent FMT, no subsequent surgeries or ICU care, no re-hospitalizations for recurrent CDI, and no associated adverse effects or deaths. Primary cure was 67%, and secondary cure was 100%. Further evaluation of the role of FMT in severe and complicated CDI is indicated.

## CHANGE IN LEFT VENTRICULAR EJECTION FRACTION IN HEART FAILURE PATIENTS DISCHARGED WITH WEARABLE EXTERNAL DEFIBRILLATOR

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**Background:** Some patients with newly diagnosed heart failure (HF) with reduced ejection fraction (HFrEF) have improvement in left ventricular ejection fraction (LVEF) with the guideline recommended medical therapy and thus do not require implantable cardioverter defibrillator (ICD) during the follow up. The factors associated with increase in LVEF in HFrEF patients have been not well defined. We conducted a retrospective analysis of HFrEF patients with LVEF<35% who were discharged from Charleston Area Medical Center (CAMC) with a Wearable External Defibrillator (WCD) and analyzed the trend and factors associated with the change in LVEF.

**Methods and results:** 120 patients with HFrEF (Mean±SD, NYHA class 2.4±0.6, LVEF 23.7±7.4%) who were discharged from CAMC hospitals with the WCD for prevention of sudden cardiac death were included in the study. The average age was 62±11 yrs. The patients were mostly overweight (BMI 29.4±7.3 kg/m<sup>2</sup>) Caucasian (92%) and there was predominance of male (62.5%) population. Follow up echocardiogram completed after 86±26 days showed 7.4±11.3% increase in LVEF from the initial ones but there was minimal increase (1.3± 9.9%) in LVEF during the extended follow up period of two year (398±220 days). When we divided the patients into three groups based on their 90 day follow up LVEF (<35%, 35-50%, >50%), we observed that 30% of patients had improvement in LVEF to >35% and thus did not meet the criteria for ICD implantation and 10% patients had LVEF normalization (>50%). Upon stratification of patients based on change in LVEF (LVEF decrease or no change vs <50% increase vs > 50% increase), we found significant correlation of LVEF increase with fewer coronary revascularizations, atrial fibrillation, higher systolic blood pressure and non-smoking status. Despite having similar initial LVEF in three groups, the change in LVEF was not associated with initial LVEF, ischemic etiology, NYHA class, renal function, serum sodium or guideline recommended HF medications.

**Conclusions:** In this comprehensively characterized cohort of HFrEF patients, we observed that one third of patients had LVEF improvement to > 35% during the 90 day follow up period. Fewer coronary revascularizations, higher systolic blood pressure and non-smoking status were associated with increase in LVEF during 90 day follow up period. There was no correlation between change in LVEF with the initial LVEF, and no further increase in LVEF after the initial 90 day period during the course of two year follow up.

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REDUCING READMISSION RATES IN ACUTE PANCREATITIS THROUGH PATIENT EDUCATION AND RISK ASSESSMENT

Jordan T. Vulcano, DO, Anthony Caceres, MD

**BACKGROUND:** Early hospital readmissions are a direct burden on both our patients' wellbeing and healthcare system as a whole. Acute pancreatitis is a top offender in this category, with countless 30-day readmissions. Studies have showed a consistently higher than average 30-day readmission rates in acute pancreatitis, around 19%. This is significantly higher than the average all-cause readmission rate at Aurora hospitals. This quality improvement project/study aims to reduce the rate of acute pancreatitis 30-day readmission rates at several Milwaukee Aurora hospitals through patient education and use of a readmission risk assessment tool.

**METHOD:** Project was conducted out of Aurora Sinai, St. Luke's and West Allis Hospitals with a total of 18 patients admitted with acute pancreatitis admitted predominantly to the Internal Medicine Teaching Service (IMTS) between 2/2014-10/2014. Patients were seen within 1-2 days of admission and provided one-on-one education with handout on acute pancreatitis. In addition, a 30-day Pancreatitis Readmission Predictor (PRP) score was used to classify patient as low (5%), moderate (17%) or high (68%) risk for readmission via Epic "Dot Phrase." Subsequent readmissions, 14 day follow-up, total hospitalizations and ED visits were tracked through present. This was compared to readmission rates of a randomly selected control group of 18 patients admitted with acute pancreatitis.

**RESULTS:** Patients had a PRP score ranging from 0-4, with an average of 1 (rounded from 0.78). Of the 18 patients in the study group, only 2 were readmitted within 30 days for pancreatitis or 11.1%. The control group had 3 readmissions within 30 days or 16.7%. Patients with alcohol related pancreatitis were more likely to have a higher PRP (1.0) and readmission rate at 20% (2 out of 10).

**CONCLUSION:** A diagnosis of acute pancreatitis places the patient at a significantly higher than average risk of readmission. This quality improvement project, while small in size and scope, was able to reduce readmission rates from 16.7% to 11.1% through simple patient education and identifying those patients ready for safe discharge through a 30-day readmission risk assessment tool. Readmissions are detrimental to both the patient and healthcare system. This project serves as a starting point for reducing readmissions not only in acute pancreatitis patients but potentially other diagnosis-specific readmission initiatives.



## MERCURY POISONING AS A MIMICKER OF PHEOCHROMOCYTOMA

Meierhenry, Erin, MD; Kuo, Alan, MD; Amin, Janki, MD; El-Farra, Neveen, MD; Ptaszny, Magdalena, MD. University of California, Los Angeles

A 46-year-old man with recently-diagnosed hypertension presented with fatigue, myalgias, weakness, anxiety, diaphoresis and subjective fevers. He reported intermittent use of Chinese herbal medicine over years with increased consumption in the weeks prior to admission. The patient was consuming a heavy amount of alcohol for years but quit at the onset of his symptoms, approximately 3 weeks prior to admission.

On presentation, vital signs showed elevated blood pressure up to 218/100 and heart rate up to 100. His exam was significant for tremulousness, profuse diaphoresis and salivation. Neurologic exam was notable for poor articulation of speech, confusion, 4/5 strength in upper and lower extremities bilaterally, unsteady gait, and fine tremor punctuated by coarse shaking in bilateral upper extremities.

Complete blood count and chemistry were within normal limits. Urinalysis was notable for 3+ proteinuria. Neurologic work up including lumbar puncture, electromyography (EMG) of upper and lower extremities and brain imaging were unremarkable. A cortisol stimulation test was normal. The patient was treated empirically for alcohol withdrawal with minimal improvement. A diagnosis of pheochromocytoma was considered, however, serum metanephrines and urine catecholamines were only mildly elevated and his symptoms were constant. In addition, CT of abdomen and pelvis showed no adrenal masses. Toxicologic work up revealed an elevated blood mercury level twice the upper limit of normal and urine mercury level 25 times the upper limit of normal.

He was started on chelation therapy with penicillamine and had gradual improvement in his symptoms and blood pressure. Though his hyperadrenergic symptoms improved, he continued to have sequelae from mercury toxicity, including neuropathy, autonomic dysfunction, generalized weakness, and urinary retention.

The most common causes of hyperadrenergic state include sympathomimetic drugs (ex: amphetamines, cocaine, epinephrine, monoamine oxidase (MAO) inhibitors with tyramine-containing foods), panic disorder, and pheochromocytoma. Mercury poisoning is a rare but important mimicker of pheochromocytoma, and should be considered in patients presenting with sympathetic overdrive in the right clinical context. By inactivating catechol-*O*-methyltransferases (COMT), mercury inhibits the breakdown of catecholamines, resulting in manifestations such as tremulousness, hypertension, and diaphoresis. Other common symptoms of mercury poisoning include weakness, fatigue, paresthesias, excess salivation, gait unsteadiness, and cognitive/emotional difficulties, all of which were present in our patient.

Chinese herbal medications have often been associated with high levels of heavy metals, including mercury. Studies estimate that less than 50% of patients disclose use of herbal medications or supplements. This case illustrates the importance of obtaining a thorough history of all medication and substance use, including complementary/alternative medications.

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DEVELOPMENT OF AUTOIMMUNE DIABETES FOLLOWING THERAPY WITH PEMBROLIZUMAB FOR METASTATIC MELANOMA

First Author: Manisha Chandar, DO Second Author: Bruce Brockstein, MD

*Background:* The immune checkpoint inhibitor pembrolizumab is a monoclonal antibody directed towards the programmed cell death (PD)-1 receptor, which normally serves as a co-inhibitory modulator of T-lymphocyte activity. In the treatment of advanced melanoma, pembrolizumab has been shown to prolong progression-free survival and have lower rates of high-grade toxicity than ipilimumab. However, similar to other immune checkpoint inhibitors, increased T cell activity resulting from pembrolizumab therapy may put patients at risk for immune-related adverse events (irAEs).

*Case Report:* A 41-year old female with history only of metastatic ocular melanoma presented for pembrolizumab therapy. She had previously received proton beam radiotherapy to the liver, five cycles of doxorubicin drug-eluting beads (DEBDOX), and two cycles of ipilimumab, which had been discontinued due to the development of gastrointestinal intolerance. Approximately one week following her second dose of pembrolizumab, she presented to the Emergency Department with dyspnea, polyuria, polydipsia, and decreased appetite. She was found to be in diabetic ketoacidosis, without any known history of diabetes (glucose 530, pH 6.92, and HCO<sub>3</sub> of 7 mEq/L). Further labwork demonstrated a β-hydroxybutyrate level of 8.1 mmol/L, C-peptide level <0.1 ng/ml and significantly elevated anti-glutamic acid decarboxylase (anti-GAD) antibodies, supporting an autoimmune etiology of her presentation.

*Discussion:* It is known that immune checkpoint inhibitors such as pembrolizumab may put individuals at risk for irAEs, however the development of new-onset diabetes as a result of therapy had not been recognized previously as a known side effect of pembrolizumab. This case suggests that the development of autoimmune diabetes and diabetic ketoacidosis may be a rare but significant adverse event mediated by pembrolizumab and other PD-1 inhibitors.

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BURKITT LYMPHOMA MIMICKING AS TOLOSA HUNT SYNDROME

First Author: Chan Yeu Pu, MD; Kumar Rohit, MD; Neil Bhalerao, MD

**Introduction:** Tolosa-Hunt (TH) syndrome is a rare clinical syndrome, characterized by painful ophthalmoplegia with annual incidence of about one case per million. It is caused by an idiopathic granulomatous inflammation of the cavernous sinus or orbital apex complex and is extremely responsive to steroids. TH syndrome is a diagnosis of exclusion and extensive work up is recommended to prevent misdiagnosis. We present a case of case of Burkitt lymphoma mimicking TH syndrome.

**Case:** A 48-year-old male presented with bilateral ptosis and headache for 10 days associated with bilateral eye pain and drooping of his eyelids. He had fever with chills, night sweat and unintentional weight loss of 20lbs in 3weeks. His vitals were within normal limits. Cranial nerve examinations revealed normal visual acuity, bilateral fixed nonreactive dilated pupils and bilateral extraocular muscle palsy. Cranial nerves V, VII-XII, motor, sensory and cerebellar examination was normal. There was no palpable lymphadenopathy. Significant laboratory findings included platelet 44k/uL, hemoglobin 11.4g/dL, white cell count(WBC) 3.8l/uL, c-reactive protein 35.6mg/dL and lactate dehydrogenase 4400U/L. MRI brain revealed abnormal tissue within sella and parasellar with bilateral cavernous sinus extension. On Lumbar puncture cerebrospinal fluid was clear with protein 79, glucose 72, WBC 2uL, negative VDRL, varicella zoster and herpes simplex DNA serology. Endoscopic sphenoid sinusotomy, biopsy and culture were done. Pathology showed high-grade B-cell lymphoma, immunohistochemical stains were positive for CD10, CD20BCI-6, EBER (Ebstein-Barr Virus in-situ) and MUM-1. CT neck, chest, abdomen and pelvis found axillary and external iliac lymphadenopathy. He was started on R-CODOX-M/IVAC chemotherapy and significant neurological improvement was documented on follow up.

**Discussion:** Tolosa-Hunt syndrome is caused by an inflammatory process of unknown etiology. The compression due to the inflammation causes secondary dysfunction of structures within cavernous sinus especially CN III, IV and VI. TH syndrome typically has unilateral presentation but 4-5% cases have bilateral involvement. TH syndrome has vast differentials including neoplasm, vascular abnormalities, autoimmune or infectious. Extensive work up not limited to imaging, lumbar puncture, biopsy, autoantibodies, cultures and PCRs are recommended before diagnosis of TH syndrome is made. Lymphoma should be considered especially if patient has B-symptoms, elevated LDH or evidence of lymphadenopathy. Initiation of steroid therapy with presumptive diagnosis TH syndrome without excluding lymphoma can adversely affect tissue diagnosis leading to treatment delays. It is recommended that these drugs should be withheld until tissue is obtained for diagnosis.

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CASE OF NOD2-ASSOCIATED AUTOINFLAMMATORY DISEASE SUCCESSFULLY TREATED WITH SULFASALAZINE

First Author: Maya Estephan, MD Additional authors: Qingping Yao MD, Jason Springer MD

Autoinflammatory diseases (AIDs) are characterized by unprovoked episodes of inflammation, without high titer autoantibodies or antigen specific T cells. Several monogenic AIDs have been described in the adult population, such as Familial Mediterranean Fever (FMF), Tumor necrosis factor receptor-associated periodic syndrome (TRAPS), and Blau syndrome. In 2011, Yao et al described a new disease entity consisting of periodic fevers, skin disease, inflammatory arthritis, serositis, sicca-like symptoms and elevated acute phase reactants in seven patients, and this disease has been designated NOD2 associated autoinflammatory disease (NAID). NAID is associated with nucleotide-binding oligomerization domain containing 2 (NOD2) gene mutations, with the IVS8+158 NOD2 sequence variant detected in all patients.

A 23-year-old Caucasian male was referred to our Rheumatology Clinic in August 2013 for polyarthralgia and recurrent fevers. He reported a 6 month history of flu-like symptoms associated with recurrent fevers as high as 102.3 Fahrenheit for 2 weeks of duration, mainly occurring at nighttime. He had a negative influenza screen at that time. In March 2013, he noted a painful swollen lymph node behind his left ear, which was treated with a course of azithromycin without improvement. A pan Computer Tomography scan of the neck, chest, abdomen and pelvis did not reveal any other lymphadenopathy. The infectious work up was negative, including blood cultures, Human Immunodeficiency Virus, Cytomegalovirus, bartonella, tularemia, tuberculosis and acute Epstein Barr Virus infection. A biopsy of the auricular lymph node showed mild diffuse hyperplasia with central sclerosis without malignancy. Treatment with colchicine was attempted but without improvement. Genetic testing for TRAPS and FMF was negative, however it was positive and heterozygous for IVS8+158C>T and R702W. The patient was diagnosed with NAID and was started on sulfasalazine 1000 mg twice daily with resolution of his symptoms within 2 months. He remained afebrile at 6 months follow-up.

The clinical phenotype of our patient includes recurrent fever, polyarthralgia, flu-like symptoms, and elevated acute phase reactant. His clinical phenotype and genotype were consistent with NAID. NOD2 has several potential functions, including mediating responsiveness to the bacterial cell wall component muramyl dipeptide, activating nuclear factor kappa B (NFkB), and regulating apoptosis. To our knowledge, this is the first case of NAID described outside the initial cohort at Cleveland Clinic and the first case successfully treated with Sulfasalazine. Sulfasalazine prevents the stimulation of T cells and activation of NFkB in maturing dendritic cells. It also inhibits chemotaxis of neutrophils, reduces superoxide and proteolytic enzyme production and reduces angiogenesis. These are all possible mechanisms through which sulfasalazine regulates the immune response in NAID.

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THE SECRET COST OF BEAUTY

First Author: Inga Harbuz-Miller, MD

Hypercalcemia is commonly encountered on routine blood work or in symptomatic patients. The most common causes of hypercalcemia are primary hyperparathyroidism and malignancy, leaving about 10% to other etiologies. We present a rare cause of hypercalcemia.

A 55-year-old woman with no significant medical history was referred to the endocrinology office for asymptomatic hypercalcemia. She denied taking any vitamin or mineral supplements of any kind. A complete physical examination was unrevealing. Serum chemistry results revealed calcium 10.8mg/dL, which was without much change from prior levels “for years” per the patient. Additional workup revealed PTH 24pg/ml (15-65), 25-VitaminD (calcidiol) 36.9ng/ml (32-100) and an elevated 1-25 Vitamin D (calcitriol) of 106.8pg/ml (10-75). Thyroid function tests, CBC, liver and renal function were normal. Additional workup excluded granulomatous disease and lymphoma. Upon further questioning, the patient disclosed having had silicone breast implants placed in 1989, with ensuing hypercalcemia starting several years later. A mammogram revealed increased irregular breast density consistent with free silicone. A breast MRI confirmed bilateral sub-glandular silicone breast implants with intra- and extra-capsular rupture without evidence of malignancy. Owing to the planned removal of silicone and implants, and the mild degree of hypercalcemia in an asymptomatic patient, treatment with steroids was not initiated.

Silicone-induced hypercalcemia due to granuloma formation has been cited in the literature since 1964 and has a similar mechanism as calcitriol-induced hypercalcemia in granulomatous diseases like sarcoidosis or malignancy like lymphoma. In addition to breast implants, silicone injections for a variety of procedures are growing in popularity, and include sites such as the face, buttocks, and penis. With the increase in silicone of varying grades of purity being injected worldwide, the suspicion of silicone granulomatous hypercalcemia after body augmentation should be high.

## A RARE ASSOCIATION OF HYPOPITUITARISM WITH POLYCYTHEMIA AND CARDIAC MANIFESTATIONS

First Author: Gargi Patel, MD Ryan Wilson MD, Allegra Lee MD, Lissette Cespedes MD Dana Herrigel MD,

**Introduction:** Hypopituitarism carries an annual incidence of 2.1 – 4.2 per 100,000 persons. Approximately 25% of presentations are idiopathic. Hypopituitarism rarely presents with hematologic complications, and is most often associated with anemia. We present an unusual case of panhypopituitarism associated with polycythemia and profound electrocardiographic changes.

**Case Report** A 28-year-old woman with no prior history presented with acute confusion and agitation, and found to be hypoglycemic (blood glucose 37mg/dl.) She reported a recent 40lb weight loss, fatigue, and amenorrhea for 6 months prior to presentation. She denied prior pregnancies, hirsutism, galactorrhea, headache, or visual field disturbances. On admission, vital signs were normal, with a resting heart rate of 85. Laboratory data showed Hb 22.7 g/dL, hematocrit 67.3%, platelet count 82 thousand/ul, white blood cell count of 6.7 thousand/ul with normal differential. Further evaluation revealed cortisol level <1mcg/dL, with inappropriate response to cosyntropin. In addition, Somatomedin C, ACTH, Prolactin, FSH, LH, T3, T4, and TSH were all inappropriately low. MRI of the pituitary revealed a gland on the lower limits of normal in size but without evidence of pituitary adenoma, hemorrhage, infiltration, or infarction.

Our patient developed sinus bradycardia to 36 bpm, QTc prolongation to 580 ms, and anterior precordial T wave inversions at the onset of high dose steroid treatment. This coincided with a drop in T3, likely due to consumption of triiodothyronine in the setting of supraphysiologic steroids. With additional thyroid hormone supplementation and reduction to physiologic corticosteroids, EKG changes completely resolved within 2 weeks.

Erythropoietin level was undetectable, and CT of the abdomen and pelvis found no renal or adrenal mass. Bone marrow biopsy revealed possible early signs of fibrosis; JAK2 mutations were negative. At 2 month follow up, polycythemia had improved.

**Discussion:** The underlying cause of our patient's hypopituitarism is unclear. It is possible that primary polycythemia induced transient hyperviscosity may have led to a remote infarct, given the inappropriately small size of the pituitary on MRI. Low Erythropoietin levels support this hypothesis along with bone marrow biopsy showing mild fibrosis despite negative JAK2 and Exon 12 mutations. A second explanation is the hormonal deficiencies inducing polycythemia. Some literature describes hormonal interaction with bone marrow function, typically manifesting as anemia, not polycythemia. This seems more likely explanation given that with hormonal treatment there appears to be an improvement of hematologic abnormalities.

Moreover, electrocardiographic changes resolved with appropriate treatment. Case reports have linked both adrenal insufficiency and hypothyroidism to EKG changes, which can further be supported by our case. Hypopituitarism rarely causes hematologic abnormalities, and to our knowledge no prior cases have described hypopituitarism with polycythemia. This case is unique for its multi-system effects driven by hypopituitarism, which resolved following treatment.

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ANTI-NMDA RECEPTOR ENCEPHALITIS CLINICALLY PRESENTING AS PSYCHOSIS

First Author: Christian L Castaneda, MD Second Author: Radhika Vachhani, MD

**Introduction:** The clinical presentation of anti-NMDA receptor encephalitis presents a challenge to physicians. Disorganized thought process, delusions, and hallucinations are diagnostic criteria for many prevalent psychiatric disorders. As a result, these patients are often misdiagnosed. Awareness of this disease has been growing rapidly. In this case report, we describe a patient with NMDA receptor encephalitis that had an improvement in behavioral symptoms after a multimodal treatment regimen. We discuss the importance in considering this diagnosis due to its high morbidity and increasing incidence.

**Case Study:** A 26 year-old man was admitted to the hospital with one episode of tonic clonic seizure and acute onset of confusion, long-term memory loss, and paranoid delusions. Head CT, MRI, and laboratory tests for altered mental status were found to be normal. Patient denied recreational drug use and urinary toxicology was negative. Video EEG revealed right temporal slowing and he was given a diagnosis of right temporal lobe epilepsy. He was discharged, and 10 days later readmitted for confusion and physical aggression. He seized on day 10, was intubated, and started on midazolam and levetiracetam. A lumbar puncture was performed which was unrevealing. The possibility of autoimmune encephalitis was considered and he was started on a course of steroids. Repeat lumbar punctures were ordered with paraneoplastic panel. A course of plasmapheresis was initiated for the presumptive diagnosis. On his twenty-seventh day of admission, serology revealed autoantibodies toward NMDA receptors in both the serum and the CSF. Patient received full body CT, PET scan, and testicular ultrasound to identify any underlying neoplasms; no malignancies were found. A five-day course of intravenous immunoglobulin and chemotherapy was initiated. After 2.5 months at the hospital, the patient began to show signs of improvement and was eventually discharged to a rehabilitation center.

**Discussion:** The majority of anti-NMDAR encephalitis cases are reported in young females, median age between 18.5-23 years old, with an underlying ovarian teratoma. Given the severity and high morbidity caused by the disease, early diagnosis and treatment is fundamental. As patients often present with acute psychiatric symptoms including personality changes, hallucinations, and persecutory delusions, a medical emergency may not be suspected until later in the course of the disease. Particularly, a young female presenting with acute psychosis should be monitored closely for signs of autonomic instability or seizure activity. Prompt diagnosis and treatment may save her life or prevent permanent cognitive impairments. Treatment includes tumor resection and immunotherapy including plasma exchange, IVIG, and corticosteroids. With the increasing number of reported cases of anti-NMDAR encephalitis, the possibility of this diagnosis should have greater significance during diagnostic work-up of a patient with encephalitis-like symptoms.

A RARE CASE OF ADULT T- CELL LEUKEMIA/ LYMPHOMA, DIAGNOSIS AND TREATMENT WITH ANTIVIRAL THERAPY

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Adult T-cell leukemia/lymphoma (ATLL) is a rare and aggressive T-cell lymphoma, linked to infection by human T-cell lymphotropic virus - 1 (HTLV-1). The incidence of ATLL in the United States is approximately 0.05 cases per 100,000 people. A higher incidence of the malignancy is documented in areas endemic for HTLV-1 such as Japan, Caribbean, West Africa, South and Central America and Southeastern United States. HTLV-1 is spread via sexual contact, blood contact or from mother to child via breastfeeding. There is a slight male predominance overall and median age of diagnosis is in the sixth decade. Here, we present a case of Adult T cell Leukemia/ Lymphoma in a patient of Caribbean origin.

Case presentation: A 59-year-old lady of Caribbean origin with congenital Kyphoscoliosis and history of sickle cell trait presented to her primary care doctor with a mass in her neck, generalized malaise and night sweats for a month. Laboratory tests showed leukocytosis with lymphocyte predominance (WBC – 40K /mm<sup>3</sup>; 80% - lymphocytes with smudge cells but no blasts), hemoglobin of 12.1 g/dl, hematocrit of 38.3%, platelet count of 211k/cu mm and elevated LDH of 451. She was immediately referred to our tertiary care center for further evaluation. On physical examination, she had a left upper cervical mass, upper border abutting the angle of the mandible, 3 x 3 cm in size, hard, non-tender, not fixed, minimally mobile. She had no axillary or inguinal lymphadenopathy. Bone marrow biopsy was done, flow cytometry of which revealed mature CD4 positive T cells. Differentials included ATLL versus T-cell prolymphocytic leukemia (T-PLL). In view of her Caribbean descent ATLL was suspected. Among multiple viral serologies sent for testing, HTLV-1 serology was positive. ATLL is classified according to the Shimoyama criteria into acute, lymphoma, chronic and smoldering categories, with the earlier two having aggressive course with poor prognosis. Our patient belonged to the category of chronic ATLL. Patient was started on antiviral therapy with Zidovudine and Interferon alfa 2b. She tolerated it well and was discharged home on this regimen.

Discussion: Human T-cell lymphotropic virus type 1 associated adult T-cell leukemia/lymphoma (ATLL) is an aggressive, chemotherapy-resistant malignancy. Antiviral therapy with Zidovudine with addition of Interferon alfa can be used as a first line regimen for acute, chronic, and smoldering types of ATLL. In chronic or smoldering ATLL, use of a combination of Zidovudine and Interferon alfa resulted in a 100% five-year survival rate. Acute, lymphoma, or unfavorable chronic type ATLL needs chemotherapy. Vigilance and a high index of clinical suspicion of malignancy by her primary physician in this case are commendable. Early referral to tertiary centers for definitive diagnosis and initiation of therapy are crucial for improving long term prognosis and survival.

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A RARE CASE OF ECCRINE ACROSPIROMA TRANSFORMING INTO METASTATIC ECCRINE SPIRADENOMA

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Eccrine acrospiroma is an infrequent sweat gland tumor that rarely undergoes malignant transformation. Eccrine acrospiroma is so uncommon that its prevalence and its rate of transformation into metastatic disease cannot be found in literature.

This is a case of 56 year old male electrician with no significant medical history who, in April of 2009, noticed a chronic callus on his left thumb that began bleeding. The lesion was treated as an infection until eventual excision. Pathology of this callus returned as an eccrine acrospiroma. The margins were negative, thus no further treatment was performed. In May of 2010, the patient presented with a 3 month history of an enlarging left axillary mass. The mass was surgically excised and pathology returned as a metastatic malignant eccrine spiradenoma. Chest computed tomography demonstrated disease confined to the left axillary region. He was referred to the James Cancer Center where he underwent radiation in July of 2010. In December 2010, the patient represented with two nodules involving the inner aspect of his left upper arm. Pathology of the excised nodules returned as in-transit metastatic disease. In January 2011, he noticed a recurrent nodule that involved his left thumb. Core biopsy revealed an aggressive digital papillary adenocarcinoma. Due to multiple recurrences, he was referred to Memorial Sloan-Kettering Cancer Center for further evaluation. He underwent a positron emission tomography scan and thumb magnetic resonance imaging. Both scans were negative for further metastasis. Per the recommendation of Memorial Sloan-Kettering Cancer Center, he underwent partial amputation of the left thumb and excision of in-transit metastatic lesions of the left upper arm. Over the last five years, he has undergone 10 additional surgeries to remove in-transit metastatic lesions.

This case is a presentation of not only a rare diagnosis, but highlights the need to aggressively treat eccrine acrospiroma upon diagnosis and the challenges associated with managing malignant transformation and metastasis.

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HEPATIC INFLAMMATORY PSEUDOTUMOR IN A PATIENT WITH PRIMARY SCLEROSING CHOLANGITIS  
MASQUERADING AS CHOLANGIOCARCINOMA

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**Introduction:** Inflammatory pseudotumor (IPT) of the liver is an infrequent but benign lesion that often mimics malignancy. We report a rare case of a large hepatic IPT masquerading as an intrahepatic cholangiocarcinoma in a patient with primary sclerosing cholangitis (PSC).

**Case description:** A 71 year old Caucasian male with a 8 year history of PSC, presented to the GI clinic for follow-up visit. He was asymptomatic, and except for mild non-tender hepatomegaly, his physical exam was unremarkable. Routine laboratory workup revealed abnormal liver chemistry including elevated alk. phos. (431 IU/L), ALT (129 IU/L), AST (111 IU/L), total bilirubin (2.9 mg/dL), direct bilirubin (1.3 mg/dL), CEA at (93.9 U/mL), and CRP of (12.4 mg/L). MRI of abdomen demonstrated a large well-circumscribed, bilobed mass measuring 14.3 x 7.1 x 9.4 cm in the caudate and right lobe of liver that was hyperintense on T1-weighted images and hypointense on T2 weighted images, a new finding that was highly suspicious for malignancy. Liver biopsy of the mass revealed extensive lymphoplasmacytic inflammatory infiltrate and significant fibrosis, but no evidence of malignancy. Follow-up MRI of abdomen 3 months later showed stable size & appearance of mass, and the diagnosis of hepatic IPT secondary to PSC was made.

**Discussion:** IPT is a benign lesion characterized by inflammatory infiltrates and areas of fibrosis that can be mistaken for malignancy. First described by Baker and Pack in 1953, only several hundred cases of hepatic IPT have documented in medical literature. The exact pathogenesis remains unknown, but it has been suggested that radiation, chemotherapy, and infectious etiologies may play a role in development. Inflammatory disorders may also play a role in development, as hepatic IPT often occur in the setting of chronic biliary disease like PSC and IgG4 sclerosing cholangitis. Even in previously reported IPT cases in patients without PSC, histology showed significant destructive sclerosing cholangitis mimicking PSC suggesting a relationship between both disorders. Hepatic IPT have been associated with nonspecific symptoms such as jaundice, abdominal pain, fever, and weight loss as well as nonspecific laboratory abnormalities including elevations in ESR, CRP, leukocyte count, bilirubin, AST, Alkaline phosphatase, anemia, and thrombocytopenia. While imaging modalities can identify IPT, hepatic IPT can be difficult to distinguish from malignancy on imaging studies alone; tumors markers can be useful to exclude malignancy but may also be elevated. In such circumstances, biopsy with histologic analysis may be warranted to help confirm diagnosis. Expectant management with follow-up imaging has been increasingly utilized as spontaneous regression and/or reduction of size have been reported.

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PHIMOSIS AND FEVER

Mohammed Raja, Amir Azarbal, Vineet Veitla

53-year old Caucasian male with history of Hypertension, Diabetes and Coronary artery disease, presents with 1 week of fever, nausea, vomiting, diarrhea, dyspnea and rash. 6 weeks ago had circumcision for phimosis then went jet skiing at a lake and developed infection of the surgical site. Initially improved with oral antibiotics, and symptoms began 1 week later at which point his condition deteriorated and was admitted to ICU. On presentation, patient was septic with Fever of 101.3 F, with diffuse mottling rash and bilateral conjunctival suffusion. Serum creatinine was 6.2 and chest x ray showed bilateral pulmonary infiltrates. Patient was started on emergent hemodialysis and was placed on broad spectrum anti-microbials with fluconazole, vancomycin, meropenem and azithromycin. Over the next few days, patient's condition initially worsened with rising total bilirubin, creatinine and increasing pulmonary infiltrates on chest x ray. Finally patient's condition gradually improved with decreasing total bilirubin as well as creatinine level. He was eventually taken off hemodialysis and was transferred out of the ICU to a regular room. Extensive work up including imaging and serological tests as well as cultures did not reveal a definite diagnosis. One lab send out however, returned positive for Leptospira antibody. Leptospirosis is a zoonosis caused by a spirochete. Incidence is ten times higher in tropical than temperate regions. Classically has had triathlon outbreaks in the past. Rodents are the most important reservoirs. Humans are usually infected through exposure to contaminated soil or water from infected rodent urine. Overall has a low incidence in the U.S with highest cases reported in Hawaii. Has a variable clinical course, mostly mild and self-limited but some cases may be fatal. Usually presents with sudden onset of fever, rigors, myalgias and other non-specific symptoms. Conjunctival suffusion, when present, is a highly specific sign, in up to fifty percent of cases. Most common complications include: liver failure, renal failure, and pulmonary hemorrhage with up to seventy percent mortality. Diagnosis is mainly based on a high index of suspicion with epidemiologic exposure and clinical manifestations. Serology and molecular tests are non-specific with high false positives. Mild cases can be treated as outpatient with oral Doxycycline or Azithromycin. For severe cases IV Penicillin or Doxycycline may be used. Prevention is based on mainly proper sanitation and avoiding food and contaminated water sources. Domestic and farm animal vaccination is also available.

## NOT YOUR USUAL ACUTE ABDOMEN

Krishna Venkata MD, Sumant Arora MD, Kannan Thanikachalam MD, Talha Nawaz MD, Jewell Halanych MD

**Introduction:** Appendicular adenocarcinomas are rare, but must be considered in the differential of acute abdominal pain.

**Case description:** A 47-year-old woman with no significant past medical history presented with 2-day history of severe diffuse lower abdominal/pelvic pain associated with fever, chills and anorexia without weight loss. Past surgical history included a caesarian section 17 years ago. Vital signs were within normal limits except tachycardia of 105/min. Physical examination revealed moderate abdominal distention with tenderness in lower abdominal quadrants bilaterally and involuntary guarding and rebound tenderness. Initial laboratory data showed WBC of 13,000/microliter with 82 % neutrophils, normal urinalysis and normal lipase and amylase levels. CT abdomen/pelvis with contrast revealed a 11cm right adnexal mass containing fat, calcifications, and air which abutted the thickened wall of the terminal ileum. Transvaginal pelvic ultrasound showed normal uterus, ovaries and a right adnexal mass with free fluid and air in pelvis suggestive of an inflammatory process. She was started empirically on intravenous cefazolin and metronidazole, and morphine for pain control.

The patient underwent exploratory laparotomy that revealed a large perforated appendicular tumor. An ileo-cecal resection with ileo-colonic anastomosis was performed with peritoneal wash for mucinous material and antibiotics were discontinued. Surgical biopsy confirmed the right adnexal mass to be mucinous adenocarcinoma of appendix with no lymph node involvement (T4,N0,M1). Oncology was consulted and given high likelihood of intraperitoneal metastasis, patient was started on chemotherapy with 5-Fluorouracil (5-FU), leucovorin, and oxaliplatin. Follow up CT abdomen/pelvis and nuclear PET scan at 1 year showed no mass or enlarged lymph nodes suggestive of residual or recurrent tumor.

**Discussion:** Primary tumors of appendix are rare, found in less than 1% of surgically removed appendices and account for 0.5-1% of all intestinal neoplasms. Major histological varieties include: carcinoid, adenocarcinoma, adenocarcinoids, cystadenomas and cystadenocarcinomas. Mucinous adenocarcinoma is the most common histological variant followed by intestinal type of adenocarcinoma. Appendicular adenocarcinomas usually present with clinical picture of acute appendicitis however, may also present as ascites, abdominal mass, or generalized abdominal pain. Cancer is found incidentally in surgery for other reasons in less than 20% cases. Standard treatment includes right colectomy while simple appendectomy can be considered only if the tumor is confined to the mucosa. Despite the lack of clear data regarding the role of adjuvant chemotherapy, many oncologists recommend 5-Fluorouracil (5-FU) based chemotherapy for node positive intestinal type adenocarcinomas. The 5-year disease specific survival rate for mucinous adenocarcinoma of appendix is 58%. Our case emphasizes importance of keeping an open mind while developing differential diagnoses in any case of acute abdomen.

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## RUNNING OUT OF SIGHT

Jessica August, MD Bujji Ainapurapu, MD Tirdad Zangeneh, MD Jennifer Huang, MD

Neuroretinitis is a rare condition involving the optic nerve that can be caused by a broad differential of infectious and inflammatory causes. This case represents a one-of-a-kind diagnosis that emphasizes the profound effect that a good history can have on narrowing the differential diagnosis by taking clues directly from the patient.

The patient is a 28 year old female nurse who presented with chief complaint of stabbing right eye pain associated with severe right sided temporal headache. She also reported central vision deficit when rising from a seated position in the affected eye. Review of symptoms was positive for three self-resolving episodes of fever with maximum temperature of 101F over the past two months associated with myalgia and headache that she attributed to viral syndrome. She also had recent travel to southern Washington state where she enjoyed running in the forested area that surrounds her parent's home. She denied joint pain, rashes, changes in hair distribution, and other symptoms suggestive of autoimmune cause. Upon initial presentation, her physical exam was notable only for right sided deep cervical lymphadenopathy. Laboratory studies were within normal limits. Neurology recommended trial of a Triptan for suspected migraine headache and referred her for MRI of the brain that showed chronic sinus disease of the sphenoid sinus and posterior ethmoid air cells. She was seen by ophthalmology who found evidence of peripapillary edema in the affected eye for which neuro-ophthalmology was consulted and made the final diagnosis of neuroretinitis without macular star. A broad differential of the possible infectious causes including viral, bacterial, fungal, and parasitic causes were tested using serology. IgG for *Borrelia hermsii* was found to be positive at 1:64, correlating with her recent travel and likely tick exposure while running in the forest of Washington. She was treated with a course of ceftriaxone and doxycycline and her symptoms completely resolved.

*Borrelia Hermsii* is the most common cause of tick-born relapsing fever in North America with the *Ornithodoros hermsi* tick as its vector. Our patient had been exposed to the wooded areas of the Pacific Northwest where this spirochete and its host commonly live. There have been case reports of uveitis due to *Borrelia hermsii* however, to our knowledge, this patient represents the first diagnosis of neuroretinitis. Her presentation highlights the importance of exploring the details of a patient's history to lead you toward the diagnosis.

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NOT ALL SLE FLARE ARE TREATED THE SAME

First Author: Amirali Kiyani, MD, and Konstantinos Parperis, MD

**Introduction:** Systemic lupus erythematosus (SLE) is a chronic autoimmune disorder with myriad of manifestations and is often called the great masquerader; however, rarely, there are other diseases than can mimic a SLE flare which requires a high index of suspicion to diagnose and treat.

**Case presentations:** Patient is a 41 year-old female from Middle east with history of SLE on high dose prednisone, hydroxychloroquine, and Mycophenolate, who presented to ER with fever, chills, nausea and abdominal pain for the past 2 days. Patient was initially diagnosed with SLE nearly 7 years ago manifested with malar rash, hair loss, arthralgia, Raynaud's, sclerodactyly, positive ANA / SSB / dsDNA, low c3, c4 and positive Scl-70. Patient had been hospitalized several times in the past 4 years with similar recurrent symptoms. She was hypotensive and tachycardic on previous admissions during which she was treated with broad spectrum antibiotics and high dose steroids for possible underlying infection causing a SLE flare. During these episodes elevated ESR and CRP. However, imaging and cultures, including CSF analysis, failed to identify a source of infection. She was most recently treated for clostridium difficile infection.

On arrival, T: 39.8, HR: 110, BP:130/79. Patient was started on IV antibiotics but all cultures came back negative, and despite high dose steroids patient was febrile. Rheumatology service was consulted and it was concluded that there might be a second concurrent process present such as periodic fever syndromes (likely familial Mediterranean fever). She was empirically started on colchicine and symptoms improved significantly within 48 hours. After discharge, she followed up with rheumatology clinic and was found to have a heterozygous mutation in MEFV gene. Thus, FMF was confirmed based on clinical criteria, genetic testing and response to colchicine. She has not experienced any attacks while on colchicine in the last year.

**Discussion:** Familial Mediterranean fever (FMF) also called recurrent polyserositis, often presents with brief recurrent episodes of peritonitis, pleuritis, and arthritis, usually with accompanying fever. Nonsense or missense mutations in the MEFV (Mediterranean fever) gene appear to cause the disease in many cases. MEFV produces a protein called pyrin. Its exact function is unknown, but Pyrin forms a part of the NLRP3 inflammasome complex, and dysfunctional pyrin causes increased interleukin 1 $\beta$  production through hyperactivated inflammasome. In a Turkish study with 2716 FMF patients, only 4 cases (0.1%) of SLE were identified. It is suggested that the rarity of SLE with FMF patients might be because of the high levels of C-reactive protein which mediates the removal of apoptotic cells. Colchicine is the drug of choice for acute attacks and prophylaxis. Major long term complication is secondary amyloidosis.

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CANCER, CHEMOTHERAPY AND NAUSEA: IS THE ALWAYS THE SAME OLD STORY?!

First Author: Amirali Kiyani, MD, Keng-Yu Chuang, MD

**Introduction:** Nausea and vomiting are common side effects of chemotherapy; however, high index of suspicion is needed to promptly diagnose rare but potentially life-threatening condition presenting with nausea as the only symptom.

**Case report:** A 54-year-old diabetic female with history of non-small cell lung cancer presented to the ED with nausea and vomiting for the last month, worse especially over the last few days. She was on cisplatin for chemotherapy. She also received *H. pylori* triple therapy recently. Her past surgical history is significant for left lower lobectomy with partial resection of the diaphragm and primary repair. She reported moderate epigastric, LUQ pain as well as bilious vomiting and diarrhea. She denied fever, chills, hematemesis, melena, recent travel or sick contacts. Physical exam was remarkable for epigastric and left chest wall tenderness. Lab results revealed severe hypokalemia and hypomagnesemia. CXR showed marked elevation of the left hemi-diaphragm as well as thick-walled air-filled structure overlying the left lower thorax, which may represent the stomach with an elevated left hemi-diaphragm. An esophagram was performed which showed the fundus of the stomach below the diaphragm and the antrum of the stomach above the diaphragm. The contrast could not empty into the duodenum after multiple positioning attempts. A CT scan confirmed mesenteroaxial volvulus of the stomach with gastric outlet obstruction. Patient was immediately taken for exploratory laparotomy. Reduction of gastric volvulus, repair of diaphragmatic defect, and left tube thoracotomy were performed. On postoperative day 5 the chest tube was removed. The patient tolerated diet and pain was well controlled. Patient continued on chemotherapy with no further complications

**Discussion:** Patients with acute gastric volvulus usually present with Borchardt triad of epigastric pain, retching without vomiting, and inability to place a nasogastric tube into the stomach. Gastric volvulus is classified as primary (idiopathic) or secondary depending upon its etiology, organoaxial or mesenteroaxial according to axis of rotation, and acute or chronic depending upon the clinical presentation. With mesenteroaxial volvulus, the antrum becomes displaced above the gastroesophageal junction. The rotation is usually partial (<180°) and is not generally associated with a secondary anatomic defect. Risk factors include: Age>50, diaphragmatic defects, gastroesophageal surgery, neuromuscular disorders, increased intra-abdominal pressure, conditions leading to diaphragmatic elevation (such as left lung resection). Mesenteroaxial volvulus could lead to severe strangulation and obstruction.

Acute gastric volvulus is a surgical emergency. The goals of treating patients with gastric volvulus are restoring the stomach back to a more normal anatomic position, repairing any associated anatomic abnormalities, and preventing future stomach rotation.

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LUPUS AND NON-VEGETATIVE MITRAL REGURGITATION

First Author: Shruti Mony, MD Ashbeel Samuel, MD , Gabriel Colceriu, MD

**INTRODUCTION:** Systemic lupus erythematosus (SLE) is an autoimmune disease exhibiting great diversity in presentation. Pericarditis and endocarditis are the most common cardiac manifestations secondary to SLE while isolated valvular disease is very rare. We present a unique case of a young male with SLE causing multi-organ dysfunction and severe mitral regurgitation.

**CASE DESCRIPTION:** A 34 year old Hispanic male with a past medical history of SLE, Antiphospholipid syndrome (APS) and stroke presented with symptoms of recurrent hemoptysis, shortness of breath and fatigue for 3 months. He had multiple outside hospital admissions for shortness of breath wherein he was found to have massive pulmonary hemorrhage and acute kidney injury requiring temporary hemodialysis. He also underwent a kidney biopsy which confirmed crescentic lupus nephritis. He failed therapy with high dose IV steroids, rituximab, cytoxan, and plasmapheresis. Due to his refractory status and respiratory decline he required intubation, following which he was transferred to the critical care unit at our hospital for further evaluation.

On admission, he was in acute distress and ventilator dependent. His laboratory data revealed hemoglobin/hematocrit - 6.6/19gm/dl, platelet count 50,000/uL, and creatinine 4.68 mg/dl (from 2.45 mg/dl 1 month ago). His chest X-ray was consistent with pulmonary hemorrhage. For further evaluation, he underwent a transthoracic and transesophageal echocardiogram which revealed severe mitral regurgitation and moderate pulmonary hypertension, no vegetations were noted. He was deemed a poor candidate for further cardiac procedures and was managed medically. In addition to daily high dose IV steroids, and hemodialysis he received mycophenolate mofetil. But his hospital course was further complicated with thrombocytopenic purpura(TTP) requiring a repeat cycle of plasmapheresis. Despite all aggressive measures patient failed to improve and he succumbed secondary to his illness.

**DISCUSSION:** Among the various cardiac manifestations of SLE, fibrinous pericarditis and verrucous endocarditis are the most recognized, but lesions of the myocardium and coronary vessels may also occur. Our case is unique as the patient was diagnosed with severe mitral regurgitation without any prior vegetation or associated cardiomyopathy. In reviewing literature, there has been only one case reported of SLE causing isolated mitral regurgitation. A high index of suspicion needs to be maintained for SLE and cardiac involvement, as early diagnosis may avoid devastating consequences.

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DUODENAL PLASMACYTOMA: A RARE MANIFESTATION OF MULTIPLE MYELOMA

Wedad Rahman, Dileep Atluri, Farshad Aduli

Plasmacytomas originate from a neoplastic proliferation of plasma cells. Primary cases are more common and occur as solitary plasmacytomas of the bone or as solitary extramedullary plasmacytomas. Secondary plasmacytomas are rare and present as extramedullary manifestations of malignancies such as Multiple Myeloma, where plasma cell proliferation is typically confined to medulla. We report a case of secondary plasmacytoma in a patient with high-risk Multiple Myeloma.

A 66-year-old male with a 5 year history of IgG lambda Multiple Myeloma who had previously undergone two autologous stem cell transplants presented with a one day history of intractable nausea and vomiting preceded by three weeks of diffuse abdominal pain . Admission labs were unremarkable except for mild anemia, thrombocytopenia and elevated creatinine. A Computed Tomography (CT) of the abdomen and pelvis with oral contrast suggested the presence of a proximal duodenal obstruction and subsequent Esophagogastroduodenoscopy (EGD) encountered an obstruction in the second part of the duodenum and could not pass beyond it. Multiple biopsies taken from the narrowing later revealed lambda light chain restriction with in situ hybridization and positive IHC staining for CD 138 suggesting the presence of a duodenal plasmacytoma in the setting of longstanding multiple myeloma.

Extramedullary spread of Multiple Myeloma can occur in up to 20% of advanced disease cases and confers a poorer prognosis as it typifies stroma-independent growth. Gastrointestinal plasmacytomas account for nearly 7% of all extramedullary plasmacytomas and can occur in every segment of the GI tract. Diagnosis of GI plasmacytoma hinges upon biopsy confirmation as the endoscopic appearance of plasmacytomas varies significantly. Current treatment guidelines recommend high dose chemotherapy followed by autologous stem cell transplantation although immunomodulatory drugs such as Lenalidomide with Bortezomib and dexamethasone has also shown rapid cytoreduction and survival advantage in some studies. Localized radiation or surgery should be reserved for complications such as GI obstruction or bleeding. Extramedullary myeloma of the GI tract remains a challenge to treat due to the lack of prospective studies and aggressive nature of disease

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WOUND BOTULISM IN THE SETTING OF IV HEROIN USE

First Author: Andrew Frank Bernard, DO Gurpreet Sihota, DO Morteza Chitsazan, DO

Wound botulism is a rare form of botulism characterized by descending flaccid paralysis as a result of neurotoxins produced by *Clostridium botulinum*. Per the CDC, the incidence of wound botulism has increased due to the increase in use of black-tar heroin, especially in California.

A 51 year old male presented to the ED for progressive weakness in all extremities, dysphagia, slurred speech and bilateral ptosis, as well as progressive shortness of breath on exertion. Patient has history of IV heroin use, with last use prior to arrival. Given his symptoms, he had a stroke workup performed. Echocardiogram, carotid duplex, CT head were all negative. On exam, he was noted to have multiple injection marks on bilateral legs and buttock area, with CT bilateral lower extremities showing possible abscess formations. Patient continued to have worsening respiratory decline, resulting in transfer to the ICU for respiratory failure requiring intubation. Neurology and Infectious Disease were consulted; both were concerned for wound botulism. As a result, the CDC was contacted, and botulinum antitoxin was sent and administered. Infectious workup, including hepatitis panel, HIV, and blood cultures were all negative. Gradually the patient started improving in strength and breathing and was successfully extubated. He continued to improve with physical therapy and was discharged home with outpatient follow up.

This case illustrates the increasing incidence of wound botulism in the setting of increasing IV heroin use in the past several years, especially in California as per the CDC. It is important to be cognizant of the risk despite its rare incidence in the U.S. as the presentation may mimic other common neuromuscular diseases, such as Guillain-Barre, myasthenia gravis, or Lambert-Eaton syndrome.

NOT JUST A LOOSE TOOTH: PARATHYROID ADENOMA PRESENTING AS FACIAL BROWN TUMORS

Katherine Chen, MD Jane Ma, MD Artin Krekory Ohanian, MD Nasser Mikhail, MD

**Introduction:** The two most common causes of hypercalcemia are hyperparathyroidism followed by malignancy. Clinical manifestations of hyperparathyroidism typically result from hypercalcemia, with features such as constipation, nephrolithiasis, polyuria, and neurologic disturbances. Persistently high parathyroid hormone levels can also cause skeletal symptoms including osteopenia, bone cysts, and focal lesions of resorption and fibroproliferation called Brown tumors.

**Case presentation:** This is a 45-year old female with no significant past medical history who presented with painful facial swelling. She first developed a painless mass over her right mandible two years prior to presentation. One month before admission, she developed painful swelling near the left maxilla associated with intermittent bleeding and a loose tooth. She also endorsed intermittent headaches, fevers, chills, fatigue, unintentional weight loss, constipation, and diffuse transient bone pain. Exam was remarkable for a firm 2-cm mass palpable over the right maxillary sinus, a tender 1-cm mass over the right superior alveolar ridge, and a tender 3-cm mass over the left maxilla with overlying gingival erosion and bleeding. Head CT revealed multiple mandibular and maxillary expansile, cystic-appearing lucent lesions. Serum biochemistry demonstrated marked hypercalcemia with a calcium level of 14.4 (reference range 8.9-10.3) as well as hypophosphatemia and elevated alkaline phosphatase. PTH returned markedly elevated at 1202 (reference range 15-65), prompting a neck ultrasound and 4D parathyroid CT scan, which demonstrated a 1.6-cm parathyroid mass. PTHrP later resulted as negative, and Vitamin D levels were normal. The patient's hypercalcemia was treated with aggressive intravenous fluids, calcitonin, and zoledronic acid. Interestingly, the patient never had neurological manifestations of hypercalcemia. Otolaryngology was consulted for urgent parathyroidectomy, and pathology confirmed the diagnosis of a right inferior parathyroid adenoma. Pathology from a biopsy of the left maxillary mass ultimately revealed numerous multinucleated giant cells with interstitial hemorrhage, consistent with Brown tumors secondary to hyperparathyroidism.

**Discussion:** In contemporary practice, Brown tumors are rarely seen because hyperparathyroidism is usually identified and treated on the basis of incidental, asymptomatic hypercalcemia. Longstanding untreated hyperparathyroidism can induce osteitis fibrosa cystica from osteoclast activation and calcium resorption. Brown tumors develop when focal areas of bone undergo increased resorption followed by fibrocellular proliferation and accumulation of multinucleated giant cells, often accompanied by small focal fractures and hemorrhage. This case depicts facial brown tumors an unusual presenting manifestation of parathyroid adenoma. Although uncommon in the era of rapid serum biochemistry assays, brown tumors should be considered in the differential for bony lesions in the setting of hypercalcemia.

## THE COUGH THAT WOULDN'T GO AWAY

First Author: Satya Das, MD Second Author: Ahmad Anshasi, MD

**INTRODUCTION:** Anaplastic Large Cell Lymphoma (ALCL) represents an uncommon subtype of Non-Hodgkin's Lymphoma (NHL). It makes up 3% of all NHL occurrences and 30% of NHL in pediatric patients (1, 2). Clinical presentation and prognosis is driven in large part by the presence or absence of the anaplastic lymphoma tyrosine kinase (ALK) gene with ALK positive patients carrying a significantly better prognosis (3). Endobronchial involvement is exceedingly rare with only nine cases reported in a prior literature review (4). We report, to our knowledge, the third documented case of ALCL-associated endobronchial polyposis in a patient older than 65 years of age (5, 6).

**CASE REPORT:** A 66 year old man presented with eight weeks of fever, night-sweats, cough and twenty pound weight loss. Multiple courses of antibiotics had not improved his symptoms. Physical exam was notable for bilateral anterior cervical lymphadenopathy and crackles at the right lung base. He was admitted to the hospital and started on empiric antibiotics for presumed community acquired pneumonia. Tuberculosis was simultaneously ruled out with three negative sputum AFB smears. He experienced daily fevers up to 103 degrees with a blossoming leukocytosis that peaked at 49,000 K/ $\mu$ L. CT chest showed diffuse bilateral mediastinal, axillary and supraclavicular lymphadenopathy and a right lower lobe infiltrate with bronchovascular thickening (Figure 1). CT abdomen revealed multiple mesenteric and retroperitoneal lymph node conglomerates. Biopsy from two cervical neck lymph nodes was obtained and he underwent bronchoscopy in the setting of bronchovascular thickening on chest CT and unremitting cough. Innumerable endobronchial polyps were visualized from the carina distally into right mainstem (Figure 2), bronchus intermedius (Figure 3), and lower lobe basilar segments; biopsies and bronchial washings were obtained. Pathology from both endobronchial and cervical lymph node biopsies confirmed ALK positive ALCL. PET CT demonstrated significant FDG avid uptake in the aforementioned lymph node conglomerates and the patient was initiated on cycle one of cyclophosphamide, hydroxydaunorubicin, oncovin, and prednisone (CHOP) given his rapidly proliferative disease. After completing cycle one, he was discharged home and continued on therapy as an outpatient. He developed a complete response after three cycles of CHOP and currently remains disease free after completing six cycles.

**DISCUSSION:** ALCL remains a rare disease entity within the NHL subgroup with ALK positive disease representing an even rarer entity in the non-pediatric (>18) population. Endobronchial involvement should be considered in patients presenting with clinical symptoms of dyspnea, chest pain, and persistent cough and radiographic findings of intra-luminal bronchial nodules or possible bronchovascular thickening. ALK positive ALCL patients generally have very robust responses to chemotherapy but in high risk patients who recur, targeted ALK inhibitors such as crizotinib may play an important role.

## RAPIDLY PROGRESSIVE NEUROLOGIC DECLINE AND PAPULAR RASH PRESENTING IN A PATIENT WITH LYMPHOMA

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**Introduction:** West Nile Virus (WNV) was first recognized in the United States in 1999, however WNV encephalitis has become endemic and is now the most common viral encephalitis in the United States. The recognition of neuroinvasive WNV by physicians is crucial due to the estimated 2-12% mortality rate and significant long-term morbidity.

**Case:** Mr. B is a 67 year-old male with past medical history significant for mantle cell lymphoma who presented with a papular rash followed by an acute onset of ascending bilateral lower extremity weakness. On the day of presentation, his family found him on the ground and unable to stand, with mild confusion. In the emergency department the patient presented with high fever to 103 degrees, intermittent disorientation, and bilateral lower extremity weakness without meningismus. Broad-spectrum antibiotics were initiated, yet over the course of three days the patient's Glasgow Coma Score deteriorated from 14 to 6. The patient had diffuse weakness, absent upper and lower extremity reflexes, aphasia and dysphagia, and an erythematous papular rash on the trunk, back and lower extremities. Lumbar puncture was initially notable for a neutrophilic cerebral spinal fluid (CSF) and negative gram stain. One week later, the CSF WNV IgM antibody returned positive. Once the diagnosis of neuroinvasive WNV was made, all antibiotics and antivirals were discontinued. The patient made significant neurologic progress over his one-month hospital course, regaining his ability to speak and follow complex commands. On discharge, however, there were persistent deficits from baseline, including significant lower extremity weakness.

**Comment:** This case demonstrates a classic and particularly profound example of neuroinvasive WNV. Our patient presented with fever, lower extremity weakness, and rash, which rapidly progressed to debilitating encephalopathy and diffuse flaccid paralysis. The neutrophilic CSF found in our patient is well described in neuroinvasive WNV, despite its viral etiology. Less than 1% of those infected with WNV develop neuroinvasive disease. The best-described risk factors for neuroinvasive disease are advanced age, malignancy, and organ transplantation, consistent with this case of an older patient with underlying lymphoma. Prognostication to patients and families becomes important once the diagnosis is made. Age (>50) is the most important predictor of lasting complications. In the Houston West Nile Cohort followed since 2002, 86% of patients and 43% of patients with WNV encephalitis had neurologic deficits 1-3 years and 8-11 years post-infection, respectively. Wide variability in the literature between individual studies makes prognostication challenging, but based on a recent review of all known WNV studies, persistent long-term sequelae are likely. The most frequently observed sequelae are muscle weakness, fatigue, myalgia, memory loss, depression and difficulty concentrating. Patients and families must be counseled that recovery will be slow and that patients may continue to make progress even past one year.

A MIND GONE VIRAL: AN UNUSUAL CAUSE OF ACUTE ENCEPHALOPATHY

Christine Haynes, MD, MPH, UCLA and Annie Zhang, MD, UCLA

A 26-year-old previously healthy male was brought in by paramedics after a seizure in the setting of a recent febrile illness. Seven days prior to admission he developed fevers, abdominal pain, nausea, vomiting, and diarrhea. The morning of admission he had a witnessed convulsive episode with loss of consciousness for about two minutes. He bit his lips, had no bowel or bladder incontinence, and was confused for 15 minutes afterwards. His medical history revealed a diagnosis of G6PD deficiency, an allergy to cefaclor, occasional Tylenol use, and no other medical problems. He was unemployed but had previously worked for a telephone company, had no recent travel, smoked five cigarettes daily, denied alcohol or drug use, and was sexually active with women.

On exam he was afebrile with normal vital signs. He was tremulous but in no acute distress, answering questions appropriately but oriented only to self. His mother noted unusual irritability and foul language. His sclera were mildly icteric. His cardiopulmonary exam was unremarkable. His abdomen was soft with mild hepatomegaly. His cranial nerves, strength, and sensation were intact. His gait was ataxic.

His initial labs showed leukopenia, normal hemoglobin and platelet levels, acute kidney injury, elevated aminotransaminases, hyperbilirubinemia, hyponatremia, a metabolic acidosis, and a markedly elevated creatinine kinase. Urine toxicology screen was negative. His lumbar puncture demonstrated less than one red blood cell, less than one white blood cell, and normal protein and glucose. MRI brain was unremarkable. EEG demonstrated nonspecific frontal intermittent rhythmic delta activity.

He was empirically started on vancomycin, aztreonam, and acyclovir, which were subsequently discontinued when his CSF bacterial cultures as well as HSV and VZV PCRs were negative, as was a meningoencephalitis panel. After 48 hours of IV fluid resuscitation and observation, his mental status normalized, as did his creatinine, hyponatremia, bicarbonate, and aminotransaminases. The working diagnosis was viral encephalitis of unclear etiology. However, after five days his HIV RNA PCR returned with 2.3 million copies, and he was diagnosed with acute HIV infection.

This case demonstrated a particularly dramatic acute retroviral syndrome affecting multiple organ systems, including atypical features of encephalitis, hepatitis, rhabdomyolysis, and acute renal insufficiency. It illustrates the myriad ways acute HIV infection can manifest. Given the persistently high number of HIV positive patients who remain undiagnosed, there is a need for ongoing awareness of potential symptoms of acute HIV infection. In one retrospective observational study, only 5 of 19 patients with acute HIV infection were correctly diagnosed,<sup>1</sup> another prospective trial of 290 patients with primary HIV infection found that 30% had atypical symptoms.<sup>2</sup> These further emphasize the importance of recognizing acute HIV infection, leading to testing and diagnosis in order to both reduce transmission and initiate prompt treatment.

<sup>1</sup> Schaker T, Collier AC, Hughes J, et al. Clinical and Epidemiologic Features of Primary HIV Infection. *Ann Intern Med.* 1996; 125(4): 257-264.

<sup>2</sup> Braun DD, Kouyos RD, Balmer B, et al. Frequency and Spectrum of Unexpected Clinical Manifestations of Primary HIV-1 Infection. *Clin Infect Dis.* 2015; 61(6):1013-1021.

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## WEATHERING THE DIAGNOSIS OF THYROID STORM

First Author: CPT Christian L Horn, MC USA (Associate) Second Author: LTC Patricia Short, MC USA (Fellow)

**Introduction:** Thyroid storm is a rare, life threatening condition with a variety of manifestations and clinical presentations. Early identification of this challenging diagnosis will decrease the time to appropriate treatment and help to prevent disease morbidity and mortality.

**Case:** A 26-year-old previously healthy G1P1 woman 13 months postpartum presented to the emergency department for a four week history of intermittent, sharp epigastric abdominal pain with no association with foods. Over the last week she complained of pruritis and new onset jaundice. Review of symptoms revealed a 25-pound unintentional weight loss over the course of the last three months, despite polyphagia. Upon presentation, she was afebrile, tachycardic to the 140's, alert and oriented answering questions appropriately. Physical exam revealed scleral icterus and multiple skin excoriations. Labs drawn in the emergency department were significant for undetectable thyroid stimulating hormone, free T4 that was too high to measure, total bilirubin of 4.2g/dL, direct bilirubin of 3.8g/dL, and elevated alkaline phosphatase, alanine aminotransferase, and aspartate aminotransferase. Right-upper-quadrant ultrasound revealed the presence of cholelithiasis with a gallbladder wall thickness of 3 mm, but no evidence of biliary ductal dilation. She was given intravenous propranolol, methimazole, and prednisone due to concern for possible thyroid storm. She subsequently underwent magnetic resonance cholangiopancreatography which revealed the presence of a 4 millimeter obstructing gallstone within the common bile duct, without any duct dilation. Given the presence of choledocholithiasis explaining the jaundice and abdominal pain, and the absence of any CNS alterations, the diagnosis of thyroid storm was revised to thyrotoxicosis complicated by choledocholithiasis. She underwent endoscopic retrograde cholangiopancreatography with sphincterotomy to alleviate the biliary obstruction. A few days later, she underwent laparoscopic cholecystectomy and had complete resolution of her liver function tests abnormalities. Thyroid ultrasound and thyroid stimulating immunoglobulins confirmed a diagnosis of Grave's disease.

**Discussion:** This case illustrates two important features in the diagnosis of thyroid storm. Upon presentation, this patient met Burch and Wartofsky's criteria for thyroid storm with tachycardia greater than 130, recent history of parturition, abdominal pain, and unexplained jaundice. With the discovery of choledocholithiasis, the jaundice and abdominal pain was readily explained and she no longer met the criteria for thyroid storm. Additionally, one of the most common features of thyroid storm is CNS involvement, which our patient never demonstrated. Understanding the myriad of ways that thyroid storm may present and performing a full work-up in patients at risk for thyroid storm will help aid in the correct diagnosis and treatment plan.

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GOUT GONE WILD! TOPHACEOUS GOUT IN THE SPINE.

First Author: Stephanie Hsiao, MD Sarah Haserodt, MD, Maria M. Patino, MD, Jennifer L. Beachey, MD, Eugene E. Lee, MD., MPH

Introduction: Tophaceous gout typically involves peripheral joints, but in poorly controlled state, can presents as axial gout arthropathy with spinal cord compression.

Case Description: A 46-year-old female with history of poorly controlled tophaceous gout involving multiple joints, chronic back pain, and asthma, was admitted for an asthma exacerbation. On admission, patient was found to have significant thoracic back pain with left arm and bilateral lower-extremities numbness, concerning for myopathy. On further questioning, patient reported her back pain and associated extremities numbness have been slowly progressing over the past year. She denied saddle anesthesia, bladder, or bowel incontinence.

An MRI of the thoracic spine revealed a soft tissue mass with partial encroachment of T7-T8 neural foramen and another soft tissue mass at T10-T11 with mild impingement of the thoracic spinal cord, concerning for metastatic malignancy. Neurosurgery was consulted and there was no indication for emergent surgical intervention. A CT-guided core biopsy of the T7-T8 soft tissue mass revealed crystalline material, most likely urate crystals. Chart review showed that the patient has failed both allopurinol and febuxostat in the past for uric acid level suppression. Her uric acid levels have been ranging between 5-9 mg/dL during the two years prior to admission. Patient is currently awaiting pegloticase infusion and has close follow up with neurosurgery to monitor ongoing symptoms of spinal cord compression. Patient may need surgical intervention if pegloticase is ineffective to control axial gout.

Discussion: This case illustrates the importance of keeping axial gout arthropathy high on the differential diagnosis for patients with a history of gout who present with back pain and neurologic deficits. More common than generally recognized, the prevalence of axial gout has been shown in the literature ranging between 14-35% in patients with history of gout. However, there are only few case reports of axial gout and it remains an under-diagnosed condition because of the ambiguous imaging findings on CT or MRI and a general lack of understanding as part of the disease state. Axial gout typically takes years to develop and may be a preventable condition with strict uric acid level suppression. Unfortunately, this patient was refractory to the typical xanthine oxidase inhibitors and now has tremendous disease burden. Uricase infusion therapy provided earlier in her disease course may have prevented axial invasion of gout. The efficacy of pegloticase in treating refractory chronic peripheral gout has been well studied but not in axial gout, given the lack of clinical cases. Pegloticase infusion in this patient may provide further insights into the long-term management and possible reversal of axial gout.

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FAST AND FURIOUS: BREAST CANCER ON STEROIDS

Annie K. Hung, MD, Jenny R. Aronson, MD, Eugene E. Lee, MD

**Introduction:** Ectopic ACTH secretion is a known paraneoplastic syndrome and is most commonly associated with small cell lung cancer and neuroendocrine tumors. It is a rare complication of breast cancer, less than 1% of cases, and is associated with rapid cancer progression and poor prognosis.

**Case Description:** A previously healthy 32 year old G0P0 African-American woman with known moderately differentiated, triple negative (ER-, PR-, HER2-), metastatic invasive ductal carcinoma, presented with altered mental status, hypertensive emergency, and hyperglycemia. She had previously been treated with carboplatin and paclitaxel, along with denosumab for extensive bone metastases. In addition, she had received whole brain radiation for intracranial metastases. Given her current presentation, a lumbar puncture was performed that showed no evidence of leptomeningeal disease. Head MRI showed stability of the brain metastases and unfortunately evidence of posterior reversible encephalopathy syndrome (PRES) likely related to her malignant hypertension. Her blood pressures remained labile, requiring four different antihypertensive medications. She was also found to have elevated blood sugars and diagnosed with new onset diabetes mellitus. In the setting of her refractory hypertension and hyperglycemia, ACTH and cortisol levels were drawn and found to be significantly elevated. Dexamethasone suppression test failed to decrease her cortisol levels. Cushing syndrome secondary to ectopic ACTH secretion was diagnosed, likely associated with her advanced metastatic breast cancer. She was initially treated with ketoconazole, but developed elevated transaminases and therapy was changed to metyrapone with normalization of serum cortisol levels. She progressed to fulminant liver failure, continued to deteriorate, and expired shortly after transition to comfort care.

**Discussion:** There are less than ten previously reported cases of paraneoplastic Cushing's syndrome in the setting of breast carcinoma. In general, cancer-related hypercortisolemia is associated with significant mortality and lack of response to anticancer therapy. Prompt recognition of this syndrome is necessary to avoid its rapidly evolving, life-threatening complications including hypokalemic alkalosis, malignant hypertension, and severe diabetes mellitus. Abnormal secretion of cortisol has also been shown to have immunosuppressive effects with decreased levels of lymphocytes and natural killer cells, exposing patients to opportunistic infections. Although Cushing's syndrome is a rare complication, breast cancer is the second most commonly diagnosed malignancy among American women and its death rates are higher than those for any other cancer, besides lung cancer. Due to the high prevalence of breast cancer, it is important to know that it can be associated with Cushing's syndrome. Early intervention can directly impact morbidity and mortality, as some of the associated complications, such as PRES and opportunistic infections, can directly impact quality of life. The management of ectopic ACTH syndrome in the setting of malignancy is complex and requires not only control of the hypercortisolemia but treatment of the underlying cancer.

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A CASE OF IV TPA PROVOCATION IN OBSCURE GASTROINTESTINAL BLEED

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Case Presentation: 89 year-old male with history of atrial fibrillation on warfarin and recently discharged with hemorrhagic gastritis presents with recurrent gastrointestinal bleed. During initial hospitalization, two endoscopies were performed which showed no source of bleeding. Coumadin was held and the patient was discharged after stabilization of his hemoglobin. One week later, he presented with melena found to have hemoglobin 5.6. He was transfused 8 units of PRBC in the first 24 hours of hospitalization. Endoscopy revealed duodenal ulcer, which was clipped and the patient stabilized. Work up included tagged RBC scan and abdominal aortogram, however, no additional source of bleeding was found. He became hypotensive on hospital day 8 requiring vasopressors. Hemoglobin dropped from 8.2 to 5.6 in one day. He was resuscitated with fluid and blood products. Bleeding resolved and he remained hemodynamically stable. In attempt to identify the source of bleeding, the patient was given a 5000 unit IV heparin bolus and heparin drip. Tagged RBC study performed was unrevealing. On hospital day 9, he was placed on a tPA drip at 1mg/hr plus 5 mg bolus prior to angiography. He received 11 mg total IV TPA prior to angiography. Extravasation was seen at the left gastric artery and 4 coils were placed after which his bleeding resolved. He was discharged from the hospital without additional bleeding.

Discussion: Obscure gastrointestinal bleed (OGIB) is defined as recurrent bleeding from the gastrointestinal tract after negative evaluations with upper and lower endoscopies. This represents about five percent of all GI bleeds. Common etiologies of OGIB are fundic varices, peptic ulcer, angioectasia, Dieulafoy lesions, and Cameron erosions. Specialized techniques such as dedicated small bowel enteroscopy, enteroclysis, technetium Tc 99m radionuclide scanning, and angiography may aid in localizing the source of bleeding in these patients. Unfortunately, technetium Tc 99m radionuclide scanning or angiography requires active bleeding for a positive result. Provocative angiography is a technique in which bleeding is provoked by a thrombolytic agent and has been reported to induce bleeding in 20% to 60% of patients. Most provocative studies in the literature include heparin or intra-arterial tPA, however, in cases of OGIB where conventional methods fail to identify the source, provocative angiography with intravenous tPA is an effective option. Although there is no evidence that provocative angiography may change outcomes in OGIB, we conclude that it can be a safe modality that should be utilized in patients with life-threatening OGIB that had negative endoscopic and radiological investigations.

Conclusion: This case presented a diagnostic challenge in a patient with recurrent OGIB. Initial attempts at identifying the source with conventional diagnostic techniques were unsuccessful. The source of the bleed was identified after IV tPA administration without adverse outcomes.

## CELIAC DISEASE PRESENTING AS ISOLATED ACUTE WEIGHT LOSS IN AN ELDERLY MAN

Priya Jayachandran, MD; Haifaa Abdulhaq, MD

**Introduction:** Celiac disease is a small bowel disorder characterized by mucosal inflammation and villous atrophy, caused by dietary gluten sensitivity. This classically leads to malabsorption and nutritional deficiencies. However, the disease can manifest across a broader spectrum. Here we describe an atypical case of celiac disease presenting in an elderly man as isolated acute weight loss.

**Case Description:** A 75 year old Caucasian man presented with complaint of 21 pounds weight loss over 2 months. He denied abdominal pain, early satiety, emesis, diarrhea, and melena. He had no prior medical history. He was adopted and his family history was unknown. Physical exam revealed only bilateral inguinal lymphadenopathy.

His Hgb was 12.6 and ferritin 16. CMV by PCR, toxoplasma antibody, HIV, LDH, ESR, ANA, and DS DNA were all negative or normal. He underwent an esophagogastroduodenoscopy and colonoscopy to evaluate for malignancy. EGD showed grossly normal mucosa of the esophagus, stomach, and duodenum. No biopsies were taken. The colonoscopy was also normal. A CT scan showed mild mesenteric and inguinal lymphadenopathy. He was seen by Surgery and taken to the OR for diagnostic laparoscopy and mesenteric and right inguinal lymph node excisional biopsies. The pathology from all the specimens was reactive changes. PET/CT showed mild, non FDG avid lymphadenopathy and he was referred to Oncology. Given his anemia, celiac workup was initiated. Transglutaminase IgA level was >100, gliadin IgA and IgG were both >100, and reticulin and endomysial IgA were also positive. He was started on a gluten free diet and his energy level improved. He was seen again in clinic 6 months later and had gained 15 lbs. He was no longer anemic. No further workup for malignancy was pursued.

**Discussion:** Although classically a disease of children, celiac disease often presents later in life, but most often before 40 years (1). Adult patients rarely present with severe diarrhea and metabolic disturbances. More often, they have nonspecific symptoms such as fatigue, borderline iron deficiency, mild elevations in serum aminotransferases, or are asymptomatic (2).

Studies describe an increasing prevalence of celiac disease with age. An Italian survey documented that 15 percent of newly diagnosed patients are older than 65 and often have symptoms for 11-19 years prior to correct diagnosis (3).

Here we have described an atypical presentation of celiac disease in a 75 year old patient with acute weight loss and mild anemia. A malignancy workup due to lymphadenopathy was negative. On endoscopy his duodenal mucosa did not appear atrophic or inflamed, but biopsies were not taken. He was eventually diagnosed with celiac via serum titers and improved rapidly with a gluten free diet. It is important to keep celiac disease in the differential even with patients who do not present with a classic picture.

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TO ANTICOAGULATE OR NOT?: A CASE REVIEWING GRANULOMATOSIS WITH POLYANGIITIS  
INDUCED ATRIAL FIBRILLATION

First Author: Gurdeep Jhaj, MD Second Author: Mahvand Sadeghi, MD, Msc Third Author: Liza Gill, MD Fourth Author: Anil Perumbeti, MD Fifth Author: Rick Loftus, MD

Case Description: 65-year-old female with hypertension and smoking history presented with a gradual onset of painful, edematous feet for 4-days. She continued to develop erythema and a constant, nonradiating pain in her feet. The patient also noted an increase in her baseline dyspnea, migratory arthralgias, and recurrent sinusitis. On exam, she had sinus tachycardia, inspiratory wheezing, and edematous, erythematous feet. Labs were significant for anemia, microscopic hematuria, and elevated CRP. She desaturated on room air while ambulating, with a PaO<sub>2</sub> of 42mmHg on ABG. Chest CTA showed scattered pulmonary nodules of varying size. She was given supplemental oxygen and admitted with suspicion for infectious, vasculitic, and neoplastic etiologies.

On admission she had ongoing dyspnea and intermittent episodes of tachycardia. An EKG revealed new onset atrial fibrillation with rapid ventricular rate requiring diltiazem drip. A vasculitic panel showed a positive ANA, c-ANCA, and Proteinase3 Ab, which were highly suggestive of Granulomatosis with Polyangiitis. A CT guided lung biopsy revealed necrotizing granulomatous inflammation with acute vasculitis, confirming GPA. IV steroids were started, with immediate improvement in respiratory and extremity symptoms. The patient was discharged on oral corticosteroids, rituximab with oral diltiazem and a short course of Rivaroxaban with cardiology follow-up.

Four days following discharge, the patient presented with hypoxemia requiring intubation. A bronchoscopy revealed diffuse alveolar hemorrhage due to bleeding granulomas. Despite aggressive treatment, the patient expired from respiratory failure.

Discussion: AFib is the most common arrhythmia with estimated 2.7–6.1 million and is associated with a five-fold higher risk of stroke. AFib from pulmonary pathology is well established and has been documented with GPA even without cardiac involvement. In our patient with CT confirmed pulmonary nodules and biopsy proven GPA, the new onset AFib was attributed to her GPA nodules.

In our patient who had an underlying etiology for AFib, there is less data on how to approach anticoagulation. Data in hyperthyroidism and patients with new AFib in the preoperative cardiac surgery period can be utilized in decisions to anticoagulation. In both these groups the decision for anticoagulation is still guided by the CHA<sub>2</sub>DS<sub>2</sub>-VASc score and an assessment of bleeding risk. In our patient the major risk for bleeding was diffuse alveolar hemorrhage which has an incidence of 8-18% in GPA.

She did not have an active bleed at the time of her AFib diagnosis and she did have a CHA<sub>2</sub>DS<sub>2</sub>-VASc score of 3 so it was a reasonable decision to start the patient on anticoagulation. Her echocardiogram ruled out valvular etiologies for her AFib and so the initiation of a novel oral anticoagulants (NOAC) was not contraindicated. This case presented a unique clinical decision initiating anticoagulation with a NOAC in a patient with GPA for AFib with a high risk for stroke and bleeding who expired from diffuse alveolar hemorrhage one week after AFib diagnosis and initiation of rivaroxaban.

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THE DISAPPEARING HEART BLOCK

First Author: Alan K. John, MD Second Author: Hartaj Girn, MD

Introduction: Aortic root abscess is a relatively rare condition which usually occurs as a complication of aortic valve endocarditis. It is more common with prosthetic aortic valves. The proximity of such abscesses to important structures of the heart make it vital to quickly recognize and manage severe complications.

Case: A 61-year-old man presented to the emergency department with ten days of worsening back pain, fever, and chills. He had a history of bicuspid aortic valve with severe stenosis and ascending aortic aneurysm status post bioprosthetic aortic valve replacement and graft repair of the ascending aorta four years prior to presentation. The patient was initially noted to be febrile, normotensive, and tachycardic. Physical Exam was notable for poor dentition and a grade 2/6 systolic ejection murmur at the right upper sternal border. The initial EKG showed sinus rhythm without Atrioventricular (AV) block. Blood cultures were positive for *Strep. viridans*. A transesophageal echocardiogram demonstrated valve vegetations with a large aortic root abscess in close proximity to the mitral annulus. The patient was planned for non-urgent surgical intervention. EKGs then progressively demonstrated first degree heart block, second degree heart block, Mobitz type 1 and type 2, and ultimately complete heart block. A temporary transvenous pacemaker was placed. The patient underwent surgical intervention with aortic root abscess debridement and bioprosthetic aortic valve implantation. There was improvement to first degree AV block with a PR interval of 266 msec post-operatively.

Discussion: This case highlights a course of rapidly progressive AV block in the setting of a large aortic root abscess secondary to endocarditis of a bioprosthetic aortic valve. The proximity of an aortic root abscess to the nodal and infra-nodal conduction system necessitates careful monitoring with serial EKGs. Transvenous pacing is a viable option for temporary correction of acute AV block when emergent surgical intervention is not available. Resolution of acute high grade AV block is possible following surgery.

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AORTO-BRONCHIAL FISTULA CAUSING RECURRENT HEMOPTYSIS 21 YEARS POST HEART TRANSPLANT

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Introduction: 37 year old male with a double outlet right ventricle, pulmonary stenosis, right aortic arch, underwent a descending aorta to right pulmonary artery shunt with an azygous vein graft at one year of age, followed by an orthotopic heart transplant at the age of 16 and mitral valve replacement with an Onyx valve at the age of 27.

Case Description: Patient presented with recurrent episodes of hemoptysis, with his first bronchoscopy showing diffuse mucosal friability suggestive of viral pneumonitis. Hemoptysis was assumed secondary to his supratherapeutic INR at admission in the setting of possible pulmonary infection in an immunocompromised host . He was started on broad spectrum antibiotics, antivirals, and antifungals. Repeat bronchoscopy showed clotted blood in the right upper lobe but no active bleeding. Soon after this patient had an episode of large volume hemoptysis and went into asystolic cardiac arrest with successful resuscitation. A pulmonary angiogram showed an aortobronchial fistula for which an endovascular thoracic aortic stent graft was placed. A metallic clip was observed at the site of the fistula from his previous surgery.

Discussion: The differential included bleeding due to a high INR, HSV pneumonitis, CMV pneumonitis, nasopharyngeal bleeding, tuberculosis, and marijuana related fungal infection. Multiple computed tomography (CT) scans of the chest and bronchoscopies did not identify the source of the bleeding until finally a pulmonary angiogram identified the aortobronchial fistula.

Aortobronchial fistula is a rare cause for hemoptysis in patients with previous surgery of the descending aorta. Patients may initially present with small volume hemoptysis culminating in massive hemoptysis and death. Imaging and bronchoscopy is not always conclusive and a high index of suspicion is necessary to diagnose this life threatening condition.

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TRICK OF THE EYE: WHEN ORBITAL CELLULITIS IS A LACRIMAL GLAND PLASMACYTOMA

First Author: Jane Park, MD Second Author: Eugene Lee, MD

**INTRODUCTION** In patients with multiple myeloma, the vast majority of extramedullary lesions are known to occur in upper respiratory and gastrointestinal tract. Here we present an unusual case of an extramedullary plasmacytoma in the lacrimal gland.

**CASE DESCRIPTION** The patient is an 83 year old male with known multiple myeloma, treated with decadron and revlimid, who presented with swelling and erythema of the right periorbital region. A CT of the orbits with contrast identified a 2.6cm subperiosteal rim-enhancing collection in the superolateral right orbit concerning for abscess and new lytic lesions. An emergent incision and drainage was performed and operative wound cultures grew MSSA and coagulase negative *Staphylococci*. Pathology obtained during the surgery revealed a plasmacytoma of the lacrimal gland.

The patient continued on revlimid for his multiple myeloma with lacrimal gland involvement and scheduled for outpatient follow up. Unfortunately, he was unable to tolerate further therapy and expired with hospice.

**DISCUSSION** Extramedullary plasmacytomas (EP) are rare (7%) and present as part of multiple myeloma as opposed to its distinct cousin, the solitary extramedullary plasmacytoma (SEP) which represent 3% of plasma cell malignancies. The overall incidence of either EP or SEP in the lacrimal gland is unknown, but the condition is exceedingly rare. It is important to differentiate MM with EP from solitary extramedullary plasmacytomas, however, because of significant differences in treatment and survival.

Given the poorer prognosis with EP, it raises the possibility that some cases may be treatment-related transformation of multiple myeloma.

This case elucidates the importance of:

- - Evaluating for extramedullary plasmacytomas in patients with orbital cellulitis.
- - Distinguishing between SEP and EP in multiple myeloma which carries a much worse prognosis.
- - Recognizing that new plasmacytomas in those receiving therapy indicates worsening progression.

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CHERNOBYL CALCIUM CRISIS: A TALE OF THREE NEOPLASMS

Elisa Rogowitz, MD

The Chernobyl Nuclear Power Plant Disaster of 1986 caused a dramatically increased incidence of endocrine cancers with the potential for unusual clinical presentations. This case highlights an extraordinarily rare carcinoma found growing between two other neoplasms in a patient with radiation exposure.

A 65 year old male Ukrainian immigrant presented to his internist for progressive generalized weakness, myalgias, and fatigue. Over the past four months, he has such disabling that he could not rise from bed, forcing him to retire prematurely. He reported expecting early disability and likely cancer as he lived in Chernobyl at the time of the explosion. His only relevant past medical history was on ultrasound-guided FNA of a thyroid nodule that showed benign tissue one month prior. At presentation his physical exam was unremarkable. Surprisingly, lab studies showed a serum calcium 16.7 mg/dl. He was emergently admitted and the hypercalcemia confirmed. He received intravenous hydration, zoledronic acid, and calcitonin. Additional diagnostic work-up revealed PTH 1420 pg/mL (ref range 14-72 pg/mL), Vitamin D-25 20.5 ng/mL, creatinine 2.4 mg/dL (baseline 1.4mg/dL), and albumin 3.3g/dL. Phosphorous, TSH, and free T4 were normal.

A 4D Neck CT scan localized a 4cm x 3.5cm left inferior parathyroid mass that displaced the esophagus and extended into the superior mediastinum. A multinodular left thyroid lobe with spiculated foci was also seen. The patient had an en-bloc resection of the left inferior parathyroid, left hemi-thyroid and isthmus. Intra-operative monitoring of PTH showed the level fell to 118 pg/mL. This was considered to sufficient to exclude another hyperactive PTH-secreting site. Pathology disclosed three distinct neoplasms: parathyroid carcinoma; papillary thyroid carcinoma; and thyroid adenoma. He is disease-free six months after resection.

Parathyroid carcinomas are exceedingly rare. They have an estimated incidence of 2-5 per 10 million persons. They can affect any of the four parathyroid glands but have a strong predilection to the inferior glands. When PTH > 5 times the upper level of normal, it is highly suggestive of parathyroid carcinoma. This patient's PTH was almost 20 greater. In persons exposed to Chernobyl radiation, the most common neoplasm is papillary thyroid carcinoma (93.1%) and thyroid adenoma/hyperplasia (18.8%) which are often found to co-exist. Exceptionally parathyroid carcinoma is found. A recent study correlated high PTH and calcium levels in the Chernobyl clean-up crew. Parathyroid carcinomas pose a diagnostic challenge, both clinically and histologically, due to their indolent early course and variable features on pathology. This case illustrates the need for thorough and careful investigation for concurrent primary cancers in a patient with radioactive substance exposure and a significant decline in health.

## ADRENAL INSUFFICIENCY IN A YOUNG MALE

First Author: Sahar Sherf, MD Second Author: Dorothy Martinez, MD

**Introduction:** Adrenal insufficiency is most commonly due to autoimmune disease, infection, metastatic disease, or drugs. Less commonly, it can result from X-linked adrenoleukodystrophy (ALD)/adrenomyeloneuropathy (AMN), a rare peroxisomal disorder of beta-oxidation caused by mutations in the ATP-Binding Cassette gene at Xq28. This disorder results in accumulation of very long chain fatty acids (VLCFAs) in tissues, particularly the central nervous system, Leydig cells of the testes, and the adrenal cortex. ALD/AMN affects 1 in 16,800 and accounts for ten percent of adrenal insufficiency cases.

**Case:** A 40 year old male initially presented to the neurology clinic for headaches, bilateral foot and thigh paresthesias, diffuse twitching, and gait instability. Neurological exam was notable for impaired attention and recall, hyperreflexia, and decreased vibration. He exhibited diffuse hyperpigmentation of skin and loss of hair in his bilateral lower extremities. Cranial MRI showed increased signal in the cortex, internal capsule, and brain. Of note, his 42 year old brother had died of a motor neuron disease diagnosed on autopsy. Given his family history an ALS gene study was performed and was negative. Exome sequencing then identified a hemizygous c.231G>A nonsense variant for the ABCD1 gene associated with X-linked recessive ALD/adult-onset AMN. VLCFAs were found to be elevated (hexacosanoic acid (C26:0) 3.1 umol/L, ratio of hexacosanoic acid to tetracosanoic acid (C26:0/C24:0) 1.69 and to docosanoic acid (C26:0/C22:0) 0.05). ACTH was then found to be elevated at 1351 pg/mL (normal 6-59 pg/mL); evening cortisol level was 8 mcg/dL. He was referred to the endocrinology clinic for adrenal insufficiency secondary to ALD. The patient noted generalized fatigue/weakness, lightheadedness, mild positional dizziness, salt cravings, and anorexia. He was hypotensive to 95/69 on initial visit. He was started on daily fludrocortisone and prednisone with significant improvement in symptoms.

**Conclusion:** This rare X linked disorder has two main phenotypes. ALD typically begins in childhood and rapidly progresses to quadriplegia. AMN, more likely in our patient given later onset and milder form with slower progression, typically presents between 20 and 40 years of age with weakness, spastic paraparesis, and polyneuropathy. While there is no correlation between the duration or severity of endocrine dysfunction and the severity of the myeloneuropathy, approximately 66% of male AMN patients have resultant Addison disease. We present a case of a young male with adrenal insufficiency due to likely AMN subtype of ALD.

It is important to consider ALD/AMN in a young male with idiopathic adrenal insufficiency. While suspicion is higher in those with vague neurologic symptoms, up to 60% of young men with ALD have no or few neurologic abnormalities at the time of diagnosis of adrenal insufficiency. More challenging, biochemical evidence of adrenal insufficiency can be present for up to two years before the development of clinical signs. Diagnosis of ALD/AMN with elevated VLCFA levels and molecular genome sequencing and rapid initiation of corticosteroid replacement therapy in those with adrenal involvement is essential.

## CHAPTER WINNING ABSTRACT

Cory W Taylor, Omar S Darwish, Sonali L Iyer, Dang K Duong

Symptomatic cholelithiasis is a common ailment with a classic presentation, though occasionally physicians encounter a more rare manifestation. This vignette illustrates classic features of the disease and underscores the importance of clinical judgment. Here is presented a case of a young Hispanic man with marked transaminitis and a stone-cold stoneless endoscopic ultrasound, ultimately reflecting an entity known as gallstone hepatitis.

A 29 year-old Hispanic man presented with a one-week history of right upper quadrant (RUQ) post-prandial pain. He also noticed three days of darkened urine and yellow eye discoloration. He denied weight loss, night sweats, alcohol, fever, diarrhea, dysarthria, or arthralgia. Examination revealed obesity (BMI 36), scleral icterus, RUQ tenderness, and a negative Murphy sign. Laboratory data demonstrated normal albumin with total bilirubin 7.1 mg/dl, AST 217 U/L, ALT 485 U/L and AlkPhos 255 U/L. Abdominal ultrasound showed a normal gallbladder, multiple layering gallstones, and no common bile duct dilatation. An endoscopic ultrasound (EUS) with intent for endoscopic retrograde cholangiopancreatography (ERCP) was performed. It, too, demonstrated multiple gallbladder stones without ductal dilatation or choledocholithiasis. Given the absence of obstructive findings, the ERCP portion of the procedure was cancelled. The patient's clinical and laboratory status worsened (peaked levels: AST 265, ALT 657, t-bili 14.4) and attentions turned to a possible parenchymal liver process. Magnetic resonance cholangiopancreatography (MRCP) revealed cholelithiasis, again, without evidence of ductal stones or dilatation. An autoimmune panel was negative. Additional tests for viral hepatitis, alpha 1-anti-trypsin, mononucleosis and hemochromatosis were unrevealing. Ultimately, a liver biopsy was performed. Pathology suggested ductal obstruction or drug-induced liver injury, without histologic features to support other causes. Given the extensive evidence against an intrahepatic etiology, gallstone disease again emerged to the forefront of the investigation. A repeat EUS revealed 10 mm CBD dilation with four stones distally. ERCP with sphincterotomy was performed, producing all four visualized stones along with a number of smaller stones and debris. The patient recovered, and was subsequently discharged with arrangements for outpatient cholecystectomy.

In conclusion, a young man presented with a common disorder dynamically camouflaged from otherwise highly sensitive tests. This, in turn, prompted an incredible degree of unnecessary, expensive, and even invasive testing that could all have been avoided with increased reliance on clinical judgement. The diagnosis was there – the diagnostics were not. Gallstone hepatitis is a risk factor for developing ascending cholangitis. It reflects the emerging pathophysiology of gallstone-induced cholestasis and requires interventional action based on physician instinct, even sometimes in the face of uncertainty.

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A SWEET, SWEET RASH: AZATHIOPRINE-INDUCED SWEET SYNDROME IN A PATIENT WITH  
ULCERATIVE COLITIS

Vivian Tien, MD Willis Bowman, MD Kanal Singh, MD

Sweet syndrome, or acute febrile neutrophilic dermatosis, is an inflammatory disorder characterized by abrupt onset of fever and painful rash. Most frequently associated with malignancy or underlying autoimmune disease, it can also be secondary to drug reaction. Here, we describe a case of Sweet syndrome in a patient with multiple risk factors, demonstrating the importance of a complete history in what may have otherwise seemed to be a simple case of infectious colitis.

A 69 year-old man with a history of ulcerative colitis, on azathioprine and budesonide, presented to the Emergency Department with chills, fatigue, and diarrhea of 3 days' duration. On review of systems, he noted a slightly tender rash on his arms, legs, neck and chest that had developed 5 days prior. Physical examination was notable for fever, tachycardia, benign abdominal exam, clear lung fields, and an erythematous macular papulopustular rash on the patient's neck and elbows. Stool tested positive for *Clostridium difficile* toxin, prompting hospitalization and initiation of metronidazole for presumed *C. difficile* infection. Of note, the patient started azathioprine two weeks earlier during routine follow-up with his gastroenterologist. The patient's hospital course was marked by persistent fever, tachycardia, and tachypnea, particularly concerning for sepsis. However, antibiotic therapy was broadened without clear clinical benefit and signs and symptoms ultimately improved after holding azathioprine and budesonide out of suspicion for adverse drug reaction and infection. Histopathology from a lesional skin biopsy obtained on admission revealed infiltrative neutrophilic dermatosis of the dermis and epidermis consistent with Sweet's syndrome. Ultimately, final blood, urine, and skin biopsy cultures were negative and patient was discharged with resumption of budesonide and cessation of azathioprine. Two weeks after discharge, he reported near resolution of the rash and return to his usual state of health.

This case highlights the importance of thorough medication reconciliation and clinical vigilance to identify adverse drug reactions. The patient was treated with multiple antibiotics but his symptoms were most likely secondary to drug-induced Sweet syndrome, which can pose a diagnostic dilemma as it may mimic other disorders. Sweet syndrome is diagnosed by both clinical and pathologic criteria, including signs and symptoms of inflammation, and the characteristic rapid onset of rash characterized by dense neutrophilic infiltrates. Azathioprine has been documented as a causative agent of Sweet syndrome, particularly in patients with underlying inflammatory bowel disease. Some have proposed that a new clinical entity, azathioprine hypersensitivity syndrome, be described to more clearly link the systemic reaction to the offending drug.

## SURFER'S MYELOPATHY: A RARE NONTRAUMATIC SPINAL CORD INJURY ASSOCIATED WITH NOVICE SURFERS

First Author: Angelica L Zen, MD Ault, Mark, MD

Surfer's myelopathy is a nontraumatic spinal cord injury associated with surfing that is becoming increasingly recognized. Because it can have devastating consequences, providers should be aware of the signs and symptoms of surfer's myelopathy and counsel their patients appropriately. A 23-year old female presented with new onset bilateral lower extremity paralysis after surfing for the first time. While walking onto shore, she became very weak and had to be carried out. 45 minutes later, she had severe and progressive weakness of her lower limbs and numbness traveling proximally. She reported no trauma while surfing.

The patient's vitals were normal. Weight was 54.6 kg (BMI 18.3). Exam showed decreased sensation of the trunk at T8 and down. She had 1/5 right hip flexion, but otherwise no lower limb strength. Bulbocavernosus reflex was intact. Rectal tone was decreased. Lower extremity deep tendon reflexes were absent. Lumbar spine was mildly tender.

Hypercoagulable studies, rheumatologic work-up, vitamin B12, RPR, and HIV were normal. Neuromyelitis optica antibodies were mildly elevated, but ophthalmologic exam was normal. MRI spine showed gray matter edema centrally from the T7-8 level extending inferiorly to the level of the conus, with some distal cord expansion, concerning for an underlying cord infarct. She was started on IV solumedrol 1g daily for 5 days.

The patient was discharged to acute rehab, and had improvement in bilateral lower extremity function, though she had residual spasticity and weakness requiring ambulation with an assistive device. She regained bowel and bladder functioning.

Since the first case series published by Thompson in 2004, there have been approximately 35 cases of surfer's myelopathy reported in the literature. Symptoms include back pain, urinary retention, sensory deficit, and lower limb weakness during or shortly after surfing. Patients are usually first time surfers and thin, suggesting that lack of trained musculature may be a risk factor. MRIs show hyperintense signal abnormalities extending from the midthoracic region to the conus.

The etiology of surfer's myelopathy is not certain; however, the leading hypothesis is that hyperextension of the spinal cord while laying prone leads to compression or vasospasm of the spinal arteries, leading to transient ischemia. At greatest risk for ischemia is the midthoracic spine, which is a watershed region and has poor vascular supply, predominantly by a single Adamkiewicz artery. This artery also traverses through the intervertebral foramina, making it susceptible to mechanical compression. Novice surfers may be at higher risk due to poor technique and exaggerated spine extension.

Outcomes vary from full recovery to complete paraplegia despite rehabilitation. Many patients receive empiric high dose steroids, although this has not seemed to change outcome. Future research is needed regarding appropriate management and measures to prevent surfer's myelopathy.

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SEVERE RHABDOMYOLYSIS: AN UNAPPRECIATED CONSEQUENCE OF WATER INTOXICATION

Nazanin Montazeri, MD, Katarzyna Plaza, MD, Jason Cheung, MD, James Gibson, MD.

We present the case of a 32-year-old male with severe rhabdomyolysis following rapid correction of acute hyponatremia secondary to water intoxication. The patient's past medical history included psychogenic polydipsia, obsessive-compulsive disorder, generalized anxiety disorder, and anorexia nervosa. He was found on the ground following a presumed seizure. He was incontinent of stool and had evidence of tongue bite. The patient had consumed a large amount of free water earlier in the day. His serum sodium level was measured at 106 mmol/L; down from 124 mmol/L only 9 hours earlier. Less than 48 hours earlier, his serum sodium was 134 mmol/L. Given the presumed seizure, the patient was treated with hypertonic saline. He received a total of 200 cc of 3% hypertonic saline, resulting in an increase of his serum sodium to 116 mmol/L. His serum sodium level then corrected to 135 mmol/L within fifteen hours with fluid restriction as the only intervention.

Creatinine kinase (CK) levels were measured at the time of the presumed seizure and were elevated at 2489 U/L (normal < 200 U/L). The CK levels increased the following day, but this was felt to be attributable to restraints that were needed for agitation. Within 48 hours of the event, his CK increased to 39,775 U/L, eventually peaking at 64,899 U/L. He was treated for rhabdomyolysis with intravenous fluids and urine alkalinization. The patient recovered with no evidence of renal impairment.

This case highlights a rare and largely underrecognized cause of rhabdomyolysis in the context of severe hyponatremia from water intoxication.

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AN UNUSUAL CASE OF PROPTOSIS: IGG4-RELATED DISEASE IN THE SPOTLIGHT

First Author: Shannon Ruzycski, MD Jay L. Patel Margaret M. Kelly

**Introduction:** IgG4-related disease (IgG4-RD) is a newly recognized disorder in which IgG4-positive plasma cells infiltrate tissues with subsequent fibrosis and tumefactive enlargement. Many organ-specific diseases, previously thought to be unrelated, are now recognized as manifestations of this systemic disease. We describe a case of rapid lacrimal gland enlargement due to IgG4-RD, which was successfully treated medically. In addition we discuss how to recognize and diagnose this uncommon but treatable disorder.

**Case Description:** A 66-year-old male without significant medical history presented to the Urgent Eye Clinic with one week of painless right eye proptosis and epiphora. Computed tomography of the head demonstrated a homogenous, 3.7 x 1.7 cm enhancing mass arising from the right lacrimal gland that displaced the lateral rectus muscle. Urgent surgical biopsy was performed. Pathology demonstrated a lymphoplasmacytic infiltrate with storiform fibrosis and occasional eosinophils. Immunohistochemistry showed that IgG4-positive plasma cells were increased and constituted more than 40% of the total plasma cells. Serum immunoglobulin subclass analysis revealed an IgG4 level of 3.50 g/L (normal range 0.01-1.40 g/L). A diagnosis of IgG4-RD was made, and treatment was initiated with high-dose prednisone.

**Discussion:** IgG4-RD is a recently described, systemic fibroinflammatory disease defined by characteristic pathologic lesions. A recent consensus report (*Mod Pathol 2012;25:1181*) emphasized the importance of histological findings as the diagnostic criteria. The number of IgG4-positive plasma cells must be elevated (the threshold depending on the biopsy site); with the proportion of IgG4-positive plasma cells greater than 40% of total. In addition, there should be increased lymphocytes and fibrosis. Obliterative phlebitis is a characteristic finding, but was not present in our case. Diagnosis also requires exclusion of malignancy and granulomatous inflammation. Increased serum IgG4, whilst useful if present, is not required for diagnosis, and in the absence of typical histology, is a non-specific finding.

Since the first description of the disease-defining IgG4-positive plasma cell infiltrate in a landmark case series of autoimmune pancreatitis in 2003, IgG4-RD has been reported to affect nearly every organ. Thus, physicians in any specialty may encounter IgG4-RD. Clinical presentation varies by the affected organ, and most commonly is due to mass effect due to organ enlargement or subacute failure secondary to fibrosis. The most commonly affected organs are the pancreas and salivary glands. A high index of suspicion is required in order to uncover a diagnosis of IgG4-RD due to its non-specific presentation. It is likely that many cases go undiagnosed, despite this being a readily treatable condition, with the majority of cases responding to glucocorticoids.

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THE INVASIVE LIVER ABSCESS SYNDROME PRESENTING AS SEPTIC ARTHRITIS

First Author: Shannon Ruzycski, MD Bonnie Meatherall, MD

**Case Description:** A 54-year old man with hypertension and recently diagnosed diabetes mellitus presented with a subacute onset of left knee pain and swelling. He also had a four week history of drenching night sweats, fevers, and unintentional weight loss of 5 kilograms. On exam, his left knee was erythematous with active and passive range of motion limited by pain. Culture of synovial fluid demonstrated the presence of *Klebsiella pneumoniae* which was sensitive to ceftazidime. The patient was treated with intravenous ceftazidime and underwent irrigation and debridement of the affected joint on his second day of admission. Despite this therapy, the patient continued to have elevated white blood cell count and documented fevers on days three through five of admission. Repeat blood cultures grew *K. pneumoniae*. On further history, the patient revealed that he had spent three weeks in the Philippines visiting family four months prior to his presentation. Abdominal ultrasound demonstrated a large liver abscess, which was drained. Culture of the abscess fluid also grew *K. pneumoniae*. The patient was then diagnosed with the invasive liver abscess syndrome secondary to presumed hypervirulent *K. pneumoniae*. With drainage, the patient improved clinically and was discharged well with six weeks of intravenous ceftazidime.

**Discussion:** Epidemiologic studies indicate that between 80-90% of all septic arthritis are caused by *Staphylococcus aureus*, *Streptococcus* species, or *Neisseria gonorrhoea*. Gram negative organisms are rarely reported to cause bone, joint or soft tissue infections in patients without history of immune compromise, trauma, or intravenous drug use. Identification of gram negative organisms in synovial fluid may indicate serious, unrecognized disease. Single case reports and small case series suggest connections between specific organisms and certain diseases. For example, septic arthritis caused by *Fusobacterium* species has been linked to undiagnosed periodontal disease and tonsillar abscess. Familiarity with connections between unusual causes of septic arthritis with underlying comorbidity may prompt the clinician to pursue these diagnoses.

Hypervirulent *K. pneumoniae* is an emerging pathogen initially described in a small case series in Southeast Asia in the 1980's. It is phenotypically and genetically distinct from the more familiar *K. pneumoniae* strains that cause pneumonia and urinary tract infections. Since its identification as the cause of the invasive liver abscess syndrome, hypervirulent *K. pneumoniae* has been reported with increasing incidence in North America. Clinically, the invasive liver abscess syndrome is characterized by a primary liver abscess with hematogenous spread of infection to distant sites, most commonly the eyes, central nervous system, and lungs. Patients with unusual sites of *K. pneumoniae* infection should undergo abdominal ultrasound as diagnosis of the invasive liver abscess syndrome leads to a change in management.

## EXTRACORPOREAL MEMBRANE OXYGENATION FOR THE TREATMENT OF SEVERE REFRACTORY HANTAVIRUS CARDIOPULMONARY SYNDROME

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**Introduction** Hantavirus cardiopulmonary syndrome (HCPS) is a rare viral infection transmitted by infected rodents. HCPS is characterized by rapid onset of adult respiratory distress syndrome, cardiovascular collapse, acute renal failure and disseminated intravascular coagulopathy. Despite optimal supportive treatment with invasive ventilation methods and hemodynamic support, mortality remains high. In recent years, extracorporeal membrane oxygenation (ECMO) has proven to be an effective supportive therapy for severe HCPS.

**Case Description** We present the case of a previously healthy 22-year-old man admitted to an intensive care unit (ICU) in Montreal, Canada, on June 25 2015 with rapid onset respiratory failure and circulatory collapse.

The patient presented with a two-day history of vomiting, bloody diarrhea and fevers at 40 degrees Celsius. On initial exam his blood pressure was 140/80, heart rate of 130 beats per minute and saturation 95% on room air. At that time, his physical exam was non-contributory. Over the next 18 hours, he rapidly developed profound hypoxemic respiratory failure, requiring intubation and invasive ventilation. Despite controlled ventilation and paralysis, his oxygen saturation remained low at 75%. Using a pulmonary artery catheter, he was also found to have a severely depressed cardiac index despite fluid resuscitation and high dose inotropic support. His initial chest x-ray showed severe bilateral interstitial and alveolar infiltrates. He was initiated on broad spectrum antibiotics for severe community acquired pneumonia and urgently transferred to a tertiary care center, where veno-arterial ECMO was initiated eight hours after transfer. Blood, stool, and urine cultures were negative. Multiplex polymerase chain reaction was negative for adenovirus or other respiratory viruses. However, his Hantavirus IgM and IgG serology came back positive from the Canadian National Microbiology Laboratory. The patient's family subsequently confirmed that rodents were present in the platforms under the tents where the patient slept at night, during military training exercise in Wainwright, Alberta from April 23 to June 1<sup>st</sup> 2015. Within 36-48 hours, his cardiac function and gas exchange improved. ECMO was successfully withdrawn after a total duration of 112 hours. Subsequently, the patient was extubated and was discharged home from the medical ward on July 9<sup>th</sup> 2015 without sequelae.

**Discussion** HCPS can present with refractory cardiopulmonary collapse with high associated mortality rates. The incidence of HCPS has doubled in Canada in recent years, rising to more than 10 cases per year in the last two years. Supportive therapy using ECMO as a bridge to recovery has retrospectively been shown to significantly reduce mortality in patients with severe HCPS. This case highlights the importance of a detailed exposure history and reminds physicians of the presenting features and treatment modalities for HCPS. Early consideration for transfer to an ECMO capable tertiary care center should be considered in patients with suspected HCPS and progressive hemodynamic deterioration.

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STROKE-LIKE SYMPTOMS FOLLOWING A PERCUTANEOUS CT-GUIDED LUNG BIOPSY: PERHAPS A MORE COMMON PROBLEM THAN PREVIOUSLY REALIZED

First Author: Katie L Kaput, DO

**Introduction:** Percutaneous CT-guided biopsy is a minimally invasive procedure that is increasingly being used to investigate worrisome pulmonary lesions. It is indicated to evaluate new or enlarging solitary nodules, multiple nodules in patients without malignancy, persistent focal infiltrates and hilar masses. Complications of the procedure are rare, but potentially underestimated and associated with significant morbidity. We present the case of a cerebral air embolism due to percutaneous CT-guided lung biopsy, followed by a discussion of this procedural complication and its future implications.

**Case:** An 80-year-old female, with known longstanding tobacco dependence, was referred to IR for CT-guided lung biopsy of a 3.5 x 2.7 cm cavitory lesion. The procedure was performed by an experienced radiologist. The target lesion was localized and a total of three biopsies were taken using a coaxial 19-gauge introducer with a 20-gauge Cook Quick-Core biopsy device. The patient tolerated the procedure well, but shortly after, developed acute onset left sided hemiparesis. A stroke protocol was initiated and a CT head was obtained. The CT demonstrated numerous curvilinear structures in the region of right anterior cerebral artery, suggestive of air embolus. The patient was then stabilized and transferred for hyperbaric treatment. The remainder of her hospital course was complicated by epilepsy, inability to maintain adequate nutrition and disability due to persistent left sided deficits. The final biopsy revealed stage I adenocarcinoma.

**Discussion:** In recent years, annual screening for lung cancer with low dose CT has increased the detection of pulmonary lesions, with the major question being the probability of malignancy. Management options include surveillance, nonsurgical biopsy, surgical biopsy or surgical resection. Various models dependent on lesion volume and doubling time exist to assist with management, but present a dilemma for elderly patients, with multiple comorbidities who are at highest risk. CT-guided biopsy is considered a less invasive option. Though it is not without risks; and despite a low incidence of previously reported cases of systemic air embolism (i.e. 0.07%), as more nodules are identified and the number biopsies increases, so may this complication.

Given the significant morbidity and mortality associated with this condition, efforts should be made to identify risk factors. Risk factors our patient may have encountered include the coaxial biopsy system, the number of biopsies and presence of a cavitory lesion. In the coaxial biopsy system, the outer cannula is open to the atmosphere for a fraction of a second. If multiple specimens are taken, air can be introduced into the system for a few seconds. When the needle is properly placed, this is not an issue. However, this can be a problem for cavitory lesions which require multiple biopsy attempts. Use of a hemostatic valve has been mentioned, but is not commercially available and more data is needed to identify patient, lesion, and technical factors which may help reduce the frequency of this dreaded complication.

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NORWEGIAN SCABIES MASQUERADING AS PLAQUE PSORIASIS: A COMMON MISDIAGNOSIS

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Norwegian scabies is a rare but severe form of scabies that causes erythematous plaque-like lesions covering many different areas of the body including the hands, feet, and scalp. It typically occurs in the immunosuppressed but should be considered in any patient who presents with itching and progressive plaque-like lesions.

A 34 year-old male with a history of alcohol abuse initially presented to an outside hospital with six weeks of a scaly rash on his hands, feet, and elbows. He was diagnosed with psoriasis and given a prednisone taper and amoxicillin at discharge. One month later, he was admitted to a different hospital after his rash and itching worsened. Exam showed silver plaques with surrounding erythema covering his feet, knees, buttocks, groin, hands, elbows, and scalp. He was unable to ambulate due to fissures in the plaques of his feet causing severe pain. Labs were notable for mild thrombocytopenia, normal WBC count, and a negative HIV test. Dermatology was consulted and determined the rash to be consistent with severe eczematous dermatitis with secondary impetiginization. A shave biopsy was performed on his right lower abdomen, and he was discharged with oral prednisone, topical triamcinolone and topical terbinafine. Following discharge, biopsy results returned showing scabies. He returned to the emergency department three weeks later with worsening extremity pain and rash. He was administered five total doses of oral ivermectin and topical permethrin cream daily. By the time of discharge nine days later, skin erythema remained but all of his plaques had resolved.

Norwegian scabies is a rare form of disseminated scabies that occurs as a result of hyperinfestation of the parasitic mites. The characteristic erythematous patches often cause it to be mistaken for other common dermatologic conditions including eczema and psoriasis. Recognized risk factors include HIV infection, Down syndrome, and autoimmune diseases. This patient's risk factor was likely his alcohol abuse. Biopsy should be performed in all difficult to diagnose skin lesions in order to expedite appropriate medical treatment. Rapid diagnosis and treatment in Norwegian scabies can prevent spread of disease and quickly relieve a patient's symptoms.

IGG4-RELATED DISEASE AS AN UNUSUAL UNIFYING DIAGNOSIS OF LUNG AND KIDNEY  
PSEUDOTUMORS AND PROGRESSIVE RENAL FAILURE

First Author: Ana Onuchic, MD, David Yoder, DO, Peruvamba Venkatesh, MD. University of Connecticut Health Center Internal Medicine Residency, Farmington, CT.

**Introduction:** IgG4-related disease (IgG4-RD) is a recently recognized clinicopathologic entity typically characterized by formation of tumefactive lesions, dense lymphoplasmacytic infiltrate with abundant IgG4-positive plasma cells on histopathology, and elevated serum IgG4 levels. This immune-mediated systemic disease often has its diagnosis prompted by radiologic findings or organ failure. The diverse spectrum of clinical presentations frequently makes it both an atypical and difficult diagnosis.

**Case description:** A 70-year-old African American male with a history of hypertension, diabetes mellitus, stage III chronic kidney disease and mild dementia presented to the hospital with decreased oral intake, weight loss of about 20 lbs in the preceding few weeks, and transient upper respiratory symptoms one week prior. Laboratory data revealed acute kidney injury with serum creatinine of 5.9 mg/dL (baseline 1.5 mg/dL), BUN 79 mg/dL, and a random urine protein of 158 mg/dL. Computed tomography of the abdomen revealed an abnormal soft tissue mass in the left kidney, retroperitoneal lymphadenopathy and multiple pleural-based lung masses, concerning for malignancy. Clinical suspicion was initially highest for renal malignancy with pulmonary metastasis, but subsequent biopsy of a representative pleural-based lesion revealed a dense lymphoplasmacytic infiltrate, raising suspicion for IgG4-RD. Subsequent finding of elevated serum IgG4 level of 252.5 mg/dL supported this unusual diagnosis. After significant continued decline of the patient's renal function, he was treated empirically for IgG4-related kidney disease. After 2 days of oral dexamethasone 20mg daily, the patient's serum creatinine, which had peaked at 8.5 mg/dL, started to improve. A renal biopsy was performed and revealed patchy, dense lymphoplasmacytic infiltrate with numerous plasma cells expressing IgG4 (up to approximately 75-100 per high power field), associated focally with fibrosis and glomerulosclerosis. The patient was transitioned to prednisone 1mg/kg/day, and his serum creatinine improved to 3.1 mg/dL over 8 days.

**Discussion:** In this case, IgG4-RD was found to be the underlying diagnosis of malignant-appearing masses in the lung and kidney, with associated progressive renal failure. A high degree of suspicion and appropriate recognition of this novel disease process is critical, as steroid therapy often yields a prompt and profound clinical response, as seen in this case. It is also important to avoid erroneous management of tumefactive lesions, which can be easily misdiagnosed as malignancies on initial evaluation.

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PSEUDOCYLO-PNEUMOTHORAX: A RARE COMPLICATION OF RHEUMATOID ARTHRITIS

First Author: Jatan Shah, MD Second Author: Natasha Bhimani, MD Third Author : William Frederick, MD Last Author: He Zhang, MD

Introduction Pleural effusions are common in patients with rheumatoid arthritis (RA) but pseudochylothorax or spontaneous pneumothorax are rare and may be discovered incidentally or present insidiously. Here, we report a case where both pseudochylothorax and pneumothorax were present in an asymptomatic patient with chronic RA and discuss the diagnosis, pathophysiology and management options.

Case presentation A 56-year-old male with a three-year history of RA and chronic nonproductive cough presented after a chest x-ray performed outpatient revealed a new right-sided hydropneumothorax. He denied any history of trauma, malignancy or tuberculosis. Physical exam revealed diminished bibasilar breath sounds, hyperresonance at the right base and rheumatoid deformities of both hands. Lab investigations showed eosinophilia, elevated rheumatoid titer, ESR and CRP. Blood and sputum were negative for bacteria, mycobacteria or fungi. CT chest revealed large right-sided hydropneumothorax with pleural thickening and left-sided pleural effusion devoid of masses or lymphadenopathy. Thoracentesis revealed lymphocyte dominant chylous effusion with cholesterol 198 mg/dl, triglyceride 161 mg/dl, LDH 3540 IU/L, protein 5.4 and glucose 3 mg/dl. Open thoracotomy demonstrated extensive dense adhesions of both the visceral and parietal pleura with loculated effusion. Pleural fluid showed no evidence of malignancy or infection and biopsy revealed significant chronic inflammation. Debridement and decortication were performed and chest tubes were placed. Patient was subsequently discharged once the lung had re-expanded and effusion had resolved.

Discussion Pseudochylothorax (chyliform effusion or cholesterol pleurisy) is an exudative effusion that gradually becomes enriched with cholesterol. In majority of the cases it presents with thickened fibrotic pleura resulting from chronic pleuritis of at least 5 years which then “traps” cholesterol from the cell wall of degraded erythrocytes and neutrophils present in the pleural fluid. Diagnostically, fluid triglycerides levels <110 mg/dl, cholesterol levels >200mg/dl, glucose level < 40mg/dl, protein level >4g/dl and LDH level >700 IU/L are typically seen. Treatment of underlying pleuritis is essential with NSAIDs or steroids but decortication may be useful in symptomatic effusion or large pleural thickening. Spontaneous pneumothorax is thought to be due to rupture of necrobiotic nodule forming a persistent bronchopleural fistula (BPF). Diagnosis is made based on new air fluid level or persistent air leak. Management can be challenging and may require surgical intervention.

Conclusion It is important to identify pseudochylo-pneumothorax and associate it with RA for early detection of pleurisy and BPF. This will help initiate prompt treatment and thus prevent restrictive pleural changes to improve the overall quality of life.

## ARTHRITIS IN A FEMALE RETURNING FROM THE TROPICS, A LOCAL CULPRIT

First Author: Ahmed Babiker 1, MBBS Antonio G. Corona 1, MD, Nouman Akbar 1, MD, Ayia Babiker 2, MBBS; Jason Bailey 1, DO.

**Introduction** Minocycline is a synthetic tetracycline commonly used to treat acne vulgaris. Minocycline differs from the other tetracycline antibiotics in that it has a unique dimethylamino that allows for the formation of the quinone iminium ion. Minocycline is associated with a range of autoimmune entities, it is postulated that the immunologically-driven adverse reactions could be related to the abnormal production of reactive metabolites.

**Case Presentation** We present a case of a previously healthy 29 year-old Caucasian female who presented with joint pains, fatigue, elevated liver function tests and inflammatory markers after vacationing in Saint Lucia. She denied any drug or alcohol abuse, she had been in a monogamous relationship and had no recent tick bites or animal exposure. Her review of systems was significant for anorexia, throat pain, early morning joint stiffness and myalgias. Her physical examination was remarkable for small joint arthritis and faint livedo reticularis skin rash. Initially the patient was worked up for Chikungunya, Dengue, Lyme disease and reactive arthritis all which were negative. The patient was then referred to a rheumatologist to explore a possible autoimmune etiology for her symptoms. A comprehensive autoimmune workup was ordered. The results of the work up were unremarkable apart from positive antistreptolysin O antibodies, an elevated C-reactive protein (CRP) and aldolase. Interestingly though her ANA, which was negative on initial presentation, was now positive with a titre of 1:1280. However anti-histone (AHA), anti-dsDNA, Anti-Smith, a-Jo, anti-CCP antibodies were negative. Given her poor response to non-steroidal anti-inflammatory drugs and positive ANA, an empiric trial of steroids was attempted. The patient quickly reported symptomatic relief. It was at this time the patient reported that she had been taking oral Minocycline one month prior to the development of her symptoms. A presumptive diagnosis of minocycline-induced lupus with autoimmune hepatitis was made and the patient transitioned to long-term Hydroxychloroquine with continued symptomatic improvement.

**Discussion** Drug-induced lupus erythematosus (DILE) should be suspected in any individual with the clinical features of SLE, exposure to a known offending agent and a positive ANA test. Minocycline is associated with a range of autoimmune entities, the two most prevalent are DILE and autoimmune hepatitis which can often coexist together. Unlike other causative agents, Minocycline induced DILE is typically not associated with a positive AHA. The constellation of malaise with rash, myalgias, and arthritis in a returning traveler brings many etiologies to mind and one must consider specific geographically relevant conditions. However, one must not overlook the importance obtaining a complete history and physical examination. This case highlights the importance of history taking and the unique presentation of minocycline-induced lupus with associated autoimmune transaminitis.

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## HYPERBARIC OXYGEN IS EFFECTIVE FOR TREATMENT OF PNEUMATOSIS INTESTINALIS DUE TO SYSTEMIC SCLEROSIS

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Pneumatosis intestinalis (PI) is the presence of gas within the bowel wall. Acute PI is associated with acute illness and bowel necrosis. Chronic PI may be asymptomatic and associated with medical conditions such as infectious diseases and rheumatologic conditions. PI has been described in patients with systemic sclerosis. Bowel rest, antibiotics, and pro-kinetics have been proposed as treatments and have had varying success. Hyperbaric oxygen (HBO) therapy has also been described, but research on its effectiveness is limited. We present a case of complete resolution of pneumatosis intestinalis after hyperbaric oxygen therapy.

A 29 year old woman with systemic sclerosis, gastroparesis, and chronic abdominal pain was transferred to our hospital for worsening abdominal pain, nausea, vomiting, and inability to tolerate oral feeding. She had been maintained on total parenteral nutrition. One year before her admission, she was diagnosed with pneumatosis intestinalis localized to the ileum. Her prior treatment included bowel rest and broad-spectrum antibiotics, but she did not improve radiographically or clinically. On admission, she was afebrile and mildly tachycardic with a heart rate in the 110s. Her leukocyte count and lactate were normal. On physical exam, she had sclerodactyly, calcinosis cutis, and microstomia along with diffuse abdominal tenderness. A radiograph of her abdomen showed marked and diffuse PI. CT showed pneumatosis extending from the jejunum to the rectum with evidence of free air in the peritoneum. There was no evidence of bowel necrosis. She received two HBO treatments over the course of two days. After her first treatment, she demonstrated marked clinical improvement. A follow up radiograph showed complete resolution of the pneumatosis. She received an additional HBO treatment resulting in resolution of her abdominal pain and return of her ability to tolerate an oral diet.

In patients with chronic medical conditions and associated PI, symptoms may cause a significant reduction in their quality of life. Hyperbaric oxygen therapy has been used successfully to treat PI. HBO reduces the partial pressure of non-oxygen gasses within the cysts. Absorption of oxygen results in decreased cyst volume. Supplemental inhaled oxygen might have a similar effect, but is also associated with ocular, pulmonary, and other toxicities. Hyperbaric oxygen avoids the effects of hyperoxia. No complications have been reported from HBO therapy. Health care providers should recognize hyperbaric oxygen therapy as a safe, effective, and non-surgical treatment of pneumatosis intestinalis. More studies are needed to determine treatment length to ensure resolution of the bowel wall air and to keep patients symptom free.

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AN UNCOMMON PRESENTATION OF MULTIPLE MYELOMA IN A DEPLOYED SOLDIER

Akira Shishido, MD Camellia Hernandez, MD Sharon Ahluwalia, MD

Multiple myeloma accounts for 10% of hematologic cancers making it one of the most common malignancies diagnosed in the United States. Classically, multiple myeloma presents with hematologic abnormalities, bone pain or fractures, hypercalcemia, or renal failure, and nearly one third of all cases are diagnosed incidentally. Less commonly patients may present with neuropathy, pancytopenia, or infections – all of which stem from the disease process of hyper-secretory plasma cell overgrowth and resulting immunodeficiency. Here, we present an uncommon presentation of multiple myeloma that initially was thought to be a primary immune deficiency due to hypogammaglobulinemia.

While deployed in Afghanistan, a 43-year-old male Air Force Officer presented to a battalion aid station with symptoms of a viral gastroenteritis. He had a history of chronic loose stools and abdominal discomfort over the preceding 5 months, but was able to continue his daily activities and exercise. However, after an outbreak of gastroenteritis on his base, he developed an exacerbation of abdominal cramping and multiple liquid stools per day. His only medication was doxycycline for malaria prophylaxis. Upon evaluation at the base clinic he had an unremarkable exam, but was noted to be anemic with a hematocrit of 30.9. He was medically evacuated to a hospital in Germany where additional testing revealed macrocytosis of 104, as well as low vitamin B12 and folate levels of 107 and 5 respectively. A celiac disease panel revealed a low IgA level of 19 and subsequent quantitative antibody levels revealed severe hypogammaglobulinemia. Given his history of gastrointestinal symptoms and what appeared to be profound immunodeficiency, he was medically evacuated to Water Reed Medical Center in Bethesda, MD for a suspected diagnosis of common variable immune deficiency. Stool testing for viral, bacterial and parasitic infection were negative, and a blood smear showed macrocytic anemia but no other abnormalities. Gastroenterology performed upper and lower endoscopies that revealed no abnormalities. Immunology performed serum and urine protein electrophoresis and free light chain testing which revealed a monoclonal protein spike and elevated kappa free light chains. Oncology performed a bone marrow biopsy that revealed hypercellular marrow comprised of 90% plasma cells. He was diagnosed with Multiple Myeloma and was initiated on appropriate treatment.

Typically patients with hypogammaglobulinemia, whether primary or secondary, present with recurrent bacterial infections of either the respiratory or gastrointestinal tract. Given the patient had been taking daily doxycycline for prophylaxis of malaria while deployed, we suspect he may have masked his underlying immunodeficiency and prolonged his diagnosis. This case illustrates the variability of presentations of common disorders and how an operational military environment may complicate diagnostic efficiency.

## EXERCISE ASSOCIATED CARDIAC ASYSTOLE IN A HEALTHY INDIVIDUAL

First Author: Piyush Sovani, MD Second Author: Monvadi B Sricahi, MD

Exertional syncope due to underlying cardiovascular conditions is a well-recognized and reported condition. However, syncope due to cardiac dysfunction in individuals with no structural heart disease is extremely rare. One serious and potentially fatal cause of this is exercise associated cardiac asystole. We report a case of an athletic, healthy young individual who developed post-exercise associated cardiac asystole after a stress test, suggesting a cardioinhibitory mechanism during recovery.

A 35 year-old African-American man with no significant medical history presented with exertional syncope. One day while he was jogging, the patient noted a “curtain” falling over his field of view. Shortly thereafter he had a witnessed syncopal episode and fell face first onto the ground. He regained consciousness quickly without any confusion or disorientation. He went to the ER, where he denied chest pain, palpitations, dyspnea, and constitutional symptoms. An ECG showed marked T-wave abnormalities. He was advised to seek a cardiology evaluation, limit physical exertion, and was discharged from the ER. Upon following up in the cardiology clinic, his EKG showed sinus bradycardia, regular rhythm, normal axis and intervals, and deep, symmetric T-wave inversions in V1-V5 with biphasic T-waves in leads II/III/avF/V6. Given the marked T-wave abnormalities on ECG, a non-obstructive apical variant of hypertrophic cardiomyopathy was suspected. The patient was advised to limit exercise to moderate activity and a cardiac MRI was pursued that showed septal enhancement consistent with myocarditis but no evidence of hypertrophic cardiomyopathy or arrhythmogenic right ventricular cardiomyopathy. An exercise stress echocardiogram was performed next, which showed no ischemia. However approximately 2 minutes into recovery there was sudden slowing of the patient’s heart rate, and he was noted to have intermittent periods of sinus arrest with a slow ectopic escape rhythm. The patient lost consciousness during this time period and reported a prodrome of lightheadedness and nausea. Temporary pacing pads were placed and patient was in the process of getting IV access in order to give atropine. He was unconscious for a total of 2 minutes before spontaneously reverting back to normal sinus rhythm.

Exercise associated cardiac asystole (EACA) in patients without cardiovascular disease can be a fatal disorder. It is extremely rare but may explain some cases of sudden cardiac death where no underlying cause is found. It is likely the result of over-activation of the parasympathetic system. Although it is extremely rare, the incidence seems to be underreported and may be responsible for sudden cardiac death in athletes in which there is no known cause. The underestimated prevalence could be due to lack of awareness, no significant findings on diagnostic tests, and frequently fatal presentation. Increased awareness of this condition can lead to proper diagnosis. Currently, treatment is empiric, consisting of avoidance of strenuous activity, pharmacologic interventions, and pacemaker implantation. Longer follow up in patients is needed in order to form concrete guidelines in terms of management.

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A PULSATING CASE OF WORSENING CONSTIPATION AND URINARY RETENTION

First Author: Fnu Asad-ur-Rahman, MBBS Second Author: Nadia Echevarria, MD Third Author: Cheng Fang, MD  
Fourth Author: George Everett, MD

Background: Pelvic arteriovenous (AV) malformations are a rare entity in males. Mass effect or vascular accidents are their most common presentation. Our case of progressive constipation, vague lower abdominal pain and obstructive urinary symptoms was caused by a large internal iliac artery AV malformation.

Case Presentation: A 38 year old Caucasian male weighing 74 kg, without significant past history presented to the Emergency Department with progressive lower abdominal discomfort and constipation for 3 weeks. He reported concomitant urinary straining and abdominal fullness for 1 week. He was otherwise healthy but did heavy weight lifting especially leg presses of over 360 kg in the previous 4 months. A bruit was noted in the right lower quadrant and suprapubic region. Computed tomography (CT) abdomen revealed a 12.8 X 10.8 cm pelvic mass displacing the urinary bladder and compressing the colon, suspicious for pelvic aneurysm, AV fistula or AV malformation (AVM). Angiography revealed a large AV malformation from the anterior and posterior divisions of right internal iliac artery that was embolized. Subsequently, his symptoms improved dramatically. A CT Angiogram chest and magnetic resonance (MR) angiogram head/neck did not reveal other AVMs. A follow up angiogram done 2 weeks later revealed no residual flow as well as complete thrombosis of the AVM. He will have follow-up in the future with serial angiography to assess for recurrence.

Discussion: Pelvic AVMs are twice as common in females as compared to males. The underlying etiology could be congenital or acquired. Acquired causes include surgery, trauma, increased systemic estrogen or neoplasia. It may present as abdominal pain, obstructive symptoms of bowel and/or bladder, hematuria, hematochezia, deep venous thrombosis (DVT) or high-output cardiac failure. Angiography is the preferred diagnostic modality to accurately define size, vascular supply and collaterals, and allow endovascular embolization. Symptomatic/enlarging AVMs warrant urgent intervention viz. intra-arterial embolization, surgical excision or both. Follow-up angiograms are needed after treatment, since recurrence is seen in upto 50 % of cases. Although it is difficult to ascertain the exact etiology of our patient's AVM, his recent increase in lower extremity exertion could have played a part in the development or increase in size of a congenital AVM. To our knowledge, this is the first case of pelvic AVM potentially attributable to excessive lower extremity exertion.

Conclusion: Bowel and/or bladder obstruction can rarely be caused by pelvic vascular ectasia. Its rarity, vague symptoms and deep location make this entity a diagnostic challenge. Excessive lower extremity exertion can be a risk factor for the development of acquired or worsening of underlying congenital pelvic AVM.

FROIN'S SYNDROME MASQUERADING AS GUILLIAN-BARRE SYNDROME

First Author: Mohammed N Bhuiyan, MD Second Author: Saif Al Yaseen, MD Third Author: Shaun Isaac, MD

**Introduction:** Spinal Epidural Abscess (SEA) is a medical emergency that can result in permanent neurological damage and ultimately death. This disease process can be difficult to identify in early only on due to its non-specific presentation. Only a minority of patients present with a symptomatic triad of back pain, focal neurological deficits, and fever. Imaging with MRI is crucial to establish diagnosis and intervention. We report a case of spinal epidural abscess that proved to be a diagnostic challenge.

**Case:** Patient is a 57-year-old male who was initially admitted for hyperglycemic hyperosmolar state (HHS). He was found to be febrile with leukocytosis. He was transferred to the floor with broad spectrum antibiotics therapy on board, after glycemic control was achieved. Hospital day 2 patient began complaining of mild lower back pain. Blood cultures came back positive for staphylococcus aureus. Suspected sources included patients AICD and abdominal aortic graft from a previously repaired aneurysm. Trans-esophageal echocardiogram was negative for vegetations. His lower back pain progressively worsened throughout his hospital course with evolution of lower extremity weakness. Physical exam was significant for thoracic para-spinal tenderness, diminished lower extremity reflexes and strength. Suspicion of SEA was considered and CT with intravenous contrasts was ordered to evaluate the spine. MRI was contraindicated due to patient's cardiac device. The imaging study was negative, PET CT scan followed to look for sites of increased signal, which might be a sign of inflammation around an infectious source. Mild signals were detected in patient's aortic graft; clinical relevance was indeterminate. No prominent findings were noted in the spine. Complete paralysis and areflexia rapidly developed with sensory deficit to a T-9-10 level, autoimmune demyelinating syndromes were under consideration. The patient was treated empirically for Guillain-Barre' syndrome (GBS) using intravenous immunoglobulin, while results for lumbar puncture were pending. Preliminary results showed a protein of 1010 with mild pleiocytosis, normal glucose. Transverse myelitis arose as a possibility, in addition to GBS. To rule out an obstructive process CT Myelogram was ordered which displayed a blockage of CSF flow at a T-10 level. The patient was started on dexamethasone and a STAT neurosurgery consult was placed. T-10 laminectomy revealed an abscess, which was evacuated and cultured. The CSF findings of markedly elevated protein with normal glucose can be seen with impediments to CSF flow in the spinal canal, such as a tumor or abscess. Protein subsequently pools towards the lumbar region, thus lumbar punctures will display elevated protein. Xanthochromia and pleiocytosis may also be present. This phenomenon is known as Froin's syndrome.

**Discussion:** This case reinforces the necessity of focusing on the clinical picture in the face of negative tests. Spinal epidural abscesses can be difficult to diagnose, particularly in patients who cannot receive MRI. Moreover, detecting Froin's syndrome is clinically relevant.

## A POSSIBLE MECHANISM FOR SODIUM-GLUCOSE CO-TRANSPORTER 2 INHIBITOR-ASSOCIATED EUGLYCEMIC KETOACIDOSIS (EKA)

Shane Bobart MD, Benjamin Gleason MD, Jonathan Schroeder DO, Nydia Martinez MD, Keith Norris, MD, PhD, Sandra F. Williams MD

Background: Sodium-glucose co-transporter 2 (SGLT2)-inhibitors are now part of the armamentarium for the treatment of type 2 diabetes mellitus (DM). They represent an attractive option because they result in weight loss and improved glycemic control by inducing glycosuria, via inhibition of glucose re-uptake in the proximal renal tubule. These agents however, have now been implicated in over 20 cases of euglycemic ketoacidosis (EKA). Most cases reported an antecedent period of decreased oral intake and treatment with both intravenous insulin and dextrose.

Case Presentation: We report the case of a female in her mid-40's with Type 2 DM on metformin, liraglutide, and canagliflozin, presenting two days after elective cosmetic surgery with dyspnea, nausea and vomiting. She had clinical and laboratory evidence of severe ketoacidosis, associated with significant glycosuria. Notable labs were pH 7.0, beta-hydroxybutyrate 45 mg/dL, urine glucose >1500 mg/dL, serum bicarbonate 6 mmol/L and anion gap 33, despite serum glucose of only 180 mg/dL and minimally elevated serum lactate. Initial treatment included intravenous hydration and insulin infusion. However, following Endocrinology consultation insulin was discontinued approximately 3 hours after initiation when anion gap was 19-20. Intravenous fluids with dextrose were continued with subsequent complete resolution of clinical and laboratory evidence of ketoacidosis within 18-24 hours after admission. We believe that this is the first report of SGLT2-associated euglycemic ketoacidosis (EKA) treated primarily with dextrose thus supporting a mechanism unrelated to diabetic ketoacidosis.

Discussion: A precise mechanism underlying the etiology of ketoacidosis associated with these drugs has not yet been identified. Based on the observation that most reported cases of ketoacidosis with use of SGLT2 inhibitors share features of antecedent decreased oral intake, absence of severe hyperglycemia and presence of severe glycosuria, we propose that this ketoacidosis may stem from interferences with metabolic compensation as a result of the significant glycosuria induced by these agents. Excess urinary glucose wasting, likely depletes glycogen stores and has been reported to inhibit renal gluconeogenesis. Subsequent inhibition of hepatic gluconeogenesis perhaps occurs via hepato-renal cross-talk analogous to that which is postulated to occur in the hepato-renal syndrome. In the absence of available glycogen for glycogenolysis, and superimposed on an environment where gluconeogenesis is being suppressed, EKA will be triggered during periods of fasting or severe carbohydrate restriction. Clinicians should therefore exercise caution in the use of these agents during anticipated glucose deficient states in order to prevent EKA.

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A RARE FORM OF AMYLOIDOSIS IN A PATIENT WITH GOUT

Gerard Chaaya, MD, Ashwini Komarla, MD, Clifford Eng, MD, Allison Carilli, MD

The amyloidoses are a group of rare conditions characterized by extracellular deposition of various types of misfolded and insoluble proteins. In contrast with other forms of chronic inflammation, gout is not usually associated with an increased incidence of amyloidosis. We present a rare case of localized amyloidosis in a patient with chronic tophaceous gout.

A 68-year-old man with a past medical history of hypertension, diabetes mellitus type II and tophaceous gout on maintenance allopurinol and colchicine as needed for flares, presented to the clinic for a 6-month history of left second toe mass that progressively became painful due his shoe wear. A 0.8cm x 0.9cm x 0.5cm non-pulsatile, non-fluctuant, pink to yellow, waxy-appearing nodule was appreciated on the dorsolateral aspect of the second left toe just distal to the DIP. On examination, it was soft and non-tender. The mass was surgically removed and stained positive with Congo red on pathologic examination. Immunohistochemistry revealed the deposition of Amyloid P as well as immunoglobulins Kappa and Lambda light chain (Amyloid L). A workup to rule out systemic involvement of amyloidosis revealed an unremarkable complete metabolic panel and urinalysis. Complete blood count was unremarkable except for a mild normocytic anemia. Antinuclear antibody, rheumatoid factor, anti-cyclic citrullinated peptide antibody, serum and urine protein electrophoresis and echocardiogram were unremarkable. A bone marrow biopsy showed no abnormalities. As no evidence for systemic amyloidosis was detected, the final diagnosis was primary localized cutaneous nodular amyloidosis (PLCNA). This patient's gout has been moderately controlled with a uric acid of 5.6 mg/dl and a couple of flares yearly. The patient remains otherwise asymptomatic and is being followed-up closely for the development of systemic disease.

Three types of primary cutaneous amyloid are recognized. Lichen and macular amyloidoses are the most common and are associated with the deposition of amyloid in the papillary dermis. The third rarer type is PLCNA, in which the dermis, subcutis and blood vessel walls are diffusely infiltrated with amyloid L protein in the form of monoclonal immunoglobulin light chains. Treatment of symptomatic nodules includes surgical excision, cryotherapy, or electrodesiccation. It is critical to closely follow these patients for the development of systemic disease (recognized in 7 to 50% of patients). Although gout is a very common disease, the association with amyloid is rare, with eight cases being reported to date. The reasons for this uncommon association are that gout attacks are self-limiting with short inflammatory duration, the low amyloidogenic effect of the protein serum amyloid A that is increased in gout, and the widespread use of colchicine that inhibits the formation of protein AA in animal models. This case presents a rare cause for a nodular mass and the possible association with a common disease, gout.

“BLACK ESOPHAGUS” OR GURVITS SYNDROME: A RARE COMPLICATION OF DIABETIC KETOACIDOSIS.

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**Introduction:** Acute esophageal necrosis (AEN) also known as “black esophagus” or necrotizing esophagitis, is a rare syndrome characterized by a striking diffuse patchy or circumferential black appearance of the esophageal mucosa that preferentially affects the distal esophagus and terminates at the gastro-esophageal junction. Only 88 patients over a span of 40 years have received this diagnosis, and the prevalence of this disease ranges from 0.001 to 0.2% of cases in literature. It more commonly affects men (4:1 ratio) in the sixth decade of life. It is associated with a high mortality rate, approaching 32%. We present a case of AEN presenting in the setting of diabetic ketoacidosis (DKA), affecting both the proximal and distal esophagus.

**Case Report:** A 65-year-old male presented to the hospital with altered mental status, confusion, agitation and psychosis. He was found to have blood glucose of 989 with positive serum acetones, an anion gap of 30 and was diagnosed with DKA. He was started on insulin infusion and supportive care. On day 3, he developed coffee ground emesis and melena with an associated hemoglobin drop from 10.5 to 7.9. Emergent esophagogastroduodenoscopy (EGD) was done which showed circumferential black and necrotic mucosa in the cervical esophagus. Consequently, the scope was withdrawn for fear of perforation, and biopsies were not obtained. The patient was kept nil-per-os (NPO), started on nasogastric (NG) suction and IV proton pump inhibitors (PPI). His DKA resolved and he was weaned off the insulin drip with closure of the anion gap. On repeat EGD three days later, the endoscopic appearance was greatly improved, with only patchy areas of residual necrosis and diffuses ulceration, without any stricture or stenosis. No history of caustic ingestion was documented.

**Discussion:** Our case illustrates Gurvits Syndrome in the setting of DKA. A history of diabetes mellitus (24%), malignancy (20%), hypertension (20%), alcohol abuse (10%) and coronary artery disease (9%) places patients at risk of developing AEN. Clinically, AEN can present with upper gastrointestinal bleeding, epigastric/abdominal pain, vomiting, dysphagia, fever, nausea, and syncope. AEN may arise in the setting of multi organ dysfunction, poor perfusion, sepsis, DKA, alcohol intoxication, gastric volvulus, traumatic transection of the thoracic aorta, thromboembolic phenomena and malignancy. Management of this condition comprises of treating the underlying etiology, maintaining hemodynamic stability, NPO, intravenous proton pump inhibitors, fluoroscopically guided NG tube placement and blood transfusions as needed. Complications include esophageal perforation, mediastinal abscess, stricture formation and death. The present case serves to illustrate the point that upon endoscopic examination, as with caustic ingestion, the presence of circumferential necrosis should prompt immediate withdrawal of the scope, in order to avoid esophageal perforation. This is one of the few known indications for immediate scope withdrawal and termination of the EGD study.

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DOOMED TO REPEAT: AN INTERESTING CASE OF A MODERN DISEASE PRECIPITATING A HISTORICAL ONE

First Author: Edwin Hayes, MD Prakhar Vijayvargiya, MD Teresita Casanova, MD

Tetanus, while rare in developed nations, remains an important disease process where mortality and morbidity are critically linked to prevention tactics. This case underlines the importance of prophylaxis and a new indication worth considering for immunoglobulin administration.

A 64-year old Haitian man with peripheral neuropathy from diabetes was admitted to the hospital for 1 week of progressively worsening swelling and redness of the right great toe associated with fever. He could not recall a precipitating traumatic event or prior tetanus vaccination. On examination he had 2 x 2 cm wound on the plantar surface of the right big toe with purulent drainage extending to the deep fascia. Blood work revealed leukocytosis of 15,100/mm<sup>3</sup>. X-ray of the right foot showed subcutaneous gas with extension to the first metatarsophalangeal joint. Empirical vancomycin, piperacillin-tazobactam, clindamycin and tetanus vaccine were administered. Patient underwent immediate amputation of the hallux and metatarsal debridement. Through the night he experienced progressive difficulty opening his mouth. The following morning, he developed severe stiffness in the neck and back. It was confirmed that neuroleptic medication was not administered during the amputation. Tetanus was suspected prompting neurology and infectious diseases consults. Clindamycin was switched to metronidazole and patient received 500mg IM tetanus immunoglobulin. By this point, he could only open his mouth by 2cm and developed risus sardonicus and opisthotonus. Dantrolene and IV diazepam provided immediate improvement but he required intubation for airway protection and eventually received midazolam and propofol drips for long-term sedation. After two weeks, the patient developed pulseless electrical activity requiring resuscitation. Subsequent neurological exam suggested anoxic brain injury. Patient's family decided to withdrawal life support and he passed away/expired subsequently.

CDC guidelines suggest immunoglobulin for an uncertain or inadequate history of tetanus vaccination with a wound that is not considered clean or minor, including "contaminated with dirt, feces, soil, and saliva; puncture wounds; wounds from crushing, tears, burns, and frostbite". Our patient exhibited subcutaneous gas, often manifested by clostridium species, and we propose all wounds with subcutaneous gas be considered for immunoglobulin administration. Concerning dosage, classical literature suggests 3000-6000 units, but more recent literature suggests IM 500 units are adequate. A retrospective study in JAMA of 545 cases of tetanus from 1965 through 1971 showed equal effectiveness between 500, 3000, or 10,000 units.

In an age of growing diabetes and subsequent foot infections, when the value of vaccinations are questioned by the general public, cases such as this demonstrate the importance of judicious prophylaxis for diseases that are often considered historical.

## A LITTLE HICCUP

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A 61 year-old male with a history of gastroparesis, chronic obstructive pulmonary disease, and mononeuritis multiplex on chronic steroids and bi-weekly intravenous immunoglobulin had been diagnosed with three large chains of esophageal varices during a diagnostic esophagogastroduodenoscopy (EGD) for abdominal pain. At the time he had undergone placement of 5 bands and was being seen again, now three months later, for repeated EGD with planned banding. The three large chains of esophageal varices were identified again and a single band was fired. The patient had almost immediately developed endoscopically confirmed complete distal esophageal occlusion. Visualized liquids and secretions were unable to pass this area. The EGD was subsequently aborted. Upon being aroused from procedural sedation the patient reported substernal chest pain, hiccups and inability to swallow his secretions. His vital signs were within normal limits with no oxygen desaturation, tachycardia or increase in respiratory rate. Physical exam was remarkable only for bilateral thenar atrophy and mild loss of pain sensation in lower extremities attributed to his mononeuritis multiplex. Chest x-ray and EKG did not reveal any acute cardio-pulmonary disease process or evidence of esophageal perforation. The patient was subsequently admitted while awaiting resolution of the occlusion by expected necrosis of the banded tissue.

The patient was initially managed with strict null per os, intravenous fluids, pain control and anti-emetics, but continued to have severe hiccups and periodic substernal, self described spasms as well as complete intolerance of his secretions. After a few days of unsuccessful conservative management, he underwent a barium esophagram which showed no improvement of his obstruction. Despite conservative measures and the addition of sublingual nitroglycerin, symptoms did not abate. Intravenous glucagon at 4mg every four hours was initiated, resulting in improvement in hiccups and spasmodic chest pain. By day seven, he was able to manage his secretions. Repeat barium esophagram demonstrated complete resolution of the obstruction. He was able to be discharged home on a regular diet.

This case demonstrates a very rare yet clinically significant complication of esophageal variceal ligation, associated with significant morbidity. There are only four previously reported cases of complete esophageal occlusion after endoscopic variceal ligation, despite common utilization of this procedure. The rarity of this condition leads to limited therapeutic strategies. Previous reports propose pain control and watchful waiting with reassessment for resolution of dysphagia and odynophagia. We propose intravenous glucagon, a therapy previously used for food bolus impaction due to its effects on reducing peristaltic contraction of the mid and distal esophagus, as a potential therapeutic tool for symptom control in complete esophageal occlusion following esophageal variceal ligation.

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METRONIDAZOLE INDUCED ENCEPHALOPATHY: A RARE CASE

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Metronidazole is a commonly used antibiotic with activity against anaerobes and protozoa that easily penetrates the central nervous system. Metronidazole induced encephalopathy (MIE) is a very rare toxic encephalopathy that poses a diagnostic challenge because of its rarity and characteristic MRI features often cinch the diagnosis.

A 51 year old female with a past history of alcoholic cirrhosis was diagnosed with a right frontal brain abscess and was managed medically with Vancomycin (dosed to therapeutic troughs), Ceftriaxone (2g daily) and Metronidazole (500 mg three times daily). Brain MRI at 15 days showed interval reduction in abscess size and surrounding edema. However at 27 days of treatment, she presented with acute onset of ataxia and dysarthria. She was afebrile, anicteric and with mild ascites. There was no asterix and she was oriented to all spheres with intact recall. Neurological examination was significant for marked horizontal nystagmus, postural instability, mild dysarthria and ataxia. Laboratory studies were consistent with her baseline biochemical features of hepatic cirrhosis, her MELD was 19 and notably there was no leucocytosis or hyperammonemia. Her metronidazole level was 110 mcg/ml. Repeat brain MRI was conducted to exclude brain abscess progression or a new process. It showed increased signal intensity in the dentate nuclei bilaterally on T2-weighted images obtained with FLAIR and near resolution of the right frontal abscess. At that point her diagnosis was very unclear and a literature review of these imaging findings narrowed the differential to MIE versus Wernicke's encephalopathy (WE). However, in addition to her lack of confusion she had quit alcohol use 4 months prior and was compliant with thiamin supplements; making WE unlikely. Discontinuation of metronidazole resulted in the improvement of the dysarthria and ataxia over a period of approximately two weeks. Clindamycin was substituted for metronidazole to complete therapy.

MIE appears to not be dose or duration dependent and is most times reversible with drug cessation. The mechanisms that underlie the neuronal toxicity remain unclear. T2 hyperintense lesions of the dentate nuclei on MRI are classic, but can occur in other conditions such as WE. The frequency with which metronidazole is used makes it imperative that clinicians be aware of this unusual adverse effect. Moreover this drug usually reaches a peak of 12 mcg /ml, 2 hours after a 500mg oral dose. This is significantly lower than our patient's level of 110mcg/ml drawn almost 24hours after her last dose. This may be largely due to the fact that the drug is metabolized in the liver and therefore caution should be exercised in its administration to patients with hepatic dysfunction.

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ACUTE PANCREATITIS AS A RARE INITIAL PRESENTATION OF GRANULOMATOSIS WITH POLYANGIITIS

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Granulomatosis with polyangiitis (GPA) is a systemic vasculitis with prominent reno- pulmonary manifestations that very rarely presents with involvement of other organ systems. There have been only four previous reports of acute pancreatitis as the first presentation of GPA.

A 54 year old previously healthy male presented with severe epigastric pain and vomiting. He denied any alcohol use. There was mild epigastric tenderness on exam. Initial investigations revealed WBC of  $13.3 \times 10^9$  /ml (normal:  $4.5-11 \times 10^9$  /ml) and lipase of 1045 U/L (normal: 0-60 U/L). Hepatic and kidney profiles as well as other hematological parameters were normal. An abdominal CT scan showed a diffusely enlarged pancreas with surrounding inflammatory changes. Abdominal sonography was unremarkable. A diagnosis of acute pancreatitis of unknown etiology was made. He recovered with supportive care and was discharged.

Over the following 5 months, he saw several specialists for seemingly unrelated concerns. He first visited an otolaryngologist for persistent sinusitis and serous otitis media of his right ear. He then started experiencing frequent spells of severe left ankle and hip pain and was seen by a rheumatologist. There was no objective evidence of synovitis and investigations for rheumatoid and viral arthritis were negative. He also developed paresthesia of his left leg and right hand for which he was referred to a neurologist and nerve conduction studies were inconclusive. It was also noted that he had a normocytic anemia with Hemoglobin 9 g/dl (normal: 11-14 g/dl). He was evaluated by a Hematologist who ruled out iron or vitamin deficiencies. A bone marrow biopsy was done which was normocellular and negative for malignancy. He was also observed to have declining renal function with BUN 8.9 mmol/l (normal: 1.8-7.1mmol/l) and Cr 208  $\mu$ mol/l (normal: 60-110  $\mu$ mol/l), a high c- ANCA (titer 1:320) and proteinase - 3 (60 U/ml; normal: 0-3.5 U/ml). At this time he was re- admitted for an elective renal biopsy which showed pauci- immune glomerulonephritis in keeping with GPA. He was given a three dose course of pulse steroids followed by oral prednisone and cyclophosphamide.

Over the 5 months following his presentation with acute pancreatitis, our patient developed multi system complications that ultimately led to a unifying diagnosis of GPA. Similar to previous reports, the diagnosis was initially elusive and the patient had a rapidly progressive course. The pathophysiology is thought to be part of the underlying systemic vasculitis and there is no standard recommended treatment. Clinicians must therefore be aware of this rare, but serious entity and remain vigilant in cases of pancreatitis when the etiology is unclear.

PRIMARY SJÖGREN'S SYNDROME-ASSOCIATED NONSPECIFIC INTERSTITIAL PNEUMONIA WITH CYSTIC FORMATION: AN UNUSUAL PRESENTATION

First Author: Maria I Linares, MD Second author: Ehimen Aneni, MD Last author : Ari Ciment, MD

**INTRODUCTION** Primary Sjögren's syndrome (PSS) is a chronic inflammatory autoimmune disease. Interstitial lung disease (ILD) can be an extraglandular complication and patients are typically screened to rule out ILD. The ILD in such cases usually precedes the diagnosis of PSS. We present an unusual presentation of biopsy proven Nonspecific interstitial pneumonia (NSIP) along with cystic formation in a patient with serologic evidence of Sjogren's syndrome.

**CASE DESCRIPTION** 27 year-old previously healthy African-American female presented with indolent fever, breathlessness and dry cough for one year. She had been treated for recurrent pneumonia with antibiotics and reported transient improvement. One month prior to her admission, her dyspnea worsened and she started to experience intermittent left hip pain. She denied rashes, photosensitivity, sicca symptoms, Raynaud's phenomenon, weakness, recent travels, exposure to airborne organic antigens, smoking or family history of lung disease. Physical exam disclosed fever, tachycardia, mild hypoxia, lungs with diffuse rales and wheezing. Severe clubbing was noted in hands and feet. She had pain on motion of her left hip. Blood work showed mild pancytopenia, elevated ESR and CRP. High resolution computed tomography (HRCT) revealed diffuse centrilobular ground glass opacities with traction bronchiectasis, numerous pneumatoceles and mediastinal-hilar lymphadenopathy. BAL was negative for infectious process. HIV testing was negative. Further workup revealed positive Rheumatoid factor, ANA and Anti-Ro/SSA antibodies. Serum ACE, aldolase, creatinine phosphokinase and complement were normal. Histology from VATS revealed NSIP with a combination of cellular and fibrosing features and a plethora of noninfectious cysts. She was treated with systemic steroids with impressive symptomatic improvement within the next few weeks.

**DISCUSSION** NSIP is the most common type of ILD secondary to connective tissue disease (CTD). NSIP could be an early manifestation of latent CTD and many times, it can actually precede the diagnosis of CTD as seen in our case. Our patient had some of the key features for NSIP on HRCT including bilateral, symmetric ground glass opacities but had a preponderance of pneumatoceles which are not typical. In previous studies, about 10% of Sjögren's syndrome patients have cystic lesions in their lungs. A ball-valve mechanism related to stenosis of the bronchioles and fragile alveolar walls due to severe infiltration of inflammatory cells or amyloid deposits is postulated to account for such cystic PSS lung disease. Our patient's pancytopenia, arthralgia coupled with positive Anti-Ro/SSA antibody favored the PSS-associated NSIP diagnosis. Treatment with corticosteroids is recommended to hinder progression of the disease and can have a dramatic response as seen in this case.

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## THE GEM THAT CITO-LYSED: A RARE COMPLICATION OF GEMCITABINE

First Author: Alvina Munaf MD; Sameen Khalid MD; George Everett MD; Ahmed Zakari MD.

**Introduction:** Hemolytic Uremic Syndrome(HUS) includes the classic triad of microangiopathic hemolytic anemia, thrombocytopenia and acute renal failure. Unlike children with HUS where 90% of the cases are preceded by diarrheal illness, adults with HUS have a variety of etiologies. Cytotoxic drugs including Gemcitabine, a nucleoside analogue, have also been implicated in causing HUS. Gemcitabine has shown promising results in a wide range of malignancies. The incidence of Gemcitabine-induced HUS (GiHUS) is reported to be between 0.02 and 1.2% but the mortality is as high as 50-60%. We report a case of GiHUS in our institution to heighten the awareness of this potentially lethal complication of Gemcitabine.

**Case:** 66-year-old male patient with past history of pancreatic adenocarcinoma and hypertension presented for nausea, vomiting and generalized weakness. Pancreatic adenocarcinoma was suspected from the presence of 1.7 cm lesion in head of pancreas on magnetic resonance cholangiography and confirmed with endoscopic biopsy a year earlier. He was not a surgical candidate and was treated palliatively with 9 cycles of Gemcitabine and Paclitaxel. The dose of Gemcitabine was 2000 mg initially but reduced to 1400 mg from cycle 6th due to cytopenias. Physical Examination was normal other than epigastric tenderness. Laboratory investigations showed WBC of 9500/uL, Hb 6.1g/dL, Platelets 69,000/uL and creatinine 1.7mg/dL (baseline 0.6) with FENA 0.38%. Renal ultrasound didn't show any obstruction. Urinalysis was positive for 1+blood and 1+albumin. Over the next few days even with aggressive IV fluids, blood and platelets, the anemia, thrombocytopenia and renal failure worsened. Serum LDH was found to be elevated to 1700u/L, retic count was 7.8% and peripheral smear showed microcytic anemia with frequent schistocytes consistent with a microangiopathic hemolytic process leading to a diagnosis of GiHUS. No other potential causes were identified. Plasmapheresis was offered but patient declined further treatment and opted hospice care.

**Discussion:** The pathogenesis of GiHUS is not well-understood but damage to vascular endothelium directly or indirectly through complement activation is a likely cause. The mean duration between initiation of Gemcitabine and onset of HUS has been reported to be 7.4 months. In some cases discontinuation of the offending agent resulted in remission while other patients required plasma exchange, hemodialysis, steroids and immunosuppressive therapies, but despite aggressive care this complication proves to be fatal in the majority.

GiHUS is rare but a serious complication of Gemcitabine. As anemia and thrombocytopenia are frequently attributed to myelosuppression in patients on antineoplastics, we recommend high index of suspicion and close monitoring of kidney function in patients on Gemcitabine. Early detection of HUS is crucial so that the causative agent can be stopped promptly.

## UNCOVERING MEGACE INDUCED ADRENAL INSUFFICIENCY

First Author: Sweta Shah, MD Matthew Snyder, PharmD Christy Thai, PharmD, BCPS Sowmya Nanjappa, MD

**Case Presentation:** A 65-year old male with hypertension and stage IV clear cell renal cell carcinoma (RCC) with metastases to the brain, lung, bones, and pancreas was admitted for evaluation of a recent fall. Surgical history included a left nephrectomy and adrenalectomy. He was started on therapy with axitinib 1 month prior to admission. Upon evaluation, patient was found to have altered mental status and hypotension with a blood pressure of 71/49 mmHg. After ruling out potential etiologies, including dehydration, electrolyte imbalances, new brain metastasis, and hypothyroidism, a workup for adrenal insufficiency was performed. Morning serum cortisol was  $<1.0$   $\mu\text{g/dL}$ . A cosyntropin stimulation test revealed serum cortisol levels of  $5.9$   $\mu\text{g/dL}$  and  $8.9$   $\mu\text{g/dL}$  at 30 and 60 minutes, respectively, with a pre-test plasma adrenocorticotropic hormone (ACTH) of  $<5$   $\text{pg/mL}$ , indicating secondary adrenal insufficiency. After further review of the patient's medication list, it was noted that he was recently prescribed megestrol (megace) 1 month ago. This was thought to be the likely culprit of adrenal insufficiency, and the medication was discontinued. Oral hydrocortisone 20 mg every morning and 10 mg every evening was initiated, and the patient's blood pressure significantly improved. Treatment with steroids was completed 3 months post-discharge. At that time, repeat serum cortisol and ACTH were  $20$   $\mu\text{g/dL}$  and  $68$   $\text{pg/mL}$ , respectively, thus confirming the resolution of megestrol induced adrenal insufficiency.

**Discussion:** Adrenal insufficiency is a serious, life-threatening disorder that stems from primary adrenal failure or secondary adrenal disease owing to impairment of the hypothalamic-pituitary-adrenal (HPA) axis. The origin of our patient's disorder was clouded by several atypical factors, including prior removal of his left adrenal gland, tyrosine kinase inhibitor use, and metastatic RCC. Megestrol, a synthetic progestin with anti-estrogenic properties, is often used to help cachectic cancer patients increase their appetite and gain weight. It is thought to cause suppression of the HPA axis at the hypothalamus through negative feedback, resulting in low serum cortisol and plasma ACTH levels. While megestrol induced appetite stimulation is useful in patients like ours, this benefit is often outweighed by risks such as thrombosis, hyperglycemia, osteoporosis, hypogonadism, and secondary adrenal insufficiency.

**Conclusions:** Although adrenal insufficiency is a reported potential adverse effect of megestrol, it is not well recognized in clinical practice. Given the potentially severe complications of adrenal insufficiency, this case underscores the importance of recognizing the possibility of endocrine complications induced by megestrol. Questions regarding the timeframe for HPA-axis recovery after discontinuation of megestrol are still unknown. However, our case describes a patient who successfully recovered 3 months after discontinuation of the medication.

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## COCOON IN ABDOMEN

First Author: Olayinka Afolabi, MD Second Author: Rajan Kapoor, MD

**Case:** A 49 y/o male with history of hypertension, end stage renal disease due to bilateral nephrectomies in 1989 when he was diagnosed with bilateral renal cell cancer. He was started on peritoneal dialysis (PD) in 1989 up until 1990 when he received his first kidney transplant. He lost this allograft in 1995 but fortunately received his second renal transplant that year. His second transplant failed in 2002 and he was placed back on PD. In 2006, the patient presented with syncope along with weight loss, generalized edema, and fatigue. He was found to have PD catheter related peritonitis with staphylococcus aureus. The PD catheter was removed and intermittent hemodialysis (IHD) was initiated. He recovered after this prolonged hospitalization and was discharged on IHD as he had thickened peritoneum which was not amenable to PD at that time. Soon after recovering from his peritonitis, he had severe malnutrition and weight loss with multiple episodes of small bowel obstructions (SBO) requiring hospitalizations up to 30 times that year. An open laparotomy showed multiple bowel strictures and adhesions and a biopsy of the peritoneum confirmed diagnosis of Sclerosing Encapsulating Peritonitis (SEP). The patient was started on Tamoxifen which decreased the frequency of his SBOs. Currently, 9 years after his diagnosis he has regained his weight and the frequency of SBO episodes have significantly reduced to 3-4 per year, and each hospitalization with SBO episodes recovers with conservative management only.

**Discussion:** SEP is a rare but serious complication of PD and is associated with high morbidity and mortality. The mortality rate, which can be as high as 56%, is related to malnutrition and sepsis. Prevalence estimates vary between 0.5% and 7.3% but may be as high as 17.2% in those patients on PD for 15 or more years. The features of SEP include extensive intraperitoneal fibrosis and encasement of bowel loops referred to as “cocooning”. It results from underlying pathogenic processes including inflammation and peritoneal adhesions. SEP is an insidious and gradual syndrome. It can present with non-specific symptoms of abdominal pain, nausea, constipation, ascites, weight loss and loss of dialysis adequacy. The diagnosis should be confirmed with radiologic imaging in patients with a long history of PD, signs and symptoms consistent with SEP.

**Conclusion:** SEP is a serious life threatening complication of PD. Clinicians need to make prompt diagnosis and obtain surgical referral in suspicious cases. Herein, we present a rare case of SEP with long term survival and clinical improvement with tamoxifen.

**AUTOIMMUNE HEPATITIS: AN UNCOMMON PRESENTATION WITH RAPID PROGRESSION AND DETRIMENTAL SEQUELAE.**

First Author: Venkata Sai Gogineni, MD Second Author: Hayley Walter, MD

**Introduction:** Autoimmune hepatitis is a generally progressive, chronic condition that has a marked variability in its clinical course and presentation. Early recognition of clinical manifestations can help to anticipate complications and initiate the appropriate treatments. We present a patient with autoimmune hepatitis who unfortunately falls in a rare end of the spectrum of this disease who had significant barriers to healthcare.

**Case Presentation:** A 35-year-old obese, Hispanic female with no past medical history presents to the Emergency Department with complaints of worsening tea-colored urine, jaundice, yellow eyes, nausea and persistent vomiting for the past several weeks. Patient admits that she experienced the same symptoms in 2011, during which she was treated for acute EBV infection with underlying autoimmune hepatitis. The patient was lost to follow up with Gastroenterology for outpatient management of her newly diagnosed autoimmune condition. Now, she presents with recurrence of similar symptoms. The laboratory workup identified elevated transaminases and total bilirubin, in addition to elevated prothrombin time and INR. Computed tomography (CT) abdomen identified a mildly cirrhotic liver. Lab findings were consistent with autoimmune hepatitis. Hepatitis A, B, C serologies were negative. Daily corticosteroid therapy was initiated, however, PT/INR continued to rise. Discussions about liver transplantation were initiated while she attempted to obtain emergent health insurance. She would not be considered a transplant candidate without health insurance in place.

Within a short time span, she developed acute hypoxemic respiratory failure. The patient was promptly intubated and transferred to the intensive care unit. She subsequently had a complex course of severe sepsis with fungemia, fulminant liver failure, and cerebral edema. Transport to liver transplant center was declined. The patient expired due to septic shock and respiratory failure.

LABS	Admission 09/2011	Admission 09/2015	Admission 10/2015
AST	1,550 IU/L	1128 IU/L	127 IU/L
ALT	1,211 IU/L	533 IU/L	146 IU/L
Total Bili	9.9 mg/dl	10.9 mg/dl	12.6 mg/dl
INR	1.41	3.1	2.76
Anti- Smooth muscle Ab	1:80	1:20	---
F- Actin IgG Smooth Muscle Ab	68	70	---

Anti mitochondrial Ab IgG	---	124.1 units	---
Anti-LKM-1	---	Negative	

Table 1: Laboratory findings on admission

PT sec	30.1	31.3	32.4	34.2	37.6	38.1	27.7*	32.8
INR	3.03	3.19	3.34	3.59	4.06	4.13	2.72*	3.40

Table 2: PT/INR rising trend despite starting prednisone therapy. Note the \* denotes the values measured after administration of FFP and Vitamin K.

**Discussion:** Unlike the more common subclinical nature of autoimmune hepatitis, this patient’s rapid decline and presentation with acute liver failure marks an uncommon but very possible state of clinical manifestation. The patient’s barriers to medical therapy along with an acutely rising PT/INR indicated a very poor prognosis. It was evident that her state of condition could only benefit from a liver transplantation. However due to her late and abrupt onset of encephalopathy and cerebral edema, this intervention was no longer an option. It is also important to recognize the seriousness of the patient’s failure to follow up for treatment of her condition. The course of her disease could have very well taken a different pathway if she had adhered to medical advice and barriers to health care were overcome. This case emphasizes the importance of the physician to educate patients about the seriousness of their disease and importance of acquiring health insurance, even in young, seemingly healthy patients.

## COSTOCHONDWRONGUS

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**Introduction:** The SAPHO syndrome (synovitis, acne, pustulosis, hyperostosis, osteitis) is a rare, although likely under recognized, chronic inflammatory condition related to psoriatic arthritis. Because the signs and symptoms of SAPHO syndrome are nonspecific, the diagnosis can be challenging. We present a case of a young man who underwent numerous evaluations over the course of five years before the diagnosis of SAPHO was made.

**Case Presentation:** A 30-year old man was referred to rheumatology clinic for evaluation of neck, shoulder and anterior chest wall pain present of 5 years duration. He reported a sharp sternal pain that was aggravated by movement with associated intermittent swelling and warmth of the area. The pain had persisted despite numerous courses of non-steroidal anti-inflammatories (NSAIDs) medications and was beginning to interfere with his daily life. He had a normal dermatologic exam and inflammatory markers were within normal limits. Computed tomography (CT) scan of the chest was obtained and revealed a 16 mm round lucency with irregular margins at the sternomanubrial joint. Magnetic resonance imaging (MRI) of the chest confirmed the presence of a soft tissue density between the sternum and the manubrium which enhanced with administration of gadolinium. Subsequent biopsy demonstrated dense fibroconnective tissue without evidence of infection or malignancy. Although the patient had no evidence of skin involvement, the patient's osteoarticular findings satisfied diagnostic criteria for SAPHO syndrome. Treatment with a tumor necrosis (TNF)- $\alpha$  inhibitor was initiated given failure of NSAIDs to control his symptoms.

**Discussion:** The SAPHO syndrome is a rare inflammatory musculoskeletal disorder of unknown etiology with an estimated prevalence of 0.04%. The syndrome is characterized by hyperostosis and inflammatory osteitis of the axial skeleton, most commonly the anterior chest wall. It is associated with a wide spectrum of neutrophilic skin lesions. Because skin findings may be mild or absent, as in our case, the diagnosis may not be considered and therefore delayed. Diagnosis is based on appropriate clinical and radiological findings. Recognition of SAPHO syndrome is important in order to avoid unnecessary testing, prolonged antibiotic treatment and invasive procedures.

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AMAUROSIS FUGAX AS THE PRESENTING SYMPTOM OF A LEFT ATRIAL MYXOMA

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**Background:** Amaurosis fugax and other neurological signs and symptoms are rare manifestations of left atrial (LA) myxomas. Though heart failure symptoms most commonly present with LA myxomas, embolic events resulting from these tumors can lead to serious CNS complications in a small subset of patients.

**Case:** A 48 year old male with past medical history of hypertension and tobacco use was referred for a left ventricle (LV) thrombus found on echocardiogram during work up for transient episodes of vision loss. The patient had multiple episodes of unilateral vision loss for 1 week which lasted 30 minutes each episode before resolving. Six months previously, he experienced chest tightness associated with nausea and vomiting for which he never sought medical attention. Cardiac MRI revealed a multi-lobed mass measuring 4 sq cm in the LA. He exhibited no signs or symptoms of heart failure or secondary pulmonary hypertension, but cardiac echo showed an ejection fraction of <20% with severe global LV dysfunction. He was diagnosed with amaurosis fugax secondary to embolic events from the mass. Surgical resection and pathology yielded the diagnosis of atrial myxoma.

**Discussion:** Myxomas are the most common primary cardiac neoplasm, and the majority of them arise in the LA. These benign tumors vary widely in size and weight. A proportion of myxomas can present with signs of systemic emboli as a result of their friable or villous nature.

The most common and classic presentation of LA myxoma includes symptoms of mitral valve obstruction and heart failure. Constitutional symptoms such as fever and weight loss can also be seen. Neurological symptoms are the least common presentation of atrial myxomas and are a result of tumor embolization. In a Mayo clinic retrospective review, only 12% of a series of patients with myxomas exhibited neurological symptoms, the majority of which manifested CNS deficits as the initial presentation of myxoma. Limited case studies have described vasculitis-like presentations of atrial myxomas, while others have described retinal artery occlusion or ischemic stroke as initial presentations. Amaurosis fugax (or transient monocular visual loss) without evidence of other neurological signs such as syncope or focal weakness have rarely been described to date as an initial presentation of an atrial myxoma.

We are describing a case of amaurosis fugax as a presenting symptom of LA myxoma in a patient without heart failure or constitutional signs. Neurologic or ophthalmologic manifestations of myxomas are rare but serious complications and should promptly be investigated with echocardiography or cardiac MRI. Imaging studies are generally sufficient for a presumptive diagnosis of myxoma and surgical resection is the definitive treatment. The need for prompt recognition and treatment of LA myxoma is highlighted in this study in order to prevent potentially fatal neurologic complications.

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A TALE OF A TUBE AND A POT- A CASE OF TAKOTSUBO CARDIOMYOPATHY OCCURRING AFTER MRI

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Takotsubo Cardiomyopathy (TCM) is stress induced cardiac dysfunction which is being increasingly recognized in patients with physical or neuropsychological stress. This cardiomyopathy receives its name from a Japanese word '*takotsubo*' which translates to "octopus pot," resembling the shape of the left ventricle during systole on imaging studies. Here we present a case of TCM occurring in a patient after MRI which is a first reported case of its kind in literature.

A 59 year-old Caucasian female with past history of hypothyroidism, hypertension and coronary artery disease came to emergency room (ER) with complaints of chest pain. This patient had undergone an MRI of lumbar spine for her lower back pain about 4 hours earlier. She said that she is mildly claustrophobic but before her MRI she took an over the counter sleep medication and "slept well in the tube while getting MRI". While coming back home she started having these chest pain which did not go away even after taking sublingual nitroglycerine. It is then that she decided to come to ER. On exam, patient's vitals were stable and physical examination was unremarkable. Cardiac troponins were found to be elevated at the level of 0.766 ng/mL which trended down over next 24 hrs. Electrocardiogram at admission revealed normal sinus rhythm with diffuse T-wave inversions in limb leads.

Urgent transthoracic echocardiogram demonstrated normal size left ventricle (LV) with mild concentric LV hypertrophy. LV systolic function was moderately to severely reduced with ejection fraction of 25-30%. There was akinesis of LV apical and anterior wall and severe hypokinesis of septal wall. Patient also underwent urgent coronary angiogram which revealed patent coronary arteries and left ventriculogram which showed regional apical wall motion abnormalities suggestive of Takotsubo cardiomyopathy (TCM). Patient was later discharged home on beta blocker and Angiotensin Converting Enzyme Inhibitor. A repeat ECHO after 2 weeks showed improved LV ejection fraction with no apical wall motion abnormalities.

TCM mimics acute coronary syndrome at the time of presentation. Although, cardiac dysfunction in TCM is typically transient and reversible but patients usually undergo cardiac work-up and interventions like ECHO and coronary angiogram. Pathogenic mechanism of TCM is still unclear but catecholamine release and sympathetic hyperactivity has been considered as one of the most likely mechanisms. Prognosis is usually excellent but hypotension and ventricular arrhythmia have been described in some of the patients with TCM.

Our case illustrates that transient cardiac dysfunction can occur even with minimal psychological and emotional stress. Physicians should have high suspicion for TCM in patients presenting with signs and symptoms of acute coronary syndrome after emotional stress which could avoid unnecessary cardiac work-up in these patients.

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TRIMETHOPRIM-SULFAMETHOXAZOLE-INDUCED RHABDOMYOLYSIS IN AN IMMUNOCOMPETENT PATIENT

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**Case:** A 64-year old African American female with bipolar disease, borderline personality disorder, hypertension, and gout was admitted to the Psychiatry unit for cognitive evaluation. On admission, she was started on trimethoprim-sulfamethoxazole (TMP-SMX) for a urinary tract infection (first day of TMP-SMX = Day 1). Her creatine phosphokinase (CPK) was initially 192 U/L but was rechecked on Day 5 when new muscle rigidity was observed, at which point it had increased to 2,586. She was transferred to Medicine due to concern for neuroleptic malignant syndrome (NMS). She did not report muscle aches but had a hypoactive demeanor despite being hyperactive and occasionally psychotic at baseline. Physical examination revealed normothermia and only minimal upper extremity rigidity. Her CPK peaked at 26,404 on Day 6. Her creatinine also increased from her baseline of 1.0 to 1.3. A diagnosis of rhabdomyolysis was made. Maintenance IV fluids were started, and TMP-SMX was discontinued with resulting resolution of elevated CPK and acute renal failure. A return to baseline demeanor was also noted, as she became more conversant and had occasional psychotic episodes. Given that the patient exhibited neither the hallmarks of NMS nor the classic triggers for rhabdomyolysis, she was believed to have developed TMP-SMX-induced rhabdomyolysis.

**Discussion:** Rhabdomyolysis is a severe and potentially fatal condition involving muscle necrosis and significant CPK elevations and is associated with a worse prognosis if diagnosis is delayed and/or acute renal failure is present. Statins are a frequently documented cause of medication-induced rhabdomyolysis. However, rhabdomyolysis secondary to TMP-SMX is not a well-known phenomenon, with only a handful of case reports documenting its occurrence. The majority of these case reports were in patients in immunocompromised states, namely HIV/AIDS or allogeneic stem cell transplants. This is only the third case report of TMP-SMX-induced rhabdomyolysis in an immunocompetent patient, in which rhabdomyolysis occurred within 5 days of antibiotic usage. Given that TMP-SMX is an inexpensive and frequently utilized medication both in the inpatient and outpatient setting, a heightened awareness of the possibility for this serious complication to occur in the general population is warranted.

PSEUDO-SEPTIC ARTHRITIS OF THE HIP: AN UNCOMMON PRESENTATION OF A COMMON RHEUMATIC DISEASE.

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A 31 year old female, with a history of infertility, reported arthralgias and morning stiffness in her bilateral elbows and wrists for three months. This started at roughly the same time of her initiating infertility treatments. She presented however with acute left hip pain to our emergency facility. Examination revealed severe tenderness to the left hip. She had a white cell count of  $14.7 \times 10^9/L$ . Her CRP was 0.85 mg/dL (0-0.5) and ESR was 55 mm/hr (0-20). Plain radiographs were unremarkable and she was treated symptomatically. She returned the following day with worsening pain. There was extreme left hip tenderness and limitation in range of motion. Blood cultures and STD screening were negative. Her WBC was  $13.7 \times 10^9/L$ , ESR 68 mm/hr, and CRP 10.6 mg/dL. An MRI demonstrated left hip joint effusion, with enhancement suggestive of infection. Orthopedics proceeded with incision and drainage for suspected septic arthritis. Hip washout showed frank pus. Synovial fluid analysis showed a cell count of 45,955 with negative cultures, and she was treated empirically with Vancomycin and Ceftriaxone for two weeks. Seven weeks later, the patient presented again acutely with two days of moderate aching contralateral hip pain, reminiscent of her previous presentation of the left hip. She had significant tenderness to active and passive range of motion without erythema or warmth. Her WBC was  $9.9 \times 10^9/L$ , ESR 58 mm/hr, and CRP 2.3 mg/dL. Plain radiographs were negative. Arthrocentesis demonstrated frank pus with a cell count of 40,942. Cultures and crystals were unremarkable. Lyme and ANA serology were negative. Interestingly, RF and the IgA/IgG CCP serology were positive at 978.9 IU/mL and >250 EU, respectively. We diagnosed rheumatoid arthritis and treated with DMARD therapy. She has not had another attack since.

Our patient presented with pseudo-septic arthritis. She had a vague history of morning stiffness, in symmetric joints, involving the hands, and was eventually found to be RF and CCP positive after undergoing orthopedic washout for a presumed septic joint. While patients carrying this diagnosis of rheumatoid arthritis can present with pseudo-septic arthritis, it is rare as the initial presentation. Even without her new hip complaints, she already met four of seven criteria for rheumatoid arthritis in a classification system that is 93.5% sensitive and 89.3% specific. Due to the involvement of an unusual joint, her diagnosis and treatment was delayed. Could she have manifested destructive joint disease if she had not developed this acute presentation? This illustrates the importance of a thorough history and the need for a wide differential to prevent irreversible joint damage in this population.

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MEMBRANOUS NEPHROPATHY ASSOCIATED WITH GASTROINTESTINAL STROMAL TUMOR

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Introduction: Membranous nephropathy (MN) is one of the most common causes of the nephrotic syndrome in adult non-diabetic patients. Secondary MN warrants attention for an underlying malignancy.

Case: A 69-year-old man was referred to nephrology clinic for nephrotic syndrome. By history, he was diagnosed with nephrotic syndrome after developing edema and proteinuria at age 56. He declined Western-style treatment and opted instead to undergo a homeopathic remedy in Brazil; this was associated with resolution of symptoms. He received regular follow-ups with no recurrence of proteinuria until 5 months prior, when he noticed pedal edema and severe proteinuria. He denied skin changes or joint symptoms. He has no diabetes and there was no exposure to nonsteroidal anti-inflammatory drugs. Laboratory included serum albumin 2.2 mg/dl, BUN 21 mg/dl, creatinine 1.0 mg/dl, LDL 230 mg/dl, and triglyceride 101 mg/dl. Urine protein excretion was estimated as 3.5 g/day. A diagnosis of nephrotic syndrome was made. Tests for hepatitis B and C viruses, syphilis and human immunodeficiency virus were negative. Antinuclear antibody titer was elevated as 1:320 (homogeneous and speckled pattern), but anti-dsDNA antibody was negative and serum complement (C3 and C4) levels were within normal range. Serum protein electrophoresis and immunofixation showed no evidence of monoclonal protein. Kappa/lambda ratio was normal. Computed tomography (CT)-guided renal biopsy revealed an immune complex-mediated glomerulopathy with a membranous pattern of injury. The deposits were negative for anti-phospholipase A2 receptor (PLA2R), which favored a secondary MN. CT imaging incidentally detected a 7.6 cm duodenal wall mass suspicious for gastrointestinal stromal tumor (GIST). No duodenal abnormality was discovered in the CT 7 years prior.

Discussion: In MN, 25% of adult cases are secondary and attributed to a variety of conditions such as chronic infections, systemic autoimmune diseases, and medications. Although the direct causal relation between malignancy and MN is controversial, recent reports suggest that the risk of malignancy among patients with MN is up to 12 times higher than in the general population. Therefore, a work-up for a malignancy is warranted if no other causes of MN found. We suggest that this secondary MN is associated with GIST because (1) there are no infectious or systemic conditions despite extensive testing, and (2) the GIST showed rapid growth at the time of the development of the nephrotic syndrome. One prior case of MN in a patient with a GIST has been reported in the literature.

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PROGRESSIVE MUSCLE WEAKNESS IN A MIDDLE-AGED MAN

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**INTRODUCTION:** Inflammatory myopathies are rare inflammatory diseases with common signs and symptoms such as weakness and muscle pain and elevated creatine kinase (CK). The different subtypes vary in presentation and prognosis.

**CASE:** A 43 year-old man presented to a community health center with two months of progressive fatigue, weakness and pain of his proximal arms and legs without fevers, chills, or joint pain. He took no medications. One month prior a limited physical exam was remarkable only for 'rash.' Labs showed elevated liver enzymes with negative hepatitis serologies. He was given ten days of prednisone and referred by his PCP to the internal medicine team. On presentation the patient reported improved strength and pain, but could not lift heavy objects and struggled to climb stairs. He endorsed mild dysphagia, and decreased energy. The rash on his arms and neck had resolved. He did not use tobacco, alcohol or drugs. Family history was unremarkable.

Vital signs and cardiopulmonary exam were normal. Extremities were non-tender, with normal bulk and tone, decreased strength in the neck and proximal upper and lower extremities, and decreased sensation to pinprick on the right forearm and hand; reflexes were normal. Labs showed markedly elevated CK, AST, ALT, and antinuclear antibody with normal, ESR, CRP, and creatinine. EKG, CXR, and abdominal ultrasound were unremarkable.

Based on his progressive proximal weakness and pain over months, elevated CK, ANA, and normal inflammatory markers, a presumptive diagnosis of necrotizing autoimmune myositis (NAM) was made. Positive anti-signal recognition particle antibodies (anti-SRP) and negative anti-Jo-1 antibodies further supported this. MRI showed left thigh edema and enhancement consistent with myositis. Biopsy was ordered but not done because of cost. We started him on four weeks of high dose prednisone followed by a taper. His strength improved and his CK normalized.

**DISCUSSION:** Our patient had classic symptoms of myositis. Key features—acute onset, symmetric muscle weakness, and elevated CK with normal inflammatory markers—pointed us toward NAM. Positive anti-SRP antibodies supported NAM, even without biopsy-proven necrosis. The absence of a characteristic rash steered us away from dermatomyositis; his age, the acuity of his symptoms, and the lack of bulbar signs made inclusion-body myositis unlikely. Polymyositis, a diagnosis of exclusion, typically develops slowly.

There is limited data regarding the treatment and prognosis of NAM. Usually patients are given high dose steroids (1mg/kg) for 4-6 weeks, and if they cannot tolerate a taper, will be transitioned to a steroid-sparing agent, such as methotrexate, IVIG or rituximab. These agents can also be tried if the patient is steroid-unresponsive. Ten-year survival is 90%, however, prognosis for specific sub-classes is unclear. We are hopeful our patient will do well given his rapid response to steroids and ability to tolerate the steroid taper.

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CHRONIC INFLAMMATORY DEMYELINATING POLYNEUROPATHY (CIDP): A RARE AND UNCOMMON MANIFESTATION OF SYSTEMIC LUPUS ERYTHEMATOSUS (SLE)

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**Introduction:** Chronic Inflammatory Demyelinating Polyneuropathy (CIDP) is an uncommon and a rare manifestation of Systemic Lupus Erythematosus (SLE). Only 19 patients with concurrent diagnosis of SLE and CIDP are reported in the medical literature until now. Multiple factors including early diagnosis of CIDP and presence of multiple antibodies associated with SLE predict a good response to intravenous immunoglobulin (IVIG).

**Case description:** 40-year-old African American female with past medical history of SLE diagnosed at age 40, on hydroxychloroquine presented with tingling sensation and progressive weakness in her feet and ankles after 4 months of SLE diagnosis. The sensory and motor symptoms progressed in an ascending fashion resulting in impaired balance without bowel or bladder involvement. Physical exam significant for weakness of all extremities decreased temperature, pinprick and vibration sense of distal extremities, areflexia and wide based unsteady gait. Laboratory investigations revealed positive ANA, Anti-SM antibody, Anti- RNP, Anti-SSA, Anti-ds-DNA, ESR of 75 mm/hr, low C4 compliment, leukopenia and anemia. No paraproteins were identified in serum electrophoresis. EMG showed axonal demyelinating polyradiculoneuropathy, abnormal peroneal distal latency with very low amplitude and disappearance of F waves consistent with CIDP. Patient was treated with IVIG 2 gm/kg daily for 5 day and prednisone 60 mg daily for total of 7 days while continuing hydroxychloroquine. She had marked improvement and regained ability to ambulate with minor assistance. She was discharged home on tapering prednisone dose with rheumatology follow up.

**Discussion:** About 50 % of the patients with concomitant diagnosis of SLE and CIDP achieve substantial clinical response to IVIG and remainders have minimal response. Certain characteristics including early CIDP diagnosis, involvement of all 4 extremities, hyporeflexia or areflexia, slowed motor nerve conduction velocity of the peripheral nerve, SLE involvement of critical internal organs and the presence of multiple antibodies associated with SLE predicts a good response to IVIG. Various proposed mechanisms of CIDP in SLE patients include abnormalities in blood vessels that supply the epineurium resulting in nerve fiber loss and interstitial amorphous infiltrate that separated nerve fibers along with reduction in the number of myelin sheaths. Treatment include steroids, IVIG and immunosuppressive agents including azathioprine and cyclophosphamide even though most patients are treated successfully with steroids.

**Conclusion:** CIDP in SLE is a rare presentation. Early identification of disease, SLE involvement of internal organs and multiple antibodies associated with SLE as seen in our patient predicts better outcomes. The presence of underlying SLE in CIDP reflects more severe immune dysfunction (mainly neuropathy) resistant to conventional therapy requiring addition of immunosuppression. Most patients are treated successfully with steroids if diagnosis made early.

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ONE VICE, BITTEN TWICE: THE DANGERS OF ADULTERATED COCAINE

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Levamisole is a veterinary antihelminthic agent, and was previously used in combination with fluorouracil to treat colon cancer. However, it was withdrawn from the drug market in the United States and Canada in the early 2000s due to severe adverse effects, most notably agranulocytosis. Today, levamisole is found as a diluent in cocaine and is reported to enhance its euphoric effects.

We present the case of a 54-year-old gentleman, with previous history of cocaine abuse, who was admitted to the hospital due to complaints of multiple small areas of rash and bruising. This extended initially from his shoulders to his abdomen and hips. He was otherwise asymptomatic. The patient revealed that he had recently relapsed and used cocaine 4 days prior to admission. He previously had a similar episode of petechial rash leading to extensive ulceration and skin necrosis requiring multiple debridements and grafting by plastic surgery. He also used cocaine at this time, which was confirmed to be adulterated with levamisole. On examination, the patient was noted to have significant petechial and purpuric lesions over the chest, back, shoulder, upper and lower extremities. Urine toxicology was positive for cocaine. Blood work revealed mild anemia, leucopenia and acute kidney injury. It also revealed positive anti-neutrophil cytoplasmic antibodies, myeloperoxidase antibodies, and proteinase-3 antibodies, suggesting vasculitis. Skin biopsy, however, was not consistent with vasculitis, revealing thrombosis and necrosis of the superficial dermal vessels. He later developed worsening renal injury with nephrotic-range proteinuria and casts consistent with acute tubular necrosis. Steroids and immunosuppression were considered but decided against due to a chronic, infected left hip ulcer. The patient's skin lesions progressed and eventually became demarcated. Plastic surgery was consulted early for excision and eventual grafting of the necrotic areas. However, despite aggressive care, the patient eventually developed fungemia and Klebsiella bacteremia, leading to septic shock, multi-organ system failure and his eventual death.

The presentation of this case leads to some unique lessons. First, the problem of cocaine adulterated with levamisole leading to significant medical issues, including skin necrosis, bicytopenia and renal failure, may become more common given the increasing amounts of cocaine found to have levamisole present. Repeat exposures of the agent may cause symptoms with quicker onset and severity. The complexity of this patient's case, in particular the juxtaposition of renal failure possibly requiring immunosuppression against the patient's chronic, active wound infection, required early consultation, extensive interservice communication and close monitoring and may lead to treatment dilemmas. It also reveals the importance of early recognition of these findings with early surgical consultation, as these patients requiring close monitoring and aggressive supportive care due to the potential for high morbidity and mortality.

## KAPOSI SARCOMA: A CAUSE OF GASTROINTESTINAL BLEED IN ACQUIRED IMMUNE DEFICIENCY SYNDROME (AIDS)

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**Background:** Despite a significant fall in incidence since the introduction of acute retroviral therapy (ART), Kaposi sarcoma (KS) remains one of AIDS-defining malignancies. It is also the most common gastrointestinal (GI) malignancy in AIDS patients due to a high rate, up to 40%, of gastrointestinal involvement. Patients are often asymptomatic but may rarely present with gastrointestinal bleed.

**Case:** We report the case of a 44 year old Caucasian male who presented to the emergency department with a two day history of fever, chills and non-bloody diarrhea. The patient had a history of human immunodeficiency virus (HIV) infection and was noncompliant with ART. He had been diagnosed with cutaneous KS three months prior to presentation and received one course of paclitaxel chemotherapy with subsequent regression of his malignant skin lesions. Physical examination was significant for fever, tachycardia and tachypnea. Labs revealed anemia (hemoglobin 7.6g/dl, MCV 77.4fL), thrombocytopenia (Platelet 33k/mm<sup>3</sup>) and elevated lactic acid (3.8mmol/l). Chest x-ray was normal. He was admitted to the intensive care unit for treatment of presumed sepsis. Blood cultures were drawn followed by administration of broad spectrum antibiotics. He had two episodes of melena on the second day of hospitalization causing an acute drop in hemoglobin down to 6.5g/dl. He remained hemodynamically stable and was transfused two units of packed red blood cells. The gastroenterology service was consulted and he underwent esophagogastroduodenoscopy and colonoscopy the following day. The procedures exhibited extensive lesions throughout the stomach, duodenum and colon with an appearance consistent with KS. No diagnostic biopsies were obtained due to the vascular nature and friability of the lesions. The patient improved clinically and antibiotics were discontinued after negative work-up for an infectious source. He was discharged home in a stable condition with scheduled follow up at the oncology clinic for chemotherapy.

**Discussion:** KS most commonly involves mucocutaneous tissues but visceral involvement is not unusual, especially in the AIDS population. ART has been shown to decrease the incidence of visceral involvement of KS in AIDS patients with known cutaneous disease. Often asymptomatic, GI KS are occasionally large, friable and result in bleeding, perforation or obstruction. Diagnosis is confirmed via endoscopy and treatment depends on the extent of the disease. Therapeutic modalities include ART, radiation and chemotherapy, alone or in combination. Antiretrovirals have been shown to decrease the proportion of new lesions, promote regression of existing lesions, and improve survival with or without chemotherapy. Systemic chemotherapy, mostly with liposomal anthracyclines, is reserved for cases with more widespread disease. Our case illustrates the need for increased awareness and a high index of clinical suspicion for GI KS in AIDS patients presenting with GI bleeding.

IGG-4 RELATED DISEASE PRESENTING AS LACRIMAL GLAND ENLARGEMENT IN A PATIENT WITH ESTABLISHED SJOGREN'S SYNDROME

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Autoimmune syndromes can share clinical manifestations but usually have distinct pathogenic, diagnostic and treatment modalities. Sjogren's syndrome and IgG4 related disease are two such syndromes that can present a diagnostic challenge.

An eighty-eight year old female was being treated for SS-A positive Sjogren's syndrome complicated by sicca symptoms and inflammatory joint symptoms for many years. Her past medical history was significant for untreated hepatitis C and interstitial lung disease (ILD) presumed related to Sjogren's syndrome. Her Sjogren's was treated with lubricant eye drops and hydroxychloroquine. She presented to her ophthalmologist with six months of worsening bilateral ptosis and swelling of her eyelids. Physical examination revealed enlarged lacrimal glands. Blood count, metabolic panel, erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) were all normal. Anti-nuclear antibody (ANA) was positive at 1:80 and SS-A was positive again. Magnetic resonance imaging (MRI) of her orbits revealed bilateral symmetrically enlarged lacrimal glands with suggestion of lymphoid infiltration. Given particular concern for lymphoma, a tissue diagnosis via lacrimal gland biopsy was quickly pursued. Biopsy showed large areas of fibrous band cicatrization with intervening areas of chronic inflammation associated with follicular lymphoid hyperplasia and a lymphoplasmacytic infiltrate. The biopsy showed no morphologic or immunohistochemical evidence of malignant lymphoma. Staining for IgG and IgG4 demonstrated a predominance of IgG4 positive plasma cells with an IgG4/IgG ratio of greater than seventy-five percent. A subsequent serum IgG4 level was elevated at >300 mg/dL (normal range 4-86). The histology along with elevated serum IgG4 level pointed towards the diagnosis of IgG4 related disease (IgG4-RD). On further questioning, she did have a past episode of pancreatitis decades prior without clear etiology. The patient was started on prednisone 40 mg/day with marked improvement in lacrimal enlargement, eyelid swelling, and ptosis just two weeks later. Steroid sparing agents such as methotrexate or azathioprine were deferred in the setting of her hepatitis C and ILD. The patient continued to do well on a tapering dose of steroid. IgG4-RD is a relatively newly described immune-mediated condition with a ubiquitous set of disease manifestations ranging from local glandular enlargement to retroperitoneal fibrosis and autoimmune pancreatitis.

Here we describe an unusual presentation of IgG4-RD in a patient with established Sjogren's syndrome with a high clinical concern for lymphoma. While elevated IgG4 levels have been described in patients with Sjogren's, to our knowledge this is the first case of biopsy-proven IgG4-RD in a Sjogren's patient.

DIFFUSE PAPULONODULAR SKIN LESIONS IN A RENAL TRANSPLANT PATIENT

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Cutaneous manifestations of cryptococcal infections are protean and there is no characteristic quality to the skin lesions that manifest from the infection.

A 65-year-old woman with allogenic renal transplant 20 years prior and chronic renal insufficiency was admitted for lesions on her face, upper chest and arms appearing as dome-shaped papules, with central umbilication. *Molluscum contagiosum* was suspected initially but symptoms did not improve and the lesions became more enlarged and necrotic. One week prior, her dose of prednisone was increased from 5mg to 10mg daily but mycophenolate mofetil (500mg BID) and tacrolimus (4mg BID) were unchanged. The rest of her physical exam was unremarkable, while her labs were notable for hemoglobin 7.6, WBCs 3.66 TH/UL: 61.4% neutrophils, 7.9% monocytes, 28.7% lymphocytes, 1.4% eosinophils, 0.4% basophils, and an elevated creatinine from baseline of 2.0 to 5.0 mg/dL. CXR was notable for a right upper lobe mass with prominence of the paratracheal regions bilaterally and a CT chest with nonspecific calcified nodules. Skin biopsy showed “numerous yeast with surrounding clear halos and PAS positive stain suggestive of cutaneous cryptococcosis” despite the absence of an inflammatory response as normally expected. Cryptococcal antigen was 1:10,420. The patient was diagnosed with disseminated cryptococcal infection without CNS involvement and started on liposomal amphotericin B. Consideration was given to the addition of flucytosine but was precluded by her creatinine elevation. No new lesions developed in four days of starting treatment, suggesting therapeutic efficacy, and her systemic, pulmonary and skin lesions gradually improved. Repeat cryptococcal titers were 1:320.

Cryptococcosis is a fungal infection more commonly seen in immunocompromised hosts. It typically infects the lungs, central nervous system and skin, however skin manifestations vary. Most lesions have an erythematous or ulcerative quality. The confounding appearance similar to *Molluscum contagiosum* may have delayed treatment of patients in this setting. Any skin finding in an immunocompromised patient should trigger a possible diagnosis of cryptococemia. Literature reviews have demonstrated the majority of transplant patients who had cryptococcal infection were renal transplants; of this, the majority tend to have infections in the late post transplant period with a mean time of 1.6 years. While taking tacrolimus decreases the chance of CNS involvement, it significantly increases the risk of skin involvement. Literature suggests in non-HIV infected patients with severe cryptococcus or cryptococcal meningoenzephalitis, the combination of amphotericin B with flucytosine has the greatest fungicidal activity compared to monotherapy or other therapeutics determined by clinicians.

ROMANO-WARD SYNDROME IN 27 YEAR OLD FEMALE MISDIAGNOSED WITH A SEIZURE DISORDER.

First Author: Baqer Haider, MBBS

**Introduction:** Congenital Long QT syndrome (LQTS) is an inherited cardiac arrhythmia characterized by abnormal QT prolongation on electrocardiogram (ECG) that may cause syncope or sudden death usually due to ventricular fibrillation. The syncope can often times be confused with a seizure disorder leading to the burden of anti-epileptic medications in patients. We present a case of a 27 year old female with unknown biological parents, who presented to the ER with generalized weakness and found to have Romano-Ward syndrome.

**Case Description:** A 27 year old female presented to the ED with multiple episodes of vomiting for one day, associated with headaches, blurry vision and generalized weakness. Her only home medicine was phenytoin for a presumptive seizure disorder. On admission, she was hypotensive and bradycardic. Her ECG showed wide QRS complexes with a prolonged QT interval (552ms). Patient was alert and oriented, labs demonstrated impaired renal function likely secondary to dehydration, urine drug screen was negative. Six months ago, the patient was hospitalized for a witnessed syncopal episode at which point she had fell and was reported to have been shaking, cyanotic and received resuscitation breaths. Although EEG and MRI were unremarkable, she was clinically diagnosed with primary generalized epilepsy and started on phenytoin. She smokes tobacco, but denies alcohol and any other illicit drug use. She is adopted and does not know her biological parents. While in the hospital, she was resuscitated until she became hemodynamically stable. Electrolytes were replenished. Echocardiogram showed decreased ejection fraction (20-25%). Review of ECGs from a previous hospitalization revealed that prolonged QT was present then as well. Prolonged QT was deemed to be likely congenital and unrelated to medications. While hospitalized, she experienced multiple episodes of nonsustained ventricular tachycardia and torsades de pointes and was placed on nadolol. After consideration, she received a subcutaneous ICD for prevention of sudden cardiac death. She agreed to have genetic testing, which was found to be negative, however she was counseled for LQTS testing in her child.

**Discussion:** Romano-Ward is an autosomal dominant genetic disease caused by mutations of ion channel genes of cardiac myocytes which results in delayed ventricular repolarization and manifests as syncope and possibly sudden cardiac death by ventricular fibrillation. A characteristic feature of this syndrome is the precipitation of arrhythmias by bradycardias, which may occur nocturnally and during episodes of emotional stress. Although it can be confused with seizure disorders it is essential that clinicians keep a high index of suspicion for cardiac related causes including LQTS as they maybe life threatening. Although genetic testing supports the diagnosis of LQTS it has a sensitivity of 75%.

## BEHCET'S DISEASE: A CASE PRESENTATION

First Author: Stephanie Iusim, MD, MPH Second Author: Vadim Bul, MD Third author: Dr. Brian Boulay, MD, MPH

A 20 year old male with a history Crohn's disease (CD) complicated by recurrent intestinal perforations presented to our facility for diarrhea, abdominal pain and for evaluation of his ileostomy. He reported a history of oral ulcers but not genital ulcers, abnormal ocular or skin findings. His medications included methotrexate and infliximab. He underwent a small bowel follow-through showing narrowing at the terminal ileum but his ileoscopy was without abnormalities. A flexible sigmoidoscopy was then performed showing patchy scattered inflammation and erythema but no ulcers. Rectosigmoid biopsies showed chronic colitis with cryptitis but no granulomas. His ocular exam was without abnormalities and no oral ulcers were present. He was initially discharged on azathioprine and prednisone but was readmitted several months later for a flare requiring high dose steroids. At this time, the patient also had evidence of scrotal ulceration requiring debridement by urology and underwent ileoscopy showing inflammatory polyps. Post-procedurally he developed a small bowel perforation requiring partial small bowel resection. He made a fully recovery with significant improvement in his symptoms. Although the patient did not fit criteria for Bechet's disease (BD), his diagnosis of CD was replaced with BD based on his recurrent gastrointestinal perforations, oral and genital ulcers. He was discharged on prednisone and infliximab and had good long-term outcomes. Behçet disease (BD) is a rare, chronic, multi-systemic, autoimmune disease characterized by recurrent oral aphthous ulcers, genital ulcers, uveitis, and skin lesions commonly encountered in young males.

Only approximately one-third of BD patients will have gastrointestinal involvement. Common intestinal symptoms are diarrhea, nausea, vomiting, and abdominal pain. Unfortunately, distinguishing intestinal BD from Crohn's disease can be quite difficult as clinical and endoscopic manifestations are quite similar. However, recent endoscopy studies comparing the two disease processes have found that round ulcers, focal involvement, presence of less than six ulcers, absence of gastrointestinal aphthous lesions, and lack of cobblestone appearance were endoscopic findings more commonly found in BD compared to CD. Treatments for acute CD and BD include mesalamine or sulfasalazine and high dose steroids while common maintenance therapy includes azathioprine, infliximab or adalimumab. While treatment is similar, it is important to differentiate between the two disease processes as prognosis greatly varies. Unlike with Crohn's, approximately 50% of patients with BD will fail medical therapy and require surgical intervention for perforations, fistulae formation, and massive gastrointestinal bleeds. Recurrence of intestinal manifestations of BD is common even after surgical procedures with 30% of patients requiring repeat procedures. Therefore, although rare, BD should be considered in patient with signs and symptoms of inflammatory bowel disease in conjunction with oral or genital ulcers, dermatological findings or ocular abnormalities, even when patients may not fit diagnostic criteria for Behcet's.

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'ABSTINENT AND INTOXICATED' A RARE CASE OF AUTO-BREWERY SYNDROME

Krishan Kataria MD, Sana Grover MBBS, Renato Alcaraz MD

**Introduction:** Gut fermentation or Auto-Brewery syndrome is a rare disease that affects immunocompromised patients. Patients present with a negative history of alcohol consumption and an elevated serum ethyl alcohol level.

**Case presentation:** A 25-year-old man was found unresponsive at his home. In the emergency department, his Glasgow coma scale was 6, for which he was intubated and started on mechanical ventilation. He was taking metformin for a history of diabetes mellitus, type 2. His vital signs were remarkable for a respiratory rate of 25/minute and blood pressure of 91/47 mm Hg. Physical examination was limited as the patient was under sedation. The comprehensive metabolic panel was remarkable for glucose of 173 mg/dl; aspartate transaminase and alanine transaminase were elevated at 90 U/L and 271 U/L respectively. The patient's serum alcohol level was 0.710 g/dL (normal-undetectable). We made a diagnosis of acute metabolic encephalopathy secondary to ethyl alcohol overdose. On day 2, the patient was liberated from mechanical ventilation. On subsequent evaluation, he mentioned a three-month history of intermittent episodes of right sided abdominal pain, headache, and dizziness. He strongly denied a history of alcohol intake and his family also confirmed that he was a teetotaler. A computed tomography of the abdomen showed findings consistent with fatty liver. His serum alcohol level slowly declined during hospitalization. Given the significant elevation of serum alcohol level and plausible denial of alcohol use, a diagnosis of gut fermentation syndrome was pursued. Esophagogastroduodenoscopy was done to obtain gastric and esophageal aspirates for fungal culture. A stool sample was also sent for fungal culture. Later, both these samples showed growth of *Candida albicans*. This supported the diagnosis of gut fermentation syndrome, for which patient received treatment with a 21-day course of fluconazole and nystatin. On regular outpatient follow-ups, his serum alcohol levels were unremarkable, and he remained asymptomatic.

**Discussion:** Gut fermentation or Auto-Brewery syndrome is a rare disease caused by overgrowth of certain species of *Candida*, including *C. Albicans*, *C. Krusei*, *C. Glabrata*, and *Saccharomyces cerevisiae*. These fungi metabolize carbohydrate to ethyl alcohol in the gut and lead to elevated serum levels of alcohol. Patients present with signs and symptoms of alcohol intoxication and a negative history of alcohol intake. Fungal cultures of stool and the gastric aspirate specimen can help in establishing the diagnosis. In some patients, carbohydrate challenge with certain specific foods may reproduce symptoms, and the serum alcohol level may rise, suggesting this diagnosis.

Since this disease is very rare, physicians require a high level of suspicion and awareness to diagnose this entity. If undiagnosed, the auto-brewery syndrome can pose negative personal, professional, emotional and sometimes, legal implications for patients and their families. A short course of anti-fungal medications can prevent recurrence.

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BACK PAIN; THINK ABOUT SOMETHING MORE THAN JUST MUSCLE, SPINE OR NERVE.

Ranju Kunwor, Ann Marie Canelas, Department of Internal Medicine, MacNeal Hospital, Berwyn, Illinois.

Arachnoiditis is a rare chronic inflammatory condition of arachnoid mater. Its rarity and lack of enough trials challenge the treatment which is confined to just pain management. It is usually secondary to adverse reaction to chemical (epidural/ spinal anesthesia), infection, direct injury to spine or complication of spinal surgery. Symptoms of arachnoiditis are seen weeks to months after the putative insult.

67 year old female with past medical history of Osteoarthritis and sciatica presented with worsening back pain and lower extremity pain for one month. Pain is more in the lower back and is worse than her usual sciatica symptoms. She describes her pain as sharp, stabbing, pin prick sensation radiating to left lower leg. For last two days, she has developed trouble ambulating due to extreme pain and numbness, that compelled her to come to Emergency department. She has a past surgical history of laminectomy forty two years ago. Patient states that her MRI five years ago was normal. Physical examination revealed tenderness in lower lumbar spine. Both the active and passive movement of lower extremities was painful with paresthesia. Reflexes were intact. Lumbar X-ray show mild spondylosis, without fractures. We continued her home medication tramadol and gabapentin; added oxycontin and lidocaine patch. Patient is admitted to neuro care unit with frequent neurological checks. Her pain was fairly controlled with all the analgesics. MRI showed post surgical changes in L4-L5 and findings suggestive of adhesive arachnoiditis at the level of L5. There was no associated enhancement of cauda equina nerve root. Neurosurgery recommended pain management only. Patient was discharged to home with home Physical therapy/rehabilitation along with pain medicines. Follow up appointment is scheduled in pain clinic. Patient symptoms continued, gabapentin dose was increased with little help. An epidural injection will be attempted to relieve the symptoms.

This case illustrates that arachnoiditis symptoms may start as late as 42 years after the causal event. Although our patient did not have any paralysis, bowel or bladder involvement, it is very important to make patient aware about the progressive nature and severity of the condition. It should be encouraged to discuss this disabling but exceedingly rare complication of spinal surgeries as a part of obtaining informed consent. Arachnoiditis is a debilitating condition with no specific treatment. Outcome is generally poor even with microsurgical lysis of nerve roots. Studies have shown that epidural injections of analgesics may even deteriorate the condition. Since adhesions are already formed at the time of diagnosis, steroid injections are not promising for the treatment of this unfortunate disease.

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INDUCING HYPOTHYROIDISM – AN UNUSUAL DRUG INTERACTION

First Author: Lima Lawrence, MD Aleida Rodriguez MD Natasha Shah MD Armand Krikorian MD, FACE Tahira Yasmeen MD, FACE

Introduction: Mycobacterium avium intracellulare (MAI) is a nontuberculous mycobacterial infection that is treated with multi-drug therapy including rifampin. Rifampin is a potent inducer of hepatic enzymes with multiple drug interactions and hormone derangements. Rifampin induced hypothyroidism in euthyroid patients with Hashimoto's thyroiditis is well described. We present a case where thyroid stimulating hormone (TSH) levels escalate due to rifampin therapy in a patient with previously controlled hypothyroidism. Our aim is to increase awareness of the effect of rifampin on thyroid function.

Case Description: A 71 year-old female with a past medical history of hypothyroidism, chronic obstructive pulmonary disease and recently diagnosed MAI infection presented with recurrent pneumonia. She was started on standard therapy for MAI including rifampin 450 mg daily, ethambutol 800 mg daily and azithromycin 500 mg on Monday, Wednesday and Friday. Patient was diagnosed with hypothyroidism over 20 years ago and maintained on levothyroxine (LT4). Her thyroid hormone replacement regimen consisted of LT4 50 mcg five days a week and 75 mcg two days a week. A few weeks after initiation of rifampin, the patient reported symptoms of constipation, cold intolerance and severe fatigue. On physical exam, there was no thyromegaly or proptosis. Laboratory evaluation demonstrated a progressive increase in TSH over two weeks from a normal value of 1.557 mU/L (0.4 and 4.0 mU/L) before initiation of rifampin, to 14.889 mU/L. Free thyroxine level was normal at 1.0 ng/dL (0.7 to 1.9 ng/dL). Subsequently, LT4 dose was increased to 75 mcg six days a week with instructions to repeat a TSH level in 6 weeks.

Discussion: Rifampin has been well-documented to cause clinically significant drug interactions and alterations in hormone levels. Case reports of rifampin-induced hypothyroidism during tuberculosis (TB) treatment exist in the literature. We report a rare case of rifampin-induced hypothyroidism during MAI treatment. A Japanese team reported a case series of three euthyroid patients with Hashimoto's thyroiditis with mycobacterium tuberculosis infection who developed hypothyroidism with elevated TSH that resolved after rifampin was discontinued. Our patient's progressive rise in TSH and transition from clinical euthyroidism to symptomatic hypothyroidism is likely related to increased thyroid hormone requirement due to enhanced hepatic metabolism and biliary excretion mediated by rifampin. Rifampin is a well-known inducer of cytochrome P450 hepatic enzymes and may result in increased T4 requirement. Physicians should be aware that patients with underlying thyroid disorders on rifampin may be vulnerable to developing worsening hypothyroidism. Thyroid function should be monitored after initiation and termination of therapy with rifampin.

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LOOKING BEYOND THE ITCH: WHEN A RASH IS NOT JUST SKIN DEEP

First Author: Kushal Naha, MD Second Author: Sowjanya Naha, MD Third Author: Adi Gidron, MD

A 39 yo Caucasian female presented with recurrent and progressive generalized pruritus of several years duration associated with a gradually darkening skin rash. The rash and pruritus were exacerbated by hot weather, sun exposure and exercise. She also complained of marked facial flushing after consuming alcohol, depression and tearfulness and chronic constipation. She had been using over the counter antihistaminics for years for her pruritus and noted that both the rash and itching would abruptly worsen whenever she ran out of it. She denied any constitutional symptoms such as weight loss, fevers or night sweats. Physical examination revealed a hyperpigmented macular rash over her arms, trunk and thighs, but was otherwise unremarkable. Review of her medical record showed that she had previously underwent punch biopsy from her thigh which had shown numerous mast cells consistent with urticaria pigmentosa. A bone marrow examination was performed which revealed multifocal dense infiltrates of predominantly spindle shaped mast cells accounting for an estimated 25% of bone marrow cellularity. The cells stained strongly positive with Giemsa and CD117 immunohistochemical staining. Polymerase chain reaction (PCR) study was positive for KIT (D816V) mutation. In addition, her serum tryptase was elevated to 74.9 ug/L. This patient therefore satisfied one major and three minor criteria for systemic mastocytosis. In the absence of 'B' and 'C' findings, she was diagnosed with Indolent Systemic Mastocytosis (ISM), treated with combined H1 and H2 blocker therapy and kept on follow up.

Systemic mastocytosis is a rare hematological disorder characterized by the accumulation of excessive mast cells in one or more tissues. Chronic and episodic mast cell mediator release result in generalized pruritus, neuropsychiatric and gastrointestinal symptoms that are typically exacerbated by temperature changes, exercise and alcohol ingestion. One of the underlying pathogenetic mechanisms, which was seen in this patient, is an activating mutation of the cell surface KIT receptor resulting in stem cell factor-independent activation and clonal expansion. Treatment strategies include antihistaminics, anti-leukotrienes and even chemotherapy, based on the severity which might range from indolent disease seen in this case, through smouldering disease, aggressive disease, to mast cell leukemia and mast cell sarcoma.

This case report serves to illustrate a classical presentation of systemic mastocytosis with multiple innocuous symptoms readily overlooked especially in the settings of a busy practice. Through this report, we hope to raise awareness of this condition among physicians while simultaneously highlighting the importance of considering all the symptoms of a patient however disparate, when making a diagnosis.

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MENINGITIS MYSTERY

First Author: Sowjanya Naha, MBBS, MD Second Author: Andriy Havrylyan, MD Third Author: Dima Dandachi, MD

We report a case of recurrent meningitis in a 59 year old male who presented with fever and confusion. His past medical history included mild mental retardation, hypothyroidism and hyperlipidemia. Three months prior to this presentation, the patient had been admitted with similar symptoms and was diagnosed with acute meningitis on the basis of abnormal cerebrospinal fluid (CSF) studies. He had completed a course of vancomycin, ceftriaxone and ampicillin with resolution of his symptoms. He was deaf and mute from birth. On clinical examination, he was febrile but had no localizing signs of infection. Basic blood work showed leukocytosis.

Considering his recent presentation with meningitis, repeat lumbar puncture and CSF analysis was performed, which showed neutrophilic pleiocytosis consistent with bacterial meningitis. Blood and CSF cultures were sterile. Retrospective questioning failed to reveal any risk factors for recurrent meningitis or history suggestive of congenital immunodeficiency. The patient showed a good response to empirical therapy with vancomycin, ampicillin and cefepime for 2 weeks. Broad-range bacterial polymerase chain reaction (PCR) of CSF was subsequently reported positive for *Streptococcus salivarius*. A member of the viridans family, *Strep salivarius* is known to cause bacteremia and endocarditis but is a relatively unusual pathogen for meningitis, due to its relatively low affinity to the leptomeninges.

Of the 68 reported cases of *Strep salivarius* meningitis, two-thirds were iatrogenic in origin having occurred after epidural and spinal anesthesia and diagnostic myelography. Interestingly, our patient did not undergo any of these procedures; while a lumbar puncture was performed on his previous admission, it was followed immediately by broad spectrum antibiotics which would have effectively sterilized the CSF. This case report also highlights the diagnostic utility of the broad-range bacterial PCR in culture negative meningitis.

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DRESS SYNDROME FOLLOWING METFORMIN ADMINISTRATION

First Author: Chibuzo C Odigwe, MD Prakruthi Voore, Aibek Mirrakhimov, Dana Rifai, Nkem Iroegbu

The DRESS drug rash, eosinophilia and systemic symptoms (DRESS) syndrome is a severe and potentially life threatening cutaneous hypersensitivity reaction characterized by extensive mucocutaneous eruption, fever, hematologic abnormalities including eosinophilia with or without atypical lymphocytosis and organ involvement.

The estimated incidence is thought to range from 1 in 1000 to 1 in 10,000 with mortality up to 10% in some series. A 40 year old man with a past medical history of psoriasis, hypertension, hyperlipidemia and recently diagnosed type 2 diabetes mellitus presented with complaints of a rash that had been going on for 3-4 weeks prior to presentation. The rash was described as diffuse, pruritic, red and scaly. There was no mucosal involvement. He had no associated fever, night sweats, weight loss, joint pains, headache, visual changes or photosensitivity. Two weeks before the onset of the rash he was started on metformin for diabetes mellitus. Prior to that he was taking only rosuvastatin 20mg daily, and Lisinopril 2.5 mg daily. His psoriasis was intermittently treated with 0.1% triamcinolone topical cream. Physical examination revealed extensive erythematous eroded papules coalescing into plaques with surrounding hyperpigmented patches noted on the face, neck, trunk, arms and legs, involving about 25% body surface area with associated onycholysis and cervical lymphadenopathy without hepatosplenomegaly. Complete blood count revealed a WBC 17.2 (having increased from 7 about 8 weeks prior), Hemoglobin was 15.9, and mean cell volume 87.7, platelets 313. Differentials showed an eosinophilia 37% (absolute count 6364 cells/mm<sup>3</sup>).

Comprehensive metabolic panel was unremarkable. Hepatitis Panel, HIV and RPR screening were all negative. Quantiferon gold assay was negative. Skin biopsy revealed excoriated psoriasiform spongiotic dermatitis, with bacterial cultures revealing a heavy growth of methicillin sensitive *Staphylococcus aureus* (MSSA). PASF (Fungal stain) was negative. A drug rash with eosinophilia and systemic symptoms (DRESS) was diagnosed as he met 3 out of the 5 Regiscar criteria for the DRESS syndrome, and Cephalexin was initiated to treat the bacterial superinfection. On follow up visits, the rash resolved and there was an improvement in fatigue. Lymphadenopathy resolved. Repeat CBC about 5 weeks later (WBC count of 8.1, Hemoglobin of 15.3, platelets 267, with 19% eosinophils and absolute count of 1612. To the best of our knowledge this is the first report of DRESS syndrome following metformin administration. Our patient had noticed a worsening of the rash about 1-2 weeks following commencement of metformin. He had 3 out of the five symptoms described in the REGISCAR criteria for diagnosis of the DRESS syndrome and he experienced remarkable improvement upon discontinuation of metformin.

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HERPES ESOPHAGITIS IN AN IMMUNOCOMPETENT PATIENT ON DOXYCYCLINE

Moni Roy, MD., John Farrell, MD.

**Background:** HSV esophagitis is an uncommon viral infection seen in immunocompetent patients. We present a case of severe HSV esophagitis in an immunocompetent patient after treatment for recent community acquired pneumonia with doxycycline. We also discuss the possible triggers of esophageal herpes infections including pill esophagitis that may have triggered reactivation of HSV-1 in our patient leading to severe herpes infection in spite of his immunocompetent status.

**Case presentation:** An 81 year old man with a history of COPD and atrial fibrillation, no history of systemic steroid treatment was hospitalized with complaints of productive cough, fever, and dyspnea on exertion. Exam revealed temperature of 101.2 C and crackles at the left base on inspiration. The oral mucosa and posterior pharynx were normal on exam. Chest radiograph that was notable for consolidation of the left lower lung fields. Empiric antimicrobial treatment was initiated with oral doxycycline 100 mg tab twice a day. On day 7 of treatment he returned with odynophagia, painful perioral skin lesions and oral ulcers.

**Methods:** Examination was notable for crusted ulcers over lips and perioral region, extending beyond midline. Initial testing included esophagogastroduodenoscopy (EGD), blood and sputum cultures, and CMV, HSV, VZV, HIV serology. HSV PCR was also sent.

**Results:** Herpes simplex, Varicella Zoster, Cytomegalovirus IgM titers were negative. Cytomegalovirus IgG negative. HSV IgG and HSV-1 PCR positive. HIV 1 & 2 antibody and antigen screen negative. EGD was notable for superficial circular ulcerations ranging from 5-10mm in size diffusely throughout the esophagus. Pathologic examination of esophageal biopsies was notable for intra-nuclear inclusion bodies, consistent with HSV infection.

**Treatment** consisted of acyclovir, initially IV, followed by oral treatment with improvement in odynophagia.

**Conclusions:** Both Herpes and doxycycline are known causes of ulcerative esophagitis. HSV esophagitis is commonly seen in immunosuppressed patient but is a rare finding in immunocompetent hosts. A negative antibody titer does not rule out infection. In our patient, the diagnosis was confirmed by endoscopy guided biopsy though HSV serology remained negative. It was unclear how this immunocompetent patient acquired the infection. One hypothesis being superinfection of esophageal pill esophagitis ulcers due to extension from preceding skin oro-labial lesions. Other possible cause may be reactivation of HSV virus that is known to remain dormant in ganglions and nerve endings after primary infection. Though pill esophagitis is mostly due to direct corrosive effect of pills, cases of vascular degeneration leading to mucosal sloughing due to doxycycline have been reported. Another possible cause of esophagitis in our patient may have been likely mucosal sloughing due to doxycycline leading to exposure of herpes virus harbored nerve endings. Early Clinical diagnosis and starting treatment for herpes esophagitis in immunocompetent patient remains a clinical dilemma and in spite of negative test results clinical decision making takes precedence.

## BILATERAL ADRENAL HEMORRHAGE: A CAUSE OF HEMODYNAMIC COLLAPSE IN HEPARIN-INDUCED THROMBOCYTOPENIA

First Author: Nasir Saleem, MD Second Author: Mahjabeen Khan, MD Third Author: Sanober Parveen, MD Fourth Author: Arvind Balavenkataraman, MD

**Background:** Heparin-induced thrombocytopenia (HIT) is a life-threatening complication caused by exposure to heparin. It is mediated by autoantibodies to platelet factor-4 causing platelet destruction, activation and thrombosis. Given their rich arterial supply and a single central vein, the adrenal glands are particularly susceptible to congestive hemorrhage following venous thrombosis. Adrenal hemorrhage associated with HIT tends to be bilateral, resulting in severe clinical manifestations ranging from abdominal pain and lethargy to catastrophic hemodynamic collapse.

**Case:** A 76-year old female with past medical history of rheumatoid arthritis (RA) and hypothyroidism presented initially to the ED with fatigue, abdominal pain, nausea, diarrhea and generalized weakness for 4 days. She had been taken off chronic prednisone therapy recently. A CT abdomen showed normal adrenals and she was discharged home on prednisone 30 mg daily. She received prophylactic unfractionated heparin during her hospital stay.

She presented again 10 days later with extreme weakness and listlessness. Vital signs included a heart rate of 101 beats/minute and BP of 102/54 mmHg. Her abdomen was soft and non-tender. Basic metabolic panel revealed mild hyponatremia (Na: 124 mmol/l) and normal potassium (4.7 mmol/l) and glucose (112 mg/dl) levels. Her platelet count had dropped from 187k/mm<sup>3</sup> to 62k/mm<sup>3</sup> raising suspicion for HIT. A serotonin release assay was positive confirming the diagnosis of HIT and she was started on argatroban infusion. Given persistent sinus tachycardia and her procoagulable state a CT chest was ordered to rule out pulmonary embolism (PE). Although no PE was detected the CT scan incidentally revealed bilateral adrenal masses that were absent 10 days earlier. MRI of the adrenals confirmed these to be bilateral adrenal hemorrhage (BAH).

ACTH-stimulation test revealed a low serum cortisol at 1.6 mcg/dl and she was put on IV hydrocortisone. Given her stable hemoglobin and no evidence of ongoing bleeding the argatroban infusion was maintained. This was later discontinued after adequate bridging to coumadin for 3 days with a target INR of 2-3. A repeat MRI done at the time of discharge showed no progression in the size of adrenal hemorrhage. She was discharged on oral hydrocortisone and is to avoid all forms of heparin products in the future.

**Discussion:** BAH is a life-threatening, paradoxical complication associated with HIT, a prothrombotic state. The resulting adrenal insufficiency may manifest as abdominal pain, fatigue, lethargy or severe hemodynamic instability. The development of new BAH in our patient as confirmed by the initial and subsequent CT scans are consistent with HIT associated BAH, and would explain her worsening fatigue and lethargy. She was receiving physiologic doses of prednisone for her rheumatoid arthritis which likely avoided an adrenal crisis and overt hemodynamic collapse. However, undiagnosed cases of HIT associated BAH tend to be fatal and a high index of suspicion is needed for early diagnosis and prompt treatment, primarily aimed at repletion of glucocorticoids and close monitoring of enlarging hemorrhage.

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SARCINA IN THE STOMACH, IS IT AN INNOCENT BYSTANDER OR A SUSPECT?

First Author: Raiya Sarwar, MBBS Second Author: Janet Jang, MD; Davendra P Ramkumar, MD

**Introduction** *Sarcina ventriculi* is a gram positive organism, which has a well established pathogenic role in veterinary sciences. It has also been found in feces of healthy humans consuming a vegetarian diet. Recently, there have been reports showing association between *Sarcina* in the stomach and gastric ulcers. It is rare bacteria to be isolated from gastric specimens and its pathogenic role in human is yet to be established.

**Case description** Patient is a 51 year old Caucasian woman with past medical history significant for type 1 diabetes mellitus and diabetic nephropathy leading to pancreatic and renal transplant 14 years prior, who presented with chief complaint of dull epigastric pain, non-bilious, non-bloody vomiting for the past two months. Physical exam showed epigastric tenderness. Laboratory investigation was consistent with iron deficiency anemia. The patient was started on a proton pump inhibitor (PPI). She underwent upper endoscopy, which revealed a large quantity of food material in the upper body and fundus with two large ulcers seen in the antrum. Biopsy revealed severely active antral gastritis with extensive ulceration and numerous microorganisms morphologically resembling *Candida* and *Sarcina*. Metronidazole and fluoroquinolone were given for four weeks and PPI dosage was increased. One month later, repeat endoscopy showed partially healed, but persistent ulceration with biopsy revealing atypical lymphoid cell proliferation negative for malignancy. *Sarcina* was not identified further. Cross-sectional imaging showed no signs of gastric perforation/emphysematous gastritis. After being treated with high dose PPI for two months, repeat endoscopic examination showed persistent ulceration with a large phytobezoar and gastric outlet obstruction. Subsequently, the patient was treated with N-acetylcysteine for the phytobezoar and was continued on high dose PPI.

**Discussion** *Sarcina ventriculi*, first observed in 1842 by Goodsir is an anaerobic gram-positive coccus with characteristic arrangements in tetrads or octets. Its role as a gastric pathogen in humans and direct mucosal injury is not well established. Recently, there has been emergence of reported cases with gastric ulcers, gastric outlet obstruction, and its complications of gastric perforation or emphysematous gastritis. This suggests a need for a further investigation of its pathogenic role in humans and establishment of management guidelines. There was no further identification of *Sarcina* species after completion of antibiotics in our case; we presume this was responsible for its eradication. The cause of the persistent ulceration in this case is unclear, and possibly mechanical in origin. *Sarcina* appears to be a marker for severe gastric complications related to physical or functional obstruction of the stomach, and its detection should prompt appropriate work-up and management.

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## HOW TO SHAKE OFF A CHOLESTATIC PICTURE

First Author: Matthew Steinman, MD

**Case Description:** The patient is a 43 year-old male who presented complaining of one week of fever, abdominal pain, cough, myalgia, nausea, jaundice, and dark colored urine. The patient went to Convenient Care originally with symptoms of a cough and a fever of 101.1 Fahrenheit was diagnosed with an upper respiratory tract infection and was given a course of antibiotics. However, his symptoms persisted and he eventually came to the Emergency Department. He denied any history of alcohol or IV drug abuse, incarceration, blood transfusions, recent travel, sick contacts, tick bites, or a family history of known liver disorders. The patient had one tattoo placed four years ago in a licensed parlor where a clean syringe was used. On admission to the hospital, he was found to have a white blood cell count of 27 with 45% bands, total bilirubin 11.7, alkaline phosphatase 139, aspartate aminotransferase of 38, and alanine transaminase of 31. His urine analysis showed elevated urobilinogen. A computerized tomographic (CT) scan of the abdomen demonstrated bibasilar lung infiltrate. A right upper quadrant ultrasound demonstrated no evidence of common bile duct dilation, but showed sludge within the gallbladder without evidence of wall thickening or pericholecystic fluid. The patient was started on meropenem. General surgery did not recommend acute surgical intervention. Gastroenterology was consulted and the patient was worked up for acute viral or autoimmune hepatitis which was negative. A magnetic resonance cholangiopancreatogram (MRCP) showed no biliary dilation or obstruction, but demonstrated periportal edema. Over several days, the patient's fever spikes and jaundice resolved and the leukocytosis and bilirubin levels normalized. Upon further investigation, the patient was consuming large quantities of whey protein as a dietary supplement. The whey shakes were thought to be the culprit for the cholestatic liver injury in this patient.

**Discussion:** The patient's case demonstrates an acute cholestatic liver injury associated with the use of whey protein supplement. The difficulty of diagnosing drug-induced liver injury is emphasized. The patient is a healthy 43-year old man who presented with fever, abdominal pain, and jaundice. He did not use recreational drugs or alcohol. Labs revealed an elevated bilirubin with minimally elevated transaminases. Serologic workup was negative for viral hepatitis and autoimmune liver diseases, and Wilson's disease was excluded. MRCP was unremarkable. A PubMed search had one similar case of cholestatic liver injury associated with whey protein and creatinine supplement. Similarly, there was a dramatic clinical improvement with the discontinuation of the nutritional supplement. In patients with an acute cholestatic picture, physicians should inquire about dietary supplement usage and consider immediate cessation of all potentially contributing products.

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CORAL REEF AORTA- A CAUSE OF RESISTANT HYPERTENSION AMENABLE TO SURGERY

Victor Udechukwu M.D, Venkatesh Ravi M.D, Stefan Tchernodrinski. M.D

Resistant Hypertension remains a common problem on the general medical wards and emergency rooms; this case vignette elucidates the need for imaging to evaluate for aortic atherosclerosis in cases of negative work up for the usual causes of poorly controlled or resistant hypertension. Coral reef aorta describes the rocky hard atherosclerotic lesion in the suprarenal part of the aorta.

**CASE PRESENTATION:** The patient is a 72-year-old woman who presented with shortness of breath and abdominal pain. She had a history of poorly controlled hypertension. Patient was currently on 5 different antihypertensives. Patient also had a history of heart failure with preserved ejection and recurrent admissions with hypertensive emergency. Blood Pressure(BP) on admission was 230/92mmHg. Examination revealed bilateral crackles in the lung bases, abdominal bruit. Patient was found to be in hypertensive emergency with flash pulmonary edema. Labs show creatinine elevation. Patient was assessed as having acute kidney injury. Work up for Pheochromocytoma, Cushing's disease and primary aldosteronism proved negative. Patient had no history of snoring or suspected obstructive sleep apnea. A repeat BP check revealed BP of 190/110 mmHg in the upper extremity and BP of 80/40 mmHg in the lower extremity. CT imaging revealed a dense isolated mass of calcification in the abdominal aorta above the level of the suprarenal arteries extending into the suprarenal, celiac and superior mesenteric arteries. She was diagnosed with coral reef aorta and underwent suprarenal aortic endarectomy. Post surgery, patient's BP improved and patient required only Nifedipine for blood control.

**DISCUSSION:** Cases of Coral reef aorta have been reported in literature. . Patients classically present with severe hypertension, usually requiring at least 2 or more antihypertensives, intermittent claudication, abdominal angina. Some also presented with complications of sustained severe hypertension- heart failure, renal failure. Diagnosis made with CT imaging. Treatment is usually surgical. Blood pressure control is greatly improved post surgery

**CONCLUSION:** Evaluation of patient with resistant hypertension should include blood pressure measurement in the upper and lower limbs, assessment to ensure medication compliance; rule out common causes of secondary hypertension. Consider CT imaging to evaluate for coral reef aorta if work up still remains non diagnostic.

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CASE OF SEVERE HYPONATREMIA IN A PATIENT ON A HERBAL FIVE DAY KIDNEY DETOX REGIMEN

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**Abstract:** This is a case of a patient who developed severe hyponatremia after commencing a five day kidney detoxification regimen. This regimen consisted of drinking over a gallon of fluid daily, in addition to taking a preparation of herbal tea with Uva Ursi Leaves, Juniper Berries and several other ingredients. On the last day of this detoxification regimen, he presented to the Emergency Department with a critical serum sodium level of 111 mmol/L associated with neurological symptoms. The purpose of this case report is to highlight the potential serious adverse effects associated with what is considered by the public to be benign herbal medicine.

**Introduction:** In recent times, the surge of public interest in herbal and alternative medical therapies has brought to the fore the possible harm that could result from some of the popularly advertised therapies. Herbal kidney detoxification regimens are promoted as a way to cleanse the kidneys, dissolve kidney stones and promote health.

**Case Report:** A 67 year old male with no significant past medical history presented to the emergency department with tremors and lethargy for one day. He was on a kidney cleansing regimen and had followed the instructions to the letter. On physical examination he was hypertensive with blood pressure of 158/84 mmHg, tachypneic with a respiratory rate of 28 cycles per minute, pulse of 88 beats per minute, temperature of 98 F, and oxygen saturation of 98% on room air. His cardiovascular and respiratory examination were unremarkable. Neurological examination was remarkable for tremors and a degree of agitation. Initial laboratory evaluation revealed a serum sodium of 111 mmol/L. Patient was admitted to the ICU and treatment for hyponatremia was initiated with 3% Normal Saline followed by different concentrations of NS. Serum sodium was monitored closely. After 49 hours from the time of admission, the patient clinically improved, serum sodium increased to 129 mmol/L and intravenous fluid administration was discontinued.

**Discussion:** This detoxification regimen induced a state similar to beer potomania. The patient's diet for 5 days only consisted of water and herbal preparation. In our patient the use of the various herbal teas induced a diuretic response with a degree of dehydration. The high hypotonic fluid intake normally would suppress ADH, but since the patient was dehydrated his ADH was not suppressed. Further the low osmolal load resulted in water retention and hyponatremia. The normal kidney can excrete urine of varying concentration ( 50 mosml up to 1200 mosmol). Older patients have more restricted range. Our patient's lack of osmoles in his diet resulted in hyponatremia even though his water intake was not excessive and was not beyond the ability of the normal kidney to excrete in the presence of normal osmolal load. Herbals, complementary nutritionals and micronutrients are all categorized as dietary supplements by the Dietary Supplements Health and Education Act of 1994. With increasing advertising and publicity, there has been a rise in the number of products, increasing from 4000 in 1994 to more than 55000 in 2012. The FDA is tasked with oversight of dietary supplements but the regulatory framework is different from that of prescription or over the counter pharmaceuticals, making it challenging to monitor the safety of supplements and herbal medicines.

**Conclusion** There is a need to raise awareness amongst the public about the potential serious metabolic and electrolyte derangements associated with herbal detoxifying regimens. In this case the patient developed severe acute hyponatremia which was potentially life-threatening.

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THE MAN WHO COUGHED UP LUNG

Xixi Zhao, M.D., Vikaas Kataria, M.D., Shobha Rao, M.D., and Mark Yoder, M.D.

**Case Description:** A 48-year-old man with a history of congestive heart failure (CHF) secondary to severe mitral regurgitation presents with persistent cough for 4 years. The patient described the cough as productive of a “meaty substance,” resembling lung tissue coated with blood and thick secretions. The tissue would become stuck in his throat, causing him to choke, become short of breath and sometimes faint. Only after he expectorated this substance would he experience respiratory relief until he produced another one. He noticed an increased production after developing pneumonia 3 months ago. On day 4 of admission, the patient expectorated a large bronchial cast. Cytology revealed rare bronchial cells and scattered macrophages. Bronchoscopy was preformed but no additional casts were located.

**Discussion:** Plastic bronchitis is a rare and enigmatic disease, characterized by the expectoration of bronchial casts. Case reports describe patients mistaking casts for undigested food, and 1 patient in particular mistook it for squid because of its large branching pattern and rubbery consistency. It can mimic status asthmaticus and foreign body aspiration when the casts obstruct major airways, causing acute respiratory failure. Although the first report of bronchial casts dates A.D. 131, there remains a dearth of information on the subject. The pathogenesis of cast formation is unclear and therapeutic options are limited and largely based on anecdotal experience. A major advancement was made when Seer et al. categorized patients into 2 groups based on histologic examination. Type I or “inflammatory” casts consist of fibrin with eosinophilic infiltrates and are associated with inflammatory diseases of the lung (e.g. asthma and pulmonary infections). Type II “acellular” casts comprise mainly mucin and are typically found in patients with congenital heart disease following the Fontan procedure. Some experts postulate that trauma to the lymphatic channels or increased inflammatory response provokes the formation of the casts, and others attribute it to increased pulmonary venous pressure leading to mucus hypersecretion; however, most cases are idiopathic. In our patient, although stains were not performed to classify the cast, we hypothesize that his are due to a combination of factors. Increased pulmonary venous pressure in the setting of mitral regurgitation may be the underlying etiology, with worsening due to recent pneumonia. The patient is being considered for the Clinical Outcomes Assessment of the MitraClip Percutaneous Therapy (COAPT) Trial in an effort to reduce mitral regurgitation, and potentially the formation of bronchial casts. This case suggests a possible novel mechanism of this disease, reinforces its potentially lethal nature and highlights the paucity of data regarding its management.

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CHERNOBYL DIARIES: AFTERMATH STILL BEING REPORTED.

First Author: Beenish Zulfiqar MD, Bilawal Ahmed MD

Introduction: The Chernobyl Nuclear Power plant accident was the worst nuclear power plant accident in history in terms of cost and casualties. There are several identifiable groups of individuals who were exposed to radioactive contamination, namely cleanup workers, evacuees and residents of contaminated territories.

Case: A 49-year old Polish man with a history of hypertension was sent to the emergency department (ED) by his primary care physician (PCP) after his complete blood count report showed WBC of 286,000. Patient had experienced 40 pounds unintentional weight loss in the last 6 months. He reports 3 weeks history of progressive night sweats, chills, diaphoresis and weakness, hence visited his PCP. He has no prior history of such symptoms. He denies any recent travel. In the Emergency Department (ED) his vital signs were stable. On physical exam he had palpable splenomegaly up to the umbilicus which was confirmed by CT abdomen. No abdominal or pelvic lymphadenopathies were identified on CT. Hemoglobin was 7.2, WBC 309 (differential showed 9% blast cells), platelets 242, uric acid 8.2, lactate dehydrogenase (Ldh) 1185. On further questioning he stated that he moved to United States at the age of 23 (1989). Before that he served in the Poland military from 1983-1989. He was exposed to Chernobyl nuclear plant and lived 300 km from the site. He denies any further exposure and states that he did not participate in any cleanup operations. He did take liquid iodine treatment at that time. He denies any history of cancer or blood disorder in his parents or siblings. Tumor lysis workup including chromosome analysis, leukocyte alkaline phosphatase (LAP) and Jak 2 mutation were sent. Patient underwent 3 sessions of leukopheresis. Workup showed that Jak 2 mutation was negative and Bcr-Abl antibody was positive. Bone marrow biopsy showed hyper cellular marrow with marked granulocytic hyperplasia with progressive maturation and positive BCR/ABL, consistent with chronic myelogenous leukemia. Patient was started on treatment with dasatinib (tyrosine kinase inhibitor) on discharge.

A number of studies of the health effects of radiation from the Chernobyl accident have been conducted in the last 25 years, mostly in the three most affected states of Belarus, the Russian Federation and Ukraine, on cleanup workers. They have been reported to be at greatest risk of thyroid cancer and leukemia. These were mainly men employed in 1986 and 1987, aged 20–45 years and who lived in the 30-km exclusion zone.

In this case the patient didn't belong to the affected state nor participated in the cleanup operation and lived far beyond the exclusion zone. It indicates that the incident had far reaching radioactive effects than documented with aftermath still being reported almost after 30 years.

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HIVES GONE BAD: A CASE OF LIFELONG URTICARIA PIGMENTOSA TURNED MAST CELL SARCOMA

First Author: Sarah M Jeong, MD Second Author: William B. Fisher, MD

Introduction: Mastocytosis is a heterogeneous group of disorders characterized by clonal mast cell proliferation and involves the skin in 80% of cases. In 60% of cases, cutaneous mastocytosis appears in childhood, usually takes an indolent course, and spontaneously regresses before puberty. Mast cell sarcoma (MSC) is a rare and aggressive subtype of mastocytosis characterized by a sarcoma-like tumor consisting of cytologically malignant mast cells and has been shown to progress to mast cell leukemia. To date, there have been less than 10 reported cases of MSC, all with a median survival of 12 months. Thus far, there is no curative treatment.

Case: A 27-year-old female presented with left-sided tender cervical lymphadenopathy and three small subcutaneous nodules on her left parietal scalp. Her medical history was significant for persistent urticaria pigmentosa diagnosed at age 5, which manifested as intermittent episodes of severe whole-body pruritis and flushing, and had since been largely controlled with topical treatments and hydroxyzine. Around age 7, she underwent a skin biopsy which showed cutaneous mastocytosis with typical mast cell morphology. At age 22, she noticed a 1cm pink nodule on her left parietal scalp, which was biopsied and demonstrated cutaneous mastocytosis with an atypical mast cell infiltrate. She was treated with corticosteroid injections into the nodule. Over the next 5 years, her disease remained quiescent and controlled with hydroxyzine.

On this presentation, the patient's lymphadenopathy had been accompanied by two episodes of drenching night sweats; however she never noticed fevers, chills, malaise, or weight loss. She underwent a short course of cephalexin with no improvement. Her CBC post-antibiotic treatment was normal. She underwent CT of the neck which showed extensive left sided cervical and supraclavicular lymphadenopathy, the largest lymph node measuring 3.1x2.3x3.9cm with central necrosis. Lymph node biopsy revealed atypical proliferation of pleomorphic and multinucleated mast cells as well as eosinophilic microabscesses. This case was reviewed in comparison to her two prior biopsies and showed pathological and immunohistochemical progression of her disease. She was subsequently diagnosed with MSC. Bone marrow biopsy showed frequent aggregates of atypical mast cells, rendering her disease systemic. She is currently undergoing chemotherapy treatment with 2-chlorodeoxyadenosine (cladribine).

Discussion: Mast cell sarcoma is an extremely rare and aggressive disease that is often misdiagnosed due to its rarity and lack of diagnostic features. This case represents a malignant transformation of what is normally an indolent disorder. Clinicians should be aware of the malignant potential of mastocytosis and suspicion should be raised with unexplained lymphadenopathy or subcutaneous nodules in any patient who harbors a history of cutaneous mastocytosis.

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BRUGADA, BRUGADA; WHAT IS ALL THE FEVER ABOUT?

First Author: Olufemi Olorunda, MD, MPH Second Author: Rodney Yuhico, MD

**Introduction:** Brugada syndrome is an autosomal dominant genetic disorder associated with sodium channelopathies, syncope, sudden cardiac arrest and characteristic electrocardiogram (ECG) changes. The typical ECG changes are consistent with a pseudo-right bundle branch block pattern and ST-segment elevation in V1-V3. There are two established patterns on the ECG with Type I Brugada characterized by a coved pattern in leads V1-V3 and Type II showing a saddle back pattern. Studies have shown that fever can unmask the characteristic ECG changes in some patients.

**Case Presentation:** A 19 year old male with a past medical history of infantile Respiratory Syncytial Virus, post-concussive seizures, and multiple visits to the Emergency Room (ER) for syncope and chest pain presented with a near syncopal episode while at a fireworks display. He experienced similar symptoms the night before and according to his wife, had a syncopal episode with concomitant seizure like activity. He endorsed pleuritic chest pain, fever, chills, nausea, and right upper quadrant abdominal pain. He also endorsed a significant family history of sudden cardiac deaths of cousins on both sides of the family.

Vital signs disclosed a maximum temperature of 103.6 degrees F while in the ER with ECG findings showing sinus tachycardia and typical Brugada pattern Type I with coved ST segment elevation in V2.

The patient's clinical course was complicated by another spike in temperature to 101.1 degrees F. Blood and urine cultures were negative for any source of infection and all other diagnostic tests were negative. The fever of unknown origin was deemed to be a viral syndrome. The patient was afebrile for 48-72 hours after which a subcutaneous Implantable Cardioverter Defibrillator was placed.

**Discussion:** The association of Brugada syndrome and syncope is very well established with very few cases describing seizures as the initial presentation. Given the various etiologies for syncope, non-structural heart disease is sometimes an afterthought. In our case, the family history of sudden cardiac deaths was a red flag; however all the patient's ECGs were negative in his previous ER visits. It is likely that the fever in our patient unmasked the Brugada changes seen on the ECG.

Sudden cardiac arrest is the initial presentation in as many as 30% of cases. It is reasonable to consider a heart monitor or referral to an electrophysiologist in a young patient with multiple visits to the ER for syncopal episodes as well as a significant family history of sudden cardiac deaths and negative diagnostic tests.

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THE IDENTIFICATION OF A STEROID CELL TUMOR IN A PATIENT WITH PRESUMED MENOPAUSE

First Author: Carol Rupprecht, MD Co-Author: Nancy Noel, MD

**Introduction:** When a woman over age 45 presents with irregular menstrual cycles and mood changes, often no additional laboratory evaluation is indicated.

**Case:** A 49-year-old Spanish-speaking female presented to our clinic for follow-up of non-insulin-dependent diabetes mellitus. Despite compliance with low-dose metformin, her hemoglobin A1c had increased from 6.4 to 7.9 over 10 months. She had gained seven pounds, which she attributed to exercising less. She endorsed mild depression which had begun two years prior when her father died and had improved over time. She had new complaints of facial hair and acne. She had not menstruated in 1.5 years but had not yet experienced hot flashes. Her mother had reached menopause in her late 40s. Her medical history included essential hypertension and a postpartum ovarian cystectomy. Physical examination revealed shaved facial hair. Morning cortisol was normal; her FSH was 6.5 mIU/mL, not consistent with menopause. The patient returned a month later with heartburn; pelvic examination at this visit, done to obtain a Pap smear and for further evaluation of her premenopausal amenorrhea, revealed clitoromegaly. Further blood tests included high total testosterone at 613 ng/dL, LH at 4.3 mIU/mL, and normal DHEA-S at 119 mcg/dL. For five months she presented for follow-up of diabetes but delayed in obtaining a transvaginal ultrasound; hair began to grow on her shoulders. Transvaginal ultrasound later revealed a solid left adnexal mass. Its morphology score was 5, associated with an increased risk for malignancy. Three months later the mass was visualized on CT scan with central enhancement. At time of total abdominal hysterectomy with bilateral salpingo-oophorectomy, her tumor measured 4.2 x 4.0 x 3.5 cm with no involvement of sampled lymph nodes or the omentum. A pathologist characterized it as a nonspecific steroid cell tumor.

**Discussion:** Laboratory evaluation is not often indicated for a female presenting with likely menopause. However, this patient's mild acne and hirsutism plus history of ovarian cyst prompted us to check an FSH level, primarily to differentiate between PCOS and menopause. We did not expect this would lead to a diagnosis of a sex cord-stromal ovarian neoplasm; incidence is 1 per 500,000 women. Few case reports of steroid cell tumors or Sertoli-Leydig cell tumors describe the presentation of women who are neither clearly premenopausal nor known to be postmenopausal. This case prompts us to consider a wide differential diagnosis for amenorrhea in middle-aged women, especially in those with any atypical symptoms or history.

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TO CLOT OR TO BLEED?... A RARE CASE OF LUPUS ANTICOAGULANT AND FACTOR INHIBITOR

Sushma Tatineni M.D., Naveen Manchanda M.D.

Lupus anticoagulant hypotherbinemia syndrome (LAHS) is a rare cause of coagulopathy and bleeding of unknown etiology. LAHS and other factor deficiencies should be considered in the work up of otherwise unexplained causes of bleeding.

A 45-year-old male with known history of coronary artery bypass graft (CABG) surgery without complications in 2011 and diabetes mellitus type 2 required hematology consultation for refractory coagulopathy. The patient was admitted for work-up of benign liver lesions and had elevated international normalized ratio (INR) of 3.0 despite adequate Vitamin K and fresh frozen plasma administration preventing ability to undergo biopsies.

Initial physical examination demonstrated a stable patient with epigastric tenderness to palpation and minor extremity ecchymoses. Prothrombin time (PT) was elevated to 26.5 seconds and activated partial thromboplastin time (aPTT) was elevated to 47.5 seconds. Mixing study demonstrated low factor II levels (13%) with presence of an inhibitor. Workup also revealed positive antinuclear antibodies, antiphospholipid, and anti-double stranded DNA antibodies. Treatment was initiated with high dose prednisone therapy along with cyclophosphamide. The factor II level had increased to 24% and INR normalized with resolution of abdominal pain, melena, and bruising. However, the patient stopped cyclophosphamide due to cost concerns. During outpatient follow up, the prednisone dose was decreased due to significant side effects and hydroxychloroquine was added.

This case represents a newly acquired coagulopathic disorder, as the patient had completed a long-term aspirin regimen after his CABG in 2011 without complication. With elevation in both PT and aPTT, this coagulopathy's origin is in the common pathway of the coagulation cascade. Factor II repletion via fresh frozen plasma did not correct the INR due to the presence of an inhibitor to factor II as a cause of bleeding, as revealed by the mixing study. The low factor II levels likely decreased activated protein C levels; causing discordant bleeding in the setting of positive antiphospholipid antibodies. Some literature suggests the anticoagulant effect is due to the direct inactivation of factor II from antiphospholipid antibodies through binding of plasma proteins present on factor II. The rare association of antiphospholipid syndrome with decreased factor II activity with bleeding has been documented. In a review of 74 cases of LAHS, 89% had hemorrhagic diathesis and 62% had minor bleeding. The prevalence of factor II inhibitor in patients with antiphospholipid syndrome ranges from 13.9-74% depending on the populations that were studied. Due to the rarity of acquired factor inhibitor, standard therapies do not exist. Immunosuppressive therapy has shown to be beneficial for treatment of this coagulopathy, and was effective management for this patient. In patients with bleeding disorders refractory to usual therapies, a mixing study should be considered to evaluate for coagulation factor inhibitors as the etiology.

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AN ORANGE A DAY KEEPS THE DOCTOR AWAY.

First Author: Jad El Masri, MD

Scurvy has become an increasingly rare clinical diagnosis in developed countries. Presentations may vary from the more common, often cited, follicular hyperkeratosis and perifollicular hemorrhages, to bleeding gums and dental caries, to sometimes life threatening GI bleeds and subcutaneous hemorrhages.

A 65 year old man with a history of alcohol abuse, with no known other medical condition, presented with atraumatic bluish discoloration of the left posterior thigh, accompanied by bilateral leg pain and swelling to a local Emergency Department (ED). He was given pain medication and was discharged home. He presented one week later with worsening leg swelling. He had more diffuse purpura, so leukoclastic vasculitis was suspected. He underwent a biopsy of one of the lesions that returned negative for a vasculitic process. The pain and swelling continued to worsen and he developed shortness of breath after which he re-visited a local ED where he was found to be severely anemic. He was transferred to our hospital for evaluation of a bleeding disorder.

The patient was not able to walk and had significant bilateral leg pain. He was found to have bilateral hematomas extending from the upper thigh to below the knees. His left knee was warm and had limited range of motion. His hemoglobin had dropped from 15.1 to 7.9 g/dL in the course of the 12 days since his initial presentation. Workup was negative for hemolysis, with normal iron studies, vitamin B12 and Folate levels. Blood smear revealed macrocytic anemia. Factor VIII level was normal. Coagulation labs were normal with normal liver enzyme levels but an increasing bilirubin level (predominantly indirect) from 1.2 to 1.9 mg/dL. HIV and hepatitis serologies were negative. SPEP was negative. Rheumatologic workup including C3, C4, ANCA, ANA and cryoglobulin screen was negative. CT chest and CT abdomen, done on separate occasions, did not reveal any masses. MRI of the left tibia and fibula was done and revealed circumferential subcutaneous soft tissue edema that was non-specific, with fatty infiltration of the muscles. His knee was evaluated by the orthopedics team and did not need to be tapped. The patient's Vitamin C level was checked and was <5 umol/L. He was started on Vitamin C supplementation. Currently, he is doing well with no new episodes of bleeding.

This case illustrates a rare case of life threatening subcutaneous bleed that required several transfusions. It is a reminder that vitamin and mineral deficiencies should be on the differential, especially when more common etiologies have been ruled out. This is particularly true in elderly patients, patients who are severely malnourished and in those with a history of alcohol and substance abuse.

RECURRENT MYCOBACTERIUM AVIUM COMPLEX INFECTION: AN UNUSUAL RESERVOIR

First Author: Amanda Grippen Goddard, DO Second Author: Zuhair Ballas, MD

A 64-year-old Caucasian female with a past medical history significant for osteoarthritis, iron deficiency anemia, and depression presented to the immunology clinic with a four-year history of productive cough, recurrent fevers, chills, night sweats, weight loss and decreased appetite. Previously, she was evaluated by the infectious disease and pulmonology services. Bronchoalveolar lavage (BAL) culture grew *Mycobacterium avium* complex (MAC); she was started on clarithromycin, ethambutol, and rifabutin. Despite taking these antibiotics for nearly two years, she continued to have daily productive cough and frequent fevers up to 102-103° F. BAL and sputum cultures persistently grew MAC after four weeks of incubation. Computed tomography (CT) of the chest revealed worsening reticulonodular infiltrates with early bronchiectasis. Lung biopsy was unremarkable. Her clinical course was punctuated with periods of improvement and worsening suggestive of seeding. High dose prednisone (20-40 mg/day) improved her symptoms better than antibiotics alone, although a maintenance dose of 5 mg prednisone was not beneficial.

Upon presenting to the immunology clinic, a thorough physical exam was negative except for diffuse rhonchi. Oral exam revealed a complete set of dentures that were removed and examined. They were found to be covered in a grayish film. Laboratory data was significant for a white blood cell count of 2900 (3700-10,500), neutrophils 1890 (2188-7800), lymphocytes 480 (875-3300), hemoglobin 11.2 (11.9-15.5), ESR 67 (0-20) and CRP 1.2 (<0.5). Extensive immune testing did not reveal any abnormalities. Genetic testing was not supportive of mutations that increase susceptibility to systemic infections with nontuberculous mycobacterium (Mendelian Susceptibility to Mycobacterial Diseases). Cultures taken from the dentures grew MAC. Shortly after she discontinued wearing the dentures, the fevers resolved and her cough significantly improved. After eight weeks of not wearing her dentures, repeat ESR was found to be 8 mm/hr.

Dentures are not commonly regarded as foreign bodies and may easily be missed on the physical exam. This patient demonstrates the importance of routinely examining and culturing dentures especially in the setting of recurrent oropharyngeal and pulmonary infections in a pattern suggesting seeding. Biofilms are described as a coherent cluster of bacterial cells, embedded in a self-produced matrix, that are more tolerant of antibacterials and host defenses than free-floating bacteria. They can lead to serious medical consequences including recurrent infections, antibiotic resistance and permanent tissue damage such as seen in cystic fibrosis. They characteristically cause chronic and difficult to treat infections rather than acute infections. As was seen in our patient, the most effective form of treatment is removal of the inciting foreign body. If this treatment strategy is not possible, the patient should receive chronic suppressive antibiotic therapy.

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FORGOTTEN BUT NOT GONE! SECONDARY SYPHILIS MANIFESTING AS TENOSYNOVITIS

First Author: Felicia Ratnaraj, MD Additional authors: David Brooks, Arun Nagabandi MBBS., Mahmoud Abu Hazeem MBBS.

**INTRODUCTION:** Tenosynovitis, inflammation of a tendon and its synovial sheath, is a rare manifestation of secondary syphilis and if diagnosed early, is reversible.

**CASE PRESENTATION:** A 52 year old male with past medical history of untreated syphilis presented with gradual onset of swelling and pain of the right fourth metacarpophalangeal joint (MCP). He reported a history of painless penile lesions after having sexual intercourse with a new partner approximately five months ago. The lesions resolved after being treated with sulfamethoxazole/trimethoprim. An RPR done at that time came back positive with a high titer; however, patient was lost to follow-up.

On examination, patient had an edematous, non-erythematous right fourth proximal interphalangeal (PIP) joint. The patient was started on broad spectrum antibiotics to treat empirically for infectious tenosynovitis. X-ray of right hand showed severe degenerative changes at the fourth PIP joint with medial subluxation of the middle phalanx, and periosteal reaction. An urgent irrigation, debridement and exploration of the right hand into the tendon sheath was performed. Post-surgery, a magnetic resonance imaging of the right hand showed soft tissue edema extending along the palmar aspect of the digit into the wrist, and tenosynovitis of the flexor pollicis longus tendon. Blood cultures, HIV and hepatitis panel were negative. RPR was reactive with a titer of 1:64. A confirmatory FTA-ABS test was completed, rendering a positive result. Based on his history of untreated syphilis, dormancy followed by clinical scenario of swelling of the right fourth finger and a high RPR titer, he was diagnosed with secondary syphilis manifesting as tenosynovitis. The patient was given benzathine penicillin 2.4 million units IM with complete resolution of symptoms in four weeks.

**DISCUSSION:** Syphilis is a chronic infection caused by transmission of bacterium *Treponema pallidum* via direct contact with an infected lesion during sexual intercourse. The early lesions of primary and secondary syphilis are very infectious. Untreated, acquired syphilis progresses through several stages. Primary stage consists of a chancre and regional lymphadenopathy. Secondary syphilis typically appears about six weeks after spontaneous healing of the chancre, but there can be an overlap. Patients with secondary syphilis can have various systemic manifestations.

One-third of the patients with secondary syphilis develop musculoskeletal complaints. Often, it involves the skull, shoulder girdle and/or the long bones. Common symptoms include localized or diffuse bone pain relieved by movement. Previous cases have presented X-rays demonstrating lytic lesions, or pathologic fractures, all of which were seen in our patient.

Treatment of choice for *Treponema pallidum* is Penicillin G, a single IM injection of 2.4 million units of benzathine penicillin G, in patients with primary, secondary, and early latent syphilis with complete resolution.

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MIGRATORY PULMONARY CAVITIES CAUSED BY RECURRENT LUNG INFARCTION DUE TO TAKAYASU'S ARTERITIS.

Yusuke Saishoji, Shin Yamashiro, Mitsuyo Kinjo, Kiyoshi Kinjo

Takayasu's arteritis is a chronic systemic inflammatory disease that mainly affects medium to large size arteries such as aorta and its major branches. It may sometime involve pulmonary arteries. We report a case of recurrent pulmonary infarction due to Takayasu's arteritis presenting as migratory cavitory lesions.

A 30 year-old woman presented with severe productive cough and occasional fever for three years. Physical exam was remarkable for a respiratory rate of 18 and low oxygen saturation (94% on ambient air). Chest X ray and Computed tomography showed multifocal cavitory lesions. Contrast Computed tomography demonstrated absence of the left pulmonary artery circulation and a thickened left pulmonary artery. Ventilation/Perfusion scan showed no perfusion to the left lung. Pulmonary angiogram showed total occlusion of the left pulmonary artery.

Review of her past medical history disclosed the following. Three years ago, she presented with fever, cough, hemoptysis and left lower lobe infiltrates. She was treated with antibiotics but failed to respond. Computed tomography of the chest and bronchoscopy were performed and pulmonary embolism was diagnosed. She had no risk factors for pulmonary embolism and coagulopathy work-up was negative. She took warfarin for six months. One year ago, she presented with a left lower lobe cavity: lung abscess was diagnosed and she was treated with antibiotics. The cavity healed with a residual air cyst, but two months later a new mass lesion appeared in the right upper lobe. The mass resolved without treatment three months later.

Lung infarction sometimes presents with cavitory lesions. Therefore, we diagnosed Takayasu's arteritis of the pulmonary artery causing recurrent infarction which presented with migratory cavities.

Takayasu's arteritis may affect pulmonary arteries and present as recurrent lung infarctions. Careful review of previous history and imaging studies helped establish the correct diagnosis.

DOCTOR, I CANNOT KEEP MY OXYGEN SATURATION UP! A CASE OF METHEMOGLOBINEMIA.

First Author: Asad Pervez, MD Second Author: Raiya Rehman, MBBS Third Author: Ghulam R Mohyuddin MD  
 Fourth Author: Allan Fleming, MD

**Introduction:** We describe a case of methemoglobinemia in a patient with persistent hypoxia.

**Case description:** 67-year-old lady was admitted to hospital with multiple compression fractures of spine. Her pain was controlled with hydromorphone, fentanyl and lidocaine patches. Past medical history was significant for multiple myeloma. She was receiving dapsone for PJP prophylaxis, in combination with chemotherapy. Interestingly, she was found to be hypoxic on admission. She denied chest pain, dyspnea, cough and hemoptysis. Respiratory and cardiovascular examination was normal.

Lab workup was significant for hemoglobin level of 8mg/dl, related to multiple myeloma. Chest X-ray and CT chest were normal. A V/Q scan was performed which was low probability for pulmonary embolism. Meanwhile, her oxygen requirements rose to 10L/min. Methemoglobin level was checked and it was elevated at 16.2%. G6PD level was checked and she was given appropriate dose of methylene blue. Oxygen saturation improved immediately and within an hour, oxygen was discontinued completely. Lidocaine patch and dapsone were discontinued. Repeat methemoglobin several hours later was 2.1%.

**Discussion:** Methemoglobinemia is a state of hemoglobin, which contains iron particles in ferric form. Ferric particles cannot bind oxygen. This results in increased affinity for oxygen in remaining ferrous particles in hemoglobin tetramer. Oxygen disassociation curve shifts to left and oxygen delivery to tissues is affected.

Dapsone and topical anesthetic agents like lidocaine, benzocaine and prilocaine are most common culprits for drug induced methemoglobinemia. Other causes include inhaled nitric oxide and aniline derivatives. Following table outlines treatment options based on serum methemoglobin levels.

Methemoglobin level	Treatment
<20%	Discontinue offending agent, observe.
>20%	Methylene blue or Vitamin C

Methylene blue was given to this patient with methemoglobin level of 16.2% because of baseline anemia. Methemoglobinemia causes a state of functional anemia and causes enhanced dysfunction in patients who are already anemic. We suspect this played a role in her high oxygen requirement in setting of a relatively low serum percentage of methemoglobin.

Methemoglobinemia is a potentially life threatening condition and it is important for physicians to know its clinical presentation and appropriate management.

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A RARE CASE OF MUTUALLY EXCLUSIVE KRAS AND BRAF MUTATIONS COEXISTING IN A PATIENT WITH METASTATIC COLON ADENOCARCINOMA

First Author: Anusha Vittal, MBBS Corresponding Author: Anup Kasi Loknath Kumar, MD

**Introduction:** Activating mutations in human Kirsten rat sarcoma viral oncogene (KRAS) occur frequently in metastatic colorectal cancer (mCRC). KRAS mutations result in constitutive activation of the RAS-RAF-ERK pathway conferring resistance to anti-EGFR therapy. B-Raf murine sarcoma viral oncogene homolog B1 (BRAF) is downstream of RAS. Mutations in BRAF, most commonly V600E, occur in upto 10% of mCRCs. KRAS and BRAF mutations are not only prognostic but also predict lack of response to anti-EGFR therapy. KRAS and BRAF are generally described as mutually exclusive in CRC.

**Case Report:** A 29-year old Hispanic woman with no significant medical, family and smoking history presented with complaints of abdominal pain, nausea, fatigue and constipation. Laboratory tests revealed iron deficiency anemia and mild transaminitis. Imaging studies revealed marked hepatomegaly with multiple hepatic lesions and sigmoid colon mass. Biopsy of the hepatic lesions showed adenocarcinoma of colonic origin. CEA was elevated at 284. Surprisingly, mutational analyses revealed CRC positive for both KRAS (codon 12) and BRAF mutations (V600). Hence, she was ineligible for anti-EGFR therapies. Anti-VEGF therapy was avoided due to the presence of a colouterine fistula. FOLFOX chemotherapy was administered for 2 cycles but her cancer failed to respond. Unfortunately, her disease progressed rapidly and she expired within 3 months from the time of her first diagnosis.

**Discussion:** KRAS and BRAF mutations are rare enough to be considered virtually (albeit not entirely) mutually exclusive. Coexistent mutations appear to be a very unique molecular and clinical subset with aggressive clinical course, which is in dire need of new therapeutic strategies. It is unknown which of the two mutations is the dominant oncogenic driver. Adding biologic agents to chemotherapy improves efficacy of treatment in mCRC. EGFR inhibitors such as Panitumumab and Cetuximab are approved for mCRC with wild type KRAS and BRAF. Either KRAS or BRAF mutation confers resistance to EGFR inhibitors. Vemurafenib and Dabrafenib are potent BRAF inhibitors that are very effective treatments in BRAF mutant melanomas. However, their role in BRAF mutant mCRC is unknown. Preclinical data suggest that EGFR reactivation occurs with selective BRAF inhibition that may lead to absence of response to BRAF inhibitor. So there may be a role to combine BRAF and EGFR inhibitors and this approach is being currently evaluated in clinical trials. Our case illustrates the need to assess for coexisting KRAS and BRAF mutations to not only understand the biology but also to evaluate the benefit of BRAF inhibitor +/- EGFR inhibitor in CRC.

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FSGS REFRACTORY TO IMMUNOSUPPRESSIVE THERAPY RESPONDS AFTER HCV TREATMENT

Jimmy Gentry, MD Hanna Mawad MD

Case: The patient is a 32-year-old male with a history of IV drug abuse who presented with 7 months of lower extremity edema and dyspnea on exertion. Labs indicated renal insufficiency with a creatinine of 1.89 mg/dL, hypoalbuminemia with a serum albumin of 1.1 g/dL, proteinuria greater than 10g per day, and a hepatitis C viral load of 14 million. Initially his hepatitis C infection was not treated due to his worsening renal function. He was started on cyclosporine and prednisone for FSGS and his volume status was controlled with albumin and furosemide. Despite temporary improvements in renal function, he continued to have worsening edema, proteinuria, and multiple readmissions for renal failure over the next 6 months. Hepatitis C viral load had also increased to 69 million over this time. With consistent improvement in his renal function following albumin and furosemide therapy, he was discharged with an outpatient regimen of albumin infusions 3 times weekly in addition to oral furosemide. His renal function then stabilized and he was able to avoid dialysis. After stabilization of his renal function he underwent therapy for his hepatitis C consisting of interferon and ribavirin. He was then restarted on cyclosporine and prednisone with drastic improvement in his proteinuria. He continues to have nearly normal urinary protein content, stable chronic kidney disease, and is currently being titrated down on his immunosuppression in the outpatient setting.

Discussion: Chronic hepatitis C virus infection is associated with various types of renal disease. Most common among them are essential mixed cryoglobulinemia leading to membranoproliferative glomerulonephritis (MPGN), MPGN without cryoglobulinemia, and membranous glomerulonephritis. Focal segmental glomerulosclerosis is rarely associated with HCV infection. Herein is presented a case of FSGS associated with chronic hepatitis C infection that did not respond to initial immunosuppressant therapy. His hepatitis C was stabilized with interferon and ribavirin and the challenges encountered during the treatment were discussed. This case strongly suggests that FSGS can be a result of HCV and immunosuppressive therapy may not be as effective until the virus is eliminated. In this particular patient the issue of worsening renal function initially excluded the option of HCV treatment and an unconventional approach had to be taken. In addition to glomerular damage from FSGS, massive proteinuria also caused repeated episodes of renal hypoperfusion leading to acute kidney injury. This was circumvented with albumin infusions as an outpatient with successful stabilization of renal function. Allowing for his HCV to be treated, he was better suited for immunosuppressive therapy and showed a favorable response.

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TREATMENT OF T2DM WITH SODIUM-GLUCOSE COTRANSPORTER LEADING TO EUGLYCEMIC DIABETIC KETOACIDOSIS

First Author: Victor Gasia Alfonzo, MD, PGY-1, Internal Medicine, Baton Rouge General Abdelghani Ramsy, PGY-3, Internal Medicine, Baton Rouge General

Sodium–glucose cotransporter 2 (SGLT-2) inhibitors are the most recently approved antihyperglycemic medications used as an adjunctive treatment in managing type 2 diabetes. This new class of drugs have been associated with diabetic ketoacidosis (DKA) with uncharacteristically mild to moderate glucose elevations (euglycemic DKA [euDKA]). The atypical clinical presentation makes euDKA difficult to recognize.

A 40 year-year-old woman with past medical history significant for T2DM controlled with metformin 500 mg BID and canagliflozin 300 mg qday, presented to the emergency department with a 1-week history of increasing fatigue and generalized weakness. Patient had also been complaining of diffuse abdominal pain, associated with nausea, vomiting and decreased oral intake. The symptoms persisted despite using antiemetic medication at home. The patient noted blood glucose at home in the 150s with adherence to medication regimen. Physical exam was unremarkable except for, dry mucous membranes, tachycardia and mild epigastric tenderness. Laboratory data yielded a blood glucose of 125, bicarbonate of 10, venous ph of 6.630 and calculated Anion Gap of 26. Ketonemia was noted, Beta hydroxybutyrate was 39. C- peptide was WNL. Ethanol, methanol and ethylene glycol was negative. Lactic acid was 1.4. No source of infection was detected. Patient was started on DKA protocol with IV fluids and insulin and canagliflozin was discontinued. After all interventions above ketoacidosis eventually resolved. Patient’s symptoms resolved as well completely.

This case illustrates the potential complications seen with the use of (SGLT2) inhibitors in diabetic patients. Increased renal clearance of glucose mediated by the SGLT-2 inhibitor led to deceptively low blood glucose levels in the setting of acute illness. Due to the odd presentation of this DKA case with the abnormally low to normal serum glucose level, timely identification of ketoacidosis with increased anion gap is critical to institution of appropriate medical therapy to avoid further metabolic derangements.

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## DIAGNOSIS AND THERAPEUTIC MANAGEMENT OF HYPOTHYROIDISM-INDUCED CHOLESTEROL PERICARDITIS

First Author: Amanda L Bennett Co-Authors: Todd Rosenthal David J. Elizardi Eiman Jahangir

**Introduction:** Cholesterol pericarditis is a rare cause of pericardial effusion defined by the presence of cholesterol crystals in the pericardial fluid and reactive pericardial cells with foreign body giant cells. The etiology of cholesterol pericarditis remains unknown due to its infrequency.

**Case Description:** We present the case of a 37-year-old male with no past medical history who complains of 6 months of progressive weakness, dyspnea, and progressive weight gain (10 lbs in one week, 35 lbs in one month). According to the patient, four days prior to admission, routine thyroid studies demonstrated mild hypothyroidism although laboratory values were not available for review and he was started on levothyroxine supplementation. On the evening of presentation, he experienced profound worsening of dyspnea, palpitations, and weakness. Chest x-ray demonstrated a markedly enlarged cardiac silhouette. Echocardiogram demonstrated a large circumferential pericardial effusion, with RV collapse and respiratory variation on mitral inflow consistent with acute pericardial tamponade requiring urgent pericardiocentesis.

**Management:** Pericardiocentesis yielded 2,650 mL of serous, yellow colored fluid containing cholesterol crystals (48 mg/dL, serum cholesterol = 99 mg/dL) and giant foamy macrophages. A pericardial drain was placed and maintained for 6 days until output decreased to ~10 mL/day. Severe scrotal and lower extremity edema was also noted. He was started on intravenous Furosemide and diuresed a net negative 44.1L. CT imaging of the chest did not demonstrate evidence of damage to the thoracic duct; his HIV, PPD, and Rheumatoid factor, tests were all negative. Serial echocardiographic studies demonstrated resolution of the effusion without reaccumulation. He was discharged with levothyroxine, furosemide and potassium supplementation. Subsequent follow up has demonstrated an initial 8 kg weight increase over 11 days but no recurrence of pericardial effusion on echocardiogram at 3 months.

**Discussion:** This case demonstrates the therapeutic management of cholesterol pericarditis with tamponade. Cholesterol pericarditis has been seen in combination with myxedema related to hypothyroidism, thoracic duct trauma, rheumatoid arthritis, and tuberculosis. Pericardial effusions associated with cholesterol pericarditis accumulate slowly but may ultimately progress to restrictive and tamponade symptoms and may recur. The most effective known therapy in these cases is identification and treatment of the causative agent. As hypothyroid-induced cholesterol pericarditis is the most frequently observed form, effective treatment involves therapy until euthyroid state is achieved.

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ONE OF THE BEST MIMICKERS OF METASTATIC DISEASE.

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**Introduction:** Histoplasmosis is a common endemic mycosis and its clinical presentation varies greatly posing a diagnostic challenge.

**Case Description:** A 48-year-old, retired military officer presented to Emergency Department with complaints of generalized abdominal pain, intermittent headaches, weakness, vomiting and diarrhea along with 60 pounds weight loss in the last 3 months. His past medical history was significant for hypertension and an 80 pack year smoking history. His physical exam revealed hypotension, hepatosplenomegaly and generalized abdominal tenderness. His labs showed hyperkalemia, anion gap metabolic acidosis, acute kidney injury with creatinine of 4.5mg/dL and adrenal insufficiency. CT Thorax and Abdomen without contrast revealed pulmonary nodules, diffuse abdominal lymphadenopathy, massive splenomegaly and 7 cm bilateral adrenal masses. MRI of the brain showed 3 ring enhancing lesions. Our workup for HIV came back positive, however, lumbar puncture, flow cytometry and all other infectious disease workup were unremarkable. His renal function improved with hydration. After ruling out pheochromocytoma, core biopsy of left adrenal gland was performed which revealed Histoplasmosis. We initiated treatment with Fludrocortisone, IV Amphotericin, combined anti-retroviral therapy (CART) and empirically covered him for Toxoplasmosis. The patient's symptoms, laboratory findings and repeat imaging studies showed marked improvement.

**Discussion:** Histoplasmosis can present as an asymptomatic, pulmonary, progressive disseminated or primary cutaneous disease. The progressive disseminated form of the disease is rare and usually occurs in the immunocompromised patient. In Louisiana and other endemic regions, Histoplasmosis should be considered in a patient with [mediastinitis](#), pulmonary nodules, [lymphadenopathy](#), [hepatosplenomegaly](#), chorioretinitis, adrenal enlargement and calcifications.

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NOT JUST FOR KIDS: RECOGNIZING AN IMPORTANT GENETIC DISORDER IN ADULT PATIENTS

Lesley B. Gordon, MD

**Introduction:** As internists we must be cognizant of late-presenting so-called “pediatric” illnesses.

**Case Description:** A 24-year-old man with type 1 diabetes mellitus presented to the Emergency Department with oozing lower extremity wounds in the setting of six months of edema. He reported a 30-lb weight loss over the same time period, severe diarrhea with fecal incontinence, and intermittent RUQ abdominal pain. On exam he appeared emaciated and had RUQ tenderness, diffuse anasarca, and superficial erosions on lower extremities. He was admitted for hyperglycemia (548 mg/dL) and early diabetic ketoacidosis. His AST/ALT on admission were 118/261 U/L. Within two days his aminotransferases became markedly elevated (5916/1662 U/L). He did not exhibit encephalopathy, nor did he have hyperbilirubinemia or coagulopathy. CT-Abdomen from several months prior showed massive hepatomegaly without nodularity or associated splenomegaly as well as an atrophic pancreas and thickened duodenum. Viral hepatitis and HIV serologies were negative; ultimately, testing for alpha-1 antitrypsin deficiency, hemochromatosis, and autoimmune hepatitis was also negative. Duplex ultrasound did not reveal Budd-Chiari; there was no evidence of right heart failure by exam or echocardiogram. Skin and duodenal biopsies were negative for amyloid. Due to intense headaches, he underwent a head CT that revealed severe sphenoidal sinusitis. His stool studies were consistent with steatorrhea and a therapeutic trial of pancreatic enzyme replacement proved beneficial. He had a borderline sweat chloride test for cystic fibrosis (CF) and his initial genetic panel returned positive for N1303K, a “severe” mutation with respect to the pancreas. His blood was also sent for genotyping. His aminotransferases decreased gradually although his alkaline phosphatase rose modestly.

**Discussion:** This patient’s acute hepatocellular injury is classic for ischemic hepatitis (i.e., aminotransferases ~ 50-100 x ULN, late-peaking alkaline phosphatase, rapid resolution), which can result from subclinical hepatic hypoperfusion particularly with underlying abnormal liver architecture or functionality. His broader constellation of findings was consistent with CF, including pancreatic atrophy with associated diabetes mellitus and exocrine pancreatic insufficiency, sinusitis, and liver disease. CF, with over a thousand known underlying mutations, is now appreciated as a spectrum of phenotypes. Though previously thought to be rare outside of childhood, initial recognition in adults is becoming more common. These patients are more likely to have nonclassic features that can easily be missed if not on the radar of the clinician. Early referral and management of these patients will improve quality of life, reduce morbidity/mortality, and potentially impact their family planning.

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RAPIDLY PROGRESSIVE DEMENTIA IN A HEALTHY MAN: AUTOIMMUNE LIMBIC ENCEPHALITIS

Vineet Agrawal

Purpose: To describe an uncommon cause of altered mental status who presents with rapid onset changes

Case Presentation: The patient is a 61 year old man with no prior history who was referred to the hospital by his primary care provider for increasing episodes of confusion and right upper extremity jerking. Episodes had initially started 2 months prior in the setting of stress and consisted of acute onset confusion and memory loss. One such episode resulted in a motor vehicle accident and prompted inpatient admission. Upon initial evaluation, vitals were within normal limits. Mini mental status exam was 23/30 with deficits in memory, attention, and calculation. Physical examination was notable for a euvolemic thin gentleman with normal naming, speech, comprehension, cranial nerves, motor exam, coordination, reflexes and gait. Multiple episodes of 1-2 second-long periodic right upper extremity jerking movements were noted during examination. Initial laboratory studies were notable only for hyponatremia (Na 123). Extensive biochemical testing for thyroid function, liver dysfunction, syphilis, autoimmune studies, and HIV were negative. Lumbar puncture was notable for no WBCs or RBCs and normal glucose and protein with negative cultures and viral PCRs. EEG testing showed no evidence of epileptiform discharges. MRI of the brain showed mild parenchymal volume loss in the post-central gyrus but otherwise normal. PET-CT showed diffuse hypo-metabolism throughout the cortex with mid-brain sparing. A repeat lumbar puncture was performed and found to be positive for voltage gated potassium channel antibodies, and specifically LGI-1 antibody. Notably, 14-3-3 protein was negative. The patient underwent plasmapheresis with subsequent reduction in periodic myoclonic movements and improvement in MMSE to 28/30. He was referred to a specialized neurology clinic. He was eventually started on systemic immunosuppression with continued improvement in symptoms.

Discussion: Voltage gated potassium channel antibody-associated encephalitis is a rare form of limbic encephalitis that is characterized by mood changes, disorientation, abnormal movements, and seizures. Hyponatremia due to SIADH is a common finding in these patients. Early recognition and expedited referral for concerns of autoimmune limbic encephalitis can result in full recovery with minimal permanent sequelae.

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SEVERE AORTOILLIAC DISEASE COMPLICATED BY ABDOMINAL AORTIC ANEURYSM THROMBOSIS WITH ACUTE RENAL INFARCTION

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**INTRODUCTION:** Abdominal aortic aneurysm (AAA) thrombosis is related to occlusive iliac disease and often presents with intermittent claudication. While mural thrombosis, which generally does not preclude blood flow, is frequently observed in 70-80% of patients with AAA, complete vessel occlusion is a comparably rare event associated with high mortality. Likewise, acute renal infarction is a serious medical emergency on its own. However, the diagnosis is often delayed or missed as it is uncommon resulting in treatment delays and permanent loss of renal function. We present a case of proximal propagation of infra-renal AAA thrombosis with near complete vessel occlusion leading to acute renal infarction.

**CASE DESCRIPTION:** A 63-year-old African American male with history of hypertension, CVA with residual right upper monoparesis, PVD and dyslipidemia presented with a two-day history of an 8/10 colicky peri-umbilical pain associated with diarrhea, nausea and vomiting. Vomitus consisted of greenish, non-bloody fluid with one episode of brownish watery stool. He denied sick contacts, fever or chills. Notably, he has been off his medications for three months. He is a current smoker with 60 pack years. On admission, his BP was markedly elevated at 212/129mmHg with grade 2 hypertensive retinopathy which was initially attributed to his noncompliance. Cardiopulmonary examination as well CMP and CBC were within normal limits.

However, on day two, he reported worsening claudication and right foot pain at rest with abdominal pain now predominantly in left lower quadrant associated with leukocytosis. Abdominal CT scan with contrast revealed acute left renal infarct and a 2.8x3 cm AAA just above the bifurcation with penetrating atherosclerotic ulcers for which he was started on therapeutic anticoagulation to prevent further renal infarction. He successfully underwent open aorto-bifemoral bypass in the setting of severe aortoiliac disease complicated by PVD and AAA thrombosis with acute renal infarction. Significant improvement in symptoms was noted and he was discharged home a few days later.

**DISCUSSION:** Manifestation of chronic AAA thrombosis as intermittent claudication is a rare but well-recognized issue in clinical practice. Several factors, like thromboembolic disease, appear to be associated with AAA thrombosis. However, obstructive iliac disease plays the most important role in chronic manifestation. Renal infarction is a serious cause of acute nephron loss that is potentially reversible by reperfusion therapy, but the diagnosis is often missed or delayed. Neither renal infarction nor proximal propagation of AAA thrombosis was considered initially in this case. This highlights the importance of considering renal infarction in the differential diagnosis in patients presenting with acute abdominal pain in the setting of PVD.

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RARE SQUARED – AN EXTRAGONADAL GERM CELL TUMOR IN A PATIENT WITH A MITOCHONDRIAL DISORDER

First Author: LT Ayeetin Azah, MD and CPT Joseph Roswarski, MD

The majority of the recognized oncogenes and tumor suppressor genes implicated in tumorigenesis are found within nuclear DNA. However, there is a growing body of literature describing the causative influence of germline and somatic mutations in mitochondrial DNA in cancer formation and mutagenesis. We present a case of primary mediastinal non-seminoma germ cell tumor in a patient with an inherited mitochondrial disorder.

A 36-year-old Caucasian male with maternal family history of mitochondrial encephalomyopathy with lactic acidosis and stroke-like symptoms (MELAS) was transferred from a medical center in Germany to Walter Reed National Military Medical Center for evaluation of a five-week history of right chest and shoulder pain as well as dyspnea on exertion. His initial chest x-ray revealed a mediastinal mass. He was hemodynamically stable and had no signs of superior vena cava syndrome on exam. His routine laboratory data was remarkable for mild leukocytosis. Additional pertinent labs included lactate dehydrogenase, 361 mg/dL; alpha fetoprotein, 732 ng/mL; beta human chorionic gonadotropin hormone, 5028 mIU/mL. CT imaging of the chest, abdomen, and pelvis with contrast showed a 13.5 x 8.7 x 8.2 cm lobulated mass in the right anterior mediastinum causing mass effect on the heart and superior vena cava with multiple bilateral pulmonary nodules. He underwent CT-guided core needle biopsy of the mass, which was consistent with a non-seminoma germ cell tumor. Bilateral testicular ultrasound showed normal sonographic appearance of the testes and brain MRI was negative for any intracranial pathology. The diagnosis of primary mediastinal non-seminoma germ cell tumor was made and he was started on bleomycin, etoposide, and cisplatin chemotherapy for a planned total of four cycles. Baseline audiogram prior to chemotherapy showed mild bilateral sensorineural hearing loss, which may be related to his diagnosis of MELAS.

Mutations in mitochondrial DNA have been implicated in the etiology of human cancers, with breast cancers being most commonly identified. Primary mediastinal non-seminoma germ cell tumors are rare tumors with a 5-year overall survival of 45%. Per review of the literature, this is the first reported case of primary mediastinal non-seminoma germ cell tumor occurring in a patient with an inherited mitochondrial disorder.

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A RARE CASE OF OBSTRUCTIVE SLEEP APNEA SECONDARY TO A RETROSTERNAL GOITER

First Author: Deepti R Baheti, MBBS Second Author: Maksym Puliaiev, MD Third Author: Rupinder Singh, MD

**INTRODUCTION:** Literature, as early as 1965, describes an association of obstructive sleep apnea (OSA, described then as “alveolar hypoventilation during sleep”) with thyroid disorders. Of all the thyroid disorders linked with OSA, hypothyroidism is the most common abnormality studied. Lingual thyroid, goitrous Hashimoto’s thyroiditis have also been reported in association with OSA, although infrequently. This is one of the few case reports in English medical literature where OSA was the presenting feature of a near fatal, mediastinal mass that turned out to be a retrosternal goiter(RSG) diagnosed, albeit, 5 years later.

**CASE PRESENTATION:** A 55 year old obese woman with history of hypertension presented with excessive daytime sleepiness. Sleep study showed moderate OSA (RDI-21.6). TSH done at that time was 0.496 uIU/ml. She was non-compliant with nasal CPAP & was lost to follow up. 5 years later, she returned to our hospital, after a chest X-ray performed at an urgent care center for flu-like symptoms showed a mediastinal mass. Her exertional dyspnea had progressed to a point where she was symptomatic at rest. She also noted easy fatigability & paresthesias of both upper extremities. She did not have dysphagia, unintentional weight loss or changes in her voice. She did recall having a mildly enlarged thyroid 20 years ago. Physical exam showed a 1 cm tracheal deviation to the left with a palpable fullness over the left lobe of the thyroid without any discrete nodularity. Lung sounds were clear with no stridor or audible wheeze. A large mass originating from the inferior pole of the right thyroid lobe was seen on CT(with and without contrast) neck and thorax, with a paratracheal extension into the right mediastinum up to the mainstem bronchus. Her thyroid function tests (TFT), despite a large, near-fatal goiter were normal. A right hemithyroidectomy with mediastinal exploration was done. Pathology showed multinodular hyperplasia.

**DISCUSSION:** The incidence of RSG as a percentage of total thyroidectomies ranges from 2 to 19%. RSG can cause laryngeal edema and compression leading to OSA. Unlike cervical goiters, RSG may not produce any visible enlargement and can be missed. TFT used routinely to screen thyroid disorders in patients with OSA can be normal, as in our patient. After a diagnostic sleep study, a thorough workup should be done to delineate the etiology of OSA. While CT scan is the gold standard for diagnosing RSG, it carries the risk of radiation exposure. Laryngoscopy may be an attractive alternative test to screen OSA patients with airway compromise early on.

**CONCLUSIONS:** OSA can be a potential harbinger of RSG and research should focus on laryngoscopy as a screening tool along with TFT to aid early diagnosis.

## A TALE OF TWO TUMORS

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Gastrointestinal carcinoids are the most common neuroendocrine tumors. The rarest type of carcinoid tumor is the ampullary located carcinoid which accounts for 0.05% of all carcinoids [1-3]. The rarity of this entity makes it a diagnostic challenge.

A 61 year old lady presented after two episodes of generalized tonic-clonic seizures with left sided hemiparesis. She reported recurrent headaches for two weeks, anxiety attacks, bouts of watery diarrhea, and recent weight loss. Examination showed mild left sided facial droop.

Head MRI with contrast showed a 3.4x2.6x2.3 cm solid non-enhancing right temporal lesion with edema and mass effect. History of recent weight loss and diarrhea prompted a metastatic workup. CT of the abdomen and pelvis with contrast showed a 2.5x2.3 cm soft tissue mass within the stomach causing thinning of the greater curvature. EGD showed a prepyloric mass with normal gastric antral mucosa on biopsy. An ampulla of vater mass was also identified. Subsequent ERCP showed a 3 cm ampullary mass with pancreatic ductal dilatation. Biopsy showed a well differentiated Grade-I carcinoid tumor with strong diffuse staining for synaptophysin and weak focal staining for chromogranin. 24hr Urine 5-HIAA(5.9mg, N<8) was normal and Chromogranin-A levels(94ng/ml, N <93) was found to be slightly elevated.

In addition, she underwent right temporal craniotomy with complete resection of a benign grade I meningioma. Follow-up EUS after 2 months showed a heterogeneous non-circumferential mass in the ampulla measuring 30 by 18 mm in maximal cross-sectional diameter with sonographic evidence suggesting invasion into the pancreas. The patient is planned to undergo a modified Whipple's procedure.

This case illustrates a rare primary ampullary neuroendocrine tumor found incidentally on evaluation of a brain tumor along with the value of good diagnostic instincts. Ampullary carcinoids have annual incidence of 0.07/100,000 and usually present as jaundice, abdominal pain, or weight loss but without clinical/biochemical features of carcinoid tumors [3]. Histopathological diagnosis with ERCP is the gold standard but has low preoperative diagnostic rates due to the sub-mucosal nature of the tumor [3]. Whipple's procedure is the treatment of choice because of its propensity to invade lymph nodes and unreliable tumor staging [4].

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## HEMOLYTIC ANEMIA ASSOCIATED WITH AN OVARIAN MASS

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**Introduction:** Hemolytic anemias are associated with various malignancies, primarily hematological. We discuss a case of hemolytic anemia with cystic ovarian mass.

**Case:** 65 year old woman presented with pelvic cramping and dysuria to a local emergency department. A transvaginal ultrasound showed a 11.4cm x 12.8cm x 10cm ovarian cyst. CA-125 was elevated. Total abdominal hysterectomy with bilateral salpingo-oophorectomy was planned.

Preoperative hematocrit was 18%. MCV 96.4 fL, ferritin 482.4 ng/ml, LDH 501 unit/L, reticulocyte count 3.8%, haptoglobin 34 mg/dL, total bilirubin 1.8 mg/dL and direct bilirubin were 0.44 mg/dL. It was noted that blood clotted in the EDTA tubes instantaneously after phlebotomy, resulting in spuriously low hemoglobin levels (up to 3.7 g/dL). Cold agglutinins were considered and blood specimens were hereon kept at 37 - 40 C. The samples did not coagulate.

Direct Agglutination Test (DAT) was positive for C3 and negative for IgG. Antibody screen revealed a cold agglutinin. Thermal range study showed 4+ reactivity at 4C, 22C and 30C with no reactivity at 37C. There was no M spike on serum protein electrophoresis. Diagnosis of hematological malignancy was entertained. CT chest/abdomen/pelvis re-demonstrated the mass causing bilateral hydronephrosis without significant lymphadenopathy. Flow cytometry on peripheral blood was negative for abnormal B or T cells.

On resection, histology showed Diffuse Large B Cell Lymphoma (DLBCL). Bone marrow biopsy showed hypercellular marrow with no lymphoma. R-CHOP, a standard chemotherapy regimen for aggressive B-cell lymphomas, with Bortezomib was administered. After five cycles, the cold agglutinins were undetectable.

**Discussion:** Cold Agglutinin Disease (CAD) is rare (one in million), and characterized by autoimmune hemolytic anemia (AIHA) caused by cold reacting antibodies to erythrocytes. Diagnosis was considered due to hemolytic anemia and positive Coombs testing for anti-C3, and negative for anti-IgG.<sup>1</sup> The strength of reactivity and thermal activity confirmed the diagnosis. CAD is associated with Mycoplasma, Infectious Mononucleosis, hematologic malignancies and nonhematologic malignancies, primarily adenocarcinomas. Minority of secondary AIHA in Non Hodgkin's Lymphoma (NHL) are associated with cold antibodies (1.1-4.8% of all NHL - usually Lymphoplasmacytic Lymphoma and Chronic Lymphocytic Lymphoma).<sup>2</sup> We present an unusual case of localized DLBCL masquerading as CAD.

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ANTI-SYNTHETASE SYNDROME-RELATED INTERSTITIAL LUNG DISEASE IN AN OTHERWISE HEALTHY SWIMMER

First Author: Ahmadreza Moradi, MD Ariane Davis, MBBS Robert Dobbin Chow, MD

**Introduction:** To describe the clinical course of a patient with intercurrent infection and fever of unknown origin (FUO) who was diagnosed with the anti-synthetase syndrome (ASS) associated interstitial lung disease (ILD) positive for anti-alanyl-tRNA synthetase (anti-PL12) antibodies.

**Case Description:** Upon returning from a trip to Cambodia, a previously healthy 79-year-old woman developed upper respiratory symptoms. A chest x-ray demonstrated bilateral lower lobe interstitial changes, and she was treated with a course of antibiotics. She slowly improved, and was able to resume her usual vigorous lifestyle, which consisted of swimming half a mile a day. One year later, she developed fever, chills, chest pain, and increased shortness of breath and was treated with a course of antibiotics. Four months later, she had fever, cough, flu-like symptoms, and arthralgia, and was again treated with a course of antibiotics. Physical examination only revealed bilateral dry crackles on inspiration. Pulmonary function testing showed a FVC percent predicted of 89%; FEV1 percent predicted of 105%; TLC percent predicted of 94%; and a DLCO percent predicted of 65%. A CAT scan showed basilar ground-glass infiltrates and the bronchoscopy showed no evidence of active infection. She had a negative QuantiFERON with negative AFB cultures, despite positive PPD. However, she tested positive for PL12 antibody and was diagnosed with anti-PL12 ASS ILD.

Thus, the patient was treated with low dose mycophenolate, given her thin body habitus. Again, she developed fevers and chills, and was treated for suspected pneumonia. A detailed evaluation suggested that her symptoms were attributed to flare of ILD in the setting of lower than desired immunosuppression. She was then treated with 3 days of pulse steroids, and the mycophenolate dose was increased.

**Discussion:** Positive test results for anti-PL12 antibodies are particularly rare. ASS could present as an isolated diffuse ILD. Therefore, clinicians should consider an isolated ILD form of anti-PL12 syndrome on their differential diagnosis list in such a clinical context. Anti-P12 ASS ILD could mimic the presentation of pneumonia and might cause recurrent fever. In these patients, recurrent episodes of fever are not always infectious and might be part of the inflammatory process of the disease.

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A CASE OF LUNG ABSCESS MASQUERADING AS AN INTRA-THORACIC STOMACH

First Author: Jaskeerat Singh, MD

**Introduction:** Diaphragmatic/hiatal hernia is mostly secondary to trauma or occurs in patients with past history of multiple abdominal surgeries with gastric bypass being the commonest one. Lung abscesses are infections of the lung parenchyma commonly caused by aspiration pneumonia. Here we present an unusual case of a large lung abscess which was initially read radiographically as hiatal hernia with intra thoracic fluid filled stomach.

**Case report:** 39 y o female with PMH of multiple abdominal surgeries, including a recent gastric bypass, on high doses of oral hydromorphone presented with severe shortness of breath, cough and fever. Chest X-ray showed left sided opacification which on CT scan was read as intrathoracic fluid filled stomach. Patient was being evaluated for surgery when she went into respiratory failure requiring intubation and mechanical ventilation. Another CT scan was sought but this time with an oral contrast and interestingly NG tip was seen below the diaphragm. Thus the chest findings were assumed to be from a loculated cavitary lesion with air fluid level. Also no contrast was seen in the intrathoracic region. The abscess was finally drained with a chest tube, and was treated with culture specific antibiotics.

**Discussion:** Initial CT Scan in the above mentioned patient was read as Large fluid-filled structure occupying most of the left hemithorax appearing to be an intrathoracic stomach, due to a hiatal hernia. On the next day giving her oral contrast confirmed the presence of stomach below the diaphragm. CT is the most sensitive and specific imaging modality to diagnose a lung abscess. Contrast should be administered, as this enables the identification of the abscess margins, which can otherwise blend with surrounding consolidated lung. Although this patient had typical signs and symptoms of a lung abscess, lack of a questioning attitude to the radiological findings lead to a delay in insertion of a chest tube and thus early drainage of the abscess.

**Conclusion:** This case highlights the difficulties that may be faced while diagnosing a patient with a lung abscess. Intra-thoracic stomach can mimic radiological findings of a lung abscess and thus can present a challenge to appropriate medical management.

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## A SHOCKING CASE OF ADRENAL INSUFFICIENCY

First Author: Jaskeerat Singh, MD

**Introduction:** The relationship between electroconvulsive therapy (ECT) and central adrenal insufficiency has not yet been well established. Here we present a case of central adrenal insufficiency that developed quickly after ECT.

**Case description:** A 32 year old woman with a past medical history of severe major depression, generalized anxiety and seizure disorder, was referred from a psychiatric facility to the emergency department after a fall that occurred secondary to dizziness, which she has had multiple episodes of during the preceding month. On examination, she had pronounced orthostatic hypotension. Notably, this patient had received twelve sessions of ECT two months prior to the onset of these symptoms. A diagnosis of central adrenal insufficiency was made based on a low morning cortisol of 2.6 ug/dl, undetectable ACTH level, and an adequate response of serum cortisol to the low-dose ACTH stimulation test (26 ug/dl). Her symptoms and orthostatic hypotension significantly improved on initiation of hydrocortisone.

**Discussion:** The most common cause of suppression of adrenocorticotropic hormone (ACTH) is iatrogenic, secondary to the use of glucocorticoid medications to treat a large variety of illnesses. There was no use of steroids any point of time in the above mentioned case. Another cause of ACTH deficiency is in the setting of hypopituitarism secondary to traumatic brain injury (TBI), which usually resolves over time. The question is whether the effects of ECT are similar to that of TBI resulting in hypopituitarism. Conversely, there were two separate studies that revealed serum levels of TSH, ACTH, and Prolactin increase immediately after ECT sessions. There have even been case reports where ECT completely resolved hypothalamic-pituitary suppression caused by long term glucocorticoid use. In the medical literature, there is only one other case report of ECT induced central adrenal insufficiency.

Due to the chronology of this patient's development of central adrenal insufficiency soon after ECT, it is likely indicates a causal relationship.

**Conclusion:** ECT induced disruptions in hypothalamic-pituitary-adrenal hormonal regulation are common, mainly in the form of immediate release of hormones after a session. However, dysfunction of this axis after ECT is rare. There is scant evidence to support other types of hypothalamic-pituitary dysfunction such as central diabetes insipidus in patients who have received several sessions of ECT. Further research should be aimed towards the relationship between these neuroendocrine responses and the effects of ECT.

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## DOWNHILL ESOPHAGEAL VARICES DUE TO SUPERIOR VENA CAVAL STENOSIS

First Author: Srilakshmi Vallabhaneni, MBBS Second Author: Richard B. Williams MD FACP Third Author: Kenolisa Onwueme MD PhD

**Introduction:** Superior vena caval stenosis is a well-documented complication of central venous dialysis catheters. Cannulation of the vessel causes endothelial injury and subsequent fibrosis upon healing. This is further complicated by the rapid blood flow associated with hemodialysis, which induce endothelial proliferation, potentially leading to venous stenosis. Rarely, the obstruction is severe enough to cause the formation of downhill esophageal varices. Here we describe a patient with ESRD and upper GI bleed from endoscopy proven downhill esophageal varices due to superior vena caval stenosis.

**Case Presentation:** A 43 y/o female with ESRD on hemodialysis was admitted after having 4 episodes of frank blood in her vomitus on the day of presentation. Hematemesis was associated with epigastric pain, constant, radiating to the back and relieved by PPI in the emergency department. There was no previous history of similar episodes. She had a fistula in the left upper arm, which was done seven years ago. Her vitals were stable and on physical examination she had pale conjunctiva and engorged veins on the upper chest. The patient had an urgent endoscopy performed which showed Grade F3 varices extending from 10cm to 25cm in the proximal esophagus with a white nipple sign (indicating recent bleed). A superior vena cavogram was done which showed significant SVC stenosis for which an angioplasty was performed. She was discharged later with a follow-up appointment to be seen in three months for repeat angiography.

**Discussion:** This report describes a patient with ESRD who experienced a potentially life-threatening bleed from downhill esophageal varices, which developed secondary to trauma associated with central dialysis catheter insertion. Downhill, or upper esophageal varices, were first described by Felson and Lessure in 1964, and represent a rare cause of proximal gastrointestinal tract hemorrhage. They are dilated veins in the upper esophagus that most frequently form secondary to superior vena cava obstruction. To date, malignancy is the most common source of obstruction. Iatrogenic causes such as pacemaker insertion and hemodialysis access leading to stenosis of the SVC are far rarer. Acute upper gastrointestinal bleeding is the most common presentation of downhill varices.

**Conclusion:** To date, there are no definitive recommendations on how to screen for downhill varices, however we recommend a high index of suspicion for upper gastrointestinal bleeds in all hemodialysis patients with history of central venous access.

## DISSEMINATED VZV IN THE IMMUNOCOMPROMISED PATIENT

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Varicella zoster virus (VZV) is a rare but important cause of severe infection in immunocompromised patients. The spectrum of presentation can range from mild illness to fulminant respiratory failure resulting in death. A high level of clinical suspicion is required for diagnosis. Prompt initiation of anti-viral therapy is crucial to reduce morbidity and mortality.

A 41-year old African American woman with a deceased donor kidney transplant 3 months ago, on mycophenolate mofetil and tacrolimus, presented to the ER with a 3 week history of low grade fevers and cough with sputum. Initial Chest CT showed bilateral multifocal areas of consolidation. Bronchoalveolar lavage and blood tests for BK Virus, HSV, Aspergillosis, TB, Nocardiosis, and Histoplasmosis were negative. A diagnosis of 'atypical pneumonia' was made and she was treated with azithromycin and ceftriaxone with improvement in symptoms. Concomitantly, a rise in serum creatinine raised concern and a kidney biopsy revealed cellular and antibody mediated rejection. Given her improvement in respiratory symptoms, treatment of graft rejection was started. Anti-thymoglobulin and high dose steroids were administered, and she was started on plasmapheresis as an outpatient. The following week, patient returned to ER with a new onset diffuse rash and worsening shortness of breath, requiring intubation and mechanical ventilation.

The new onset of a polymorphic, vesicular rash and hypoxia then led a clinical diagnosis of disseminated VZV with pneumonia. Intravenous acyclovir was started immediately, along with broad spectrum antibiotics for prevention of secondary bacterial infections. A bronchio-alveolar lavage, PCR of skin lesions and high titres of VZV IgM in blood confirmed the diagnosis of disseminated VZV. On completion of antiviral therapy, patient was successfully extubated. A repeat CT Scan showed substantial decrease in previously apparent areas of consolidation.

This case illustrates the atypical manifestations of disseminated VZV in immunocompromised patients. Few case reports describe respiratory symptoms prior to a visible rash. Therefore, it is prudent to consider this diagnosis in transplant patients with mild respiratory symptoms even before appearance of rash. Her case was also challenging because improvement in symptoms of her initial presentation allowed for the treatment of the graft rejection with steroids. This prompts the question of whether steroids masked symptoms of a brewing VZV pneumonia or augmented its reactivation. Consideration of vaccination in patients prior to transplant is important to decrease the morbidity associated with disseminated VZV as immunization with a live virus vaccine is contra-indicated post transplant.

NOT YOUR AVERAGE SKIN LESION: POST TRAUMATIC CALCIPHYLAXIS IN A NONUREMIC RENAL TRANSPLANT PATIENT ON CHRONIC WARFARIN THERAPY

First Author: Scott Ferrara, DO John Bostrom, MSIV

Introduction: We present a case of calciphylaxis in a post renal transplant patient with excellent graft function after a mechanical fall responsive to intravenous sodium thiosulfate.

Case: 58 year-old man with HCV cirrhosis (liver a transplant in 1997), ESRD from calcineurin toxicity (kidney transplant in 2005) on sirolimus and mycophenolate, and atrial fibrillation on warfarin since 2010. He presented with worsening right lower extremity pain, redness, and swelling over 5 weeks. The symptoms started after a mechanical fall sustained while walking his dog. The fall resulted in abrasions to the posterolateral portion of his right calf. He was treated with two courses of oral antibiotics (amoxicillin and cephalexin) and had a lower extremity ultrasound negative for DVT prior to presentation in the emergency department (ED). In the ED he described stabbing pain, exacerbated by light touch, with occasional yellow discharge. He was tachycardic with otherwise unremarkable vitals. He had no leukocytosis, a baseline anemia, no electrolyte abnormalities and excellent renal graft function. He was admitted to the transplant nephrology service. Antibiotic coverage with vancomycin and cefepime was started. Sirolimus was discontinued to promote wound healing and he was started on cyclosporine instead. An MRI was negative for osteomyelitis or abscess formation. Bacterial and fungal blood cultures remained negative. Dermatology was consulted and performed a punch biopsy. The biopsy was negative for bacterial, fungal or mycobacterial elements. The biopsy revealed epidermal necrosis and foci of small vessel calcification consistent with a diagnosis of calciphylaxis. A workup for the etiology of the calciphylaxis was done. Calcium metabolism disorders, hypercoagulable states, and cryoglobulinemia were ruled out. Warfarin was considered the etiology as a diagnosis of exclusion. Warfarin was discontinued and he was started on enoxaparin. Intravenous sodium thiosulfate was started three times weekly and continued after discharge. Three months later he was seen in dermatology clinic with a 50% improvement in the surface area of his rash and significant improvement in his pain level.

Discussion: Calciphylaxis is a disorder of unclear pathophysiology causing calcium deposition in the intima of small vessels within the dermis. It most commonly presents as a stellate rash with necrotic foci, in the proximal lower extremities. It has been reported in 1-4% of ESRD dialysis patients. Additional risk factors include calcium homeostasis disorders, hypercoagulability, obesity, diabetes and warfarin use. We present a particularly rare case of calciphylaxis in a patient post transplant with excellent graft function with a trauma as a precipitating event. The most promising therapy through an unclear mechanism is intravenous sodium thiosulfate. Our patient responded well to this treatment. The role of sodium thiosulfate should continue to be studied as it has shown to be beneficial in a disease with a historically high mortality due to sepsis and adverse effects of pain medications.

PERSEVERANCE BY A PERVASIVE DISEASE (PPD)

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**INTRODUCTION:** Miliary tuberculosis is characterized by disseminated *Mycobacterium tuberculosis* (TB) infection, with radiological findings suggestive of millet seeds. The incidence is about 2% of extra pulmonary TB. Pott's disease is the involvement of the spine by *Mycobacterium tuberculosis*. It usually is comprised of osteomyelitis and arthritis involving multiple vertebrae. Paraplegia, impaired sensation, nerve root pain, cauda equina syndrome, among others, are the feared complications.

**CASE:** A 43 year old Brazilian male who immigrated to the US 16 years ago, with his last travel to Brazil having been in 2006, presented with intermittent fevers and drenching night sweats for 8 months, along with low back pain and weight loss of 40 lbs. He was exposed to active pulmonary TB at age 26, but documented a negative PPD skin test at the time of immigration. He was on no medications and had no pertinent family history or risky behaviors. Examination at admission revealed a comfortable gentleman with fever of 101.4°F and limited anterior flexion of spine due to pain, without point tenderness, masses, or skin changes. Laboratory data showed a hemoglobin of 13.1gm/dl, WBC 5900 with 35% bands and negative blood cultures. Serology for HIV was negative. Quantiferon Gold proved positive. A chest X-ray was unrevealing, but a CT of the lumbar spine raised concerns for an epidural abscess and osteomyelitis at L4/L5. Laminotomy with decompression of the abscess and bone biopsy revealed a negative gram stain, AFB smear and cultures. Fevers continued to spike up to 103°F, and vancomycin was started empirically with no response. A chest CT done one week after admission revealed multiple bilateral pulmonary nodules in a miliary pattern. BAL via bronchoscopy was negative, as were a transbronchial biopsy for gram stain, AFB smear and initial cultures. Histopathological examination revealed necrotizing granulomas with bacilli consistent with *Mycobacterium tuberculosis*. The patient was started on isoniazid, pyridoxine, rifampin, pyrazinamide, and ethambutol and showed clinical improvement without further febrile episodes within one week. Cultures for *Mycobacterium TB* were reported negative after 8 weeks.

**DISCUSSION:** Miliary TB has likely involved our patient's lungs and spine. Given the preliminary negative culture results from his bone and epidural abscess, we cannot as yet diagnose Pott's disease with complete confidence, but the lack of evidence of any other infection and the patient's response to antituberculous medications makes it most likely.

According to the literature, this kind of presentation of miliary TB with Pott's disease is rare. In 62-90% of cases with Pott's disease there is no extra-spinal involvement. On a closing note, this case with widespread involvement in an immunocompetent patient reiterates the need for a high degree of clinical suspicion to diagnose TB.

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MANAGEMENT OF SPONTANEOUS UMBILICAL HERNIA DISRUPTION IN A CIRRHOTIC PATIENT.

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There is controversy regarding the timing of an umbilical hernia repair in patients with alcoholic liver disease and ascites.

A 70 year-old male cirrhotic (Child Pugh C) patient was referred to the Emergency Department with abdominal pain and giant erythematous umbilical hernia (since two years ago) with massive ascites discharge from the umbilicus (20 liters). There was no history of bleeding, fever, hepatitis or trauma to umbilicus region. Physical examination was remarkable with grade 3 ascites, portal hypertension syndrome, grade 3 hepatic encephalopathy, malnutrition, a giant non-bleeding ruptured umbilical hernia with massive ascites discharge and two inguinal hernias.

Treatment was initiated with terlipressin, albumin, parental antibiotics and colloids to manage liver function. We wait 72 hours until the patient's renal, liver and hemodynamic functions improved. Four days later, the patient underwent laparotomy to repair the umbilical hernia and two more inguinal hernias. He recovered well and seven days after surgery, the patient was discharged. He continues follow up at the outpatient clinic.

This case illustrates the importance of managing liver function and hemodynamics in patients with spontaneous rupture of hernia due to massive ascites before surgery.

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MISDIAGNOSIS OF MYCOBACTERIUM CELATUM: TB OR NOT TB?

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*Mycobacterium Celatum* is a slowly-growing nontuberculous mycobacterium (NTM) shown to cause symptoms similar to pulmonary *Mycobacterium Tuberculosis*. Certain strains have been shown to cross-react with the probes used to detect *M. tuberculosis*, making this a diagnostic challenge.

A 56 year old gentleman with a past medical history of common variable immune deficiency (CVID), bronchiectasis on inhaled tobramycin therapy, and chronic obstructive pulmonary disease on 2 litres of home oxygen, presents to hospital with refractory *Clostridium Difficile* diarrhoea. He had failed medical management with oral antibiotics and subsequently received 4 fecal transplants over a 2-week period with no effect. He finally responded to long term oral vancomycin therapy. Prior to discharge, his hospital course was complicated by increasing oxygen requirements, from his home 2L/minute to 6L/minute. Lung auscultation revealed diffuse rhonchi and decreased breath sounds in the right lung fields. He was afebrile and his labs reflected his diagnosis of CVID with a variable white count of 2.5 to 6 x10<sup>9</sup>/L. His sputum remained clear/white but increased in amount and frequency. Chest x-ray was performed which showed diffuse consolidation throughout the right lung consistent with pneumonia and he was started on IV antibiotics for treatment of hospital acquired pneumonia (HAP). 3 days later, the patient still remained short of breath and hypoxic so computed tomography (CT) scan was performed of the chest. This showed consolidation within the right lung apex with areas of central lucency suggesting cavitation. Serial sputum samples were positive for acid-fast bacilli and nucleic acid amplification testing (NAAT) identified *M. Tuberculosis* rRNA. The patient was started on isoniazid, ethambutol, pyrazinamide and rifampin while awaiting culture results. During this time, contact tracing and public health interventions were performed. Two weeks later, high performance liquid chromatography showed a pattern consistent with *M. celatum* and the 4-drug regime was switched to ethambutol, azithromycin and moxifloxacin and the patient was eventually discharged from hospital after a 6-week admission.

This case illustrates the potential for *M. celatum* to mimic *M. tuberculosis* in both its clinical presentation and laboratory testing due to the similar oligonucleotide sequence contained in both. Despite its rarity and the reported high sensitivity of the NAAT to diagnose *M. Tuberculosis*, there is an increasing number of case reports suggesting early differentiation could reduce unnecessary treatment and public health intervention associated with misdiagnosed TB.

ACUTE STEMI SECONDARY TO POSTPARTUM CORONARY ARTERY DISSECTION, AN EXTREMELY RARE BUT A FATAL TRAUMATIC EVENT!! SHOULD WE BE MORE AWARE OF IT?

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Introduction: Spontaneous coronary artery dissection (SCAD) is an uncommon etiology of acute coronary syndrome and sudden cardiac death. It primarily affects young healthy women with no known atherosclerotic risk factors, with 30% cases occurring in peripartum or postpartum period. It mainly involves the left main stem or left anterior descending artery or both. The cause of primary SCAD is still unclear and majority of cases are diagnosed on autopsy.

Case Presentation: A 35 year old african american female patient, G5P3-0-1-3, with no significant past medical history presented eight days post partum with sudden onset left sided chest pain that started two hours prior to admission and woke her up from sleep. She described the chest pain as sharp, crushing, 10/10 in severity and radiating down her left arm. She was diaphoretic and dsypneic but denied any other symptoms. Vital signs were stable and physical exam was normal. EKG on presentation showed antero-lateral ST segment elevation myocardial infarction. Two troponin readings were more than 40 ng/ml. Left heart cardiac catheterization revealed proximal to distal dissection of left anterior descending coronary artery. Three overlapping drug eluting stents were placed and TIMI-3 flow was re-established following which her symptoms resolved. 2D Echo showed severe left ventricular dysfunction with ejection fraction of 25%. She was started on aspirin, ticagrelor, lisinopril, carvedilol and rosuvastatin. Patient was discharged on lifevest as she was having frequent runs of non sustained ventricular tachycardia (NSVT). She was counseled on medication compliance, smoking cessation and permanent birth control with tubal ligation. We also recommended cardiac rehabilitation and outpatient follow up for monitoring of left ventricular systolic function.

Conclusions: SCAD does not have any warning signs or specific identifiable risk factors, therefore it is difficult to diagnose before it actually causes myocardial infarction. Timely diagnosis of this potentially fatal condition helps in deciding the appropriate treatment which is conservative for distal lesions, percutaneous coronary intervention (PCI) for single vessel dissection, and coronary artery bypass grafting (CABG) for left mainstem dissection or multi-vessel involvement. Our patient underwent PCI. She followed up eleven months later with significant improvement in left ventricular ejection fraction. Her lifevest was discontinued and she was advised to continue medical therapy.

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NAIL TO THE HEART: NO BIG DEAL.

First Author: Yashwant Agrawal MD Second Author: Sandeep Patri MD Third Author: Monoj Konda MD Fourth Author: Hardik Chhatrala MD Final Author: Vishal Gupta MD MPH

Introduction: Penetrating nail gun injuries to the heart are extremely rare and usually fatal with a reported mortality of 25% due to rapid hemodynamic compromise. Our patient is a rare case that survived a nail gun injury to the heart requiring no intervention.

Case: A 32-year-old male with no significant medical history presented to the ER with non-radiating, burning type substernal chest pain of 7/10 intensity which started 30 minutes back. He reported a nail gun accidentally being discharged at work few hours before presentation. The nail had penetrated his right chest next to the right sternal border. He removed the nail which he described as being 3.5 inches in length and continued working until the day's end. Examination was significant for puncture wound at right sternal border in the right fourth intercostal space and pericardial friction rub over the precordium. EKG revealed diffuse ST-segment elevation consistent with pericarditis. Chest X-ray showed widened mediastinum with possible hematoma of the right hilum and upper mediastinum. Labs including troponins were normal. CT chest revealed moderate pericardial effusion and no other significant findings. Cardiology was consulted and an emergent TTE was performed. It showed penetration injury at right ventricular apex with moderately dilated cavity and moderate pericardial effusion, larger on the RV free wall. No tamponade physiology or evidence of hemodynamic compromise was identified. He was started on 50 mg of indomethacin three times a day to prevent Dressler's syndrome and admitted to ICU for continued management. Serial TTE were performed which revealed decreasing size of the pericardial effusion. He was continuously monitored in the ICU and with no hemodynamic compromise, discharged after 3 days on indomethacin to be taken for a month. He had a follow up appointment 3 months later, when a TTE was done showing complete resolution of his pericardial effusion.

Discussion: In this case the injury was to the right ventricle as a puncture wound. This was small enough that it led to a slow leak into the pericardial space without causing any significant hemodynamic compromise. Had it been a larger penetration it would have led to catastrophic hemodynamic instability from massive hemo-pericardium and tamponade physiology, which is commonly seen in these patients. Penetrating injuries of the right heart usually have lower mortality compared to those of the left heart as it is low pressure system. Hemodynamically stable patients are managed conservatively by obtaining a chest X-ray, TTE, CT chest to reveal the extent of cardiac injury and to rule out injury to other mediastinal structures. Hemodynamically compromised patients warrant urgent pericardiocentesis or surgical intervention to relieve the tamponade physiology and if required heart-lung support. Mediastinal survey to repair co-existing injuries should also be performed in such patients. This case highlights the importance of obtaining appropriate imaging studies in penetrating chest wall injuries and utilization of anti-inflammatory medications to prevent expected complications.

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FATALITY IN KIKUCHI-FUJIMOTO DISEASE: A RARE PHENOMENON

First Author: Bianca Barbat, MD Second Author: Ruby Jhaj, MD Third Author: Daniyeh Khurram, MD

Kikuchi-Fujimoto disease (KFD), or histiocytic necrotizing lymphadenitis, is a rare, self-limiting condition characterized by regional lymphadenopathy, fever, night sweats, and upper respiratory symptoms. Although a viral or autoimmune pathogenesis has been suggested, the etiology remains unknown. Recognition of KFD is essential because it can easily be mistaken for lymphoma, tuberculosis, or carcinoma.

Here we report the case of a healthy, 21-year-old female who presented with a two day history of shortness of breath, fever, and malaise. A chest x-ray demonstrated multilobular pneumonia and treatment with ceftriaxone and azithromycin was initiated. Clinically worsening, the patient required intubation for respiratory distress. A CT chest, abdomen, and pelvis was performed revealing significant cervical and axillary lymphadenopathy, bilateral lung consolidation, and a moderate pericardial effusion. To rule out other significant pathology, such as lymphoma, a cervical lymph node biopsy was performed revealing necrotic regions composed of karyorrhectic debris with abundant histiocytes consistent with KFD. The patient was provided with high dose steroids; unfortunately, she developed disseminated intravascular coaguloapthy (DIC) and died.

Typically, KFD has a benign self-limiting course. Symptomatic and supportive treatment is usually adequate. In severe cases, high dose steroids have been shown to be effective. On extremely rare occasions, KFD may progress to mortality, such as in our patient. A proposed mechanism for DIC in KFD involves a massive cytokine release by activated mononuclear cells within lymph nodes affected by the disease. In our review of the literature, this is the fourth documented case where KFD has led to DIC and fatality.

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BILATERAL RENAL ARTERY STENOSIS PRESENTING AS POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME

First Author: Hardik Satish Chhatrala, MD MPH Christopher Di Felice, MD; Yashwant Agrawal, MD; William Nichols, MD

Posterior Reversible Encephalopathy Syndrome (PRES) which manifests as headaches, visual disturbance, altered sensorium, and seizures is characterized by posterior cerebral white matter edema on MRI. It is commonly associated with hypertensive encephalopathy. Recognizing bilateral renal artery stenosis (RAS) as an underlying etiology needs high clinical suspicion.

A 62-year old lean woman with history of labile hypertension, tobacco abuse and headaches was found unresponsive at home. She was brought to the ED where she had two generalized tonic clonic seizures requiring intubation. Her BP of 240/130 was controlled with intravenous nicardipine to an initial target BP 180/100. Serum creatinine was elevated at 1.3 (baseline 1.0). Lumbar puncture ruled out meningitis. When CT brain was unremarkable, an MRI brain was done which showed extensive abnormal elevated T2 and FLAIR signal in a pattern suggesting PRES.

Continuous electroencephalogram ruled out further subclinical seizures. Owing to slow neurological recovery, the patient required five days of mechanical ventilation. Since admission, she required oral metoprolol, losartan, and amlodipine at high doses along with intravenous nicardipine drip to maintain her BP under 150/90. Clonidine and hydrochlorothiazide replaced nicardipine on her transfer to general medical floor. Encephalopathy gradually cleared around Day 10. While exploring for causes of secondary HTN, a renal ultrasound with doppler noted focal high velocity jet at 4.6 m/s in the main left renal artery (LRA) with a normal size kidney and an atrophic right kidney. MRA for renal arteries revealed high-grade stenosis near the LRA origin but widely patent distal artery and high-grade stenosis of right renal artery at the origin with diffuse stenosis of distal artery.

Significant atherosclerotic aortoiliac occlusive disease as well as mesenteric occlusive disease was also noted. Patient underwent LRA and superior mesenteric artery stenting. BP improved remarkably post procedure requiring only two low dose antihypertensives. Patient was discharged home. This case illustrates PRES in the setting of renovascular HTN as a rare complication of bilateral RAS and the value of searching of causes of resistant HTN. Recognizing and treating severe RAS is key to prevent hypertensive crisis in future.

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HIGH-DOSE INSULIN AS AN INOTROPIC AGENT

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**Introduction:** Medication overdose is a common presentation in patients admitted to the ICU, and most cases do not warrant contacting poison control. Recognizing when to do so, however, can be life-saving as evidenced by this case of polypharmacy overdose successfully managed with high-dose insulin (HDI) therapy as an inotropic agent. Review of recent literature shows that HDI is emerging as a first-line treatment for severe cases of either beta-blocker or calcium channel blocker overdose. This case demonstrates its efficacy in an overdose involving both medications concomitantly.

**Case:** A 50-year-old woman was admitted to the ICU after being found unconscious surrounded by empty bottles of beta-blockers, calcium channel blockers and narcotics. Naloxone given en route to the hospital resulted in little response. She was intubated for airway protection upon arrival and shortly thereafter developed bradycardia. Glucagon and calcium therapy were initiated along with intravenous fluid administration with limited response. Her hemodynamic status continued to decline and she developed cardiogenic shock. Vasopressor therapy was initiated with dopamine and norepinephrine. Poison control was contacted to review the case and they recommended high-dose insulin therapy and faxed complete algorithms to the ICU. HDI achieved hemodynamic stability. She fully recovered from the overdose and is now undergoing treatment for depression.

**Discussion:** Beta-blocker and calcium channel blocker overdose is common and potentially life-threatening if severe enough to cause cardiogenic shock. Conventional therapies including fluids, vasopressors, calcium and glucagon do not always achieve hemodynamic stability. Case series have demonstrated success with high-dose insulin therapy with early initiation when conventional treatments fail in cases of beta-blocker or calcium channel blocker overdose. High-dose insulin (bolus of 1 unit/kg followed by an infusion of 0.5-2.0 units/kg/h) works by increasing inotropy, causing vascular dilatation and increasing intracellular glucose transport. Therapy requires co-administration of concentrated dextrose to prevent hypoglycemia, necessitating frequent lab monitoring. In our case, the patient required D20 to maintain euglycemia. Permissive hypokalemia (as low as 2.8 without supplementation) is also a mainstay of therapy in order to prevent hyperkalemia upon discontinuation of HDI. This case demonstrates a successful outcome using HDI as an inotrope in an overdose involving both beta-blockers and calcium channel blockers. HDI is just now emerging as a superior treatment in severe cases of calcium channel blocker or beta-blocker overdose with development of poison induced cardiogenic shock. For best outcomes, it should be considered as a first-line agent at the onset of cardiogenic shock rather than waiting for a patient to become refractory.

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## A RARE CASE OF MESENTERIC PARAGANGLIOMA

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Paragangliomas or extra adrenal pheochromocytomas are rare neuroendocrine tumors that arise from the extra-adrenal autonomic paraganglia so they can be found anywhere from the upper neck to the pelvis. Most paragangliomas secrete catecholamines, but they can also be nonfunctional. Paraganglioma as a mesenteric tumor is extremely rare and has been published as case reports only.

A 66 year-old man with a history of hepatitis C was being followed in the infectious disease clinic. He was noted to have a mesenteric mass measuring 4x5x6 cm on a routine abdominal computed tomographic scan that was done to evaluate his liver disease. The mass was hypervascular with small areas of low attenuation; it was anterior to the pancreatic head and posterior to the colon. The patient refused further evaluation for the mass. Two years after the initial presentation, he had another follow up abdominal computed tomographic scan. This showed enlargement of the mesenteric mass. Subsequently, he underwent core biopsies of the mass. The immunohistochemical and the morphologic findings supported the diagnosis of low-grade neuroendocrine neoplasm and a paraganglioma was favored. He underwent laparoscopy with excision of the mass. The mass was composed of nests of cuboidal cells compartmentalized by a rich vascular network compatible with the diagnosis of paraganglioma.

Paragangliomas represent only 10% of chromaffin tissue tumors, and the mesenteric form seems to be a rare occurrence, but should be among the preoperative differential diagnosis of abdominal masses of unknown etiology. Most paragangliomas are benign, but about 15 to 35 percent are malignant. Metastatic spread is the only reliable indicator of malignant potential. The recognition of these tumors as a cause of an abdominal mass is extremely important although it could be a true diagnostic dilemma even for the pathologist; however, cytologic appreciation of neuroendocrine features of the tumor cells may help to avoid an erroneous diagnosis of carcinoma or sarcoma. Surgical excision is the treatment of choice for mesenteric paraganglioma. Radiation therapy is reasonable alternative to surgery if resection would require sacrifice of critical vascular and/or neural structures. Chemotherapy has been proven ineffective in previous cases.

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SEVERE AND SUDDEN THROMBOCYTOPENIA: A POTENTIALLY DEADLY COMPLICATION OF GP IIB/IIIa INHIBITOR THERAPY

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**Introduction:** Glycoprotein IIb/IIIa inhibitors are commonly used in percutaneous coronary interventions. While adverse events related to bleeding have been well-described in the literature, severe and sudden thrombocytopenia is another important, albeit uncommon, adverse reaction to this class of medication. We present a patient who developed severe thrombocytopenia within hours of receiving eptifibatide during cardiac catheterization.

**Case Presentation:** A 56-year-old-male presented to the hospital for acute lower limb ischemia and underwent angioplasty on his femoral artery. During that hospitalization, he had a positive stress that warranted cardiac catheterization with placement of a drug-eluting stent in the left anterior descending coronary artery. The morning of the procedure, the patient's platelet count was 302,000. He received a double bolus and drip of eptifibatide as is standard practice prior to heart catheterization. Afterwards he became hypotensive, developed gum bleeding, and petechiae. Repeat bloodwork revealed a platelet level of 3,000. Peripheral blood smear confirmed thrombocytopenia without schistocytes and without platelet clumping in the test tube. Heparin-Induced Thrombocytopenia was considered less likely, however a heparin platelet factor 4 antibody was tested and was negative. The patient was transfused two units of platelets, given IVIG and steroids and his platelet count began to rise two days later.

**Discussion:** Given the widespread utilization of GPIIb/IIIa inhibitors during catheterization, medication-induced thrombocytopenia is an important consideration as it can be severe and life-threatening. This diagnosis should be included in the differential for sudden, severe thrombocytopenia. Rapid treatment and monitoring, as was done in our case, can prevent morbidity and mortality.

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TRANSMURAL GASTRIC HEMANGIOMA: AN USUAL CAUSE OF ACUTE ABDOMEN.

First Author: Daniel E Ezekwudo, MD,Ph.D; Srinandan Guntupalli MD; Asma Taj MD

**Introduction:** Gastric haemangioma accounts for about 0.05% of all gastrointestinal (GI) neoplasms. There is currently no pattern to their presentation. However, given the submucosal localization of gastric hemangiomas, their high vascularity and the need for biopsy, significant challenges are encountered in confirming diagnosis. Currently, endoscopic ultrasonography (EUS) and contrast-enhanced computed tomography (CT) have been shown to play crucial roles in establishing a diagnosis. Here, we report the case of a 53-year-old male patient who was admitted due to epigastric pain.

**Case presentation:** A 53-year old patient presented with non-radiating, constant epigastric pain of 2 day duration. He also complained of nausea and vomiting for same period. On the first day he was stabilized in the emergency room and discharged with Prilosec. However, he returned to the emergency room the following day due to worsening abdominal pain with nausea and vomiting. His past medical history is significant for myocardial infarction, hypertension and type II diabetes mellitus. Physical examination: good general clinical status, stable vital parameters. No palpable mass was detected during abdominal examination. The laboratory test results, including liver and renal function tests, complete blood count, coagulation profiles and tumor markers were within normal limits. Abdominal ultrasound revealed a liver mass in the posterior left lateral lobe measuring 69 X 60 X 34 mm which was suspicious for malignancy. This finding was in turn confirmed by contrast-enhanced computed tomography (CT) of abdomen followed by endoscopic ultrasound –guided fine needle aspiration. Cytology report revealed rare degenerated atypical cells in the background of inflammatory cells and macrophages suggesting a cystic component. Patient subsequently underwent surgical resection.

**Discussion:** Hemangiomas are congenital in nature originating from the mesenchymal tissues. They may involve the skin, internal viscera, or both, with a predilection for the head and neck region (60%), the truncal region (25%), and the extremities (15%). However, liver remains the most frequent site of involvement among the internal viscera (0.4% - 20%). The mode of presentation varies from upper GI bleed to acute abdomen as presented in this case. Other gastric submucosal masses such as stromal tumors, leiomyomas and lipomas should be kept in mind in the differential diagnosis of hemangiomas due to their frequent submucosal localization. Surgery remains the curative treatment. Currently, wedge resection, partial or total gastrectomy constitute the standard treatment modalities. In summary, transmural gastric hemangiomas are rare and benign tumors of the stomach. Use of endoscopy in conjunction with CT is beneficial for diagnosis, furthermore, surgical treatment along and histopathological studies occupy an important place in the establishment of a definitive treatment.

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A SPONTANEOUSLY REGRESSED SEMINOMA PRESENTING AS A UNILATERAL INGUINAL MASS

First Author: John Fleming, DO Christopher DiFelice, MD, Hardik Chhatrala, MD, Joanne Baker, DO

**Introduction:** Testicular cancer is relatively rare, accounting for approximately 1-2 percent of all male cancers. Germ cell tumors (GCTs) account for 95 percent of testicular cancers with seminomas being the most common histologic subtype. Metastatic disease at presentation is an unusual occurrence seen in less than fifteen percent of patients. The retroperitoneal lymph nodes are the typical site of spread while inguinal node involvement is far less common.

**Case Description:** A 42 year old man presented to the clinic with the chief complaint of a groin mass. He had no constitutional symptoms. Pertinent historical findings included hydrocelectomy at age sixteen, and family history of large cell lymphoma. The physical exam was remarkable for a large right-sided inguinal mass that was firm, fixed, non-tender and non-reducible. No overlying skin changes were apparent. No additional masses or lymphadenopathy were found.

CT scan of the abdomen and pelvis showed right internal iliac and inguinal adenopathy. An open biopsy of the right inguinal node was performed; results were consistent with metastatic seminoma. Testicular ultrasound revealed a smaller right testis with mild diffuse heterogenous echogenicity suggesting atrophy and fibrosis. Radical orchiectomy of the right testis revealed extensive atrophy and scarring within multiple sections of the testicular parenchyma.

The patient was referred to oncology to begin systemic chemotherapy with cisplatin and etoposide. He completed four cycles with follow-up CT showing resolution of all lymphadenopathy.

**Discussion:** Testicular metastasis typically occurs via efferent lymphatics draining alongside the gonadal vessels to the retroperitoneal para-aortic lymph nodes. Involvement of the iliac and inguinal lymph nodes in testicular cancer is unusual; however, it can be related to local invasion of the tumor into the tunica vaginalis or scrotal skin, bulky retroperitoneal lymphadenopathy, and previous surgical manipulation of the inguinoscrotal region. The latter is the most likely mechanism of spread in our patient who had a history of hydrocelectomy.

Another unique feature to our case is the absence of a testicular mass on exam. This can likely be explained by spontaneous regression of the primary tumor. This is a well-recognized phenomenon marked by a distinct constellation of findings on biopsy. In a large case series of patients who were found to have metastatic disease at presentation without a palpable testicular mass, biopsy specimens following orchiectomy showed irregular foci of scarring, distinct from the adjacent parenchyma, in association with widespread testicular atrophy.

This case poses a diagnostic challenge for the internist who may not suspect inguinal adenopathy arising from testicular cancer. A thorough history to include previous inguinal surgery may aid in uncovering the diagnosis.

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NOT ALL SCLERODERMA IS THE SAME SCLERODERMA

First Author: John Fleming, DO Christopher DiFelice MD, Mark Schauer MD

**Introduction:** Scleroderma, also called systemic sclerosis, is actually a group of disorders characterized by abnormal inflammation and subsequent overproduction of collagen resulting in skin thickening and hardening, along with multi-organ system involvement, and vascular dysfunction. Women are affected four to nine times more often than men and are generally diagnosed between ages of twenty and fifty years old. Although clinical presentation is the foundation of diagnosis, there are numerous serologies that help support a diagnosis of scleroderma, for example: ANA, Anti-Scl-70, Anticentromere antibodies, anti-RNA polymerase III.

**Case Report:** 75 year old white male initially noticed finger swelling and polyarthralgias eight months prior to admission. Subsequently he developed worsening skin thickening advancing proximally to mid-forearm and mid-thigh. He was diagnosed with scleroderma. The skin thickening progressed until his walking was impaired by the reduced range of motion at his knees and ankles. He could not flex his fingers into a fist and exhibited facial involvement with decreased mouth opening at 40mm. Intermittent skin pruritis occurred over the affected areas. He complained of progressively worsening dyspnea on exertion and underwent HRCT which confirmed interstitial pulmonary fibrosis. He was initially treated with diltiazem, losartan, methotrexate, and prednisone. His hypertension worsened and continued to be difficult to control. The addition of mycophenolate mofetil increased his range of motion somewhat. However, his renal function progressively declined during the four months after diagnosis. He underwent renal biopsy which demonstrated ATN and findings consistent with scleroderma renal crisis. While awaiting the results of this biopsy, his dyspnea and fatigue acutely worsened, and he was admitted to the inpatient medical floor. Hemodialysis was initiated. Captopril was used to control his hypertension. Serology revealed that he was PM-SCL negative but he was anti-RNA Polymerase III strongly positive at >150.

**Discussion:** This case is unusual for several reasons. First, the patient was an elderly male. Scleroderma usually presents in younger women. His disease, anti-RNA polymerase III positive scleroderma, is rapidly progressive. Typically other sclerodermas demonstrate a more indolent course. In addition, he was Scl-70 negative and yet had severe systemic disease including skin, lung, and renal manifestations, which is a hallmark of anti-RNA polymerase III positive scleroderma. Anti-RNA polymerase III positive scleroderma is a significant risk factor for scleroderma renal crisis which this patient developed. In addition, scleroderma renal crisis can be precipitated by corticosteroids which he received. It should be kept in mind that not all scleroderma is the same and that there are significant differences in disease manifestations and progression which can in part be predicted by serology.

AN UNUSUAL CASE OF CHOLANGIOCARCINOMA PRESENTING AS NON-HEALING DUODENAL ULCER

First Author: Ewa Gniado, MD Lohit Garg, MD Veslav Stecevic, MD

Bile duct carcinoma is a rare carcinoma arising from the epithelial cells of intrahepatic and extrahepatic bile ducts. Cholangiocarcinoma usually presents with obstructive jaundice, fever and weight loss. We present a case of cholangiocarcinoma presenting as hematemesis and nonhealing duodenal ulcer.

A 71-year-old female with past medical history of aortic stenosis and sigmoid diverticulosis presented to our hospital with 2 episodes of hematemesis. She also complained of abdominal pain for 4 months, accompanied by nausea and loss of appetite for 1 month. She denied melena, pale stools, dark urine, fever, heartburn, or weight loss. She had a colonoscopy and esophagoduodenoscopy (EGD) performed 2 years prior to the admission, which were unremarkable. She was in no acute distress and her vitals were stable. On physical exam there was no icterus, or pallor. Abdomen was soft, nontender, and no masses were palpated. Her laboratory tests revealed anemia. Liver function tests were unremarkable except chronic mild alkaline phosphatase elevation. The patient was started on pantoprazole infusion and scheduled for EGD that revealed normal esophagus, non-bleeding erosive gastropathy, a single non-bleeding cratered duodenal ulcer with mass effect and severe edema causing stenosis in the first part of the duodenum distal to bulb. Biopsies were negative for inflammation, dysplasia or carcinoma. She was discharged on omeprazole twice daily. She underwent another EGD in 2 months, with findings of persistent duodenal stenosis and a large mass with no bleeding in the duodenal bulb. She was scheduled for a CT abdomen/pelvis and endoscopic ultrasound (EUS). CT showed increasing prominence of the biliary tree and both intra and extrahepatic dilation without a definite pancreatic head mass or common bile duct (CBD) abnormality. EUS revealed 1.5 x 1.5 cm subepithelial mass in the 2nd portion of the duodenum. Unfortunately, fine needle aspiration of the duodenal mass was non-diagnostic. She underwent magnetic resonance cholangiopancreatography (MRCP) that displayed significant intrahepatic and extrahepatic biliary ductal dilatation, and abrupt narrowing of the CBD at the level of the pancreatic head without evidence of an enhancing mass or lesion. One week after MRCP she presented to the hospital with a 2-day history of new onset jaundice. Lab testing revealed transaminitis, and direct hyperbilirubinemia. She underwent pancreaticoduodenectomy with results showing moderately differentiated invasive adenocarcinoma of the distal CBD, with invasion of duodenal wall, pancreas, peripancreatic soft tissue and spread to lymph nodes.

This case illustrates an unusual presentation of extrahepatic cholangiocarcinoma with nonhealing duodenal ulcer. We suggest that in the presence of anemia, non-specific gastrointestinal symptoms and nonhealing duodenal ulcers, a diagnosis of cholangiocarcinoma is considered. Moreover, this case serves to highlight the principle that the persistence of symptoms despite appropriate therapy should prompt clinician to establish a clear diagnosis.

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DIFFUSE ALVEOLAR HEMORRHAGE ASSOCIATED A NOVEL ORAL ANTICOAGULANT

First Author: Brian J Grondahl, DO; Megan Benedict, DO, Keren Shahar, MD; Heather Laird-Fick, MD

Introduction: Novel anticoagulants such as Rivaroxaban, a direct Factor Xa inhibitor, have presented advancement in the management of venous thromboembolism and atrial fibrillation. The most common complication of Rivaroxaban treatment is bleeding, and diffuse alveolar hemorrhage (DAH) has only been reported in one case in the literature. We present a case of Rivaroxaban associated diffuse alveolar hemorrhage.

Case Report: The patient is a 45 year-old woman with a past medical history of Systemic Lupus Erythematosus (SLE) complicated by lupus nephritis, antiphospholipid syndrome (APS) with aortic and renal artery thrombosis, and autoimmune hemolytic anemia. The patient was anticoagulated with Rivaroxaban for arterial thrombosis. Her renal function was stable with a glomerular filtration rate of 51. She was on anticoagulation with 20mg of rivaroxaban daily for the preceding 19 days. She presented to the hospital with complaints of shortness of breath and hemoptysis. Her condition deteriorated quickly and she was transferred to the intensive care unit and was intubated and mechanically ventilated. Imaging of the chest revealed bilateral opacifications. Laboratory studies revealed an acute on chronic anemia with a hemoglobin of 5.8 (baseline of approximately 8.5). A bronchoscopy was performed and confirmed the diagnosis of diffuse alveolar hemorrhage. Rivaroxaban was held and she was treated with red blood cell transfusion, high dose steroids, and intra-pulmonary Factor VII concentrate. Her renal function declined and it was felt that this was partly due to worsening lupus nephritis. She was started on cyclophosphamide. The patient recovered and has been stable on follow up visits for the past several months.

Discussion: DAH associated with Rivaroxaban is a rare complication, one other case is reported in the literature of a man with a history of granulomatosis with polyangiitis being treated for deep venous thrombosis on Rivaroxaban. Pertinent to this case, APS is also associated with diffuse alveolar hemorrhage, either as a primary etiology or in combination with other connective tissue disorders such as systemic lupus nephritis. The incidence of DAH associated with APS is unknown, but at least 50 cases were presented in one series.

There are only scarce case reports of diffuse alveolar hemorrhage associated with nearly all anticoagulants, including warfarin, dabigatran, apixapan, and low molecular weight heparin. We believe that the novel anticoagulants should be used cautiously in the subset of patients with lung disease and conditions that may predispose to the potentially fatal complication of DAH, including APS. In addition, at this time arterial thrombosis is not an FDA approved indication for the use of Rivaroxaban. The reporting of these complications and post marketing data collection and reporting of complications is essential to patient safety with new pharmaceuticals.

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PANCREATIC CANCER: WHAT ELSE OTHER THAN ADENOCARCINOMA?

First Author: Leila Khaddour, MD

Introduction: Adenocarcinoma represents more than 95% of pancreatic cancer. Neuroendocrine neoplasms comprise no more than 5%. Very rarely, primary connective tissue cancers of the pancreas can occur and may require specific diagnostic approach.

Case presentation: A 52-year-old relatively healthy male presented with one-month of progressive fatigue, and pruritus. Physical exam was unremarkable except for jaundice. Initial Labs showed elevated liver enzymes with cholestatic pattern. CBC showed significant thrombocytopenia with normal white cell count and hemoglobin. Abdominal and pelvic CT scan with contrast demonstrated diffuse intrahepatic and extra-hepatic biliary ductal dilatation, in addition to a 5 cm mass of the pancreatic head. Further laboratory work up with CA19-9 noted to be normal. He underwent endoscopic retrograde cholangiopancreatography for stent placement of the common bile duct. Brushing biopsies showed no evidence of malignancy. Two FNA biopsies failed to prove the histology nature of this neoplasm. The case was discussed by multidisciplinary tumor board who agreed that the tumor was not resectable, favoring neoadjuvant chemotherapy. Finally, CT guided core biopsy along with pathology and immunohistochemistry studies confirmed the diagnosis of large B-cell non-Hodgkin lymphoma. Further lymphoma work up including PET scan was consistent with stage II-E primary pancreatic lymphoma. Bone marrow studies showed increased mature megakaryocytes with no infiltration. He successfully underwent complete remission after 6 cycles of chemotherapy. Moreover, thrombocytopenia completely resolved after 2 cycles of chemotherapy, supporting the diagnosis autoimmune thrombocytopenia secondary to lymphoma.

Discussion: Primary pancreatic lymphoma (PPL) is very rare, accounts for less than 0.5% of all pancreatic malignancies. Diagnostic criteria for PPL include lymphomatous spread limited to the pancreas and peripancreatic lymph nodes and lack of splenic, hepatic, and superficial or mediastinal lymph nodes involvement. Histologic confirmation is required to establish the diagnosis of pancreatic cancer. Considering that 95% of pancreatic cancers are adenocarcinomas, patients who have potentially resectable pancreatic tumor do not necessarily undergo a preoperative biopsy before proceeding directly to surgery. However there has been increased recognition of benign and malignant disease entities presenting as pancreatic mass, diagnosed postoperatively would not warrant surgical resection in the first place. Hence extensive preoperative workup should be considered by rebiopsy if initial studies were inconclusive, especially in patients presenting with large pancreatic mass, B symptoms, and low CA 19-9 level. Furthermore, immune thrombocytopenic purpura ITP secondary to non-Hodgkin lymphoma (NHL) has been reported. The key feature of ITP is increased number of megakaryocytes in the bone marrow in response to the peripheral destruction. ITP secondary to NHL usually shows no response to steroids. In contrary complete resolution has been observed in cases when lymphoma was successfully treated with chemotherapy.

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ACUTE PANCREATITIS? THINK TWICE!

First Author: Leila Khaddour, MD

Introduction: Acute abdominal pain is one of the most common presentations. Detailed history and physical exam are crucial to augment studies, henceforth reach accurate diagnosis.

Case presentation: 41 year-old male with past medical history of type 2 diabetes who presented to our hospital with three days of severe epigastric pain, nausea and vomiting. Three weeks prior to his admission he complained of sore throat, fever, and myalgia, associated significant unintentional weight loss. He acknowledged chronic marijuana use, remote history of heroin use, and high-risk sexual behaviors. Physical exam was remarkable for mild epigastric tenderness and cervical lymphadenopathy. Labs showed mild thrombocytopenia and elevation of serum lipase at 120 U/L with normal liver and kidney functions as well normal electrolytes and lipid panel. Patient was admitted for possible acute pancreatitis. However abdominal imaging revealed normal pancreas. Considering his social history, HIV screening algorithm was obtained and was consistent with early HIV infection. Elevated lipase was transient and related to acute HIV syndrome.

Discussion: Acute pancreatitis is defined by: characteristic abdominal pain combined with elevation in serum lipase or amylase to at least 3 times of normal and/or indicative abdominal imaging findings. Nonspecific elevation of serum lipase has been reported with numerous pathologies such as renal failure as well several other conditions other than pancreatitis. Elevated serum lipase is found in 12% of patients admitted to the hospital with non-pancreatic abdominal pain. More interestingly, it has been reported that about 60% of asymptomatic HIV patient had an abnormal amylase or lipase measurement on at least one occasion.

Moreover, acute HIV syndrome mimics other viral infections. Diagnosis of acute HIV is important, since prompt initiation of antiretroviral therapy reduces the likelihood of HIV transmission to others and can reduce the size of the latent HIV reservoir, potentially making patients eligible for future HIV eradication strategies. In early HIV infection, the viral RNA level is typically very high and the CD4 cell count can drop transiently.

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HIGH GRADE SEROUS CARCINOMA OF OVARIES DIAGNOSED IN AN UNUSUAL WAY, FOLLOWING AN ABNORMAL PAP SMEAR

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**Introduction:** Pap smear is an effective screening tool for early detection of cervical cancer. Numerous studies are reported in literature where an abnormal Pap smear was associated with diagnosis of cancers other than cervical cancer like primary uterine, ovarian, mesenteric sarcoma, breast and gastric cancers. Some studies also examined the possibility of using Pap smear as a screening tool for ovarian and endometrial cancers although further research on this area is still needed. We present an interesting case where an abnormal Pap smear on routine screening led to the diagnosis of extra-uterine cancer. To the best of our knowledge this is the first case to be reported in this particular combination of clinical presentation, test results and final diagnosis.

**Case:** A 59 year old postmenopausal woman presented to our outpatient clinic following an abnormal Pap smear, interpreted as adenocarcinoma (favoring endometrial origin), on routine screening. She was largely asymptomatic and no adnexal masses were found on physical exam or radiologic imaging. Endocervical and endometrial biopsies also indicated a primary uterine neoplasm and patient underwent a hysterectomy with bilateral salpingo-oophorectomy. The final diagnosis was stage T3cN1M0 high grade serous carcinoma (HGSC) of bilateral ovaries with normal fallopian tube and endometrial histology. This case is unique in the way it was diagnosed and the combination of test results prior to surgery that were misleading towards a primary uterine neoplasm.

**Discussion:** HGSC of ovary is the most common type of ovarian cancer accounting for 70-80% of all malignant ovarian neoplasms. A retrospective study reported that ovary, gastrointestinal tract and breast were the three most frequent primary sites of cancers diagnosed with abnormal Pap smears, accounting for 28 of the 33 cases (85%). A case series of 8 patients reported the deceptive nature of some tubal/ovarian HGSC where initial cervical and uterine samples suggested a primary endometrial cancer and post-surgical diagnosis revealed HGSC of tubal or ovarian origin and a transtubal spread was considered the likely mechanism. There are multiple case reports of extra-uterine malignancies presenting as an abnormal Pap smear on routine screening but the clinical scenario with combination of following three features makes our case unique.

1. Asymptomatic patient presenting with an abnormal finding of adenocarcinoma on screening Pap smear
2. No sign of adnexal mass on physical exam or radiologic imaging.
3. HGSC carcinoma on post-surgical histology

This case illustrates the importance of abnormal Pap smears in diagnosis of extra uterine cancers.

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NEUTROPENIC FEVER, AN UNCOMMON PRESENTATION OF A COMMON DEFICIENCY

First Author: Sadichhya Lohani, MD Pradeep Khanal, MD Kristle Haberichter, DO Dilip Khanal, MD

Vitamin B12 deficiency is a very common medical problem. Megaloblastic anemia and neurologic manifestations are the usual presenting symptoms. We present a case of a young lady who presented with pancytopenia, neutropenic fever and severe sepsis secondary to vitamin B12 deficiency.

A 27-year-old previously healthy female presented with worsening fatigue, exertional shortness of breath, fever and chills for one week. On presentation, her blood pressure was 86/49 mm Hg, heart rate 124 per minute and temperature was 103.1 °F. Physical examination was normal except conjunctival pallor and loss of vibration sensation in lower extremities. White blood count (WBC) was 0.6 bil/L, neutrophil 0.1 bil/L, hemoglobin 5.3 g/dl, mean corpuscular volume 94 fL, and platelet 17 bil/L. Liver enzymes were mildly elevated with indirect hyperbilirubinemia, low haptoglobin, and elevated lactate dehydrogenase. Reticulocyte count was normal. Coomb's test, paroxysmal nocturnal hemoglobinuria (PNH) screen and autoimmune workup, were negative. Serology for Parvo virus and HIV were negative. She was resuscitated with intravenous (IV) fluids and started on IV cefepime after blood and urine cultures were obtained. Subsequent work up revealed vitamin B12 level of 97 pg/ml with normal folate level. Homocysteine and methylmalonic acid level were both elevated. Bone marrow evaluation showed hypercellularity with left shift and megaloblastic changes without evidence of hematologic malignancy. Intrinsic factor antibodies were positive. With the diagnosis of pernicious anemia, daily vitamin B12 injection was started. She had some improvement in fatigue and exertional shortness of breath. She became afebrile in three days. Infectious workup was negative and no focus of infection was identified. In one month follow up in clinic, her symptoms had resolved, hemoglobin was 11.8 g/dl and WBC count improved to 6.9 bil/L.

Neutropenic fever is a commonly encountered clinical problem with malignancy, chemotherapy and radiotherapy. Vitamin B12 is an essential cofactor in maturation of erythrocytic, granulocytic and megakaryocytic lineages and its deficiency can lead to pancytopenia. However, severe neutropenic fever is very uncommon as the initial presentation of vitamin B12 deficiency. Another interesting point in this case was that our patient had normocytic anemia despite having very low vitamin B12 levels. Hemolysis seen in this case was likely due to ineffective erythropoiesis. Through this case, we want to emphasize that vitamin B12 deficiency should be considered in the differential diagnosis in cases of severe neutropenia and fever. Absence of macrocytosis and presence of hemolysis does not rule out vitamin B12 deficiency. Early diagnosis and treatment of this easily reversible disorder is very important in preventing adverse patient outcome.

## A CHALLENGING OVERDOSE

First Author: Issa Ali Makki, MD Second Author: Fatima Ali-Ahmed, MD; Kyle J Feldmann, MD, Aaron D. Berman, MD, FACC

Calcium channel blockers (CCB) toxicity can be devastating. CCB are the leading cause of cardiovascular drug overdose with a mortality rate as high as 38%. Due to the profound refractory bradycardia and hypotension, treating patients with a CCB overdose is often very difficult and challenging.

A 65-year-old female with history of hypertension and atrial fibrillation presented with altered mental status. On presentation, the patient was hypotensive and bradycardic at 34 bpm. She stated, "I took a few extra pills of my beta blocker." She was drowsy, but alert and oriented and requested to be DNR/DNI. The patient quickly deteriorated, developing profound refractory bradycardia and hypotension, despite aggressive IV fluids, atropine, naloxone, glucagon pushes, and maximum support with dobutamine. She was lethargic and unresponsive, with no family members to be found, she was deemed a suicide attempt and was intubated. A trans venous pacemaker (TVP) was placed but would not capture. Given the patients lack of response to therapy, her medication bottles were reviewed again, revealing empty bottles of Amlodipine 10 mg and diltiazem XL 240 mg. The patient was quickly bolused with calcium gluconate followed by a continuous infusion and switched to norepinephrine. Afterwards, the TVP started capturing; however she continued to display refractory hypotension requiring maximum support of three pressors. She was placed on a high dose insulin drip of 1 unit/kg with glucose and weaned off pressors. The insulin drip was titrated upwards until the hypotension resolved. After a few days of high dose insulin, the patient made a full recovery and is currently receiving counseling.

CCB are divided into two categories, dihydropyridines (amlodipine), which mainly block the L-type calcium channels in the vasculature and nondihydropyridines (diltiazem), which selectively block L-type calcium channels in the myocardium. In this patient, the combination of these two agents led to depression of the conduction system and severe vasodilatation leading to severe bradycardia, inability to pace, and refractory hypotension. Initial treatment for CCB overdose includes airway maintenance, fluids and atropine. However, patients often require further intervention, such as IV calcium, high dose insulin/glucose and TVP. CCB are highly protein bound, therefore dialysis provides no benefit. Glucagon is of questionable usefulness in CCBs overdose. The proposed mechanism of insulin, involves improved smooth muscle contractility by increased efficiency of carbohydrate uptake, which counteracts CCBs blockade. This optimizes the glucose-dependent energy formation required to overcome CCBs overdose. As demonstrated in our case, CCB overdose can be devastating, unpredictable and complicated due to the profound refractory bradycardia and hypotension. It is difficult to recommend one specific strategy; however urgent administration of fluids, calcium, atropine, vasopressors, and insulin therapy appear to be the mainstay approach.

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INVASIVE PNEUMOCOCCAL DISEASE: BEYOND AUSTRIAN TRIAD

First Author: Himabala Medasani, MBBS Alex C Essenmacher, MD Narendra N khanchandani, MD

Austrian triad describes the association of pneumococcal meningitis, pneumonia, and endocarditis. We describe a rare scenario where apparently healthy gentlemen developed invasive disease with diskitis and arteritis in addition to pneumonia and meningitis. The infection progressed rapidly to involve the right common iliac artery aneurysm adjacent to the disk leading to rupture. We postulate that pneumococcal arteritis accelerated the aneurysm expansion leading to rupture, emphasizing the need for vigilance and timely intervention to prevent mortality.

A 58-year-old male who presented with altered mental status grew *Streptococcus pneumoniae* in his blood cultures, sputum, and CSF resistant to penicillin but sensitive to ceftriaxone. Antibiotics were de-escalated to IV ceftriaxone. Trans-esophageal echo-cardiogram was negative for endocarditis. Computed tomography revealed infra-renal abdominal aorta dilatation of 3.2cm, right common iliac artery aneurysm of 3.3cm, and left common iliac artery aneurysm of 2 cm. The patient had persistent back pain, and MRI of the lumbar spine showed discitis at L5-S1. The pathology result was positive for acute discitis with inflammatory exudate but with negative cultures. Repeat CT revealed progressive dilation of the right CIAA to 5.8cm.. A 12-week course of IV ceftriaxone therapy was begun. After discharge, the patient was found unresponsive in acute care facility; CT workup revealed further dilation of the right CIAA to 8.3cm along with hemoperitoneum suggesting a rupture of right CIAA aneurysm. There was no change in other two aneurysms. Endovascular repair of all three aneurysms was undertaken with stable postoperative course.

The incidence of pneumococcal pneumonia in association with meningitis and endocarditis, the Austrian triad, has greatly fallen with modern antibiotics. Review of the literature suggests that iliac artery aneurysms <3cm in size expand at an average rate of 1.1mm/y whereas those >3cm expand by 2.6mm/y. Our patient's aneurysm expanded at a rate much higher than the above, potentiating our theory of contiguous involvement leading to rupture despite appropriate antibiotic therapy. Cultures were negative due to concurrent administration of antibiotic therapy.

Endovascular infection of an existing aneurysm is a serious but rare entity. Scant case reports describe discitis possibly extending to contiguous arteritis. This case substantially adds to the evidence of contiguous involvement, as we radiographically demonstrate rapid expansion of the affected artery aneurysm. Considering this, it is important to investigate beyond Austrian Triad for timely intervention to prevent mortality.

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IT'S NOT ALL IN THE NUMBERS: AN UNUSUAL PRESENTATION OF PULMONARY LEUKOSTASIS IN ACUTE MYELOID LEUKEMIA

First Author: Amy N Mertens, DO Daniel Isaac, DO, MS Paul Bozyk, MD

Patients with newly diagnosed acute myeloid leukemia (AML) have a variety of clinical presentations and complications, the most emergent being hyperleukocytosis leading to decreased tissue perfusion through vascular leukostasis. Hyperleukocytosis is defined as a total white blood cell count greater than 50,000/microL to 100,000/microL. The increased leukemic white blood cell aggregates form intravascular thrombi, leading to occlusion of the pulmonary and neurologic microvasculature. Vascular leukostasis has been documented as the single worst prognostic factor in AML patients presenting with hyperleukocytosis.

A 24-year-old previously healthy female presented with a three day history of progressive cough, dyspnea and diarrhea. She denied any sick contacts or recent travel. Her medical history was significant for tobacco abuse. She worked as a children's caregiver at a zoo and did not have any contact with animals. On presentation, she was in acute hypoxic respiratory failure. Physical exam was notable for scleral icterus. Initial laboratory studies revealed a leukocytosis of 16,200/microL with a neutrophilic predominance and slight monocytosis. CT of the chest showed diffuse bilateral patchy opacities and interstitial thickening with bilateral hilar and mediastinal lymphadenopathy. She was initially treated for community acquired pneumonia but quickly worsened with persistent high grade fevers and increased oxygen demands, requiring mechanical ventilation. She subsequently underwent bronchoscopy, which revealed only mild inflammation. All cultures and infectious serologies, including potential zoonosis and endemic mycoses, were negative. Despite broadened antimicrobial and antifungal coverage, she exhibited continued hypoxia with maximum lung protective ventilatory settings and eventually required extra-corporeal membrane oxygenation. Laboratory studies exhibited persistent neutrophilic leukocytosis peaking at 25,500/microL with a monocytosis reaching 47.4 percent. A review of her peripheral blood revealed leukocytosis, absolute neutrophilia, and large atypical monocytic cells near the periphery of the slide. The patient subsequently underwent bone marrow biopsy but expired shortly thereafter. Post-mortem cytogenetics were consistent with acute myelomonocytic leukemia.

Classically, patients with AML present with respiratory and neurological symptoms secondary to vascular stasis from hyperleukocytosis. Early recognition is essential since mortality is as high as 20-40% in the first week if left untreated. Interestingly, in this case study we present a patient with pulmonary vascular leukostasis whose lack of overt hyperleukocytosis led to a delay in diagnosis. Given the failure to improve despite broad spectrum antibiotics, an expanded differential, including AML, must be pursued. This case illustrates the importance of recognition and prompt treatment for leukostasis, which may be present even in the absence of overt hyperleukocytosis.

## A (VF) STORM IS COMING: DIFFERENTIATING BETWEEN BRUGADA SYNDROME AND BRUGADA PATTERN

First Author: Amy Mertens, DO Second Author: Thomas Verrill, MD Third Author: Michael Gallagher, MD

Brugada syndrome is an inherited autosomal dominant disorder of the myocardial sodium channel. It is associated with characteristic changes on surface electrocardiogram (ECG) and increased risk of sudden cardiac death and sustained ventricular tachyarrhythmias. Brugada pattern is defined as having typical ECG features of Brugada syndrome without associated symptoms.

A 57 year-old-female was brought to the hospital by emergency medical personnel due to cardiac arrest from ventricular fibrillation. Her only known medical conditions included hypertension and hypothyroidism. Return of spontaneous circulation was obtained after 32 minutes of advanced cardiac life support (ACLS). She was noted to have mild hypokalemia and subsequent ECG displayed normal sinus rhythm, nonspecific intra-ventricular conduction delay, and no evidence of acute or chronic ischemia. Hypothermia was induced and she was admitted to the cardiac care unit. Her electrolytes were corrected and therapy with beta blockade, aspirin, and intravenous heparin was initiated. Despite treatment with multiple antiarrhythmics, she continued to experience recurrent sustained ventricular arrhythmias. 2D Echocardiography revealed mildly reduced left ventricular ejection fraction at 45%. There were no significant regional wall motion abnormalities. Serial ECGs revealed findings suspicious for type 1 Brugada syndrome. Cardiac catheterization was negative for obstructive coronary disease. The patient was subsequently diagnosed with Brugada syndrome. Therapy with intravenous isoproterenol and quinidine was initiated and an ICD was implanted without further episodes of ventricular arrhythmia storm. The patient eventually underwent genetic testing revealing SCN5A congenital long QT syndrome type 3 (LQT3) mutation.

Brugada syndrome is responsible for 4-12% of all sudden cardiac death. The prevalence of Brugada syndrome among patients with Brugada pattern on ECG has not been well studied; a meta-analysis of 30 published reports of patients with Brugada pattern on ECG demonstrated a 10 percent event rate at 2.5 years. Though the Brugada pattern is occasionally observed on surface ECG, the criteria for Brugada syndrome are rarely met. Brugada syndrome must be differentiated from conditions that lead to Brugada-like pattern including right bundle-branch block, early repolarization, and arrhythmogenic right ventricular cardiomyopathy. Risk stratification is based on specific symptoms and spontaneous changes on surface ECG revealing Type I Brugada pattern. However, risk evaluation in asymptomatic individuals is controversial and needs to be further studied.

While both Brugada pattern and syndrome have identical findings on surface ECG, only the Brugada syndrome is associated with symptoms including sudden cardiac death and ventricular tachyarrhythmias. Though sometimes challenging, the distinction is critical in order to identify patients at risk of sudden cardiac death. In patients who are identified with Brugada syndrome, genetic testing may allow for family screening and risk stratification.

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CAN LIGHTNING STRIKE TWICE? INTRAUTERINE LIGHTNING STRIKE AS A POTENTIAL SOURCE OF WOLFF-PARKINSON-WHITE SYNDROME AND MULTIFOCAL ATRIAL TACHYCARDIA

First Author: Leo E Reap III, DO

Wolff-Parkinson-White syndrome (WPW) and multifocal atrial tachycardia (MAT) are separate electrophysiological conduction disorders that predispose to symptomatic tachyarrhythmia. While they are relatively common disorders encountered in cardiology, they are rarely seen together as the etiology of supraventricular tachycardia.

A 28-year-old woman underwent atrial tachycardia ablation for lifelong episodes of persistent exertional tachycardia. She had been born via emergency Cesarean section due to severe fetal tachycardia at 240 bpm. Throughout her life she experienced recurrent episodes of shortness of breath and palpitations precipitated by exertion and stress. Though unifocal atrial tachycardia was presumed to be the source of her symptoms, ablation of the atrial foci unmasked a concomitant underlying Wolff-Parkinson-White syndrome as well as an additional underlying atrial foci. She underwent subsequent ablation of both the accessory pathway and ectopic foci with improvement in her symptomatology. Given the highly unusual nature of concomitant multifocal atrial tachycardia and Wolff-Parkinson-White syndrome, a thorough history of all risk factors was taken. Her family history was only positive for atrial fibrillation in her grandmother at 70 years of age. No other family members had a history of WPW or symptomatic tachyarrhythmia. Her only potential teratogen was a lightning strike her mother suffered in the shower when she was approximately 4 weeks gestation. Her mother stated that a lightning bolt had struck a nearby water main and the current passed through the shower head, the water flowing directly upon her abdomen. She was taken to the hospital but had an uneventful recovery and delivered at 40 weeks gestation. Given that this exposure was during fetal cardiogenesis and that the current passed directly through her mother's abdomen, it is highly possible that the electrical exposure may have led to impaired cardiac development and a predisposition to subsequent tachyarrhythmia. Also, as WPW and MAT are very rarely seen concomitantly, the lifelong nature of the patient's symptomatology in addition to fetal tachycardia requiring Cesarean section suggests an intrauterine exposure as the etiology of both disorders.

This case illustrates the importance of electricity as a potential teratogenic source of congenital tachyarrhythmia and the critical importance of a thorough patient history. While electrical injury in the pregnant patient is rare, lightning strike as a source of congenital WPW and MAT has never been reported.

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SPORTS FANS BEWARE: CARDIAC ARREST WHILE WATCHING FOOTBALL

Travis Tagami Adrian Mercado Lohit Garg Ismail Hader

Long QT syndrome (LQTS) is a manifestation of a group of disorders that cause a long QT interval and cardiac arrhythmias. There are currently thirteen types of congenital LQTS that have been identified. LQT1-3 make up close to 75% of all cases. We present a case of probable LQT2 diagnosed after cardiac arrest.

A 22 year old previously healthy female was watching an annual rivalry football game at home when an unexpected play happened. She remembered being extremely excited prior to losing consciousness. Her boyfriend performed CPR prior to EMS arrival. Her initial rhythm was torsades de pointe and she was delivered one shock. She converted to sinus tachycardia, however was minimally responsive. She underwent hypothermia protocol. Her medical history was significant for occasional feelings of shortness of breath and palpitations over the previous year. She was not taking any medications, she had a younger brother with a bicuspid aortic valve and supra-ventricular tachycardia. She did not smoke, or use illicit drugs. Her vital signs were within normal limits. On exam her heart rate was tachycardic and regular without any audible murmurs or gallops. Her EKG demonstrated sinus tachycardia. Continuous EEG monitoring showed a diffuse anoxic brain injury pattern. After 24 hours of cooling protocol she was rewarmed and exhibited signs of full neurologic recovery. She was extubated the next day. A cardiac MRI showed no evidence of cardiomyopathy or congenital disease. Reviewing an EKG from one year earlier demonstrated a prolonged QT interval. She was placed on a non-selective beta blocker and underwent placement of a subcutaneous implantable cardiac defibrillator and was eventually discharged home.

LQT2 is the most likely diagnosis in this case due to the nature of her presentation. LQT1 usually causes arrest during exercise (62%), while intense emotion (43%) or auditory stimuli (26%) are the most common causes in LQT2. LQT3 is described as causing cardiac arrest most commonly while sleeping. She remembered feeling intense emotion prior to losing consciousness which fits with LQT2. Congenital cardiac arrhythmias should always be in the differential of unexplained syncope or cardiac arrest.

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SEVERE ADENOVIRUS PNEUMONIA IN AN IMMUNOCOMPETENT ADULT

Travis Tagami Adam Skrzynski Paul Johnson Carl Lauter Bishr Al-Ujayli Matthew Sims

**Introduction:** Adenovirus is known to cause respiratory and gastrointestinal illness in children and transplant patients, however severe disease is rare among healthy adults. We present a case of disseminated adenovirus causing severe acute respiratory distress syndrome in an immunocompetent adult.

**Case:** A 45-year-old male presented with a three-day history of non-productive cough, fevers, nausea, and diarrhea. His seven-year-old son had a similar illness in the week prior. He had no recent travel or change in diet and was working up until the day of presentation. He had a history of type I diabetes mellitus and hypertension. He was a non-smoker and worked in construction. He was tachycardic and febrile to 38.5 degrees Celsius with normal oxygen saturation. He had mild splenomegaly and left upper quadrant tenderness. A complete blood count was remarkable for thrombocytopenia. Initial chest x-ray demonstrated a right-sided infiltrate. Rapid flu, sputum culture and gram stain were negative. His respiratory status worsened over the next 24 hours despite broad-spectrum antibiotic therapy. A chest CT was performed and showed worsening right upper and lower lobe consolidation. He had a bronchoscopy which showed diffuse inflammation. Bronchial lavage cultures were negative, however a viral panel was positive for adenovirus by PCR amplification. Serum quantitative PCR was positive at >2 millions copies. Renal function declined and the patient was placed on dialysis. His clinical status continued to worsen and he was placed on extracorporeal membrane oxygenation (ECMO). IV cidofovir and PO probenecid were started for treatment of severe disseminated adenovirus. He initially improved and was able to wean off vasopressors, however four days after the initiation of ECMO therapy he was found to have large intraparenchymal hemorrhage in the left parietal-occipital lobe. He was deemed a poor surgical candidate and care was eventually withdrawn.

**Discussion:** Adenovirus is a ubiquitous pathogen that normally causes mild and self-limited symptoms in immunocompetent adults. As is illustrated in this patient, it is important to think of this virus as an occasionally aggressive pathogen that can cause severe and disseminated disease. This case stresses the urgency of recognizing the presence of common diseases in patients presenting in uncommon ways.

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## ATRIAL ABLATION AND SIADH

First Author: Brian Tyson, MD Second Author: Farhan Qureshi

SIADH is a common condition within hospitalized patients, with a very broad differential. A variety of medications, organ pathologies, and paraneoplastic syndromes are commonly investigated, however it is rarely associated with a cardiac etiology.

A 71 y/o male presented to the Cardiovascular ICU after undergoing an atrial ablation with isolation of the pulmonic veins for atrial fibrillation. The procedure itself was complicated by the development of a moderately sized pericardial effusion which required placement of a pericardial drain catheter. Roughly 650cc of bloody fluid was drained in the cardiac lab. The catheter drained an additional 400cc of fluid over the next 12 hours. Serial hemoglobin and basic metabolic panel (BMP) were ordered to monitor the patient, and it was noted that the patient's sodium decreased by 10 units over that same period of time, from 141 mEq/L to 131 mEq/L. The patient began complaining of some mild numbness and tingling in his lower extremities. The results of the BMP, demonstrated that the patients sodium continued to decrease, down to 129 mEq/L. We placed the patient on fluid restrictions except for small sips of water, believing the hyponatremia was related to the amount of fluids he received in his procedures the previous day. Despite above measures the patient sodium decreased another 6 mEq/L, from 129 to 123, and the patient complained again of just mild tingling. At this point we gave the patient a 500cc challenge of 0.9% normal saline, and ordered a BMP a few hours later, which showed his sodium continued to decrease down to 119. Additional serum and urine studies were performed which demonstrated a serum osmolarity of 250mOsm/kg and a urine osmolarity of 899mOsm/kg. This was consistent with SIADH, with worsening hyponatremia with intravenous normal saline. The patient was placed on fluid restrictions again, along with consulting the nephrology team. Soon after, the pericardial drain catheter was removed as the patients output from the pericardial effusion had diminished. Subsequent lab tests after the removal of the catheter demonstrated a marked improvement in the patient's sodium level (128 mEq/L) and the patients urine osmolality decreased to 112mOsm/kg, with resolution of the patient's numbness and tingling.

This case represents marked hyponatremia following an atrial fibrillation ablation complicated by a pericardial effusion. It is interesting to note that once the catheter was removed the patients hyponatremia resolved. Of note, the patient was on flecainide prior to and after the ablation procedure, therefore it is unlikely to have caused this patients hyponatremia (cases have described flecainide induced hyponatremia).

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SUCCESSFUL THERAPY WITH LEDIPASVIR/SOFOSBUVIR FOR HEPATITIS C REACTIVATION IN A HEMATOPOIETIC STEM CELL TRANSPLANT RECIPIENT.

Marian Wahba, Malini Surapaneni, Pranatharthi H. Chandrasekar

**Background:** Hepatitis C virus (HCV) infection is a unique challenge in stem cell transplant (SCT) recipients. Traditionally, chronic hepatitis C was not treated in patients undergoing SCT as immediate post-transplant complications are uncommon. Treatment with interferon-based regimens is contraindicated with immunosuppression and have unfavorable side effect profile. With the recent era of interferon-free regimens, the approach to HCV infection management in SCT recipients needs to be addressed.

**Case description:** A 53-year-old African-American woman with chronic hepatitis C genotype 1a presented four months post allogeneic SCT with fatigue, new onset ascites, and pedal edema. The patient had mildly elevated serum transaminases and decreased albumin with normal bilirubin. She was pancytopenic and transfusion dependent. Serum HCV RNA PCR increased from 198,642 copies/ml pre-transplant to 100 million copies/ml post-transplant. Hepatic Doppler ultrasound showed no evidence of sinusoidal venous occlusion. FIBROSpect II revealed F2-F4 indicating significant fibrosis and cirrhosis. Given this clinical presentation, therapy with Ledipasvir/Sofosbuvir was started to treat the HCV infection. The patient tolerated treatment with no side effects; she reported symptomatic improvement of her fatigue, and ascites resolved gradually after starting the treatment. Red blood cell and platelet transfusion requirements decreased over 30 days, and the liver function tests normalized. Serum HCV RNA became undetectable in one month. The patient completed her 12 weeks treatment course successfully.

**Conclusions:** From our review, this is the first documented case of successful use of Ledipasvir/Sofosbuvir in a SCT recipient. Management of hepatitis C in SCT recipients is changing with the recent availability of well tolerated, highly effective therapy of HCV.

## A PECULIAR CAUSE OF STROKE IN A YOUNG WOMAN: BILATERAL INTERNAL CAROTID ARTERY OCCLUSION

First Author: Linda Wang, DO, Sadichhya Lohani, MD, Noora Kazanji, DO, Rebecca Grysiewicz, DO, Pradeep Kaminoulu, MD

Unilateral internal carotid artery occlusion is commonly found in the elderly. Complete occlusion of bilateral internal carotid arteries is not only rare among the elderly, but nearly unheard-of in young adults.

A 30-year-old African-American female presented with a severe right-sided frontal headache of sudden onset. Her initial neurological examination was normal. Her initial blood pressure was 156/122 mmHg. Computed tomography without contrast of her head did not reveal hemorrhage or infarction. One hour later, she developed slurred speech, left facial droop, left-sided weakness and decreased sensation, most notable in the upper extremity. Magnetic resonance imaging (MRI) with and without gadolinium and magnetic resonance angiogram of her brain without gadolinium revealed acute ischemia involving the right insula, corona radiata and cortical frontal regions. Interestingly, there was also apparent occlusion of the bilateral internal carotid arteries and filling of the anterior circulation mainly by the posterior communicating arteries. Carotid ultrasound demonstrated bilateral distal internal carotid occlusion with no evidence of atherosclerotic disease and a thrombus visible in the middle left internal carotid artery. Carotid arteriogram confirmed bilateral cervical internal carotid occlusions. Initially, permissive hypertension was allowed followed by gradual reduction in blood pressure. After comprehensive workup, she was diagnosed with fibromuscular dysplasia. She underwent inpatient physical therapy and subsequently discharged to continue maintenance therapy for lingering deficits.

Acute cerebrovascular accidents in young patients should prompt clinicians to pursue further and more thorough evaluation than the classic evaluation for stroke. Bilateral carotid artery occlusion is a rare phenomenon that typically affects patients over the age 60 with a history of vascular disease. It is extremely uncommon in younger patients without significant risk factors (auto-immune disease, vasculitis or hypercoagulability). Despite the initial unclear presentation in our patient, an early MRI was considered within 6 hours of presentation which ameliorated confirmation of ischemic stroke. However, the perplexing question remained as to why a young woman would develop extensive bilateral carotid artery stenosis with an acute thrombus. In patients with no other significant risk factors, fibromuscular dysplasia is the predominant etiology for bilateral carotid artery occlusion. Fibromuscular dysplasia is a diagnosis of exclusion and known signs and symptoms are hypertension and migraines, as were present in our patient. Hence, our case illustrates the importance of completing a comprehensive workup in identifying the etiology of bilateral internal carotid artery occlusion in order to prevent future recurrence of cerebrovascular events. Lastly, along with pursuit for unusual causes, our case reinforces the importance of identifying the common risk factors such as hypertension, diabetes, smoking, and migraine in a young adult with stroke.

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DANGERS OF USING ONLINE SUPPLEMENTS: THE HAZARDS OF WITHDRAWING FROM PSYCHOTROPIC DRUGS

First Author: Linda Wang, DO Bianca Brunelli, DO Kerry Spero, DO Vaibhav Sahni, MD

The use of over-the-counter and online supplements is very popular among the general population. Many of these supplements are not regulated by the FDA and thus the specific ingredients and dosages of the supplements are often unknown. These supplements are usually not benign agents and frequently can have dangerous side effects or interactions with other medications.

27-year-old Caucasian male presented to the emergency room with the intention of ending his physical dependence of phenibut. He had been using phenibut for six months to self-medicate for anxiety. After four months of use, he began experiencing physical tolerance to the drug and insomnia. He tried to wean himself off of phenibut but reported symptoms of tachycardia, diaphoresis, visual hallucinations and seizures. Patient also reported urinary retention, nausea, decreased appetite, and blood in the stool. Physical exam was remarkable for tachycardia, hypertension, and tremors. Initial laboratory findings were within normal limits except for signs of dehydration and 1+ protein found in urinalysis. Poison Control was contacted and recommended IV fluids and oral lorazepam. The patient was placed under Alcohol Withdrawal Assessment Protocol and transferred to the intensive care unit. Medical management consisted of placing the patient back on his home medications and managing symptoms of withdrawal with lorazepam. The patient was started on olanzapine after psychiatric consult. Patient's withdrawal course was unremarkable except for one isolated episode of blurry vision that occurred on day 4 of his course. The patient spent 4 days in the ICU and a total of 7 days in the hospital. The patient was discharged with prescriptions of his hospital medications and follow-up with his primary care physician.

Phenibut (also known as fenibut or phenybut) is a neuropsychotropic drug which was discovered and implemented in Russia in the 1960s. It primarily acts on  $\gamma$ -aminobutyric acid (GABA) receptors and has pharmacological and physiological properties similar to Baclofen, a para-Cl-derivative of phenibut. Phenibut primarily has anxiolytic and tranquilizing effects, among other effects. Phenibut has not been approved by the FDA for use in the United States; it is available for purchase online as a medicinal supplement. This case is unique in that very few cases of phenibut withdrawal have been managed by hospitals, and of the few cases that have been reported, many are found outside of the United States. This case was managed differently than other case studies and it may give other physicians an idea of how to manage phenibut withdrawal. This case also highlights the need for physicians to collect thorough histories on their patients regarding medication use, especially the use of substances not prescribed from a physician including dietary supplements and even substances found in food such as energy drinks.

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WHEN THE BIG BAD WOLF BITES: LUPUS WITH HEMOPHAGOCYTOSIS

First Author: Daniel V Dudenkov, MD Author: Floranne Ernste, MD

Hemophagocytic syndrome (HPS), or hemophagocytic lymphohistiocytosis (HLH), is a rare systemic, inflammatory disorder of macrophage activation that leads to hemophagocytosis in the reticuloendothelial system. It is diagnosed when five of the following eight clinical features are met: fever; splenomegaly; cytopenia (at least 2 cell lines); hypertriglyceridemia or hypofibrinogenemia; hemophagocytosis in bone marrow, lymph node or spleen; low natural killer cells; hyperferritinemia; or elevated soluble CD25. Secondary (or reactive) HPS, which is the more common form of HPS in adults, is typically seen in association with infection, malignancy or autoimmune disease. Acute lupus hemophagocytic syndrome is a term defined by the presence of a systemic lupus erythematosus (SLE) flare in the setting of HPS with no evidence of infection or malignancy.

A 38-year-old Bhutanese refugee from Nepal, previously healthy, presented to an outside hospital with a 6-month history of sore throat, dysphagia, fevers, diffuse arthralgias and abdominal pain, and a 1-month history of diffuse skin lesions. She was transferred to the Mayo Clinic Saint Mary's Hospital with a diagnosis of HLH and limited records. On physical exam, she was somnolent with mild tachycardia but otherwise vitally stable. Skin exam revealed erythematous, nonblanching lesions on the ears, knees and toes. Joint exam revealed diffuse joint tenderness and Jaccoud's arthropathy of the hands. Labs revealed pancytopenia; acute kidney injury with proteinuria; elevated ESR, CRP, triglycerides, ferritin, coagulation tests and liver function tests; and low fibrinogen and natural killer cells. A thorough infectious work-up including viral and fungal serologies, blood cultures and urine cultures was negative. PET/CT showed diffuse lymphadenopathy and splenomegaly, with increased uptake in the axial and appendicular bone marrow. Autoimmune labs revealed significantly elevated antinuclear antibodies and antibodies to dsDNA, SSA/Ro, Smith, and RNP, as well as low complement levels. Biopsy of a skin lesion revealed histologic findings consistent with SLE. Axillary lymph node biopsy was negative for malignancy and demonstrated necrotizing histiocytic lymphadenitis, which is often associated with lupus. Bone marrow biopsy revealed hemophagocytic histiocytes. The patient was treated with pulsed high-dose IV methylprednisolone and one dose of IV cyclophosphamide, with clinical improvement. At discharge she was continued on a prolonged steroid taper, hydroxychloroquine and mycophenolate mofetil.

This case demonstrates a rare occurrence of HPS secondary to acute SLE, with clinical improvement after treatment with aggressive immunosuppression. HPS secondary to autoimmune disease has typically been associated with juvenile idiopathic arthritis or adult onset Still's disease. Early recognition of HPS is essential to making a prompt diagnosis and instituting therapy since HPS can rapidly progress to multi-organ failure and death. When a diagnosis of HPS is made, it is important to work-up infection, malignancy and autoimmune disease, because the treatment also involves management of the underlying cause.

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ST-ELEVATION FROM AN UPSIDE-DOWN STOMACH

First Author: Mithulan Jegapragasan, MD Yan Bi, M.D., Ph.D., Rajiv Gulati, M.D., Ph.D.

**INTRODUCTION:** ST elevation has a long differential of potential causes. One of the most life-threatening and time sensitive is myocardial ischemia. However, non-cardiac etiologies of ST elevation can require just as timely management.

**CASE PRESENTATION:** An 80-year-old female presented with acute severe substernal pain and nausea preceded by a history of chronic post-prandial retching. Her past medical history was significant for large paraesophageal hiatal hernia with failed laparoscopic hiatal herniorrhaphy in 2009, dyslipidemia, and hypertension. Electrocardiogram revealed ST-elevation in V1-3 and Troponin-T was elevated. Physical examination was unremarkable. In view of concerns for acute myocardial infarction, she underwent emergent coronary angiography, which revealed unobstructed coronary arteries. Intra-procedural fluoroscopy was however notable for a dynamic left-sided intrathoracic mass, with motion dyssynchronous with the cardiac cycle. Subsequent non-contrast computed tomography demonstrated this to be a large intrathoracic (“upside-down”) stomach, with acute outlet obstruction. Upper endoscopy revealed a twist in the stomach resulting in partial obstruction. Following therapeutic decompression with nasogastric tube placement, the patient underwent definitive open repair and Nissen fundoplication. Pre and postoperative chest X-rays illustrate successful reduction of the intra-thoracic stomach.

**DISCUSSION:** Paraesophageal hernias are uncommon, comprising only 5% of all hernias that occur through the esophageal hiatus. An intrathoracic stomach occurs when a large portion of the stomach herniates through the hiatus and can result in serious complications, including volvulus, gangrene and perforation. The most common clinical presentation is reflux and post-prandial chest pain. Diagnosis is normally made through fluid levels on a radiograph or with cross sectional imaging. Treatment is surgical repair if patients have symptoms. If obstruction is present, emergent open or laparoscopic reduction and repair are indicated.

ST elevation is seen in myocardial infarction but also occurs in the setting of many other conditions, including gastrointestinal pathology. This case serves to broaden our differential for ST elevations in patients with known hiatal hernias.

A QUESTION OF THROMBUS OR TUMOR: A CASE OF PULMONARY ARTERY INTIMAL SARCOMA MISDIAGNOSED AS MASSIVE PULMONARY EMBOLISM

First Author: Hannah C Nordhues, MD Second Author: Kelly Pennington, MD Mentor: Darlene Nelson, MD

**Introduction:** Pulmonary artery intimal sarcoma is a rare tumor with poor prognosis. It is often identified as filling defect on computed tomography angiography (CTA) leading to frequent misdiagnosis as acute or chronic pulmonary embolism. Here, we present a case of pulmonary artery sarcoma misidentified as a massive pulmonary embolism.

**Case Presentation:** A previously healthy 24 year-old mother presented to an outside emergency department with progressive shortness of breath and syncope in the setting of recently diagnosed pulmonary embolism. Three weeks prior to presentation, she was admitted for shortness of breath, pleuritic chest pain, and hemoptysis. She was diagnosed with unprovoked bilateral pulmonary emboli and started on a heparin bridge to warfarin. Following dismissal, she maintained a therapeutic INR; however, her symptoms progressed culminating in two syncopal episodes prompting re-presentation to her local emergency department. CTA of the chest demonstrated worsening bilateral pulmonary artery filling defects in the main pulmonary artery and left pulmonary artery with multiple distal defects in both lower lobes. Echocardiography revealed a severely dilated right ventricle with right ventricular systolic pressure of 108 mmHg. She was transferred to our intensive care unit for worsening “pulmonary emboli” and pulmonary hypertension while on therapeutic anticoagulation. Initial physical examination revealed a tachycardic (117 bpm), obese female breathing comfortably on 4L/min nasal cannula oxygen. No lower extremity swelling or erythema was appreciated. Lungs were clear to auscultation bilaterally. The remaining physical examination was normal. Laboratory studies were remarkable for leukocytosis, supra-therapeutic INR (4.8), elevated NT-Pro BNP (6105 pg/mL), and negative troponin. Special coagulation profile was notable for Factor V Leiden heterozygosity. Upper and lower extremity duplex ultrasound was negative for thrombosis. Similarly, inferior vena cava ultrasonography was negative for thrombus. Cardiac surgery, vascular medicine, and interventional radiology were consulted for possible thromboendarterectomy versus clot removal with AngioVac; however, given the chronicity, it was decided to pursue catheter directed thrombolysis. On hospital day three, she underwent catheter directed thrombolysis without any improvement in clot burden. Consequently the underlying malignancy was considered. Unfortunately she developed progressive respiratory failure and circulatory collapse requiring intubation. She had a PEA arrest and expired on hospital day 4. Postmortem evaluation revealed, pulmonary artery intimal sarcoma arising from the left side of the main pulmonary artery with multiple tumor emboli with associated pulmonary infarcts.

**Discussion:** Although pulmonary artery intimal sarcoma is a rare diagnosis, practitioners need to be aware of this possibility when a patient presents with presumed thromboembolism not responsive to anticoagulation or thrombolysis. Other clues to possible intimal sarcoma may include the following: few or no risk factors for thromboembolism, high sedimentation rate, and nodular parenchymal infiltrates on CT. Early recognition can lead to avoidance of unnecessary thrombolysis, appropriate surgery, chemoradiation, and prolonged survival.

UTTERLY OBVIOUS: A CASE OF LISTERIA MONOCYTOGENES ENDOGRAFT INFECTION PRESENTING AS FAILURE TO THRIVE

First Author: Kelly M Pennington, MD Melissa Myers, MD Abinash Virk, MD

Introduction: Listeriosis is serious foodborne illness caused by *Listeria monocytogenes*, most commonly affecting immunocompromised hosts, pregnant women, neonates, and the elderly. Manifestations generally include meningitis, rhombencephalitis, and bacteremia; however, focal infections (i.e. native valve endocarditis, prosthetic joint infections, and perianal abscess) have been reported. Prosthetic endograft infection alone is an extremely rare event with substantial morbidity and mortality. Causative organisms usually include *Staphylococcus aureus*, *Enterococcus*, *Streptococcus*, and *Escherichia coli*. Here, we present a case of aortic endograft infection secondary to *Listeria monocytogenes*.

Case Presentation: A 76 year-old dairy farmer with a past medical history significant for abdominal aortic aneurysm repair with Dacron graft in 2010, renal cell carcinoma with right nephrectomy, and mild mitral stenosis presented to the emergency department with a nine-month history of progressive weakness, functional decline, low back pain, and 40 pound weight loss. Physical examination showed an afebrile obese male with a systolic ejection murmur radiating to the carotids and right upper quadrant abdominal tenderness upon deep palpation. No midline spinal tenderness, rashes, or peripheral stigmata of endocarditis were appreciated. Other systemic examination was normal. Laboratory studies revealed mild normocytic anemia, leukocytosis with neutrophilia, hyperbilirubinemia (Direct Bilirubin=2.5 mg/dL) with mild transaminitis, and stage I acute kidney injury. Computed tomography (CT) scan of the abdomen was remarkable for prominent portacaval, gastrohepatic, upper mesenteric root and para-aortic lymph nodes with an intact aortic graft without stranding or inflammation. His c-reactive protein was notably elevated at 124 mg/L (reference range < 8). Blood cultures returned positive for *Listeria monocytogenes* at 16 hours in four bottles drawn from two peripheral sites. Ampicillin was initiated, but repeat blood culture remained positive at 24 hours. Transesophageal echocardiogram was negative for vegetations. Secondary to his persistent low back pain and concern for low grade endograft infection, positron emission tomography (PET CT) was obtained and revealed increased FDG uptake around graft in the distal aorta. He was deemed high risk for graft removal surgery and elected to undergo 6 weeks of ampicillin therapy followed by chronic suppression therapy.

Discussion: Prosthetic graft infection with *Listeria monocytogenes* is a rare but potentially fatal complication. To date, eight cases of *Listeria* prosthetic graft infection, two cases representing aortic endograft infection, have been reported worldwide. Although rare, practitioners need to be alerted to the ability of *Listeria* to cause focal infections including graft infections. Moreover, endograft infection needs to be on the differential diagnosis when patients with a history of endograft placement present with failure to thrive and back pain.

## A CURIOUS CASE OF COLD TOES

Korosh Sharain, MD Vincent S. Rajkumar, MD

CASE: A 65-year-old man with hypertension, hyperlipidemia, and gout presented with worsening bilateral foot pain and black toes. He described 8/10 burning foot pain which began one month prior to presentation that was treated with NSAIDs and gabapentin without significant relief. Over the subsequent weeks he developed blue discoloration of the tips of his toes which eventually turned black. He described worsening pain in the cold for which he would place blankets on his feet. He denied any preceding trauma, fevers, night sweats, or weight loss. On presentation, he was afebrile, blood pressure was 191/115 mmHg and heart rate was 110 bpm. His exam was notable for 2+ bilateral lower extremity edema to the mid-leg, nonpalpable dorsalis pedis pulses, livedo reticularis and dry gangrene of the distal toes bilaterally. Labs were significant for hgb of 9.0 g/dL, WBC of  $13.7 \times 10^9/L$ , creatinine 2.3 mg/dL, ESR 140 mm/1-hr, and CRP 71 mg/L. Urinalysis had RBCs without casts or dysmorphic cells and predicted a 24-hr protein of 6.4g. He was initially evaluated with lower extremity ultrasound and Dopplers, and ankle-brachial indices which were normal. Vasculitis evaluation with ANCA, ANA, and HCV were negative. An echocardiogram did not demonstrate an embolic source. A peripheral blood smear was performed to evaluate his anemia which demonstrated rouleaux formation; therefore, suspicion arose for a paraproteinemia. Serum protein electrophoresis and immunofixation demonstrated an IgG kappa monoclonal gammopathy with a kappa/lambda free light chain ratio of 241. Cryoglobulins were also present at 15%. A bone survey demonstrated a left femoral neck lytic lesion. A subsequent bone marrow biopsy demonstrated 80% involvement by plasma cells. Therefore, he was diagnosed with type I cryoglobulinemic vasculitis from multiple myeloma and light chain cast nephropathy. He was treated with plasmapheresis along with cyclophosphamide, bortezomib, and dexamethasone. His cryoglobulins disappeared after two treatments of plasmapheresis. He also eventually underwent stem cell transplant for his multiple myeloma.

DISCUSSION: This case highlights the importance of understanding the differential diagnosis of distal extremity gangrene. Common causes of distal extremity gangrene includes arterial occlusion from atherosclerotic disease or embolic phenomenon, severe venous occlusion, vasculitis, or infectious causes. However, cryoglobulinemia must be considered when venous and arterial studies are unrevealing. A history of worsening pain with cold temperatures and persistence of cyanosis despite the temperature should heighten clinical suspicion. Cryoglobulins are serum immunoglobulins that precipitate at temperatures

## DID TOFACITINIB CAUSE DISSEMINATED HISTOPLASMOSIS?

First Author: Bonnie C Sohn, MD

Introduction: Tofacitinib, an oral anti-rheumatologic agent approved in 2012, exerts its anti-inflammatory effect through inhibition of the JAK-STAT signaling pathway. In this patient, tofacitinib may have promoted dissemination of *Histoplasma* through inhibition of macrophage fungicidal capacity.

Case: A 59-year-old woman from Minnesota with rheumatoid arthritis (on tofacitinib, methotrexate, prednisone, and prophylactic valgacyclovir) presented to clinic in April 2015 with 2 weeks of productive cough and oral ulcers. She was treated with azithromycin and an increased valgacyclovir dose. Two weeks later, she returned for persistent dry cough, oral ulcers, fever, and confusion. MRI of the brain was normal. Tofacitinib and methotrexate were discontinued. Five days later, she presented to urgent care with an acutely worsened respiratory status, additional oral ulcers, and new rash.

Vital signs were T 36.9 °C, HR 121, RR 20, BP 136/90, and SpO<sub>2</sub> 94% on room air. Exam revealed lungs with occasional crackles, lateral punctate tongue lesions, and erythematous macules on extremities and abdomen. Chest CT revealed no acute pneumonic infiltrates. Initial rheumatologic and infectious workups were negative.

Her rash progressed to erythematous nodules over hospital day 3-5, and *Histoplasma* studies returned abnormal: urine antigen 21.10 (if > 0.5, positive), blood antigen 9.41 (if > 0.4, positive), serum antibody positive, mycelial phase titer of 1:256 (if > 1:32, suggests active disease), and H & M bands positive (if both bands positive, suggests active disease).

Itraconazole was initiated on hospital day 3. She subsequently developed hypoxia (SpO<sub>2</sub> 60%) on room air, and chest CT revealed faint patchy ground-glass opacities throughout the lungs, presumed to be an immune reconstitution inflammatory syndrome (IRIS). On hospital day 11, she improved and was discharged on oxygen. Over the following months, her erythema nodosum, oral lesions, and hypoxia resolved.

Discussion: *Histoplasma capsulatum* is endemic in Minnesota but typically a self-limited disease. However, tofacitinib, concomitantly administered with methotrexate and prednisone, likely augmented this patient's risk for an unusually severe course with constitutional symptoms, cough, oral ulcers, and erythema nodosum.

In vitro studies have demonstrated that tofacitinib targets dendritic cells, CD4+ T cells, and activated B-cells, which leads to inhibition of multiple cytokine pathways. This interruption of her cell-mediated immune response, and therefore suppression of macrophage activation, likely contributed to dissemination.

Opportunistic infections reported from phase II, III, and ongoing long-term extension studies of tofacitinib include VZV (multi-dermal / ophthalmicus), TB and non-TB mycobacteria, esophageal candidiasis, CMV (infection / viremia), cryptococcus (pneumonia / meningitis), *Pneumocystis jiroveci* pneumonia, and BK virus associated encephalitis. As we enter the post-marketing surveillance phase, it is important to be aware of opportunistic infections, for a delay in their diagnosis may lead to dismal outcomes.

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DISSEMINATED MYCOBACTERIUM CHIMAERA INFECTION IN AN IMMUNOCOMPETENT HOST  
FOLLOWING ASCENDING AORTIC ANEURYSM REPAIR

Nicholas Tan, MD Omar Abu Saleh, MBBS Rahul Sampath, MBBS Dragan Jevremovic, MD, PhD Andrew Badley, MD

Introduction: *Mycobacterium Chimaera* (*M. Chimaera*) is a slow-growing non-tuberculous mycobacterium (NTM) species that is normally associated with respiratory infections. Disseminated disease in immunocompetent hosts is rare but may occur in the setting of prior cardiovascular surgery.

Case Description: A 66 year-old man presented with several months of fatigue, weight loss, intermittent productive cough, and low-grade fevers. Relevant medical history included prior tobacco use and an ascending aortic aneurysm treated with a prosthetic graft 3 years prior to presentation. Initial laboratory workup was remarkable for hypercalcemia, elevated creatinine, and thrombocytopenia. Differential diagnoses included chronic infections, systemic autoimmune disease, and malignancy. Blood cultures (aerobic, anaerobic and fungal), endemic fungal serologies and antigen assays, hepatitis serologies, HIV, and TB Quantiferon tests were negative at the time. ESR and CRP levels were mildly elevated and antinuclear antibody titers, serum and urine protein electrophoresis were unremarkable. Echocardiogram showed normal ventricular function with no valvular abnormalities. Chest CT showed stable emphysematous and postoperative changes with no pulmonary nodules or infiltrates. A bone marrow biopsy was performed which revealed non-caseating granulomas and normal trilineage hematopoiesis. A presumptive diagnosis of sarcoidosis was made and he was started on oral prednisone 20mg daily.

The patient's symptoms did not improve despite steroid therapy. 3 weeks later, his bone marrow and blood cultures returned positive for NTM; this was later speciated as *M. Chimaera* by sequencing. Ophthalmic exam was remarkable for bilateral chorioretinitis. Immunological workup including lymphocyte subset panel, serum immunoglobulins, and vaccination titers yielded no evidence for a primary immunodeficiency; tests for autoantibodies to interferon-gamma and GATA-2 mutations also returned negative. A whole body PET/CT scan revealed diffusely abnormal FDG uptake in both lungs. In addition, an FDG-avid focal collection anterior to the aortic graft anastomosis was appreciated, raising suspicion for a potential nidus of his disseminated disease.

A CT-guided fine needle aspiration of the collection was deemed challenging and unsafe due to its proximity to the aorta. The mortality/morbidity risk associated with graft explantation was also felt to be unacceptably high. Hence, conservative management with antibiotic therapy (clarithromycin, rifampin, ethambutol, and amikacin) was initiated, with consideration for surgical intervention should his clinical course fail to improve.

Discussion: Disseminated *M. Chimaera* infection is extremely rare in immunocompetent hosts. However, recent literature described outbreaks among patients who underwent cardiovascular surgery; airborne contamination of heater-cooler unit water tanks used in cardiopulmonary bypass machines was implicated as the likely source of infection. Incubation time ranged from 1.5 to 3.6 years post-surgery. Outcomes of disseminated infection are guarded – 60% experienced breakthrough infections despite antibiotics and surgical exploration, and mortality was 40% overall. Among the NTM related infections, *M. Chimaera* may be an etiological consideration in the setting of prior cardiovascular surgery.

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A CRYPTIC PRESENTATION OF CRYPTOCOCCUS NEOFORMANS

First Author: Mazie Tsang, MD

Introduction: Spells can be a diagnostic enigma requiring consideration of a wide differential, including neurologic, cardiogenic, autonomic, infectious, and psychogenic etiologies. Here, we present a patient who had an unexpected cause of spells that should be considered in an immunocompromised patient.

Case: A 73 year old man—with a history of pulmonary sarcoidosis on chronic prednisone, stroke, and recent knee arthroplasty from which he was recovering at a swing bed—was admitted for spells, delirium, mental status changes, headaches, fevers, and cough. His delirium began shortly after being transferred to a swing bed facility, where he was noted to have a spell both at the facility and at an outpatient clinic visit. During his spells, his family noted that his upper extremities stiffen up prior to him losing consciousness. Upon admission, he was found to be febrile and coughing, so he was initiated on broad-spectrum antibiotics. He was placed on continuous electrocardiogram monitoring with no arrhythmia noted. Neurology was consulted and witnessed a spell, which was deemed to be consistent with a seizure, so he was started on levetiracetam. Magnetic resonance imaging and angiogram (MRI/A) of the brain showed new multi-territory strokes and vascular changes concerning for vasculitis; he was nearly started on high dose methylprednisolone before his cerebrospinal fluid and blood cultures grew *Cryptococcus neoformans*. Infectious Disease was consulted—he was started on Amphotericin B and flucytosine, and his broad-spectrum antibiotics were discontinued. Repeat MRI suggested that his vasculitic-like changes and multiple strokes were thought to be secondary to his central nervous system (CNS) infection. He was eventually transitioned to fluconazole and flucytosine prior to discharge to his swing bed facility.

Discussion: Cryptococcal meningitis typically is an indolent process that occurs over a period of weeks, with patients most commonly presenting with fever, malaise, confusion, and headache. Our patient who presented with spells was found to have multiple strokes that mimicked CNS vasculitis, which is a very unusual presentation of cryptococcal meningitis. One of the largest retrospective studies identified 28 patients with cerebral infarctions due to chronic meningitis, of which 39% had cryptococcal meningitis. Although there is a high index of suspicion for cryptococcal meningitis in patients with HIV and low CD4 count, it and other opportunistic CNS infections should be considered in the differential in patients on chronic immunosuppression who present with CNS vasculitis and stroke, especially if they are not improving despite other appropriate therapies. In conclusion, *Cryptococcus neoformans* is uncommonly a mimicker of CNS vasculitis, and our case emphasizes its unusual presentation and the importance of considering CNS infections in immunosuppressed patients who present with spells or stroke.

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## A RARE DIAGNOSIS THAT'S HARD TO SWALLOW

First Author: Allison L Yang, MD Second Author: Karthik Ravi, MD

Dysphagia is a common chief complaint in the primary care setting. Esophageal lichen planus is a rare cause of dysphagia and long segment esophageal strictures, and is often misdiagnosed and underrecognized, leading to significant delays in treatment.

A 79-year-old woman presented to clinic with progressive dysphagia to solid foods. She had been treated for a food impaction (pork chop) one year prior. She reported symptoms of dysphagia initially to meats and bread and later to fish and soft foods. At the time of evaluation, she was only able to eat small mouthfuls of mashed food and required several glasses of water with each meal. She denied odynophagia, regurgitation or reflux, or emesis. She had a seven pound weight loss in the three weeks prior to presentation. She was a lifetime nonsmoker and had no family history of esophageal malignancy. Physical examination revealed a thin woman and was otherwise unremarkable.

EGD showed a benign-appearing, intrinsic severe stenosis, which was dilated. Following the dilation, there was significant sloughing of the esophageal mucosa around the stricture site. Esophagram showed diffuse esophageal narrowing with the narrowest area measuring 6 mm. Biopsies showed parakeratosis, patchy intraepithelial lymphocytosis and dyskeratotic cells. The constellation of mucosa sloughing on EGD, diffuse narrowing on esophagram, and pathology was consistent with a diagnosis of esophageal lichen planus. She was treated with repeat endoscopy with dilation, triamcinolone injection to the esophagus, and started on budesonide. In the primary care clinic, the evaluation of dysphagia begins with differentiating oropharyngeal versus esophageal dysphagia. Esophageal dysphagia to solids is secondary to a mechanical obstruction due to peptic strictures or esophageal cancer (progressive symptoms), or esophageal rings or eosinophilic esophagitis (non-progressive symptoms). Dysphagia to both solids and/or liquids suggests a motor disorder such as scleroderma (progressive disease in the setting of chronic heartburn), achalasia (progressive diseases with regurgitation and/or respiratory symptoms), or esophageal motility disorders (intermittent symptoms).

Lichen planus is an uncommon diagnosis that can involve the skin, oral mucosa, genitalia, scalp, nails, or esophagus. Cutaneous lichen planus most frequently affects middle age adults and the etiology is not known. Esophageal involvement is very rare and is characterized by endoscopic findings of peeling, friable mucosa, white plaques, and stricture formation in the upper and mid-esophagus. Barium swallow often shows a long segment smooth stricture. Treatment usually includes topical or systemic steroids and stricture dilations.

This case highlights a rare cause of dysphagia and the importance of accurate diagnosis with EGD and/or barium swallow and early management with steroids.

## A FEVER FROM AFRICA: AN UNUSUAL PRESENTATION OF HEMOPHAGOCYTIC LYMPHOHISTIOCYTOSIS

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**Introduction:** A patient with fever of unknown origin is a diagnostic and therapeutic challenge, especially when confounded by a clinical syndrome suggestive of an African illness. We present a complicated presentation of a case of hemophagocytic lymphohistiocytosis (HLH).

**Case Description:** A 41-year-old male with no past medical history presented with fever, chills, arthralgia and macular rash on his trunk and distal extremities. His symptoms began while working off shore in Equatorial Guinea. Physical exam showed severe synovitis of his bilateral ankles, wrists and proximal interphalangeal joints. Laboratory work revealed leukocytosis, microcytic anemia, elevated lactate dehydrogenase, elevated creatinine, elevated transaminases, and signs of significant inflammatory response including elevated erythrocyte sedimentation rate, C-reactive protein and a ferritin >100,000 mg/L. Antinuclear antibody, rheumatoid factor, hepatitis and HIV was negative. We treated empirically with doxycycline and quinine for Malaria and vancomycin and meropenem for sepsis. Bone marrow biopsy revealed prominent clear inclusions within erythroid precursors. No hemophagocytes were noted. Serologic testing returned negative for Chikungunya virus, malaria, Parvovirus, Epstein Barr, dengue, Cytomegalovirus, and Arbovirus. After admission, patient's symptoms severely worsened. Antibiotics were discontinued, and he was started on stress-dose methylprednisolone to suppress immune response. Bone marrow specimens were sent to the Center of Disease Control for additional viral testing including an "African Viral Panel." These results were also negative. Computed tomography revealed splenomegaly. Interleukin 2 returned elevated (4720) and his natural cell activity resulted low to absent thus meeting 5 of 8 criteria of HLH.

HLH was presumed to be viral induced, likely from an unknown African virus. We started him on dexamethasone and etoposide per the HLH 2014 protocol. Subsequently, his fevers resolved and ferritin trended down. He was discharged home two weeks later.

**Discussion and Conclusion:** Initially, this patient's clinical picture suggested severe sepsis secondary to viral illness such as Chikungunya, Malaria or another African viral illness given his recent travel history. It is difficult to distinguish HLH from sepsis syndrome. However, with fever of unknown origin and ferritin > 3,000 µg/L, HLH should also be considered. Elevated ferritin is also seen in systemic juvenile rheumatoid arthritis, adult onset stills disease and hemochromatosis. Prompt diagnosis of HLH is critical as it is often a fatal disease. Diagnostic criteria include 1. Fever = 38.5°C, 2. ferritin > 500 ng/ml, 3. elevated soluble interleukin 2, 4. low to absent natural killer cell activity, 5. Bicytopenia, 6. Splenomegaly, 7. fibrinogen < 150 mg/dL or fasting triglycerides >265 mg/dL, 8. hemophagocytosis in bone marrow, spleen, lymph node, or liver. Five criteria are required for the diagnosis. In adults, HLH is most commonly secondary to infection, hematologic disorders, malignancy or rheumatologic diseases. This patient likely had viral induced HLH that we were unable to identify. No reports of African viral illness have been associated with HLH.



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SYSTEMIC LOXOSCELISM: A RARE CAUSE OF COOMBS POSITIVE HEMOLYTIC ANEMIA

First Author: Keith W Murdock, MD Second Author: Jericho L. Bell, MD Third Author: Jeffery J. Grondin, MD Fourth Author: Scott M. Letellier, MD

Introduction: Loxoscelism is a term used to describe systemic illness caused by the envenomation by recluse spiders. Although it commonly presents with small necrotic lesions, it has been known to rarely cause multi-organ failure, DIC and hemolytic anemia (1). Here we report a case of systemic loxoscelism as a rare cause of autoimmune hemolytic anemia (AIHA).

Case report: 47-year-old African American female presented to the emergency room with a 1-week history of non-radiating back pain between her shoulder blades. Prior to her back pain, she noted a small area of redness and swelling on her back. She worked at a nursing home, which she reported had a brown recluse spider infestation, and believed she had a spider bite. She sought outpatient medical care when she began having intermittent fevers and was treated with clindamycin. The lesion did not improve and developed into larger eschar. She returned to medical attention for nausea, fatigue, muscle weakness, and generalized abdominal pain. Physical exam was remarkable for a 1cm x 1cm dry eschar overlying the right scapular region with 2mm area of erythema surrounding it and sublingual icterus. Labs on admission demonstrated an elevated bilirubin (primarily indirect), leukocyte count of  $32 \times 10^9/L$  (5% blast, 6% bands, many nucleated RBC's), hematocrit of 19.3%, lactate dehydrogenase of 1126units/L and haptoglobin <10mg/dL. Her direct Coombs was positive.

Initial differential diagnoses included autoimmune hemolytic anemia, chronic myeloid leukemia, and drug induced hemolytic anemia. Upon admission to the hospital, the patient received blood transfusions. Peripheral smear was without schistocytes, had multiple nucleated RBC's and left shifted population of neutrophils. She continued to be anemic despite transfusion, and was started on 1 mg/kg prednisone for AIHA associated with a brown recluse spider bite. Her leukocytosis, anemia, eschar and symptoms normalized after completion of a steroid course.

Discussion: Systemic loxoscelism is a rare cause of Coombs positive hemolytic anemia. When confronted by a hemolytic anemia, in a patient with a spider bite or develops a suspicious eschar, a clinician should consider systemic loxoscelism as a cause. Additionally, with signs of systemic illness, the patient should be hospitalized for treatment and supportive care (2). In our case, the patient was able to achieve a full recovery after treatment with prednisone and close observation.

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CANDIDA PARAPSILOSIS PROSTHETIC VALVE ENDOCARDITIS PRESENTING AS ST ELEVATION MYOCARDIAL INFARCTION

Ian Ross MD, Jaime Bolda MD, Gerome Escota MD

Introduction: Fungal prosthetic valve endocarditis is an uncommon disease, accounting for only 1.3-6% of all cases of infectious endocarditis. *Candida albicans* is the most common causative pathogen, and *Candida parapsilosis* is the most common non-*albicans* species. Fungal endocarditis embolizes more frequently than bacterial endocarditis and can rarely present as an ST elevation myocardial infarction.

Case Description: A 57-year-old male with a history of aortic bileaflet mechanical prosthetic valve and intravenous drug use presented to the ER with complaints of chest pain, syncope, and abdominal pain. An ECG showed 2 mm ST elevations in the inferior leads. Immediate cardiac catheterization was aborted due to coagulopathy. The patient's myocardial infarction was managed medically. Echocardiography showed a 1.4x0.8 cm echogenic mass on the anterior leaflet of the mechanical aortic valve. Subsequent imaging showed hemoperitoneum, superior mesenteric artery and splenic mycotic aneurysms, as well as renal infarcts. Blood cultures grew *Candida parapsilosis*. The patient was treated with micafungin and fluconazole and underwent valve replacement. His aneurysms and hemoperitoneum were treated surgically.

Discussion: *Candida parapsilosis* is an important pathogen in fungal endocarditis, and is especially prevalent in intravenous drug users. Coronary septic embolism is a rare cause of ST elevation myocardial infarction and was first described by Virchow. It is estimated to occur in only 0.5-10% of cases of infective endocarditis and the left anterior descending artery is most commonly affected. Fungal endocarditis is associated with a higher incidence of embolic events than bacterial endocarditis. Elements in the history and presentation that can aid in diagnosis include a history of intravenous drug use, valve replacement, or concurrently observed embolic/hemorrhagic phenomena. Combined medical and surgical therapy is the mainstay of treatment, with isolates usually susceptible to amphotericin and azole antifungals.

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CRYPTOCOCCAL ENDOCARDITIS IN A HIV-SERONEGATIVE PATIENT WITHOUT VALVULAR HEART DISEASE

First Author: Danicela Younce, MD

Fungal endocarditis is an uncommon but serious complication of immunocompromised states. Here I present a case of cryptococcal endocarditis in a HIV-seronegative patient without preexisting valvular disease.

A 57-year-old Caucasian man with a history of diabetes, coronary artery disease, alcoholic cirrhosis and hepatitis C presented with worsening confusion for five days. He was discharged from the hospital ten days earlier after evaluation for dizziness and headaches which was thought to be due to dehydration and poor oral intake. Soon after admission he became nonverbal and had new onset left gaze deviation with right hemiplegia. Emergent head CT was negative for intracranial hemorrhage and he received tPA for suspected ischemic stroke. Post tPA MRI showed multiple infarcts involving bilateral basal ganglia, left frontal lobe, and cerebellar hemispheres concerning for cardioembolic disease. Also evident were multiple foci of intraparenchymal and intraventricular hemorrhage thought to be due to tPA administration. The patient was intubated and transferred to the ICU. He began spiking fevers and broad spectrum antibiotics with meningitis coverage were started. His blood cultures however, remained negative as did infectious work up of his ascitic fluid and urine. Transthoracic echocardiogram (TTE) showed a vegetation on the left AV leaflet. Lumbar puncture showed xanthochromia, high opening pressure, low glucose, high protein and high WBC count. CSF culture grew *Cryptococcus neoformans* and his blood cryptococcal antigen titer was elevated. He was started on amphotericin B and flucytosine for cryptococcosis with meningoencephalitis and endocarditis. His course was complicated by pleural effusions, sepsis and pneumonia resulting in acute hypoxic respiratory failure. Given poor prognosis, his family elected to make him comfort care and he died a day after antifungal treatment was initiated.

Based on review of the available literature, this represents a rare case of *Cryptococcus neoformans* endocarditis in a HIV-negative patient without preexisting valvular heart disease or undergoing treatment with immunosuppressive agents. In eight documented cases, where HIV status was negative or unknown, four had undergone prosthetic valve surgery, three had known rheumatic heart disease and the last case was on immunosuppressive therapy for malignancy. The diagnosis was made by evidence of vegetation on TTE, multifocal cardioembolic strokes, positive cryptococcal serum titer and clinical correlation. The predisposing factor in this patient was his advanced cirrhosis which rendered him immunocompromised. This diagnosis should be considered in all immunocompromised patients with or without valvular heart disease where there is evidence of endocardial vegetation on cardiac imaging.

## CHAPTER WINNING ABSTRACT

Lyndsey Heise, MD, Jason Shiffermiller, MD, MPH, Steven Paulmeyer, MD, Michael Smith, MD

A 72 year-old man presented with three to four months of progressive dyspnea. He went on to develop a requirement for supplemental oxygen and required four liters per nasal cannula. The patient underwent a coronary artery bypass graft three months prior to presentation which was complicated by a pericardial effusion and atrial fibrillation. His other active conditions include diabetes mellitus, COPD, and chronic lymphocytic leukemia with treatment held for cardiac surgery. Exam revealed mild respiratory distress with trace peripheral edema, normal cardiac auscultation, and absent breath sounds in the lower left lobe. On admission, hemoglobin was 7.7 compared to a baseline of 10. Chest x-ray revealed loculated pleural effusion that previously had been evaluated and attributed to post-operative complication following the coronary artery bypass graft. Thoracentesis was performed and resulted in bloody pleural fluid that was exudative in nature. Pleural fluid hematocrit was 9%, markedly lower than serum hematocrit making it inconsistent with a pure post-operative bleeding process. PET scan indicated avid glucose uptake of left pleura. A left video-assisted thorascopic surgery, pleural decortication, and pleural biopsy were performed. Two large bore chest tubes were placed with sero-sanguinous output for three days to allow resolution of effusion. Pathology examination of the pleura revealed chronic organizing fibrinous pleuritis with foci of small lymphocytic lymphoma/chronic lymphocytic leukemia cells. Patient was discharged eight days after surgery with no supplemental oxygen requirement and a stable hemoglobin with plan for re-initiation of chemotherapy.

## Learning Objectives

1. Recognize the differential diagnoses of a bloody pleural effusion.
2. Understand possible intrathoracic complications of chronic lymphocytic leukemia.

Discussion: Pleural effusions are very commonly encountered by physicians and can be categorized by a variety of different features that will help with development of differential diagnoses. Pleural effusions can be classified by bilateral or unilateral presence. Right versus left sided pleural effusions can indicate different etiologies by consideration of the adjacent organ systems. Additionally, gross appearance of the pleural fluid can help refine the differential diagnoses. A bloody pleural effusion is suggestive of etiologies such as malignancy, pulmonary embolus, trauma, and pulmonary vascular malformation.

Chronic lymphocytic leukemia can manifest in a wide variety of ways. Some of the most serious complications of chronic lymphocytic leukemia occur intrathoracically and can mimic other conditions commonly encountered by physicians such as congestive heart failure, COPD, and community-acquired pneumonia. If a patient has a known or suspected chronic lymphocytic leukemia, it must be considered in the differential diagnoses of each of these conditions. Chronic lymphocytic leukemia, however, is a very rare cause of pleural effusion. Pleural effusions are a common finding on chest x-ray and a differential list for bloody effusions will help tailor the investigation allowing earlier implementation of appropriate treatment.

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PERICARDIAL TAMPONADE: A RARE LIFE THREATENING COMPLICATION OF HYPOTHYROIDISM

First Author: Shweta Kukrety, MD Jeff Murray MD, Abhilash Anikapelli MD, William P Biddle MD

Introduction: Pericardial effusion in overt hypothyroidism is common with the incidence ranging from 3-6 % in mild cases to 30-80 % in severe cases of hypothyroidism. However, cardiac tamponade due to hypothyroidism is a rarely seen complication as the fluid accumulates gradually, allowing for the pericardium to distend.

Case Description: A 52 year old gentleman with a past medical history of hypothyroidism and hypertension, presented to the Emergency Room with worsening shortness of breath over a 1-2 week period. The patient was non-adherent to his medications. Social history was significant for daily marijuana and methamphetamine use. On examination, his blood pressure was 100/60 and heart rate was 80. He had jugular venous distension, distant heart sounds, pulsus paradoxus and 3+ bilateral pedal edema. Chest X-ray revealed enlarged cardio-mediastinal silhouette. Subsequently, a transthoracic Echocardiogram was performed which showed severe Left ventricular hypertrophy (LVH) with a large circumferential pericardial effusion of 3 cm in size with diastolic collapse of the right atrium and right ventricle, indicative of pericardial tamponade. A complete laboratory panel was obtained which was significant for a TSH of 200 micro international units per Litre (normal 0.5 - 4.70) with low T4 levels. Our patient was initiated on intravenous thyroxine replacement. He underwent surgical drainage of the effusion with pericardial window formation due to predominant posterior location of the effusion.

Discussion: Cardiac tamponade occurring secondary to severe hypothyroidism is rare and a high index of clinical suspicion is required for timely diagnosis and management. Hypothyroidism should be suspected in the absence of sinus tachycardia in the setting of tamponade. Management of cardiac tamponade depends on the hemodynamic status of the patient. Cardiac tamponade with minimal hemodynamic compromise may be treated conservatively, with close hemodynamic monitoring, avoidance of volume depletion, and thyroxine replacement. Patients with severe hemodynamic compromise require drainage of the pericardial fluid. Pericardial fluid removal can be accomplished by either catheter pericardiocentesis or open surgical drainage with or without pericardial window formation. Echocardiographically-guided pericardiocentesis is a preferred approach due to its ability to be performed more rapidly and with lower complication and mortality rates. In our patient echocardiographically-guided pericardiocentesis would have been technically challenging as much of the effusion was posterior. Hence the patient underwent open surgical drainage with pericardial window formation.

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THYROID ABSCESS: AN UNUSUAL CAUSE OF HYPERTHYROIDISM

First Author: Ahl Jeffrey Caseja, MD

**Introduction:** Infection of the thyroid gland is a rare occurrence and is uncommonly associated with hyperthyroidism. We report a case of a thyroid abscess with subsequent hyperthyroidism in an otherwise immunocompetent patient.

**Case Description:** A 34-year-old female with no significant past medical history presented with fevers, chills, and progressive left-sided, non-radiating throat pain of 10-day duration. The pain worsened with swallowing and movement of the neck. On presentation, blood pressure was 128/80 mmHg, heart rate 114 bpm, and temperature 100.3 F. Physical examination revealed an ill-defined tender mass in the left anterior neck, without drainage or erythema. Tremors, diaphoresis, and hyperreflexia were absent. Her thyroid function tests were significant for a TSH of <0.100 uIU/ml (0.35-4.94), FT4 of 2.38ng/dl (0.70-1.48), FT3 of 3.7pg/ml (1.7-3.7), and TSI of 17% (0-139). Computed tomography of the neck showed a rim-enhancing fluid collection with an epicenter at the left thyroid lobe. HIV test was nonreactive. The abscess of the neck was incised and drained. She was initially treated with Vancomycin, Ceftriaxone, and Metronidazole. Wound culture grew B-hemolytic streptococcus sensitive to penicillin. She was discharged with a 7-day course of Cephalexin along with Propranolol. The patient was seen in follow-up one month later, and her labs showed normal TSH of 2.70 uIU/ml, FT4 of 1.1ng/dl, and an FT3 of 2.7pg/ml

**Discussion:** The thyroid gland is relatively resistant to infection due to its rich vascular and lymphatic supply, a high iodine content, and the thyroid capsule. Thyroid abscesses are far less common than other inflammatory conditions of the thyroid gland. Prompt diagnosis is key, as it is potentially life threatening.

Abscesses most commonly develop in children secondary to piriform sinus fistula formation. In adults, they develop in immunocompromised patients via hematogenous spread. Gram-positive (*S. aureus, streptococci*), gram-negative (*H. influenza, Enterobacteriaceae*), and anaerobic bacteria (from oropharyngeal flora) are the most typically identified pathogens.

Unlike what we found in this case, serum levels of T4, T3, and TSH are generally normal. Management of hyperthyroidism in the setting of an abscess involves symptom control with beta-blockers. There is no role for treatment with anti-thyroid medication as there is no increase in thyroid production. Traditional management of a thyroid abscess involves open surgical drainage and antibiotics as was done for our patient. Recent reports demonstrated good outcomes using less invasive procedures such as percutaneous image-guided drainage with catheter irrigation or intra-cavitary antibiotics. If extensive necrosis develops, or if infection persists despite adequate antibiotics, thyroid lobectomy may be necessary.

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CHAPTER WINNING ABSTRACT UNILATERAL CONDUCTION HEARING LOSS DUE TO CENTRAL VENOUS OCCLUSION

Philip Ribeiro MD, Swetal Patel MD, Rizwan Qazi MD

**Abstract:** Central venous stenosis is a well known complication in patients with vascular access for hemodialysis. We report 2 cases of patients on hemodialysis with arteriovenous fistulas developing reversible unilateral conductive hearing loss secondary to critical stenosis of Central Veins draining the arterio-venous dialysis access. A proposed mechanism for the patient's reversible unilateral hearing loss is pterygoid venous plexus congestion leading to decreased Eustachian tube patency. Endovascular therapy was conducted to treat the stenosis and both patients hearing loss was returned to near normal after successful central venous angioplasty.

**Introduction:** End stage renal disease is prevalent worldwide. As such, the advent of hemodialysis has become one of the more prominent medical advances of the century. There were 3.2 million patients on hemodialysis for ESRD at the end of 2013 [7]. This number is rapidly growing, and along with this rapid growth come a multitude of complications. In recent years the field of vascular access for patients requiring hemodialysis has had numerous advances in arteriovenous fistulas and grafts. However, despite advances, there are still many complications and unforeseen pathologies.

In current literature, central vein disease (CVD) is defined as greater than 50% narrowing of the thoracic central veins. These veins include the superior vena cava (SVC), brachiocephalic (BCV) and subclavian (SCV) [5]. The incidence of CVD has been reported to be as high as 23% in the total dialysis population and 41% in those with access-related complaints [6]. Many fistulas do not sustain dialysis due to poor maturation, thrombosis, or critical stenosis [3,4]. Central Venous stenosis and occlusion are a result of high vascular wall shear stress due to the presence of AVF or AVG upstream and vascular compression or distortion frequently seen in aging and due to previously placed central venous catheters [1]. Once critical stenosis has developed patients are at risk for central venous occlusion, which can result in face, neck, or arm swelling, along with other severe disabling complications [2,11].

After careful literature review, there is a paucity of data regarding the development of reversible unilateral conductive hearing loss secondary to critical stenosis of venous access. This disabling symptom of central venous occlusion has gone under recognized. We two case reports of reversible unilateral conduction hearing loss due to central venous occlusion. Both of these patients were on hemodialysis and hearing returned to near normal after central venous angioplasty.

**Case Report:** *Case #1* Our first case is of a 66 year old male with ESRD on hemodialysis for the past 8 years. His vascular access is a left upper arm brachiocephalic arteriovenous fistula. He presented to our vascular access center with left arm swelling. He also complains of hearing loss from his left ear off and on for the past five months. On further inspection, we noticed a wick of cotton in the patients left ear that he places before sleeping because he hears a continuous, loud streaming noise. Physical examination showed a left upper arm Brachial Cephalic fistula which was hyperpulsatile on palpation and does not collapse on raising the arm. The patient was taken for an angiogram of his AV fistula, which showed a 90% stenosis of the left innominate vein. He underwent

successful venous angioplasty with a 14mm x 40mm atlas venous angioplasty balloon to 10% residual. Patient had significant improvement in left arm swelling, and most noticeably had significant improvement in hearing immediately following angioplasty in the recovery area.

*Case #2* Our second case is of a 71 year old female with past medical history of hypertension, diabetes mellitus type II, and ESRD on hemodialysis for the past 2 years. Her vascular access is a left upper arm brachiocephalic arteriovenous fistula. She presented to our vascular access center with left arm and left sided facial swelling. She also complained of gradual left sided hearing loss for the past 2 months. Physical Examination revealed a left upper arm Brachial Cephalic fistula which was hyperpulsatile on palpation and did not collapse upon raising the arm. The patient was taken for an angiogram of her AV fistula, which showed a 80% stenosis of the left innominate vein. She underwent angioplasty with a 14mm x 40mm atlas venous angioplasty balloon to 20% residual. Immediately post procedure she had significant improvement in her hearing. She has since returned to our access center three times with similar complaints of arm swelling and hearing loss both of which resolve after central venous angioplasty.

**Discussion** Unilateral reversible conductive hearing loss is an uncommon but under recognized consequence of central venous occlusion. The exact mechanism of deafness, although not known, is likely due to swelling and congestion of the eustachian tubes [ET].

The Eustachian tube is a narrow tube that connects the middle ear to the back of the nose. Blockage of the Eustachian tube isolates the middle ear space from the outside environment. The lining of the middle ear absorbs the trapped air and creates a negative pressure that pulls the eardrum inward. When it becomes stretched inward, patients often experience pain, pressure, and hearing loss. Long-term blockage of the Eustachian tube leads to the accumulation of fluid in the middle ear space that further increases the pressure and hearing loss [10].

There are several muscles that allow for opening of the eustachian tube. The principal and perhaps only dilator of the tube is the tensor veli palatini (TVP)[9]. The TVP functions to tighten the anterior part of the soft palate and assist in opening the pharyngotympanic tube. Studies have shown that abnormal insertion of the TVP into the cartilage of the Eustachian tube was found to cause a functional obstruction that led to an increase in incidence of otitis media. This anatomical relationship between the TVP and the Eustachian tube is well described in literature.

Concurrently, the pterygoid plexus is the main venous drainage for the eustachian tube [9]. The proposed mechanism for our patient's reversible unilateral hearing loss is congestion of the pterygoid venous plexus (PVP) leading to decreased Eustachian tube patency. Oshima et. al [8] study used MRI to evaluate the anatomical relationship of the muscles of the neck and the PVP before and after neck compression. The results showed that the lateral pterygoid muscle became enlarged after neck compression. Simultaneously, the volume of the PVP observed between the medial pterygoid muscle and tensor veli palatini muscle was increased. The increased volume of the PVP led to protrusion of the ET anterior wall to the luminal side, and thus decreased ET patency [8]. Given this anatomical relationship, we postulate that in our patients, central vein occlusion led to PVP congestion, which in turn led to compression of the ET.

The loss of ET patency is what likely contributed to our patients' unilateral hearing loss. After adequate venous outflow was restored with angioplasty our patients' hearing was restored. These symptoms can be quite disabling to patients and significantly affect their quality of life. Timely recognition of the cause and treatment can reverse deafness, improve patient's quality of life, and prevent disability from becoming permanent.

MESENTERIC ISCHEMIA: A RARE DYSAUTONOMIA COMPLICATION OF GUILLAIN-BARRÉ SYNDROME.

First Author: Ravi Raj Shah, MD Second Author: Terin Howard Martinjak, MD

Guillain-Barré syndrome (GBS) is a rare acute immune mediated polyneuropathy with an incidence of 1-2 per 100,000 worldwide each year. Dysautonomia is commonly seen with GBS affecting up to 70% of these patients, however it can result in fatal and life threatening complications secondary to severe orthostatic hypotension, cardiac arrhythmias and in some cases death. Therefore, early recognition, understanding, and treatment of dysautonomia complications is a very important part of clinical management.

79 yr. old Caucasian male with history of pyelonephritis, diabetes mellitus type II, and hypertension presented with a four-day history of ascending lower extremity weakness. Pertinent physical exam findings were hyperalgesia, hyperpathia, 2/5 strength and areflexia of bilateral lower extremities. Diagnostic lumbar puncture was done with a high protein level of 251 indicative of GBS. CT imaging of lumbar spine showed no acute pathology. Patient was treated appropriately with plasmapheresis with 4 sessions. Despite these efforts, the patient developed autonomic dysfunction leading to episodes of hypotension. Renal function deteriorated to BUN/Creatinine ratio of 100/6.2 and the patient developed symptoms of uremia. Patient subsequently developed severe abdominal pain and peritoneal signs on physical exam; a stat CT abdomen and pelvis revealed pneumatosis coli and intestinalis with mesenteric and portal venous gas. General Surgery performed an emergent exploratory laparotomy with an unextended right hemicolectomy and small bowel resection was performed due to extensive necrosis most consistent with mesenteric ischemia.

Autonomic dysfunction is a complication associated with GBS up to two thirds of cases. Cardiovascular dysautonomia is commonly seen when there is extensive motor involvement with GBS and changes in hemodynamic status are expected. These hemodynamic changes are thought to be related to 17OH corticosteroids, renin levels, atrial natriuretic factors, aldosterone levels, CSF dopamine, and serotonin metabolites amongst other factors. Renal failure is a common dysautonomic complication which significantly increases mortality in these patients. Although relationship between dysautonomia and GBS is established, there have not been any cases describing pneumatosis coli and mesenteric ischemia secondary to dysautonomia. The case described above is a significant example of how dysautonomia can be detrimental and catastrophic. More studies are needed to establish the exact underlying etiology of this dysautonomia, the prognosis associated, and development of early diagnostic tools to prevent this life threatening complication.

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TB OR NOT TB: AN UNLIKELY PATIENT ENCOUNTER IN RURAL NEW HAMPSHIRE

Jason H Malenfant MD, Sukeerti G Kesar MD

**Introduction:** Tuberculosis (TB) remains a widespread and often fatal disease in the developing world, but its incidence has continued to decline in the United States since the early 1990s. Of the 9945 cases of tuberculosis reported in the U.S. in 2012, approximately 20% of these were miliary TB. Here we present a case of confirmed miliary TB in a patient whose exposure remains a mystery.

**Case Report:** A 90 year old French-speaking female with a past medical history significant for chronic pancytopenia, HTN, and dyslipidemia, presents in transfer from an outside hospital after being admitted there for a syncopal episode and fall. CT work up revealed a psoas abscess. IR drainage of the abscess had revealed acid-fast bacilli. Further history brought to light a 3 month history of decreased appetite, low grade fevers and night sweats, and nausea/vomiting. She also estimated a 12 lb weight loss over the past month. She was a nun who had been living in convents for the past 67 years, most recently in the same convent in Vermont for the past 8 years. She had worked in nursing homes but no prisons or shelters. Her only travel history was a trip to Europe 40 years earlier. She did not have a history of any recreational drug use, had a negative HIV screen and no known exposures to tuberculosis.

Initial vital signs were all within normal limits and exam revealed a well-appearing elderly female with psoas drain in place expressive of purulent material. She was admitted to the medicine floors with airborne precautions and treatment with isoniazid, rifampin and pyrazinamide was initiated. On day 3 of her hospital course she had two near syncopal events when standing after feeling dizzy. That afternoon she began to have waxing and waning mental status and an increasing O<sub>2</sub> requirement to 12L. Her abdominal exam was notable for a newly tense and tender abdomen. Her BP declined to 80/30s range with minimal improvement to the 90/40s range with IVF boluses. Labs drawn during this event revealed a stable hemoglobin but increased WBC count to 20.6K (from 7.5K) as well as increasing direct bilirubin (3.0 from 0.1) and creatinine (1.92 from 0.73). The patient had advanced directives that specified no aggressive therapy including invasive lines or BiPAP, and with help from her sisters who were present at bedside, she was made CMO (comfort measures only) and passed away shortly after. An autopsy was unable to be performed due to the Vermont Department of Health policies. A post-mortem bronchoscopy was performed and Mycobacterium tuberculosis complex was isolated at 4 weeks.

**Discussion:** Given the rather sudden decompensation that was observed in this patient, there were two theories posed regarding her cause of death. The first theory explored the cause as being secondary to multi-organ failure from sepsis as a result of her widespread disease. The second theory posited that the patient suffered from immune reconstitution syndrome (IRS), a paradoxical worsening of clinical status after the initiation of anti-tuberculosis therapy. Though the pathogenesis of IRS in HIV patients on HAART therapy is well explained, the underlying physiology is less clear in TB patients, though its incidence has been well documented. Though this case ended in tragedy, it proved to be a valuable learning experience for trainees in a medical center where this disease is uncommonly seen.

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MULTIPLE RING ENHANCING LESIONS ON MRI OF THE BRAIN IN TRANSPLANT RECIPIENT: A DIAGNOSTIC DILEMMA

First Author: Md Usman Ali MD, Mohammed Al-Jumayli MD, Daniel Eichorn, Khurram Zubair MD, Daniel Goldsmith MD

**Introduction:** Post-transplantation primary central nervous system lymphoma (PT-PCNSL) is a very rare tumor that can present from months to years after transplantation. Although it is frequently encountered in a renal transplant recipient, can certainly present in liver and other solid organ transplant recipients too. Its symptoms can be varied ranging from non-specific symptoms such as headache, gait disturbance, change in mental status to the focal neurological deficit. Inflammatory markers such as C-reactive protein, LDH, and ESR may be elevated whereas CSF analysis often is inconclusive. Here we present a case of PT-PCNSL, who was initially misdiagnosed and treated as brain abscess until brain biopsy proved it otherwise

**Case:** A 41-year-old Hispanic female with a past medical history of bacterial endocarditis, renal transplant (1994) secondary to glomerulonephritis treated with mycophenolate and tacrolimus presented with a transient change in mental status. She had stable vitals without neurological deficits and the spontaneous return of baseline mental status. Her MRI showed two ring-enhancing lesions in the right temporal and parietal lobe. Considering her previous history of endocarditis, current immunosuppression, MRI findings and non-specific neurological manifestation, she was treated as a brain abscess without any significant response. Her blood culture remained negative, and CSF analysis was unremarkable. Therefore, she underwent brain biopsy that characterized it to be diffuse large B-cell lymphoma that was assumed to be post-transplant. Her immunosuppression was withheld; subsequently she underwent tumor resection followed by Rituximab therapy with an effective response, and she is currently disease free

**Discussion:** Although PT-PCNSL is a very rare entity, it is increasingly recognized in transplant recipient as the number of transplant recipients rises, and better survival outcomes are achieved. Early diagnosis is a harbinger of a better outcome. Therefore high index of clinical suspicion should always be exercised in this patient population. Chronic immunosuppression plays a critical role in the etiopathogenesis of these neoplasms and often, the treatment is withholding immunosuppression itself. MRI is a better diagnostic modality than CT; however, it can still be difficult to diagnose accurately these ring-enhancing lesions given the wide-range of the differential. As a result, brain biopsy becomes mandatory to establish the diagnosis in most cases. PT-PCNSL is hard to diagnose, easy to miss and rather difficult to treat with variable prognosis.

**Conclusion:** Although the differential diagnosis of ring-enhancing lesions on MRI includes glioma, metastatic malignant lesions, bacterial and non-bacterial abscesses, toxoplasmosis, and multiple sclerosis plaques, clinicians must consider PT-PCNSL highly in transplant recipients.

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## HARK, THE HERALD BLEEDING INTERVAL!

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A primary aortoenteric fistula (PAEF) is a connection between the aorta and gastrointestinal tract not secondary to surgical cause. Surgical intervention at early signs of presentation is necessary to prevent exsanguination.

A 56-year-old woman with hypertension, hyperlipidemia, diabetes and smoking history presented with one week of increasing abdominal and back pain with melena. She was febrile to 38.2°C. Abdominal computed tomography (CT) revealed extensive atheromatous disease of the abdominal aorta, concern for aortitis and duodenal ulcer. Blood cultures grew *Escherichia coli*. Upper endoscopy did not show an ulcer. The patient went home to complete a course of ciprofloxacin and metronidazole for concern of aortitis.

Five years later, the patient presented with abdominal pain and rectal bleeding. Upper endoscopy showed possible gastrointestinal stromal tumor with additional active bleeding in the duodenum, requiring cauterization and clipping. Blood cultures grew *Klebsiella pneumoniae*. CT angiography revealed contiguity between the fourth part of the duodenum and the anterior wall of the abdominal aorta, representing a PAEF. She went into hemorrhagic shock and was taken for emergent laparotomy with resection of affected areas of aorta and duodenum and placement of a rifampicin-soaked aortic graft. Pathology of the fistula specimen revealed an inflammatory process of granulation tissue and fibrosis consistent with foreign body reaction to cholesterol debris. The patient was discharged with six weeks of intravenous ertapenem and did well.

According to a large autopsy study, the incidence of PAEF in the general population is 0.07%. The classic PAEF triad includes massive gastrointestinal bleed, pulsatile abdominal mass, and abdominal or back pain, though only approximately 11% of patients with PAEF have the triad.

A herald bleed is defined as the initial hematochezia or hematemesis foretelling an impending massive bleed from an aortoenteric fistula. Mortality is 100% without surgical intervention. In patients who present with herald bleed, the time interval between herald bleed and massive exsanguination is greater than six hours in 70% of patients, but only 29% have an interval of greater than one week. Our patient had an extremely rare presentation since she had a five-year interval between herald bleed and massive gastrointestinal bleed. This patient's PAEF was most likely a result of severe atherosclerotic disease, and the fistula may have clotted off for the five-year interval. Postoperatively, patients with PAEF should be treated with broad-spectrum antibiotics for four to six weeks if they are bacteremic and at least one week if not bacteremic. This case is important and unusual because of the successful outcome despite an extended time interval between herald and massive bleeds.

## PARANASAL SINUSITIS LEADING TO BRAIN ABSCESS

First Author: Ciandra Dsouza Spiro Mavromates, Abyi Tessema, Sunil Sapru

A brain abscess is a focal collection of infected material within the brain parenchyma. Infection can arise through direct spread from a contiguous body site or through hematogenous seeding. Numerous pathogens can be the causative organism; however *Streptococcus* and *Staphylococcus spp.* are the most common. The viridans group of streptococci, specifically *Streptococcus anginosus* is particularly associated with abscess formation following primary infection of the paranasal sinuses and subsequent cavernous sinus invasion. A meta-analysis comprised of 9,699 patients, by Brouwer et al., found that predisposing conditions consisting of metastatic or contiguous foci of infection were present in 86% of brain abscess cases. Among these conditions sinusitis was the second most common, encompassing 10% of study patients.

38 year old female G0P1 33 weeks gestation presented with a one week history of headache, blurry vision, low grade temperatures and neck stiffness. Laboratory analysis revealed leukocytosis and physical exam was remarkable for signs of meningeal irritation. Lumbar puncture was attempted in the ED but was unsuccessful. Differentials included bacterial versus viral meningitis. Imaging showed right sided sinusitis with occluded paranasal sinuses. MRI was positive for gyriform acute infarctions, right fronto-temporal acute infarcts, bilateral cavernous sinus invasion and severe segmental stenosis of the carotid siphons. In light of these findings the patient was started on merrem, daptomycin, amphotericin, caspofungin and the decision to admit to the ICU was made. She was then taken to the operating room for a functional endoscopic sinus surgery, right maxillary anostromy, right total ethmoidectomy, right frontal sinusotomy, right sphenoidectomy. The patient was then treated in the ICU with antibiotics, post surgically. She was also started on keppra for seizure prophylaxis. Her course was complicated by fetal distress requiring an emergent c-section. Progressive MRI showed sinous venous thrombosis which required anti coagulation with a heparin drip. Repeat imaging showed formation and progression of abscesses. Flagyl was added to her course; neurosurgical intervention included cerebral angiogram for concern of mycotic aneurysms but dysplatic MCA was found. Cultures grew out *Streptococcus angionosus* and *Streptococcus viridans*. She was treated with Rocephin and discharged to rehab.

The initial manifestations of brain abscess are usually nonspecific, often delaying diagnosis. Headache is the most common symptom, occurring in 69% of patients, followed by neck stiffness, fever and changes in mental status or neurologic deficits. Improvements in cranial imaging, neurosurgical technique and antimicrobial therapies have decreased fatality from 40% to 10% over the last 5 decades and increased the rate of full recovery to 70% of patients.

## THE ACROMEGALIC HEART

First Author: Rishi Handa, MD Second Author: James Cassuto, MD Third Author: Borislav Kheyson, MD Fourth Author: Arshi Handa, MD Fifth Author: Jacqueline Darcey, MD

Acromegaly is a rare clinical syndrome which occurs as a result of excessive secretion of growth hormone (GH) and insulin-like growth factor-1 (IGF-1). It can manifest as heart failure, which is one of the most severe complications and is associated with increased mortality. We present a patient whose respiratory symptoms prompted him to seek medical attention, possibly leading to a diagnosis that would have otherwise been missed.

A 47-year-old man with a history of an abnormal electrocardiogram (ECG) revealing “an enlarged heart” but with a normal stress test performed prior to an abdominal hernia repair 4 years ago presented to the emergency department with complaints of a non-productive cough for 1 week, recently worsening shortness of breath (SOB) on exertion, and right sided pleuritic chest discomfort. He denied exertional chest pain or pressure, however he did mention 2 pillow orthopnea and worsening SOB on exertion for approximately 1 year. He was clinically in heart failure. His chest x-ray was consistent with pulmonary vascular congestion. He also had a troponin-I rise of 0.090 (<0.045 ng/mL) and an ECG showing sinus tachycardia, a left bundle branch block, and left ventricular (LV) hypertrophy with repolarization abnormalities. An echocardiogram revealed eccentric LV hypertrophy with severe LV dilation, a LV ejection fraction of 10%, severe left atrial dilation, and grade 2 diastolic dysfunction. He underwent coronary angiography which was grossly negative. Despite the patient initially denying any progressive physical changes, his physical exam was revisited. He was noted to have very few teeth, which he attributed to his diagnosis of “soft teeth” approximately 10 years ago. On further questioning he mentioned he stopped wearing his wedding band because he was “tired of getting it resized.” The combination of his physical exam (including an enlarged jaw, a prominent forehead, and enlarged hands and feet) and non-ischemic heart failure presentation raised the suspicion of acromegaly. His IGF-1 was grossly abnormal. Magnetic Resonance Imaging of the brain showed a 1.2 x 1.2 cm lesion within the sella centrally, consistent with a macroadenoma.

This case demonstrates how the presentation of a patient with the novel diagnosis of decompensated heart failure lead to the identification of acromegaly based on physical exam cues. It is important to note, although rare (~3%), acromegaly can present with heart failure. IGF-1 and GH directly increase contractility of myocardial tissue and cause elongation of the cardiac action potential, resulting in cardiac hypertrophy. Heart failure in acromegaly is associated with higher mortality and therefore should be recognized early. Medical management to normalize cardiac function should be given utmost priority. Even though our patient presented with heart failure signs and symptoms, all patients suspected or diagnosed with acromegaly should ideally have a baseline cardiac work-up performed.

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TAKOTSUBO CARDIOMYOPATHY IN THE SETTING OF MYASTHENIC CRISIS

First Author: Mohamed Hassanein, MD Ola Adams, MD Stephen Tiekou, MD Mohammed Al-Jumayli, MD Bipinpreet Nagra, MD James Ware, MD Paul Stillwell, MD Daniel Goldsmith, MD

Background: Takotsubo cardiomyopathy (TTC) is recognized as transient left ventricular dysfunction following various hyperadrenergic states such as emotional or physical stressors. Here we describe a case of Takotsubo cardiomyopathy in a 65 years old man who was hospitalized for treatment of pneumonia and myasthenia gravis (MG) exacerbation.

Case report: The patient was a 65 years-old man with a history of hypertension, dyslipidemia and recently diagnosed myasthenia gravis, who presented to the hospital with complaints of two days of non-productive cough and worsening shortness of breath. At home the patient was on Mestinon 60 mg po TID and Mestinon 180 mg po daily at night for the myasthenia gravis. On admission vital signs were: BP 160/79. HR77. RR18. Temp98.7 O2 sat 100 % on 2 L NC. Patient was comfortable with an unremarkable physical exam. Laboratory workup was significant for WBC 20.4, bands 9%, segments 80% and troponin of 0.22 which was trending up in the following pattern 1.260 to 1.168 to 1.172. CT chest with contrast showed significant consolidation of the left lower lobe extending to the left hilum. Echocardiogram showed severe mid to distal anteroseptal hypo kinesis. Ejection fraction = 45-50%. The patient was admitted to the intensive care unit for severe pneumonia and NSTEMI. Rocephin and zithromax were initiated for treatment of community acquired pneumonia. Patient's vital capacity was closely monitored however it rapidly deteriorated over the following 18 hours as a result of exacerbation of his myasthenia gravis and the patient was intubated. Repeat echocardiogram 24 hours later showed severe hypokinesia of the mid to distal septum and apex. Ejection fraction was 30-35%; these features were suggestive for Takotsubo cardiomyopathy. Medical management for cardiomyopathy was initiated however the patient could not tolerate it as he developed bradycardia with the beta blocker and acute kidney injury that prevented the initiation of ACE inhibitors. Follow up echocardiogram on 22nd day of admission showed normal chamber size, normal wall thickness and ejection fraction= 50-55%. The patient responded well to the supportive management and he was discharged to sub-acute rehabilitation facility in stable condition.

Discussion: This case demonstrated that patients with MG crisis may be at potential risk of developing TTC and careful clinical and echocardiography monitoring is necessary while treating them. Takotsubo cardiomyopathy can resolve spontaneously if the MG crisis is appropriately treated in spite of the lack of goal-directed cardiomyopathy management.

## PLASMAPHERESIS AS AN EFFECTIVE MODALITY FOR TREATMENT OF HYPERTRIGLYCERIDEMIA INDUCED PANCREATITIS

First Author: Mohamed Hassanein, MD Nabil Mesiha, MD Majlinda Xhikola, MD Tania Calzada, MD Emily Chen, MD Daniel Goldsmith, MD.

**Background:** Hypertriglyceridemia is the third most common cause of acute pancreatitis, accounting for 1 to 4% of cases presenting to the hospital. A serum triglyceride (TG) level of approximately 1000 mg/dL or greater increases the risk of acute pancreatitis (AP), although some patients have developed AP at lower levels. The risk of developing acute pancreatitis is approximately 5 percent with TG >1000 mg/dL and 10 to 20 percent with TG >2000 mg/dL. The optimal approach to hypertriglyceridemia induced pancreatitis has not been established in with designed clinical trials.

**Case Report:** A 48 Hispanic female presented with epigastric pain, radiated to the back, associated with nausea and 3 episodes of non-bloody vomiting. There was no fever, change of bowel habits or urinary symptoms. Her past medical history was significant for alcohol induced pancreatitis 10 years prior. She took no medication, and last alcohol drink was a few weeks previously. There was a positive family history of dyslipidemia. On physical examination T 97.8, BP 140/87, Heart rate 108, RR 22 and BMI 32. Mucous membranes were dry. There was significant epigastric tenderness with no rebound tenderness. Laboratory findings: Lipase of 1682 Units/L, amylase 504 Units/L, Total Bilirubin 0.8 mg/dl, WBC 21.2 cells/mm<sup>3</sup>. Hb 12.2, lactic acid 3.7 mg/dl, Triglyceride 3321 mg/dL. The blood sample was extremely lipophilic and needed to be tested multiple times to obtain accurate results. Abdominal computed tomography showed severe inflammatory stranding abutting the pancreas and duodenum, compatible with pancreatitis. Ultrasound of the gall bladder showed no cholelithiasis or cholecystitis. Conservative treatment of AP was started including bowel rest, aggressive fluid resuscitation, and parenteral analgesia. The patient was still severely symptomatic so plasmapheresis was initiated. After 2 sessions of plasmapheresis, the patient improved significantly and she was able to start oral intake and required less parenteral analgesics. By the fifth day of hospitalization, TGs decreased to 347 mg/dL, and she was able to tolerate a full liquid diet and oral analgesics. Statin therapy and finofibrates were initiated. The pancreatitis resolved and she was discharged home on the seventh day of hospitalization.

**Discussion:** This case demonstrates plasmapheresis was an effective modality in treating HTG-induced acute pancreatitis. Medical therapy of severe hypertriglyceridemia is effective but may take time to achieve a result that allows resolution of AP. Intravenous insulin is frequently used for rapid correction of severe hypertriglyceridemia; however use of plasmapheresis in the acute setting is recognized as a viable alternative. Our experience suggests that plasmapheresis is rapid and effective treatment modality not only for the hypertriglyceridemia but for the AP as well. A large case control analysis or prospective clinical trial is warranted to establish the best approach to this challenging condition.

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GUILLAIN-BARRE SYNDROME FROM NOVEL IMMUNOTHERAPEUTIC AGENT-NIVOLUMAB

First Author: Aasems Jacob, MD Dileep C. Unnikrishnan, MD Shil Patel, MD Braghadheeswar Thyagarajan, MD

Nivolumab, an anti PD1 monoclonal antibody, is the first FDA approved immunotherapeutic agent for treating squamous cell lung carcinoma. Autoimmune demyelinating polyneuropathy also known as Guillain-Barre syndrome (GBS) can very rarely occur as a side effect of nivolumab (incidence rate undocumented). The presenting symptoms of the same are usually vague and often overlapping with other conditions making the diagnosis of GBS challenging.

A 68-year-old female presented to the hospital with 4 days history of fatigue, back pain and generalized weakness. She has poorly differentiated squamous cell carcinoma of the lung, which was initially treated with carboplatin and abraxane. She developed cancer metastasis to brain and was resected with gamma knife surgery. She was started on Nivolumab and had regression of the lung and brain lesions after 4 cycles, but the drug had to be discontinued secondary to pneumonitis that responded to high dose steroid therapy. During admission, her physical exam was significant for 3/5 power in lower extremities and 5/5 in upper extremities with intact sensations. The lower limb weakness progressed and she had bilateral tingling sensations, which led to suspicion of spinal metastasis. However, MRI of the spine was reported normal. She was monitored in the neuro-unit with neurological exam every four hours. After 36 hours of admission, she also developed bilateral arm weakness. Examination at the time revealed power of 1/5 in lower and 2/5 in upper extremities along with areflexia. The ascending paralysis picture and areflexia prompted us to make a diagnosis of GBS. The cerebrospinal fluid study showed albuminocytologic dissociation confirming the diagnosis. Hence, IV immunoglobulin therapy was initiated. The patient however developed respiratory muscle paralysis and required endotracheal intubation. She did not have any prior diarrhea or GBS provoking viral illness. No recent immunizations were documented and she was found to be negative for HIV, syphilis and Lyme infection. Sarcoidosis was also ruled out with normal ACE levels. Although the incidence rate of GBS with Nivolumab is not documented, careful review of literature revealed that checkpoint inhibitor monoclonal antibodies could rarely result in Guillain-barre syndrome even at sub-therapeutic doses or occasionally after discontinuation of the drug. Plasmapheresis was started since her condition worsened after interval improvement. Effective early intervention led to halting of progression of the GBS and gradual resolution of her symptoms.

This case points to the emerging side effects of the anti-cancer monoclonal antibodies and autoimmune demyelinating polyneuropathy should be considered in the differentials when patients on checkpoint inhibitor monoclonal antibodies present with vague symptoms like weakness or fatigue. Overlapping neurological symptoms can make the diagnosis challenging, but frequent and thorough neurological examination can help in early diagnosis and treatment of this otherwise fatal condition.

METHADONE AND SEVERE C. DIFF COLITIS: DRAWING FROM THE LOPERAMIDE EXPERIENCE

First Author: Elizabeth S John, MD Second: Ramy Sedhom, MD Third: Ranita Sharma, MD

Introduction: Clostridium difficile (C. diff), a toxin-producing anaerobic bacteria, is transmitted among humans through the fecal-oral route and results in symptoms ranging from mild diarrhea to fatal colitis. There are many risk factors for adverse outcomes - increasing age, concomitant antibiotic use, hypoalbuminemia, acute kidney injury, and anti-motility agents. Specifically, loperamide, a mu-receptor opioid agonist, is known to worsen C.diff infection (CDI), however this is the first case of CDI in the setting of methadone, a drug that works similarly to loperamide.

Case: A 59-year-old African American woman with a past medical history of diabetes, hypertension, and cerebrovascular accident without residual deficits, on chronic methadone for detoxification (100 mg), presented with a 3 day history of diffuse abdominal pain accompanied by multiple episodes of vomiting and non-bloody diarrhea. She denied recent hospitalization or antibiotic use. Physical examination revealed temperature 102.5 F, pulse 126, respiratory rate 18, blood pressure 105/80, SaO2 96% on room air. Abdominal exam was significant for diffuse tenderness and distention without rigidity. Notably, the patient demonstrated psychomotor retardation, and fell asleep while answering questions multiple times, which prior medical records attributed to her high daily dose of methadone 5 mg/5mL, 100 mg. Her labs were significant for a leukocytosis (14,600), elevated creatinine (2.1), and albumin less than 2.5. CT of the abdomen/pelvis revealed pancolitis. As the patient met sepsis criteria, fluid resuscitation and empiric antibiotics of IV Levofloxacin and Metronidazole were initiated. C. diff stool toxin assay came back positive, and repeat labs after fluid resuscitation were consistent with a severe CDI. Oral vancomycin, rectal vancomycin, and intravenous flagyl were started. Her methadone was continued to avoid withdrawal symptoms at a lower dose. The patient's hospital course included persistent vomiting, worsening abdominal distension, and increased leukocytosis. Bowel sounds were absent. KUB revealed a non-obstructive bowel gas pattern. A nasogastric tube was placed for decompression and surgery was consulted for severe complicated CDI with ileus. Initially, she was slow to respond to available treatment. A decision was made to quickly taper her methadone. Subsequently, both subjective and objective measures of infection improved over the course of several days. After successful taper of her methadone, from her outpatient regimen of 100 mg daily to 30 mg while hospitalized, her ileus, leukocytosis, and acute kidney injury resolved.

Discussion: To date, there have been no documented reports of severe CDI in the setting of methadone, a drug widely used in opioid dependent patients. Like loperamide, it is a long-acting mu-receptor agonist of smooth muscle organs, notably those in the gastrointestinal tract. The delayed transit induced by opioids may create a more conducive setting for bacterial epithelial penetration and local microbial proliferation, thus prolonging the toxic manifestations of bowel infections. The increased transit time may also cause slower and reduced delivery of the oral vancomycin to the colon. As CDI can lead to fatal complications, every measure to ensure proper and efficacious treatment should be taken, including the discontinuation or tapering of medications such as anti-motility agents, that can further exacerbate infection and lead to adverse patient events.

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LYME DISEASE: THE NEW GREAT IMITATOR

First Author: Nasir KHAN, MD Second Author: Anthony Carlino, MD

Lyme disease is a vector-borne multisystem illness caused by the spirochete *Borrelia burgdorferi sensu lato*. Its pathophysiology owes not only to the infection but also to the damage incurred by host immune responses leading to chronic inflammatory state as seen in neuroborreliosis. *B burgdorferi* is a highly neurotropic organism that causes a wide spectrum of central nervous system (CNS) manifestations. Cerebral vasculitis is a rare complication of Lyme disease (Estimated in 0.3% of neuroborreliosis cases). We describe a case of Lyme disease induced autoimmune CNS vasculitis.

A 30-year-old female landscaping consultant presented to Saint Barnabas Medical Center with a five day history of gradual onset generalized headache, mild photophobia and right upper extremity tingling. Initial routine laboratory tests were within normal limits except for an ESR of 68. Extensive head and neck imaging studies including computed tomography with and without contrast, magnetic resonance (MR) imaging and MR-angiography of the head and neck vessels were unrevealing. However, ELISA assay for Lyme disease was positive and the result was confirmed by Western blot analysis. She was started on Ceftriaxone but her symptoms actually worsened. CSF analysis showed pleocytosis but a low Antibody Index (AI); no organisms were seen on microscopy and PCR analysis of CSF for Lyme disease was negative. Autoimmune workup including ANA, rheumatoid factor, compliment levels and dsDNA was all within normal limits. Subsequently, a cerebral angiogram confirmed small and medium vessel CNS vasculitis. The patient was started on high dose prednisone. Her symptoms responded rapidly to steroids and she was discharged home asymptomatic on ceftriaxone and steroid taper.

The putative mechanisms for CNS injury in Lyme disease other than the infection itself include vasculitis, neurocytotoxicity and autoimmune damage via molecular mimicry involving both innate immune responses through Toll-Like Receptors as well as T and B cell mediated specific responses much like in case of *Treponema pallidum*. Manifestations can range from clinically silent disease to highly unusual presentations, mimicking many primary and secondary neurological conditions. Indeed, Lyme disease can masquerade as CNS vasculitis and hence, it is yet another great imitator. A low threshold for clinical suspicion is required for patients presenting with atypical neurological clinical features.

Vasculitis may complicate the clinical course of Lyme borreliosis. Whether elicited directly by the spirochete or by secondary autoimmune mechanisms, vasculitis occurs in association with localized or disseminated organ damage. It should be suspected in the right patient population especially when antibiotic treatment does not improve the clinical course in Lyme borreliosis. A high degree clinical suspicion is required and we recommend prompt consideration of immunosuppression therapy in established neuroborreliosis that fails to respond to antibiotics.

## FALSE POSITIVE HIV RESULT AND LOW CD4 IN BABESIOSIS

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**Background:** Babesiosis is a disease, prevalent in the Northeast during warm months, May through September. The majority of those infected with *Babesia microti* are asymptomatic. However, the elderly, immunocompromised and asplenic patients can develop life-threatening complications including intravascular hemolysis. Patients with HIV are known to have high morbidity and mortality, as CD4<sup>+</sup> T helper cells are the subpopulation chiefly responsible for protection against *Babesia microti*. Therefore, all patients with symptomatic babesiosis are usually screened for underlying HIV. EIA (enzyme immunoassay) is the screening test with high sensitivity. Some infections, such as malaria, and autoimmune diseases, such as systemic lupus erythematosus, are known to cause false positive results on HIV screening. We report two consecutive cases of symptomatic babesiosis with false positive EIA.

**Case Presentation:** Case #1. A 29 years male, who works as a landscaper, presented with one week history of fever, chills, night sweats, myalgia, headache and dizziness. On admission temperature was 101,5 and vital signs were otherwise stable. Hb was normal and wbc was 4.7 with 16% bandemia. His peripheral smear confirmed babesiosis parasitemia of 1.1%. Atovaquone and azithromycin were started and we observed steady improvement of his symptoms. HIV screening was performed on admission and EIA came back positive. On further questioning, we identified no risks factors for HIV. A western blot confirmation was pending and CD4 count was ordered. Western blot was negative, but absolute CD4 count was 20 and Cd4/Cd8 ratio was 0,08 At his 2 week outpatient visit, he was asymptomatic. The CD4 count and CD4/CD8 ratio normalized.

Case #2. An afebrile 62 years old male presented with one week history of generalized fatigue and dizziness. He was diagnosed with severe hemolytic anemia secondary to babesiosis with 11.5% parasitemia. He was started on atovaquone and azithromycin. HIV EIA was positive with total CD4 suppressed at 412. The CD4/CD8 ratio was normal. The western blot was negative. On follow up visit in the outpatient clinic patient the patient was asymptomatic and his CD4 normalized.

**Discussion:** We are well aware that some infectious and autoimmune diseases are associated with false-positive EIA results for HIV. Malaria, for example, may cause this false positive result due to hypergammaglobulinemia from immunological stimulation. We postulate that babesiosis, similar to malaria, is another condition where we need to be aware of the possibility of false positive EIA result for HIV. Also, our cases demonstrated that CD4 count should not be used as a surrogate test for HIV diagnosis, especially in patients with acute immunologic response to infection.

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AN UNUSUAL CAUSE OF LYMPHADENOPATHY IN SICKLE CELL DISEASE

First Author: Meyreles-Chaljub Gustavo, MD Guragai Nirmal, MD Sonia FNU, MD Cholankeril Michelle, MD.

**INTRODUCTION** Sickle Cell Disease (SCD) is due to a point mutation in the  $\beta$ -globin chain resulting in hemoglobin S. Red blood cell transfusions (RCT) are the mainstay in treatment of acute and chronic complications of SCD. Secondary hemosiderosis (SH) is an uncommon, yet possible complication in these patients. The inflammatory state in SCD results in the release of Interleukin-1 and Interleukin-6, decreasing iron absorption and enhancing iron retention in the reticulo-endothelial system through hepcidin induction. SH in SCD is commonly found in the liver, joints, skin, and kidneys; rarely involves the cardiac or endocrine systems.

**CASE REPORT** We present the case of a 25 year-old African American female with past medical history of SCD and chronic deep vein thrombosis (DVT) of the left upper extremity (UE). Due to her severe vaso-occlusive crisis since childhood, she has chronically received RCT. She presented to our hospital complaining of diffuse joint pain and swollen left UE for two days. She denied trauma, fever, chest pain or respiratory distress. On examination, conjunctival pallor and diffuse joint tenderness was observed. Initial blood work showed hemoglobin of 7.4G/dL and reticulocyte count of 7%. She was started on intravenous fluids, hydromorphone, folic acid and serial hemoglobin and reticulocyte count monitoring. On day five of admission, physical examination revealed non-tender, mobile left supraclavicular mass not present initially. Computerized tomography of the neck with contrast revealed cervical, bilateral supraclavicular, hilar and mediastinal lymphadenopathy. Due to possible sarcoidosis in the setting of generalized lymphadenopathy, angiotensin converting enzyme level was sent and resulted normal. Lastly, excisional biopsy of the left supraclavicular node reported lymph node sinus and follicular centers to be populated with pigmented macrophages accompanied by plasma cells with Prussian blue stain positive for iron. Subsequent serum ferritin level was found to be 1,487ng/ml.

**DISCUSSION** Chronic RCT reduces stroke incidence and recurrence in SCD patients as well as hemolytic crisis. However, complications like SH, are sometimes unavoidable. We present an unusual case of iron deposition in lymph nodes secondary to transfusional support. This has not yet been reported in literature, making our case unique. We illustrate the importance of understanding the indications of RCT in SCD patients in order to avoid SH and its many life-threatening complications. High clinical suspicion is required to identify patients at risk, as serum ferritin offers poor correlation to actual end-organ damage by iron deposition. Early initiation of therapy with chelating agents is known to prolong survival and prevent complications in those receiving chronic RCT.

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SEROTONIN SYNDROME: AN UNDER RECOGNIZED LIFE-THREATENING DRUG INTERACTION

First Author: Zainab A Mirza, MD Naseer Ahmed MD, Mohamed Elswaf MD

Objectives: Recognize and review management of serotonin syndrome.

**Case:** A 95 year old nursing home resident with a history of dementia, anxiety, and recurrent urinary tract infections was chronically taking duloxetine, quetiapine, and clonazepam. She developed another urinary tract infection with vancomycin-resistant enterococcus and was started on linezolid. Over the next 6 days, she developed progressive diffuse tremors, restlessness, and increased confusion and was sent to the ER. VITALS: BP 109/94 HR 111 Rectal Temp 101°F. She was given lorazepam then haloperidol without improvement and her temperature increased to 103.4°F. Her worsening condition prompted admission to the ICU. EXAM: generalized tremors, dry mucosa, rigid extremities with hyperreflexia, inducible knee and ankle clonus, and down going Babinski bilaterally. Her blood work showed Na153mmol/l, lactic acid 6.6mmol/l, acute transaminitis AST 210 IU/L and ALT 127 IU/L, and acute renal failure with rhabdomyolysis. Concern for serotonin syndrome (SS) was raised with the possibility of underlying sepsis. She received broad spectrum antibiotic coverage with vancomycin and imipenem. A nasogastric tube was placed and she was given several doses of cyproheptadine, but with minimal response. Her condition worsened requiring to be intubated and paralyzed. Her renal failure continued to worsen despite aggressive fluid resuscitation. However, the family decided against dialysis and chose to withdraw care.

**Discussion**This patient developed serotonin syndrome after initiation of linezolid therapy due to an inadvertent drug interaction with duloxetine. The incidence of SS in patients on linezolid and serotonergic agents is reported as 0.24-4.0%. Other drugs implicated are monoamine oxidase inhibitors, tricyclic antidepressants, selective serotonin reuptake inhibitors, and serotonin-norepinephrine reuptake inhibitors causing excessive serotonin activation. The syndrome is a clinical diagnosis and requires a high level of suspicion as it can potentially be rapidly fatal with multi-organ failure. It is characterized by autonomic dysfunction, hyperpyrexia, neuromuscular instability, and mental status changes. Severe episodes can produce rhabdomyolysis secondary to excessive muscle activity leading to acute renal failure. Initial management includes controlling hyperthermia with external cooling, hydration, and benzodiazepines. Cyproheptadine, a non specific serotonin antagonist can be used as an adjunct however currently it is unknown if it modifies patient outcome. Patients with a temperature higher than 41C should be intubated. There is a limited role for traditional antipyretics, as the mechanism of the fever is due to muscle tone rather than central thermoregulation. Further management includes total sedation to control muscle rigidity while the drugs wear off. The epidemiology is difficult as many physicians are unaware of serotonin syndrome as a clinical diagnosis. Hence, a greater awareness of drug combinations and attention to medication history is needed with a high index of suspicion to prevent and identify this syndrome.

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RESPIRATORY FAILURE AFTER SEPTOPLASTY AS A CONSEQUENCE OF OBSTRUCTIVE SLEEP APNEA

First Author: Padma Raghavan Pillai, MD, MBBS Second Author: Kristin Fless, MD

**Introduction** Post-obstructive pulmonary edema is a well-recognized and potentially life threatening complication of acute airway obstruction. It is essential to recognize this entity which is more frequently seen in patients with obstructive sleep apnea (OSA) as there is good outcome associated with proper treatment.

**Case** A 45 year old male was admitted from an outpatient surgical center after septoplasty and turbinectomy. He developed respiratory failure and flash pulmonary edema post extubation and was re-intubated. There was some report of upper airway bloody secretions on re-intubation. His medical history was significant for obesity with a BMI of 40, Obstructive sleep apnea (OSA) on Continuous positive airway pressure (CPAP) of 14 cm H<sub>2</sub>O at home. There was no significant family history. He did not smoke or drink alcohol.

Examination showed a responsive, obese male, comfortable on propofol drip and saturating 100% on PRVC with 60% FiO<sub>2</sub>, 650 ml TV and PEEP 8cmH<sub>2</sub>O. He had bilateral nasal packing which was dry and intact. There were bilateral coarse rhonchi on auscultation. Chest radiograph showed bilateral pulmonary opacities with consolidative changes in the right upper lobe. He was admitted to ICU for management of non-cardiogenic pulmonary edema and was continued on mechanical ventilation. He was extubated the next day and was placed on face tent during the day and CPAP at night. Examination revealed Mallampati class 3 pharynx and neck circumference of 19.5 cm. He was discharged two days later.

**Discussion** Non-cardiogenic pulmonary edema is a common cause of post-operative respiratory failure for OSA patients. It is estimated that less than 20% of patients on positive airway pressure (PAP) for OSA have PAP applied in the perioperative period [1]. The mechanisms for upper airway obstruction in these patients are many and include loss of cortical input to pharyngeal muscles with anesthesia, topical nasal anesthesia which causes loss of mechano-receptor reflexes in the pharynx that are usually activated by increased nasal resistance, an increase in pharyngeal edema during recumbent surgery, and loss of functional residual capacity of the lung and tracheal tug during anesthesia induction [3]. In this patient, PAP use with nasal packing was challenging and alternative mask interfaces could have been considered to administer PAP.

Post obstructive pulmonary edema is a non-cardiogenic edema caused by massive negative intrathoracic pressure developed against a closed upper airway (Muller maneuver) [2]. Patients with sleep apnea, chronic airway obstruction, head and neck surgeries have an increased risk. Large negative intrathoracic pressure increases venous return thus increasing pulmonary capillary pressure in combination with decreased left ventricular compliance secondary to septal bounce and negative inotropic effect of the intrathoracic pressure.

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ARE ALL LARGE TONGUES FROM AL AMYLOIDOSIS?

First Author: ROBERTO H RAMIREZ, MD, GLENMORE LASAM MD, GINA LACAPRA MD

63 years old African American female with ESRD, congestive heart failure, poorly controlled hypertension was admitted for increasing shortness of breath. Patient had BP 208/112, JVD, crackles bilateral bases, and peripheral edema. EKG: ST elevations(V3, V4) BNP: >5000 Patient was diagnosed with STEMI, CHF and hypertensive crisis and treated accordingly. She developed upward gaze, confusion and jaw trismus which progressed to nonresponsiveness to verbal stimuli. EEG revealed nonconvulsive status refractory to all antiepileptics. Mental status improved led to responsiveness only to painful stimuli. Macroglossia was then noticed. Echocardiogram showed thickened left ventricle/dense myocardial speckling suggestive of amyloidosis. Protein electrophoresis and immunofixation were normal, making AA and AL less likely. Genetic testing for TTR was not sent. She became more alert, but then developed a complication from her tracheostomy and passed away. Autopsy was declined.

Discussion: Amyloidosis frequently goes unrecognized for long periods of time due to its wide variety of nonspecific clinical manifestations and its associations with different diseases. Macroglossia is mostly seen in AL amyloidosis, although there are few reported cases of macroglossia present in TTR amyloidosis. Cardiac involvement is mostly seen in AL and TTR amyloidosis hereditary type (Val122Ile allele). 50% of AL patients have clinically significant cardiac involvement but there is limited data on TTR. The most common cardiac manifestation is heart failure in both AL and TTR, atrial fibrillation, poor ventricular diastolic function, and poor atrial mechanical function. AL is more severe, more prone to develop atrial fibrillation and tends to present later in life. The TTR variant has a prevalence of 3.5% in the African-American and African-Caribbean population. 25% of cardiac amyloidosis in African-American and African-Caribbean present with this allele mutation. Echocardiography is the initial noninvasive test of choice although hypertensive cardiomyopathy can appear similar. Most common findings in amyloidosis are increase in left ventricular wall thickness and diastolic dysfunction. Increased echogenicity, described as “sparkling” or “granular.” Definitive diagnosis is amyloid deposits by Congo red stain on an endomyocardial biopsy or another organ. The genetic test for TTR is helpful in differentiating the two entities. Loop diuretics are a mainstay for AL and TTR cardiac amyloidosis. ACE inhibitors, calcium channel blockers and beta blockers have not shown proven benefit. In AL amyloidosis therapy involves chemotherapy and/or autologous stem cell transplantation (ASCT). In TTR amyloidosis treatment is liver transplantation.

Recommendation: Cardiac amyloidosis should be considered in any adult with unexplained heart failure and an echocardiogram showing increased wall thickness. TTR amyloidosis should be suspected in any patient of African-American/African-Caribbean descent over the age of 50 who has unexplained left wall thickening on echocardiogram.

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MISDIAGNOSIS: THE CLOT THICKENS

First Author: Amee Sodha MD Second Author: Katherine Chiapaikeo-Poco DO Fellow: David Shiu DO Attendings: Chengqun Shao MD, Christina Migliore MD

Introduction: In common practice, certain algorithms are utilized to risk-stratify patients and to avoid the overuse of diagnostic testing. However, physicians should understand the limits to these tools and that concomitant diagnoses do exist and may disguise a more severe condition if not considered. In the case below, we introduce and discuss an interesting case of symptomatic anemia masking a common, but acutely grave and life-threatening diagnosis.

Case: This is a 38-year-old African-American female with history of pulmonary embolism (PE), uterine fibroids and menorrhagia who presented to our emergency department with acute-onset shortness of breath. Pertinent history and hospital course includes a two year history of progressive fatigue and dyspnea on exertion which was initially attributed to severe anemia secondary to menorrhagia and anxiety. At the time, a differential of PE was deemed 'unlikely' when risk-stratified by her modified Wells score of <4. More convincingly, her PE rule-out criteria (PERC) were completely satisfied, therefore a D-dimer was never pursued. Two years later, the patient presented with acutely worsened dyspnea which prompted a CTA, showing multiple, bilateral PE. She was started on enoxaparin, which she stopped prematurely because of uncontrolled menorrhagia. Due to premature cessation of anticoagulation, she presented four months later in our emergency department, as stated at the beginning, with similar symptoms, with a repeat CTA showing existing bilateral PE and new right heart strain. A subsequent echocardiogram estimated a severely high pulmonary artery pressure of >95 mmHg and severe right ventricular dilatation. Hypercoagulable studies were negative, with no evidence of underlying malignancy or autoimmune disease, so her PE was classified as unprovoked. Given the diagnosis of probable severe pulmonary hypertension (PH) classified as WHO Group 4, she was deemed too unstable for gynecological intervention and was started on oral progesterone to control her bleeding. Her condition would necessitate further investigation and treatment, including potential life-long anticoagulation, right heart catheterization and medical management for her PH.

Discussion: This case underscores the shortcomings of strict adherence to algorithms and tools with high sensitivity. Given a low pre-test probability based on her Wells Score and PERC, therefore low suspicion, a life-threatening disease was uncovered late in her clinical course. Unfortunately, adherence to evidence-based medicine and an algorithmic approach can mislead us to attribute the symptomatology to other processes, such as anemia and anxiety as in this case. It is important to have a high index of clinical suspicion based on other comorbidities, which likely would have helped our patient receive appropriate treatment sooner.

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LATE NEUROSYPHILIS PRESENTING AS MYELOPATHY AND SPINAL CORD MASS

Karyna M Neyra MD, Isaac K Soliman MD, Nilaya Bhawsar DO, Vinit Gupta MD

**Introduction:** Syphilis is referred to as “the great imitator” because it can affect any organ system and present in unusual ways, requiring a high level of suspicion to diagnose it. We present a case of neurosyphilis which presented as myelopathy secondary to what was initially suspected to be a spinal cord tumor.

**Case Description:** A 71-year-old male presented with complaints of right foot drop, gait instability, and paresthesia of the thorax, abdomen, and bilateral lower limbs worsening over several months. Physical examination demonstrated decreased strength in the right lower extremity, hyperreflexia, and positive Babinski’s sign. Proprioception was preserved bilaterally. MRI of the spine showed an intramedullary enhancing lesion at the level of T6-T7 in the right hemicord measuring about 10 mm in maximum dimension with extensive surrounding cord edema. CSF analysis was remarkable for 125 white cells per uL with lymphocyte predominance and 24 oligoclonal bands. Other scans, including MRI of the brain and CT scan of the chest, abdomen, and pelvis were unremarkable. The patient was treated with systemic steroids (dexamethasone) and physical therapy, which resulted in modest improvement of his symptoms. A subsequent MRI performed four weeks later showed a decrease in the size of the lesion and resolution of the cord edema. The patient later divulged that he had been diagnosed with syphilis roughly 40 years prior. Further work-up revealed reactive serum RPR (1:256) and CSF VDRL (1:32), confirming the diagnosis of myelitis secondary to neurosyphilis.

**Discussion:** Symptomatic syphilitic myelitis is a rare presentation of neurosyphilis. Diagnostic evaluation of this patient was complicated by the atypical symptoms for neurosyphilis, unusual findings on MRI, ambiguous CSF analysis, and positive response to steroid therapy. This case illustrates the importance of considering syphilis in the differential diagnosis of enhancing spinal cord lesions.

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HEAD OVER HEART: A CLINICAL VIGNETTE DISCUSSING ACROMEGALY AND CARDIAC DYSFUNCTION

First Author: Fadwa Sumrein, DO, Neha Verma MD, Sharon Bushi MD, Ronak Patel MD,

Acromegaly is a rare disease with insidious onset and progression, usually presenting with development of coarse features, increase in hand size and arthralgia. Due to the supra-physiological levels of growth hormones (GH), patients present with clinical features including cardiac hypertrophy, arrhythmias, sleep apnea, diabetes mellitus, goiter and gastric tract polyps.

In this report, we present a case of acromegaly in a middle aged man with severe systolic heart failure at the time of diagnosis. The patient is a 47 year old male with a history of hypertension and asthma. During a pre-operative work up for hernia repair four years prior to this admission he was informed that he had an “enlarged heart”. Stress test at the time was reportedly normal.. During the current admission, he presented with symptoms of progressively worsening shortness of breath and pleuritic right sided chest pain. The echocardiogram showed severe left ventricular dilation and global dysfunction with an ejection fraction of 10-15%. A cardiac catheterization revealed no evidence of coronary disease. Lab abnormalities also revealed elevated HbA1c indicating newly diagnosed diabetes. In gathering additional history, patient reported that he had an increase in head size, ring size and enlarging feet for approximately 10-15 years. After confirming an elevated level of Insulin-like growth factor(IGF) of 857 ng/mL, the most likely etiology of his systolic heart failure was acromegaly. The patient was subsequently found to have a macroadenoma of the pituitary on an MRI study. While surgery is the first line treatment for acromegaly, due to his severe non ischemic cardiomyopathy the patient was treated with somatostatin receptor ligands and referred for outpatient follow-up.

The most common cause of GH excess is a pituitary macroadenoma. Due to the slow development of the disease, the diagnosis is usually delayed 7-10 years and is commonly diagnosed after 50 years of age. Due to the increase in IGF-1 and GH, systemic manifestations include cardiac and reproductive disorders. Significant factors predictive of poor outcome are high growth hormone levels and cardiovascular disease. More than half (60%) of the patients with acromegaly die from cardiovascular disease complications. Nearly all patients have bilateral ventricular hypertrophy at the time of diagnosis, some with diastolic heart failure. Dilated cardiomyopathy with systolic dysfunction is a very rare finding and no studies are available for prognostication for these patients. Creating a patient database for these rare findings may assist in providing additional data to refer to for better understanding of acromegaly and its complications.

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## THE RISE OF CRYPTOCOCCUS LAURENTII: A CASE SERIES

First Author: Omer H Tarar Komal Motwani, Natasha Lawrence, Salman Arif, Mustafa Hasan, Adriana Grigoriu.

**INTRODUCTION:** Cryptococcus Laurentii is one of the several nonneoformans Cryptococcus that has rarely been associated with human infection. It was previously considered saprophyte and thought to be non-pathogenic to humans but in the last few years there has been increased incidence of infection due to Cryptococcus laurentii especially in the immunocompromised host. There have been limited case reports on the organism causing fungaemia, meningitis, keratitis, endophthalmitis and lung abscess. We report a series of 7 cases of Cryptococcus Laurentii infection including one in blood, one in the bile and five in the urine.

**CASE DESCRIPTION:** All the 7 cases were from our institution that presented within the past year and were found to have cultures positive for C. Laurentii. Five cases had the organism in the urine, one in blood and one in bile. There were 4 males and 3 females with their ages ranging from 27 to 87 years. 1 patient had HIV, 1 had history of renal transplant and immunosuppressant use, 1 had cholangiocarcinoma, 1 had cervical cancer with prior fluconazole use, 1 had partially treated prostate cancer and 2 had history of chronic steroid use. Out of 7 cases, 4 presented with abdominal pain and fever, 1 had hypotension with high grade fever and 2 were asymptomatic. The patient with fungaemia was treated with Amphotericin B and other 6 were administered Fluconazole. Out of the 7 patients, 1 passed away during treatment due to multiple other co morbidities while the rest responded to therapy with clinical improvement and negative subsequent cultures.

**DISCUSSION:** Non neoformans Cryptococcus species have generally been regarded as nonpathogenic saprophytes but in the past few years, there have been increasing reports of invasive infections caused by this organism. This increase is thought to be related to increase in number of immunocompromised patients over the years. Majority of these cases have been attributed to Cryptococcus Laurentii and Cryptococcus Albidus infections. Risk factors associated with C. laurentii infection are invasive devices, prior steroid exposure, prior immunosuppressant exposure, prior azole exposure, low CD4 count, exposure to pigeon excreta and neutropenia. Most of our patients had at least one of these risk factors present. There are no set guidelines for the treatment of this infection but mostly Amphotericin B or Fluconazole have been used based on the severity and nature of the infection.

**CONCLUSION:** Cryptococcus Laurentii is a rare infection that has been reported with increasing frequency recently especially in the immunocompromised patients. It can cause various invasive infections and should be considered in patients with risk factors as early identification and treatment can decrease the associated morbidity and mortality.

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**SUCCESSFUL USE OF EXTRACORPOREAL MEMBRANE OXYGENATION FOR SILICONE EMBOLISM SYNDROME AND PULMONARY HEMORRHAGE**

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17 y/o male-to-female transgender patient presented with progressive dyspnea, non-productive cough and pleuritic chest pain 2 days after injecting silicone for breast augmentation. On admission, the patient was tachycardic, tachypneic and required supplemental oxygen & was noted to have decreased breath sounds, bilateral breast tenderness and ecchymotic lesions in the upper quadrant of left breast. Laboratory results revealed leukocytosis of 21,000 cells/ $\mu$ L & D-dimer of 1.29  $\mu$ g/L. Initial chest radiograph(CXR) showed diffuse bilateral hazy airspace opacities with interstitial thickening. A pulmonary CT angiography was negative for acute emboli but revealed patchy ground glass opacities throughout both lungs. On the night of admission, the patient had several coughing paroxysms with oxygen saturation (O<sub>2</sub>sat) of 85 to 87% & multiple episodes of hemoptysis. 48 hours later, respiratory status worsened, initially requiring high flow oxygen, subsequently bi-level positive airway pressure and was given 125mg of intravenous methylprednisolone, with minimal improvement & was emergently intubated. Post-intubation the O<sub>2</sub>sat remained at 80% with FiO<sub>2</sub> of 100% and positive end-expiratory pressure (PEEP) of 16cmH<sub>2</sub>O, after which nitric oxide (NO) at 20ppm was added, but O<sub>2</sub>sat remained <90%. Repeat CXR showed worsening bilateral infiltrates. 5 hours post-intubation and NO administration, hypoxemia and acidosis worsened. The team decided to place the patient on Venovenous(V-V) ECMO support & continue with pressure-controlled ventilation. Bronchoscopy done post-ECMO showed fresh blood in both bronchi with hemorrhagic fluid return & cytology revealed rare clusters of reactive bronchial epithelial cells and alveolar macrophages. Patient was kept on IV steroids while unfractionated heparin (UFH) was used while on V-V ECMO. The patient's hemoptysis, blood gases, chest radiographs and overall clinical status progressively improved & was successfully weaned off ECMO on day 5 and then extubated on day 6.

Non-professional injection of silicone for breast augmentation or in other cosmetic procedures is a growing problem in the USA and with this comes a rising incidence of complications, especially in the pulmonary system. Silicone embolism syndrome (SES) is one of the life threatening complication which involves formation of multiple pulmonary emboli of silicone fluid. SES and fat embolism are known to have similar pathophysiology, where uptake of silicone or fat by alveolar macrophages results in modulation of pulmonary immunoregulatory mechanisms that promote an exaggerated inflammatory response, leading to ARDS or pneumonitis. Treatment of SES is largely supportive with high dose steroid administration and supplemental oxygen being the mainstay of therapy; unfortunately, few patients develop refractory respiratory failure from massive pulmonary hemorrhage that may require mechanical ventilation or ECMO. V-V ECMO was used in this patient as rescue therapy to maintain life, bridge to recovery, reduce barotrauma and allow pulmonary rest.

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A TALE OF TWO VASCULITIDES: BIOPSY-PROVEN GIANT CELL ARTERITIS FOLLOWED BY THE INDEPENDENT DEVELOPMENT OF RENAL-LIMITED MICROSCOPIC POLYANGIITIS

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**Learning Objective:** (1) Recognize the clinical presentation of large and small vessel vasculitides and (2) appreciate the rarity of their occurrence in the same patient.

**Case summary:** An 84 year-old Caucasian woman with a history of osteoporosis, atrial fibrillation and osteoarthritis presented with bilateral temporomandibular joint pain radiating to the neck and shoulders, as well as bilateral scotomas. The patient was treated with prednisone 60 mg daily and a biopsy-proven diagnosis of giant cell arteritis (GCA) was subsequently established. Prednisone treatment was gradually tapered over the course of four years, down to 1 mg daily. She was treated with zoledronic acid for steroid-induced osteoporosis. Several months later, she was noted to have a gradual worsening of her renal function (serum creatinine = 1.64 mg/dL). Shortly thereafter, the patient sustained a right intratrochanteric hip fracture following a fall and was found to have acute kidney injury (serum creatinine = 3.47 mg/dL). Urinalysis was significant for large hematuria and a urine protein-to-creatinine ratio of 1.63 mg/g creatinine. Serologies were notable for the presence of a perinuclear anti-neutrophil cytoplasmic antibody (p-ANCA) at a titer of 1:320 and antibodies to myeloperoxidase (MPO). Renal biopsy demonstrated a pauci-immune focal crescentic glomerulonephritis. Extensive review of systems, physical exam and diagnostic studies revealed no evidence of extra-renal disease at the time, and the patient was diagnosed with renal-limited microscopic polyangiitis (MPA). She underwent intramedullary nail fixation for her hip fracture and was restarted on prednisone 60 mg daily. Despite counseling, she subsequently refused definitive treatment with either rituximab or cyclophosphamide for MPA due to concern for toxicity. Her prednisone dose was tapered down to 10 mg daily at her rehabilitation facility over the course of two months. Her renal function stabilized (serum creatinine = 1.82 mg/dL) and moderate hematuria persisted.

**Conclusion:** Herein we present a patient who was diagnosed with GCA, which was confirmed by temporal artery biopsy. GCA is a large-vessel granulomatous vasculitis predominantly affecting the cranial arteries. Other organs including the lung, abdominal viscera and skin may be involved, but GCA typically spares the kidneys. She was treated accordingly with slowly tapering steroid and demonstrated good disease control. Four years later she developed renal-limited MPA, which is an unrelated small-vessel ANCA-associated vasculitis. Both GCA and MPA have an epidemiological predilection for older adults of European descent. Two previously published case reports from Japan demonstrated the concurrent development of GCA and MPA. To our knowledge, we presently report the first case describing the consecutive, independent development of MPA following treatment for GCA.

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BALANCING PATIENT SAFETY, PATIENT AUTONOMY, AND RESOURCE UTILIZATION - A DIALYSIS PATIENT'S CHOICES CREATE AN ETHICAL DILEMMA.

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Case report. A 40 year-old hemodialysis-dependent woman presented to the hospital for the third time in two weeks needing urgent dialysis after she could not keep an outpatient dialysis chair due to repeated interpersonal conflicts with dialysis clinic staff. A psychiatrist evaluated her, and found her to have personality traits affecting her interactions at dialysis centers. During her first two admissions, our patient had a prolonged stay because outpatient dialysis could not be secured by the inpatient team, and she left the hospital against medical advice when she was medically stable. This hospitalization she stated she planned to leave AMA again when necessary.

A care conference with the Ethics Committee, hospital medicine, risk management, nephrology, and nursing from the hospital dialysis unit was held to discuss her case. The ethicist noted that this patient was decisional and could make choices about her care, including a choice to modify her behavior to be compliant with outpatient dialysis. Her choice not to didn't obligate the medicine team to continue her hospitalization once she was otherwise safe for discharge. Rather, their duty was to inform the patient of the risks of not receiving scheduled dialysis and that UNMH cannot provide scheduled dialysis.

The patient was educated about the conference findings and instructed to return to an emergency room to be evaluated for emergent dialysis if she felt symptoms reflecting this, but informed that she wouldn't be admitted unless she met criteria for emergent dialysis. Though displeased with not being able to schedule her return, the patient expressed understanding. She was discharged the following day.

Discussion. There is significant risk and cost associated with hospitalization. Patient autonomy should be preserved in the process of medical decision making, but autonomy carries with it responsibility. Informed, competent patients may make choices. But rational use of resources as well as the ethical principles of nonmaleficence and beneficence may constrain those choices. There is not a great deal of literature on options for inpatients who have lost access to outpatient dialysis because they have been difficult or disruptive. The Renal Physicians Association has published a clinical practice guideline on the initiation of and withdrawal from dialysis to help guide provider decision-making. In 1982, the President's Commission for the Study of Ethical Problems in Medicine and Biomedical and Behavioral Research stated that "informed consent does not mean that patients can insist upon anything they want," and went on to state that, while patients had the right to refuse some or all medical treatment, they do not have the right to particular treatments. "Difficult or disruptive patient" are not uncommon. This will continue to be a problem in the world of outpatient dialysis presenting ethical challenges for practicing internists.

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WITH A HEAVY HEART

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**Introduction:** Cardiac mural masses are classified as myomas, sarcomas, or other metastatic lesions. About 9-27% of patients with advanced lymphoma have cardiac involvement. These masses seed from the vena cava and invade the pericardium but rarely penetrate the myocardium. The incidence of right sided cardiac lymphomas is 8 times greater than those in the left. Right sided masses that obstruct the outflow tract can lead to dyspnea, lower extremity edema and ascites.

**Case Presentation:** We present a 49-year-old male with past medical history of diabetes mellitus type II and polysubstance abuse with two weeks of intermittent, substernal chest pain, shortness of breath, lower extremity swelling, abdominal pain, fevers, chills, and night sweats. On presentation, he was afebrile but tachycardic with blood pressure ranged = 105-121/73-88. He required 5 L of oxygen to maintain adequate oxygen saturation. Physical exam revealed a generally ill-appearing male with wet bilateral lung crackles and profound lower extremity edema. Laboratory results were significant for leukocytosis ( $16.3 \times 10^3$ ) favoring neutrophilia, elevated troponin (8.69 ng/mL) and pro-BNP (10,359PG/ML). EKG showed T wave inversion in the anterioseptal leads. Transthoracic echocardiogram revealed left ventricular ejection fraction >70% with a large heterogeneous mass in the right ventricle. The mass extended through the myocardium, invaded the pericardial space, and entered the right atrium. Severe mechanical tricuspid stenosis was also noted. CT chest, abdomen, and pelvis revealed several pulmonary emboli and large bilateral renal masses. Biopsy of the renal mass diagnosed diffuse large B cell lymphoma, stage IVe.

Medical oncology was consulted and determined that chemotherapy treatment carried a 90% mortality rate due to reduction of tumor burden causing right ventricular wall rupture, massive pulmonary embolus, or fatal arrhythmias. However, if the patient elected to forgo treatment, prognosis would be equally fatal. The patient ultimately opted for chemotherapy, which remarkably decreased the right ventricular mass and improved hemodynamic function.

**Discussion:** This case demonstrates a rare cause of heart failure secondary to right ventricular lymphomatous mass with near complete resolution of symptoms following chemotherapy. It also reminds providers of the importance of the shared decision making model. When presented with a situation where both pursuing and forgoing medical treatment can be fatal to a patient, shared decision making should be encouraged.

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TO STRIP OR NOT TO STRIP? THAT IS THE QUESTION.

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**Introduction:** Differentiating between constrictive pericarditis and restrictive cardiomyopathy presents a unique diagnostic dilemma in medicine. Both processes can present with identical symptoms and diagnostic data often overlap. The practitioner must differentiate between these two hemodynamic profiles by using objective data and correlating those with the clinical presentation. The following case highlights the difficulty in navigating patient management when diagnostic data fails to clearly differentiate between two very similar disease processes.

**Case Presentation:** A 69 year old Hispanic male with known history of tuberculous pericarditis, status post pericardial window, was transferred from an outside hospital for evaluation of pericardial stripping after developing symptoms of heart failure. On admission the patient was borderline hypotensive at 90/66mmHG and hypoxic, requiring 3.5L of oxygen. Physical exam revealed bilateral 3-4+ lower extremity edema to the hip, distant heart sounds, hepatomegaly, lung crackles and JVD to the mandible. BNP was 3,548. EKG showed normal sinus rhythm and diffuse low voltage. Initial echocardiogram revealed an ejection fraction of 51-55% and a moderate-sized pericardial effusion with pericardial thickening, concerning for constrictive pericarditis. However, the final report suggested a restrictive process given no respiratory variation in transmitral flow, leading to initiation of aggressive diuresis for heart failure secondary to restrictive cardiomyopathy.

Diuresis was continued for two days with minimal response. Cardiac catheterization was performed for more definitive data, which revealed no coronary artery stenosis and concordance in pressure tracings, consistent with restrictive cardiomyopathy. Aggressive diuresis was continued for 1 week given catheterization results, however the patient remained profoundly fluid overloaded and hypoxic. At this time, clinical concern for constrictive pericarditis was mounting given lack of response to diuresis. Repeat echocardiography was performed which revealed septal deviation with respiratory variation, consistent with constrictive pericarditis. Pericardial stripping was performed and the patient ultimately had vast improvement in his symptoms.

**Discussion:** The management of this case was difficult due to the lack of clear evidence supporting a single diagnosis. With a history of effusive pericarditis and pericardial thickening per echocardiogram the patient's presentation was more consistent with constrictive pericarditis. However, initial echocardiogram and invasive catheterization reports suggested restriction, so the patient was initially treated as such. Differentiating between restrictive cardiomyopathy and constrictive pericarditis is difficult for the most seasoned internist. Specialty studies utilized to differentiate between the two are susceptible to a number of variables that can ultimately alter final data interpretation. In the face of conflicting diagnostic data, the practitioner must focus on the patient, allowing clinical presentation to guide management, rather than relying solely on diagnostic tests and their results.

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A CASE OF TEMPERATE PYOMYOSITIS IN A HEALTHY YOUNG RUNNER

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23 year old previously healthy male, long distance runner, presented to the emergency department with left hip pain of two weeks duration. He was initially examined by his sport medicine doctor. A hip x-ray was done and was unremarkable. He was treated for possible muscular sprain. His pain was gradually progressing, increasing with walking and improving with rest. No history of recent viral illness, trauma, IV drug abuse, skin breakdown, rash or insect bites. On examination he had a temperature of 36.7 c, pulse of 57 bpm, respiratory rate of 18 breath/min, oxygen saturation of 99% on room air and blood pressure of 120/69 mm Hg. He had tenderness in inner thigh muscles and lumbar area but no swelling was noted. Psoas sign was positive bilaterally. His muscular strength was diminished in the hip flexors bilaterally, no sensory deficit noted. He was found to have leukocytosis with neutrophilic predominance, elevated ESR and CRP. MRI of the pelvis and lumbar spine showed multiple fluid collections within the musculature of the thighs, pelvis, and rectus abdominis muscles. He was diagnosed with pyomyositis. Ultrasound guided aspiration of the rectus abdominis yielded purulent fluid. Culture of the fluid grew MSSA. Results of the blood and urine cultures showed MSSA as well. A screening test for HIV was negative. A transesophageal cardiac echo-cardiogram revealed no valvular vegetations. The patient was treated with IV antibiotics for 42 days and his condition improved.

Discussion: Pyomyositis is an acute bacterial infection of skeletal muscle, typically involving the larger muscles of the lower extremities and trunk. It is endemic in tropical areas. By contrast it is uncommon in non tropical areas. Temperate pyomyositis has been described in patients who have HIV disease, diabetes, hematopoietic disorders and cancer with undergoing chemotherapy and in IV drug abusers. It has been divided in to three stages including invasive stage, purulent stage and late stage. Most of the patients are first seen in the purulent stage because of the presence of fever, chills and progressive pain. MRI is the imaging modality of choice for the diagnosis of pyomyositis. It is a life threatening condition and diagnosis in early stages is challenging and requires high clinical suspicion. A delay in diagnosis can lead to sepsis and death. Systemic antibiotic is the mainstay of treatment and can eliminate the need for surgical drainage in selected cases.

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## A RARE CASE OF HYALINIZING GRANULOMA OF THE LUNG

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**Introduction** Pulmonary hyalinizing granuloma (PHG) is a rare benign disease characterized by fibrosing nodules formed by lamellar collagen hyaline. The exact cause of this condition is unknown. Most of the lesions are not initially correctly diagnosed. The aim of this case presentation is to focus on clinical presentation, diagnosis and treatment of PHG.

**Case description** An 85-year-old male with a history of pulmonary arterial hypertension, congestive heart failure, paroxysmal atrial fibrillation and asthma, was seen in the pulmonary clinic for persistent dyspnea and wheezing despite the use of inhalers for his asthma. A CT scan of the chest without contrast was done and revealed mediastinal and hilar lymphadenopathy with calcifications, bronchiectasis and scarring in the right lower lobe. A flexible bronchoscopy with EBUS was performed and showed subcarinal and paratracheal lymphadenopathy. Biopsy was taken and cytology was negative for malignancy. A few months later, he was hospitalized with severe shortness of breath and found to have acute right main stem occlusion. Repeat bronchoscopy showed an obstructive right main stem endobronchial lesion. The patient underwent laser curettage of the endobronchial mass. The pathology from the right main stem bronchus mass revealed hyalinized material and acute inflammatory necrosis with focal calcifications and focal granulation tissue. No malignancy was identified. A repeat CT scan of the chest showed findings most consistent with granulomatous disease, and resolution of the right mainstem bronchial obstruction. Extensive testing for causes of granulomatous disease came back negative.

**Discussion** The exaggerated immune response to antigenic stimuli by infection or autoimmune processes is the presumed pathophysiology of PHG. The clinical presentation of PHG includes cough, fever, fatigue, dyspnea and pleuritic chest pain. However, patients may be completely asymptomatic. The accurate diagnosis of PHG can only be made with histopathological examination. Typically there is deposition of hyaline tissue masses accompanied by sparse lymphocytic infiltrate. Single lesions tend to be stable and resection is often curative. There is no definitive treatment for multiple nodules, although improvement with glucocorticoid use has been reported. The prognosis of patients with PHG is generally excellent with no significant impact on longevity.

**Conclusion** PHG is a rare fibrosing nodular disease that has few complications and an excellent prognosis. PHG should be considered in the differential diagnosis of pulmonary nodules. While PHG nodules are usually solid, they may be cavitory or contain calcifications.

## PANCREATITIS AND FACIAL PALSY WITH REACTIVATED EPSTEIN-BARR VIRUS (EBV) INFECTION

First Author: Amandeep Singh Aujla Other Authors: Nelky Ramirez, MD Shobhana Chaudhari, MD

**Introduction:** Epstein-Barr virus (EBV) is a gamma-herpes virus known to cause mononucleosis like syndrome and various malignancies including Burkitt lymphoma and nasopharyngeal carcinoma. The illness is usually self-limited and resolves in 2-4 weeks. But multiple complications have been reported in literature. We report syndromic presentation of reactivated EBV infection presenting as acute pancreatitis, pancytopenia, pharyngitis, splenomegaly with late onset facial palsy.

**Case description:** A 34 year-old male with no significant medical history presented with epigastric abdominal pain and non-bilious vomiting for 3 days. Review of symptoms revealed subjective fever, sore throat and watery diarrhea for 1 week. He is an occasional drinker and reported last drink 1 month ago. He was not taking any medications at home. Examination was relevant for tachycardia, pharyngeal erythema without lymphadenopathy and mild tenderness in epigastric region. Laboratory work-up revealed elevated lipase levels; normal calcium and triglycerides and negative toxicology. Imaging was remarkable for gallstone and pancreatitis without necrosis. Management was started with bowel rest, intravenous fluids and pain control. Magnetic resonance cholangiopancreatography ruled out any obstruction or stones but revealed splenomegaly. Next day; he complained of pain in right side of jaw and was found to be febrile with minimal parotid swelling and tenderness. Hemogram showed mild pancytopenia with low reticulocyte index (0.2). Repeat contrast enhanced CT didn't show any interval changes. Patient continued to have elevated lipase levels with intermittent fever for 12 days and all blood/stool/urine cultures were negative. Serologies came negative for mumps, coxsackie, CMV, hepatitis-A, hepatitis-B, hepatitis-C and HIV. HSV-1 IgG antibody was positive but PCR came negative. EBV antibody titers were equivocal for past versus reactivated infection. Autoimmune work-up came negative for Lupus, Sjogren and IgG-4 diseases. On 14<sup>th</sup> day of hospitalization, he developed lower motor neuron facial nerve palsy and was started on prednisone. Sarcoidosis was ruled out and patient was discharged based on clinical improvement. Repeat EBV serology at interval of 4 weeks is suggestive of reactivated infection. Pancytopenia is resolved and facial palsy is improving.

**Discussion:** Diagnosis of EBV infection is typically clinical but atypical presentation needs to be confirmed with serology. Rising titers of antibodies with seroconversion of anti-Early Antigen supports the diagnosis of reactivated infection in our patient. Pancreatitis and neurological sequelae including isolated facial nerve palsy have been reported individually with EBV in literature. Pancytopenia is also a common presentation of EBV infection secondary to increased T-cell activation and Interferon-gamma related bone-marrow suppression. It is a rare presentation of EBV infection with two uncommon complications together. Management is usually supportive except noted benefit of steroids in facial palsy.

## EUGLYCEMIC DIABETIC KETOACIDOSIS: THE CLINICAL CONCERN OF SGLT2 INHIBITORS

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Sodium–glucose cotransporter 2 inhibitors (SGLT-2) are the newest class of anti-glycemic approved to treat type 2 diabetes mellitus (T2DM). Off label use in type 1 diabetes mellitus (T1DM) is increasing, presumably due to the increasing obesity in T1DM patients. Euglycemic DKA (EuDKA) is a fast growing concern with this new drug.

A 39 year old obese woman with presumed T2DM for many years presented with three days of nausea/vomiting and abdominal pain. She had been non-adherent to prescribed multiple dose of insulin (MDI). One week prior to presentation, she had been started on canagliflozin and liraglutide to increase medications adherence. She denied alcohol use, starvation, or recent illness. Examination: Temperature 97.7 F, blood pressure 120/80 mmHg, heart rate 133 bpm, respiratory rate 24/min. Patient was ill appearing, had dry mucus membranes, diffusely tender abdomen.

Labs: bicarbonate 6 meq, anion gap 25, lactic acid 1.4 mmol/L, blood glucose 167 mg/dl, HbA1c 11.1%. Urine dipstick was positive for ketones. Arterial blood gas: PH 7.08; PCO<sub>2</sub> 22 mmHg; HCO<sub>3</sub><sup>-</sup> 6.5 mEq/L. Given her unimpressive level of blood glucose, the diagnosis of DKA was unrecognized initially. The patient received intravenous fluid hydration and antiemetics without improvement. Upon admission to MICU, dextrose water 5% /half normal saline, and an insulin drip were initiated. Her DKA resolved after 36 hours and she was discharged on MDI.

EuDKA is rare syndrome defined as DKA without marked hyperglycemia. The hypothetical mechanisms of EuDKA differ in both T1DM and T2DM. In T1DM, SGLT-2 inhibition leads to a lowering of the insulin dose by the clinician or the patient to prevent hypoglycemia. Reduction in circulating insulin levels contributes to the development of DKA. In T2DM, SGLT-2 inhibitors increase urinary glucose excretion and decrease the blood glucose, stimulating glucagon and depressing insulin release. This hormonal shift enhances lipid oxidation at the expense of carbohydrate oxidation and stimulates lipolysis. Augmented free fatty acid delivery to liver promotes ketogenesis.

One case series study of EuDKA from canagliflozin found that six out of twelve patients had T1D or late autoimmune diabetes in an adult. Our patient tested positive for Glutamic Acid Decarboxylase autoantibodies (GAD65).

There have been >70 cases reported to FDA since the release of this drug class. Most patients and clinicians fail to recognize the symptoms of DKA, given near normal blood glucose. Insulin reduction, low caloric/fluid intake, current illness, starvation, and alcohol use were other factors leading to the development DKA.

Increased awareness of EuDKA caused by SGLT2 inhibition is warranted. Given the increasing incidence and the insidious nature in which the diagnosis can be missed, vigilance is necessary to decrease morbidity and potentially mortality.

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## HICCUPS AS A HARBINGER FOR HSV ENCEPHALITIS

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**Case Presentation:** A 54 year old previously healthy male presented with one day of altered mental status. Two days prior to admission, the patient had new onset of intermittent hiccups, occurring 10-15 times per day, without other associated symptoms. The morning of presentation, he was found by co-workers to be disoriented with incoherent speech, and was warm to touch, prompting EMS to be called. On arrival, the patient was febrile to 102.4, ill appearing, with intermittent hiccups, and oriented only to name. There were no meningeal signs, focal neurological deficits, skin rashes or oral or genital lesions. Initial labs showed a leukocytosis of 11.2 with 26% lymphocytes. The patient was started on Vancomycin, Ceftriaxone and Acyclovir for empiric coverage of meningoencephalitis. A head CT showed no abnormalities. Lumbar puncture was significant for lymphocytic pleocytosis, normal glucose, elevated protein and negative gram stain and culture. MRI revealed a large area of T2 enhancement involving the anteromedial left temporal lobe, posterior left frontal lobe and the insular cortex. HSV DNA PCR of CSF fluid later returned positive for HSV 1. The patient had gradual improvement in mental status on sole therapy with Acyclovir; however he continued to have daily frequent hiccups throughout his hospital course.

**Discussion:** HSV is implicated in roughly 10 percent of encephalitis cases yearly and portends a high degree of morbidity and mortality. Without therapy, studies have estimated 70% mortality; while this has been shown to decrease to 25% with treatment, survivors frequently have neurological sequelae. The most common presenting symptoms are acute onset of fever, altered mental status, focal neurological signs and seizures. Rarely, there have been case reports of HSV encephalitis preceded by a prodrome of hiccups such as the case we present here. Hiccups have been associated with wide array of pathologies ranging from CNS infections, medication side effects, and thoraco-abdominal processes involving the vagal or phrenic nerves. The pathophysiologic relationship between HSV and hiccups has not been clearly delineated, but is postulated to be related to brainstem inflammation.

**Conclusion:** HSV Encephalitis is a highly fatal disease that typically presents with fevers, altered mental status and focal neurological signs; however it can rarely be associated with prodromal hiccups. Early suspicion, diagnosis and treatment is key due to the high degree of associated morbidity and mortality. While the differential diagnoses associated with hiccups are vast, this prodromal symptom should raise clinical suspicion for a significant underlying disease process.

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PULMONARY EMBOLISM OR PERICARDIAL EFFUSION? THE UTILITY OF CHEST CT FOR URGENT DIAGNOSIS AND MANAGEMENT

First Author: Samir Bhalla, MD Second Author: Rajiv Bhagat, MD Third Author: Patrick Cocks, MD

**Case Presentation:** A 36 year old female with history of triple negative breast adenocarcinoma presented with progressive dyspnea on exertion for 10 days. Chest CT demonstrated a right pleural effusion and innumerable metastatic lesions throughout the thorax including a 6x7x8cm mediastinal mass invading into the right atrium and compressing the SVC and IVC. Additional images revealed a filling defect within the right pulmonary artery representing an acute pulmonary embolism. Palliative oral chemotherapy with Xeloda and external beam radiation were initiated; the patient was also anticoagulated with LMWH for pulmonary embolism. On hospital day seven, the patient was noted to be tachycardic to the 140s with alteration in her mental status. She was normotensive without pulsus paradoxus, and auscultation of her lung fields was non-focal and unchanged from admission. Given the extent and pericardial location of her mediastinal disease, initial diagnostic considerations included pericardial effusion and recurrent pulmonary embolism. Due to unavailability of urgent echocardiogram, the patient was sent for repeat chest CT which revealed a large and likely bloody pericardial effusion with right ventricular collapse and contrast reflux into the vena cavae consistent with tamponade. Protamine was administered and the patient was transferred to the CCU where she underwent urgent pericardiocentesis with drainage of roughly 300cc of bloody fluid.

**Discussion:** Hemorrhagic pericardial effusions are most associated with trauma, recent cardiac surgery, aortic dissection, malignancies involving the pericardium, and tuberculosis in endemic areas; rarely have there been cases of spontaneous hemorrhage due to anticoagulant therapy. We present the unfortunate case of a patient with a mediastinal metastases who developed a hemorrhagic pericardial effusion and tamponade in the setting of anticoagulation for pulmonary embolism. This case highlights the difficulty in diagnosing a pericardial effusion and tamponade. Classic physical exam findings such as Ewart's sign, pulsus paradoxus, and EKG findings of low voltage and electrical alternans, have low sensitivity and specificity for effusion and tamponade. While TTE is the diagnostic modality of choice, new evidence supports the utility of chest CT to identify pericardial effusions, elucidate the etiology of these effusions, and to rule out alternative diagnoses.

**Conclusions:** A unique aspect of this case is the development of spontaneous hemorrhagic pericardial effusion in patient with extensive thoracic malignancy. This case demonstrates the utility of the CT chest in identifying pericardial effusions and tamponade physiology while simultaneously ruling out recurrent pulmonary embolism in a patient with risk factors for both pathologies. While echocardiography remains the favored imaging modality, chest CT can be obtained quickly in most medical centers, with high sensitivity for pericardial effusions and capability of ruling out alternative pathologies.

LOW BACK PAIN IN PSYCHIATRIC PATIENT: A RARE CASE OF LUMBAR PARASPINAL COMPARTMENT SYNDROME

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**Introduction:** Low back pain is a common and usually self-limiting health problem. When it is associated with neurologic symptoms, more serious underlying conditions need to be investigated. Lumbar paraspinal compartment syndrome (LPCS) is an extremely uncommon condition defined as increased pressure within the closed paraspinal muscle space, resulting in rhabdomyolysis and ischemic pain. LPCS has been reported in various settings including strenuous exercises, direct trauma and non-spinal surgery. Below, we present a case of LPCS which is first reported to be associated with antipsychotic medication use.

**Case presentation:** A 57-year-old male with history of obsessive-compulsive disorder (OCD) presented with acute progressively worsened lower back pain and unsteady gaits with multiple falls for past two days. Patient denied direct trauma to his back. No alcohol/illicit drugs abuse. Home medications included clomipramine, fluvoxamine and clonazepam.

Initial vitals showed HR 120 bpm; BP 89/57 mmHg; and afebrile. Physical examination revealed fine tremors in tongue and fingertips, increased tone and rigidity in legs, with moderate tenderness in lumbar spine and bilateral paraspinal regions. Labs were remarkable for creatine kinase (CK) 3792 IU/L; serum creatinine 3.4 mg/dL; and slightly elevated transaminases. His hemoglobin was 15.4gm/dL. Computed tomography (CT) of the head, thoracic spine, and lumbar spine without contrast were unremarkable. Treatment started with intravenous fluid, analgesics for rhabdomyolysis and antipsychotic medications were stopped for possible side effects. In the next 48 hours, patient's back pain worsened. Repeat CK was 56000 IU/L, AST 633 U/L; ALT 255 U/L with stable hemoglobin. Bromocriptine and bztropine were started for dysautonomia and cholinergic symptoms. MRI of thoracic and lumbar spine without contrast revealed abnormal signal throughout the paraspinal musculature which may represent intramuscular edema and/or hemorrhage. Orthopedic consultant suggested continuing conservative treatment. Patient's symptoms and CK level showed gradual improvement. The CK level returned to normal range at 6-week follow up.

**Discussion:** In our case, patient presented with commonly described side effects from antipsychotic medication associated with lower back pain and rhabdomyolysis. Due to persistence and worsening of symptoms, an MRI was ordered and revealed lumbar paraspinal muscle abnormalities. After excluding all the known causes, it is very plausible that the development of LPCS was contributed by his antipsychotic medication. Although clomipramine and fluvoxamine can cause neurotoxic syndromes associated with rhabdomyolysis, there is no report of developing compartment syndrome. When psychiatric patients present with lower back pain and rhabdomyolysis, LPCS syndrome should be considered in the differential diagnosis. Early recognition, diagnosis and treatment could prevent irreversible damage and long-term functional sequelae.

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DERMATOMYOSITIS AND HIV INFECTION: A RARE ASSOCIATION

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Dermatomyositis is one of the groups of idiopathic inflammatory myopathies that share common features of immune mediated muscle injury with incidence less than 2 per 100,000. There is an increased incidence of rheumatologic diseases in Human Immunodeficiency Virus (HIV) infection. Yet, there have been very few reported cases in medical literature of Dermatomyositis and HIV. We present a case of dermatomyositis in a young male with HIV infection without other well-described predispositions.

A 19-year-old male, recently diagnosed with dermatomyositis, presented to medical clinic in mid 2009 with complaints of acne, hair loss and proximal muscle weakness. He was on prednisone, methotrexate and folate. He admitted being sexually active with both male and female partners with infrequent use of condoms. Subsequently he complained of sore throat, fever, anal pain and discharge, but he refused HIV testing claiming he had tested negative elsewhere. He was seen throughout 2010-2011 for oral candida, scabies and anal lesions that were later diagnosed to be condyloma acuminata. He was also found to be RPR and FTA positive. In early 2012 he agreed to be re-tested for HIV, his HIV EIA and western blot were positive with CD4 of 182 cells/mcl and HIV RNA of 1337310 copies/ml. He was started on HAART. He was treated with trials of prednisone, methotrexate, mycophenolate and azathioprine without success and was started on monthly IVIG. Despite aggressive treatment, he was admitted in mid 2015 for an episode of dermatomyositis flare, which prompted a right vastus lateralis muscle biopsy, which was consistent with dermatomyositis. He was discharged to follow up as an outpatient for rituximab therapy. He continues to take HAART and follows up in Infectious disease clinic.

HIV infection influences the pathogenesis of autoimmune diseases by activating polyclonal B cells with autoantibodies formation and by increasing expression of auto-antigens due to direct infection of endothelium and by molecular mimicry between HIV and endothelium. Early stages of HIV are associated with flares of autoimmune diseases, which lessen as autoimmunity is suppressed with the progression of HIV. Also, HAART-induced immune reconstitution may exaggerate the immune responses, and lead to exacerbations. Our patient gave history of a negative HIV test while being treated for dermatomyositis, though he may have been having acute retroviral syndrome. Physicians need to have high degree of suspicion in diagnosing HIV seroconversion in patients presenting with autoimmune diseases like dermatomyositis, especially in high risk patients. Treatment remains a challenge, as there has to be fine balance between immunosuppression and retroviral immune reconstitution.

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AN INTERESTING CASE OF A LARGE CELL PARANASAL NEUROENDOCRINE TUMOR

First Author: Ryan K Dean, DO Aashrai Gudlavalleti, Amit Dhamoon

**Introduction:** Neuroendocrine tumors (NETs) are a group of heterogenous epithelial neoplasms that produce peptides and amines. Despite their heterogeneity, they share common features, including growth pattern and expression of histologic biomarkers. While NETs can arise in almost every organ, the nasal and paranasal sinuses are relatively rare sites. Because of the relative paucity of reported cases of sinonasal NETs, there is no definitive management regarding this rare neoplasm. In addition to its rarity, this is an extremely aggressive and recurrent disease and its management needs to be shared to develop treatment guidelines.

**Case Presentation:** An 81-year-old male initially presented to the ED after he was involved in an MVA. A CT head incidentally found a right posterior ethmoid mass, which was asymptomatic at the time. One month later, at an initial ENT evaluation, the patient's extra-ocular muscle movements (EOMs) were grossly intact without any ptosis. A flexible nasopharyngoscopy demonstrated an area of thick mucosal erythema and an endoscopic biopsy was planned. Over the next two weeks, the patient progressively developed dull headaches, severe right eye pain, severe extra-ocular muscle limitation and worsening ptosis and returned to the ED. An MRI showed a large nasal passage mass with extension into the right orbit, right maxilla, cribriform plate and into the right intracranial fossa without extension into the overlying right frontal lobe, with significant growth in comparison to the prior CT. The patient received high dose steroids and an endoscopic biopsy was performed. The immediate frozen section biopsy was reported to be a small blue cell tumor, most likely esthesioneuroblastoma, at which time radiation oncology started the patient on a 7-week course of radiation. The final biopsy result ultimately showed a large cell neuroendocrine tumor. Radiosensitizing cisplatin chemotherapy was started after the final results, in addition to the planned 30 fractions of radiation.

**Discussion:** NETs of the paranasal sinuses are rare but remarkably aggressive, with a high rate of recurrence. Regrettably, the medical community is lacking in sufficient literature regarding definitive guidelines on its management. Few studies suggest that surgery followed by chemoradiation may be beneficial in overall survival. However, in our patient, the mass had extended into the cribriform plate and initial surgery was not an option. Notwithstanding, the patient has initially had a positive response to chemoradiation therapy. This case illustrates the aggressiveness of a rare sinonasal NET and its initial response to chemoradiation. An extensive follow up with the patient is planned to document his response to the treatment. This will contribute significantly to the limited data on epidemiology and management of sinonasal NETs.

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A CASE OF ACUTE DISSEMINATED HISTOPLASMOSIS AFTER CYSTOSCOPY IN IMMUNE COMPETENT PATIENT

First Author: Ayman Elbadawi, MD Second Author: Karthik Vadamalai, MD Third Author: Hamdy Ahmad, MD Last Author: Ann Falsey, MD

**Introduction:** *Histoplasma capsulatum* usually causes asymptomatic infection of the lung. About 5 % of acute infection develops into disseminated histoplasmosis, mostly in immune-compromised and patients at extremes of age.

**Case report:** 65 year old male presented with a one week history of high grade fever, fatigue and confusion which began abruptly two days after cystoscopy for recurrent urinary tract infection. Past medical history included pulmonary sarcoidosis diagnosed by mediastinal lymph biopsy (didn't receive steroids), diabetes and hypertension. On admission he was febrile and confused with stable vital signs. Physical exam was notable only for rhonchi on chest exam. Initial workup included negative Head CT and lumbar puncture. Blood work revealed normal metabolic and liver function tests with progressive anemia, thrombocytopenia and atypical lymphocytosis of 15-20%. Chest and abdominal CT showed modestly enlarged mediastinal and retroperitoneal lymph nodes felt consistent with prior history of sarcoid. Blood, urine and respiratory cultures all were negative for bacteria and EBV, CMV, HIV testing were negative. A bone marrow biopsy was done given the abnormal lymphocytes in peripheral smear, revealing budding yeast consistent with *Histoplasma capsulatum*. Histoplasma antigen was positive in urine and eventually blood and bone marrow grew *H.capsulatum*. Patient was started on amphotericin-B for diagnosis of disseminated histoplasmosis. After a 2 week period of amphotericin B, patient was switched to oral Itraconazole to complete six months course of treatment.

**Discussion:** *H. capsulatum* enters the human host by inhalation in to the lungs. In immunocompetent patient the lung macrophages ingest the fungi and are responsible for initial asymptomatic dissemination to various organs. Development of cellular immunity becomes the prime factor in containing these fungi inside the granuloma in these organs, discovered mainly from autopsy studies. There are case reports of chronic disseminated histoplasmosis especially in the elderly patients with no obvious immunosuppression. Specific defects in macrophages and T cells, in the processing of *Histoplasma* antigens are described as mechanism for this process. Several case reports have shown that histoplasmosis can involve the genitourinary system, where it's mostly asymptomatic and rarely present with testicular abscess, prostatic abscess or epididymitis. Given the temporal association with the cystoscopy in our patient, we hypothesize that latent infection in the prostate may have been disrupted by the urologic procedure leading to dissemination of fungi into bloodstream. The previous diagnosis of sarcoidosis may have been latent histoplasmosis. Atypical lymphocytosis has not been previously described in patients with histoplasmosis. In conclusion, disseminated histoplasmosis should be kept in our differential for fever of unknown origin in elderly immunocompetent patients with hematologic abnormalities.

## IDIOPATHIC CHYLOUS ASCITES IN A PATIENT WITH CRYPTOGENIC ORGANIZING PNEUMONIA: A CASE REPORT

First Author: Yasaman Eslaamizaad, MD, Sunil Saith MD, Tshering Amdo, MD

**Introduction** Chylous ascites (CA) is the accumulation of milk-like lymphatic fluid with elevated triglyceride (TG) levels in the peritoneal cavity. CA is known to develop secondary to traumatic injury or lymphatic obstruction by malignancy, causing chyle leakage through lymphatic vessel walls. CA can also be caused by disease conditions, including tuberculosis, liver cirrhosis and nephrotic syndrome. There are few reported cases of idiopathic CA. Cryptogenic organizing pneumonia (COP) describes idiopathic bronchiolitis obliterans organizing pneumonia (BOOP), a distinct pattern of lung reaction to injury. We describe a patient with non-traumatic, non-malignant CA and COP, whose hospital course was complicated by acute respiratory failure.

**Case Presentation** A male in his mid-forties with a medical history significant for hypertension, chronic obstructive pulmonary disease, pulmonary hypertension, atrial fibrillation and end stage renal disease secondary to IgA nephropathy was referred to the hospital for exchange of a leaky dialysis catheter.

Catheter exchange was complicated by septic shock and acute type I respiratory failure requiring intubation. Chest radiograph and CT revealed bilateral pleural consolidation and effusions. Patient was started on pressors and antibiotics. Physical examination was remarkable for a distended abdomen. Abdominal CT revealed massive ascites. Paracentesis revealed cloudy, white fluid with elevated TG level of 764 g/dl. Serum ascites-albumin gradient (SAAG) was 0.8. Fluid and smears were negative for acid-fast bacilli and QuantiFERON®-TB and HIV testing were negative. Pancreatic and liver enzymes were within normal limits. Echocardiogram demonstrated normal ejection fraction. Rheumatological workup including complement and Anti-Ro and Anti-La was unremarkable. Venous ultrasounds and bilateral upper and lower extremity lymphoscintigraphy were negative for any lymphatic flow interruption.

Upon completion of antibiotic course, patient was extubated, but required reintubation for hypercapnic respiratory failure. Chest radiographs demonstrated worsening bilateral opacifications. When compared to 11 days prior, CT imaging revealed increasing diffuse ground-glass appearance bilaterally, with partly consolidative airspace disease within the right upper and lower lobes. The lung biopsy revealed negative cytology and demonstrated cellular and fibrosing interstitial pneumonia with organization in the right upper and middle lobes, confirming COP. Ultimately, patient failed multiple weaning trials and required tracheostomy.

**Discussion** CA typically occurs as a result of lymphatic system interruption in the setting of trauma, obstructing malignancy or lymphatic anomaly. Rarely, there are atraumatic causes of a chylous ascites, which can be sequela of numerous disease processes. Our case illustrates the diagnosis of a rare, non-traumatic, non-malignant CA with concurrent COP after the patient's septic shock and pneumonia did not improve with mechanical ventilation and antibiotics.

## A RARE CASE OF STATIN-INDUCED NECROTIZING AUTOIMMUNE MYOPATHY LEADING TO RESPIRATORY FAILURE

First Author: Deepthi Gandhiraj, MD Second Author: Dipak Chandy, MD

Side-effects of statins such as myopathy and transaminitis usually improve after discontinuation or dose modification. Statin-Induced Necrotizing Autoimmune Myopathy (SINAM) is a very rare, severe side-effect where symptoms and marked elevation of creatinine kinase (CK) persist after stopping statins, requiring immunosuppression for treatment. We cared for a patient with SINAM progressing to respiratory failure.

A 70-year old male with hyperlipidemia was started on atorvastatin. Within three weeks, he started having muscle pain and soreness. He had CK of 1400 and atorvastatin was stopped. But he continued to have difficulty lifting weights which progressed to difficulty walking over one month. He was admitted after a fall. He had strength of 1-2/5 in the proximal and 4-5/5 in the distal muscles of his extremities. Admission labs revealed CK 9200 U/L. MRI was consistent with myositis of bilateral thigh muscles. Anti-nuclear Ab, myositis panel and viral markers were negative. Anti-3-hydroxy methyl glutaryl CoA reductase receptor (HMGCR) antibody titer > 200 units was consistent with SINAM. Thigh muscle biopsy, with blotchy areas of necrosis, varying regenerative fibers and no evidence of inflammation was typical of SINAM. Patient was started on intravenous methylprednisolone and intravenous immunoglobulin for 4 days followed by high-dose oral prednisone. CK dropped to 6000 but weakness progressed leading to dyspnea, respiratory acidosis and intubation within a few days. Deep vein thrombosis (DVT) complicated by upper gastrointestinal bleed (UGIB), while on anticoagulation, required placement of an IVC filter. Due to these complications, immunosuppression was held. Over the next 2 weeks, patient could not be weaned and tracheostomy was performed. Repeat MRI showed continued evidence of myositis indicating an ongoing disease process. He was started on a weekly methotrexate regimen. After four weeks, muscle weakness improved with increasing periods off mechanical ventilation. Patient was eventually discharged to a long-term acute care facility on methotrexate and ventilator support.

There are 6 grades of statin-induced myotoxicity based on severity. SINAM is the rarest and most severe form of toxicity where muscle weakness and marked elevation of CK continue to progress after stopping statins. It is not known whether dose and duration of treatment affects severity. In the majority of patients, diagnosis is made by elevated serum Anti-HMGCR antibody levels. Muscle biopsy is characterized by myonecrosis without significant inflammation around non-necrotic fibers. Along with discontinuation of statins, immunosuppression is often required. Prednisone ± methotrexate is mainstay of therapy though multiple immunosuppressants may be required. Patients often require long-term immunosuppression. Involvement of respiratory muscles has been rarely reported in SINAM and so identification and prompt intervention are critical. High-intensity inspiratory muscle therapy has been shown to improve respiratory muscle performance. Studies focusing on different manifestations and treatment strategies of SINAM will help better manage such patients.

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SCREENING FOR CELIAC DISEASE IN PATIENTS WITH SARCOIDOSIS?

First Author: Dalvir Gill (SUNY Upstate Medical University Hospital) Second Authors: Jaswinder Virk, Sakshi Dutta (SUNY Upstate Medical University Hospital), Allam Fatme (VAMC)

**Introduction:** Sarcoidosis and celiac disease (CD) are both immune disorders, which have been associated with class II haplotype HLA-DR3, DQ2. Literature shows this combination is more prevalent in the Irish population. CD involves the small bowel and can lead to multiple vitamin deficiencies. More importantly there is an association between CD and cancers, mainly B cell lymphoma and gastrointestinal cancers. Diagnosis of CD in a patient with sarcoidosis has special importance including implication for the treatment of both diseases.

**Case discussion:** 35 year old woman, diagnosed with biopsy proven sarcoidosis, developed a complex clinical course over time. She started experiencing recurrent watery diarrhea, accompanied with abdominal pain and generalized weakness. Physical exam revealed thin chronically unwell female, with BP of 115/76 | pulse 94 | temperature 97.7 °F (oral) | respiratory rate 16/minute | BMI 20.49 kg/m<sup>2</sup> | oxygen saturation 100% on room air. She also had evidence of alopecia, non specific dermatitis and clear lungs. Labs revealed hemoglobin of 11.3 g/dL, ferritin of 11 ng/mL, and 25-hydroxy vitamin D of 20 ng/mL. Stool tested negative for ova/parasites, Clostridium difficile, WBCs, cultures and viral studies. Celiac Disease (CD) work up revealed, elevated transglutaminase IgA 57 units (sensitivity 90 to 98%; specificity 95 to 97%), and elevated IgG deamidated gliadin antibodies, 77 units (sensitivity 92 %; specificity 100 %). Duodenal biopsy was suggestive of CD, with marked villous blunting, increased lymphocytes and epithelial damage, and lamina propria expanded by numerous plasma cells and crypt hyperplasia. She improved symptomatically on a gluten free diet and with replacement of her deficient vitamins.

**Conclusion:** We present a patient with sarcoidosis and CD, given the genetic predisposition for these diseases to coexist especially in patients with Irish background; the clinician caring for the patient should be vigilant for such scenarios. Simple measures such as dietary modifications can result in immense improvement for the patient. We also need to be mindful of the long-term sequelae of CD, such as intestinal lymphoma. Hence, if the patient with sarcoidosis has non-specific gastrointestinal symptoms such as bloating after meals, abdominal cramps, and diarrhea, one should strongly consider screening for coexisting CD. Given the prevalence of CD in this target population, screening could be done using relatively inexpensive tools such as IgA endomysial antibodies, or anti-tissue transglutaminase antibodies. We feel screening for CD in patients with sarcoidosis that are complaining of GI symptoms will greatly benefit the patient and the health care system. Hence, we strongly feel that larger studies are required to validate this recommendation.

## WEIGHT LOSS SUPPLEMENT INDUCED PANCREATITIS

First Author: Angela C Grigos, MD Jamila Benmoussa, MD Jaspreet Sandhu, Medical Student Matthew Clarke, MD

Obesity is a major health burden in the United States. Herbal supplements as weight loss options are increasing in popularity, since they are available at a low cost. However, herbal supplements lack FDA regulations and little has been published regarding their side effects. We present an unique case of acute pancreatitis secondary to garcinia cambogia (GC).

A 61-year-old female with history of diabetes mellitus type 2, hypertension, chronic kidney disease, chronic hepatitis C presented with mid-epigastric pain for three days. She denied alcohol consumption or history of gallstones. Upon presentation vitals were normal, BMI 49.9 kg/m<sup>2</sup>. Laboratory results: triglycerides 104 mg/dl, glucose 94 mg/dl, calcium 8.7 mg/dl, albumin 3.1 g/dl, protein 7 g/dl, AST 68 U/L, ALT 75 U/L, lipase 13,332 U/L. She started taking a weight loss supplement GC two weeks ago. CT scan of the abdomen demonstrated mild peri-pancreatic fat stranding, consistent with acute pancreatitis. MRCP showed unremarkable intrahepatic, extra-hepatic biliary ducts and no gallstones. Home medications included Metformin, Glipizide, Metoprolol and HCTZ, with no recent changes for the last two years. On hospital day 4, she had resolution of the pain and lipase levels trended down after discontinuation of GC.

GC, a fruit found in Asia and Africa has been recently popularized by the Dr. Oz program as a dietary supplement for weight loss. Hydroxycitric acid is the active ingredient and it help in weight loss by competitively inhibiting the enzyme ATP citrate lyase, ultimately altering the synthesis of fatty acids, cholesterol and triglycerides. It promotes fat oxidation, normalizes lipid and lower leptin levels in obese patients.

Instances of acute pancreatitis following GC usage are limited. Social media websites linked two cases of acute pancreatitis to GC. In this case virtually all possible etiologies of acute pancreatitis were ruled out, leaving the recent usage of GC as the only significant factor. Several studies have shown a possible link between GC and oxidative stress. It was observed that GC significantly up-regulated hepatic superoxide dismutase and glutathione peroxidase mRNA expression with concomitant increase in lipid peroxidation in the liver. This suggests that an increase in antioxidant gene expression by GC seems to be a compensatory response to increased oxidative stress. It is established that reactive oxygen species play an important role in the pathogenesis of acute pancreatitis. Specifically, increase in the rate of free radical generation mediated by lipid peroxidation can lead to oxidative damage and pancreatic inflammation.

This case is demonstrates a possible association between the weight loss supplement GC and acute pancreatitis. Importantly, it illustrates the need for increasing population awareness about the side effects of non-FDA approved herbal supplements used for weight management.

SPONTANEOUS THYROID HEMORRHAGE - UNUSUAL CAUSE OF ACUTE AIRWAY OBSTRUCTION

First Author: Kulothungan Gunasekaran, MBBS, Swetha Murthi MBBS MPH, Narmadha Panneerselvam MBBS, Nazir Lone MD

Introduction: Even though highly vascularized, spontaneous bleeding into thyroid gland is very rare. Here we describe a case of spontaneous thyroid hemorrhage precipitated by warfarin causing acute airway obstruction.

Case: 91 y.o. female with past medical history of HTN, a-fib on warfarin, hyperthyroidism-Grave's with large goiter presented with complaints of acute SOB. She had dyspnea at both rest and on exertion. She had trouble getting air in through the throat. She noticed a pea sized area on her neck that was red and had become tender over time. She denied chest pain, hematemesis, fever, chills. She was initially able to swallow liquids but had developed difficulty later. She denied any trauma to the neck or problems with bleeding/clotting. O/E: Normotensive, tachycardic, afebrile and not tachypneic. She had right neck tenderness and enlarged thyroid with large bilateral nodules. She had bilateral air entry with diffuse rhonchi and crackles but no stridor noted. CXR showed cardiomegaly with mild pulmonary vascular prominence. Labs showed INR 2.0, BNP 3759, Troponin <0.10. CT neck showed enlarging hemorrhage in to the right lobe of the thyroid gland with some mass effect on the trachea. Her anticoagulation was reversed with vitamin K 10 mg IV and 3 units of FFP. She was initially intubated for airway protection, later extubated and discharged home for follow-up elective thyroidectomy.

Discussion: Anticoagulation with warfarin is commonly indicated in patients with atrial fibrillation to prevent thrombus formation and ischemic strokes. Administration of warfarin is often associated with the risk of bleeding. Established risk factors include advanced age, alcoholism, hypertension, stroke, cerebrovascular disease and malignancy. Intracranial hemorrhage is the most serious bleeding complication in warfarin-treated patients, but bleeding into other critical anatomical areas have also been reported. Bleeding into the thyroid gland resulting in acute airway compromise is a rare lethal condition. It can cause sudden upper airway obstruction that may require intubation. Additionally, hemorrhage into thyroid gland can result in release of preformed thyroid hormones and can cause acute hyperthyroidism. However, this is usually self-limiting and does not warrant any treatment. Bleeding into the thyroid gland is best diagnosed with CT scan. Management may include partial or total thyroidectomy as well as further treatment with thyroid hormones to prevent hypothyroidism. With an increase in number of patients receiving warfarin, physicians should be aware of these rare life threatening bleeding complications, enabling them to provide prompt recognition and management of these conditions.

## ATYPICAL CASE OF INCLUSION BODY MYOSITIS

First Author: Divya Jayakumar, MBBS, Priya Prakash, MD, Sachin Sule, MD, Amy M. Wasserman, MD

**INTRODUCTION:** Inclusion body myositis (IBM) is a condition belonging to the group of idiopathic inflammatory myopathies. IBM usually occurs as an isolated condition, but in rare cases, may be associated with other autoimmune disorders. It distinguishes itself from the other inflammatory myopathies by clinical presentation and pathological findings. Our case represents an atypical presentation of IBM which led to delay in diagnosis and management.

**CASE:** A 40 year old African American man presented to rheumatology clinic with a one year history of frequent falls, muscle pain, fatigue and weight loss. He also had progressive difficulty in climbing stairs and getting up from a seated position. He was clinically diagnosed with dermatomyositis 6 months ago, though biopsy was deferred, and was subsequently treated with high doses of prednisone without resolution of symptoms. On presentation, he was noted to have significant temporal wasting, hyperpigmented excoriated lesions on face, scalp and knuckles, but no Gottron's papules or V neck sign. Musculoskeletal examination revealed symmetric bilateral proximal muscle weakness, most prominent in the hip flexors. There was no distal muscle weakness or muscle atrophy observed. Nail fold capillaroscopy showed aberrant capillaries. Labs were remarkable for Hemoglobin of 9.7, Erythrocyte sedimentation rate of 55 and Creatine kinase of 708. ANA screen by ELISA was positive, IFA and extractable nuclear antigens were negative, as was the myositis panel. Computed tomography of chest/abdomen/pelvis did not show any evidence of malignancy. STIR MRI noted diffuse edema, predominantly in the quadriceps extensor musculature suggestive of myositis. Muscle biopsy of the quadriceps extensor was done and histopathology found perimysial and endomysial connective tissue infiltration by mononuclear inflammatory cells. There was variation in muscle fiber size with both segmental necrosis and regenerating fibers. There were rimmed vacuoles in modified Gomori trichrome stained sections and fibers with subsarcolemmal deposits. These findings were consistent with Inclusion body myositis.

**DISCUSSION:** IBM is usually rare in patients younger than 50 years, and is seen mostly in Caucasians. It classically presents with asymmetric involvement of the distal muscle groups. Our patient had symmetric involvement of proximal muscles causing diagnostic dilemma and initiation of inappropriate therapy. Delay in diagnosis occurs when clinical presentation is atypical and with overlapping features of autoimmune diseases. A positive ANA may be found in 20% of IBM patients and these patients may also have other autoimmune processes. IBM poses a therapeutic challenge to clinicians as it is not often responsive to glucocorticoids and conventional immunosuppressive therapy. It is essential to recognize the atypical presentation of diseases like IBM, which will allow redirection of therapy. This case reiterates the importance of muscle histopathology in myopathy unresponsive to immunosuppression, which will initiate early intervention and prevents functional and physical deterioration from disease.

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RECONSIDER “IDIOPATHIC” THROMBOCYTOPENIC PURPURA WHEN IT IS REFRACTORY

First Author: Tina Kapadia D.O., Adam Rothman M.D., Patricia Dharapak M.D.

Case Presentation: A 77 year old Jehovah’s Witness woman presented with one month of petechial rash, epistaxis and oral mucosal bleeding. There was no recent travel or infectious illness. She denied toxic ingestions. Exam was remarkable only for diffuse petechiae. Labwork showed new severe thrombocytopenia (2 K/UL), a normal peripheral smear, elevated LDH (754 U/L) and non reactive HIV. Abdominal CT revealed splenomegaly and 3.2 x 4.6 cm retroperitoneal lymphadenopathy. Chest CT was normal. Lymph node aspirate showed normal cytology and flow cytometry. With a working diagnosis of idiopathic thrombocytopenic purpura (ITP), she was treated with IVIG and 1mg/kg/d Prednisone with a brisk platelet response to 150 K/UL. In the following two months, despite also starting Romiplostim, the patient was readmitted twice with recurrent petechiae and thrombocytopenia (10 K/UL), each time coinciding with an attempt to taper off Prednisone. A repeat abdominal CT showed enlargement of the retroperitoneal adenopathy with necrosis and encasement of the infrarenal IVC. The patient underwent splenectomy and resection of the retroperitoneal mass. Pathology revealed non-Hodgkin’s follicular B-cell lymphoma, stage 3. Unfortunately, the patient suffered post-surgical complications requiring serial hospitalizations such that chemotherapy could not be initiated. Her thrombocytopenia remained refractory to treatment, and she ultimately died.

Discussion: Autoimmune phenomena are associated with non-Hodgkin’s lymphoma (NHL). They can precede the diagnosis of NHL but can also occur concurrently or after treatment. Refractory ITP is an uncommon initial presentation. Studies have indicated a 0.76% prevalence of ITP in patients with NHL with a male/female ratio of 1.75. Half of the cases occurred prior to the diagnosis of lymphoma. The standard incidence ratio for developing NHL when ITP is diagnosed after the age of 65 is 7.9% (95% CI 6.1-10.1). Compared with IVIG and steroids, which produce poor or transient platelet responses, chemotherapy is the best treatment in patients with NHL and ITP. The mechanism of efficacy is thought to be elimination of the IgM anti-platelet antibody producing lymphoma cells. Splenectomy is also effective. ITP preceding NHL responds well to steroids, IVIG, and/or splenectomy. As in our patient, ITP occurring at or after the diagnosis of NHL, however, is poorly responsive to these treatments. In these cases, sustained remissions of ITP are often only observed in cases of surgical removal of the lymphoma or after chemotherapy. Our case therefore highlights the importance of early recognition of the association between ITP and NHL, as it can drastically affect outcome and treatment options.

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TOO MUCH SPICE ISN'T ALWAYS NICE: A CASE OF K2 INDUCED CHEST PAIN

First Author: Tina Patel Kapadia, DO

**Case Presentation:** A 26 year old male with a prior history of asthma, DVT, and PE presented to the ER with severe, constant, pressure-like, left sided chest pain after smoking K2 the night before. The pain was associated with palpitations and shortness of breath. Initial vital signs demonstrated a heart rate of 104 bpm and a normal cardiopulmonary exam. Electrocardiogram revealed a normal sinus rhythm with early repolarization. Given the patient's prior history of clots, a CT angiogram of the chest was obtained, which was negative for PE. Laboratory results were notable for an initial troponin of 0.044 ng/mL and a urine toxicology positive for cannabinoids. The patient's troponin peaked at 0.367ng/mL. He was treated with heparin drip, aspirin, and a statin with resolution of his chest pain. Further ischemic evaluation revealed an echocardiogram with normal left ventricular function and no regional wall motion abnormalities. Ultimately, the patient left against medical advice and cardiac catheterization was not pursued.

**Discussion:** K2, also known as Spice, is a mixture of herbs and spices treated with synthetic cannabinoids. It is typically smoked for its ability produce similar effects as tetrahydrocannabinol (THC), and undetectability on urine drug screens. An estimated prevalence of K2 use in adolescents and adults in the US and UK is between 6.5% and 12.6%. Unfortunately, K2 mixtures are not uniform and their health implications are not well understood.

The detrimental effects of THC is over stimulation of the sympathetic nervous system and inhibition of the parasympathetic system leading to increased cardiac output, heart rate, and vasoconstriction. Synthetic cannabinoids often have an increased affinity for cannabinoid receptors. Combined these effects cause decreased coronary perfusion, mismatch of oxygen demand and delivery, and presumably coronary vasospasm, which can cause myocardial ischemia.

There are a wide range of presenting side effects from synthetic cannabinoids including tachycardia, agitation, hypertension, and confusion. A recent case series published in the Journal of Pediatrics (2011) showed an association of K2 usage and the development of ST elevation myocardial infarction in healthy teenagers with subsequent normal echocardiograms and coronary angiograms. Our patient is an adult, with minimal cardiac risk factors, who presented with chest pain after K2 use and had evidence of myocardial injury. This case illustrates that synthetic cannabinoids, due to their variable nature of ingredients, are capable of causing many different adverse reactions including myocardial ischemia.

Synthetic cannabinoid use has become more prevalent. The components within this drug can vary greatly, as can their side effects, which can include chest pain and myocardial ischemia. When evaluating a patient for acute onset of chest pain, it is essential that clinicians take a detailed drug history and are aware of the harmful effects of K2.

## PELLAGRA IN HIV INFECTION

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Pellagra is a disease caused by deficiency of niacin. It is characterized by 4 "D"s: diarrhea, dermatitis, dementia, and death. Although the current incidence of pellagra in the United States is unknown, it appears to be limited to sporadic cases. Rare cases of pellagra can be seen in patients with anorexia nervosa, chronic alcoholism, colitis, carcinoid, HIV disease, or with drugs interfering with vitamin B or tryptophan metabolism. We present a case of pellagra in a patient with HIV infection.

A 58 year old female presented to the ED with increasing rash on her face, neck and in distal limbs. The rash initially appeared 4 months ago but has worsened for the last 3 days. She denied fever, vomiting, weight loss, diarrhea or oral ulcers. There was no history of headache, weakness, neck stiffness or weight loss. She denied any recent travel, insect bites or sick contacts. She had a history of hypertension, diabetes, hypothyroidism, and was recently diagnosed with HIV and HCV infection. Her CD4 count was less than  $20 \times 10^6/L$  and she was scheduled to start HAART therapy. She was a current smoker but denied alcohol or IV drug use. On admission, she was afebrile and her vitals were stable. There was a symmetrical, hyperpigmented, scaly rash around her neck and sun exposed areas in her face, upper and lower extremities. The mucous membranes were not affected. Laboratory data: WBC  $3600/mm^3$ , Hb 10.9g/dl, platelet  $209 \times 10^9/L$ , creatinine 0.62 mg/dl and glucose 248mg/dl. Her LFTs were unremarkable, RPR non-reactive, and blood culture was negative. She was initially managed with systemic steroids for possible exfoliative dermatitis. However the distribution of the skin lesions raised suspicion for pellagra, and serum niacin level measured 0.02 mcg/ml (normal 0.50-8.45mcg/ml). She was started on PO niacin 50 mg three times daily, with improvement of her rash.

HIV patients can have cutaneous lesions ranging from photosensitivity, seborrheic dermatitis, pruritic papular eruptions and secondary skin infections. In addition HIV infected patients can have low circulating tryptophan concentrations despite evidence of adequate dietary intake of this essential amino acid.

Although niacin is the main exogenous substrate for NAD and NADP formation, tryptophan is the primary endogenous substrate for the synthesis of niacin. The conversion of tryptophan to nicotinamide is normally balanced by dietary intake of both tryptophan and niacin. Niacin levels need to be checked in HIV patients with photosensitivity dermatitis. Timely replacement with niacin could reverse the devastating consequences of pellagra, and improve prognosis.

A RAPIDLY PROGRESSIVE DISSEMINATED KAPOSI SARCOMA FROM IMMUNE RECONSTITUTION INFLAMMATION SYNDROME: A CONUNDRUM FOR CLINICIANS

First Author: Eileen Kim, MD

Immune Reconstitution Inflammation Syndrome related Kaposi Sarcoma (IRIS-KS), though rare, is an important event that needs to be recognized early. IRIS-KS with pulmonary manifestations is particularly associated with a high mortality rate, and an aggressive approach to clinical management of KS-associated IRIS, including concomitant use of systemic chemotherapy, may result in better outcomes. Further studies should focus on timely assessment and management of IRIS-KS development after the initiation of highly active antiretroviral therapy (HAART).

A 30 year-old MSM with a newly diagnosed HIV presented with shortness of breath and cough. He stated that he became sick 5 weeks ago with cough and mild SOB at which point he made an appointment to see his PMD where he was diagnosed with RML pneumonia and was given 5 day course of oral antibiotics. He had also developed multiple small bruise-like lesions on his bilateral lower extremities over the past few months, with several appearing over the last 2-3 weeks. Due to persistent symptoms with hypoxemia, he was sent to ER where he was found to have extensive bilateral pulmonary nodules, axillary and inguinal adenopathy, RML opacification along with bilateral pleural effusions. On admission, he was hypoxemic at 96% on 2L NC and tachycardic at 115bpm. His labs were significant for CD4 T-cell count of 4/mm<sup>3</sup>, positive AFB culture from bronchoscopy and sputums, likely MAC, positive viral culture and low level positive PCR for CMV. With several subsequent biopsies, he was diagnosed with widespread Kaposi Sarcoma (KS) with cutaneous, pulmonary and lymph node involvement with extension into retroperitoneum. He was started on HAART, including Truvada (Tenofovir and emtricitabine), Darunavir, Ritonavir and Raltegravir after tuberculosis was ruled out. As he clinically improved after the regimen with dramatic improvement in HIV viral load from 94,010 copies/mL to 118 copies/mL in 2 weeks, he was planned for chemotherapy. Yet, he subsequently had multiple re-admissions for worsening SOB and new lesions on his penis and tongue, both of which were clinically diagnosed as KS. He quickly deteriorated into acute respiratory failure and subsequently into bradycardia and PEA arrest.

The prevalence of AIDS-related KS has markedly decreased after introduction of HAART although it remains one of the most common AIDS-defining malignancies, accounting for severe morbidity and mortality. Despite efforts to diagnose HIV infection early, a substantial number of patients still present late with CD4 T-cell count < 200/mm<sup>3</sup>, increasing risk for IRIS: it is defined as a worsening of a patient's clinical condition after initiating HAART, attributable to the recovery of the immune response to viable or nonviable pathogens. Multivariate analysis identified four independent predictors of KS-associated IRIS: clinical KS at the pre-HAART visit, detectable pre-HAART plasma KS herpesvirus DNA (KSHV or HHV-8), hematocrit <30% and pre-HAART plasma HIV-1 RNA levels. Our patient had clinical manifestation of IRIS-KS with increased number of KS skin lesions and a change in consistency or ulceration, lymphedema, involvement of mucosa other than the oral mucosa. No randomized controlled trials have established the best timing for starting HAART in patients with KS, although uncontrolled data suggest that early initiation may be beneficial.

THROMBOTIC THROMBOCYTOPENIC PURPURA WITH ADAMTS13 DEFICIENCY : IS TRIMETHOPRIM-SULPHAMETHOXAZOLE THE CULPRIT?

First Author: Pallavi Bhaskar Kopparthy, MBBS, Sonny Nijjar MD, Viveksandeep Thoguluva Chandrasekar MD, Umair Masood MD and, Zachary Shepherd MD.

**Introduction:** Thrombotic thrombocytopenic purpura (TTP) is a hematological disease, with incidence of 4 to 11 cases per million per year, characterized by microangiopathic hemolytic anemia and thrombocytopenia. Although the link between ADAMTS13 deficiency and idiopathic TTP has been well-established, the role of trimethoprim-sulfamethoxazole (TMP-SMX) in the pathogenesis of TTP is not yet well elucidated. We present a case of TTP in a previously healthy 22 year old, in whom we think TMP-SMX is the culprit.

**Case:** A previously healthy, 22 year old woman, presented with progressive shortness of breath and chest pain on exertion. She had been hospitalized for a Escherichia coli urinary tract infection and treated with Trimethoprim-Sulphamethoxazole (TMP-SMX) a week prior to hospitalization. On presentation her hemoglobin was 6.7g/dL and platelet count was 14,000/uL which was an acute drop from baseline. She also had Acute Kidney Injury along with elevated total bilirubin, lactate dehydrogenase and reticulocyte count with peripheral smear showing characteristic schistocytes and basophilic stippled erythrocytes. Hematology and oncology services were consulted and in the mean time she received pooled platelet and packed RBC transfusions. However, cell lines continued to decline, even though she had adequate response initially. Direct anti-globulin test as well as blood and urine cultures were negative, transaminases and disseminated intravascular coagulation panel were normal. ADAMTS13 lab was sent and resulted positive (< 7%, RI: >70%) for a likely diagnosis of TTP. Plasmapheresis was initiated and she underwent five consecutive days of plasmapheresis. After failure to achieve complete hematological response with plasmapheresis, a four week course of Rituximab was added, resulting in complete clinical and hematological remission.

**Discussion:** Thrombotic thrombocytopenic pupura (TTP) is characterized by a pentad of symptoms including microangiopathic hemolytic anemia, thrombocytopenia, neurological abnormalities, renal failure and pyrexia. TTP can be congenital or acquired. The etiology of acquired TTP is predominantly idiopathic, but it can be associated with drugs, pregnancy, infections and malignancies. Drug associated TTP represent around 12% of all cases and they may cause TTP either by dose related toxicity and/or an immune mediated reaction. However, very limited data is available to explain the mechanism by which TMP-SMX causes TTP. Few case reports exist postulating that either a hypersensitivity reaction involving diffuse endothelial injury/vasculitis or idiosyncratic drug reaction could be the cause, but ADAMTS13 levels in these patients were either normal or never measured. Since our patient had severe ADAMTS13 we hypothesize that TMP-SMX probably induced generation of inhibitory auto antibodies, that destroy ADAMTS 13, leading to accumulation of von willebrand multimers and subsequently TTP. More investigation is definitely warranted to investigate this query.

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DIFFUSE LARGE B-CELL LYMPHOMA INVOLVING THE PROSTATE PRESENTING AS URINARY RETENTION AND HEMATURIA

First Author: Tsering Gyalpo Lama Tamang, MBBS, Second Author: William Solomon, MD, Third Author: Jonathan Garellek, MD, Fourth Author: Abhinav Chandra, MD, Department of Medicine, Maimonides Medical Center, Brooklyn, NY

**Introduction:** Lymphomas of the prostate can be primary or secondary. Primary malignant lymphomas of the prostate account for 0.09% of all prostate neoplasms and 0.1% of all non-Hodgkin's lymphomas (NHL). Because of their rarity, they are not commonly considered in the clinical and histological differential diagnosis of prostatic enlargement. Consideration of these neoplasms is important because of their aggressive behavior and poor outcome.

**Case Report:** We present a case of 49 year old male current smoker with past surgical history of cholecystectomy who presented to Emergency Department with several weeks of difficulty in urination as well as dysuria, hematuria, subjective fever, diarrhea, nausea and vomiting. On examination, the patient was afebrile with mild suprapubic tenderness and no costovertebral angle tenderness or urethral discharge. Routine laboratory tests were within normal limits except hemoglobin 11.6 and prostatic specific antigen (PSA) 0.4ng/ml. Computerized tomography scan showed a suspicious 7 x 4 cm lobulated mass below the base of the bladder, possibly originating from the superior aspect of the prostate. There was right inguinal lymph node involvement measuring 2 cm and no distant organ metastatic disease was seen. Prostatic biopsy was performed and revealed diffuse large B cell lymphoma that was immune-reactive for CD45, CD20, BCL2 and MUM1. Bone marrow biopsy was normal. Hence, stage IIE primary NHL of prostate was diagnosed. The patient was subsequently started on R-CHOP (rituximab, cyclophosphamide, doxorubicin, vincristine and prednisone) chemotherapy regimen.

**Discussion:** Primary and secondary lymphomas of the prostate are rare neoplasms with similar presenting complaints and clinical findings to benign prostatic hyperplasia (BPH) and prostatic adenocarcinoma, including urgency, frequency, occasional hematuria, and acute retention. This makes consideration and evaluation of these neoplasms challenging given the relative prevalence of the aforementioned clinical entities. Indeed, our patient was initially treated with tamsulosin and finasteride for BPH with a poor therapeutic response culminating in further evaluation with imaging, and, ultimately, tissue biopsy. Although there are no clear guidelines to the management of prostatic lymphomas, chemotherapy is the foundation for their management.

**Conclusion:** The clinical misdiagnosis of this rare tumor is common, particularly in the elderly patients because of similar presentations to BPH or prostatic cancer. Thus lymphoma of the prostate must be included in the differential diagnosis of a patient presenting with obstruction of the lower urinary tract, especially in patients with enlargement of the gland with normal PSA level and previous history of lymphoma in other sites.

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A COLLEGE STUDENT'S ACADEMIC DECLINE: EARLY SIGNS OF NMDA RECEPTOR ENCEPHALITIS

First Author: Yonathan Litwok, MD Second Authors: Bhakti Shah, MD Karen Friedman, MD Olawumi Babalola, MD

Introduction: Anti-NMDA receptor encephalitis is a rare diagnosis, which can present with a wide variety of symptoms. This etiology should be considered in the differential of a patient presenting with mental status changes. A failure to properly identify it can have lethal consequences for the patient.

Case Scenario: A 19 year old female with no medical history, pursuing an engineering degree at a competitive university, developed behavioral changes in the middle of her college semester. Two weeks prior to her arrival at our hospital she was unable to sleep more than 1-2 hours per night and began eating only one meal per day. These changes forced her to drop out of one of her classes. Ten days prior to admission she developed a migraine headache and nausea. That same day, the patient had an episode of confusion in which she was unable to recognize a close friend. Six days prior to admission the patient's mother heard her fall and found her face down on the floor. The patient was unresponsive with her eyes closed for 2-3 minutes, with no incontinence or abnormal movements. The patient was presumed to have had a seizure although workup that day at an outside hospital was negative. The patient was discharged in a confused state and her family brought her to our institution.

Workup at our institution for her encephalopathy was negative for an infectious process or toxic etiology. As the patient's confusion and delirium worsened, paraneoplastic and autoimmune etiologies were considered. CT abdomen, confirmed with vaginal ultrasound revealed a 19.2 cm complex cystic and solid mass arising from the left adnexa. The patient's serum and CSF were positive for NMDA receptor antibodies. She underwent left salpingo-oophorectomy on hospital day 7. Pathology demonstrated high grade immature teratoma. Immunotherapy was initiated on post-op day 3. The patient was treated with corticosteroids, intravenous immunoglobulin (IVIG) and plasmapheresis.

The patient's mental status remained unchanged until she was three weeks post-op. At that point the patient's speech increased and her ability to concentrate improved. The patient's insomnia began to resolve as well. Eight weeks after discharge the patient had returned to her normal mental status.

Discussion: This patient had a very rare case of NMDA receptor encephalitis secondary to an ovarian teratoma. Symptoms often include psychiatric manifestations and behavioral changes, but can also include somatic complaints and seizures. NMDA receptor encephalitis is caused by an auto-antibody which leads to inflammation of the limbic system. Prompt intervention, including removal of the tumor, IVIG, plasmapheresis, and corticosteroids can lead to full recovery, as seen in our patient.

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E CIGARETTES - 'HARMLESS VAPOR' OR 'LIFE THREATENING INHALED TOXIN'?

First Author: Yogeeta Naidu, MD Alex Croake, MD Nikhil Mehta, MD Nabeel Alqsous, MD Mohammed Jawish, MD Deerajath Lingutla MD

**Introduction:** Electronic cigarettes are devices designed to imitate traditional cigarettes and deliver nicotine via inhalation without combusting tobacco. There is a widely held perception that they are a safer alternative. Little toxicity testing has been performed to evaluate the chemical nature of vapor generated from e-cigarettes. We present a patient who developed severe parapharyngeal swelling necessitating intubation following use of a vaporizer.

**Case Description:** A 33-year-old female presented with complains of shortness of breath, pharyngeal irritation, and voice hoarseness. She was seen one day prior at an urgent care facility, treated with single dose of intravenous Clindamycin for possible Streptococcus pharyngitis, and sent home on oral Clindamycin. Her symptoms worsened over 24 hours and she presented to our emergency department. In the ambulance she received subcutaneous epinephrine, racemic epinephrine, intravenous corticosteroids, and inhaled bronchodilators for possible anaphylactic reaction. Of note, she had used new flavors “Blue Raz Cotton Candy”, “Bubblegum”, and “Pomegranate” in her vaporizer one night before symptom onset. On examination diffuse wheezing was noted, with associated stridor. CT scan of her neck showed airway narrowing secondary to extensive soft tissue swelling. She was directly admitted to the ICU for monitoring, where she received additional hydrocortisone, diphenhydramine, and subcutaneous epinephrine. An otolaryngologist was consulted, and patient underwent laryngoscopy, which showed severe laryngeal edema. She desaturated during the procedure leading to intubation under general anesthesia. She remained stable on sedation and mechanical ventilation, and was successfully extubated after 24 hours. Extensive bacterial, viral, and immunological workup was negative. The patient gradually resumed a regular diet, and was discharged home.

**Discussion:** The compounds and flavors in e-cigarettes are vast, and their use is rapidly rising, especially amongst teens and young adults. Tierney et al<sup>1</sup> identified flavor’s chemicals in e-cigarettes and that included aldehydes (eg., benzaldehyde and vanillin) which could cause respiratory irritation. The concentration of some of these chemicals is sufficiently high for inhalation exposure via ‘vaping’ to be of toxic concern.

We present a young female who had a life threatening reaction potentially due to e-cigarette vapor. It is recommended that regulatory limits are needed for levels of some of these worrisome chemicals, as well as ingredient labeling of e-cigarette flavors. Research is needed to characterize both the presence of toxic chemicals in flavorings and the potential adverse respiratory effects of exposure to e-liquids, especially flavorings.

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A CASE REPORT OF HYPERMAGNESEMIA RELATED TO BOWEL PREPARATION LEADING TO DEATH DESPITE DIALYSIS

First Author: Yaser S Nemshah, MBBS second Author Ayesha Awais, MD Third Author Nashat Rabadi, MD Fourth Author Basharat Ali, MD

A 62-year-old African American female has a past medical history significant for hypertension, hyperlipidemia, chronic kidney disease, legionnaires' disease and gastroesophageal reflux disease. The patient was taking magnesium citrate bowel prep for a planned elective colonoscopy. Despite the initial gastric upset and vomiting, she continued taking the prep and that led to worsening of her gastrointestinal symptoms. The patient apparently had no bowel movement. In the emergency room, she was significantly hypotensive and obtunded. The blood work was significant for hypermagnesemia, hypokalemia, lactic acidosis, renal insufficiency, leukocytosis with bandemia. A CT scan revealed marked distention of the stomach, possibly suggesting gastric outlet obstruction or possibly secondary to large amount of fluid intake for the colonoscopy preparation. There was dilated small bowel distally, likely secondary to ileus. There was also evidence of pulmonary infiltrates. The patient was intubated and admitted to the intensive care unit with hypovolemic and septic shock secondary to probable aspiration pneumonia. That was managed with generous fluid resuscitation, vasopressors, gastric decompression and antibiotics. She had also elevated troponins and was treated as non-ST elevation myocardial infarction with heparin drip. The initial magnesium level was 4.2 that jumped to 6.9 within 11 hours. Initial management of hypermagnesemia with bumex, in addition to fluids, resulted in mild improvement of magnesium level. However, the follow up magnesium level has gotten worse to 9.2 that necessitated dialysis in the context of intractable shock. Despite subsequent sessions of dialysis and empiric treatment for pneumonia and myocardial infarction, the patient continued to deteriorate and was subsequently placed on comfort care measures as per the family request. The autopsy confirmed the presence of lung consolidations and coronary artery disease but failed to determine the primary cause of death. That led us to the conclusion that worsening hypermagnesemia due to ongoing absorption of bowel prep was the primary cause of death.

## NATIVE VALVE ENDOCARDITIS DUE TO VEILLONELLA SPECIES SUCCESSFULLY TREATED WITH A CEPHALOSPORIN

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**Introduction:** *Veillonella* species are anaerobic, gram negative cocci that are part of the normal mouth, gastrointestinal and urogenital flora in humans. *Veillonella* species are fastidious bacteria that have been isolated from skin and respiratory tract infections and rarely have been implicated in serious infections like meningitis, endocarditis and osteomyelitis.

**Case Summary:** A 76 year-old woman with medical history of diabetes mellitus, hypertension, chronic hepatitis C with cirrhosis and chronic kidney disease, presented to the hospital with fever, vomiting and generalized weakness for 3 days. Patient was recently admitted to the hospital and treated for urinary tract infection. On admission patient's vital signs were stable. Physical exam was significant for grade 3/6 systolic murmur in the right second intercostal space and bilateral grade 2 pitting pedal edema. The remainder of the examination was unremarkable.

Laboratory studies showed thrombocytopenia and renal failure. A transthoracic echocardiogram showed a mobile structure on anterior mitral valve leaflet measuring 0.9 cm suggestive of vegetation. Empiric therapy with vancomycin and piperacillin-tazobactam was started. On day 6, the blood culture drawn at admission grew *Veillonella* species. Patient was also diagnosed with urinary tract infection caused by extended spectrum beta lactamase producing *Escherichia coli*, therefore piperacillin-tazobactam was changed to meropenem. A trans-esophageal echocardiogram confirmed a 1.2 X 0.4 cm echo dense structure attached to the LV side of the anterior mitral leaflet. The isolate was reported sensitive to penicillin, cephalosporin antibiotics and resistant to metronidazole. At this time, vancomycin was discontinued and meropenem was changed to ceftriaxone. Repeat blood cultures remained negative. The hospital course was complicated by diarrhea due to *Clostridium difficile* which was treated with metronidazole with clinical improvement. She was discharged home after 10 days of inpatient therapy and completed 4 weeks of IV ceftriaxone at home without any adverse events. She was re-evaluated in the clinic after completion of treatment and repeat blood cultures remained negative.

**Discussion:** There are seven reported cases of endocarditis with *Veillonella* species. Four cases involved prosthetic valves and three affected native valves. In addition to antibiotic therapy, four patients required surgical intervention. Penicillin, ampicillin, gentamicin and metronidazole (alone and in-combination) have been used for treatment. Metronidazole alone may not be effective due to emerging resistance as exhibited by the isolate in our case. Only one patient among the previously reported cases was treated with a first generation cephalosporin for the initial 2 weeks followed by 6 months of oral penicillin. Although penicillin is considered the antibiotic of choice for this rare infection, we report the first case of successful treatment of endocarditis due to *Veillonella* species with once daily ceftriaxone.

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UROTHELIAL AND RENAL CELL CARCINOMAS PRESENTING AS PARANEOPLASTIC FLORID CUTANEOUS PAPILOMATOSIS

First Author: Yazan Samhouri, MD Co-authors: Gukol Samudrala, MD, Makiko Ban-Hoefen

**Introduction** Florid cutaneous papillomatosis (FCP) is an obligate paraneoplastic syndrome. We report a rare case of FCP that led to the simultaneous diagnosis of three different malignancies in a patient with a Phosphatase and Tensin (PTEN) germline mutation.

**Case description** A 55 year old gentleman with hypertension, developed hematuria and diffuse skin eruptions over a month's time. The patient has extensive family history of malignancies. On physical exam, he had innumerable fleshy white papillomas over the face, neck, and arms. Both conjunctivae were erythematous. Perianal skin was papillomatous with velvety hyper-pigmented area. Complete blood count and chemistries were normal. CT scan of chest, abdomen and pelvis revealed enhancing lesions within the right collecting system and right retroperitoneal lymphadenopathy, in addition to a 4.8 cm mass in the upper pole of the left kidney. Biopsies revealed right kidney urothelial carcinoma, right retroperitoneal metastatic urothelial carcinoma and left kidney renal papillary neoplasm. The conjunctival biopsy showed carcinoma in-situ. He was started on neoadjuvant chemotherapy with Gemcitabine and Cisplatin, followed by right nephroureterectomy and retroperitoneal lymph node dissection. The rash improved significantly, repeat imaging revealed improvement in lymphadenopathy, and stable size of the left renal mass. Unfortunately, the papillomas reappeared three months after surgery. CT scan of the abdomen and pelvis showed new retroperitoneal lymphadenopathy. Core biopsy of these lymph nodes confirmed urothelial carcinoma.

**Discussion** FCP is a rare condition, in which there are less than thirty cases reported in literature so far. It is twice as common in men as in women. It presents as eruptions of 1-3 mm diameter wart-like nodules on the hands, feet, and face. It is most commonly associated with gastric adenocarcinoma. Interestingly, our patient was simultaneously diagnosed with three different cancers, namely, urothelial carcinoma, renal cell papillary neoplasm and conjunctival carcinoma in-situ. This finding led to genetic testing which revealed PTEN germline mutation.

FCP is an obligate cutaneous paraneoplastic syndromes, similar to malignant acanthosis nigricans, and the sign of Leser-Trélat. These should be viewed as part of a continuum because they can manifest together and share similar pathogenesis. The pathophysiology of FCP is not well understood, it was supposed that the underlying malignancy induces FCP by secreting a factor similar to human epidermal growth factor or transforming growth factor-alpha.

FCP usually follows the course of the underlying malignancy. It improves after surgical resection of the cancer or chemotherapy treatment. Topical 5-fluorouracil demonstrated effectiveness as palliative treatment.

This case adds to the sparse literature on FCP and highlights their importance as neoplastic markers. The sudden onset of similar diffuse cutaneous lesions should elicit further workup to evaluate for an underlying cancer.

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TUNA IMPLICATED IN HISTAMINE OVERDOSE.

First Author: Maroun Sfeir, MD

45-year-old male patient with no significant past medical history admitted with acute onset of erythema and warmth of face, ears and neck, associated with progressively worsening occipital throbbing headache, mild difficulty breathing and palpitations. He also noticed numerous non itchy large red patches on his chest, back and upper arms. He then had 2 episodes of watery diarrhea that occurred about 2 hours after onset of other symptoms.

Upon further interrogation, patient mentioned that his symptoms began about one hour after eating dinner with 2 friends in a restaurant where he had a salad with tuna burger and one glass of red wine. None of his friends ate tuna and none developed symptoms.

On physical exam he looked uncomfortable but not in distress. He was afebrile, tachycardic at 110 beats/minute with a normal respiration at 16 breaths/minutes and a normal blood pressure of 105/55 mmHg. He had conjunctival injection, with diffuse erythema and warmth of the face, ears, neck and upper arms. His chest was clear on auscultation. His cardiac exam revealed tachycardia. His abdominal exam was normal.

His blood tests showed normal complete blood count and comprehensive metabolic panel. Chest x-ray was clear. All his symptoms and signs resolved within 6 hours after he was given 25 mg of IV diphenhydramine.

Based on the clinical picture, the main diagnosis was “Histamine Toxicity from Fish”, which was previously known as scombroid fish poisoning because the first implicated fish species were Scombridae. Histamine fish poisoning is now more appropriate since non-scombroid fish might be implicated. It is among the most common toxicities related to fish ingestion, constituting almost 40% of all seafood-related food-borne illnesses. It directly relates to improper preservation and inadequate refrigeration of the fish. Histidine decarboxylase found in *Escherichia coli*, *Morganella morganii*, and *Klebsiella* species, converts histidine, present in fish tissue, to histamine. Without adequate cooling, these bacteria multiply, increasing the histidine-to-histamine conversion rate and raising histamine levels. In fish left at room temperature, the histamine concentration rapidly increases, reaching toxic concentrations within 12 hours. Patients with histamine fish toxicity have a good prognosis. In most instances, illness severity can be well controlled with antihistamines.

In conclusion, Histamine Toxicity from Fish results from bacterial proliferation in inadequately refrigerated fish. This report emphasizes even more the importance of infection control measures and proper handling of fish to prevent toxicity.

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ETANERCEPT-INDUCED MYOSITIS: DO WE HAVE TO STOP IT? A SURPRISING OUTCOME.

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Anti-tumor necrosis factor alpha (anti-TNF) agents are widely used to treat a variety of rheumatic and autoimmune diseases including psoriasis. In rare cases, these agents can induce inflammatory myositis, which is usually managed by cessation of the anti-TNF agents and may require additional therapy with corticosteroids and immunosuppressive medications to manage the underlying disease.

Our patient presented here is a 47 year old man diagnosed with severe psoriasis and psoriatic arthritis at age 40 who was refractory to methotrexate, leflunomide and systemic steroids. He had recurrent skin and arthritic flares requiring frequent hospitalizations and intravenous steroid therapy. Etanercept 50mg subcutaneously once weekly was started, resulting in nearly complete resolution of psoriatic plaques and marked improvement of arthritis during a short period of time. Six months after initiation of anti-TNF therapy, patient presented complaining of generalized myalgia, progressive weakness in upper and lower extremities and unstable gait of 2 weeks duration. On examination, vitals were normal, musculoskeletal exam revealed synovitis of left wrist and boggiess of right hand proximal interphalangeal joints. Upper and lower proximal muscle strength was decreased to 3/5 and patient had an unstable gait. Deep tendon reflexes were normal. Laboratory tests showed elevated transaminases with aspartate transaminase of 173 U/L, alanine transaminase 497 U/L. The serum creatine kinase was found to be 5666 U/L, lactate dehydrogenase 1265 U/L, C-reactive protein 51.69 mg/dL, ESR 112 mm/hr, and antibody to JO-1 was negative. A muscle biopsy was performed and showed perifascicular muscle fiber atrophy with perimysial inflammatory activity. This presentation was suggestive of Etanercept-induced myositis. The patient was advised to stop Etanercept and was started on prednisone 60 mg once a day. His muscle strength fully recovered, gait improved with no imbalance and serum creatine kinase normalized within three months. Despite our recommendation to stop Etanercept, patient continued using it due to his concern of experiencing a possible flare of psoriasis. Oral steroids were subsequently tapered off in the next couple of months and patient has remained stable on Etanercept.

Etanercept is used in the treatment of moderate to severe psoriasis and psoriatic arthritis. There have been rare cases of anti-TNF-induced inflammatory myositis. Cases described in the literature usually have a good outcome after discontinuation of anti-TNF, however a challenge remains to treat the underlying auto-immune disorder, psoriasis or psoriatic arthritis once the anti-TNF therapy is discontinued. This is the first description to our knowledge of an anti-TNF-induced myositis responding favorably to a short course of steroids despite continuation of anti-TNF therapy.

## HYPOTHERMIA IN HODGKIN'S LYMPHOMA- AN UNUSUAL PHENOMENON

First Author: KARTHIK VADAMALAI, MD Second Author: DIPEKKA SONI MD MENTOR: ROBIN REID MD

**Introduction:** Hodgkin's lymphoma classically presents with lymphadenopathy and B symptoms (Fever, night sweats and weight loss). Hypothermia is rare in this disease and there are about 17 reported cases in literature. Here, we report an elderly woman diagnosed with Hodgkin's lymphoma, who developed hypothermia after a single dose of naproxen.

**Case:** 89-year-old African-American female presented to the emergency department with complaints of generalized fatigue, dizziness and low-grade fever with night sweats for two months. On admission, temperature- 39.2°C, BP- 130/80mmhg, PR- 89/min. Intermittent bradycardia and hypotension was noted. Work up for this fever of unknown origin including CBC, urine analysis, basic metabolic panel, and liver function tests were normal, except for elevated CRP and ferritin. Blood, urine and CSF cultures were negative for bacteria and fungi. Extensive radiological investigations including full-body magnetic resonance imaging (MRI) proved to be non-diagnostic. In the hospital, she continued to have high-grade fevers of 40-42°C, which was managed symptomatically with acetaminophen. Due to persistent fevers, naproxen was given, which resulted in a drop of temperature from 41.8°C to 32.8°C in few hours and was managed with warming blankets. An oncologic evaluation led to whole body PET/CT that showed positive lymph nodes in cervical area. Excisional biopsy revealed classical Hodgkin's lymphoma cells with CD-15 and CD-30 positivity and no sub-classification was possible. Chemotherapy with AVD (Adriamycin, Vinblastine, Dacarbazine) excluding Bleomycin was initiated. Unfortunately, treatment course was complicated by tumor lysis syndrome, neutropenic fever and septic shock, leading to multi-organ failure and death.

**Discussion:** Hypothermia in Hodgkin's lymphoma is a rare entity and also considered as a poor prognostic factor. Out of the 17 cases reported, 12 patients had hypothermia with initiation of chemotherapy, one with acetaminophen, one with prednisone and three had no clear precipitating cause. 7 out of 10 patients with reported prognosis died. In our case, hypothermia was secondary to naproxen use. Various theories have been proposed for this hypothermia, including para-neoplastic autonomic neuropathy, direct tumor involvement of the hypothalamus, chemotherapeutic drug use, altered thermoregulation from cytokines and pyrogens from the use of antipyretics. In our index patient, MRI brain did not show tumor involvement of hypothalamus, the episode of hypothermia occurred before initiation of chemotherapy and patient had hypotension with no beat-to-beat variability in EKG, supporting the cause of autonomic dysfunction as possible etiology. While definite etiology of hypothermia in Hodgkin's lymphoma remains unclear, physicians should be aware of this rare presentation and look for signs of autonomic dysfunction.

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ATLL-ASSOCIATED HYPERCALCEMIA IN A PATIENT OF CARIBBEAN DESCENT.

First Author: Sangeetha Venugopal, MD

Hypercalcemia of malignancy is a frequent cause of inpatient hypercalcemia and may be associated with hypercalcemic crisis. Here we present a case of Adult T-cell Leukemia/Lymphoma (ATLL) presenting as severe hypercalcemia in an elderly patient with Human T cell Lymphoma/Leukemia Virus-1 (HTLV-1) infection.

80 year old Jamaican female with past medical history of secondary hyperparathyroidism due to chronic kidney disease and other medical comorbidities was admitted for evaluation of lethargy. She was found to be in hypercalcemic crisis with albumin-corrected calcium level of 16.1 mg/dl (8.5 -10.5 mg/dL). The patient was initially managed with aggressive intravenous hydration and calcitonin. The serum calcium proved to be refractory and she was later treated with pamidronate which significantly lowered the calcium levels.

The initial serological studies revealed an elevated PTHrP: 59 pg/ml (14-27 pg/mL), low PTH: 8.0 pg/mL (10-65 pg/mL) and normal 25-OH vitamin D and its metabolites suggesting a non-PTH mediated cause for hypercalcemia. Skeletal survey showed healed non ossifying fibroma in left femur while whole body imaging did not reveal any mass lesions that could potentially cause hypercalcemia. Given that the patient was an immigrant from Jamaica who presented with hypercalcemic crisis, she was evaluated for hematologic malignancy and was found to be positive for HTLV-1. Flow cytometry revealed premature lymphoid cells positive for CD4, CD25, and negative CD7 indicative of ATLL which is associated with a remote infection of HTLV-1. High dose glucocorticoids was added to the medical regimen which resulted in resolution of hypercalcemia.

Due to the patient's overall poor functional status, chemotherapy was not administered. She was discharged from the hospital in stable condition.

This patient posed a diagnostic dilemma as she did not have any of the usual presenting features of acute ATLL in the form of generalized lymphadenopathy or hepatosplenomegaly.

Although hypercalcemia occurs in more than 50% of patients with acute ATLL, initial presentation with hypercalcemic crisis is rare. The humoral hypercalcemia in ATLL is caused by mechanisms that act synergistically, involving osteoclast differentiation. Some of the well-studied mechanisms include receptor activator-kB ligand, osteoprotegerin, PTHrP and proinflammatory cytokines. This case illustrates that unexplained hypercalcemia in a person of Caribbean descent should raise the suspicion for HTLV-1 infection and ATLL.

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## LACERATION OF THE LIVER AND INFERIOR VENACAVA COMPLICATING CARDIOPULMONARY RESUSCITATION

First Author: Sangeetha Venugopal, MD

External cardiac compressions administered during cardiopulmonary resuscitation (CPR) has been known to be associated with complications of varying severity. Most of the complications involve the chest and the thoracic wall. Less frequently, the viscera and vascular structures are involved.

49 year old woman was admitted to the critical care unit for altered mental status following possible Valproic acid overdose. She had hyperammonemia with evolving abnormal liver tests & thrombocytopenia. The patient subsequently developed seizures followed by bradycardic asystolic arrest. She had four sequential cardiac arrests with eventual return to spontaneous circulation following CPR and requiring multiple pressors for hemodynamic support. Serial blood work revealed the patient had a precipitous drop in her hemoglobin from 13.6 g/dl to 3.4 g/dl. Bedside Sonogram revealed fluid below diaphragm, in Morrison's pouch and both flanks with suspected bleed into the peritoneal cavity. Bedside focused echo revealed no hemopericardium. Chest X-ray showed minimally displaced fractures of fourth and sixth left ribs. Patient underwent emergency exploratory laparotomy to identify the source of bleed. Upon exploration she was noted to have caudate lobe laceration with active bleeding. A Pringle maneuver was performed and the right upper quadrant was packed. Patient was subsequently transferred to quaternary care facility for definitive repair. On second exploratory laparotomy it was noted that there were lacerations of the caudate lobe of the liver, the umbilical ligament, and retro hepatic venacava. Hemostasis was achieved by removing the left caudate lobe of liver and retro hepatic venacava. Patient's post operative course was complicated by multiple organ failure. The patient eventually succumbed after advance directives were put in place by family and further aggressive care was withdrawn.

Review of literature shows that liver injuries occurred in 0.6% of the cardiac arrest patients, most commonly involving the left lobe of the liver and almost always in the setting of coagulopathy (Meron et al). Among the vascular injuries that occur secondary to CPR, only 0.6% of the injuries involved the venacava (Miller et al). However there is paucity of data regarding simultaneous injury of liver and venacava

This case demonstrates that there should be heightened suspicion for visceral bleeding in patients requiring escalating hemodynamic support post CPR and Focused ultrasound should be a part of initial evaluation to identify, if there is any source of bleed.

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## MANIFESTATION OF DIFFUSE COLONIC POLYPOSIS AS LACTOCOCCUS ENDOCARDITIS

Taylor Bazemore MD, Kahli Zietlow MD

**INTRODUCTION:** Although generally considered an opportunistic pathogen in humans, *Lactococcus* is known to be responsible for systemic infections including bacteremia, peritonitis, and osteomyelitis. Infective endocarditis from *Lactococcus* is a relatively rare clinical entity and most often occurs in association with prosthetic heart valves.

**CASE:** A 45-year-old-male with hepatitis C, unspecified anemia, and recent Bentall repair of an aortic root aneurysm presented with two months of generalized malaise that he described to be similar to a prior episode of anemia. On arrival, he was febrile to 39.5 and found to have a IV/VI systolic murmur, with labs significant for WBC of 12.9 and hemoglobin of 10.2.

Admission blood cultures grew *Lactococcus garviae* and repeat cultures remained positive for the following three days. CT of the abdomen/pelvis showed splenic and left renal infarcts concerning for embolic phenomena. Both trans-thoracic and trans-esophageal echocardiograms demonstrated thickened valvular leaflets and peri-aortic thickening but no vegetations. The patient did not have echocardiographic evidence of intra-cardiac infection and met only four minor Duke's Criteria for endocarditis: predisposing heart valve, fever, persistently positive blood cultures, and embolic phenomena. Nevertheless, his clinical presentation was concerning for endocarditis, and he was treated with a six-week course of ceftriaxone and gentamicin.

Readmission for persistent, symptomatic anemia soon after the patient's discharge prompted a colonoscopy that revealed innumerable, 3-12mm polyps throughout the colon, concerning for Familial Adenomatous Polyposis or some other polyposis syndrome. Pathology demonstrated tubular and tubulo-villous adenomas with high-grade dysplasia. The patient ultimately underwent total colectomy.

Following completion of antibiotic therapy, surveillance chest CT demonstrated partial aortic valve dehiscence with surrounding fluid collection concerning for pseudo-aneurysm. The patient underwent replacement of the aortic root and prosthetic valve. Intra-operatively, there was near-complete dehiscence of the valve conduit from the annulus and significant destruction of the aortic annulus with large pseudo-aneurysm, confirmed on pathology. These operative findings are indicative of intra-cardiac infection, satisfying the remaining Duke's Criteria and confirming the diagnosis of *Lactococcus* endocarditis.

**DISCUSSION:** Several prior reports describe *Lactococcus* infections in association with underlying gastrointestinal disease, including cases of patients with colonic polyps or colorectal cancer, as well as patients with non-neoplastic lesions including diverticular and ulcerative disease. In our patient, the diagnosis of *Lactococcus* endocarditis proved to be a harbinger of a more serious, underlying neoplastic disease. We raise the question of whether the diagnosis of *Lactococcus* endocarditis should evoke suspicion and encourage evaluation for gastrointestinal malignancy. By establishing the association between this infection and the risk underlying colorectal carcinoma, expedient colonoscopy in patients with *Lactococcus* endocarditis may allow for earlier diagnosis and treatment of cancer in patients with occult disease.

### SYNCOPE AND SUBSEQUENT CRANIAL NERVE III PALSY IN AN INCARCERATED PATIENT

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*Mycobacterium tuberculosis* (MTB) infection of the central nervous system (CNS) can mimic other neurologic pathology, including stroke and vasculitis. MTB infection must remain on the differential for any patient with risk factors for MTB infection who presents with non-specific neurological findings.

A 43-year-old incarcerated male with a medical history including systemic lupus erythematosus (SLE) and deep vein thrombosis (DVT) presented with right eye ptosis following a syncopal event the previous day. The patient reported five weeks of arthralgias, joint swelling, morning stiffness, non-exertional chest pain, alopecia, rash, dysuria, fatigue, anorexia, weakness, and headache. The patient's medications included Cellcept, Plaquenil, prednisone, and warfarin. His prednisone dose had been recently increased from 10 mg to 15 mg daily to treat an SLE flare. Subsequently, the patient developed fevers, chills, and night sweats. One day before presentation, the patient had a syncopal episode and hit his head. Upon evaluation the following day at the prison's medical facility, he was found to have right eye ptosis and a dilated, fixed right pupil.

Upon presentation to the hospital, the patient was febrile to 38.7°C and tachypneic, with a respiratory rate of 25; blood pressure and heart rate were normal. He was somnolent and complaining of a severe headache. Physical exam demonstrated a right cranial nerve III palsy and diffuse left sided weakness. The remainder of his exam was normal. The patient had a white cell count of 15.4, an INR of 5.3, a c-reactive protein level of 4.1, and an erythrocyte sedimentation rate of 18. Chest x-ray revealed a diffuse reticular-nodular pattern. Results from brain MRI were suggestive of a medium vessel vasculitis without evidence of thromboembolic stroke. Cerebrospinal fluid (CSF) analysis showed 128 nucleated cells with 92% neutrophils and 3% lymphocytes, glucose <20 mg/dL, protein >120 mg/dL with a negative gram stain. Ultimately, acid fast bacillus (AFB) cultures and MTB nucleic acid amplification were positive from sputum and CSF. Further review of the patient's medical record revealed that he had received monitored treatment for latent MTB ten years prior to his presentation, indicating that his meningeal MTB infection was due to reactivation. The patient was treated with rifampin, isoniazid, pyrazinamide, and ethambutol therapy, as well as moxifloxacin for improved CNS penetration. He was ultimately discharged back into the federal prison system for continued management.

This case illustrates the importance of screening for MTB infection in high risk populations as well as including MTB infection on the differential for patients presenting with non-specific findings. In this patient, the initial differential diagnosis was broad and included thromboembolic and hemorrhagic stroke, lupus cerebritis, and other CNS infections. Careful history and physical exam, along with thorough laboratory and radiologic evaluation revealed the diagnosis and aided in expeditious treatment.

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AN ATYPICAL PRESENTATION OF STRONGYLOIDES INFECTION IN A 92 YEAR OLD VETERAN OF WWII

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Strongyloidiasis is an infectious disease caused by a nematode parasite, *Strongyloides stercoralis* resides in the small intestine followed by continuous autoinfection as a result of larvae production by adult worms within the small intestine. Subsequent hyper-infection syndrome is possible involving the pulmonary and gastrointestinal systems along with further dissemination to other organs.

A 92 year old Caucasian male presented to the VA hospital complaining of 3 months of weight loss and anorexia. During WWII, he spent two years in the south pacific as a prisoner of war and after escaping, lived in the jungles of New Guinea for 15 months prior to being rescued. The patient presented at age 92 with a past medical history of hypertension accompanied by Stage III chronic kidney disease and chronic iron deficiency anemia. His chief complaint was worsening appetite and continuous weight loss over a three month period from 72Kg to 63Kg. The patient denied nausea, vomiting, or diarrhea, chest pain, shortness of breath, palpitations, lower extremity edema and denied any fever, chills, or cough. Due to a finding of persistent eosinophilia (up to 8%), with an absolute eosinophil count of over 300 cells/mm<sup>3</sup> coupled with his history of having been in the endemic region of south Asia, serological testing for strongyloidiasis was carried and revealed a positive Strongyloid titer. Consequently, the patient was started on Ivermectin treatment at a dose of 0.2mg/kg. One month later, Strongyloid titer was negative although eosinophilia values remained high (0.34K/ul and 5.3% for EOS # and EOS %, respectively). Over the next two months these values decreased significantly and eventually EOS # reached a normal range. During that time, the patient's appetite improved and his weight returned to his prior baseline.

Strongyloidiasis is endemic in several developing countries in sub-Saharan Africa, Southeast Asia and Latin America. Although it is rare in developed countries, it is present in some localized areas, such as the Appalachian region of the United States where prevalence of the disease ranges between 2.5 to 4%. Physicians should also be conscious of certain high risk sub-populations like veterans, individuals from endemic regions, and those that are immune-suppressed. This case highlights the need for healthcare providers to be conscious of the possible presence of otherwise asymptomatic parasitic infections that even decades after initial infection can evolve into potentially fatal forms of hyper-infection.

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ARRHYTHMOGENIC RIGHT VENTRICULAR CARDIOMYOPATHY AS A CAUSE OF SUDDEN CARDIAC DEATH IN A YOUNG ADULT

First Author: Ruchi Jain, DO

A 38 year old male with no significant past medical history presented to the emergency department after experiencing a witnessed out of hospital cardiac arrest. The patient's coworkers obtained an automated external defibrillator (AED) and shock was advised. The patient received one shock. Emergency medical services (EMS) arrived and the patient underwent two rounds of cardiopulmonary resuscitation and received one dose of epinephrine before return of spontaneous circulation was obtained. Initial electrocardiogram (ECG) by EMS showed ST elevations in the inferior leads (II, III and aVF). Upon arrival to the emergency department, the patient's vital signs were within normal limits. His initial labs were unremarkable and his urine drug screen was negative. He was taken for emergent left heart catheterization which showed noncritical stenoses in the coronary arteries. No intervention was done. The decision was made to initiate therapeutic hypothermia and the patient was cooled for 24 hours. After rewarming, the patient was alert and was extubated. He reported no recent history of chest pain, palpitations, dizziness or lightheadedness. The decision was made to obtain a cardiac magnetic resonance imaging (MRI) study which revealed regional right ventricular dysfunction with hypokinesis. The quantitative right ventricle ejection fraction was 30%. These findings met MRI criteria for ARVC. An implantable cardioverter defibrillator (ICD) was placed prior to discharge. The patient was to follow up with cardiology for genetic testing.

ARVC is an under-diagnosed cause of ventricular arrhythmia and sudden cardiac death in young adults. The average age of diagnosis is 30 and gene mutations have been found in up to 40% of those affected. Microscopically, there is replacement of right ventricular myocardium with fibrous and fatty tissue. The scarred myocardium is a nidus for ventricular arrhythmias. Exercise can precipitate arrhythmias and patients are encouraged to avoid strenuous activity. ECG is a poor screening tool and can be normal in up to 50% of patients. ICD implantation is appropriate for secondary prevention in patients who have had a sustained ventricular arrhythmia or who have suffered sudden cardiac death. It may be appropriate for primary prevention in certain patients deemed to be high risk although no precise indications have been defined in this population and guidelines are based on expert opinion.

## RIBOFLAVIN DEFICIENCY MANIFESTING AS RECURRENT KETOACIDOSIS

First Author: Kahli Zietlow, MD Keithara Davis Cara O'Brien, MD

We present an 18-year-old male with no significant past medical history who developed recurrent episodes of ketoacidosis. His symptoms during these episodes were malaise, fatigue, nausea, and vomiting. He had three such presentations in three months and endorsed approximately 20 lbs of weight loss during this time period. He was a college freshman and worked in an organic chemistry lab. He had two healthy siblings and no contributing family history. On his third presentation to the emergency room, he was tachycardic with a heart rate of 134. He was thin but not cachectic. His physical exam was otherwise unremarkable. Blood work was notable for pH of 7.15 on venous blood gas, bicarbonate of 11, beta-hydroxybutyrate of 6.44, and a normal blood glucose. His anion gap was 19 and osmolar gap was 36. He had undetectable serum ethanol levels. A volatile acid screen did not detect methanol, ethylene glycol, or isopropyl alcohol.

Given his recurrent presentations and potential access to organic solvents at school, we were suspicious of surreptitious ingestion. Because he was underweight, we also considered an eating disorder with intermittent episodes of fasting ketoacidosis. However, the patient adamantly denied any ingestions, intentional starvation, or bingeing behavior. We did note that the patient primarily ate fried potatoes throughout his admission. Although we were concerned for a possible factitious disorder, we consulted medical genetics to exclude inborn errors of metabolism. Medical genetics recommended a plasma acyl-carnitine profile, which demonstrated marked elevations of long-chain species suggestive of multiple acyl-CoA dehydrogenase deficiency or a mitochondrial disorder. Given these results, we put the patient through a fast. After 24 hours of fasting, he became symptomatic and his blood work demonstrated worsening metabolic derangements. He was started on empiric riboflavin, which is often used to treat inborn errors of metabolism, as it is a cofactor in multiple pathways. The patient's episodes resolved after riboflavin supplementation and subsequent genetic testing for underlying genetic disorders was negative. He was ultimately diagnosed with riboflavin deficiency secondary to his diet, which consisted almost exclusively of potato chips and hash browns.

Riboflavin (vitamin B2) is ubiquitous in the Western diet, though notably unavailable in many preparations of fried potatoes. Isolated riboflavin deficiency is extremely rare, and a well-defined clinical syndrome does not exist. The differential diagnosis for ketoacidosis includes diabetic ketoacidosis, starvation, alcohol, other ingestions, and inborn errors of metabolism. Our patient presented with recurrent ketoacidosis of uncertain origin. To our knowledge, this is the first case of riboflavin deficiency presenting as recurrent ketoacidosis.

DIAGNOSING PULMONIC VALVE INFECTIVE ENDOCARDITIS WITH THE USE OF POSITRON EMISSION TOMOGRAPHY-COMPUTED TOMOGRAPHY SCAN

First Author: Kahli Zietlow, MD Taylor Bazemore, MD

Pulmonic valve infective endocarditis (IE) is extremely rare, estimated to account for less than 2% of all cases of IE. It is also diagnostically challenging, as visualization of pulmonic valve vegetations can be limited with both transthoracic and transesophageal echocardiography (TTE and TEE) due to the anterior position of the pulmonic valve. Recent studies show positron emission tomography-computed tomography (PET-CT) imaging may hold promising diagnostic value.

A 50-year-old man with a history of Tetralogy of Fallot, repaired with pulmonary valvuloplasty and ventricular septal defect patch, followed by surgical bioprosthetic pulmonic valve replacement, presented with several days of fevers and malaise. He was hemodynamically stable, had no signs of decompensated heart failure, and had no stigmata of endocarditis on exam. Initial blood cultures grew methicillin-susceptible *Staphylococcus aureus* (MSSA). Chest CT showed subpleural nodules suggestive of septic pulmonary emboli. Neither TTE nor TEE showed valvular regurgitation, abscess, or vegetation, but the patient had definite IE by the modified Duke's criteria for endocarditis. Despite appropriate antibiotic treatment, he experienced persistent, daily fevers associated with rigors and leukocytosis.

We initiated an extensive workup for fever of unknown origin. Review of his travel and social history was unremarkable. Basic laboratory workup and review of peripheral blood smear were normal. Infectious workup, including blood cultures, HIV testing, and *Clostridium difficile* stool testing, was negative, with the exception of the above mentioned MSSA. Imaging studies, including brain and spinal magnetic resonance imaging (MRI) and CT of the chest and abdomen, were normal. A tagged-white blood cell scan demonstrated intense tracer uptake in lungs, consistent with presumed septic emboli visualized on prior. All non-essential medications were sequentially discontinued to exclude drug fever. Almost one month into his hospitalization, we performed a PET-CT scan, which demonstrated circumferential fluorodeoxyglucose avidity involving the right ventricular outflow tract (RVOT), concerning for prosthetic valve IE.

The patient subsequently underwent sternotomy that revealed extensive vegetative involvement of his RVOT and prosthetic pulmonic valve, which was partially dehisced. Polymerase chain reaction of the material detected *Staphylococcus aureus*. He underwent redo pulmonic valve replacement and reconstruction of RVOT and completed a six-week antibiotic course, with excellent recovery.

Given the technical limitations of standard imaging modalities in the diagnosis of pulmonic valve IE, PET-CT imaging may reveal findings that can guide management of patients with pulmonic valve endocarditis or infection of intracardiac prosthetic material. Although PET-CT is not part of the traditional workup for IE, it can be a useful imaging modality when there is a high index of suspicion for IE with negative echocardiography findings.

IMMUNOSUPPRESSION FOR KIDNEY TRANSPLANTATION LEADS TO POST-TRANSPLANT DIABETES. IS TACROLIMUS A DOUBLE-EDGED SWORD?

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Introduction: New-onset diabetes mellitus after transplantation (NODAT) has been recently reported to occur in 13.4% of patients after solid organ transplantation, with a higher incidence in patients receiving tacrolimus than cyclosporine (16.6% vs. 9.8%). Diabetes ketoacidosis however is a rare, but dangerous initial presentation of such adverse effect.

Case Description: A 44-year-old Caucasian male, with no past medical history of diabetes mellitus presented with diabetes ketoacidosis, three months after receiving a deceased-donor kidney transplant for end stage renal disease secondary to autosomal dominant polycystic kidney disease. His immunosuppressive regimen included tacrolimus, mycophenolate sodium and low dose prednisone (5 mg). Patient presented to the emergency department with nausea and polyuria. He did not have family history of diabetes mellitus. Physical exam was unremarkable except for mild overweight. Laboratory work-up revealed hyperglycemia, high anion gap metabolic acidosis, significant ketosis and ketonuria. Glycated hemoglobin (A1C) was 9.8% compared to 4.8%, 30 days post-transplant. Tacrolimus trough level was therapeutic. Glutamic acid decarboxylase (GAD-65) autoantibodies were negative. The patient received intravenous fluids, a bolus of intravenous insulin followed by continuous insulin infusion which was soon switched to subcutaneous insulin. Daily insulin requirements were approximately 40 units. He was educated about his new diagnosis and discharged on diabetic diet and insulin therapy with appropriate follow-up.

Discussion: Maintenance immunosuppressive therapy is essential to prevent rejection in renal transplant recipients. Calcineurin inhibitors play an integral role in immunosuppressive regimens, with tacrolimus being the preferred agent over cyclosporine, as several studies showed lower incidence of acute rejections with its use. Both calcineurin inhibitors cause toxicity to pancreatic islet beta cells and may directly affect transcriptional regulation of insulin expression. Some evidence suggests tacrolimus causes more severe swelling-vacuolization, endoplasmic reticulum stress and apoptosis of pancreatic islet beta cells. Tacrolimus's diabetogenic effects threaten the health and longevity of the allograft by predisposing the recipients to microvascular and macrovascular diabetes complications which consequently reduce allograft survival. Tacrolimus's contribution to new-onset diabetes ketoacidosis, a consequence of insulin deficiency in Type 1 diabetes mellitus, seems to be a rarely reported adverse event, yet adds to the accumulating evidence of the reduced allograft survival being observed since its introduction as the immunosuppressant of choice. The development of diabetes mellitus with ketoacidosis in patients on therapeutic tacrolimus levels, with no risk factors for diabetes, highlights the need for alternative immunosuppressive agents that won't compromise patients' allografts long-term survival at the expense of inducing a devastating chronic disease that patients and physicians would end up regretting.

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## A SHOCKING FINDING FOLLOWING COLON PERFORATION

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AL amyloidosis is an extremely rare disease in young adult patients, with 99% of all cases diagnosed in those over 40 years old. Additionally, bowel perforation caused by amyloidosis is a very rare complication of the disease. We present a case of a 33 year old man with bowel perforation secondary to AL amyloidosis.

A 33 year-old man presented to the emergency department with sharp waxing and waning left lower quadrant abdominal pain. Medical history included progressive chronic kidney disease with initiation of peritoneal dialysis only 3 months earlier. After extensive laboratory workup, including an unremarkable serum and urine protein electrophoresis, the kidney disease was presumed to be due to chronic uncontrolled hypertension, so a kidney biopsy had not been obtained. Upon admission he denied fevers, chills, easy bruising, melena, hematochezia, nausea, vomiting, or constipation. The abdomen was soft, nondistended and exhibited tenderness with palpation in the left lower quadrant with rebound. Computed tomography (CT) showed descending and sigmoid colon diverticula with surrounding inflammation. Antibiotics were initiated and the peritoneal dialysis catheter was removed. Peritoneal fluid cultures would grow *Streptococcus sanguinis* and multi-drug resistant *Escherichia coli*. An exploratory laparoscopy failed to show gross perforation. Over the next two weeks his condition deteriorated and repeat CT revealed free air and suggested colonic perforation. Subsequently, he required a total colectomy and ileostomy. Pathological studies from the resected bowel showed colonic perforation with extensive amyloid deposits with apple green birefringence upon polarization. A bone marrow biopsy showed plasma cell predominance with excess production of kappa light chains, confirming AL amyloidosis.

Amyloidosis is a multisystem disease that frequently involves the GI system but is typically asymptomatic. When symptomatic, weight loss, GI bleeding, and diarrhea are most often seen. GI tract perforation secondary to amyloidosis was first reported in 1960, and there have only been a handful of cases reported since then, with presentation in a patient this young almost never seen. In our patient, amyloidosis was not considered prior to the colon perforation largely due to his age. The case illustrates the relevance of including amyloidosis in the differential of a wide range of syndromes, even in young adult patients. More broadly, it serves as a reminder that even in the setting of a seemingly obvious explanation for a clinical syndrome, one must still maintain an inquisitive viewpoint and consider alternative diagnoses and testing.

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ENDOBONCHIAL CARTILAGE RUPTURE: A RARE CAUSE OF LOBAR COLLAPSE

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Emphysematous endobronchial cartilage rupture is a rare clinical condition. To our knowledge there are no reported cases in the medical literature. The only reported cases of bronchial cartilage rupture were clinical scenarios related to traumatic causes including blunt and penetrating chest trauma and double lumen endotracheal intubation. We report a case of spontaneous endobronchial cartilage rupture leading to respiratory failure.

A case of 62-year-old male with 40 pack-years history of smoking presented to our emergency department with the chief complaint of sudden onset shortness of breath for five hours. The patient stated that he did have shortness of breath and cough at baseline limiting his activities of daily life but this time dyspnea was sudden onset and severe. He denied any trauma to chest, fever, chills, chest pain, or recent upper respiratory tract infection.

On physical examination, the patient had tachycardia, tachypnea, with labored breaths and prolonged expiration and rhonchi. Arterial blood gas showed evidence of hypoxia and hypercapnia. He was started on noninvasive positive pressure ventilation (NIPPV) in emergency department for severe dyspnea. Chest X-ray showed severe hyperinflation and flattening of both of diaphragm with vague suggestion of a small density in the right lung field. D-Dimer was 0.74mcg/ml. Given his sudden onset shortness of breath and clear lung fields and elevated D-Dimer a CT-angiogram was done to rule out pulmonary embolism which showed severe emphysema and near complete collapse of right middle lobe (image 1). No extra luminal mass was noticed, however dense material within the bronchus was identified suggestive of mucus plus or intraluminal mass. A bronchoscopy was done to rule out malignancy given patient's extensive smoking history. Bronchoscopy revealed partial right middle lobe atelectasis and rupture of cartilage at sub carina of medial segment of right middle lobe. There was no evidence of endobronchial mass or extrinsic compression (image 2). A biopsy was aborted due to possibility of injury to lung parenchymal tissue supported by ruptured cartilage. Patient's shortness of breath improved with conservative management and supportive care.

Emphysematous endobronchial cartilage rupture is a rare clinical condition, which can present in patients with severe emphysema with sudden onset shortness of breath. It is proposed that severe hyperinflation of lungs due to emphysema can lead to rupture of endobronchial cartilage and subsequent lobar collapse as in our case. Bronchoscopic findings include finding a collapsed lung lobe and a visible ruptured endobronchial cartilage.

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REVERSIBLE POSTERIOR LEUKOENCEPHALOPATHY SYNDROME IN AN ADULT PATIENT WITH SICKLE CELL ANEMIA

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**Introduction:** Reversible posterior leukoencephalopathy syndrome (RPLS) is a clinical syndrome marked by headaches, visual disturbances, altered mental status and seizures with characteristic findings on neuroimaging. MRI of the brain often reveals symmetric white matter edema in the parieto-occipital regions, though lesions of the cortex can occur with more severe cases. Only a few cases have been reported in sickle cell patients. This particular case describes an adult sickle cell patient with ascending cholangitis that subsequently developed RPLS.

**Case:** A 37 year-old African American female with history of homozygous sickle cell disease was hospitalized for acute cholecystitis and generalized sickle cell pain crisis. Her hospital course was complicated by the development of ascending cholangitis, while she was on broad spectrum intravenous antibiotics. On hospital day ten her mentation and coherence worsened and her blood pressure increased to 183/87. She experienced two subsequent seizures necessitating intubation and transfer to the intensive care unit. Initial CT scan of her brain was normal, however MRI of the brain showed marked multifocal signal abnormalities throughout the cortex and subcortical white matter of both cerebral and cerebellar hemispheres consistent with severe RPLS. Lumbar puncture revealed yellow tinged cerebrospinal fluid, elevated protein of 127 mg/dL, a white blood cell count of 61 mm<sup>3</sup>, and a red blood cell count of 1595 mm<sup>3</sup>. Prior to exchange transfusion her hemoglobin S was 53.7% by electrophoresis. She received urgent exchange transfusion and percutaneous drainage of her gall bladder. Blood cultures remained negative, but gall bladder culture grew coagulase negative *Staphylococcus*. Within 24 hours, her mental status returned to baseline and she was extubated. Repeat MRI 3 weeks later showed radiographical resolution.

**Discussion:** RPLS exact mechanism remains unknown, but it is associated with severe, acute hypertension. There are currently two competing theories regarding the pathophysiology behind RPLS. The favored is the hypertension/hyperperfusion theory in which acute hypertension leads to cerebral autoregulatory failure, subsequent transient cerebral hyperperfusion and then white matter edema. A competing theory states systemic toxicity and endothelial dysfunction leads to vasoconstriction, vasogenic edema, and ischemia particularly in watershed regions. In this particular case the altered mentation, sickle cell pain crisis and ascending cholangitis preceded the development of RPLS, favoring the systemic toxicity theory. Treatment for RPLS is typically supportive and is often reversible, with resolution of radiographic changes in weeks to months after the insult. In this case, treatment of this patient's underlying sickle cell crisis and ascending cholangitis lead to rapid improvement in her mental status.

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HENOCH-SCHONLEIN PURPURA (HSP) PRESENTING AS A LARGE PERICARDIAL EFFUSION IN AN ADULT

First Author: Atif K Hassan, MD Second Author: Romesa Sajjad

**INTRODUCTION:** Henoch-Schonlein Purpura (HSP) is the most common form of systemic vasculitis in children. It is characterized by a tetrad of palpable purpura, arthralgia, abdominal pain and renal disease. It rarely presents with significant pericardial disease, especially in adults, and is a rare entity with only a few reported cases.

**CASE DESCRIPTION:** A 49 year old Male presented with a two month history of progressive dyspnea, polyarthralgia, fatigue, and skin rash. Physical exam showed cardiac dullness beyond the apical point of maximal impulse, elbow joints with limited range of motion and swelling, periungual erythema and palpable purpura over his extremities. He was found to have a large pericardial effusion on an echocardiogram with respiratory variation requiring drainage. Laboratory evaluation revealed markedly elevated C-Reactive Protein of 225. Biopsy of the skin purpura was consistent with IgA leukocytoclastic vasculitis with intense deposition of IgA within the superficial dermal blood vessel walls. Pericardial fluid was consistent with acute inflammation with several neutrophils but no growth on culture. The patient was started on high dose steroids with marked improvement in his symptoms including a decreased pericardial effusion. A repeat echocardiogram was obtained which showed only trivial pericardial effusion with no signs of tamponade.

**DISCUSSION:** HSP is a rather common disease in children with systemic hypersensitivity vasculitis in the skin and other organs including the kidneys. It rarely involves the myocardium or pericardium and is even rarer to present as a large pericardial effusion, especially in an adult. HSP presenting as pericardial disease has only been reported in a few cases. Although the pericardial involvement of Henoch-Schonlein Purpura seen in this patient is rare, any collagen vascular disease can present with pericardial effusion and might respond to appropriate anti-inflammatory therapies including steroids if recognized promptly.

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PHENTERMINE: MIRACLE WEIGHT LOSS DRUG OR SINISTER STIMULANT?

First Author: Priyanka Jain, MD Second Author: Anandhi Sudhakaran, MD Third Author: Kevin Ranum, MD

Posterior reversible encephalopathy syndrome (PRES) is a rare condition of unclear etiology that manifests with headaches, visual disturbances, altered consciousness and seizures. While it is known to be associated with acute severe hypertension (HTN), immunosuppressive therapy and a variety of electrolyte and hematologic disturbances, there have been few reports of the association between PRES and phenyl-tertiary-butylamine, also known as Phentermine.

A 66 yo F with a past medical history significant for diabetes mellitus type 2, HTN and morbid obesity on Phentermine, was admitted to the hospital after being found unconscious by her husband. She then developed tonic-clonic seizures and had to be intubated for airway protection. On admission, her BP was found to be 235/97. She was immediately started on a cardene drip with good response. Neurology was consulted. Pt was started on keppra for treatment of seizures. MRI brain revealed increased signal intensity on FLAIR & T2-weighted sequences within the supratentorial white matter with a minor degree in the pons as well as subtle increased signal intensity on FLAIR sequence involving portions of the cortex near the vertex of the head in the right frontal, right parietal and left frontal region. EEG was also done which revealed a diffusely slow background with a mixture of delta and theta frequencies consistent with moderate encephalopathy as well as intermittent right parietal discharges indicating cortical irritability. Based on presentation and clinical findings, it was determined pt likely had PRES. She was extubated successfully and was seizure-free throughout her stay. Good blood pressure control was achieved with calcium-channel blockers, angiotensin-receptor blockers and diuretics. Pt was discharged in stable condition to an extended care facility with strict instructions to discontinue use of Phentermine and to follow up with neurology in 4-6 weeks.

While PRES has an unclear etiology, it does have many associated conditions. This case presents itself as a diagnosis of exclusion as this particular patient did not have majority of these conditions, leading to the idea that use of Phentermine could be related to PRES. As this drug is used widely across the country, it is important to recognize that the use of Phentermine could have significantly debilitating effects.

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## AN INTERESTING CAUSE FOR ACUTE PANCREATITIS

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The patient is a 32-year-old male with history significant for type I diabetes mellitus that presented with nausea and severe epigastric pain for the previous 24 hours. Additional history included previous cholecystectomy secondary to biliary colic with the presence of cholelithiasis, no recent trauma to the abdomen, arachnid bites, viral illnesses, minimal alcohol intake, and no tobacco use. Medication included home insulin glargine and insulin lispro with no recent changes to prescription medications.

Vitals were stable with physical examination revealing epigastric tenderness. Laboratory results were significant for a lipase of 4670 U/L with CT imaging of the abdomen showing pancreatic inflammation confirming the diagnosis of acute pancreatitis. Both the liver function panel and lipid panel were within normal limits. A right upper quadrant ultrasound was consistent with cholecystectomy and no biliary tract dilation.

Upon further questioning, the patient noted taking L-Arginine supplement 1000 mg daily for recent erectile dysfunction for the past 3 weeks. A brief literature search yielded the use of L-Arginine in mice to induce acute pancreatitis.

The patient improved with standard management of acute pancreatitis. Upon discharge, the patient was instructed to discontinue L-Arginine and has since not had a recurrent episode of acute pancreatitis.

L-Arginine is marketed commercially as a supplement, which may alleviate conditions such as erectile dysfunction, male infertility, along with other conditions. However, L-arginine has been used, at higher doses, as a major model to induce acute pancreatitis in rats for animal studies.

The exact mechanism of L-arginine induced pancreatitis is unknown, but previous studies have postulated that L-arginine may increase nitric oxide effects in the pancreas through several distinct pathways: Alteration of pancreatic water and ion balance as well as zymogen activation and differentiation within the pancreas; direct DNA damage from reactive oxygen species; and changes in micro-circulation. Although the levels used to experimentally induce pancreatitis in rats, approximately 500 mg per 100 g animal weight, is much greater than the amount most supplements recommend, 1-3 g/day, there is one prior documentation in medical literature reporting an adolescent male who experienced acute pancreatitis after ingesting L-arginine 500 mg daily for five months with the aims of increasing muscle mass.

Though our patient had only been using L-arginine for about 3 weeks, it is reasonable to consider L-arginine supplementation as a possible cause for his episode of acute pancreatitis. This case serves to highlight the broad differential for acute pancreatitis etiology and to consider drug-induced etiology especially if a patient was started on medications or supplements whose effects on human physiology have yet to be fully elucidated.

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NON-TYPHOIDAL SALMONELLA BACTEREMIA - A FATAL EXTRAINTESTINAL COMPLICATION OF ENDOVASCULAR SURGICAL PROCEDURES

First Author: Paul Obeng-Okyere, DO

Non-typhoidal salmonella is a food-borne pathogen commonly known to cause intestinal infections. However, it can present as a complication of endovascular surgical procedures with fatal outcomes in patients with multiple co-morbid conditions and in the immunocompromised.

A 70 year old man with a 75 pack year smoking history, uncontrolled type 2 diabetes and severe peripheral vascular disease presented to ICU after being found unresponsive at a skilled nursing facility. Patient was in septic shock with multi-organ failure upon admission requiring mechanical ventilation and hemodynamic support with levophed and dopamine. Physical exam was particularly notable for purulent drainage from a 2 cm incision site at the right groin. CT abdomen/pelvis showed soft tissue gas in the right groin with no evidence lobulated abscess. Blood cultures grew positive for non-typhoidal salmonella bacteremia. Patient was initially empirically started on vancomycin and zosyn but was subsequently switched to intravenous cefepime mono-therapy due to its superior CNS penetration after sensitivities were known. About a month prior to presentation, patient had underwent a thoracic aortic stent placement after presenting with atypical chest pain symptoms and CT of the chest had shown a penetrating atherosclerotic ulcer of the descending thoracic aorta. Patient's postoperative course was complicated by punctate cerebrovascular accidents in the right cerebellum which prolonged his hospitalization. Additionally, there was also a 1.7 cm lobulated right middle lobe mass with small mediastinal adenopathy suggestive of malignancy on the same chest CT a month prior. Further evaluation of the lung mass with CT-fine needle aspiration was deferred due to the decision to first perform the repair of the thoracic ulcer. With the positive blood cultures on his new admission, a collaborative discussion with vascular surgery and infectious disease specialists did conclude that the likely source of infection was the endovascular stent which had been placed a month prior. A recommendation was then made to remove the stent. However, the patient was deemed too critically ill to tolerate the procedure with a further decision to defer any surgery until the patient recovered from the septic shock. Patient unfortunately clinically deteriorated and subsequently passed away on the sixth day of admission.

This case demonstrates the fatal course of non-typhoidal salmonella bacteremia especially in patients who undergo high risk endovascular procedures. Although cefepime provides excellent broad-spectrum coverage with adequate CNS penetration to treat non-typhoidal salmonella, its effectiveness to reverse the course of systemic bacteremia is limited in the setting of multiple systemic comorbid conditions. Immunocompromise from malignancy and type 2 diabetes has been particularly shown to have the most adverse prognostic factor for non-typhoidal salmonella bacteremia.

## DISSEMINATED NOCARDIOSIS IN SYSTEMIC LUPUS ERYTHEMATOSUS

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**Introduction:** Systemic Lupus Erythromatosis (SLE) is a multisystem autoimmune disease with various clinical manifestations that is associated with increased mortality related to renal failure and increased risk of infections. Disseminated Nocardiosis has been reported in SLE patients receiving heavy immunosuppressive therapy, but has also in immunocompetent subjects. The site of primary nocardia infection is usually the lung, which manifest as pulmonary cavitation. Cutaneous disease, brain, eye and thyroid involvement have also been reported. Brain abscess formation is noted to be the most common sequela of disseminated nocardiosis. Our report describes a unique case of Disseminated Nocardiosis causing hydrocephalus in a patient with SLE who has been using small dose prednisone and azathioprine only. CNS infection related to nocardiosis is associated with high mortality that may exceed 80% with a delay in instituting proper therapy.

**Case Presentation :** Our patient is a 24-year old female with a history of SLE complicated by end-stage renal disease for over 5 years related to lupus nephritis. She was using stable daily doses of prednisone 15 mg and azathioprine 50 mg. The patient presented with dyspnea and cough for 1 week, and new subcutaneous nodules. The patient's physical exam findings included decreased air entry and bronchial breath sounds at the left lung base. Neck exam revealed left-sided asymmetry and fullness. Her skin exam disclosed tender and mobile subcutaneous nodules ranging in size from 1 to 2 cm. Her white blood cells count (WBC) was 23,500/mm<sup>3</sup>, with 90% neutrophils. Her C-reactive protein was elevated at 207 mg/l (normal <7 mg/l), erythrocyte sedimentation rate of 148 mm/hr (normal <20mm/hr). Computed tomography (CT) scan of the lungs demonstrated multiple cavitory lesions in the lungs. CT scan of the neck demonstrated fluid collection in the left thyroid. Drainage and fluid culture of the thyroid fluid collection revealed gram-positive branching bacilli consistent with *Nocardia otitidiscaviarum*. The patient was treated by a regimen that include amikacin, meropenem, and trimethoprim-sulfamethoxazole for 8 weeks. One month later, the patient developed confusion where an MRI of the brain showed new multiple ring enhancing lesions and hydrocephalus. A diagnostic lumbar puncture was performed. Cerebral spinal fluid analysis showed WBC 30,581 (normal <5), 97% segmented neutrophils, Glucose 0, and protein 348 mg/dl (normal 15-60 mg/dl). CSF gram stain and cultures demonstrated the same species of Nocardia.

**Discussion:** Our case highlights a rare but serious complication of disseminated nocardiosis in SLE, which is brain involvement complicated by hydrocephalus. In general, brain involvement of nocardiosis is associated with high mortality. Early identification of such opportunistic infections, especially in patients with SLE or patients receiving immunosuppressive therapy is paramount.

## DERMATOMYOSITIS FOLLOWING ZOLEDRONIC ACID RE-DOSING IN A 59-YEAR OLD FEMALE

First Author: Nicole Droz (Samuel), MD Other authors: Ali R. Abtahi, DO Stephanie D. Mathew, DO and Craig A. Rohan, MD

**Introduction:** Zoledronic Acid (ZA) is a nitrogen-bisphosphonate used in the treatment of osteoporosis. Generally well tolerated, the most common adverse reactions following infusion include pyrexia, myalgias, headache and arthralgias. To date, dermatomyositis-like reactions have not been definitively linked to ZA, however, there exists two reports in the literature describing heliotropic rash with proximal muscle weakness occurring post ZA administration. Here, we describe a third case of acute onset dermatomyositis following ZA re-dosing.

**Case Description:** A 59 year old female with a past medical history of gastro-esophageal reflux, hyperlipidemia, hypothyroidism and osteoporosis underwent her first treatment with ZA at her primary care clinic with no ill effects post-infusion. Thirteen months later, the patient returned to her family physician for re-administration. Within 24 hours, the patient developed swelling around her eyes and diffuse rash involving her forehead. She was prescribed an anti-histamine and topical hydrocortisone cream but did not have improvement in symptoms.

Upon further evaluation, the patient reported evolving fatigue and increased difficulty with activities such as standing from seated position and combing her hair. She denied changes in soaps, detergents, lotions, had no unusual environmental contacts and denied any new medications. She had no prior history of cancer, immunodeficiencies, or autoimmune disorders.

On physical exam, she was found to have a prominent heliotropic non-pustular rash with additional distribution across her shoulders, back, and upper arms (positive shawl sign). She was also found to have periungual erythema without evidence of Gottron's papules. Additionally, patient demonstrated decreased proximal muscle strength involving all four extremities.

Laboratory evaluation revealed positive anti-nuclear and anti-SSA antibodies; CPK, aldolase, and alpha fetal protein levels were within normal limits. Computed tomography of the chest, abdomen, and pelvis were negative for interstitial lung disease or malignancy. Electromyography performed in the right deltoid and thoracic paraspinalis muscles were significant for increased insertional activity, early recruitment, and sharp waves. A muscle biopsy taken from the left deltoid revealed perifascicular and perivascular invasion with CD4+ T-cells, moderate CD8+ T-cells, and few NK cells consistent with new onset dermatomyositis. Given the timing of symptoms following ZA administration, it was thought the DM was likely drug induced.

The patient was initially placed on a prednisone taper then was transitioned to immunosuppressive maintenance regimen of methotrexate, low dose prednisone and hydroxychloroquine. The patient had improvement in her baseline muscle strength but had persistence of her heliotropic rash.

**Discussion:** Drug-related dermatomyositis is a well-documented, yet poorly understood phenomena that has been associated with over 40 substances including chelating agents, NSAIDs, anti-infectious and anti-tumor drugs, statins, and the BCG vaccine. Of these agents, the most frequently documented cases have occurred in association with hydroxyurea and D-penicillamine. Only two other cases of ZA induced dermatomyositis have been reported in the literature. Analogous to our patient, both reports were found to have positive ANA titres, positive pathological findings on skin or muscle biopsy, and negative workups for malignancy. Muscular and cutaneous manifestations of dermatomyositis improved with immunosuppressive therapy and withdraw of the eliciting drug in all cases.

PSYCHOSIS: DON'T MISS ANTI-NDMAR ENCEPHALITIS

First Author: Saniya Shaikh, DO Secondary Authors: Sean Murney, DO and Kim Jordan, MD

Anti-N-methyl-D-aspartate receptor (NMDAR) encephalitis is a rare autoimmune syndrome characterized by IgG antibodies against NMDA receptors in neural tissue. Multiple neoplastic and infectious causes are reported, but the vast majority of cases occur secondary to ovarian teratomas with NMDAR antibody production. Limbic brain receptor damage and impaired glutamatergic neurotransmission result in significant psychotic and neurologic symptoms. Patients can be misdiagnosed with primary psychosis, or undergo prolonged hospitalization and extensive testing before diagnosis is made.

**Case:** A 37-year-old female presented with flu-like symptoms followed by confusion, agitation, and marked personality changes with childlike and hypersexual behavior. Though alert, speech was incomprehensible. Initial studies were normal, including brain CT and MRI, EEG, TSH, drug screen, chemistries, and serum ammonia. A lumbar puncture showed lymphocytic pleocytosis, but CSF infectious workup was negative. Extensive additional testing was normal, including ANA, ESR, RF, RPR, anti-TPO, B12, ANCA, HIV, anti-SSA, paraneoplastic panel, and copper level. Psychiatry consultants started dexmedetomidine for severe agitation, but recommended repeat testing, suggesting that symptoms were likely caused by an underlying medical condition. Lumbar puncture and CSF studies were unchanged, repeat brain MRI showed bi-hemispheric hyperintense flair signal, and repeat EEG noted bitemporal slowing (4Hz). Anti-NMDAR testing was also sent, but by two weeks, she was obtunded and IV steroids were started. Anti-NMDAR encephalitis was finally diagnosed 20 days after presentation when anti-NMDAR testing returned positive. Because of poor response to IV steroids and IVIG, she was switched to rituximab and prednisone. Transvaginal ultrasound revealed a 2.6cm left ovarian mass, confirmed by pelvic MRI. Left salpingo-oophorectomy was completed and pathology confirmed a mature teratoma. Symptoms improved and she was discharged to a rehabilitation facility six days after surgery. Unfortunately, she was readmitted after 2 months for recurrent agitation and attempts to flee the nursing home. NMDAR antibody testing was again positive. Repeat transvaginal ultrasound showed 3mm right ovarian echogenicity, but the patient elected to forego further surgical intervention. Interestingly, the echogenicity was resolved on follow-up ultrasound. Symptoms are currently controlled with antipsychotic therapy.

**DISCUSSION:** Anti-NMDAR encephalitis is most common in reproductive age females, but can affect any age and occur in men. Clinical course is characterized by four phases: prodromal, psychotic, unresponsive, and hyperkinetic phase with autonomic instability. Underlying tumors are found in 59% of patients, most commonly ovarian teratomas. Immunotherapy and tumor removal significantly improve outcomes and decrease recurrences. First-line treatment includes pulse-dose corticosteroids, plasma exchange, and IVIG. Cyclophosphamide and rituximab are used for refractory symptoms. With treatment, 80% of patients recover fully or have minor sequelae; however, relapse occurs in 20% and mortality rates range from 7-25%.

**Teaching Point:** Clinicians should consider anti-NMDAR encephalitis in young females presenting with altered mentation without obvious cause on routine medical testing and imaging.

A RARE CASE OF CLOSTRIDIUM SEPTICUM MYCOTIC AORTIC ARCH ANEURYSM WITH ENDOCARDITIS

First Author: Shoaib Shakeel Second Author: Jeffrey Weinstein

**Introduction:** Clostridium septicum is a gram positive anaerobe usually associated with colorectal malignancy. In rare cases it can cause mycotic aortic aneurysms and even endocarditis. This case presents a patient with recurrent C. septicum bacteremia and found to have both aortic valve endocarditis and mycotic aortic aneurysm.

**Case description:** The patient is an 86 year-old Caucasian male who presented to a community hospital with fever, generalized weakness and a 20lb. weight loss over 2 months. He has a past medical history significant for untreated chronic lymphocytic leukemia and type II diabetes. On admission he had leukocytosis with WBC 41 with 84% lymphocytes, 16% neutrophils and troponin elevation of 2.95. His blood cultures were positive for C. septicum. A TEE showed a 0.6cm vegetation on the aortic valve. Since C. septicum has an association with typhlitis and colorectal cancer, a CT abdomen was performed. This demonstrated a 4.7cm cecal mass. The patient underwent surgical evaluation as the mass was a likely source for the bacteremia and possibly malignant. A right hemi-colectomy was performed and pathology showed the mass to be a tubular adenoma. The patient was discharged with 4 weeks of IV ertapenem. He would re-present to the hospital with recurrent fevers, diarrhea and weakness after 1 month. Repeat blood cultures were again positive for C. septicum and a stool C. difficile PCR was positive. Repeat TEE no longer showed aortic valve vegetations. He was transitioned from IV meropenem to oral metronidazole as his symptoms improved. The patient did well at rehab, but then began to have hoarseness in his voice 1 month later. He was seen by an ENT physician and a CT of the neck revealed 7.0x6.2cm ascending aortic aneurysm. Subsequent CT chest angiogram confirmed a dissecting ascending aortic aneurysm with probable contained rupture of the aortic arch. The patient underwent aortic arch repair with elective CABG. During surgery, he was found to have a severely inflamed pulsatile mass encompassing the ascending aorta with pus throughout the pseudoaneurysm, pericardium and great vessels. Unfortunately, the patient expired intraoperatively from exsanguination.

**Discussion:** Most cases of mycotic aortic aneurysm are caused by Staphylococcus aureus but C. septicum has been documented in only a handful of reports. In addition, C. septicum endocarditis has been documented in only 2 other cases. This patient had both infections likely due to the large cecal tubular adenoma rather than adenocarcinoma. His mycotic aneurysm unfortunately was not detected on TEE only one month prior to his development of hoarseness. Despite receiving aggressive IV antibiotics, he continued to have recurrent bacteremia. Surgical intervention was attempted but the extensive nature of the infection made it a difficult repair.

## CONGENITAL LONG QT UNMASKED BY AMOXICILLIN?

First Author: Snehal Shrivastava, MD, MS, MBBS\*\* Justin Ugwu, MD, MPH\*\*, Malavika Balachandran MD\*\*, Shrinivas Katragadda MD\*

**Introduction:** Prolongation of QTc interval on EKG is a feared complication of many medications including some antibiotics. QTc prolongation with use of amoxicillin has not been reported so far. We report a case of a relatively healthy 40 year old lady who had initial episode of V-fib arrest due to QTc prolongation, unmasked by Amoxicillin use in the setting of underlying asymptomatic congenital type3 Long QT syndrome.

**Case Description:** Our patient is a relatively healthy 40 year old lady who experienced V-fib arrest during sleep. EMS found her in V-fib and was successfully cardioverted with 200J of direct current, with return of spontaneous circulation and EMS monitor displaying normal sinus rhythm. Magnesium was also administered by EMS for her initial resuscitation, she was intubated on site and transported to our ER.

In the ER, amiodarone drip was started and EKG showed prolongation of QTc interval. Magnesium was re-administered with subsequent normalization of the QTc. Upon transfer to the ICU, Propofol was used for sedation which led to next incidence of QTc prolongation in the 602 range. Magnesium was re-administered, propofol and amiodarone both stopped. All labs and CT scans came back unremarkable with no obvious trigger for her initial V-fib episode.

History obtained upon stabilization of patient revealed that patient was not taking any medications or supplements except for amoxicillin which was begun 6 days ago for a tooth infection. On the second day of amoxicillin consumption, patient noted intermittent palpitations, and tiredness which she attributed to the dental infection she was fighting.

Patient's past medical history was significant only for colectomy 3 years ago for recurrent episodes of abdominal pain and diverticulitis. EKG done at that time as a part of pre-operative work up was normal.

**Discussion:** The Food and Drug Administration's mandatory evaluation of QT/QTc prolongation and proarrhythmic potential for non-antiarrhythmic drugs came into effect in 2005. As a result, the pro-arrhythmic effect of molecules approved by FDA prior to 2005 is not well known. The ability of such molecules to unmask arrhythmias in individuals with dormant congenital arrhythmias is also unknown. Amoxicillin is one such molecule.

After evaluating all patient factors, including her history, EKGs (especially t-wave morphology), lab and imaging data, we concluded that the patient likely had congenital channelopathy causing type-3 long QT syndrome, leading to V-fib arrest, triggered by the potentiating effect of Amoxicillin.

Further observations and studies are indicated to support or refute this hypothesis.



## A RARE MECHANISM OF ACUTE CORONARY SYNDROME

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**Introduction:** Acute coronary syndrome (ACS) is caused by intravascular or extravascular coronary blood flow obstruction. Intravascular blood flow interruption by rupture of an atherosclerotic plaque is the most common mechanism. Uncommon causes include coronary artery dissection, coronary artery spasm due to drugs like cocaine. Rarely, extrinsic compression of coronary vasculature has been reported in metastatic cancers, thoracic outlet syndrome, bronchogenic cysts and dilated main pulmonary artery.

We report a case of extrinsic compression of left main coronary artery (LMCA) by dilated pulmonary artery (PA) in a patient with a history of severe pulmonary hypertension that presented as non-ST segment elevation myocardial infarction (NSTEMI) to increase awareness and highlight the diagnosis and management of this rare but important condition.

**Case Presentation:** 79-year-old male with history of end stage renal disease, severe pulmonary hypertension and chronic obstructive pulmonary disease presented with generalized weakness and progressively worsening shortness of breath for two weeks.

Vitals at presentation showed blood pressure 121/64 mmHg, respiratory rate 18/min and pulse rate 95 beats/min. Physical examination showed an old gentleman in mild respiratory distress, jugular venous distention, crackles at bilateral lung bases, +1 bilateral pedal edema and a normal neurological examination.

Electrocardiography (EKG) was consistent with ST elevation in AVR, ST depression in Lead I, II, V2-V6. CKMB and Troponin I were elevated. An emergent left heart catheterization was performed which showed non-obstructive coronary artery disease with 40% stenosis in distal left main coronary artery that was unchanged from a previous left heart catheterization 1 year ago and 40% stenosis of proximal right coronary artery. Intravascular ultrasonography (IVUS) revealed minimal luminal area 10.5mm of the distal left main coronary artery and 14mm of proximal left coronary artery. IVUS showed that the proximal left coronary artery had pulsatile compression by a dilated main pulmonary artery.

A diagnosis of non-obstructive coronary artery disease with extrinsic compression of proximal left main coronary artery by dilated and high-pressure pulmonary artery was made. The patient responded well to vasodilator therapy for pulmonary hypertension with phosphodiesterase inhibitor and prostacyclin analog treprostinil.

**Discussion:** Extrinsic compression of coronary arteries is a rare mechanism of ACS. Traditional management includes aggressive measures like coronary artery bypass graft surgery or angiography and stenting. However, our experience with conservative management of pulmonary hypertension by vasodilator therapy resulted in a good outcome.



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CONSEQUENCES OF OVERLOOKING ADVANCED CARE PREFERENCES: A CASE REPORT

Christina Molumby, MD (Associate); Oliver Cerqueira, DO

**Introduction:** One of the most important decisions that a patient can make when being admitted to the hospital is that of his/her code status. Failure to provide care that is congruent with the patient's desired goals of care has both ethical and financial implications. This case report illustrates the financial impact of noncompliance with a patient's advanced care planning preferences.

**Case Description:** A 61-year-old woman with a history of uncontrolled diabetes and tobacco abuse presented with gangrene on her right first and second toes and second degree burns on her left foot acquired after sleeping on a heating pad. On admission, the patient signed paperwork expressing her wish to not be resuscitated and/or intubated in the setting of cardiopulmonary arrest. Vascular surgery consultation was requested due to the presence of critical limb ischemia. She underwent aorto-bifemoral bypass surgery one week after admission and was admitted to the ICU postoperatively.

While in the ICU, the patient developed respiratory decompensation and was intubated despite her known code status. She was extubated, re-intubated, and extubated again over the next several days. During this time, she had multiple procedures with associated complications including: right heart catheterization, gastrostomy tube placement & replacement, surgical wound debridement with wound vac placement, exploratory laparotomy, and long-term IV antibiotics. She was eventually discharged to a long-term acute care facility (LTAC) for continued management.

The patient failed to progress with curative measures at the LTAC and was ultimately discharged to hospice care. She was seen in follow-up appointments for removal of her gastrostomy tube and requested amputation of her toes. She died approximately 5 months after her initial intubation in the hospital.

**Discussion:** Following discharge, financial data from this patient's initial hospital stay was obtained. The difference in her hospital charges pre-intubation and post-intubation is approximately \$225,000. The average length of stay (LOS) following aorto-bifemoral bypass has been reported to be 5-7 days, while the LOS in this case was 48 days.

**Conclusion:** There are moral and ethical implications involved in intubating a patient with advance care paperwork indicating they do not want to be subjected to heroic measures. This case demonstrates that there can be financial consequences to overlooking a patient's wishes, as well. Cost-conscious care should include respecting the directive of the patient.

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PRES'D FOR TIME: CONSIDERATIONS IN SOLID ORGAN TRANSPLANT

First Author: Laura E Holton, MD Jared Herr, MD, David Chang, MD, Archit Bhatt, MD, MPH, Michelle Kittleson, MD, PhD, Jignesh Patel, MD, PhD, and Jon Kobashigawa, MD

**Introduction:** Advances in solid organ transplantation have led to increasing numbers of transplant patients evaluated in primary care and inpatient settings. Immunosuppression following transplant broadens any differential diagnosis. Here, we report a case of a young man post- heart transplant who presented with blurred vision and seizures.

**Case:** A 24-year-old man with non-ischemic dilated cardiomyopathy status post total artificial heart (TAH) followed by orthotopic heart transplant (OHT) presented with hypertension and confusion. Four months earlier, he underwent TAH placement, followed by OHT six weeks before admission. Transplant was complicated by primary graft dysfunction requiring extracorporeal membrane oxygenation. He recovered well and eventually discharged home. Three days before admission, worsening hypertension required additional antihypertensive medications. Shortly thereafter, he complained of headache, blurred vision and confusion, thus was referred to the ED. Upon arrival he had a generalized tonic-clonic seizure, an extended post-ictal period and progression to status epilepticus despite loading with levetiracetam. Initial exam revealed a chronically ill appearing young man, minimally responsive to verbal stimuli, with tachycardia and hypertension. CT demonstrated multiple areas of ischemia consistent with shower emboli. This was unsurprising given recent transplant and mechanical circulatory support, thus goals were set to allow for permissive hypertension. Tacrolimus level was supratherapeutic at 19 (goal 10-12). MRI showed paramedian hemorrhage and findings consistent with posterior reversible leukoencephalopathy syndrome (PRES). Blood pressure goals were adjusted to attain normotension and he was transitioned from tacrolimus to cyclosporine. The patient rapidly improved with full recovery to baseline.

**Discussion:** PRES is a neuro toxic state associated with severe hypertension, autoimmune disease or drug toxicity. The underlying pathology is poorly understood but results in vasogenic edema generally in the parietal occipital regions. Patients present with headaches, visual changes, confusion and seizures. Diagnosis relies on imaging which reveals areas of edema in the posterior circulation and watershed areas with MRI being the gold standard. Infarction and hemorrhage can be seen in 10% and 15% of cases, respectively. Prognosis is good with symptom resolution within 1 week and radiographic resolution after 4-6 weeks.

Tacrolimus, a first line immunosuppressant commonly used in OHT, has a narrow therapeutic window. Toxicities include hypertension, nephrotoxicity, and, rarely, PRES. Multiple variables impact metabolism of tacrolimus including age, hepatic function and drug interactions. Phenytoin, a CYP3A- inducing drug, has been used as an anticonvulsant for PRES as it also increases tacrolimus metabolism and rapidly reduces levels.

In our patient, both hypertension and tacrolimus toxicity likely contributed to the development of PRES. Early recognition of PRES and withdrawal of offending agents are key steps in management. Clinicians are reminded that the differential for common presentations is greatly broadened when evaluating post-transplant patients.

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LET'S BE PARSIMONIOUS: SO MUCH MORE THAN A MIDDLE AGED MALE WITH ALOPECIA,  
CONSTIPATION AND IMPOTENCE

Briana Ketterer MD, Kamala Nyamathi MD, Alan Hunter MD

The age old sentiment of Occam's Razor and the concept of Parsimony, seeking the simplest or most unifying explanation, are decision making heuristics that weave their way into medicine quite often.

This is demonstrated in the case of a 44 year old previously healthy semi-professional athlete with 2 year history of alopecia totalis who presented with a partial small bowel obstruction and 5 days of abdominal pain on transfer from an outside hospital. His history was notable for new diagnoses of hypothyroidism, BPH and impotence within the last 6 months. On review of systems found to have a constellation of subacute symptoms which included a 4 month prodrome of constipation, lightheadedness and syncope, dry skin and xerostomia, fatigue and 35lb unintentional weight loss. Family history notable for hypothyroidism and celiac sprue. On exam, found to have orthostatic hypotension, sluggishly reactive pupils of 8mm, moderately distended tympanic abdomen and non-focal neurologic exam without ataxia, Parkinsonism or neuropathy. Unifying his personal history and family history of autoimmune disease with the clinical evidence of dysautonomia, there was an early suspicion for autonomic autoimmune ganglionopathy. With a systematic approach, work-up confirmed pure autonomic failure by tilt table test and thermoregulatory sweat testing, and revealed high titers for ganglionic acetylcholine receptor antibodies (gnAChR) without evidence of underlying disorder or neoplasia. Notably negative bone marrow biopsy, unremarkable CT and PET imaging, rectal biopsy without evidence of amyloidosis and no exposures to heavy metals.

Autonomic Autoimmune Ganglionopathy often presents as severe subacute autonomic failure in a previously healthy individual such as our patient and may follow a viral infection or some other minor insult. Some studies suggest that as high as 30% of patients with AAG and positive gnAChR have a paraneoplastic syndrome with occult neoplasm. In this case, paraneoplastic work-up has remained negative to date. The patient was treated with IVIG, plasmapheresis, mycophenolate and rituximab. gnAChR titers are down trending and he continues to slowly improve. On hospital day 73, he was discharged home on TPN with close follow-up and anticipated ongoing IVIG and steroids. This case illustrates the utility in seeking the most parsimonious diagnosis and, the value of a complete review of systems with an organized and thoughtful diagnostic approach. Autonomic insufficiency in particular may parade as a constellation of seemingly unrelated symptoms, but with a systematic approach and broad lens can be unified.

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## SYNTHETIC MARIJUANA USE AND RHABDOMYOLYSIS

First Author: Akanksha Agrawal, MD Second Author: Percy Balderia, MD Third Author: Jacqueline Bowers, MD

**Introduction** Synthetic marijuana, also known as “Spice” or “K2”, is a psychoactive designer drug that has been increasingly popular in the U.S. since 2008. It is a smokable herbal mixture with synthetic cannabimimetic agents like CP 47, 497-C8 and JWH-018 which are 4 to 5 times more potent than traditional marijuana. Hence, the adverse effects and ensuing danger can also be more pronounced. We present a case of rhabdomyolysis from the use of synthetic marijuana.

**Case Report** A 39 year old African American woman with well-controlled asthma presented to the emergency department complaining of bilateral upper and lower extremity pain. She was using K2 daily for a month until 2 days prior to presentation. This was associated with insomnia and loss of appetite. She denied taking alcohol and other illicit drugs as well as any history of vomiting, diarrhea, trauma, long term immobility, seizures, loss of consciousness, or prior episodes of similar muscle pain. Upon admission to the ED, her heart rate was 88 bpm with a blood pressure of 130/75 mmHg, respiratory rate of 18/min. She was afebrile. She was alert, oriented, and calm. There was no conjunctival injection. Laboratory testing showed a creatine kinase level of 11,224 IU/L with normal creatinine and phosphorus. Myoglobin was detected in her urine. Standard urine drug screening was negative for amphetamines, benzodiazepines, cocaine, opiates, cannabis and phencyclidine. She was admitted and received aggressive intravenous hydration. Her symptoms gradually resolved and her creatine kinase levels declined. On hospital day 3, she was discharged home and instructed to follow up with rehabilitation services.

**Discussion** The most common adverse effects associated with synthetic marijuana include tachycardia, hypertension, chest pain, hallucinations, and seizures. We present a case of rhabdomyolysis, a rarely reported effect of synthetic cannabinoid use. Few cases of rhabdomyolysis have been described so far, usually in association with seizures or severe agitation, both of which were absent in our case. With the increasing popularity of synthetic marijuana, we expect to see more cases of rhabdomyolysis. A history of synthetic marijuana use should be specifically sought in a patient with rhabdomyolysis as many people do not classify it as an illicit drug and synthetic cannabinoids are not detected by routine urine drug testing.

## TUMOR LYSIS SYNDROME IN METASTATIC COLON CANCER AFTER SINGLE FOLFOX CYCLE

First Author: Akanksha Agrawal, MD Second Author: Deepanshu Jain, MD Third Author: Marina Kishlyansky, BSc, MSc Fourth Author: Mark Morginstin, MD

**Introduction-** Tumor lysis syndrome (TLS) is a life threatening oncological complication that is often described in patients with a large tumor burden, more commonly among hematological malignancies. It has rarely been described in patients with solid tumors, and to our knowledge it has never been described in a metastatic colon cancer in response to one cycle of FOLFOX in the absence of pre-treatment with other chemotherapeutic or immune-modulating agents.

**Hospital course-** A 55-year-old African American male presented to the Emergency Department (ED) with worsening abdominal pain, oliguria, nausea, vomiting and diarrhea of 1-day duration. One-month prior, the patient was diagnosed with colon adenocarcinoma with metastases to the liver (Stage IV). Pretreatment LDH for our patient was 1196 U/L. He was started on a FOLFOX regimen the day prior, which consisted of Leucovorin (772 mg IV), 5-Fluorouracil (4632 mg IV) and Oxaliplatin (164 mg IV). In the ED, patient was found to be in acute kidney injury with creatinine of 1.9 mg/dl. In addition, patient was hyperkalemic (6.4 meq/L), hyperuricemic (20.3 mg/dl) and hyperphosphatemic (5.5 mg/dl) with normal calcium level. There was no seizure activity or cardiac arrhythmia on telemetry monitor (Cairo-Bishop grade II TLS).

Patient was admitted to the Step Down Unit with a diagnosis of Tumor Lysis Syndrome and managed with aggressive intravenous hydration, furosemide and single dose of rasburicase (6 mg). In two days, symptoms resolved with improvement in potassium (4.8 meq/L), uric acid (5.6 mg/dl) and creatinine (1.4 mg/dl). Patient was transferred to the general oncology floor and was discharged four days later with oncology follow up.

**Conclusion-** There have been so far six published cases, reporting TLS in metastatic colon adenocarcinoma. Four of these were after FOLFIRI (5-fluorouracil, leucovorin, and irinotecan) or irinotecan chemotherapy, one after cetuximab therapy and one after FOLFOX therapy in the setting of pretreatment with FOLFIRI therapy. Our case demonstrates that TLS can occur in a metastatic colon cancer patient after a single cycle of FOLFOX therapy even in the absence of any pretreatment. Our patient had all known risk factors for developing TLS like large tumor burden, liver metastases, elevated pretreatment LDH, use of combination chemotherapy drugs and dehydration. Unlike previously reported 6 cases where TLS resulted in death, our patient survived. Therefore, a clinician should maintain high index of suspicion for TLS among metastatic colon cancer patients and should do prompt intervention to prevent potentially life threatening complications like cardiac arrhythmias, acute renal failure, seizures, or death.

EXCLUDE MAY-THURNER SYNDROME FIRST BEFORE DECLARING LOWER EXTREMITY DEEP VEIN THROMBOSIS AS IDIOPATHIC.

Irfan Ahsan MD, Binish G Qureshi MBBS, Rabia Naheed MBBS, Zulfiqar Arif MD, Chris Christensen III MD

May-Thurner Syndrome (MTS) also known as Cockett's Syndrome is a rare cause (2% - 3%) of Deep Venous Thrombosis (DVT). The thrombosis results from mechanical compression of the left iliac vein against the body of the fifth lumbar vertebra by the right common iliac artery. Repetitive hyperplasia of the venous wall by compression results in spur formation that in turn causes venous flow obstruction and results in DVT.

We present a case of MTS that was successfully managed using mechanical thrombectomy with a venous stent. Our case is a 36 y/o African American non-pregnant female who presented to the hospital with left calf swelling and pain. The clinical exam was pertinent for lower extremities swelling extending up to thigh with tender and erythematous left lower leg at the calf with bilateral positive dorsal pedal pulses. Laboratory workup including CBC, CMP and coagulation profile was normal. A lower extremity Doppler ultrasound revealed occlusive acute DVT in the left common femoral and femoral veins. A diagnosis of MTS was considered. She was given IV Heparin and underwent Interventional radiology (IR) guided Infra-renal inferior vena cava filter placement and Trellis® procedure which failed to lyse clots with Alteplase® Infusion. Post lysis imaging revealed significant residual thrombus along the course of the lysis catheter involving the left common iliac, left common femoral and left superficial femoral veins. Minimal to no flow through the native left iliac vein was observed. The next day, she underwent successful left lower extremity venous mechanical thrombectomy with stenting of the left iliac vein. Post stent venogram revealed restored central flow to the iliofemoral venous system. She was discharged on oral anticoagulants.

May-Thurner Syndrome although a rare entity should be suspected especially in young patients with unilateral DVT with extensive clots especially on left lower extremity without any anteceded risk factors. The mainstay of treatment for MTS includes endovascular thrombolysis with or without prophylactic IVC filter placement followed by angioplasty/stenting of the left common iliac vein and long-term anti-coagulation.

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A CHANGE OF HEART – AN UNUSUAL CASE OF ANTIPHOSPHOLIPID SYNDROME

First Author: Nicole C Finelli, DO Second Author: Pamela L Bailey, DO

Nonbacterial thrombotic endocarditis (NBTE) is a rare condition that is most often found postmortem. It is strongly associated with advanced malignancy and inflammatory conditions such as antiphospholipid syndrome and rheumatic disorders. It is frequently diagnosed after patients present with an embolic event and the underlying cause is often difficult to uncover.

A 20-year-old woman with no past medical history presented for progressive shortness of breath and dyspnea of exertion. A transthoracic echocardiogram revealed severe aortic stenosis with an ejection fraction of 25% and left ventricular dysfunction. The patient was sent for aortic valve replacement, and the operating report revealed acute inflammation of the trileaflet aortic valve concerning for rheumatic valvulitis. A follow-up antistreptolysin O titer and streptozyme were positive, and the patient was started on a prolonged course of penicillin to avoid recurrent inflammatory valvulitis secondary to streptococcal disease. The patient returned to the hospital two weeks later for a persistent nosebleed and was found to be severely thrombocytopenic and anemic with a platelet count of 1,000/ml and hemoglobin of 7.4 g/dL. A peripheral smear revealed spherocytes and polychromasia and a positive Coombs test mounted strong evidence for an underlying immune mediated process. The patient required platelet transfusion, intravenous steroids and intravenous immunoglobulin G for stabilization and eventual improvement of the blood counts. A complete workup revealed a positive lupus anticoagulant assay, positive IgM and IgG anticardiolipin antibodies and positive anti-beta-2 glycoprotein antibodies. Given the high titers of positive antibodies, it was suspected that the patient's unifying diagnosis was antiphospholipid syndrome (APS) rather than rheumatic heart disease. The patient was continued on oral steroids and long-term anticoagulation therapy.

A strong clinical suspicion for NBTE should prompt a thorough workup not only for occult malignancy, but also for autoimmune or rheumatic diseases. The case illustrates the rare and masquerading diagnosis of APS. The patient's initial presenting cardiac manifestations are unique in that they are of a non-bacterial thrombotic etiology. Most patients with NBTE do not present with heart failure or new onset murmur, but rather secondary to embolic phenomenon. It is critically important to recognize NBTE on the differential for antiphospholipid syndrome. Ongoing management of this patient's APS is significant as well, given the need for long-term anticoagulation and oral steroids, especially considering her young age.

## AN ISOLATED CAUSE OF HYPONATREMIA

First Author: Alan Gandler, MD Allison Trainor, BS

Introduction: Isolated ACTH deficiency is a rare cause of adrenal insufficiency, which was identified during a work up of hyponatremia. This case additionally illustrates the importance of not anchoring one's diagnosis.

Case: A 68-year-old woman with a history of alcohol abuse and MI was brought in by EMS for lethargy, weakness, and dizziness after her children found her too weak to get out of bed. Per the children she had been drinking in excess of 8 beers daily, with minimal nutritional intake. The patient reported recent nausea and increased urination. Her exam was remarkable for a BP of 100/75, tachycardia, dry mucous membranes, and lethargy. Her laboratory tests were significant for serum sodium of 108, serum chloride of 62, serum osmolality of 233, urine osmolality of 334, urine sodium of 35, and a TSH within normal limits. She was initially treated for hypovolemic hyponatremia, which was gradually corrected with normal saline infusions. At this point it was presumed her hyponatremia was due to beer potomania; however, she continued to require IV normal saline at 150cc/hr to keep her serum sodium within normal limits. In addition she continued to have tachycardia, dizziness, and polyuria in excess of net negative 2L/day. A trial of fluid restriction did not resolve the polyuria, ruling out primary polydipsia. Repeat urine electrolytes revealed a urine sodium of 110 and urine osmolality of 550, inconsistent with beer potomania. The following morning, cortisol and ACTH were drawn and were 1.8 and 4, respectively, which is consistent with secondary adrenal insufficiency. To confirm the diagnosis, a high dose cosyntropin stimulation test was done which caused an increase in serum cortisol from 1.6 to 16. This is a subnormal response suggesting the adrenals are functional but atrophied from lack of ACTH stimulation. In order to rule out panhypopituitarism, serum FSH and LH were tested and showed postmenopausal levels consistent with isolated ACTH deficiency. At this point the patient was started on prednisone 2.5mg twice daily and her polyuria and dizziness resolved. She was discharged with primary care and endocrine follow up.

Discussion: This case illustrates the importance of not anchoring one's diagnosis, correlating lab results with history, and recognizing the signs of adrenal insufficiency. Upon presentation the cause of her hyponatremia appeared to be beer potomania. However, her urine osmolality and urine sodium are inconsistent with beer potomania. Typically in beer potomania causing symptomatic hyponatremia the urine is maximally dilute and has low sodium because poor solute intake impairs the kidney's ability to form a functional gradient to concentrate the urine. This case is also interesting as isolated ACTH deficiency is a rare diagnosis.

A MOTHER'S LAMENT: EMPYEMA NECESSITANS DUE TO ACTINOMYCES AND FUSOBACTERIUM IN IMMUNOCOMPETENT PATIENT

First Author: Madeeha Hafeez, MD Second Author: Ingi Lee, MD

**Introduction:** Depression and self-neglect after the death of a loved one can have devastating consequences, including infectious complications. We present the case of a mother who had self-neglect after the loss of her son and developed Empyema Necessitans; a rare complication of pleural effusion with dissection of tissue planes and chest wall abscess formation; due to Actinomyces and Fusobacterium spp. Both bacteria are part of the normal human oral flora.

**Case Description:** An 80-year-old caucasian female with 40 pack-year history of smoking presented to the emergency department with pleuritic chest pain. Her family described that since the death of her son two months earlier, she developed severe depression, ignored taking care of herself and remained in bed for long stretches. There was no recent history of trauma or dental procedures. Initial vital signs including temperature were normal. Her O<sub>2</sub> saturation was 89%. On physical examination, she appeared frail and cachectic with a withdrawn affect. Inspection of oral cavity revealed poor dentition but no thrush or ulcerations. She had decreased breath sounds at the right lung base. Laboratory blood findings were significant for leukocytosis ( $28 \times 10^9$ /liter) with 90% segmented neutrophils. A chest radiograph showed a 10 cm right-sided pulmonary opacity. A CT scan of the chest revealed right eleventh rib fracture with loculated pleural effusion. IR-guided right chest tube was inserted and 400mL of frank pus was drained. The pleural fluid culture was positive for both Actinomyces and Fusobacterium spp; mycobacterial and fungal cultures were negative. Blood and urine cultures showed no growth. A week later, she complained of right sided low back pain. A CT scan of the abdomen revealed an enhancing soft tissue lesion with partial destruction of right twelfth rib which extended from posterior aspect of right chest wall into the paraspinal soft tissue musculature consistent with abscess formation and rib osteomyelitis. IR-guided abscess drainage was performed. Chest tube was removed after complete drainage of the empyema. She was treated with intravenous Ertapenem for 6 weeks until resolution of the abscesses on CT scan and was switched to oral amoxicillin which she will continue for 12 months.

**Discussion:** Empyema Necessitans caused by Actinomyces and Fusobacterium confection is previously unreported. Literature search (PubMed) revealed isolated cases of Fournier's gangrene, periodontitis, hepatic abscess, mastoiditis, long-bone and thoracic vertebral osteomyelitis caused by this coinfection. Patients with diabetes, chronic steroid use or immunosuppression are predisposed. Our patient did not have classical predisposing factors but we believe that she became susceptible due to lack of personal hygiene. She likely had a silent aspiration event and primary lung infection by both bacteria with extension causing destruction of the ribs, development of empyema and paraspinal abscess formation.

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PULMONARY ARTERIOVENOUS MALFORMATION: A RARE CAUSE OF CRYPTOGENIC STROKE

Karlee Hoffman, Shyam Padmanabhan, Indu Poornima

**Introduction:** Pulmonary arteriovenous malformations (PAVM) are rare entities and often associated with hereditary hemorrhagic telangiectasias (HHT). There are a few cases of isolated PAVM that are not associated with hereditary disease and can present with asymptomatic hypoxemia, dyspnea, or pulmonary hemorrhage. Others may remain asymptomatic until presentation with stroke, myocardial infarction, or even death due to paradoxical embolism. Although a rare entity, PAVM is a cause of cryptogenic stroke. Clinical suspicion and early diagnosis are critical in preventing further ischemic events.

**Case Description:** A 49-year-old-female with no medical history presented with blurry vision. The patient subsequently became obtunded and imaging showed basilar artery thrombosis. Thrombolytic therapy was administered with complete thrombus resolution. A transthoracic echocardiogram (TTE) with contrast showed a right to left shunt of unclear location. Transesophageal echocardiogram (TEE) showed intrapulmonary right to left shunting. A CT pulmonary angiogram (CTPA) was ordered as an outpatient. While awaiting the CTPA, the patient had recurrent blurry vision. Head and neck MRI/ MRA were negative for pathology. CTPA showed a right lower lobe PAVM. This clearly was the cause of her strokes and she was referred for PAVM coil embolization.

The patient had no clinical evidence of HHT and a negative hypercoagulable workup. Following the coil embolization, patient has had no recurrent ischemic events.

**Discussion:** Cryptogenic stroke is defined as a brain infarction in which cardiac emboli, large artery atherosclerosis, or small artery disease are deemed not to be the source of infarction after extensive evaluation. Echocardiography is indicated to rule out intracardiac shunting. Extracardiac shunting such as an isolated PAVM is rare and only accounts for 0.5% of cryptogenic strokes.

These patients might have negative chest x-rays, normal oxygen levels, and unremarkable pulmonary physical exams. Echocardiography with agitated saline contrast injection is a noninvasive test that can identify the location of shunt based on the timing of arrival of contrast in the left atrium. Volume of shunting and the pulmonary vein of origin can be identified by TEE. CTA is currently the gold standard because it accurately locates and predicts the complexity of the PAVM.

Indications for treatment of PAVM include prevention of neurological complications, improvement in exercise tolerance and prevention of lung hemorrhage. Embolization has shown a substantial reduction in mortality and morbidity related to PAVM.

Right to left intrapulmonary shunting on TTE can also be caused by conditions other than PAVM. Pulmonary shunting can also be indicative of hepatopulmonary syndrome, congenital heart defects, or be an incidental finding in healthy individuals. In the case discussed, the occurrence of multiple strokes and the volume of intrapulmonary shunting is what led to the diagnosis of PAVM.



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POST-PARTUM PERIOD AND IDIOPATHIC THROMBOCYTOPENIC PURPURA – A NEW ASSOCIATION???

First Author: Syed Imran Jafri, MD Other Authors: Faizan Malik, MD, Irfan Ahsan, MD, Naveed Ali, MD, Michael Seidman, MD

Idiopathic Thrombocytopenia (ITP) is one of the more common causes of thrombocytopenia. There are several etiologies of thrombocytopenia during pregnancy, including gestational thrombocytopenia, HELLP, pre-eclampsia and ITP. We are reporting a case of severe thrombocytopenia in a post-partum female which turned out to be severe ITP.

A 33-year-old female presented to her obstetrician with the chief complaint of vaginal bleeding, bruises on legs and gum bleeding while brushing. The patient delivered a healthy baby girl 3 weeks before this presentation. The last platelet count before delivery was checked on week 28 of pregnancy which was 211,000/UL. The past medical history included asthma and thyroid surgery 14 years prior. She was not taking any medications at the time of presentation. She denied any recent infections or sick contacts. The patient had blood work done which showed a platelet count of 4000/UL. She was advised to come to the hospital. The pelvic ultrasound did not show any retained products of conception. All of her blood work including Liver function tests, PT, aPTT, d-dimer, Fibrinogen, ANA, RF, SPEP, Quantitative Immunoglobulins, HIV, HbsAg, HCV Ab, retic count, coombs test, Ferritin, B12 and Folate were unremarkable. She was given 1 unit of platelet on admission with no improvement and then started on IVIG and Steroids. Her platelet counts improved rapidly. She was discharged with platelet count >100,000/UL. The patient followed up with a hematologist as an outpatient. Her platelet count dropped again after a prednisone taper was started. She received treatments with high-dose steroids, IVIG and Rituximab. She was also treated for H. Pylori with the finding of positive serum studies, given the strong association between ITP and H Pylori. However, her thrombocytopenia did not respond consistently to medical management. She had splenectomy done almost 6 months after her initial presentation, with the resolution of thrombocytopenia.

ITP is a known entity in pregnancy. However, ITP has not been reported in the post-partum period. We believe our case is the first ever reported case of ITP in the post-partum period.

IVIG TO THE RESCUE: SYMPTOMATIC HYPOGAMMAGLOBULINEMIA DUE TO IMMUNOTHERAPY

First Author: Faiza Khalid, MD, UPMC-McKeesport. Sofiya Rehman, MD, UPMC-McKeesport. Shikha Gupta, MD, UPMC-McKeesport.

**Introduction:** Rituximab is an anti-CD20 chimeric antibody used to treat hematologic malignancies and autoimmune disorders. Although the initial clinical trials did not reveal significant hypogammaglobulinemia, there are increasing case reports of hypogammaglobulinemia with Rituximab therapy. This poses an increased risk of infections which do not respond to antibiotic therapy alone. We report a case of severe pneumonia who was treated with intravenous immunoglobulin (IVIg) for hypogammaglobulinemia due to Rituximab.

**Case Description:** Patient is a 62-year-old male with history of recurrent grade 2 follicular lymphoma who was on maintenance Rituximab therapy for 10 months. He was admitted to the ICU with respiratory failure, due to multifocal pneumonia, requiring endotracheal intubation. Bronchoscopy with broncho-alveolar lavage was performed, and cultures came back positive for Hemophilus influenzae and Influenza A (subtype H3). Appropriate treatment with Moxifloxacin and Oseltamivir was continued but patient deteriorated. As our patient was on immunotherapy prior to the admission, serum immunoglobulin levels were checked. Patient's immunoglobulin levels were found to be low: IgG 2.6 g/L (normal 6.9 g/L--16.18 g/L), IgM <0.13 g/L (normal 0.48 g/L—2.71 g/L) and IgA 0.52 g/L (normal 0.81g/L—4.63 g/L). Given the severity of illness and lack of improvement with appropriate antimicrobial therapy for 5 days, he was given intravenous immunoglobulin infusion at 500mg/kg. Repeat IgG level increased to 9.8g/L. Patient had significant hemodynamic improvement and repeat Chest CT done after two days of infusion revealed interval decrease in reactive airway disease.

**Discussion:** In hematological conditions, the incidence of transient or persistent hypogammaglobulinemia following Rituximab therapy is 15-40%. With the increasing use of Rituximab, it is important for clinicians to be mindful of secondary hypogammaglobulinemia and subsequent infections. Baseline immunoglobulin levels are checked before initiating treatment with Rituximab. However, there are no current guidelines on how often immunoglobulin levels should be checked while patients are on immunotherapy. Symptomatic hypogammaglobulinemia is particularly common in patients who receive maintenance therapy with Rituximab. Intravenous Immunoglobulins have been used extensively in primary immunodeficiency but no extensive data is available on their use in secondary hypogammaglobulinemia. Among the causes of secondary hypogammaglobulinemia, primary HIV infection and chronic lymphocytic leukemia (CLL), leading to low immunoglobulin levels, are the only two conditions that are FDA approved for IVIg infusion. Our patient was given IVIg due to failure of antibiotic therapy. Intravenous immunoglobulin infusion should be considered for patients who develop severe symptomatic hypogammaglobulinemia secondary to maintenance immunotherapy.

OCCAM'S RAZOR: ASYSTOLE DUE TO DYSAUTONOMIA FROM SEVERE GUILLAIN-BARRÉ SYNDROME VERSUS RIGHT CORONARY ARTERY DISEASE.

First Author: Fatima Khan, MD Second Author: Irfan Ahsan, MD Third Author: Richard Borge, MD

Guillain Barré Syndrome (GBS) is known to cause dysautonomia. Cardiac manifestations of dysautonomia include labile blood pressures, arrhythmias and asystole. We report a case of GBS patient having bradycardia, asystole and right coronary artery disease (RCA).

A 68 year old female presented for evaluation of syncope and diarrhea. Four days prior to admission, she received pneumonia and flu vaccines. Shortly after admission, she developed ascending paralysis, became quadriplegic and was diagnosed with GBS. She was intubated due to respiratory failure. She received five days course of intravenous immunoglobulins with no improvement. Later she received five plasmapheresis treatments. She developed uncontrolled hypertension so oral amlodipine, lisinopril, and intravenous labetalol were initiated. Soon she became hypotensive so her all anti-hypertensives were discontinued. Her blood pressures continued to remain labile and simultaneously, she started having episodic sinus bradycardia with heart rates below 30 beats per minute unrelated to positioning, suctioning and other nursing maneuvers. On day 17, she had an unexpected asystolic arrest with return of circulation (ROC) within three minutes of resuscitation and an external pacer was placed. E chocardiogram showed no regional wall motion abnormalities. On day 23, ST-T wave abnormalities were incidentally noted in inferior leads with elevated troponins and a cardiac catheterization was performed. Coronary angiography revealed diffusely diseased RCA with 90% stenosis and a bare metal stent was placed. Two hours after intervention patient had another asystolic arrest with ROC in 6 minutes of resuscitation. A temporary pacemaker was placed and next day a dual chamber permanent pacemaker was placed. Repeat echocardiogram showed no regional wall motion abnormalities. Due to ventilator dependence she had a tracheostomy and was discharged to a Long Term Acute Care hospital.

Dysautonomia has been noted in about 66% of GBS patient in a study. It may manifest in various organ systems innervated by parasympathetic and sympathetic nervous system. Bradyarrhythmias and asystole have been noted in about 50% of the patients with acute severe GBS on mechanical ventilation. Such patients end up requiring a pacemaker. With labile blood pressures and variable heart rate, we attributed our patient's bradycardia and asystole to the dysautonomia from acute severe GBS and not due to underlying RCA disease. Moreover, she had another asystole after the coronary intervention and no wall motion abnormalities discounting ischemia as a cause. RCA disease commonly presents as heart blocks and is known to cause bradycardia and asystole during intervention to RCA. Co-existing RCA disease in GBS patients contributing to bradycardia and asystole has not been reported but if suspected coronary angiography should be considered.

## THROMBOCYTOPENIA: "THE CULPRIT LIES IN THE STOMACH"

Faizan Malik MD, Irfan Ahsan MD, Syed Imran Jafri MD, Naveed Ali MD, Ali Ghani, MD

*Helicobacter Pylori* has been associated with Gastritis, Gastric Adenocarcinoma and Mucosa-associated lymphoid tissue (MALT) Lymphoma. Recently studies in Japan, Italy, and Colombia showed an association of Idiopathic thrombocytopenic Purpura (ITP) with *H. Pylori* and improvement with eradication. The proposed mechanism involves interaction between platelet glycoproteins and *H. Pylori* CagA protein. We report a case of ITP with excellent response to *H. Pylori* eradication therapy.

A 48-year-old African-American-male presented with bleeding of lower lip and wisdom tooth. He had a history of HIV/AIDS (on Raltegravir, Lamivudine and Zidovudine-CD4 count unknown), Cryptococcal meningitis (on Fluconazole after Amphotericin-B/Flucytosine induction), *Mycobacterium Avium* intracellulare infection and pneumocystis pneumonia currently on Dapsone for secondary prophylaxis. The patient bit his lip while eating two nights ago, progressing to hematoma formation, rupture and finally bleeding. The patient denied prior or recent bleeding from other sites, recent infectious illness, anticoagulation or non-compliance with HIV medicines. Physical examination was pertinent for tachycardia. There was large hemorrhagic bulla on the right lower lip, petechiae on soft-palate, facial hyperpigmentation and absence of oral thrush or ulcers. Laboratory studies showed leucocyte count of 2.4K/uL, hemoglobin of 8.8gm/dl, platelets of 2000/uL (prior platelets were 75K/uL) with Mean Platelet Volume >19fL, INR of 1.3 and PTT of 30sec. A presumptive diagnosis of ITP secondary to HIV was considered and multiple treatment modalities including platelet transfusion, Aminocaproic acid, intravenous steroids, IVIG, Romiplostim, and Rituximab were given with no improvement in platelet count. Even stopping HIV medicines didn't improve platelet count. Platelet trend on this regimen was 2>2>1>2>6>2>3>2>1 K/uL. Hospital course was complicated by melena and heme-positive stools. *H. Pylori* testing was positive for IgG antibody. Fecal antigen and Urea breath test couldn't be done as the patient was on PPI. Endoscopy was not performed owing to low platelet count. Bone marrow biopsy was unrevealing. Empiric treatment for H-*Pylori* by triple therapy with Omeprazole, Clarithromycin, and Amoxicillin was initiated and during ten days course, his platelet count improved with a trend of 4>9>24>49>169>172K/uL and successful improvement of his clinical symptoms.

Studies have shown sustained recovery of ITP after *H. Pylori* eradication. Physicians should be encouraged to check *H. Pylori* through fecal antigen, urea breath test and/or upper endoscopy and eradicate *H. pylori* early in thrombocytopenia to avoid unnecessary diagnostic workup and complications of delayed treatment.

ALLERGIC TO LIFE; CONTINUOUS BENADRYL INFUSION FOR PREVENTION OF MAST CELL ACTIVATION SYNDROME

Faizan Malik, Irfan Ahsan, Syed Imran Jafri, Naveed Ali, and Bilal Lashari.

Mast cell activation syndrome (MCAS) is characterized by recurrent symptoms of mast cell activation in combination with objective evidence of mast cell-mediated release. The clinical features include flushing, pruritus, abdominal pain, diarrhea, hypotension, syncope and musculoskeletal pain. Treatment options are Anti H-1, Anti H-2, Doxepin, Cromolyn and Anti-Leukotriene agents. Recently, Omalizumab has shown some efficacy in case reports. Maintenance steroids can be used in refractory cases but result in multiple side effects. We report first ever case of MCAS treated with continuous Benadryl infusion resulting in improvement of anaphylactic reactions and decreased in hospital readmission.

An 18-year-old-female with a history of MCAS presented with flushing, diarrhea, and hypotension. She had chronic swelling of her face and abdomen at baseline. She was initially stabilized by epinephrine and steroids. Patient has had multiple prior episodes of anaphylactic reactions at least twice weekly for which she used to get evaluated in the emergency room and treated per a standard protocol in place for her, given the frequency of her visits. At age 15, she had significant asthma symptoms and was found to have a mildly elevated Ig-E level. Prior skin testing resulted in vomiting, dehydration, and hospitalization. She was diagnosed with Food Protein-Induced Enterocolitis after having a food challenge test with severe anaphylaxis to eggs. A bone marrow biopsy was inconclusive without evidence of Mastocytosis and absent C-Kit D86V mutation. A serum Tryptase level has been normal between episodes with elevation up to 29ng/dl during flare ups. Plasma prostaglandin-D2 levels and 24-hour urine 11 beta-prostaglandin-F2 levels have been markedly elevated during episodes. The C1-esterase inhibitor was negative. She has been previously treated with steroids, multiple antihistamines, and Omalizumab, a monoclonal Abs which inhibits IgE binding to mast cells, but, unfortunately, it also resulted in anaphylaxis. To stress the seriousness of the situation, she had a severe anaphylactic reaction resulting in cardiac arrest with successful resuscitation. Interestingly, University of Minnesota made the recommendation of using continuous Benadryl infusion on the basis of ongoing research which had shown some efficacy. She was started on Benadryl Infusions at 5mg/hour, was increased by 2mg every 2 hours with a target goal of 15mg/hour. Patient has been episode free for 2 months now, which has not happened in years for this girl.

MCAS is an idiopathic mast cell disorder which is difficult to treat because of lack of target therapy. Benadryl was always used as Intravenous pushes but has not been reported to be used as an infusion for MCAS. We present a case of its success which can have promising future outcomes.

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HYPERALDOSTERONISM AND HIRSUTISM: WHEN OCCAM'S RAZOR DOES NOT APPLY

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Introduction: 3 beta-hydroxysteroid dehydrogenase (3BHSD) deficiency is a rare cause of congenital adrenal hyperplasia which presents with glucocorticoid and mineralocorticoid deficiency in early life. A mild non classic, late onset form is characterized by hirsutism and/ or menstrual irregularity in young adult females. The co-occurrence of 3BHSD deficiency with primary hyperaldosteronism is extremely rare. We report a case where both these disorders presented as a diagnostic challenge.

Case Description: A 25 year old female with history of hirsutism presented with debilitating generalized weakness and persistent hypokalemia over several months despite potassium supplementation. She was also found to have hypertension. Prior work up for hirsutism had ruled out thyroid dysfunction, Cushing's syndrome, androgen producing tumor, hyperthecosis ovarii, hyperprolactinemia, classic forms of congenital adrenal hyperplasia, polycystic ovarian syndrome and complete androgen insensitivity syndrome. Further investigations revealed plasma renin to be incompletely suppressed (0.55 and 0.33 ng/ml/h, N 0.15-2.33 ng/ml/h), and elevated plasma aldosterone concentration (59.9 and 37.7 ng/dL, N < 31.0 ng/dL). Abdominal CT scan showed left 1.5cm adrenal adenoma. The patient was started on amiloride and potassium supplementation. Adrenal venous sampling showed lateralization to the left adrenal gland. The patient underwent laparoscopic left adrenalectomy, with pathology confirmation of the adenoma. The patient was successfully weaned off amiloride and no additional potassium supplementation was required. ACTH stimulation revealed elevated 17 hydroxypregnenolone levels, suggesting 3 beta hydroxysteroid dehydrogenase deficiency. Since she had regular menstrual cycle, the patient chose cosmetic treatment over steroids for hirsutism.

Conclusion: The co-existence of both primary hyperaldosteronism and 3 beta hydroxysteroid dehydrogenase deficiency is exceedingly rare, with only one PubMed indexed case report, in which hyperaldosteronism was attributed to unknown "aldosterone stimulating factors," in contrast to the adrenal adenoma in our patient. In patients with concomitant hirsutism and features of hyperaldosteronism, the initial thought process among most clinicians is to utilize an Occam's razor approach, searching for a unifying medical condition. As illustrated in our patient, however, Occam's razor does not always apply. In fact, this initial assumption delayed the diagnosis and therefore patient care.

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SUCCESSFUL SHORT-TERM PERCUTANEOUS IMPELLA CP SUPPORT IN A PATIENT WITH FULMINANT MYOCARDITIS.

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**Introduction:** Myocarditis management is mostly supportive, including aggressive pharmacological interventions with vasopressors, inotropes, and at times mechanical circulatory support. To date few cases have been described using a percutaneous temporary ventricular assist device, Impella CP, in the setting of fulminant myocarditis complicated by refractory cardiogenic shock.

**Case:** A 40 year old man with a past medical history of hypertension and diabetes presented to the emergency department complaining of non-productive cough of two days duration, shortness of breath, fever, chills and chest tightness. He was initially treated for community acquired pneumonia based on a questionable left lower lobe infiltrate on the chest radiograph. Further sepsis workup was negative. The initial EKG showed sinus tachycardia and non-specific T-wave abnormalities. Troponins were elevated up to 0.12 ng/ml and an echocardiogram revealed diffuse hypokinesis with an ejection fraction (EF) <20%. The patient was taken emergently to the catheterization lab where left and right heart catheterizations ruled out any occlusive coronary artery disease and confirmed the presence of cardiogenic shock that was treated initially with vasopressors. However, the patient remained in shock and a percutaneous Impella CP was inserted through the femoral artery. His clinical course was further complicated by anemia and thrombocytopenia due to device related hemolysis requiring transfusion of one unit of packed red blood cells. On hospital day six, significant improvement of cardiac output and EF were noted allowing for removal of the Impella CP, upon which his thrombocytopenia improved without further treatment. Follow-up echocardiogram showed an EF of 40%. The entire hospital length of stay spanned 14 days and the patient was discharged to a rehabilitation facility.

**Discussion:** To our knowledge, this is the only case describing an adult patient with rapid recovery from fulminant myocarditis bridged by the insertion of an Impella CP without further need of permanent ventricular mechanical support or simultaneous extracorporeal membrane oxygenation. There is growing evidence demonstrating the safety and successful utilization of minimally invasive devices in the management of cardiogenic shock. Based on this experience, fulminant myocarditis patients might benefit from the use of these devices although further research is necessary to clarify their role.

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**MIND THE GAP: A CASE OF SEVERE METHANOL INTOXICATION**

First Author: Salik Nazir, MD Co-Authors: Shabana Ansari DO, Stephen Melnick DO, Rittu Hingorani, MD - Reading Health System, West Reading, PA

**Case Presentation:** A 37 year old alcoholic female with type 2 diabetes was brought unresponsive to the ED. On Initial vitals, she was normotensive and afebrile with agonal breathing and oxygen saturation of 88 percent on room air. Exam revealed GCS of 3, fixed dilated and nonreactive pupils. Rest of the exam was unremarkable. Patient was intubated and mechanically ventilated. CT scan brain was unremarkable and the EKG was at baseline. Initial Laboratory testing identified arterial PH (6.724, range: 7.36-7.46), Pco<sub>2</sub> (41.2 mmHg, range: 36-46 mmHg), Po<sub>2</sub> (67.3, range: 75-90 mmHg), serum bicarbonate (4.8 meq/L, range: 21-31 meq/L), high serum glucose of 361 mg/dl, anion gap (29 meq/L, range: 8-12 meq/L), beta- hydroxybutyrate (0.32 mmol/L, range: <0.27 mmol/L), serum creatinine (1.33 mg/dL, Baseline 0.7 mg/dL) and hyperlactatemia ( 9.2meq/L, range: 0.5-2.2 meq/L) suggestive of severe diabetic ketoacidosis. However, given the discordance between the anion gap and serum beta-hydroxybutyrate a concern for toxic alcohol ingestion was present. Subsequent laboratory evaluation showed high serum osmolarity (416 mOsm/kg, normal 280-290 mOsm/kg), negative blood ethanol, salicylates and acetaminophen level and high osmolar gap of 115.3 concerning for methanol and ethylene glycol ingestion. Pending volatile compound screen, fomepizole and folic acid were started, and urgent hemodialysis was undertaken. MRI brain showed restricted diffusion and hyperintense signal within putamen of bilateral basal ganglia suggestive of methanol intoxication. Patient was subsequently found to have initial methanol level of 237 mg/dL and negative for ethylene glycol. She was successfully extubated on day 2 of hospitalization with residual cognitive and visual deficits and admitted taking large amounts of windshield wiper fluid as an ethanol substitute.

**Case discussion:** Methanol intoxication presents with a high anion gap and increased serum osmolar gap. The presence of both laboratory abnormalities concurrently is an important diagnostic clue, however either can be absent, depending on the time after exposure when blood is sampled. Although methanol itself is not highly toxic, it is metabolized by alcohol dehydrogenase to formaldehyde and subsequently to formic acid. These metabolites cause the metabolic acidosis, blindness, cardiovascular instability and death attributed to methanol toxicity. It requires administration of Fomepizole or ethanol to inhibit alcohol dehydrogenase, a critical enzyme in its metabolism. Elimination of formic acid is dependent upon tetrahydrofolate and is accelerated following administration of folic acid. Hemodialysis is the best method to rapidly remove both the parent alcohol and toxic metabolites and is recommended in cases of end organ damage and high anion gap acidosis regardless of drug level.

**Conclusion:** Methanol intoxication is associated with high morbidity and mortality. It is important for clinicians to have a high degree of suspicion for this disorder as rapid decision making is crucial and often involves deciding treatment without definite drug levels.

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## GASTROPARESIS IN POSTURAL ORTHOSTATIC TACHYCARDIA SYNDROME

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Introduction: Postural Orthostatic Tachycardia Syndrome (POTS) is an orthostatic intolerance with excessive increase in heart rate which occurs on standing without arterial hypotension. A tilt test with increase in heart rate by greater than 30 beats over baseline or to more than 120 beats confirms diagnosis. About 500,000 Americans have this syndrome which is common between 14 to 45 years and women predominate. The abnormalities in autonomic regulation may either be genetic or acquired, and may be associated with other forms of dysautonomias. These patients may develop gastroparesis, defined as gastric retention of over 10% at 4 hours and/or over 60% at 2 hours on scintigraphy.

Case description: A 19 year old white female with POTS diagnosed at the age of 13 on appropriate treatment presented with 3 days of nausea, vomiting and abdominal pain. This was associated with chills, sweating, flushed face, stuffy nose and production of watery sputum. She has had multiple episodes of nausea and vomiting for years, over 6 esophagogastroduodenoscopies in 2 years, and more recently gastric emptying study with diagnosis of gastroparesis. On presentation, her physical examination revealed mild abdominal tenderness more on the left. Her basic work up was unremarkable, but for abdominal x-ray suggestive of ileus and subsequently normal abdominal computed tomography. She was seen by a surgeon and gastroenterologist with recommendation to manage conservatively without enteral feeds but on intravenous fluids, antiemetics and prokinetics for acute gastroparesis possibly triggered by gastroenteritis. Over 5 days her symptoms worsened and she was then referred to a gastric motility center. At the center she was put on nasogastric tube feeding and was scheduled for gastric pacemaker placement. She had lost over 105lbs over a period of 8 months.

Discussion: It is important to consider POTS in patients with gastroparesis. Abnormal gastric myoelectric activity has been demonstrated in patients with POTS using electrogastrigraphy suggesting involvement of autonomic/enteric neural activities mostly with a rapid pattern. It is a marker of poor prognosis especially with delayed pattern (gastroparesis), although POTS in general has been shown to have a favorable prognosis. This young patient presented in dysautonomic crises with refractory gastroparesis which is relatively rare, requiring temporary ambulatory tube feeding with eventual gastric pacemaker placement. Unlike the more invasive gastrostomy with/without fundoplication and parenteral nutrition, gastric pacemakers have been demonstrated to provide improvement and better quality of life in patients with this unusual complication of POTS.

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THE FIRST CASE REPORT DESCRIBING CROSS-REACTIVITY OF BETA-LACTAMS IN CAUSING THROMBOCYTOPENIA

First Author: Sneh Pandey, MD MBBS Other authors: Sofiya Rehman, MD Ibrahim Ghobrial, MD

**Introduction:** Drug induced thrombocytopenia (DIT) is a well known albeit uncommon entity with incidence of around 10 cases per million per year. The common drugs which cause DIT are heparins, glycoprotein IIb/IIIa antagonists, quinine, cotrimoxazole and  $\beta$ -lactams. DIT should always be considered whenever investigating thrombocytopenia as it is easily reversible and can prevent unnecessary work up. We present the first case report which reveals that the  $\beta$ -lactams may have cross-reactivity in causing thrombocytopenia.

**Case Presentation:** A 63-year-old lady presented with abdominal pain, vomiting, and fever. On examination, she had hypotension, abdominal distension and tenderness. Abdominal CT scan showed perforated sigmoid colon. She underwent resection with colostomy and closure of the recto-sigmoid stump. She developed adult respiratory distress syndrome (ARDS) necessitating mechanical ventilation. Wound culture grew extended spectrum  $\beta$ -lactamase producing E coli, hence meropenem was started. Next day, her platelet count started declining, reaching a nadir level of 55,000/ $\mu$ L from 201,000/ $\mu$ L by day 5. An extensive work-up for causes of thrombocytopenia including disseminated intravascular coagulation (DIC) and heparin induced thrombocytopenia (HIT) was negative. Meropenem was replaced with levofloxacin and metronidazole and platelet count rebounded to 269,000/ $\mu$ L, making meropenem the most likely cause. Given her persistent respiratory distress, she underwent a bronchoscopy and broncho-alveolar lavage which showed Aspergillus and Pseudomonas. She was started on caspofungin and piperacillin-tazobactam and from next day, her platelet count started declining again. An extensive thrombocytopenia work-up was repeated and found to be negative for immunological or infectious causes. Piperacillin-tazobactam was believed to be the culprit and was replaced with tobramycin following which her platelet count started improving, returning to her baseline of 239,000/ $\mu$ L.

**Discussion:** The incidence of  $\beta$ -lactam associated DIT is increasing given their widespread usage. The  $\beta$ -lactams usually cause DIT through hapten-mediated immune mechanism and thus, it is possible that there could be cross-reactivity of  $\beta$ -lactams in causing DIT though it hasn't been reported yet. The gold standard for diagnosis is subsequent rise in platelet count on stopping the suspected drug. Measurement of specific antibody against the offending drug had been reported in some studies but it is rarely done given its cost and the limited sensitivity/specificity data. Although IVIG, plasmapheresis, platelet transfusion, and steroids have all been tried for treating DIT, the only proven therapy is discontinuation of the offending drug.

To our knowledge, this is the first case report illustrating the occurrence of cross-reactivity amongst  $\beta$ -lactams in causing DIT. Further reports and studies are warranted to determine the prevalence and the nature of such relationship. Meanwhile, we advocate caution in subsequent use of  $\beta$ -lactams in patients who developed DIT with any member of this class.

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## ALBUTEROL-INDUCED LACTIC ACIDOSIS

First Author: Anna Papazoglou, MD Second Author: Soumya Patnaik, MD Third Author: Samrat Khanna, MD Senior Author: Glenn Eiger, MD

**Introduction:**  $\beta$ -agonist induced lactic acidosis is a rare and under-recognized complication of acute asthma exacerbation treatment. It is a form of type B lactic acidosis that occurs despite improvement in bronchospasm and may cloud physician judgment into increasing  $\beta$ -agonist dosing and thus propagating a vicious cycle.

**Case Presentation:** A 72-year-old woman with past medical history of mild intermittent asthma, hypertension and multiple sclerosis was brought by her family with a two day history of progressively worsening dyspnea and wheezing. The patient soon became unresponsive and pulseless. Resuscitative measures including endotracheal intubation were initiated and spontaneous return of circulation was achieved very quickly. Arterial blood gas after return of spontaneous circulation and initiation of mechanical ventilator support revealed mild lactic and respiratory acidosis (pH of 7.15, pCO<sub>2</sub> of 61 mm Hg and lactic acid of 39.8 mg/dl). The patient was admitted to the intensive care unit in stable hemodynamic condition. Physical exam revealed significant bronchospasm and continuous albuterol nebulization was administered. Chest X-ray revealed hyperinflated lungs without evidence of infection. The patient's clinical condition improved rapidly with resolution of bronchospasm and normalization of peak pressures on ventilator. However, subsequent ABGs revealed gradual worsening of lactic acidosis. There were no signs of bowel ischemia and liver profile was unremarkable. The profound lactic acidosis worsened despite discontinuation of propofol infusion and lactic acid level peaked at 100.2 mg/dl. At that time, it was felt that the high dose inhaled  $\beta$ -agonist was the etiology of the severe metabolic abnormality. Tapering of albuterol resulted in gradual normalization of lactate levels and allowed for prompt patient extubation within 36 hours of presentation.

**Discussion:** Albuterol-induced type B lactic acidosis is a rare, but important cause of severe metabolic acidosis that should be suspected in patients with asthma exacerbation treated with high dose inhaled  $\beta$ -agonists.

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SPLenic INFARCT AFTER IATROGENIC MANIPULATION, IN SETTING OF STABLE AORTIC ATHEROSCLEROSIS

First Author: Sagar Patel, MD Second author: Prasit Phowthongkum, MD

Splenic infarcts are an uncommon cause of morbidity in the general population yet in certain patient populations have a high incidence, usually patients with chronic myelogenous leukemia and myelofibrosis. In 1998, Nores et al reported 59 cases over a 30 year period at UCLA and Cedars-Sinai medical center. Splenic infarctions have also been reported in patients with extensive aortic arch atherosclerosis with or without iatrogenic manipulation. Most reported cases involved unstable atheromas that resulted in splenic infarctions.

We reported a 71 year old male presented to the ER with a 3 day history of nausea, non bilious, non bloody vomiting. Symptoms started a few hours after a vascular procedure for innominate artery dilation. Patient had an AV fistula for hemodialysis due to end stage renal disease with a chronic subclavian vein thrombosis. In ER, he was afebrile but had mild leukocytosis- WBC 12.6. CT of abdomen showed an acute splenic infarct and extensive stable atherosclerosis of the aortic arch. His symptoms were managed with anti-emetics and responded well. TEE was negative for a patent foramen ovale but showed a stable atherosclerotic plaque in ascending aortic arch. Khatibzadeh et al noted that complicated atherosclerotic plaques in the aortic arch presented an independent risk factor for systemic embolism. An article by M Elias et al, noted an incidence of embolic events to the brain of up to 12% and 33% for all embolic events within one year in patients with aortic arch atheromas.

This patient did not have any underlying myeloproliferative disease so did not have the most common risk factor for splenic infarct. Vascular procedures involving the aorta can be a potential cause of splenic infarctions which were previously reported in unstable aortic arch disease such as luminal thrombosis. Our case demonstrated that stable atheroma is a potential cause of systemic embolization. These patients typically have other co-morbidities and splenic infarcts may result in acute decompensation possibly with poorer outcomes in the long term. Use of anticoagulation for prophylaxis or treatment in such patients is controversial. We proposed that high risk patients could benefit from screening for aortic arch atherosclerosis before a procedure involving vascular manipulation.

## A CURIOUS CASE OF EVANS SYNDROME

First Author: Sagar Patel, MD Second author: Gary Huang, MD Third author: Imara Dissanayake, MD

Evans syndrome (ES) is a rare autoimmune condition in which an individual's antibodies attack their own red blood cells and platelets resulting in an autoimmune hemolytic anemia (AIHA) and ITP in the absence of a known etiology. Both of these can occur simultaneously or sequentially. Although many cases are idiopathic in origin, ES has been associated with a number of other conditions in approximately one-half of the cases, including infections (eg, HCV, HIV), systemic lupus erythematosus, lymphoproliferative disorders among others.

We present a case of a 45 year old African American male who was sent to the ED by his primary care physician with a hemoglobin of 6.4 and asymptomatic. The patient has a past history of autoimmune hemolytic anemia (AIHA), immune thrombocytopenic anemia (ITP), anti-phospholipid syndrome (APS) complicated by a previous DVT/PE, non-compliant with warfarin. His labs on presentation were Hgb 6.4, Plts 114, PT 14.3, INR 1.3, T Bil 1.3, D Bil 0.4, LDH 346, Haptoglobin <8, Cr 1.9. He is a known patient to hematology who were involved in his management. His renal function was worse than on his previous visits also with new proteinuria in the nephrotic range. Renal ultrasound was consistent with medical renal disease. ANA was positive 1:160 and double stranded DNA was negative. Anti MPO, P-ANCA, C ANCA, antiproteinase 3, atypical P-ANCA and glomerular basement membrane antibody were negative. CH 50, C3 and C4 were all low. No cryoglobulins were detected in the blood assay. Urine Electrophoresis was negative for monoclonal light chains. Nephrology recommended renal biopsy which was performed and showed findings consistent with lupus nephritis stage 4/5. The patient was transfused with PRBCs and given pulse dose steroids for 3 days after which his hemoglobin remained stable. Rheumatology decided upon chronic immunosuppression for the patient when followed up as out-patient.

ES is a rare condition because it is diagnosed in only 0.8% to 3.7% of all patients with either ITP or AIHA at onset. It is thought to be secondary to immune dysfunction and therefore a high clinical suspicion should be maintained for underlying autoimmune disease. This patient was unique in that he did not meet the criteria for SLE but demonstrated lupus nephritis on biopsy. This has a big implication in management by treating him for his underlying autoimmune condition which could help in reducing recurrence of Evans syndrome. He responded well to first line therapy with corticosteroids and did not require further acute therapy such as IVIG. He was also started on hydroxychloroquine for SLE. There should be a high suspicion for autoimmune and lymphoproliferative diseases even in asymptomatic patients with Evans Syndrome.

AN INTERNIST'S PERSPECTIVE OF A CHALLENGING SITUATION: INTRACTABLE LOWER GASTROINTESTINAL BLEEDING FROM RECURRENT SUPERIOR MESENTRIC VEIN THROMBOSIS.

First Author: Soumya Patnaik, MD Additional Authors: Alvin Htut, MD Simona Rossi, MD Jeffery Weinstein, MD

Background: Cirrhotic patients are prone to thrombosis, particularly of the portal system. Chronic mesenteric-vein thrombosis may be incidental on imaging. Lower gastrointestinal (GI) bleeding due to bowel varices from superior mesenteric vein (SMV) thrombus is rare. We present such a case, that was successfully managed with Transjugular Intrahepatic portosystemic shunt (TIPS) followed by Balloon-occluded retrograde transvenous obliteration (BRTO) of the varices.

Case vignette: A 50-year-old African-American male presented with 3 days history of syncopal episodes and bright red blood per rectum, leading to severe anemia (Hb-5.1gm/dl). An upper GI endoscopy revealed diminutive varices and portal hypertensive gastropathy. Colonoscopy showed large blood clots without any clear source. CT radionuclide bleeding scan revealed small bowel varices in right lower quadrant. Abdominal ultrasound with Doppler showed liver-cirrhosis, moderate ascites, patent main portal and hepatic vasculature but thrombosed SMV, reconfirmed by MRI.

Patient underwent TIPS with SMV thrombectomy, despite which he developed recurrent SMV thrombosis and consequent melena. TIPS was also occluded with thrombus. TIPS thrombus was removed by pharmacologic agents (TPA) and mechanical thrombectomy (angioplasty). AngioJet SMV thrombectomy was then performed. Balloon maceration and Fogarty of residual SMV-thrombus attempts were unsuccessful. Self-expanding Nitinol stent was placed in SMV. These small bowel varices were noted to communicate with right gonadal vein, a porto-systemic shunt (sequelae of chronic SMV-thrombosis). He continued to bleed, requiring several transfusions. Subsequently, he underwent BRTO procedure (Balloon-occluded retrograde transvenous obliteration) of these small bowel varices. An occlusion balloon catheter was advanced into the feeding gonadal branch. Sclerosant (5% ethanolamine mixed with iodinated contrast) was administered through balloon under fluoroscopy and varicosities were filled. After 30 minutes, sclerosant was aspirated and balloon was removed. Post-procedure, he was started on anti-coagulation, in view of recurrent thrombosis. Recovery was satisfactory, without any further bleeding episodes at 2 months follow-up.

Conclusion: BRTO is usually performed in portal hypertension for gastric varices, especially, if the patient is unable to tolerate TIPS due to elevated MELD-scores. On occasions, BRTO has, been reported to be useful in patients with bleeding small bowel varices, particularly for duodenal varices. BRTO of jejunal and ileal varices is rare. BRTO may be successfully tried in selected patients, with recurrent bleeding from small bowel varices, even during acute episodes. Rebleeding risks and serious complication rates are low.

Internists must be aware of possibility of small bowel varices due to SMV thrombosis as a cause of recurrent lower GI bleed in cirrhosis and the newer procedures that are available to manage such situations. Multidisciplinary care of such patients appropriately coordinated by the internist is essential for good outcomes.

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NEAR FATAL TORSADES DE POINTES SECONDARY TO CONCURRENT USE OF HIGH DOSE METHADONE, FLUOXETINE, AND FUROSEMIDE INDUCED ELECTROLYTE ABNORMALITIES

First Author: Sanjay K Saxena, MD Additional Authors: Qi Shi MD, Nishith Vayada MD, Sridar Sampath Kumar MD FACC

**Background:** Long QT syndrome is a disorder of myocardial repolarization associated with increased risk of Torsades de Pointes. Primary symptoms include palpitations, syncope, seizures, and sudden cardiac death. It usually results from drug therapy, hypokalemia, and/or hypomagnesemia. Without proper monitoring of drug administration and dosing, the chances of developing this potentially fatal condition are very real.

**Case Description:** A 55 year old woman with CAD post CABG, Hypertension, Hyperlipidemia, Chronic pain syndrome on Methadone 100 mg bid, Depression on Fluoxetine 20 mg tid, Diastolic Heart Failure on Lasix 120 mg bid and COPD reported to the ED following an episode of syncope at 2:30 AM. She was asymptomatic at presentation. Examination was normal except for bilateral basilar crackles. EKG showed sinus bradycardia, LVH and QTc of 537. At 8:30 AM, six hours following the initial syncopal episode, the patient developed Torsades de pointes and ventricular fibrillation. She was resuscitated per ACLS protocol with concurrent replacement of magnesium and potassium. Sixteen DC shocks were delivered during resuscitation. An emergency cardiac catheterization was done to exclude an ischemic event. Patent grafts were noted along with spasm of radial artery graft that resolved with locally instilled nitroglycerin via cardiac catheter. The patient regained consciousness after cardiac catheterization. Mechanical ventilation and supportive therapy were continued along with fluid and electrolyte management. She had an uneventful recovery and was discharged home with appropriate dose adjustment of furosemide and discontinuation of offending agents, particularly, methadone and fluoxetine.

**Discussion:** Drugs are a common cause of acquired LQTS and TdP. It is usually short-lived and terminates spontaneously. Most patients experience multiple episodes of the arrhythmia which may recur in rapid succession, degenerating to ventricular fibrillation and potential SCD. Acquired LQTS is caused by many drugs. Sensitivity to these medications may be related to genetic causes. It is important to avoid multiple drug combinations known to cause QT prolongation. Our case demonstrates the dangers of concurrent administration of QT prolonging agents with multiple drug therapy. Appropriate medication adjustments are necessary to reduce the risk of SCD.

## IS IT ALL LUPUS?

First Author: Mitali Sen, MD Second Author: Sandra Schwarcz, DO Third Author: Ruchika Patel, MD

**INTRODUCTION:** A case of known SLE and Discoid Lupus admitted for bullous skin rash which worsened morphologically despite aggressive immunosuppression.

**CASE DESCRIPTION:** 46 year old African-American female with history of Systemic Lupus Erythematosus, Discoid Lupus and Fibromyalgia since 16 years on a regime of Hydroxychloroquin 400mg, Prednisone 35 mg and Mycophenolate 1000mg and Cyclosporine 100mg and having used Rituximab, Belimumab and Acthar gel in the past presented to the hospital with nausea, body aches and a recurrence of a malar rash and discoid lesions on bilateral elbows and knees. Blood work revealed chronic leukopenia (WBC 1.5-3) with persistently low complement levels (C4-3 & C3-46) with known Anti DS-DNA and Anti-smith positivity and IgM-Cardiolipin level of 24.

On admission she received 3 day pulse dose of prednisone followed by 250 mg daily dose of methylprednisone which led to resolution of some lesions but it was soon followed by appearance of new lesions on her face, ears, eyes, chest, abdomen and thighs with morphological evolution of the rash to form painful desquamating bullous lesions filled with serous fluid with positive Nikolsky sign within a span of one week.

With the possibility of Bullous Lupus in mind, multiple samples of skin were sent for histo-pathological examination. The dosage of mycophenolate was increased to 1 gm BID and Dapsone was initiated. She was also evaluated by Ophthalmology for iritis in bilateral eyes and by ENT for bullous lesion in the external ear which were managed topically.

However, Skin Biopsy eventually revealed the presence of interface vacuolar dermatitis with cytoid bodies seen in Lupus but also described a superficial acantholysis at the granular layer which can be seen in Pemphigus foliaceus or in Staphylococcal scalded skin syndrome. Direct Immunofluorescence showed a linear IgG and IgM deposition at the Dermo-Epidermal junction. Meanwhile, microbiological examination of serous fluid grew Staphylococcus aureus. Hence, the final biopsy result suggested a combined presence of a flare of Lupus Erythematosus with a Pemphigus foliaceus and treatment was initiated accordingly.

**DISCUSSION:** This case emphasizes the importance of maintaining a broad differential diagnosis and to recognize that a patient with a longstanding history of disease such as lupus can have the presence of a comorbid condition and situation like such requires prompt identification and treatment. One should consider an alternate diagnosis when the suspected disease fails to adequately respond to aggressive treatment.

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MULTIPLE MYELOMA AS AN UNUSUAL ETIOLOGY OF BACK PAIN DURING PREGNANCY

First Author: Mansi R Shah, MD

**Introduction:** Multiple Myeloma (MM) is a malignant plasma cell dyscrasia that accounts for approximately 13% of hematologic malignancies. Although most commonly diagnosed in the sixth decade of life, the incidence is around 3% in patients younger than 40 years of age—and even less frequently found in pregnant patients under the age of 30. Diagnosis is defined by the presence of  $\geq 10\%$  clonal plasma cells in the body or extramedullary plasmacytoma plus evidence of secondary end-organ damage, which can manifest as hypercalcemia, renal insufficiency, anemia, or bony lesions. Treatment depends on risk stratification but usually involves induction chemotherapy with the ultimate goal towards early or delayed stem cell transplant.

**Case Description:** A previously healthy, G1P1 woman presented to the emergency room with fatigue and lower back pain of 3 weeks duration. Physical exam was only significant for mild tenderness at the thoracic spine region without evidence of step off or instability. Initial tests revealed severe hypercalcemia 18.7, anemia, and renal insufficiency with 10 gram proteinuria. Imaging studies revealed an area of hyperlucency on the proximal left humerus and a sacral mass concerning for multiple myeloma. Other causes of hypercalcemia were ruled out with normal parathyroid hormone level (PTH), PTH-related peptide, and vitamin D levels. Subsequent work up including CT guided biopsy of the sacral mass was consistent with a free kappa light chain plasmacytoma and serum and urine electrophoresis with immunofixation revealed free kappa: lambda light chain ratio  $>500$ , further solidifying the diagnosis of multiple myeloma. The patient's degree of hypercalcemia, beta 2 microglobulin level, and an RB1 deletion found on cytogenetics, are all associated with poor prognosis.

The patient's hypercalcemia and bony instability were initially treated with aggressive IV hydration, calcitonin, dexamethasone, and pamidronate. Though initially successful, the patient continued to have severe back pain and rising calcium levels. She underwent an urgent but successful C-section followed by rapid initiation of radiation therapy and bortezomib. Her hospital course was further complicated by cauda equine syndrome while undergoing radiation therapy requiring an emergent sacral decompression. She received additional doses of dexamethasone and bortezomib with the goal to achieve complete remission as a step towards bone marrow transplant.

**Discussion:** This case represents a unique population of patients diagnosed with multiple myeloma who present characteristic management challenges. Though rare, multiple myeloma should be included in the differential diagnosis of back pain associated with symptoms of hypercalcemia, anemia, or renal failure during pregnancy—symptoms that may otherwise be nonspecific. Consideration of maternal and fetal health should be taken when initiating treatment. Moreover, pregnancy may serve as a prognostic indicator in the setting of immune tolerance, a subject that warrants additional research.

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FEVER, ACUTE KIDNEY INJURY (AKI), THROMBOCYTOPENIA & HEMOLYTIC ANEMIA WITH SCHISTOCYTES– BABESIOSIS MIMICKING THROMBOTIC THROMBOCYTOPENIC PURPURA (TTP) SYMPTOMOLOGY

Harshal P. Shah, DO, Dhvani Y. Pandya, MD and Timothy Coyle, DO

**Introduction:** Babesiosis is an infectious disease predominantly caused by parasites of the genus *Babesia microti*, with presentation most common in individuals with splenectomy and/or immunosuppression – often manifesting systemic signs and symptoms of infection with peripheral count indicative of thrombocytopenia with transaminitis. Herein, we describe a case of Babesiosis with presentation representative of TTP in an immunocompetent, normosplenic gentleman.

**Case Report:** An 82 year old healthy Caucasian male was admitted with chief complaint of fatigue, lightheadedness, subjective fevers, generalized abdominal pain and poor appetite for two weeks. Upon admission, he had a 101.1F fever with new-onset anemia, thrombocytopenia and AKI with normal WBC. Further testing was consistent with a hemolytic anemia with negative blood cultures. A peripheral smear performed showed rare schistocytes. Given the concern for TTP, a repeat smear showed intra and extracellular inclusions with parasitic cultures positive for *B. microti*. Treatment was initiated with atovaquone, azithromycin and doxycycline for potential co-infections.

**Discussion:** In an elderly patient that presents with fevers, AKI, thrombocytopenia and hemolytic anemia; with smears showing schistocytes – a diagnosis of TTP is strongly suspected, especially with concern for underlying malignancy. However, in our patient, smears showed schistocytes with intra and extracellular inclusions with parasite cultures diagnosing *B. microti* – with 1% parasitemia. Given this finding, it becomes imperative to consider this parasitic infection as part of our differential diagnosis in patients presenting with TTP-like symptoms. Prompt recognition with peripheral smear morphology can prevent inappropriate treatment and prevent progression of disease.

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FROM TONGUE TO HEART - MYOCARDIAL METASTASIS IN SQUAMOUS CELL CARCINOMA OF THE TONGUE

First Author: Hasan K. Siddiqi Second Author: Jana Goldberg Third Author: Frank E. Silvestry

**Introduction:** Squamous cell carcinoma (SCC) of the tongue is among the most prevalent tumors of the head and neck, and carries a poor prognosis. The most common site of metastasis is the cervical region and lymph nodes; metastases to the lung, mediastinal lymph nodes, liver and bone are also described. Cardiac metastases are extremely rare. Here we present a patient with metastatic SCC to the heart that manifested as ischemic heart disease.

**Case Description:** A 51-year-old female with recurrent T1N0M0 anterior tongue SCC presented with several weeks of breathlessness, dyspnea on exertion and squeezing chest pressure. Given concern for cardiac etiology, an outpatient treadmill stress test was performed and showed thinning and akinesis of the inferior wall of the left ventricle, suggesting inferior wall infarction. She underwent CT coronary angiography, which revealed an intermediate density right-sided myocardial mass invading the myocardium of the right atrium (RA) and ventricle, and replacing the right atrial appendage and right atrioventricular septum. The mass also caused obstruction of the right coronary artery. Transesophageal echocardiogram (TEE) demonstrated similar findings, but also noted a mobile 2 cm long satellite component superimposed on the RA mass. Additionally, there was Doppler evidence of tricuspid inflow obstruction secondary to the mass. A CT pulmonary angiogram was negative for pulmonary embolism, and cardiac MRI confirmed the above findings.

She was admitted and initiated on a heparin drip due to concern for intraatrial thrombus. TEE guided biopsy was performed and tissue showed myocardium with mild myocyte hypertrophy, interstitial fibrosis and necrosis with rare atypical cells, but no viable tumor cells.

Cardiac surgery, oncology and radiation oncology were consulted, and given extensive myocardial invasion, no cardiac surgical resection option was felt possible, even for palliation. The patient and surgical team declined direct biopsy through a sternotomy. The patient decided to pursue palliative radiation (XRT), with anticoagulation for the RA thrombus. She completed 6 cycles of XRT, and reported improvement in symptoms of dyspnea and chest pressure, but had worsening cough, anorexia and weight loss. Follow up imaging five months after cardiac mass diagnosis showed new metastatic disease to the lungs, and she is now pursuing palliative chemotherapy.

**Discussion:** Cardiac metastatic tumors are rare, with an autopsy proven incidence of fewer than 20% in all malignancies. Only 19 case reports of cardiac metastasis from oral SCC were found in the literature, with most reporting mortality weeks after detection. Common presentations included dyspnea, angina, arrhythmia, syncope, embolic phenomenon, and death. Our patient is alive at 5 months after detection of the cardiac metastasis, which is the longest survival in published reports.

UTILITY OF RIGHT VENTRICULAR ASSIST DEVICE IN A HEMODYNAMICALLY UNSTABLE PATIENT WITH RV FAILURE SECONDARY TO CLASS I PULMONARY HYPERTENSION

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**INTRODUCTION** Right ventricular assist devices (RVAD) are used in the setting of right ventricular failure following cardiac surgery, left ventricular assist device implantation or after acute myocardial infarction. We report the use of percutaneous RVAD in a patient presenting with acute right heart failure secondary to idiopathic pulmonary hypertension.

**CASE A** 69-year-old woman with history of hypertension presented with four days of shortness of breath and malaise. On initial exam she was found to have SaO<sub>2</sub> of 66% on room air, tachycardic, with bibasilar rales. Chest X-ray revealed bilateral pleural effusions, EKG showed right axis deviation. Initial laboratory testing was unremarkable. Transthoracic echocardiogram revealed severely dilated and hypocontractile right ventricle (RV) with severe pulmonary hypertension (PH) and an underfilled D-shaped left ventricle (LV) with preserved ejection fraction. CT angiography of the chest was negative for pulmonary embolism.

Patient progressively became more hypoxic requiring intubation by day 3. Dobutamine was initiated for right ventricular support. She underwent right heart catheterization (RHC) revealing pulmonary artery (PA) pressure of 74/34mmHg, pulmonary capillary wedge pressure of 12mmHg, cardiac index (CI) of 2.2 liters/minute/m<sup>2</sup> and pulmonary vascular resistance (PVR) of 11 Wood units. Patient continued to deteriorate the next day requiring 100% FiO<sub>2</sub> and endured three PEA codes requiring four pressors. At this point a percutaneous RVAD (RP-Impella device, Abiomed) was placed for RV support. This device has its inflow cannula in the inferior vena cava and outflow cannula in the main PA. Shortly after placement of the RVAD, LV filling improved with improved blood pressure. We could then start pulmonary vasodilators including sildenafil and inhaled epoprostenol to reduce PVR without causing hemodynamic compromise. Pressors and ventilator requirements were weaned and the RVAD was removed after 3 days. She was continued on milrinone (inodilator) while receiving sildenafil and epoprostenol which initially maintained cardiac index around 2.5 liters/min/m<sup>2</sup>. However her respiratory requirements gradually worsened. Unfortunately the patient was not a candidate for lung transplantation due to her age and died four days after RVAD removal.

**Discussion** This case suggests the benefit of temporary RVAD in the setting of cardiogenic shock resulting from right heart failure from severely elevated PVR. RVAD support improved LV filling, thus increasing the cardiac output and aortic pressure, while raising pulmonary arterial pressures. Using an RVAD allowed us to initiate selective pulmonary vasodilators without risk of hypotension. The poor outcome was due to inability to consider her for urgent lung transplantation, the only definitive treatment in this case. To conclude, this strategy can be useful in acute decompensation from class I pulmonary hypertension, especially as a potential bridge to lung transplantation.

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DISSEMINATED STRONGYLOIDIASIS: AN UNSUSPECTED CAUSE OF MENINGITIS.

First Author: Sandra Algaze, MD Stephanie Clauss, MD

The infrequency with which disseminated strongyloidiasis is encountered and its non-specific presentation poses a diagnostic challenge.

A 50 year old man with unknown history was taken to the Emergency Department after he was found to be confused. On presentation, he was febrile, hypertensive, tachycardic, awake, yet obtunded, agitated, opening eyes spontaneously, withdrawing to pain, and producing only incomprehensible moaning sounds. Brain CT revealed no acute processes and severe cortical atrophy for his age. He was found to have hyperglycemia, and leukocytosis with neutrophilia with urine cultures positive for pan-sensitive *ESCHERICHIA COLI* and was started on levofloxacin. His mental status continued to decline and he developed severe sepsis with exam findings significant for positive meningeal signs and new onset of a maculopapular rash on his back and abdomen. Lumbar puncture was performed revealing frank purulent fluid with pleocytosis and elevated CSF protein (CSF:144K cells, PMN 78%, glucose 50, protein >600). He was started on Vancomycin, Meropenem, and Ampicillin for empiric treatment of bacterial meningitis and acyclovir for empiric treatment of viral encephalitis. He developed acute hypoxic respiratory failure requiring intubation and mechanical ventilation. As several loose stools and increasing eosinophilia were noted, stool ova and parasite analysis were sent and found to be positive for *STRONGYLOIDES STERCORALIS*. Additional tests were ordered including a bronchoalveolar lavage and repeat LP which were also positive for *STRONGYLOIDES STERCORALIS* and the diagnosis of disseminated strongyloidiasis was made. Additionally, HIV returned positive with a CD4 count of 317 and viral load of 98,333 and Hepatitis C was also positive. The patient was started on oral ivermectin daily and transitioned to subcutaneous ivermectin every other day when it became available. As the patient remained critically ill without significant improvement, albendazole was started and the patient was restarted on oral ivermectin daily. The patient remains in critical condition in the intensive care unit and has shown only minimal improvement in mental status thus far on this therapy.

Discussion: While there is a paucity of case reports describing disseminated strongyloides, *STRONGYLOIDES STERCORALIS* infection should be considered in patients presenting with bacterial meningitis, particularly if accompanied by gastrointestinal and respiratory symptoms, in endemic regions, and in immunocompromised patients. It is very important to not only consider the textbook diseases that HIV patients are at high risk for such as Toxoplasmosis and CMV, but also to consider other less common sources of infection such as Strongyloides keeping in mind that it may present as a disseminated and uncommon form of meningitis in HIV patients. In fact, Strongyloidiasis was once considered an AIDS-defining illness. Unfortunately, even with timely diagnosis and approved therapy, disseminated strongyloidiasis portends poor prognosis.

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MESENTERIC FIBROMATOSIS: A RARE AND COMPLICATED CAUSE OF ABDOMINAL PAIN

First Author: Anjanet Perez-Colon Other Authors: Ana Velazquez-Manana, Genta Ishikawa, Coral Olazagasti, Chris Taurani

**Introduction:** Mesenteric Fibromatosis (MF) is a rare intra-abdominal Desmoid tumor. We describe a rapidly progressing and complicated case of MF in a young male with no associated factors. Because of their rarity, these tumors pose a diagnostic and therapeutic challenge in which aggressive and proper management is needed to avoid life-threatening complications and increased morbidity.

**Case Description:** A 22 year-old male with no medical or surgical history presented with 4 days of sharp epigastric pain radiating to the right lower abdomen. Pain was associated with chills, anorexia, fatigue, bloating, and nausea. He had occasional alcohol use, otherwise no toxic habits and no family history. Physical exam was significant for tachycardia, mild tenderness over the epigastrium and right lower abdomen, and voluntary guarding. Urinalysis showed microscopic hematuria and amorphous phosphate crystals, labs were otherwise normal. A CT abdomen-pelvis obtained for possible nephrolithiasis showed lymphadenopathy and a 10 cm soft tissue mass encasing the distal duodenum, mid to distal jejunum, and transverse colon, and infiltrating the mesenteric fat, and superior mesenteric artery and vein. Further workup for malignancy was unremarkable, including: alpha fetoprotein, hCG, LDH, uric acid, HIV, viral hepatitis, and testicular US. EGD with EUS showed a 7cm hypoechoic irregular homogenous mass in the periduodenal area with atypical and degenerated bare nuclei on cytology. Hospital course was complicated by *Bacteroides fragilis* and *E. coli* bacteremia and colon perforation. The patient underwent emergent tumor resection en bloc, extended right ileocollectomy, and ileoileal anastomosis. Pathology revealed mesenteric fibromatosis,  $\beta$ -catenin positive, with resection margins free of tumor. Abdominal abscesses requiring percutaneous drainage and a reactive pleural effusion complicated his post-operative course. Since the resection margins were free of tumor, oncology recommended close surveillance for recurrence without cytotoxic therapy.

**Discussion:** Mesenteric Fibromatosis (MF) is a rare and locally invasive type of intra-abdominal desmoid tumor. MF has a high rate of recurrence but poor metastatic potential. Clinical presentation ranges from asymptomatic patients to most commonly a new painless mass. Few cases have been reported describing acute abdominal pain as the initial presentation and rapid progression like our patient. MF develops from the proliferation of fibroblasts in the mesentery; mutations in the  $\beta$ -catenin or the adenomatous polyposis coli genes have been described as possible mechanisms. This tumor can appear de-novo but it is commonly associated with Gardner syndrome, occurring in <10% of patients. It has also been described in rare association with Crohn's disease, pregnancy, trauma or previous abdominal surgery. As tumor progresses in size it involves more mesentery increasing its morbidity and potential of life-threatening complications; this is why we emphasize the importance of considering it in the differential of abdominal tumors. Radical resection with wide margins is the principal aim of management and is recommended even in asymptomatic patients. Alternative treatment options include radiotherapy and systemic chemotherapy for unresectable cases.

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A CONSEQUENCE OF BEING TREATED; CORONARY ARTERY DISEASE IN A YOUNG WOMAN AFTER RADIATION THERAPY

First Author: Christina Rodriguez

Heart Disease and cancer are the two most common causes of death in the world. Radiotherapy has led to the successful treatment of multiple malignancies, often leading to a definite cure. As practicing internists, it is imperative to recognize subsequent cardiovascular disease secondary to cancer treatment. With improving survival rates, delayed cardiac complications have been recognized, with one of the most severe being radiation-induced coronary artery disease.

We present the case of a 31-year-old woman with a history of hypothyroidism and Hodgkin's Lymphoma who presented to our institution with a one week history of oppressive chest discomfort and dyspnea associated to minimal amounts of exertion. Eight years prior to evaluation she underwent successful therapy of her lymphoma with 34 courses of radiation and systemic chemotherapy. She had no family history of heart failure or premature coronary artery disease. Physical examination was remarkable for jugular venous distention, an S3 gallop, decreased breath sounds at both lung bases, and trace bilateral pedal edema. Laboratory studies revealed elevated pro-BNP levels. Other laboratory tests including ANA, electrolytes, and lipid profile were unremarkable. The electrocardiogram performed upon admission revealed a left bundle branch block. A transthoracic echocardiogram study confirmed mild left atrial enlargement, global hypokinesis, and moderate left ventricular systolic dysfunction with an estimated left ventricular ejection fraction of 40%. Left heart catheterization was performed and revealed a critical 99% occluded proximal left main coronary artery. She underwent subsequent coronary artery bypass grafting with improvement of symptoms and is currently doing well with close follow-up with her primary Cardiologist.

This case illustrates the potentially serious complications of cancer therapy, in particular radiotherapy. It stresses the need for this population to receive proper cardiovascular surveillance after treatment. There are currently no established guidelines for the screening of radiation-induced coronary artery disease. Taking into consideration that radiotherapy affects all cardiac structures, and that coronary atherosclerosis is markedly accelerated, early recognition of asymptomatic patients is imperative to avoid potentially fatal complications. Radiation-induced coronary artery disease is usually present at a mean of 6-8 years after therapy, stressing the importance of careful follow up. As physicians we do not just have the responsibility to acutely treat our patients, but it's also crucial that we do not overlook the consequences and risks of our own treatments.

## A GROWN UP MAN WITH THE HEART OF A CHILD: DELAYED PRESENTATION OF CONGENITAL AV FISTULA MALFORMATION

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Coronary arteriovenous fistulas are a rare and abnormal connection between a coronary artery and another chamber or vessel. Most are due to congenital malformations and are diagnosed during childhood. Incidence accounts for 0.2%-0.4% of the population, 40% drain into the right ventricle, 26% into the right atrium, 17% into the pulmonary artery, 3% into the left ventricle, 7% into the coronary sinus and 1% into the superior vena cava. They most commonly arise from the RCA or LDA, and rarely involve the circumflex artery.

This is a case of a 62 y/o Hispanic male patient who presented in 2015 complaining of chest pain, dyspnea, lightheadness and palpitations. Past medical history was only significant for hyperlipidemia and hypertension. The patient's medications included Hyzaar, Metoprolol, Methyldopa, Amilodipine and Simvastatin. In 2011, the patient reported prior symptoms of palpitations and lightheadness. Holter was done and showed normal rhythm and PVCs. Stress test was negative for ischemia and TTE showed mild dilation of the left atrium and left ventricle, and an EF of 63%. The patient returned in 2014 complaining of lightheadness and palpitations. Cardiac studies were done. EKG showed atrial fibrillation. TTE showed mild dilation of the right atrium, left atrium, and left ventricle, and EF of 50%. The patient was placed in Xarelto and was cardioverted successfully. Cardiac studies were done in 2015 and EKG once again showed atrial fibrillation. TEE guided cardiac ablation was performed, showing a structure with continuous high velocity blood flow right next to the aortic valve. Cardiac catheterization results showed RA O<sub>2</sub> Sat 83%, PA O<sub>2</sub>Sat 77% suggesting intracardiac shunting. Additionally, a coronary arteriovenous fistula was visualized arising from mid portion of circumflex artery, draining into the superior vena cava. CT Chest angiogram revealed a tortuous dilated vessel originating from the midportion of circumflex artery and draining into the SVC via a posterior approach between the right main pulmonary artery and the left ventricle. The patient was scheduled for surgery. The surgery report showed a tortuous fistula measuring 1.5 cm diameter ending in the SVC. Ventricles were found to be poorly developed with an atrophic myocardium. Also CABGx2, RCA to RIMA and SVG to OM1 was performed. The patient was discharged home. The symptoms of chest pain, dyspnea, and palpitations resolved but asymptomatic atrial fibrillation persisted.

Coronary artery fistulas most commonly manifest with dyspnea and fatigue. Older patients may present with atrial fibrillation due to enlargement of the atria caused by the fistula. If left untreated, patients with coronary arteriovenous fistulas demonstrate 35% mortality. Congenital fistulas are present since birth and over the years they progressively enlarge. If the diagnosis is not made this increase in size will lead to the most serious complication, spontaneous rupture.

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## JUST A SORE THROAT?: A CASE OF LARYNGEAL TUBERCULOSIS

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**Introduction:** Despite advances in therapy, tuberculosis represents a major burden of disease worldwide with 9 million cases and 1.5 million deaths annually. Laryngeal tuberculosis, once a common presentation of *Mycobacterium tuberculosis* infection, has become increasingly rare since the advent of effective anti tubercular therapy. The following describes a case of laryngeal tuberculosis presenting as acute epiglottitis.

**Case report:** A 48-year-old man with a history of binge drinking and a positive PPD 20 years prior presented to the emergency department complaining of worsening sore throat, dysphagia and hoarseness for 1 week. Review of systems revealed a 15-pound weight loss and productive cough over the previous 6 months. He migrated to the United States from Guatemala 22 years prior to presentation and denied any exposure to tuberculosis. Physical examination was remarkable for hoarseness and oropharyngeal thrush. Computed tomography (CT) of the neck revealed epiglottitis and CT of the chest showed a large cavitory lesion in the right upper lobe. Flexible laryngoscopy revealed supraglottic edema as well as granular, superficial ulceration on the laryngeal surface of the epiglottis. Laboratory findings were remarkable for a lymphocyte count of 300 cells/ $\mu$ l. Anti-HIV antibody, respiratory viral panel, legionella and *Streptococcus pneumoniae* urine antigens, and blood cultures were negative. Sputum samples showed numerous acid fast bacilli and nucleic acid testing confirmed the presence of *Mycobacterium tuberculosis* organisms. His oropharyngeal symptoms improved with fluconazole and dexamethasone. He was started on isoniazid, rifampin, ethambutol, and pyrazinamide for laryngeal and pulmonary tuberculosis and was eventually discharged home with isolation precautions after coordination with the State Department of Health.

**Discussion:** Laryngeal tuberculosis (LT) is an increasingly rare manifestation of tuberculosis, now seen in only 1% of cases. Diagnosis requires a high index of suspicion, particularly in US natives or migrants who have resided in the US for a long time, as LT is frequently confused with laryngeal carcinoma in these populations. On laryngoscopy, lesions vary in both appearance and location, and may appear ulcerative, ulcerofungative, or polypoid.

While LT is typically associated with concomitant pulmonary tuberculosis, as seen in our case, up to 20% of LT cases present as primary disease. Typically LT presents in men in their forties or fifties and the most frequent complaints are dysphonia and weight loss. Risk factors include lack of prior vaccination with BCG, tobacco or alcohol use, acquired immunosuppression or malnutrition. Our patient serves as a reminder to maintain a high clinical suspicion for tuberculosis in this vulnerable population.

TORSADES DE POINTES IN THE SETTING OF HYPEREMESIS GRAVIDARUM: RARE ARRHYTHMIA BY A COMMON ISSUE DURING PREGNANCY

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**Introduction:** The risk of cardiac arrhythmias is elevated in pregnancy, even in those with structurally normal hearts and without previous medical history. Electrolyte abnormalities, including hypokalemia, are common complications of hyperemesis gravidarum which can predispose to fatal arrhythmias. We present a case of severe hypokalemia in the setting of hyperemesis gravidarum, leading to QT prolongation and subsequent Torsades de Pointes (TdP).

**Case Presentation:** A 26-year-old woman, gravida 4, para 3, at 6 weeks' gestation was brought to the emergency department (ED) for severe nausea and vomiting. Her workup revealed a positive urine HCG and she was discharged home with a prescription for ondansetron. However, her symptoms did not improve and she returned to the ED four days later where she was found to have creatinine of 6.38 mg/dL and marked hypokalemia with potassium of 2.0mmol/L as well as magnesium of 2.8 mg/dL. A 12-lead Electrocardiogram (EKG) in the ED revealed sinus tachycardia with rate of 144 beats per minute and QTc of 612 msec. Aggressive intravenous hydration and potassium supplementation was initiated in the ED and she was transferred to intensive care unit (ICU).

One hour after admission to the ICU, frequent premature ventricular contractions (PVC) were noted on telemetry with episodes of non-sustained ventricular tachycardia (VT) with runs of 3, 4, and 7 beats. Ten minutes later the patient developed TdP, initiated by a PVC, following a long R-R interval. Cardiopulmonary resuscitation (CPR) was immediately initiated and sinus rhythm was successfully restored after a single 200J biphasic shock within 2 minutes of the arrhythmia onset. She was also given 2 gram of magnesium sulfate. Status post the cardioversion the potassium was 2.4 mmol/L with a magnesium of 3.2 mg/dL. Transthoracic echocardiography on the following day revealed no significant structural heart disease and normal systolic function.

Her nausea and vomiting improved with doxylamine and diphenhydramine. On day 3 of admission, her potassium and QTc normalized to 4.5 mmol/L and 474 msec, respectively. No further ventricular tachyarrhythmia occurred and she was discharged home with an event monitor. The patient had an elective abortion the day after discharge.

**Discussion:** The risk of cardiac arrhythmias is particularly high in pregnant women even without previous health problems and with normal heart structure. Though TdP is rare among healthy subjects, this case highlights a potential complication of hyperemesis gravidarum when coupled with a medication known to prolongate the QTc (ondansetron). In cases of hyperemesis gravidum, careful monitoring of electrolytes and QTc, along with judicious use of medications known to cause QT prolongation is recommended.

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THE DANGER OF A BENIGN BILIARY STRICTURE: A CASE OF MULTIDISCIPLINARY CARE

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Interpersonal and Communication skills are among the most important of the core Internal Medicine competencies. Good communication can aid in the prevention of medical error and foster collaborative interprofessional relationships that benefit both patients and physicians. Despite this emphasis, studies show that patients often do not understand what physicians explain, and both physicians and patients have trouble identifying when there is a lack of comprehension. This poses a risk for future medical error.

GD is a 54 year old woman with a history of fibromatosis tumor of the gallbladder status post cholecystectomy (CCY) twenty years prior who presented with weakness and hypoglycemia. Her physical exam was significant for a blood pressure of 85/63, a heart rate in the 100s, and a benign abdominal exam. Laboratory examination showed a leukocytosis of 20.6 with 3% bands and an elevated alkaline phosphatase of 354 IU/L with normal AST and ALT. CT of the abdomen showed multiple hepatic abscesses, the largest at 10cm. Subsequent MRCP and ERCP revealed a common bile duct stricture.

GD responded well to fluid resuscitation. She had two hepatic abscess drains placed and was started on ciprofloxacin and metronidazole. In addition, she had a biliary drain placed due to the stricture. Afterwards she developed abdominal pain which worsened upon the action of flushing the biliary drain. These symptoms were accompanied by elevation of her alkaline phosphatase, which had previously been decreasing. Biopsy of the stricture showed no malignancy, but did show vegetable matter. Due to her persistent symptoms, a multidisciplinary meeting of Gastroenterology, Surgery, and Interventional Radiology consultants met with the primary team and reviewed imaging and hospital course together. Imaging revealed that, at the time of her CCY, her surgeon had created an internal biliary drain (likely a choledochojejunostomy) that the patient did not know about. Intestinal contents had been refluxing through that conduit, resulting in abscess formation. When the biliary drain was placed, it entered the anastomosis and caused her to have biliary obstruction. Removal of the drain immediately eased her abdominal pain. All of the drains were eventually removed and she completed ten weeks of antibiotic therapy with resolution of abscesses on follow up imaging.

Physicians today strive to practice personalized medicine, but this can be challenging when the medical history of a patient is unclear. GD did not realize she had a choledochojejunostomy, nor did her physicians. As a result, she suffered an iatrogenic biliary obstruction, increasing her pain and prolonging her hospitalization significantly. The cause of her illness was unknown until all of the specialists met, thoroughly interviewed the patient, and reviewed all of her prior imaging together. It took a large multidisciplinary effort to successfully discover what had happened, appropriately treat the patient, and ensure that she had adequate follow up and education. This case exemplifies the benefits of good interpersonal relationships and communication with colleagues, as well as with patients.

## HEMORRHAGIC PNEUMONIA: A TYPICALLY FATAL COMPLICATION OF STENOTROPHOMONAS MALTOPHILIA INFECTION

First Author: Nadia A Shaikh, MD Hussain Khawaja, MD

*Stenotrophomonas maltophilia* is a multi-drug resistant gram-negative bacillus that is a known opportunistic pathogen in the hospital setting, often colonizing equipment. Infections with *S. maltophilia* have been increasing in prevalence and most commonly manifest with bacteremia or pneumonia. Immunocompromised individuals, especially those with malignancy, HIV infection, cystic fibrosis, or admission into an ICU setting, are at highest risk of morbidity and mortality, but increasingly more cases are being reported of atypical infections in unexpected patient populations.

NM is a 59-year-old woman with a history of lung adenocarcinoma status post left upper lobectomy and on chemotherapy that initially presented with complaints of nausea, lightheadedness, and vomiting blood. Prior to that she had been in good health with no complaints. Physical exam was significant for mild hypoxia, hypotension, tachycardia, and crackles at the left lung base. Laboratory examination showed a hemoglobin drop from 9.1 G/DL to 6.6 G/DL within 24 hours of presentation. An emergent endoscopy was done which showed blood at the vocal cords, indicating pulmonary hemorrhage. CT Angiogram of the chest showed no active bleeding, but a left lower lobe consolidation concerning for pneumonia. Bronchoscopy revealed old blood at the location of her imaged pneumonia and no blood coming from her lung surgical stump.

During hospitalization, the patient received 2 units of packed red blood cells. She was initially started on vancomycin and piperacillin-tazobactam for pneumonia, which was presumed to be the cause of the hemorrhage. While on antibiotics, she developed fevers up to 102F. The bronchoalveolar lavage wash grew out 1+ *Stenotrophomonas maltophilia*, which was resistant to penicillins. The patient was then switched to trimethoprim-sulfamethoxazole. Her fevers resolved and she was discharged home with a twenty-one day course of antibiotic with close follow up from infectious disease and oncology clinics. ??

There are case reports of *Stenotrophomonas maltophilia* causing fatal hemorrhagic pneumonia in patients with hematologic malignancy or hematopoietic stem cell transplant. This particular case is unique due to the presentation of hemorrhagic pneumonia in a patient with a solid tumor. Chemotherapy and her exposure to ventilators during prior surgery left her susceptible. It is important to note that this patient had hemorrhage as the first sign of infection, rather than with any symptoms of pneumonia. Had a bronchoalveolar lavage not been done, the diagnosis would very likely have been missed. This case highlights the risks of the increasing prevalence of *S. maltophilia* infections and a widening patient population that can be affected. As such, it is important to keep a high index of suspicion in patients who meet the right clinical picture.

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POINT-OF-CARE HAND-HELD ULTRASOUND IN THE AMBULATORY SETTING TO EVALUATE SHORTNESS OF BREATH AND CHEST PAIN

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A 49 year-old female presented to clinic approximately one month following a motor vehicle collision in which she was the restrained driver. Initial evaluation in the Emergency Department (ED) was unremarkable. At presentation to clinic, the patient was experiencing persistent cough and three days of shortness of breath, fatigue, body aches, and left sided chest pain. Physical exam revealed mild tachycardia with HR around 100 bpm, decreased left sided apical breath sounds, and bibasilar crackles. Point-of-care lung ultrasound revealed presence of lung sliding artifact and multiple B-lines in the midclavicular line bilaterally, suggesting absence of pneumothorax and presence of interstitial edema. Lung ultrasound at the costophrenic angles showed no pleural effusion bilaterally. Focused cardiac exam showed dilated left ventricular cavity, severe left ventricle (LV) dysfunction and left atrial (LA) enlargement. EKG was performed and showed sinus rhythm with a left anterior fascicular block without any ST segment changes. Given the patient's chest pain and the notable reduction in systolic function of unknown duration, the patient was sent to the ED for further evaluation. Laboratory data revealed a BNP of 63, a POC troponin less than 0.02, and a D-Dimer within normal limits. Chest radiograph demonstrated cardiomegaly, pulmonary vascular congestion, and enlarged pulmonary arteries. Cardiology evaluated the patient and she underwent left heart catheterization the next morning showing severely reduced systolic function with an EF of 25% without evidence of obstructive coronary disease. She was then scheduled for further follow-up with cardiology for management of nonischemic cardiomyopathy. The use of a cardiopulmonary limited ultrasound examination (CLUE) protocol has been suggested and retrospectively examined in recent years for expedited assessment of patient's with suspected cardiopulmonary dysfunction. Traditionally, the limited 4-view exam includes a parasternal long axis view (PLAX) of the heart, 2 views of the anterior lung fields, and a subcostal view of the inferior vena cava (IVC). The PLAX view allows for examination of LV systolic function and the LA diameter. The anterior lung fields are examined for the presence of B-lines, a type of reverberation artifact caused by interstitial thickening/edema. The subcostal approach is used to examine the IVC's size and variability with respiration as a noninvasive marker for central venous pressure and intravascular volume status. Information gathered through this approach is useful to develop a global understanding of the process causing the patient's condition. In the case of our patient, point-of-care ultrasound allowed for the rapid exclusion of pneumothorax given the bilateral lung sliding and B-line artifact pattern. In addition, the findings on lung ultrasound, when combined with a focused cardiac ultrasound in the form of the CLUE protocol, accelerated triage and early identification of heart failure with reduced ejection fraction facilitating early evaluation by a Cardiologist.

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A NOVEL DRUG IN TREATMENT OF REFRACTORY CRYPTOCOCCAL MENINGOENCEPHALITIS

First Author: Scott Graupner, DO

The incidence of invasive fungal diseases has increased in the last decade and a change in spectrum of fungal pathogens has been observed. Fungal pathogens have also become increasingly resistant to what has been the standard therapy for certain types of fungal infections. Isavuconazole is an antifungal approved for use against aspergillosis and invasive mucormycosis. Recent studies have shown favorable results in use against aggressive cryptococcal infections non-responsive to standard therapies. This case explores an episode of an immunocompetent patient with cryptococcal meningitis infection refractory to standard therapy but with improved outcome using isavuconazole.

Patient was a 54-year-old male with past medical history of hypertension who presented to outside hospital with altered mental status, fever, and nausea. Patient was an employed police officer who was living a very functional life until presentation. Patient had lumbar puncture showing 300 WBC with lymphocytic predominance, high protein, and low glucose. CSF cultures came back positive for cryptococcus. Patient was started on amphotericin B and flucytosine. HIV screening was negative. Patient initially improved but symptoms worsened and he was transferred to GRU Hospital. Patient had no significant improvement while continued on antifungals. Patient had daily lumbar punctures with high pressure and voriconazole was added on day nine of hospitalization. Also around this time patient became less responsive and CT of head showed stroke in cerebellar region. Neurosurgery was consulted and decision was made to add shunt. On Day 34 of hospitalization, under infectious disease guidance isavuconazole was initiated. Patient slowly began to improve over next few weeks and began following commands and responded to family members and staff. Patient was eventually transferred to a long-term rehab facility after treatment of one month of isavuconazole and significant improvement in function capability.

This case explores how cryptococcus should be kept in the differential for immunocompetent patients, especially once bacterial meningitis has been ruled out as *Cryptococcus gattii* has been shown to effect this population. The standard of care for cryptococcal meningitis has been flucytosine and amphotericin B, which is first line treatment but shows that isavuconazole may be an effective therapy for patient that are refractory to the standard of care. Isavuconazole has shown promise in treating cryptococcus in patients who fail standard therapy and it certainly helped in the clinical situation provided in this case. Previous studies had shown good in vitro results for isavuconazole against cryptococcal isolate strains of *neoformans* and *gattii*. The patient in this case was thought to have possible *Cryptococcus gattii*, as he was immunocompetent with aggressive disease. *Cryptococcus gattii* has mostly been reported in tropical areas and the Pacific Northwest region and is associated with meningitis and meningoencephalitis in immunocompetent patients.

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AUSTRIAN SYNDROME: A FATAL TRIAD

First Author: Rachel Guthrie, DO Second Author: Sean Murney, DO Third Author: Kim Jordan, MD

Austrian syndrome is invasive pneumococcal disease (IPD) presenting as the triad of pneumonia, meningitis, and endocarditis. First described in 1862 by Heschl, *Streptococcus pneumoniae* was identified as the etiologic agent by Osler in 1881, but the syndrome was named for Robert Austrian in 1957, who defined the triad with associated 75% mortality. Since its description, approximately 70 cases of IPD have been published that fulfill the triad. We report a case of this rare syndrome resulting in death despite rapid diagnosis and aggressive treatment.

Case: A 50-year-old male smoker with type 1 diabetes mellitus presented with severe back pain, vomiting, diarrhea, fever, chills, headache, and chest pain. He admitted to cocaine use. Pneumococcal vaccination status was unknown. On admission he was hypotensive, tachycardic, with temperature of 99.5 F. Examination revealed nuchal rigidity, but no murmur, pulmonary findings, or rash were noted. Serum lactate was elevated at 4.9 mmol/L and leukocyte count was 5.23 with 94% neutrophils. Initial CXR was interpreted as no acute process, but 2 sets of blood cultures grew gram-positive cocci in chains within 8 hours. Despite vancomycin and ceftriaxone, the patient became increasingly lethargic, requiring intubation. Initial brain CT was negative. Lumbar puncture revealed gram-positive diplococci. A transthoracic echocardiogram demonstrated severely thickened mitral valve leaflets highly suspicious for endocarditis, without regurgitation. Trans-esophageal echocardiogram was not done because of severe thrombocytopenia. Repeat CXR on day 2 showed bilateral lower lobe infiltrates suspicious for pneumonia and Austrian syndrome was diagnosed. Dexamethasone was added to antibiotic therapy for his meningitis, but he developed tonic-clonic seizures. An acute infarct was noted on repeat brain CT, likely septic embolus. *Streptococcus pneumoniae* susceptible to penicillin was identified in both blood and CSF. Despite aggressive medical management, he died on hospital day 6.

Discussion: *Streptococcus pneumoniae* is a significant pathogen, causing 20 - 60% of all community-acquired pneumonia (CAP) in adults, and representing the most common cause of fatal CAP in the elderly. Pneumococcal endocarditis has decreased in incidence to 1-3% in the post-antibiotic era, but generally occurs as part of the Austrian syndrome triad when seen. Risk factors for Austrian syndrome include alcoholism, splenectomy, IV drug use, and immunosuppression. Our patient had increased risk from diabetes and illicit drug use. Though our patient had mitral valve endocarditis, native aortic valve endocarditis is most common in Austrian syndrome (80%). Typical examination findings of endocarditis are not usually seen. Despite a decrease in mortality with surgical intervention and aggressive medical support, prognosis remains poor, with reported mortality rates of 30-60%. Pneumococcal vaccination remains important given the significant mortality associated with invasive pneumococcal disease.

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THE COMPLEXITY OF HEREDITARY PARAGANGLIOMA-PHEOCHROMOCYTOMA SYNDROME FROM SDHD PATHOGENIC MUTATION

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Patients with Hereditary Paraganglioma Pheochromocytoma syndrome may develop both paragangliomas, neuroendocrine tumors symmetrically distributed along the paravertebral axis from skull to pelvis, and pheochromocytomas, a paraganglioma confined to the adrenal medulla.

A 29 year old Caucasian male presented for evaluation for left sided neck mass. Medical records over the prior 2 years highlighted anxiety, palpitations, elevated blood pressure, hyperhidrosis disorder, and headaches. Fine needle aspiration and magnetic resonance imaging suggested a carotid body paraganglioma that completely encircled the left carotid artery. Urine studies for metanephrine and normetanephrines as well as plasma catecholamine and norepinephrine levels were elevated above normal range. Metaiodobenzylguanidine (MIBG) scan showed the left carotid body lesion and multiple areas of enhanced uptake in the left adrenal, upper to mid abdominal aortocaval chains. Surgical resection resulted in removal of 7 lesions consistent with paraganglioma and a partial left adrenalectomy with pheochromocytoma. Genetic analysis revealed a SDHD mutation, a pathogenic mutation consistent with the diagnosis of hereditary paraganglioma-pheochromocytoma syndrome type 1. Left carotid body tumor was then removed, also a paraganglioma. 10 months from his initial presentation, symptoms returned and a repeat MIBG scan showed a new focus of uptake high in the left mediastinum. Video-assisted thoracoscopic excision of the lesion confirmed another paraganglioma.

This case demonstrates the complexity in diagnosis, management, and surveillance of Hereditary Paraganglioma-Pheochromocytoma Syndrome and the necessity for multidisciplinary coordination in care.

## BILE CAST NEPHROPATHY AND CHOLESTATIC LIVER INJURY IN AN ATHLETE ABUSING ANABOLIC STEROIDS

First Author: Sehrish Kamal, MD Bilal Ali, MD Muhammad Ali Khan, MD Faisal Kamal, MD Hina Akbar, MD Satheesh Nair, MD

**Introduction:** Anabolic steroid use has been associated with cholestatic liver injury. Acute kidney injury in the setting of liver disease can be caused by hypo-perfusion, acute tubular necrosis and/or hepato-renal syndrome. Experimental data and one case report has highlighted the toxic effect of elevated bilirubin on renal tubules, leading to bile cast nephropathy. We report a unique case of subacute liver failure secondary to anabolic steroids use in an athlete, leading to acute renal failure due to bile cast nephropathy. Remarkably, treatment with plasmapheresis showed improvement.

**Case Presentation:** A 52 year old Hispanic gentleman with progressively worsening hepatic and renal function was referred to our transplant center. He initially presented 4 weeks ago to an outside hospital for lethargy, anorexia, weight loss and worsening jaundice. History was significant for the use of OTC supplement containing anabolic steroids in preparation for a marathon for the last 4 months. He had no significant medical history, did not smoke or use alcohol, reported no family history of liver or kidney disease. An extended liver screen including serological testing for viral hepatitis A, B, C and E, as well as CMV, EBV, HIV, and full autoantibody profile was negative. Abdominal ultrasound, HIDA and MRCP failed to identify an alternative source of liver injury. Clinical diagnosis of 'Drug induced liver Injury' (DILI) due to anabolic steroids was made and supportive management with urodeoxycholic acid, cholestyramine and hydroxyzine was initiated. His condition continued to deteriorate with rising bilirubin levels and oliguric renal failure requiring hemodialysis (HD). Liver biopsy revealed prominent cholestasis with mild mixed inflammatory infiltrate, no steatosis or necrosis consistent with liver injury from steroid use. Interestingly, kidney biopsy showed prominent tubular accumulation and bile casts, notable for bile cast nephropathy.

On admission to our facility, physical exam was unremarkable except for profound jaundice, scratch marks on skin and hepatomegaly. Laboratory studies were notable for WBC: 10,000, HB: 11.9, Cr: 9.5, albumin: 1.7, total bilirubin: 34, ALP: 579, AST: 95, ALT:105, INR: 1.2. He was evaluated for dual liver and kidney transplant. In multidisciplinary transplant meetings, plasmapheresis was debated over and started for bile cast nephropathy. For the next 2 weeks patient received 6 sessions of plasmapheresis along with hemodialysis on alternate days. His bilirubin started trending down and patient started making urine, no longer requiring HD. Patient demonstrated significant clinical improvement and his creatinine was 2.3 and total bilirubin was 3.1 on discharge.

**Conclusion:** To the best of our knowledge, this is the first reported case of acute renal failure due bile cast nephropathy in a patient of DILI, treated with plasmapheresis. Our case manifests the need for further studies exploring it as a treatment option in such patients.

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## A TALE OF TWO TUMORS

Benjamin Maddox

Paraneoplastic neurological syndromes (PNS) are a diverse group of neurological deficits triggered by humoral responses to an underlying remote tumor. Typically, these neurological symptoms antedate the diagnosis of the malignancy, and a broad initial differential diagnosis may lead to early recognition and more favorable outcomes.

The first case is a 32 year-old woman with no known medical issues who presented to our hospital with hallucinations and confusion. She quickly deteriorated to a catatonic state, and required intubation. Initial physical exam revealed roving eye movements, no nuchal rigidity, normal cardiopulmonary and abdominal exam, and non-purposeful movements when stimulated. Lumbar puncture showed a lymphocytic pleocytosis concerning for infectious encephalitis, and she was started on broad antimicrobial and antiviral therapy. Cerebral spinal fluid (CSF) Gram stain, cultures, and viral titers were negative. Extensive head imaging, electroencephalogram, and blood cultures were unrevealing. Subsequent CSF analyses revealed anti-N-methyl-D-aspartate receptor (anti-NMDAr) antibodies. Pelvic ultrasound revealed a 1 centimeter mass in the right ovary, characterized after excision as a teratoma. She received high-dose intravenous steroids, plasmapheresis, and intravenous immunoglobulin with significant improvement in her mental and physical deficits.

The second case is a 26 year-old woman with a past medical history significant for a remote history of right oophorectomy for an ovarian cyst and 6 months of abdominal swelling, who presented to our hospital with involuntary movements of the eyes, tongue, and hands. Initial physical exam revealed normal cardiopulmonary exam, a markedly distended abdomen with a large firm mass, and minimal response to noxious stimuli. Shortly after presentation, her mental status deteriorated and she required intubation. A CT of the abdomen revealed a massive complex mass. Intraoperatively, the 40 cm mass was decompressed of 18.5 L of fluid, and surgical pathology revealed an ovarian mucinous tumor. Initial CSF analyses did not identify implicated antibodies. She received plasmapheresis with complete resolution of her altered mental status and significant improvement in physical deficits.

Paraneoplastic neurological syndromes represent a wide range of encephalopathies, myelopathies, and neuropathies. They manifest in approximately 1% of all malignancies, with nearly 10% caused by ovarian tumors. The pathogenesis is related to immune-mediated responses to neuronal proteins expressed by tumor tissues, called onconeural antigens. However, the presence of traditional intracytoplasmic onconeural antigens and antibodies have not been shown to directly correlate to paraneoplastic reaction. New research has revealed new antibodies to onconeural surface proteins, such as those implicated in anti-NMDAr encephalitis, which are a subset of PNS that respond especially well to tumor excision and immunotherapy. These cases illustrate the favorable outcomes achieved by early recognition and prompt treatment of PNS, and stress the importance of a broad differential diagnosis for the presentation of altered mental status.

RARE BREAST METASTASIS IN A PATIENT WITH ATYPICAL PULMONARY CARCINOID AND A FOLLICULAR HÜRTHLE CELL THYROID NODULE

First Author: Jaime Morris, DO J. Clay Callison, MD David Aljadir, MD

**INTRODUCTION:** Pulmonary neuroendocrine tumors encompass a broad spectrum of neoplasms that originate from embryonic neural crest cells, including typical and atypical carcinoid, large and small cell carcinoma. Atypical carcinoid tumors are defined as having carcinoid morphology with 2 to 10 mitoses per 10 high power field (HPF) and/or necrosis. The incidence of metastasis from pulmonary carcinoid is much lower than large or small cell carcinomas, estimated around 10%, and most commonly to lymph nodes, liver, bone, and, rarely to mammary glands. These tumors are typically slow growing with low glucose uptake; making <sup>111</sup>In-octreotide (Octreoscan) scans more sensitive for well-differentiated tumors, whereas <sup>18</sup>F-fluorodeoxyglucose PET (FDG-PET) scans are more sensitive for poorly-differentiated tumors.

**CASE PRESENTATION:** A 43 year old female with no preceding medical history presented with three weeks of persistent cough, fever, and hemoptysis having failed outpatient therapy for pneumonia. Initial chest x-ray was concerning for left upper lobe mass causing a post-obstructive pneumonia. Biopsy obtained through endobronchial ultrasound-guided transbronchial needle aspirates revealed a well-differentiated neuroendocrine carcinoma, staining strongly for synaptophysin. Octreoscan revealed uptake in the left hilar mass along with uptake a thyroid nodule. Fine needle cytology of thyroid nodule was consistent follicular Hürthle cell. Patient underwent thyroidectomy confirming benign follicular adenomatoid nodule with Hürthle cell change and also underwent a left upper lobectomy which showed neuroendocrine tumor with mitotic rate of 3 mitoses/10HPF, as well as perihilar lymph nodes that were positive for tumor cells. Post-surgical histologic grade was G2, moderately differentiated.

Restaging Octreoscan imaging prior to chemotherapy showed a larger left breast inferior medial lesion, previously subthreshold in original scan. The patient underwent mammography followed by needle biopsy of lesion, which originally thought to be an invasive ductal carcinoma, but upon further review, stained positive for neuroendocrine markers consistent with original tumor. Based on the National Comprehensive Cancer Network (NCCN) guidelines, the patient is currently being treated with cisplatin and etoposide. Follow-up imaging will dictate whether patient proceeds with surgical resection of the mammary lesion.

**DISCUSSION:** The breast is the most common site of primary malignancy in adult women, yet breast involvement from an extra-mammary tumor is rare, accounting for only 1-2% on breast malignancies. On review of the literature, there are less than five case reports of atypical lung carcinoid metastasizing to the breast. Of those cases, the breast metastases were discovered several years after the primary lesion. Given the differences in both treatment and outcome, it is imperative to properly stage a carcinoid tumor and to biopsy a suspected metastatic breast lesion. Novel radioactive tracers such as <sup>68</sup>Ga-DOTATATE and <sup>68</sup>Ga-DOTATOC for use in PET imaging may further improve our ability to detect these rare metastatic lesions.

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A DIFFICULT DIAGNOSIS TO SWALLOW

First Author: John Allison, MD Additional Author: Zachary Marshall Additional Author: Amitha Rao

**Case report:** A 28 year old female with no significant medical history was admitted for 4 weeks of sore throat and difficulty swallowing. One week prior to onset of symptoms she had unprotected oral sex with a male partner. She subsequently developed odynophagia and dysphagia prompting her to see her primary care physician. She was diagnosed with a viral upper respiratory infection treated with supportive care. Her symptoms remained unchanged, so she sought care at multiple clinics and emergency departments. Upon admission to our hospital, she was afebrile with cervical lymphadenopathy, erythematous pharyngeal mucosa, and bilateral tonsillar exudates without ulcerations. Her throat was swabbed for cultures, and she was treated with intravenous antibiotics to cover typical bacterial organisms as well as gonococcal pharyngitis. Initial testing (including HIV antibodies, HIV viral load, gonorrhea/chlamydia DNA probe, and bacterial cultures, and rapid streptococcal antigen detection test) was negative. As she had some improvement with antibiotics, she was discharged home; however, she returned fifteen days later with worsening odynophagia. Viral polymerase chain reaction was positive for Herpes simplex virus-2 (HSV-2). Her symptoms improved rapidly with intravenous acyclovir which was transitioned to oral valacyclovir.

**Discussion:** In immunocompetent adult patients, viral pathogens are implicated in 75% of cases of pharyngitis with a known pathogen. Bacteria, mainly *Streptococcus* spp., make up the remainder of cases. The Centor criteria is a widely accepted and well-validated tool to guide clinical decision-making for patients presenting with pharyngitis. However, the tool is often used incorrectly as it was in this case.

**Conclusions:** When patients fail to respond to conventional therapies for pharyngitis, alternative etiologies should be entertained. Furthermore, in the setting of an unprotected oral sexual encounter, sexually transmitted organisms such as *Neisseria gonorrhoeae*, HIV, and herpes simplex viruses must be considered. Herpes simplex virus-1 is relatively common in the adult population, particularly college students, but HSV-2 pharyngitis is exceedingly rare.

## A STING TO THE HEART

First Author: Erica Fidone, MD Second Author: Justin Price, MD Third Author: Evan Hardegee, MD Fourth Author: Mark Lawrence, DO

Anaphylaxis rarely manifests as a vasospastic acute coronary syndrome with or without the presence of underlying coronary artery disease. Kounis syndrome, also known as “allergic angina”, is a disorder characterized by acute coronary syndrome resulting from an allergic reaction. In rare cases, allergic angina can progress to an acute myocardial infarction, termed “allergic myocardial infarction”. It results from massive mast cell degranulation in the setting of an allergic insult with subsequent release of numerous inflammatory mediators. It is these chemical mediators that have been implicated in inducing coronary vasospasm and atheromatous plaque rupture leading to acute coronary syndrome. It is important to appropriately recognize and treat Kounis syndrome in patients with exposure to a suspected allergen.

A 61 year-old male with a known medical history of diabetes mellitus, hyperlipidemia and emphysema presented to the emergency department after being swarmed by bees. He complained of diffuse pain secondary to bee stings, but denied cardiopulmonary symptoms. Physical exam was significant for tachycardia, extensive envenomation with greater than 500 bee stings and diffuse urticaria. Initial electrocardiogram (ECG) revealed no evidence of active ischemia and initial troponin level was negative. For concern of impending anaphylaxis, he was treated with epinephrine, corticosteroids, and antihistamines. Two hours later, he developed crushing, substernal chest pain, dyspnea and diaphoresis. Repeat ECG revealed ST elevation in leads II, III, and aVF with reciprocal changes in the anteroseptal leads. Repeat troponin was 115. He received aspirin and an intravenous heparin bolus and was taken for emergent coronary angiography. Angiography demonstrated an occlusion of the right coronary artery by thrombus as well as extensive thrombus throughout the left main coronary artery and left anterior descending artery. He underwent aspiration thrombectomy of these sites along with stent placement. Diagnosis of ST-segment elevation myocardial infarction was made secondary to anaphylactic reaction from bee stings. Post-procedurally he was treated with dual-antiplatelet therapy in addition to treatment for an allergic reaction with steroids and antihistamines.

Allergic reactions to chemicals, medications, food products or even insect bites are encountered in every day clinical practice with a variety of manifestations, from a minor rash to life threatening anaphylactic reactions. Although there is no specific test to diagnose Kounis syndrome, one should have a high index of suspicion when there is a coincidental occurrence of chest pain in the setting of a recent allergic insult. Knowledge of this syndrome will enable optimal management of patients with both acute allergic and/or myocardial events. This case represents the importance of acute primary care in a case complicated by anaphylaxis and acute coronary syndrome and the rare occurrence of their co-presentation.

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UNMASKING THE BLACK SCHWAN

First Author: Brendan Gilmore, MD Second Author: Toug Tanavin, MD Third Author: Rajeev Balchandani, MD

**Introduction:** Vestibular schwannoma is a rare cause of unilateral sensorineural hearing loss. This case describes how a meticulous history and physical exam can result in the foresight to pursue additional imaging to accurately diagnosis a vestibular schwannoma and facilitate swift intervention.

**Case Presentation:** A 72 year-old African American male with esophageal squamous cell carcinoma in remission presented with six weeks of urinary incontinence, progressively worsening balance and unusual behavior. Non-contrast computerized tomography (CT) of the head showed lateral and third ventricular enlargement out of portion to cortical atrophy, which was most consistent with normal pressure hydrocephalus (NPH). However, on assessment of vestibulocochlear cranial nerve function, the patient was found to have unilateral sensorineural hearing loss. Weber test was positive. Additional history revealed that the patient had consistent asymmetric hearing loss for the last 6 months. Audiogram confirmed unilateral sensorineural hearing loss. At this point, the decision was made to obtain a magnetic resonance imaging (MRI) brain with gadolinium. It showed a 3 cm by 3.5 cm by 3.3 cm heterogeneous left cerebral pontine angle mass. The mass created a partial obstruction of the fourth ventricle, which resulted in obstructive hydrocephalus. Neurosurgery was consulted and patient underwent a left retrosigmoid craniotomy. Pathology results confirmed the diagnosis of a vestibular schwannoma. Patient did well post-operatively. Mental status and balance improved. Patient was transferred to inpatient rehabilitation floor for intensive physical and occupation therapy.

**Discussion:** In the current health care environment, high value care is becoming increasingly emphasized. It is critical to avoid unnecessary tests and imaging, while still providing the best possible care to patients. Specifically, cost-consciousness often precedes consideration for an MR. This case highlights the utility of MRI when indicated by a truly thorough history and physical. Without this clinical acumen, the patient may have been confined by a diagnosis of NPH, which may have led to misguided management and persisting morbidity for the patient.

**Conclusions:** This patient's diagnosis of vestibular schwannoma with associated obstructing hydrocephalus was set in motion by a well-executed history and physical. Misuse and overuse of diagnostic studies remains a pressing issue in high-value care. However, this case illustrates the ideal allocation of clinician resources, which can help augment management and in turn, patient outcomes.

WHEN IT'S NOT AN ALLERGIC REACTION: AN UNUSUAL CASE OF ECTHYMA GANGRENOSUM ASSOCIATED WITH PROTEUS BACTEREMIA

First Author: Andrew Hawrylak, MD Second Author: Susan Seago, MD Third Author: Edana Stroberg, DO Last Author: Megan Greene Newman, MD

**INTRODUCTION:** Ecthyma gangrenosum is an exceedingly rare dermatologic complication of bacteremia or fungemia. This case is particularly unique in that there was no co-infection with *Pseudomonas* species and our patient was not immunosuppressed. Additionally, our patient's ulcers were initially misdiagnosed as an allergic reaction to intramuscular gentamicin injections, and an important sign of occult bacteremia was missed for several weeks.

**CASE DESCRIPTION:** A 51-year-old woman with multiple medical co-morbidities including morbid obesity with body mass index of 84, hepatitis C cirrhosis, urinary retention requiring chronic indwelling foley catheter and bilateral non-obstructing renal calculi presented from her nursing home with altered mental status, tachycardia, hypotension, and urinalysis findings consistent with urinary tract infection. She had a prolonged history of recurrent multi drug resistant urinary tract infections with previous *Proteus mirabilis* bacteremia, likely secondary to her infected renal calculi; however, she was not an operative candidate due to her multiple co-morbidities. Several weeks prior to admission, she was treated empirically with ciprofloxacin and intramuscular gentamycin for presumed urinary tract infection by nursing home staff; however, her clinical status continued to worsen and she developed necrotic ulcers on bilateral upper extremities near her gentamycin injection sites.

On admission, she was started on meropenem and linezolid and initial urine and blood cultures grew out *Proteus mirabilis*. Dermatology was consulted for further evaluation of her necrotic skin ulcers and a punch biopsy illustrated acute inflammation and necrosis with florid bacterial organisms. Body tissue cultures from the ulcer sites grew *Proteus mirabilis* and *Enterococcus faecalis* and pathology was consistent with a diagnosis of ecthyma gangrenosum. A trans esophageal echocardiogram did not illustrate focal vegetation and she was treated with two weeks of intravenous meropenem. Urology was consulted for possible percutaneous nephrolithotomy, however the patient was again deemed a poor operative candidate and was ultimately transitioned to palliative care.

**CONCLUSIONS:** Ecthyma gangrenosum is a dermatologic finding of classically associated with *Pseudomonas aeruginosa* bacteremia in immunocompromised patients. Patients develop this rare finding when organisms colonize the media and adventitia of arteries or veins leading to local necrosis and ulceration of the epidermis and dermis. Although ecthyma gangrenosum has been associated with other bacteria and fungi, the majority of these case reports involve co-infection with *Pseudomonas*. Additionally, almost all documented case reports involve severely immunocompromised patients with HIV / AIDS, undergoing chemotherapy, or in the setting of acute leukemia. This report highlights an unusual case presentation and encourages physicians to maintain a broad differential diagnosis in the setting of unusual or atypical clinical presentations.

THE UNEXPECTED HEARTBREAK: DISSECTING AN UNUSUAL CASE OF ACUTE CORONARY SYNDROME

First Author: Rasheen Imtiaz, MD Second Author: Sarathi Kalra, MD Third Author: Nadine Faza, MD Fourth Author: Mark Pollet, MD Fifth Author: Nasser Lakkis, MD

Acute coronary syndrome (ACS) is a routinely encountered diagnosis for internists. However, ACS in young patients without usual risk factors can be puzzling.

A 27 year-old gravida 3, para 3 African American woman with a history of cesarean section 2 weeks ago presented to the emergency department with chest pain. She was in her usual state of health when she developed severe, burning, sub-sternal chest pain at rest associated with dyspnea, diaphoresis and nausea, lasting over 30 minutes. She denied pleurisy, hemoptysis, leg swelling or pain, radiation of pain to the back and symptoms of reflux disease. On evaluation, she was afebrile with a blood pressure of 109/30 mmHg, heart rate of 105 beats/min, respiratory rate of 19, normal oxygenation and BMI of 29. She was in moderate distress due to chest pain but the remainder of her examination including cardiac and pulmonary, was normal. An electrocardiogram showed ST segment elevation in leads V3 to V6 consistent with anterolateral ST-elevation myocardial infarction (STEMI). An initial troponin was 1.7 ng/mL. She has immediately taken for cardiac catheterization which revealed significant coronary artery dissection involving the proximal and middle left anterior descending artery (LAD) as well as the ostial left circumflex, first and third obtuse marginal arteries. She received multiple drug-eluting stents in the affected coronary arteries. Echocardiogram revealed an ejection fraction of 35% with wall motion depression in the anterolateral walls. She had a negative urine drug screen, antinuclear antibody and anti-neutrophil cytoplasmic antibodies. During her hospital course, her troponin peaked at 148 ng/mL and creatine kinase at 4266 IU/mL. Her chest pain improved and cardiac enzymes decreased at which time she was discharged with close follow-up.

Spontaneous coronary artery dissection (SCAD) is defined as a non-traumatic and non-iatrogenic separation of coronary arterial walls resulting in an intraluminal hematoma that compresses the true lumen, compromising blood supply. Most commonly, a single artery, usually the LAD is affected. SCAD can result in morbidities including ACS, ventricular arrhythmias and sudden death. Risk factors include peripartum state (most common precipitating factor in women), multiple pregnancies, hormonal therapy-particularly progesterone, extreme physical activity (most common factor in men) and diseases that predispose arterial walls to injury such as fibromuscular dysplasia, connective tissue disorders and systemic inflammatory disorders like lupus or vasculitides.

Those with acute presentations including ACS, ongoing chest pain or hemodynamic instability are recommended to undergo percutaneous coronary intervention. Emergency coronary artery bypass surgery must be considered in patients with dissections of the left main coronary artery. In the long-term, there is a significant event recurrence rate of under 20% in once affected individuals. Peripartum patients must be advised to avoid future pregnancies.

SCAD is a rare, under-recognized but important cause of ACS. Awareness of this condition is important because the management may differ.

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AN OVERLOOKED COMPLICATION OF UNCONTROLLED DIABETES MELLITUS

First Author: Sonam Jaggi, MD Fiorella Llanos-Chea, MD Nawid Sarwari, MD Adriana Rauseo, MD Philip R. Orlander, MD

**Introduction** Diabetes Mellitus is an increasing epidemic worldwide, currently affecting 8.3% of Americans of all ages. Uncontrolled diabetes is associated with the development of cardiovascular, renal, neuropathic, and retinal disease. Nevertheless, less common complications, such as diabetes myonecrosis, can increase morbidity and worsen prognosis in these patients.

**Case description** We report a case of a 38-year-old black female with history of uncontrolled hypertension and type 2 diabetes, admitted for 2-month history of worsening right lower extremity (RLE) edema, associated with right thigh pain and intermittent paresthesia. She denied any recent trauma, infections, joint pain or rash. No personal or family history of autoimmune diseases or malignancy. Physical exam was remarkable for 3+ RLE edema up to her hip, with diffuse tenderness over her right thigh. Laboratory findings were significant for leukocytosis 34.4 K, thrombocytosis 695 K, nephrotic-range proteinuria 7.3g/day, hypoalbuminemia 1.0 g/dL, and elevated globulin gap 6.2 g/dL. Her inflammatory markers were significantly elevated (ESR >100, CRP 455). Her hemoglobin A1c was 9.4%. Extensive autoimmune work-up was unremarkable. Doppler ultrasound was negative for deep venous thrombosis; however, it revealed a fluid collection. This led to surgical exploration, but no abscess was found and cultures remained negative.

CT abdomen/pelvis was negative for underlying infection or malignancy causing lymphatic obstruction. Given her inflammatory findings, a MRI of the RLE was performed, which showed markedly edematous right thigh with enlarged heterogeneous edema spanning the entire right quadriceps muscles; suggestive of infarcted necrotic muscle versus polymyositis. A subsequent muscle biopsy evidenced necrotic skeletal muscle, fascia, and fibroadipose tissue, with regional areas of necrotic neutrophil infiltration consistent with myonecrosis. Hence, patient was placed on a tighter glycemic control and supportive treatment, including rest and analgesics.

**Discussion** Diabetes Muscle Infarction (DMI) or Diabetes Myonecrosis is a rare microangiopathic complication associated with poorly controlled diabetes. It usually presents as acute pain and swelling of the affected muscle, commonly the quadriceps (62%). One of the proposed mechanisms is thought to be severe microangiopathy causing occlusion of blood vessels and in-turn ischemia. Other theories include hypoxia-reperfusion injuries, coagulation-fibrinolysis imbalance, and antiphospholipid antibodies. While ESR and CRP are elevated in 80-90% of cases, most laboratory findings are unspecific and non-diagnostic. MRI is the most valuable diagnostic test, which also helps excluding other conditions. Common findings include hypointense areas in T1 and enlarged muscle with diffuse high signals in T2. Muscle biopsy is not routinely recommended due to its post-procedural complications, such as delayed wound healing. Management is mainly supportive (rest, analgesics, optimal glycemic control), although there is a high recurrence rate. Moreover, long-term prognosis is poor as patients usually have other microvascular complications when DMI is diagnosed.

**Conclusion** DMI is a rare complication associated with poorly controlled diabetes. One must have a high-index of suspicion in order to reach the diagnosis, which is imperative because it guides us towards worsening disease and the need for strict glucose control.

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A CASE OF PULMONARY ARTERY SARCOMA (PAS) PRESENTING AS PULMONARY EMBOLISM

First Author: Michelle Janania Martinez, MD Elizabeth Bowhay-Carnes, MD Anand Karnad, MD

**Case Description:** A 59yo woman with past medical history of Diabetes Mellitus, Hypertension and hyperlipidemia presented to her primary care physician with a 2 week history of acute onset left sided chest pain and difficulty with deep inspiration. Chest X-ray was suggestive of atypical infection; she completed a course of oral antibiotics, but symptoms and X-ray findings persisted. CT Thorax with contrast reported an acute thrombus in the left lower pulmonary artery. She was hospitalized immediately, started on heparin and later completed 6 months of warfarin. Shortly after, a paroxysmal cough developed and repeat CT revealed a filling defect occupying the entire left main pulmonary artery extending into the main pulmonary artery, consistent with a tumor thrombus. FDG positron emission tomography demonstrated avidity of the tumor thrombus, felt to represent primary angiosarcoma. Core needle biopsy revealed poorly differentiated high-grade pleomorphic sarcoma, consistent with intimal sarcoma versus undifferentiated sarcoma. The tumor was determined to be unresectable and 13 months after initial onset of symptoms, she started palliative chemotherapy (docetaxel/gemcitabine and later pazopanib).

**Discussion:** Sarcomas are the most common primary tumors of the pulmonary arteries, yet they are rare and difficult to diagnose. PAS is usually asymptomatic until there is significant vessel occlusion and presents with symptoms like dyspnea, chest pain and cough. On conventional chest X-ray PAS usually manifests as hilar, mediastinal, or pulmonary artery enlargement (53%), pulmonary nodule (40%), cardiomegaly(33%), and diminished pulmonary vascularity(18%). CT pulmonary angiography is good at detecting intravascular processes but it is difficult to differentiate tumor from thromboemboli, further studies are needed.

The mainstay of therapy is radical surgical resection and neoadjuvant chemotherapy if possible. However because the tumor usually involves vital cardiac structures surgical resection can be challenging. Also initial misdiagnosis and acute cardiorespiratory symptoms requiring urgent surgery sometimes preclude neoadjuvant chemotherapy. Patients undergoing an attempt at curative resection have a median survival of 36.5 months compared with 11 months for those undergoing incomplete resection. Median survival is also better for patients undergoing surgery/chemotherapy/radiation (survival of 5-7 years has been reported), compared with those on single therapy.

In conclusion: the diagnosis of PAS represents a challenge for physicians and early diagnosis is of the utmost importance due to the poor prognosis and high mortality associated with it. The symptoms and imaging are often misleading. In patients suspected of having a pulmonary embolism who do not respond to initial anticoagulation, physicians should consider further investigation with gadolinium-enhanced magnetic resonance imaging or FDG-PET.

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ELEVATED LIPASE LEVELS IN SMALL BOWEL OBSTRUCTION

First Author: Fathima Z Kamil Faiz, MD

This is a 69 year old man with known incisional and hiatal hernias who presented with about 10 days of nausea, vomiting, anorexia as well as intermittent mild abdominal pain. In the ER, his lipase was 1012 and AST and ALT were twice the upper limits of normal. Given this picture, the patient was admitted with a working diagnosis of acute pancreatitis. On arrival to the floor, further history revealed that the pain was baseline from his ventral hernia and that the characteristics of the pain had not changed over the last 2 weeks. However, the patient reported that he had not had a bowel movement or flatus in about 10 days. Physical exam of his abdomen demonstrated decreased bowel sounds and large non-reducible ventral hernia with no focal tenderness. Given the combination of emesis and obstipation with a hernia and history of abdominal surgeries, there was concern for small bowel obstruction. A CT scan of his abdomen and pelvis showed high grade small bowel obstruction and a contained perforated sigmoid abscess. Of note, his pancreas appeared normal. The patient was taken to the OR emergently and confirmed to have small bowel obstruction with an incarcerated ventral hernia that contained about 4 feet of bowel. Additionally, patient was found to have a perforated sigmoid mass with a large leading to a pelvic abscess. Given that the mass appeared malignant, the patient underwent rectosigmoid resection with a colostomy and Hartman pouch procedure. Patient did well post-operatively and was eventually discharged with oncology follow up. Pathology showed invasive adenocarcinoma of the colon.

This case demonstrated the need for a complete history in all patients. In the emergency room and inpatient setting, clinicians are often make the diagnosis of pancreatitis based on clinical symptoms and elevated pancreatic enzymes. This patient had no risk factors to develop pancreatitis. It is unclear why small bowel obstruction would lead to an elevated lipase in the 1000s and there have only been few case reports of similar conditions. Lipase is usually released by pancreatic acinar cells and since his pancreas was not inflamed, it is uncertain where the lipase was coming from. It could be related to mucosa from his obstruction, bowel perforation or the colon cancer. Another aspect of this case was the severity of operative findings. This gentleman had high grade small bowel obstruction as well as a perforated sigmoid mass but he denied any pain on history and had no tenderness on physical exam. It may be possible that his multiple abdominal surgeries and recurrent hernias may have altered his perception of visceral pain. This may be useful to remember when evaluating patients with complex gastrointestinal anatomy.

## IRIS-ASSOCIATED PROGRESSIVE KAPOSI'S SARCOMA

First Author: Maryam Kaous, MD Cesar Arias, MD

**Introduction** Immune reconstitution inflammatory syndrome (IRIS) is thought to be a result of a newly strengthened immune system identifying previously dormant pathogens in HIV patients initiated on HAART. This subsequently leads to clinical signs of active infection and deterioration despite a good virologic response against HIV. Though most commonly seen with *Mycobacterium Tuberculosis*, IRIS has been associated with other infectious, autoimmune, and neoplastic causes as well, including Human Herpes virus-8 (HHV8+) Kaposi's sarcoma (KS). We present the case of a patient with HIV on HAART found to have disseminated KS in the setting of IRIS.

**Case Presentation** A 36-year-old man with HIV (diagnosed 5 months ago, on HAART for 3 months, last CD4 count at 220), presented with a 2-week history of abdominal pain, diarrhea, nausea, and dyspnea on exertion. Physical exam revealed a thin man in no acute distress but febrile up to 103F, tachycardic, and tachypneic, with normal pulse oximetry on room air. Patient had oral thrush, a hyperpigmented lesion on his distal soft palate, significant lymphadenopathy along cervical chains bilaterally, right lung base with decreased breath sounds, and abdomen slightly tender to palpation but normal bowel sounds. Chest x-ray showed a right-sided pleural effusion. A CT neck showed mass-like enlargement of lingual tonsils and bilateral cervical lymphadenopathy; while a CT abdomen evidenced extensive abdominal and pelvic lymphadenopathy. Laboratory findings were remarkable for a WBC of 3.2 (6% lymphocytes), CD4 at 23, and undetectable viral load. He also had negative blood and urine cultures, cryptococcal antigen, *Histoplasma* antigen, RPR, *Giardia*, *Cytomegalovirus*, AFB stain/culture, and QuantiFERON-TB Gold.

Lymph node biopsy revealed a lymph node almost entirely replaced by spindle cells, and majority of cells positive for HHV-8, consistent with KS. Our patient was restarted on HAART and treatment with doxorubicin-based chemotherapy was added, as he presented with disseminated KS. On follow up, he had clinical improvement and an up-trending CD4 count.

**Discussion** Though the pathophysiology of IRIS-KS is not well understood, documented cases of IRIS-KS usually occurred within the first 12 weeks of HAART therapy and demonstrated a relationship between rapid clinical progression of KS and HAART initiation, while HIV viral load plummeted. Literature suggests that patients with IRIS display a rapid restoration of the immune system against latent pathogens. The inflammatory responses against these antigens lead to an immunopathologic effect, causing clinical deterioration. In IRIS-KS, hypotheses suggest inflammatory cytokine and chemokine production may stimulate angioproliferation and tumorigenesis. Therapy for IRIS-KS, as other IRIS associated diseases, includes continuing HAART. If KS is disseminated, chemotherapy may be indicated, which is associated with response rates around 60-90%.

As our case illustrates, IRIS-KS is an important clinical phenomenon, which requires further study for better understanding and early therapy initiation, as progression of IRIS-KS may be rapid and fatal.

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A NOT SO MINIMAL CHANGE DISEASE

First Author: Daniel Kil, MD John Allison, MD Michelle Udayamurthy, MD Andrew Caruso, MD Lee Haruno David Martin

**Case Description:** A 63 year old African-American male with no significant medical history presented with a four day history of new-onset lower extremity and abdominal swelling. He denied shortness of breath, chest pain, orthopnea, and constitutional symptoms, but did report occasional non-drenching night sweats. He reported being otherwise healthy with no known history of baseline heart, liver or kidney disease, and sought evaluation at the suggestion of his spouse who noticed the swelling. Vital signs were within normal limits. Physical exam was significant for abdominal distension with mild ascites and shifting dullness, 3+ pitting edema in the lower extremities to the level of the knees, decreased breath sounds in the left anterior lung fields, and mild jugular venous distention. There was no lymphadenopathy or testicular masses and neurologic examination was within normal limits. Laboratory values were indicative of renal failure with nephrotic range proteinuria. A 13x9x10 cm anterior mediastinal mass was found incidentally on chest x-ray. Subsequent CT and MRI detailed extension of the mass from the left brachiocephalic vein to the left ventricle, abutting the aorta. Fine needle aspiration confirmed a large thymoma. Renal biopsy was consistent with minimal change disease likely secondary to the malignancy. Despite aggressive diuresis and high dose steroids, renal function continued to decline leading to initiation of hemodialysis. The hospital course was further complicated by recurrent left sided pleural effusions, twice requiring thoracentesis and chest tube placement. He ultimately underwent an uncomplicated total thymectomy, however, his renal function failed to improve despite tumor resection and he continued to require outpatient dialysis.

**Discussion:** This case depicts a rare presentation of thymoma-associated paraneoplastic glomerulonephritis, specifically manifesting as minimal change disease (MCD). Although an uncommon occurrence, MCD is the most frequent thymoma-associated glomerular lesion and its pathogenesis is linked to an altered immune response of T-cells. MCD associated with lymphocyte-predominant thymoma typically responds well to steroids, however, this patient did not respond. It is important to note that even with thymectomy, patients can continue to exhibit nephrotic syndrome with progressive renal decline, an association that is related to persistent T-cell dysfunction after tumor removal. Additional studies have shown that thymomas are chemosensitive diseases, therefore this case highlights the need for further investigation of chemotherapy in the management of paraneoplastic glomerulonephritis. Of importance, an emphasis on early detection of nephrotic syndrome, initiation of appropriate therapy, and pursuit of a multidisciplinary approach may prevent the progression to end-stage kidney disease.

**Conclusion:** Nephrotic syndrome in an otherwise healthy adult patient should be a red flag for possible malignancy. More specifically, thymomas are known to cause minimal change disease as a paraneoplastic syndrome. Even with surgical excision of the offending tumor, however, renal failure can progress due to persistent T-cell dysfunction.

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CARDIAC TAMPONADE AS A COMPLICATION OF PANCREATIC PSEUDOCYST

First Author: Tasnim Lat, DO Second Author: Austin Lewis, M.D.

Cardiac tamponade is a well-recognized complication of large pericardial effusions, often requiring percutaneous or surgical drainage. However, development of pericardial effusions causing tamponade secondary to a pancreatic pseudocyst may also benefit from administration of somatostatin analogues for long-term management and prevention of recurrent pericardial effusions.

A 50-year-old woman presented with right shoulder pain ongoing for one day. Her medical history included chronic pancreatitis secondary to alcoholism; her prior admission was complicated by a small pancreatic pseudocyst that was not drained. Her vital signs were significant for hypotension and tachycardia. Her physical exam was suggestive of tamponade physiology: pulsus paradoxus, jugular venous distension, and distant heart sounds. An ECG demonstrated sinus tachycardia with low QRS voltage. Bedside echocardiography demonstrated right ventricular collapse in early diastole and a large, posterior pericardial effusion. A CT Chest demonstrated a large pancreatic pseudocyst extending into the mediastinum and abutting the pericardium without definitive fistulous connection. Since her pericardial effusion was located posteriorly, Cardiothoracic Surgery was consulted for open surgical drainage. Fluid analysis was negative for infectious, autoimmune or malignant etiology. Once a pericardial window was established, she underwent drainage of her pseudocyst by Interventional Radiology. Given that the pseudocyst was communicating with the thoracic space, pericardial fluid was analyzed for lipase and amylase levels which revealed levels of 44 and 51 respectively. The etiology of her pericardial effusion was likely chemical pericarditis secondary to pancreatic enzymes originating from the mediastinal pseudocyst, for which she also underwent drainage. However, she had recurrence of pancreatic pseudocysts extending into the mediastinum and recurrence of a small pericardial effusion. She ultimately required an octreotide drip to decrease the size of the pseudocysts. She did not have re-accumulation of pericardial effusion causing tamponade thereafter. This patient had pericardial fluid analysis initially demonstrating no obvious etiology but was eventually found to have positive lipase/amylase levels suggestive of communication with the pancreatic pseudocyst. Despite pericardial window, she had recurrence of pericardial effusion and treatment of recurrent mediastinal pseudocysts were undertaken to control re-accumulation of pericardial effusion.

This case demonstrates the importance of recognizing cardiac tamponade, establishing the etiology of the pericardial effusion and providing definitive therapy to prevent recurrence of the effusion.

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PRIMARY SJÖGREN SYNDROME COMPLICATED BY MYOCARDITIS AND ACUTE HEART FAILURE

First Author: Fiorella Llanos-Chea, MD Alex H. Velasquez, MD Raymundo A. Quintana-Quezada, MD Yi Chun Yeh, MD Balan Prakash, MD

**Introduction** Cases describing anti-Ro/SSA and anti-La/SSB myocarditis are rare and typically present as secondary Sjögren Syndrome (SS) in the setting of a connective tissue disorder.

**Case** A 74-year-old white female with a history of hypertension presented with left shoulder pain, worsening fatigue and dyspnea on exertion. She was noted to have ST elevations in the inferior leads with positive troponin I of 49 ng/mL. A left and right cardiac catheterization demonstrated normal coronary arteries, a Fick cardiac output of 5.38L/min and a pulmonary wedge pressure of 34 mmHg. Echocardiogram showed markedly reduced ejection fraction of 20%. Due to persistent hypotension, she was placed on dopamine and Lasix drip for acute cardiogenic shock complicated with hypoxic respiratory failure and admitted to the intensive care unit.

Early in her hospitalization she had low-grade fevers of 101(F). There was a suspicion that myocardial inflammation was the source of her heart failure. A rheumatologic evaluation revealed a positive ANA titer of 1:80 with speckled pattern, rheumatoid factor of 74 IU/mL, anti-SSA >8.0 AI and anti-SSB of 2.2 AI. Sedimentation rate and C-reactive protein was 62 mm/hr and 31.1 mg/L, respectively. Other rheumatologic and infectious laboratory investigations were negative. Cardiac MRI showed increased late gadolinium enhancement in the inferolateral wall and transmural basal-inferior segment suggestive of edema, consistent with acute myocarditis.

Further inquiry revealed a more than 10-year history of xerostomia and xerophthalmia, but denied other symptoms concerning for systemic lupus erythematosus or rheumatoid arthritis. She was diagnosed with primary SS complicated by cardiac myositis, therefore, she was started on oral prednisone. Her heart failure and hypoxic respiratory failure resolved and she was discharged on maintenance steroid therapy. A repeat echocardiogram done three months later showed both normal systolic and diastolic function.

**Discussion** In the few cases of myocarditis and primary SS previously reported, the diagnosis was made by excluding other causes of cardiac involvement. In SS, most cases are asymptomatic, with pericardial effusion, diastolic dysfunction and pulmonary artery hypertension being the most common echocardiographic findings. Such changes are not specific to the disease and their prognostic significance is undetermined.

Three of the four cases previously reported were treated with steroids, with marked clinical and echocardiographic improvement. This cases presented with nonspecific QS waves and negative cardiac markers, while our case masquerade as an acute coronary syndrome. Although cardiac involvement is relatively common, acute myocarditis and heart failure in the setting of primary SS is a rare entity, therefore one must maintain a high index of suspicion. Corticosteroid therapy appears to be effective, with cardiac function returning to baseline in the few cases reported.

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ST-ELEVATION MI OR THYMOMA?

First Author: Humna Abid Memon, MD Anita Shah, MD Melina Awar, MD

Occlusion of a coronary artery by a thrombus is a widely recognized cause of ST-elevation on an electrocardiogram (ECG). However, up to 2.6% of patients with suspected ST-elevation myocardial infarction (STEMI) have no stenosis on coronary artery angiography, indicating a non-ischemic etiology. Coronary artery stenosis by extrinsic compression, even though rare, is included among such pathologies. We report one such case of ST-elevation in a young adult.

A 36-year-old man, with no known comorbidities, presented with a one day history of intermittent, moderate in intensity, mid-sternal, pressure-like, non-radiating chest pain. The pain had no alleviating or relieving factors and was not associated with any symptoms such as dyspnea, diaphoresis and syncope. Patient reported a similar episode of chest pain in the recent past with a resultant negative cardiac workup. On presentation he had no signs of myasthenia gravis, stable vitals and a normal cardiopulmonary exam. Nevertheless, right bundle branch block with ST elevations in the inferior leads was noted on ECG. Cardiac enzymes were trended and remained negative throughout the hospital course. Due to concern for aortic dissection, CT chest angiogram was done. It revealed a 10.0 x 8.5 x 11.0 cm anterior mediastinal mass impinging on the main and right pulmonary artery, right coronary artery, and right atrium and ventricle. As evidenced by an echocardiogram, the mass however did not result in ventricular outflow tract obstruction or reduction in cardiac output. A subsequent CT-guided core biopsy of the mass illustrated a World Health Organization Stage 1, Type A epithelial thymoma. Thus, ST-elevations due to compression of the right coronary artery by encapsulated thymoma with no coronary vessel invasion was diagnosed. Cardiothoracic surgery was consulted and recommended outpatient surgical resection. The patient remained hemodynamically stable with no recurrence of chest pain during the hospital course and was thus discharged to follow-up with cardiothoracic surgery for thymoma resection in one week.

Thymic masses constitute up to half of all anterior mediastinal masses and often present as a paraneoplastic syndrome, an incidental finding, or local symptoms such as chest pain, dyspnea, or cough. Depending on the clinical stage, treatment considerations vary from surgical resection, chemotherapy to radiation therapy. Complete surgical resection yields the best prognosis. Only one case of ST-elevation secondary to invasion of coronaries by a thymoma has been reported. This case hence illustrates a rare presentation of ST elevation, imitating a STEMI, by extrinsic compression from a Masaoka's Stage 1 thymoma. Furthermore, it demonstrates the importance of recognizing non-coronary causes of ST elevation in those with a low suspicion of acute coronary syndrome.

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THAT ESCALATED QUICKLY: A CASE OF RAPIDLY PROGRESSIVE RENAL FAILURE DUE TO ANTI-GBM DISEASE

First Author: Harsha V Mudrakola, MD Susanne McLaughlin, MD Jose Perez, MD

Introduction: Rapidly progressive glomerulonephritis is a very rare entity, with an incidence of approximately 7 cases per 1 million persons per year. The presentation is often non-specific and extensive laboratory and pathologic testing is required for diagnosis. Of those, only 3% are due to anti-glomerular basement membrane (GBM) disease.

Case Report: A 37 year old Hispanic female with no significant past medical history presented with 3 weeks of progressive fatigue, dyspnea on exertion, abdominal pain, nausea and vomiting. In the past, she had only received medical care for her three pregnancies, all of which were uneventful and ended in uncomplicated vaginal deliveries at term. Three months prior to presentation, the patient established care with a PCP. Labs at that time were significant for creatinine of 1.4 and 2+ proteinuria on urinalysis; no further workup was performed. At the time of presentation, physical exam revealed mild diffuse abdominal tenderness and signs of volume depletion. Laboratory studies were significant for BUN of 142mg/dL, creatinine of 22.2 mg/dL, bicarbonate of 9.1mEq/L. Urinalysis showed 3+ protein and 119 RBC/hpf including frequent dysmorphic red blood cells. The patient was urgently started on hemodialysis which quickly resolved her presenting symptoms. A renal biopsy was performed which revealed anti-glomerular basement disease with 95% crescents. Plasmapheresis and corticosteroid therapy were initiated, but the patient unfortunately did not recover any renal function and she remains on hemodialysis.

Discussion: The incidence of anti-GBM disease is estimated to be less than 1 case per million per year. 60-80% of patients have the well-described classic manifestations of pulmonary and renal disease. A substantial proportion of patients has renal disease alone and present with a wide array of non-specific symptoms, as this patient did. Diagnosis requires presence of anti-GBM antibody in circulation. Without pulmonary involvement manifested by hemoptysis or diffuse alveolar hemorrhage, a renal biopsy is needed to confirm the diagnosis. Management is based removing the circulating anti-GBM antibodies and immunosuppression. Prognosis depends on the stage of renal failure at presentation. This patient unfortunately presented very late and renal failure had already progressed to severe uremia, acidosis, hyperphosphatemia and anemia. Patients requiring dialysis at presentation, as this patient did, almost always subsequently require maintenance dialysis. Renal transplantation has been used in these patients, but should only be done after antibodies are undetectable. Although up to half of transplant recipients show positive staining for IgG in the allograft, progression to clinically significant disease remains rare with only a handful of cases reported in literature.

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A SLEEPING GIANT AWAKENS: A CASE OF PHEOCHROMOCYTOMA DIAGNOSED AFTER ANTIHISTAMINE INDUCED ADRENERGIC CRISIS

First Author: Serena M Okoronkwo, MD Second Author: Ali Mehr, MD

**Introduction:** Pheochromocytoma is a rare adrenal tumor causing hypertensive crisis. The classic triad of symptoms in patients include palpitations, headaches and tremor, however, not every patient manifests with this typical presentation. Here we present a case of pheochromocytoma who had adrenergic crisis after receiving antihistamine medication in the emergency room.

**Case Presentation:** A 45 year old female with history of anxiety and migraines presented to the emergency room with nausea and headache. The initial workup in the emergency room was unremarkable, and the patient was given metoclopramide and diphenhydramine for symptom relief. Shortly after these medications were administered the patient became tachycardic, hypertensive, and hypoxemic. A stat chest x-ray revealed flash pulmonary edema and the patient was intubated for respiratory distress.

A complete metabolic panel and complete blood count were all within normal limits. EKG showed sinus bradycardia and shortened PR interval. Upon review of outside records, it was noted that the patient had a history of cardiac arrest during a prior surgery. She also had a history of transient reductions in systolic heart function with pulmonary edema that occurred perioperatively--subsequent echocardiograms showed a return of normal systolic function.

Based on the patient's history, additional laboratory studies were collected which were significant for elevated plasma metanephrines and normetanephrines. Adrenal CT scan revealed a 3.1 cm right adrenal mass with 67% enhancement washout. The patient was started on phenoxybenzamine and metoprolol therapy and later underwent successful surgical resection of the mass.

**Discussion:** Pheochromocytoma is a rare adrenal tumor affecting 1 to 2 patients per million population. Patients may not always have the classic triad of symptoms. As our case demonstrates, patients may present with adrenergic crisis after induction of anesthesia and after administration of certain antihistamine medications. When pheochromocytoma is suspected, initial testing should include urine or plasma metanephrine levels. Elevated levels should prompt imaging with CT or MRI to localize the tumor.

**Conclusion:** Pheochromocytoma is a rare treatable cause of hypertension. It can be triggered by certain "innocent medications". A high clinical suspicion along with focused, detailed history taking can lead to successful diagnosis. Patients awaiting surgical resection should be started on alpha blockade followed by beta blockade to prevent intraoperative adrenergic crisis. After surgical resection, the patient and their family should be carefully monitored and have biochemical and genetic testing to screen for recurrent disease and familial syndromes associated with pheochromocytoma such as Multiple Endocrine Neoplasia type 2, Neurofibromatosis type 1 and Von Hippel Lindau.

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AN UNUSUAL CASE OF WEIGHT LOSS IN RENAL TRANSPLANT PATIENT

First Author: Anam Omer, MD Second author: Ali Raza, MD Third author: Robert McFadden, MD

**Introduction:** Norovirus is the leading cause of self-limiting gastroenteritis in immunocompetent individuals. Typical presentation includes nausea, vomiting and diarrhea that resolves with supportive care, however, immunocompromised individuals can have an unusual clinical presentation of this gastrointestinal infection.

**Case Description:** A 34-year-old Hispanic female presented with nausea, steatorrhea and unintentional weight loss of thirty-five pounds over a six-month period. She underwent renal transplantation nine years ago. Her anti-rejection medications included mycophenolate mofetil 1000mg twice a day, tacrolimus 1.5mg twice a day and prednisolone of 10mg daily. Physical examination showed a cachectic female with normal vital signs and systemic examination. Pertinent laboratory work-up showed a hemoglobin of 7.8, serum creatinine of 2.1, blood urea nitrogen of 21, amylase of 15, lipase 21, and normal liver enzymes. CT scan of the abdomen was negative for abdominal mass or evidence of chronic pancreatitis. Serological work-up for celiac disease was negative. An esophagogastroduodenoscopy showed diffuse mild inflammation and congestion with minimal blunting of villi in the entire examined small bowel. Mucosal biopsies of the small bowel showed intra-epithelial lymphocytes and Brunner gland hyperplasia. Stool PCR analyses done two months apart were both positive for norovirus. Decision was made to decrease the total amount of immunosuppression in order to eliminate the chronic norovirus infection. Six weeks after decreasing the dose of prednisone and mycophenolate mofetil, the patient had resolution of gastrointestinal symptoms and 15 pounds weight gain. Stool studies at that time were negative for norovirus.

**Discussion:** Norovirus is a calicivirus that is the most common cause of self-limiting non-bacterial gastroenteritis, (95%), in immunocompetent individuals; however it can cause chronic infection in immunocompromised patients, especially renal transplant patients.<sup>1</sup> The symptoms can vary from mild to severe gastroenteritis.<sup>2</sup> In rare cases, weight loss can be a presenting symptom, as seen in our patient. It is hypothesized that intestinal changes caused by chronic norovirus infection lead to malabsorption resulting in the weight loss seen in our patient. Hence, clinicians should keep a high likelihood of suspicion of chronic norovirus infection in immunocompromised patients presenting with non-specific gastrointestinal symptoms or significant weight loss.

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NO MATTER HOW UNCOMMON, YOU CAN ALWAYS BLAME LUPUS

First Author: Veena A Patel, MD

Rheumatologic diseases are known to affect numerous organ systems, especially SLE. Lupus is associated with numerous complications relating to multiple subspecialties, including hematology. Patients often present with cytopenias attributed to immunosuppressive therapies or the autoimmune disease itself. This is a case of a complicated patient with a long hospital course who developed worsening cytopenias due to a rare syndrome associated with SLE.

A 54 year-old African American woman initially presented with lower extremity edema and was found to have nephrotic syndrome. She also had malar rash, joint pains, oral ulcers, normocytic anemia, and +ANA with high dsDNA titer. Kidney biopsy was performed and revealed class IV/V lupus nephritis and she was admitted for further management. She was treated with cyclophosphamide, pulse steroids and required hemodialysis for oliguric renal failure.

Her hospital course was complicated by ARDS secondary to diffuse alveolar hemorrhage from uncontrolled lupus requiring intubation, plasma exchange and another course of pulse steroids. She was also found to have an acute GI bleed, which EGD revealed to be secondary to hemorrhagic gastritis. After these issues resolved, she was noted to have worsening anemia and thrombocytopenia.

On exam, vital signs were stable except for intermittent fevers with a Tmax of 101.2 F. She had no signs of overt bleeding, rashes, or hepatosplenomegaly. Her hemoglobin on admission was 8.4 g/dL and was now ranging between 5-8g/dL. Her platelet count was 327 K/uL and now ranging between 30-70 K/uL. WBC and coagulation panel were within normal limits. Coombs test and HIT panel were negative. CXR was stable. FOBT negative. Peripheral smear showed decrease in platelets and normocytic anemia, no dysplastic cells or schistocytes. And all cultures were negative to date.

At this time she had been on stress dose steroids and broad antibiotics with fungal coverage for over 2 weeks. The cause of her bicytopenia was considered to be multifactorial from SLE and medications. However, her dsDNA titer was low and her last dose of cyclophosphamide was given three weeks prior, making these causes less likely.

Her bicytopenia persisted and she developed new altered mental status with neutropenia. A ferritin level was >2000 ng/mL and triglycerides were elevated. Bone marrow biopsy was done given her worsening counts and showed hemophagocytosis. She was given the diagnosis of hemophagocytic lymphohistiocytosis/macrophage activating syndrome and was started on high dose dexamethasone and intrathecal methotrexate with subsequent improvement in her mental status and cytopenias.

SLE can commonly cause cytopenias due to autoimmune destruction of cell lines, but should improve when lupus is adequately treated. Since our patient's symptoms persisted, HLH/MAS was important to keep on the differential because the treatment differs from standard lupus treatment.



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HERCULEAN OR SISYPHEAN? CROSSFIT INDUCED ACUTE PARASPINAL COMPARTMENT SYNDROME

First Author: Chhaya Patel, MD Following Authors: Dr. Matthew Crowe, Dr. Curtis Mirkes

**Introduction:** Back pain is a common medical complaint and it is estimated that 84 % of adults have had back pain in their life. There is a large differential associated with this complaint and it requires a comprehensive history and physical examination. The differential can range from common diagnoses such as osteoarthritis to more emergent diagnoses such as spinal cord compression. Compartment Syndrome is a medical and surgical emergency which requires practitioners to evaluate and treat quickly when the diagnosis is made. Acute Paraspinal Compartment Syndrome (APCS) is a rare condition of compartment syndrome involving the paraspinal musculature and there are only ten documented cases in the medical literature.

**Discussion:** This is a case of a 23-year-old male with no past medical history who presented with back pain, bilateral lower extremity, and lower back numbness. The day previous he had completed a Crossfit work out consisting of heavy dead lifts and he noticed increasing pain with difficulty ambulating a few hours after. On initial hospital presentation he found to have a CK of 15,000 which progressed to greater than 95,000 despite IV fluid resuscitation and conservative measures. He developed worsening back pain with numbness of his lower extremities concerning for spinal cord involvement. MRI was performed demonstrating changes concerning for paraspinal compartment syndrome and possible myonecrosis. Given these findings, neurosurgery was consulted and performed paraspinal compartment pressures which measured 135 mm Hg. His symptoms and blood CK volume continued to improve and no surgical intervention was necessary.

**Conclusion:** Acute compartment syndrome occurs when there is increased pressure within a compartment that is confined by fascial membranes resulting in compromise of the circulation and function of the tissues within that space. It is generally acknowledged as a medical/surgical emergency. Acute Paraspinal Compartment Syndrome has two types: Type I and type II depending on chronicity. Type 1 has three subtypes: atraumatic, direct trauma, and secondary to non-spinal surgery. This case is an example of Type I atraumatic acute paraspinal compartment syndrome which progressed despite initial conservative treatment. The recommendations regarding intervention are few given the rarity of the disease process. Several cases document surgical intervention as the treatment of choice, which was typically employed after lack of improvement with conservative treatment. This case is an example of conservative management eventually resulting in clinical improvement. This case also displays the importance of clinical suspicion, as there are no concrete or well established guidelines regarding its diagnosis and management. It is important for practitioners to have this as part of their differential when evaluating patients for acute or even chronic back pain.

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ACHROMOBACTER XYLOSOXIDANS?

First Author: Ryle Przybylowicz, MD; Katie Jeans, MD Attending: Philip Alapat, MD

**Intro:** *Achromobacter xylosoxidans* is a rare opportunistic infection typically found in immunocompromised hosts during hospitalization. Primarily it causes bacteremia, pneumonia, and catheter-related infections. However, there have been few reported cases of *Achromobacter* in immunocompetent hosts. We describe a case of a patient with *Achromobacter* bacteremia.

**Case Presentation:** A 74 year old HIV-negative, otherwise non-immunosuppressed male with a past medical history of kidney anomaly of cross fused ectopia and abdominal/pelvic mass discovered in 2008 presented to our hospital with four days of weakness, urinary retention, and constipation. Since discovery of the mass in 2008 he had an unintentional 40-pound weight loss. He was previously treated for *Enterobacter* and Group B Strep urinary tract infections, as well as *Pseudomonas non-aeruginosa* bacteremia.

On presentation he was afebrile with normal heart rate, on room air, but hypotensive with systolic blood pressure 80-100 mmHg. Laboratory workup revealed hypovolemic hyponatremia to 117, leukocytosis to 39,000 with 95% neutrophils, UA with 160 WBCs, and lactate 6.49 concerning for sepsis. CT abdomen/pelvis revealed diffuse colonic dilation to the rectum and a cystic mass adjacent to the bladder. He was admitted to the ICU for septic shock and started on empiric cefepime and vancomycin. He developed refractory septic shock and hypoxemic respiratory failure, requiring intubation. His blood cultures grew *Achromobacter xylosoxidans* susceptible to meropenem, piperacillin/tazobactam, and sulfamethoxazole/trimethoprim. Interventional Radiology was consulted to drain the cystic mass as a possible source for his decompensation and bacteremia. On follow up imaging, the cystic mass appeared to be urinary tract diverticula that expanded over time. The drained fluid grew *Achromobacter xylosoxidans* susceptible only to sulfamethoxazole/trimethoprim and piperacillin/tazobactam, and his antibiotics were adjusted accordingly. His blood and fluid cultures after initiation of antibiotics remained negative.

**Discussion:** *Achromobacter xylosoxidans* is a Gram-negative, aerobic species that typically infects patients who are otherwise immunocompromised. The immunocompromised states previously described include HIV, cystic fibrosis, hematologic malignancies and renal failure. Our patient is unique in that he had no known malignancy or chronic immunosuppressive disease state. In addition, it is generally described as a nosocomially acquired infection. Our patient had not been to see a physician in over a year, and had not been hospitalized in the four years prior to admission. His *Achromobacter* was typical in that it was multi-drug resistant, but blood cultures after initiation of appropriate antibiotics were negative suggesting clearing of the infection.

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GONE WITH THE WIND: A RARE CAUSE OF ACUTE RESPIRATORY DISTRESS

First Author: Ryle Przybylowicz, MD; Shira Sachs, MD Attending: Chirayu Shah, MD

Introduction: Idiopathic acute eosinophilic pneumonia (IAEP) is a rare inflammatory lung disease that presents with fever, dyspnea, and bilateral pulmonary infiltrates. As this disorder is uncommon and typically affects otherwise healthy young adults, practitioners often misdiagnose IAEP as community acquired pneumonia. However, patients with IAEP can develop acute respiratory failure requiring a prompt diagnosis and appropriate treatment.

Case Presentation: The patient is a previously healthy 20 year-old woman who presented with dyspnea and chest pain. Three days prior to presentation, she developed exertional dyspnea that was acute in onset and exacerbated by minimal activity. The patient reported diffuse, non-radiating chest pain that was pleuritic in nature. She experienced chills and night sweats over this time period. Physical exam was notable for dry, bibasilar crackles. CT of the chest revealed ground-glass opacities and interlobular septal thickening in the bilateral lower lobes. An extensive autoimmune workup, respiratory viral panel, and hypersensitivity pneumonitis panel were all negative. On the second day of admission, she developed hypoxia with an oxygen saturation of 85% on room air. By the third day of admission, complete blood count was notable for new eosinophilia with an absolute eosinophil count of 2,960/uL. Eosinophilia was also present on bronchoalveolar lavage (BAL), which was remarkable for a cell count of 1,050 with 72% eosinophils. Cultures, acid-fast staining, and examination for ova and parasites from the BAL fluid were all non-revealing. The patient was diagnosed with IAEP for which she was started on 1 mg/kg of prednisone daily, with rapid improvement in her symptoms. She was continued on steroids at the time of discharge, which will be tapered over the course of one month.

Discussion: While IAEP was first described in 1989, as of 2013, less than 100 patients diagnosed with this disorder had been reported in the literature. While the etiology of IAEP is unknown, two-thirds of affected patients have a significant smoking history. The diagnostic criteria for IAEP includes the acute onset of respiratory symptoms, bilateral infiltrates on imaging, hypoxia with an oxygen saturation of less than 90% on room air, and eosinophilia, with BAL cell count and differential showing greater than 25% eosinophils. Peripheral eosinophilia is typically not found on presentation but develops over the course of treatment. The clinical course of IAEP can be severe, with up to 60% of patients developing acute respiratory distress syndrome. However, symptoms improve within 48 hours and completely resolve within one week after the initiation of corticosteroids. Recurrence is exceedingly rare and is most often described in patients who resume cigarette smoking following treatment. This case represents a classic clinical presentation of IAEP, and highlights the importance of maintaining a broad differential in young patients with respiratory distress.

NOT YOUR TYPICAL COMPLEMENTS: ATYPICAL HEMOLYTIC UREMIC SYNDROME SECONDARY TO STREPTOCOCCUS PNEUMONIAE

First Author: Katherine B. Salciccioli, MD Second Author: Anthony Wiseman, MD Third Author: Benjamin Gold, MD Fourth Author: Salman Ahmed, MD Fifth Author: Anisha Sethi, MD

Clinical Summary: A previously healthy 36-year-old woman presented to the emergency room with one day of fever and an episode of presyncope in the setting of 3 weeks of sinus pressure and headache. She denied cough, chest pain, vomiting, and diarrhea. She was febrile to 103.1° Fahrenheit, tachycardic to 123 beats per minute, and hypotensive to 92/54 mmHg. Her hemodynamics normalized with fluid resuscitation and acetaminophen. Physical exam was remarkable for tachycardia and bilateral maxillary sinus tenderness. Labs showed mild leukocytosis but were otherwise unremarkable, including cerebrospinal fluid analysis. Chest x-ray was normal and maxillofacial CT showed diffuse sinusitis. Empiric vancomycin and cefepime were initiated. 4/4 blood culture bottles grew pan-sensitive *Streptococcus pneumoniae* the following day and antibiotics were narrowed to ceftriaxone.

On day 2 of hospitalization, the patient became confused and her urine output decreased. Labs were notable for new anemia, with hemoglobin decreased from 14.0 to 11.5 g/dL, and thrombocytopenia, with platelet count decreased from 297,000 to 22,000/uL. She had acute kidney injury with blood urea nitrogen (BUN) increased from 18 to 49 mg/dL and creatinine increased from 0.8 to 4.8 mg/dL. PT, PTT, and fibrinogen were normal. D-dimer was elevated to >20 ug/mL. Haptoglobin was undetectable. ADAMST13 activity levels were normal at 61%. Peripheral smear showed many schistocytes.

The patient became progressively more acidotic and somnolent requiring intubation and mechanical ventilation for airway protection. Hemodialysis was started for anuric renal failure without improvement in her mental status. After considering ongoing severe sepsis, disseminated intravascular coagulation (DIC), and thrombotic thrombocytopenic purpura (TTP), the diagnosis of atypical hemolytic uremic syndrome (HUS) was made. The patient received eculizumab, a monoclonal antibody against terminal complement cleavage of C5, which was recently approved for treatment of atypical HUS. The morning after administration of eculizumab, the patient's mental status returned to baseline and she was extubated the following day without incident. She received two more weekly doses of eculizumab with resolution of thrombocytopenia and microangiopathic hemolytic anemia (MAHA). She began making urine but remained hemodialysis-dependent at discharge with plans to continue biweekly eculizumab infusions as an outpatient.

Discussion: Atypical HUS is an uncommon disease caused by dysregulation in the complement cascade system. It manifests as the triad of MAHA, thrombocytopenia, and acute renal failure. The diagnosis remains clinical in the absence of a Shiga-toxin producing pathogen and with preserved ADAMST13 levels. The disease is more common in pediatric patients, where it represents an estimated 20% of all HUS. Atypical HUS has been associated with severe *S. pneumoniae* infection secondary to pneumonia and meningitis in children but only rarely with sinusitis. To our knowledge, this is the first such case to be reported in an adult.

## A HEARTBREAKING CASE OF LUPUS

First Author: Tasneam Shagrani, MD Second Author: Sebastian Bruera, MD

**Introduction:** Patients with systemic lupus erythematosus (SLE) frequently have cardiac manifestations that may include involvement of the pericardium, endocardium, myocardium, conduction system, valves and coronary arteries. Myocarditis is a rare manifestation of SLE and can be complicated by conduction defects with high-degree heart block being exceedingly rare. We present an atypical presentation of lupus myocarditis complicated by symptomatic complete heart block.

**Case:** A 33 year-old female with SLE presents with 2 days of left-sided, pressure-like chest pain occurring during exertion with resolution after ten minutes of rest. Each episode was associated with shortness of breath. She denies any history of chest pain, fevers or worsening pain with inspiration. Vital signs were notable for heart rate of 108 bpm. Cardiac exam revealed sinus tachycardia, with no murmurs or rubs. EKG was notable for sinus tachycardia with no ischemic changes. Her troponin was 1.03 ng/ml and continued to rise to a peak of 8.25 ng/ml with CKMB ng/ml of 42.8. A transthoracic echocardiogram showed a preserved ejection fraction with no regional wall motion abnormalities or evidence of pericardial effusion. A left-sided cardiac catheterization showed no evidence of coronary artery disease, establishing a diagnosis of likely lupus myocarditis. She received treatment with intravenous methylprednisolone. Shortly after she had a syncopal episode and was found in third-degree atrioventricular heart block. She emergently received a permanent pacemaker and clinically improved.

**Discussion:** The heart is frequently involved in SLE with pericarditis being the most common manifestation. Lupus myocarditis is uncommon with a reported prevalence of 9% in patients with SLE. It often presents with symptoms of heart failure with global or regional hypokinesis evident on echocardiogram. This case presented a diagnostic challenge given the atypical symptoms and lack of echocardiographic findings. The patient's typical chest pains in the setting of rising cardiac biomarkers were concerning for acute coronary syndrome. Coronary artery disease is the most common cause of death in patients with long-standing SLE, with patients prone to premature coronary atherosclerosis. Echocardiography revealed no wall motion abnormalities and left heart catheterization showed no coronary disease. The diagnosis of myocarditis was established on the basis of elevated cardiac enzymes and absence of coronary atherosclerosis. Unexpectedly, our patient developed third-degree AV heart block, which we attribute as a complication of the myocarditis. While conduction abnormalities are a sequel of myocarditis in patients with SLE, complete heart block is extremely uncommon. This case illustrates an atypical presentation of lupus myocarditis with a rare and serious complication of symptomatic complete heart block. Clinicians should consider myocarditis as a cardiac manifestation in SLE patients with chest pain or elevated cardiac enzymes. Patients with lupus myocarditis should be monitored closely for potentially fatal conduction disturbances.

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DOUBLE TROUBLE: THE DIAGNOSTIC DILEMMA OF DIFFUSE ALVEOLAR HEMORRHAGE

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**Introduction:** Parasitic infection with *Strongyloides stercoralis* is typically asymptomatic in the immunocompetent host. In patients receiving corticosteroids, an autoinfectious state can occur leading to significant parasite burden and high mortality. Symptoms are characteristically gastrointestinal and pulmonic, with diffuse alveolar hemorrhage (DAH) being a rare and potentially fatal complication. We present a case of patient receiving treatment with corticosteroids for microscopic polyangiitis (MPA), who develops DAH from strongyloidiasis.

**Case:** A 47-year-old Honduran female with recently diagnosed microscopic polyangiitis presents with 2 days of shortness of breath. She reports one week of abdominal pain, constipation and non-bloody emesis. Two months prior she was diagnosed with rapidly progressive crescentic glomerulonephritis secondary to MPA. She received treatment with cyclophosphamide and high-dose corticosteroids. She remained on prednisone 80mg on her current presentation. Vital signs were: BP 110/70, HR 108, RR 24, temp 98.6F, O2 sat 88%. Her physical examination was notable for bilateral crackles on lung auscultation and diffuse tenderness to abdominal palpation. CBC was remarkable for Hgb: 6.9 g/dl, WBC: 11,600/dl with 8% eosinophils. Computed tomography of the chest showed bilateral ground-glass opacities. Shortly after presentation she developed hematemesis and was intubated for airway protection and hypoxemic respiratory failure. Bronchoalveolar lavage (BAL) revealed diffuse alveolar hemorrhage and the presence of *Strongyloides stercoralis*. EGD with duodenal biopsy showed inflammation with severe *S. stercoralis* infestation and no evidence of vasculitis in the submucosal vessels. A diagnosis of strongyloidiasis hyperinfection syndrome was established and she received treatment with Ivermectin and Albendazole. She clinically improved and stool examination confirmed parasite eradication.

**Discussion:** *Strongyloides stercoralis* is an intestinal nematode endemic to tropical regions. Infected hosts are often asymptomatic, with immunosuppressed patients being at risk for developing fatal hyperinfection syndrome (HIS). When cell-mediated immunity is depressed, autoinfection with resultant high larvae burden in the lungs and gastrointestinal tract develops. The mortality rate of HIS is as high as 90%. Manifestations include abdominal pain, vomiting, diarrhea, ileus, cough, wheezing, and rarely DAH. This case presented a diagnostic challenge as the cause of DAH was initially attributed to a known diagnosis of MPA. While DAH is a well-recognized complication of MPA, the identified *S. stercoralis* in the BAL makes the nematode the likely culprit for the patient's respiratory compromise. Recognition of strongyloidiasis as a potential cause of DAH is essential for early treatment of this highly fatal disease. To date, there are no existing guidelines regarding prophylaxis or screening for infection among immunocompromised patients. Given the high mortality rate of strongyloidiasis, clinicians should strongly consider screening or providing prophylaxis in patients with a travel history to endemic regions who are to receive corticosteroids.

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ACYCLOVIR RESISTANT HERPES SIMPLEX VIRUS (HSV) MENINGO-ENCEPHALITIS IN AN IMMUNOCOMPETENT INDIVIDUAL

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A 41 year old previously healthy Indian woman presented with a history of fevers and altered mental status for three days. Pertinent findings on examination were an ill appearing patient with a temperature of 103.6 F and neck stiffness. Computerized tomography (CT) of the head showed a small acute cortical infarct in the parasagittal aspect of the left frontal lobe. Cerebrospinal fluid analysis (CSF) was as follows- protein- 40, glucose 51, WBC- 123 (89% lymphocytes), RBC- 104 and a positive HSV polymerase chain reaction (PCR). Despite appropriately timed and dosed therapy with Acyclovir, the fever persisted. A follow up MRI demonstrated multiple hemorrhagic foci of abnormal signals in the cerebellum, left thalamus and frontal lobe. A trans-esophageal echocardiogram and a CT angiogram were negative for endocarditis and vasculitis respectively. A repeat CSF analysis performed seven days later showed the following- protein- 166, glucose 46, WBC- 620 (89% lymphocytes), RBC- 10000 with a positive HSV PCR. The patient was subsequently treated with Foscarnet and recovered dramatically as evidenced by the following CSF findings- protein-66, glucose- 42, WBC- 26, RBC- 12 and a negative HSV PCR.

HSV drug resistance should be considered in immune-competent hosts especially with the involvement of immune privileged sites, namely the cornea and central nervous system. Management of drug resistant HSV should account for: virus compartmentalization, emergence of multi-drug resistance, heterogeneity and dynamics of the viral populations. Monitoring the emergence of drug resistance is fundamental to adjust antiviral therapy. Research platforms for rapid typing of drug resistance as well as development of novel antivirals may help guide clinicians in treatment decisions.

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A CASE OF CEFEPIME-INDUCED APHASIA IN A PATIENT WITH BILATERAL PSEUDOMONAS/MRSA OSTEOMYELITIS OF THE FEET

First Author: Jeremy Walker, DO, CPT, MC, USA Second Author: Mark True, MD, Colonel, MC, USAF

Introduction: Neurotoxicity from cefepime has been documented in case reports previously, especially in patients with renal insufficiency. Prior mechanisms for the aphasia have been described as non-status epilepticus seizures. We present a case of a patient on a long course of high dose cefepime for multi-drug resistant pseudomonas and MRSA osteomyelitis of the feet causing aphasia without seizure activity.

Case Report: An 81-year-old male with history of chronic kidney disease stage 3 was transported from his acute rehabilitation for altered mental status. He was obtunded, not following commands, not answering questions and would only move his eyes. According to his wife, his mental status had been declining over the past 4-5 days. Head CT head and brain MRI did not show any acute changes to explain declining mental status. We were unable to perform a lumbar puncture due to the inability to properly position this patient secondary to mental status and lack of motor function. An electroencephalogram (EEG) demonstrated non-specific global slowing consistent with encephalopathy without epileptiform activity. Of note, he had been admitted several weeks earlier for bilateral calcaneal osteomyelitis that grew *P. aeruginosa* (sensitive only to cefepime) and MRSA for which he was on cefepime 2gm IV every 8 hours and vancomycin 1gm IV every 24 hours. These were started 8 days prior to symptom onset. Infectious Disease was also consulted for the possibility of his AMS being secondary to neurotoxicity from cefepime and for an alternative antibiotic regimen. After considering the possibility of cefepime toxicity with the Neurology and Infectious Disease services, we decided to stop cefepime and switch to doxycycline 100 mg twice a day. Within 24 hours, the patient was able to speak 1 to 2 words though was still confused. After 48 hours, he began to answer questions appropriately. On the fourth day, his motor function began to return. On the fifth day, he was able to return to his prior acute rehabilitation facility.

Discussion: Suspicion of neurotoxicity from cefepime was considered after discussion between Infectious Disease, Neurology and the primary team. Prior cases of cefepime-neurotoxicity demonstrated seizure activity on EEG, whereas this patient's study showed non-specific encephalopathy. This patient was on high doses of cefepime for 8 days to treat MDR *P. aeruginosa* which, in setting of chronic kidney disease, could cause encephalopathy. Although an infectious etiology was not definitively ruled out with a successful lumbar puncture, this patient's improved mental status within 48 hours after cefepime discontinuation makes an infectious cause much less likely than neurotoxicity.

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DON'T SKIP A BEAT: AUTOIMMUNE MYOCARDITIS ASSOCIATED ATRIOVENTRICULAR BLOCKADE

First Author: Theresa Nguyen Wenker, MD Second Author: Gregory Constantine, MD Third Author: I-Hui Chiang, MD

**Introduction:** Lupus myocarditis is an uncommon disease with variable clinical presentations. Clinicians must remain vigilant in the diagnosis and management of cardiovascular complications of autoimmune diseases.

**Case Presentation:** A 33-year-old Hispanic female with a 10-year history of systemic lupus erythematosus (SLE) and class V lupus nephritis presented with a 3-day history of intermittent, left-sided chest pressure and shortness of breath. Episodes occurred spontaneously with radiation down the left arm and neck. Symptoms were unchanged by exertion and exacerbated by deep inspiration. She denied orthopnea, paroxysmal nocturnal dyspnea, or lower extremity edema. Despite her tachycardia, she was in no distress, breathing comfortably with an oxygen saturation of 98% on room air. Physical exam revealed a regular rhythm without friction rub or murmurs. There was no jugular venous distension. Lung sounds were clear. Notably absent were malar rash and synovitis, manifestations of her prior lupus flares. Serum troponin was elevated at 1.03, peaking at 8.25. Viral serologies including Coxsackie A IgM, Epstein-Barr virus IgM, and Parvovirus B19 IgM were non-reactive. EKG revealed sinus tachycardia without ST changes. Computerized tomography with intravenous contrast was negative for pulmonary embolism. Cardiac catheterization revealed no angiographic evidence of coronary artery disease. Transthoracic echocardiogram (TTE) showed normal systolic function without wall motion abnormalities. Patient's clinical course was complicated by syncope during which telemetry revealed third degree atrioventricular block without ventricular escape. She was treated with pulse corticosteroids and mycophenolate for autoimmune myocarditis. Given her conduction abnormalities, a permanent pacemaker was implanted prior to hospital discharge.

**Discussion:** Cardiac manifestations of SLE are numerous, as all anatomical structures of the heart may be involved. Coronary artery disease remains the leading cause of death in patients with SLE due to accelerated atherosclerosis. Though uncommon, patients with SLE are at increased risk for developing both primary and secondary forms of autoimmune myocarditis. Presentations are variable, often mimicking myocardial infarction with elevated cardiac markers. Echocardiography does not establish the diagnosis but may reveal systolic or diastolic dysfunction. Importantly, normal echocardiography does not exclude myocarditis as conduction abnormalities may be sequelae or the only presenting complication and may be associated with anti-U1-RNP and Ant-Ro antibodies. First-degree block may often be transient, with complete heart block occurring rarely in adults. Endomyocardial biopsy may provide definitive histology but is invasive and often lacks appropriate sensitivity and specificity, thus it is not widely utilized. Therefore, the diagnosis of autoimmune myocarditis is largely based on clinical suspicion.

Treatment includes high dose intravenous corticosteroids and immunosuppressive therapy. Additional management is directed toward possible sequelae of myocarditis, such as complications related to congestive heart failure or arrhythmias. Little long-term data are available, and unfortunately cardiovascular involvement in patients with SLE remains a significant source of morbidity and mortality.

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THE IMITATOR OF BRAIN METASTASES: AN UNCOMMON INFECTIOUS CAUSE

First Author: Betty Yang, MD; Nicolas Cortes-Penfield, MD; Sara Fish, BS; Andrew Caruso, MD

**Learning objectives**

- 1) Recognize that CNS histoplasmosis can occur in immunocompetent patients
- 2) Identify the clinical presentation of CNS histoplasmosis
- 3) Recognize the danger of diagnosis based on radiography alone

**Case:** A 59-year-old man with a 30-pack-year smoking history and coronary artery disease presented to an outside hospital with a 30lbs weight loss, weakness, and falls. He had been well until three months prior, when he began to notice progressive weight loss, gait unsteadiness and weakness in his left arm and leg. He denied fevers, chills, headaches, change in vision, loss of consciousness, and seizures; however, his wife noticed worsening emotional lability. He had been employed by the natural gas industry, digging trenches and laying pipelines throughout Texas, Oklahoma, and Louisiana. He had no known immunodeficiency or history of recurrent or unusual infections.

On presentation his vital signs were normal. Examination of the heart, lungs, abdomen, lymph nodes and skin were unremarkable. His neurologic exam revealed slurred speech, mild palsy of the left facial nerve, and diminished strength in the left arm and leg with preserved sensation and reflexes. CT head and subsequent MRI showed innumerable enhancing masses, some with hemorrhagic rims and extensive vasogenic edema, and obstructive hydrocephalus. Based on radiologic findings he was told he had metastatic cancer with poor prognosis. A ventriculoperitoneal shunt was placed for hydrocephalus, and a brain biopsy was performed.

Pathology ultimately revealed many yeast on GMS stain with morphology consistent with *Histoplasma capsulatum*. CSF Histoplasma antibody and antigen were positive; urine Histoplasma antigen tested above the detection threshold, below the positive range. HIV was confirmed negative. The patient started liposomal amphotericin B and was transferred to the Houston VA for further care. His course was complicated by acute CVA and MSSA ventriculoperitoneal shunt infection, treated with nafcillin and shunt replacement. After completing 6 weeks of amphotericin, he was discharged on itraconazole with outpatient infectious disease follow-up.

**Discussion:** *Histoplasma capsulatum* is a dimorphic soil fungus endemic to the Ohio and Mississippi river valleys. CNS histoplasmosis generally affects immunocompromised individuals; however, 20-30% of these patients are reported to be immunocompetent. CNS histoplasmosis typically manifests as chronic meningitis or meningoencephalitis. Presentation may include headaches, altered mentation, acute focal neurologic deficits, or acute strokes. Weight loss and constitutional symptoms are also common. Because no single test is sensitive for CNS histoplasmosis, a careful exposure history and a high index of suspicion are key to early diagnosis.

This case highlights the danger of diagnosing based on radiography alone. The high suspicion of metastatic malignancy drove his management initially and may have delayed treatment.

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## NITROGLYCERIN-INDUCED ASYSTOLE

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**Introduction:** Nitroglycerin is used countless times around the world every day to treat angina. An incredibly rare adverse reaction from this medication is transient asystole.

**Case Description:** A 53-year-old male with no known cardiac history presented to the ED for two days of progressive chest pain. The patient was given nitroglycerin for therapy. After his second dose, the patient lost consciousness. Asystole was noted on the monitor. He reverted to junctional bradycardia with a pulse spontaneously and was then given a dose of atropine to raise his heart rate to a normal range. A resting EKG showed no abnormalities. The patient was admitted. His stress test the next day was normal and the patient was discharged.

**Discussion:** Nitroglycerin-induced hypotensive-bradycardia has been described in the literature only a few times. Even rarer, cases of nitroglycerin-induced asystole have been published less than a dozen times. Attempts at predicting what patients experience this rare reaction have been futile considering nitroglycerin-induced asystole can occur in patients with active ischemia or in patients with no cardiac pathology. It can also present in patients who have previously taken nitroglycerin without complications. Some researchers suggest that nitroglycerin directly affects the CNS, however studies for this theory remain inconclusive. One suggested mechanism is the Bezold-Jarisch reflex but this theory has been difficult to prove due to the sheer rarity of this adverse effect. Patients who experience nitroglycerin-induced hypotensive-bradycardia or asystole have been found in a significant percentage in groups of people with delayed phase shift reactions. However, screening for delayed phase shifts involve strenuous tilt table testing. Therefore, at this time no reasonable screen exists for nitroglycerin-induced asystole. Healthcare professionals should be aware of this incredibly rare adverse effect from a frequently-used medication and must remain vigilant when providing patients with nitroglycerin.

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IMMUNE RECONSTITUTION INFLAMMATORY SYNDROME AS A CAUSE OF COGAN'S SYNDROME IN A HIV-POSITIVE MALE

CPT Karoline Johnson, MD (Associate) Christina Schofield, MD (FACP)

Introduction: Cogan's syndrome is a rare systemic inflammatory disorder that typically presents with interstitial keratitis and audiovestibular signs and symptoms, although a number of patients also present with a life-threatening systemic vasculitis. While the cause remains unknown, it is presumed to be an autoimmune reaction to an autoantigen in the cornea and inner ear and a similar mechanism is suspected in those patients that also present with systemic vasculitis. We present a case of Cogan's Syndrome in an HIV-positive male felt to be related to Immune Reconstitution Inflammatory Syndrome (IRIS).

Case: A 44-year-old HIV-positive male with medication nonadherence experienced a decline in CD4 count from 318 to 81 cells/mL over 6 months, for which he agreed to reinstate a combination antiretroviral therapy (cART) following 2 years of treatment noncompliance. A couple days after reinstating cART, he presented to a local Emergency Room for evaluation of bilateral eye pain, blurred vision, and a headache. A day after admission he developed gait ataxia and bilateral severe sensorineural hearing loss. An MRI of his brain demonstrated bilateral cerebellar subacute infarcts. His symptoms were initially attributed to a stroke; however, further ophthalmologic and auditory evaluation demonstrated bilateral anterior uveitis, interstitial keratitis, and vestibuloauditory dysfunction, consistent with Cogan's Syndrome. The patient was started on oral prednisone and experienced an improvement of his systemic symptoms although only minimal improvement of his audiovestibular symptoms. His ocular symptoms were treated symptomatically and resolved.

Discussion: We present a case of Cogan's Syndrome in a patient with AIDS after recent initiation of cART with resultant IRIS. IRIS is a systemic inflammatory response that occurs following the initiation of cART in HIV-infected individuals. Autoimmune conditions such as thyroiditis, Graves' disease, and sarcoidosis have been reported in the setting of IRIS; However, Cogan's Syndrome attributable to IRIS has not been previously reported to our knowledge. In this case, the patient experienced a systemic inflammatory response of audiovestibular, ocular, and small vessel vasculopathy 2 days after reinstating cART. This robust inflammatory response is most consistent with the immune response observed with IRIS. A literature review found only two previous reported cases of Cogan's Syndrome in HIV patients, but neither occurred around the initiation of cART. Providers should be cognizant of the association of autoimmune conditions with IRIS in AIDS patients with recent initiation of cART.

## METASTATIC MEDULLARY THYROID CARCINOMA PRESENTING AS CHRONIC DIARRHEA

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Medullary thyroid carcinoma (MTC) is a neuroendocrine tumor of the parafollicular or C cells of the thyroid gland. These cells produce calcitonin. Bulky, metastatic disease produces calcitonin in quantities that can cause systemic symptoms such as facial flushing and diarrhea.

A 27 year old man with no significant past medical history arrives to his primary care manager with watery diarrhea of four months duration. He reports upwards of ten bowel movements daily, noting his stools are yellowish, oily, and contain mucous. The urge to defecate would wake him from sleep. He reported mild fatigue and ten pound weight loss over twelve months. Removal of dietary gluten and lactose had not improved symptoms. Four months prior the patient was in excellent health. He was referred to gastroenterology.

The patient was seen by gastroenterology two months later. Blood and stool studies evaluating for infection, gluten intolerance, inflammatory bowel disease, hyperthyroidism and malabsorption returned negative. He began loperamide and a high fiber diet. He returned two months later with minimal improvement and underwent a colonoscopy with biopsies which returned normal. Two months after his colonoscopy, the patient returned to his primary care manager complaining of swollen neck glands and was diagnosed with cervical lymphadenopathy. No further workup was performed until next month, when he again saw gastroenterology with minimal symptom improvement. The combination of swollen neck glands along with minimally improved diarrhea prompted a CT scan of the chest/abdomen/pelvis revealing a large thyroid nodule and several areas of likely metastasis including the bilateral lungs, spine, iliac crest, and liver. He was sent to otolaryngology for biopsy.

Further questioning revealed he had been experiencing dysphagia for three to four months. Biopsy of the thyroid mass revealed MTC, prompting oncology referral. Calcitonin levels were 52,894 pg/mL (normal 0.0-8.4). The patient was referred to a specialty center for surgical debulking prior to initiating chemotherapy. He underwent surgery, however only a right hemithyroidectomy was able to be accomplished. One month later he was started on vandatenib, a systemic tyrosine kinase inhibitor approved by the FDA for treatment of metastatic MTC. His bowel movements reduced to once daily after a week of therapy. Currently, he remains on vandatenib and feels well, with calcitonin levels of 825 pg/mL.

This patient's case demonstrates the importance of a broad differential diagnosis for common patient complaints. While the most likely diagnosis can be pursued after the initial visit, continued visits for the same complaint should prompt further investigation.

A PYRRHIC VICTORY NO LONGER?: A CASE SERIES OF TWO PATIENTS WITH AUTOIMMUNE DISORDER-INDUCED HEMOPHAGOCYTIC LYMPHOHISTIOCYTOSIS TREATED SUCCESSFULLY WITH RITUXIMAB AND MODIFIED HLH-2004 PROTOCOL

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Introduction: Hemophagocytic lymphohistiocytosis (HLH) is a rare disease characterized by aberrant immune hyperactivation of T lymphocytes and macrophages driven by cytokine dysfunction. Clinical features include fever, hyperferritinemia, hepatomegaly, lymphadenopathy, cytopenias, and encephalitis. HLH is categorized into primary and secondary etiologies. Primary HLH is caused by genetic abnormalities and commonly affects children. Secondary HLH is usually triggered by an infection, autoimmune process, or malignancy. Treatment per the HLH-2004 protocol includes etoposide, cyclosporine, dexamethasone and intrathecal methotrexate, which has significant toxicity. There is a paucity of case reports that less toxic treatments such as rituximab may be effective in treatment of secondary HLH. We describe two cases of HLH caused by underlying autoimmune conditions, rheumatoid arthritis (RA) and systemic lupus erythematosus (SLE), both treated successfully with rituximab as part of their modified HLH-2004 protocol.

Case 1: A previously healthy 22-year-old African American female was admitted for pancytopenia, constitutional symptoms, and arthritis. Evaluation resulted in diagnosis of SLE based on rash, inflammatory arthritis, cytopenias, positive anti-nuclear antibody, and high titers of anti-Smith and anti-ribonucleoprotein antibodies. HLH was diagnosed based on the following HLH-2004 criteria: fever, markedly elevated ferritin >7500 ng/mL, pancytopenia, elevated liver associated enzymes, hypofibrinogenemia, hepatosplenomegaly, low NK cell activity, and elevated CD25/IL-2 receptor. She was started on the HLH-2004 treatment protocol but did not tolerate treatment. Etoposide was discontinued and she was treated with rituximab. Her clinical symptoms, complete blood count, ferritin, and triglycerides all normalized.

Case 2: A 64-year-old Vietnamese female with a history of rheumatoid arthritis (RA) complicated by vasculitis was admitted with multiorgan failure requiring intubation. HLH was diagnosed based on fevers, cytopenias, elevated ferritin (highest 47,000), and hypofibrinogenemia along with a bone marrow biopsy demonstrating hemophagocytosis. She was initially treated with a modified HLH-2004 protocol to include etoposide, dexamethasone, and rituximab. Cyclosporine was held due to renal impairment. She has been without recurrence since finishing her treatment.

Discussion: The current treatment standard for HLH has significant toxicities, some of which can be lethal. There is an impetus for more tolerable treatment options. We can find only one published case of rituximab successfully used in the treatment of HLH secondary to SLE. Our case series is unique in that both patients had underlying autoimmune diseases and responded very well to rituximab therapy as part of their treatment protocol. Rituximab has a much more favorable side effect profile and if shown to work well in larger studies, may shift the paradigm of HLH treatment. This case series highlights the importance for further research into the role rituximab therapy in the treatment of HLH.

## A CASE OF EXTENDED SPECTRUM BETA-LACTAMASE ESCHERICHIA COLI PROSTATITIS: A POTENTIAL TREATMENT NICHE FOR FOSFOMYCIN

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**Background:** Fosfomycin is bactericidal agent with *in vitro* activity against multiple microorganisms commonly found to be urinary pathogens. As a phosphoric acid derivative, fosfomycin inhibits bacterial wall synthesis and is available for oral administration as a single-dose 3 gram sachet. It is currently approved for treatment of uncomplicated cystitis in women. Antibiotic treatment options for chronic prostatitis can be limited due to inadequate tissue specific penetration and resistance. The emergence of multi-drug resistant organisms (MDROs) is a growing problem in healthcare. Parenteral therapy with broad-spectrum antibiotics are often used to treat such infections. Fosfomycin may be a potential treatment option; however, there is limited data on the efficacy and optimal dosing for MDRO chronic prostatitis.

**Case:** A 73 year old male, who had recently received external beam radiation and hormone therapy for high risk prostate cancer was referred to the Infectious Disease clinic for refractory prostatitis. He initially presented to the Urology clinic one week after prostate biopsy reporting new dysuria. An extended spectrum beta-lactamase (ESBL) *Escherichia coli* was isolated from his urine. He failed several courses of nitrofurantoin and was given a treatment course of fosfomycin (3grams q72hr for 3 doses) without sustained improvement in symptoms. The ESBL *E. coli* isolate remained susceptible to fosfomycin, 3grams oral daily was subsequently prescribed. He completed a 30 day course and was re-evaluated in the ID clinic. He tolerated the course well with no reports of adverse side effects from the regimen. The main obstacle during his treatment course was difficulty obtaining the medication. Five days after completing his course of treatment he remained asymptomatic and a repeat urine culture was sterile.

**Discussion:** Previous case reports have shown promise using fosfomycin in the treatment of MDRO chronic prostatitis.<sup>1</sup> Extended courses of treatment, as long as 12-16 weeks, with dosing varying from every 72 hours to twice a day, have been described.<sup>2</sup> Other studies trialing short courses of fosfomycin for prostatitis report increased rates of failure.<sup>3</sup> This case is unique in that once a day oral dosing of fosfomycin was utilized for 30 days of therapy resulting in resolution of his ESBL *E coli* prostatitis. Oral fosfomycin may have a niche in treating MDRO urinary tract infections and prostatitis in afebrile hemodynamically stable patients without bacteremia. Potential benefits of fosfomycin therapy is the ability for oral administration, likely adequate penetration into prostatic tissue, tolerability, use renal and hepatic insufficiency, and lower cost owed to the avoidance of PICC line placement, home health nursing, and parenteral antibiotics. Further studies are needed to evaluate fosfomycin as a treatment option for MDRO prostatitis in addition to determining optimal dosing and length of therapy.

1. Grayson et. al., "Fosfomycin for Treatment of Prostatitis: New Tricks for Old Dogs." *Clinical Infectious Disease*, 2015.
2. Neune et al., "Experience with Fosfomycin for treatment of urinary tract infections caused by multi drug resistant organisms." *Antimicrobial Agents and Chemotherapy*, 2012.
3. Falagas et. al., "Fosmycin: The Current Status of the Drug." *Clinical Infectious Disease*, 2015.

A ZEBRA WITH PLUCKED CHICKEN SKIN: A RARE CONNECTIVE TISSUE DISORDER

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Heritable connective tissue disorders (HCTD) are a family of genetic diseases with diverse cutaneous and systemic manifestations. While some are well known, including Marfan syndrome and Ehlers-Danlos syndrome, less common HCTD's can evade diagnosis well into adulthood. Here we present a case of a rare HCTD, pseudoxanthoma elasticum.

The patient was a 35-year-old female evaluated for a long-standing rash on the lateral aspect of her neck bilaterally. The rash was characterized by folds of redundant skin with coalescing yellow papules and plaques forming a "plucked chicken skin" appearance. She had a similar rash on her bilateral axillas and groin. It had been present for multiple years and was asymptomatic. Her past medical history was significant for hypertension and chronic constipation. Family history was unremarkable. Review of systems was positive for bilateral posterior thigh pain occurring with exertion and relieved with rest. Her physical exam was significant for a blood pressure of 140/100, the above-mentioned rash and yellow macules on her labial mucosa. Angioid streaks were found on dilated ophthalmoscopic exam. ABI of the right and left lower extremities were 0.85 and 0.79, respectively. Chest radiograph, EKG and echocardiogram were unremarkable. The patient's distinctive rash, bilateral angioid streaks and peripheral vascular disease were consistent with a diagnosis of pseudoxanthoma elasticum (PXE). PXE is a HCTD occurring secondary to a loss of function mutation of the ABC-C6 protein. It is inherited in an autosomal recessive pattern with an estimated prevalence of 1:50,000-100,000 worldwide. Loss of ABC-C6 function leads to aberrant tissue mineralization resulting in characteristic dermatological findings ("plucked chicken skin" lesions) and systemic disease including premature coronary artery disease, peripheral artery disease, gastrointestinal bleeding and cerebrovascular accidents. Associated ophthalmologic findings range from angioid streaks to sudden and complete vision loss. PXE is diagnosed clinically. Pathohistological findings include basophilic clumping and fragmented elastic fibers in the deep dermis. PXE with angioid streaks, as seen in our patient, is referred to as Grönblad-Strandburg syndrome. There is no definitive treatment for PXE. Some evidence supports a low calcium diet (400-500 mg daily). Additional recommendations include bi-annual ophthalmologic exams with laser photocoagulation as needed and annual cardiology follow up. Bothersome skin folds can be surgically excised. Wound healing is not impaired in PXE. Our patient was advised to adhere to a low calcium diet and to follow up with ophthalmology (6 months) and cardiology (1 year).

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## CHYLOTHORAX: AN UNUSUAL TYPE OF PLEURAL EFFUSION

First Author: LT Dean Drizin, MC USN

Chylothorax is an unusual type of pleural effusion. In order to guide appropriate diagnostic testing, it is important to consider chylothorax as a potential etiology, with or without the typical milky appearance.

An otherwise healthy 73-year-old woman presented to the hospital reporting a two-week history of progressively worsening non-productive cough, dyspnea on exertion, and orthopnea. She also noted sinus congestion, sore throat, fatigue, abdominal bloating, and poor appetite. She underwent recent chiropractic cervical spine manipulation, but denied recent trauma or surgical procedures. She also denied any fevers, night sweats, abdominal pain, nausea, vomiting, weight loss, tuberculosis exposure, or history of tobacco use. Physical exam was remarkable for a respiratory rate of 23 breaths per minute, decreased right-sided breath sounds, a mildly distended abdomen, and the absence of lymphadenopathy or peripheral edema. Chest x-ray and computed axial tomography scan showed large right-sided and small left-sided pleural effusions and a possible renal mass. Thoracentesis revealed over 1600 milliliters of white, milky pleural fluid with a triglyceride level of 1,601 mg/dL. The fluid was exudative, and gram stain and culture were negative. Positron emission tomography scan showed multiple areas of soft tissue thickening and hypermetabolic activity involving the esophagus, right retroperitoneal renal fossa, small bowel mesentery, and lymph nodes. Pathological review of an excised left inguinal lymph node confirmed follicular lymphoma.

Chylous pleural effusions can be classified as traumatic or nontraumatic. As illustrated in this case, the most common etiology of a nontraumatic chylous pleural effusion is malignancy. The milky appearance of a pleural effusion can alert clinicians to the possibility of chylothorax; however, less than 50% of chylous effusions are milky, and 12% are either serous or serosanguinous, which may cause the chylous nature of pleural fluid to go unrecognized [1]. Therefore, in the absence of the typical milky appearance, clinicians should remain alert to the possibility of chylothorax, as its confirmation can guide necessary diagnostic testing.

[1] Doer CH, Allen MS, Nichols FC, Ryu JH. Etiology of Chylothorax in 203 Patients. *Mayo Clin Proc.* 2005;80(7):867-870.

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POEMS SYNDROME: A DEBILITATING SYSTEMIC DISEASE AND A ROAD LESS TRAVELED BY INTERNISTS

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Polyneuropathy, organomegaly, endocrinopathy, monoclonal gammopathy, and skin changes (POEMS) syndrome is a rare systemic disease often unrecognized in the primary care setting. Its etiology is unknown but cytokines such as Vascular Endothelial Growth Factor (VEGF) are implicated. Other paraneoplastic features include edema, papilledema, effusions, ascites, and pulmonary hypertension. The wide constellation of presenting symptoms may lead to misdiagnosis and is often confused with Chronic Inflammatory Demyelinating Polyneuropathy (CIDP). Untreated disease rapidly leads to death from neuropathic exhaustion or cardiopulmonary failure. Median survival from Mayo Clinic series of patients treated without peripheral stem cell transplantation was 13.8 years. Standard of care therapy is not established. Treatment is extrapolated from multiple myeloma regimens involving corticosteroids and alkylators such as melphalan and cyclophosphamide. Use of immunomodulatory therapy with anti-angiogenic (anti-VEGF) drugs such as lenalidomide and bortezomib are experimental. Here, we describe a patient with delayed diagnosis of POEMS syndrome who had remarkable response to lenalidomide-based therapy.

This 33-year old female was admitted for expedited evaluation of worsening bilateral lower extremity polyneuropathy with bilateral drop foot, dyspnea, and extreme fatigue for over one year. Multiple outpatient evaluations led to diagnosis of CIDP with plans for IVIG therapy. On admission, she appeared ill, cachectic, and had anasarca, skin hyperpigmentation and a steppage gait. Body CT scan showed hepatosplenomegaly, ascites, and pleural effusions without sclerotic bone lesions. Brain MRI brain revealed papilledema. Significant laboratory findings included anemia, thrombocytosis, and elevated creatinine, VEGF, prolactin, pro-BNP, and IgA levels. She had elevated kappa and lambda free light chains with a normal ratio and M spike of 0.5gm. Bone marrow biopsy showed 15-20% lambda-restricted plasma cells, and negative Congo red stain for amyloidosis confirmed with fat pad biopsy. POEMS syndrome was diagnosed based on presence of polyneuropathy, IgA lambda gammopathy, organomegaly, extravascular overload, papilledema, skin changes and elevated VEGF and prolactin levels. Therapy was initiated with cyclophosphamide and prednisone (60mg/m<sup>2</sup>) daily for five days in addition to lenalidomide (25mg daily) for every 21 of 28 days with plans for six total treatment cycles prior to autologous bone marrow transplant. IVIG was added to potentially enhance foot drop and gait recovery. Except for 25% residual neuropathy, all other previous abnormalities, including papilledema, resolved within four weeks of starting treatment.

While diagnostic criteria for POEMS exist, there is currently no therapeutic gold standard. Novel approaches that incorporate lenalidomide can result in rapid resolution of symptoms with minimal toxicity. Internists are most likely to first encounter patients with POEMS syndrome and this diagnosis should be considered in individuals with progressive peripheral neuropathy.

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SYSTEMIC LEAD TOXICITY SECONDARY TO EXTRA-ARTICULAR RETAINED SHRAPNEL PRESENTING WITH JAUNDICE AND HEPATITIS.

Ian Grasso, MD; Matthew Blattner, MD; Tyler Short, BS and John Downs, MD

Introduction: Despite greater than 60,000 non-fatal firearm injuries per year in the United States, retained shrapnel is a relatively rare cause of systemic lead toxicity with less than 100 cases reported in the medical literature since 1867. While intra-articular retained shrapnel as a cause of lead toxicity is well-described, extra-articular fragments are less well known to cause symptomatic disease.

Case Report: A 31 year old man initially presented with abdominal pain, constipation, jaundice and elevated liver transaminases approximately 3 weeks after suffering a left lower extremity injury during athletic activity. The patient was found to have steatohepatitis after extensive inpatient and outpatient gastroenterological workup to include upper and lower endoscopy, liver ultrasound, and biopsy of the liver to confirm the diagnosis. Imaging was incidentally notable for retained gunshot in the left flank and large shell fragment containing seroma in the left thigh. The patient was initially discharged with improved pain, but later presented to a primary care clinic with weight loss and continued pain. This was followed by a subsequent progression to diffuse weakness, ultimately resulting in an inability to ambulate. The patient was re-admitted to a tertiary care medical center, 3 months after the initial presentation. Physical exam was then notable for 70 lb. weight loss from initial admission and diffuse peripheral weakness with global muscle atrophy. Following a broad differential workup, he was found to have a blood lead level of 129 µg/dL, and Hgb of 7.7 g/dL with basophilic stippling on peripheral smear. The patient was transferred to the ICU for chelation therapy with dimercaperol and calcium EDTA. Lead levels initially decreased, but rose when patient was transitioned to oral therapy with succimer. Surgery was consulted for removal of multiple retained fragments, which were analyzed by the Joint Pathology Center and found to contain lead. The patient's motor function gradually improved on oral chelation and he was discharged to a sub-acute rehabilitation facility.

Conclusion: This complex case describes a rare cause for a relatively common clinical presentation, jaundice and hepatitis, and reinforces the importance of longitudinal follow up and reassessment of a patient with an unknown illness and worsening clinical condition. Diagnosis of systemic lead toxicity is challenging due to its protean clinical manifestations, and relative rarity with the advent of strict environmental lead controls and decrease in lead based paint and industrial products. Furthermore, extra-articular lead remains a rare cause of systemic toxicity, and the surgical standard of care has been to not remove these fragments in gunshot victims. This case adds to a small amount of evidence that lead screening may be of value in selected patients with extra-articular retained shrapnel, especially those with seroma and osteophyte formation in the wound.

## EHRlichIA-INDUCED HEMOPHAGOCYTIC LYMPHOHISTIOCYTOSIS CAUSING MULTI-SYSTEM ORGAN DYSFUNCTION

First Author: LT Alison B. Lane, MC USN

Hemophagocytic lymphohistiocytosis (HLH) is a life-threatening disease caused by immune overactivation and severe systemic inflammation. The syndrome can be genetic or acquired, with infectious, malignant, or rheumatologic conditions as potential underlying etiologies. Recognition of HLH, particularly when associated with infection, can be complicated by both its rarity and similarities in presentation to severe sepsis. We describe a case of HLH resulting from *Ehrlichia chaffeensis*, which is a rarely associated precipitant.

A seventy-year-old woman presented to the emergency department in late summer with six days of fevers and altered mental status. She reported a nonproductive cough and neck pain but was otherwise without focal complaints. The patient was febrile and tachycardic but normotensive. She was mildly confused with dry oral mucosa and faint left basilar crackles and an otherwise normal exam. Initial labs were significant for leukopenia to  $1800/\text{mm}^3$  with left shift, anemia, and platelets of  $11,000/\text{mm}^3$ ; acute kidney injury; and elevated transaminase levels. Chest radiograph and CT head were unremarkable. The next day, empiric doxycycline was started following an increase in serum transaminase levels and development of petechiae on her back, abdomen, and oral mucosa despite improvement in her platelet count to  $40,000/\text{mm}^3$  following a transfusion. She developed intermittent tachypnea and an increasing oxygen requirement resulting in transfer to the intensive care unit on hospital day three. Her pancytopenia worsened, requiring continued transfusions, and her renal function declined to the point of requiring renal replacement therapy. The infectious workup including serologies for tick-borne and viral illnesses remained unrevealing, and initial interpretation of a bone marrow biopsy indicated no malignancy. Ferritin was found to be  $>10,000\text{ng/mL}$ . On day four, she developed acute respiratory distress syndrome necessitating intubation and required hemodynamic support with vasopressors. A PCR for *Ehrlichia chaffeensis* returned positive. Three days later, hematopathology review of her bone marrow at a reference laboratory revealed hemophagocytosis suggestive of HLH as well as leukocyte inclusions consistent with *Ehrlichia morulae* on peripheral smear. She was started on high-dose dexamethasone and transferred to another hospital for potential initiation of etoposide per the HLH-2004 protocol. She ultimately improved with steroids alone, and after a month long hospital course was discharged.

HLH is a rare condition with variable presentation often leading to delays in diagnosis. While diagnostic criteria have been established (including fevers, splenomegaly, transaminitis, pancytopenia, hypertriglyceridemia, and hyperferritinemia), the overall presentation of severe systemic inflammation often with multi-system organ failure can overlap significantly with septic shock. Infection-triggered HLH should be considered when clinical deterioration progresses despite appropriate antibiotic treatment, even when HLH diagnostic criteria are only partially met. Treatment of secondary HLH involves suppression of life-threatening inflammation and immune overactivation with high-dose steroids and etoposide, in addition to treatment of the inciting cause.

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EX-SPLEENING A RARE DIAGNOSIS: DISSEMINATED ASEPTIC ABSCESSSES AS A PRESENTATION OF  
ULCERATIVE COLITIS

First Author: Patrick Moon, MD, Member Second Author: Jason Blaylock, MD, FACP Third Author: Nieka Harris, MD, FACP Last (senior) Author: Joseph Zeman, MD, FACP

**Background:** Aseptic splenic abscesses are a rare complication of inflammatory bowel disease (IBD). We describe a case of a 19-year-old woman with sterile splenic and intraabdominal abscesses with bilateral pulmonary lesions mimicking septic emboli as her initial presentation of ulcerative colitis.

**Case Presentation:** A 19-year-old woman with a reported history of intermittent painful bloody diarrhea presented to the emergency room with severe right upper quadrant abdominal pain. Her initial physical exam was notable only for tachycardia and a neutrophilic predominant leukocytosis of  $13,100/\text{mm}^3$  in addition to right upper quadrant abdominal pain radiating to her back. Computed tomography (CT) scan revealed a porta hepatitis abscess, prominent intraabdominal lymph nodes, and multiple bilateral pulmonary lesions concerning for septic emboli. Initial blood cultures were negative. She was started on empiric vancomycin and meropenem, but later broadened to linezolid, meropenem, and micafungin after failing to improve. Transesophageal echocardiogram, bronchoalveolar lavage, immunologic and rheumatologic workup were negative. CT-guided drainage of the porta hepatitis abscess was performed, but fluid cultures and cytology were negative for infection and malignancy.

Despite CT-guided drainage, she developed profound hypotension, hypoxia, daily fevers to  $104^{\circ}\text{F}$ , and new left upper quadrant abdominal pain with an erythrocyte sedimentation rate (ESR) of 86 mm/hr and C-reactive protein of 195 mg/L. Repeat imaging revealed enlargement of existing abscesses and multiple new splenic lesions. On hospital day 10 she received a splenectomy for continued progression of radiographic splenic abscesses. Pathologic exam revealed multiple abscesses with neutrophilic infiltrates but negative for any organisms. Following splenectomy she rapidly recovered and was discharged with four weeks of antibiotics. Two months after discharge she developed daily bloody diarrhea with tenesmus. Lower endoscopy and biopsy findings confirmed a diagnosis of ulcerative colitis.

**Discussion:** Aseptic abscesses are an exceptionally rare, but documented complication of IBD. Presenting symptoms include fevers (90%), abdominal pain (67%) and weight loss (50%), often for months without diagnosis. Aseptic abscesses are sterile neutrophilic predominant lesions, most commonly found in the spleen (93%) and abdominal lymph nodes (48%), but other organs may also be involved. Previous studies have shown transient improvement along with relapses after splenectomy in 48% of cases, but the vast majority of cases showed dramatic improvement in symptoms and resolution of abscesses with corticosteroid therapy alone. In conclusion, aseptic abscesses particularly in the spleen are extremely rare and should prompt an evaluation for early inflammatory bowel disease in the otherwise healthy young adult.

## LEPTOSPIROSIS: A DANGEROUS DIAGNOSIS TO MISS

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Leptospirosis is a bacterial infection by spirochetes of the *Leptospira* genus, transmitted commonly by the brown rat. While historically rare in the continental United States, leptospirosis is the most widespread zoonosis globally and represents a growing concern locally. *Leptospira* infections often present with nonspecific manifestations, so inaccurate diagnosis can lead to treatment delays and clinical deterioration. We present a diagnostically challenging case and discuss the key features leading to successful treatment of this emerging infection.

A previously healthy 35-year-old man presented with one week of fever, headache, myalgia, flank pain, and emesis. An avid outdoorsman, he reported a recent trip to Alabama two weeks prior to symptom onset, where he waded through a swamp during a period of heavy rainfall. Physical examination was remarkable for tachycardia, hypotension, fever (40°C), and an absence of jaundice. The patient was resuscitated with crystalloid and laboratory studies revealed a serum creatinine of 6.3 mg/dL, thrombocytopenia (63,000/ $\mu$ L), neutrophilia (90.7%), elevations of total and direct bilirubin (6.2 mg/dL and 3.8 mg/dL, respectively), and eosinophiluria.

The patient initially failed to improve on levofloxacin for presumed urinary tract infection. After ceftriaxone and doxycycline were started for empiric coverage of tick-borne illnesses and leptospirosis, the patient improved dramatically and was discharged home. Subsequently, a leptospirosis-specific assay returned positive for serum IgM.

Two decades ago, leptospirosis was of little concern in the continental U.S. However, increasing incidence led to this infection's listing as a Nationally Notifiable Condition in 2012. Transmission occurs by contact with rodent urine, so individuals engaging in outdoor activities are at higher risk. Epidemics have been reported after floods, when freshwater mobilizes *Leptospira* in the soil. After an incubation period of about three weeks, leptospirosis presents with three to seven days of fever, headache, myalgia, and emesis. If untreated, 40%-60% of patients develop acute interstitial nephritis. Ten percent progress to Weil Syndrome, a triad of kidney injury, jaundice, and pulmonary hemorrhage, associated with 10% mortality.

In this case, a young man presented with features of severe leptospirosis and responded clinically to presumptive therapy for this condition based on a high index of suspicion. The patient was treated prior to developing clinical deterioration or the jaundice and hemorrhage characteristic of Weil Syndrome. Notable in this case are the increasing incidence and evolving epidemiology of leptospirosis in this country and the importance of timely recognition of this infection in preventing significant morbidity and mortality. This patient's course illustrates the fundamental importance of a thorough history, a broad differential diagnosis, and swift action in patients with severe disease of unknown etiology.

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HHV-8-NEGATIVE, IDIOPATHIC MULTICENTRIC CASTLEMAN'S DISEASE: A RARE CASE OF FEVER OF UNKNOWN ORIGIN

First Author: Amanda Breviu, MD Second Author: Peter Hale, BS Third Author: Santosh Reddy, MD

Castleman's disease is a poorly understood lymphoproliferative disorder associated hypercytokinemia usually in the setting of HIV and HHV-8. While unicentric Castleman's disease is uncommon, the multicentric variant is extraordinarily rare, especially in the setting of HHV-8 negative serologies.

A 30-year-old male with type two diabetes mellitus and morbid obesity presented with one month of generalized weakness, fatigue and fever. This was his third hospitalization with these same symptoms; no definitive diagnosis was identified the previous two. He also complained of headache, a dry cough, and upper abdominal pain. Pertinent exam findings included abdominal tenderness to palpation in the upper quadrants with pain radiating to the back, without palpable organomegaly or lymphadenopathy. Chest x-ray, blood and urine cultures were negative. ESR and CRP were elevated at 123 and 28 respectively. CBC revealed a normal WBC count and normocytic anemia. CSF studies were pertinent for a mildly elevated WBC count. He was initially treated for aseptic meningitis thought to be secondary to NSAID usage. Of note, HIV and HHV-8 serologies were negative and IL-6 was elevated. When daily fevers persisted, CT imaging was performed and showed splenomegaly and multiple enlarged lymph nodes. PET CT imaging revealed diffuse uptake within the spleen and lymph nodes. FNA biopsies of periaortic lymph nodes were unremarkable. Lymph node excisional biopsy was consistent with a diagnosis of multicentric Castleman's disease. He was treated with rituximab weekly for four weeks as well as a prednisone taper. He subsequently had resolution of his fevers and normalization of his inflammatory markers and IL-6 levels. He later underwent radiation therapy to the abdominal region.

This case demonstrates a rare case of HHV-8 negative and HIV negative idiopathic multicentric Castleman's disease. While Castleman's has historically been associated with HHV-8 or HIV positivity, a growing number of cases, particularly of the multifocal type, have been identified which are negative for both viruses. Despite identification of this group, the pathophysiology and treatment options remain poorly understood. Interestingly, this patient still did have elevated IL-6 levels which point to a similar pathway of inflammation in absence of known viral illness. This case also highlights a diagnostic dilemma in a case of fever of unknown origin; particularly as his initial FNA was non-diagnostic and persistence was required to make his eventual diagnosis.

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## A CASE OF OGILVIE'S, OR NOT

First Author: Amanda Breviu, MD Second Author: Matthew Smith-Cohn, DO Third Author: Elwen Boud, BS Fourth Author: Ryan Murphy, MD

Ogilvie's and adynamic ileus are commonly managed diagnoses on inpatient medicine services. This case represents one where abdominal imaging led our initial diagnosis astray.

A 94-year-old man with no significant past medical history presented with a two week history of progressively worsening constipation accompanied by nausea, vomiting, and abdominal pain. He denied any hematochezia, melena, change in stool caliber or unintentional weight loss. He had never undergone a colonoscopy. He had tried stool softeners as well as a home enema without relief of symptoms.

On presentation, vitals were notable only for hypertension at 162/100. Exam was notable for mild distension and tenderness to palpation in all quadrants without guarding or rebound tenderness. Abdominal radiograph demonstrated diffuse distension of the small and large bowel with a dilated cecum consistent with Ogilvie's syndrome. CT of the abdomen and pelvis with IV contrast demonstrated normal small bowel with a large amount of stool throughout the colon without evidence of obstruction. He was admitted with working diagnosis of Ogilvie's syndrome and treated with an aggressive bowel regimen and avoidance of constipation-provoking medications. After a trial of supportive cares failed to relieve his symptoms, he underwent decompressive colonoscopy and was found to have near complete obstructing mass of the rectosigmoid colon. Pathology returned consistent with adenocarcinoma. Given his age and comorbidities, he underwent a diverting ileostomy as a palliative measure and was ultimately discharged to home.

In this case, abdominal CT results heavily swayed the initial differential towards Ogilvie's syndrome. Abdominal CT has high sensitivity and specificity in detecting an obstructing colon cancer; however, with this test, a clear obstruction was not identified. Retrospectively, this patient did not have any provoking factors that are often associated with Ogilvie's syndrome, such as surgical intervention, trauma, electrolyte abnormalities or opioid usage. He also had a high pre-test probability for colon cancer given his age and lack of prior screening colonoscopy. This case was somewhat misleading as the patient continued to pass flatus and did not have worsening symptoms of nausea/vomiting suggestive of obstruction; though he did develop worsening abdominal distension prior to final diagnosis.

In conclusion, abdominal CT with IV contrast has high sensitivity and specificity for identification of colon cancer, though should not be used to rule out a diagnosis of colon cancer when pre-test probability and clinical suspicion is high. In addition, the diagnosis of Ogilvie's syndrome should be made after exclusion of mechanical obstruction.

## HYPERVISCOSITY SYNDROME: A RARE CAUSE

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Hyperviscosity syndrome is defined as the clinical sequelae of increased blood thickness, affecting small blood vessels such as those supplying the retina and brain. Emergent recognition and treatment is mandatory to prevent long term neurologic sequelae. High blood viscosity can result from increased circulating immunoglobulins and is commonly associated with the blood cancer, Waldenstrom's Macroglobulinemia (WM).

A 63 year old Caucasian female with a long-standing history of chronic lymphocytic leukemia (CLL) presented to Hematology clinic with complaints of weakness, difficulty concentrating, blurry vision, dyspnea on exertion, and daily headaches for four months.

Her physical exam was notable for an oxygen saturation of 85% necessitating a new oxygen requirement, bilateral coarse breath sounds on lung auscultation, and sausage-shaped blood vessels on fundoscopic exam. Hyperviscosity syndrome was suspected. Serum viscosity level was 15.3 cPs (normal range 1.10-1.80 cP). Serum immunoglobulins revealed an IgM level of 5770 mg/dL (normal range 60-263 mg/dL) and an IgG of 2790 mg/dL (normal range 768-1632 mg/dL). Serum immunofixation showed both IgG kappa and IgM kappa monoclonal spikes.

She was admitted for emergent management of hyperviscosity syndrome and underwent plasmapheresis. To determine if the hyperviscosity was related to CLL or a new lymphoproliferative disorder, a bone marrow biopsy was performed. Pathology showed a small population of monoclonal CD5/CD19/CD23/CD20(dim) positive kappa expressing B-cells with phenotypic features consistent with her previously diagnosed CLL, as well as a new population of CD5/CD10/CD23 negative, CD19/CD20(bright) positive kappa expressing monoclonal B cells. Genetic studies made a secondary malignancy unlikely with negative tests for MYD88 (WM) and FISH for mutations commonly found in multiple myeloma. Therefore, a biclonal gammopathy from 2 separate CLL clones was diagnosed.

Given the similarity in the patient's presentation to WM, we elected to treat her with a regimen utilized in both WM and CLL. Ibrutinib is a B-cell receptor signaling pathway inhibitor that terminates survival signals to malignant B-cells. Ibrutinib has been FDA-approved for both WM and CLL. After 8 months of ibrutinib therapy, a repeat bone marrow biopsy showed no evidence of either CLL clone. Serum immunoglobulins were normal.

This case illustrates an uncommon presentation of one of the most common blood disorders, CLL. The incidence of monoclonal gammopathy in CLL has been cited as over 60%, but biclonal gammopathy in CLL is rare, limited to case reports. The incidence of a biclonal gammopathy in CLL causing hyperviscosity syndrome has not previously been documented. Rapid recognition of hyperviscosity syndrome and treatment of the underlying cause is critical to effective therapy and prevention of long-term neurologic sequelae.

## A CURIOSITY OF CALCIUM

Andrew Justice, MD

**Case Presentation:** The patient is a 72 year old male with a history of hypertension and numerous skin malignancies who presented with acute worsening of lethargy, fatigue, and intermittent confusion. Over the prior three months he noticed that he had become too tired to get out of bed and continue daily activities. Over the two weeks prior to presentation he didn't have the energy to get out of bed and walk around the house. He had difficulty with recent memories and often felt confused when talking to friends. He was unable to walk even 50 yards when needed. He had no dyspnea. He did have a 15 pound weight loss over 2 months and history of night sweats that were a chronic issue. He also noted deep muscle and bone pain in his legs and arms for the week prior to presentation. He had significant worsening of bone pain over his spine for the past month and worsening of his chronic back pain. He had noticed a decrease in bowel movements as well over the past 2 weeks.

**Physical Abnormalities:** The patient was fully oriented though diffusely weak. He had severe pain to deep palpation of his legs, arms, and lower back. He also had moderate tenderness to deep abdominal palpation with no palpable stool or masses. Neurological exam was otherwise non-focal with normal gait. The rest of the exam was non-contributory.

**Lab Results:** Calcium of 12.8, iCa of 1.75, PTH: 5, 1,25 Vit D 188.0, SPEP/UPEP: negative, PTHrP: negative. Beta 2microglobulin: 4.8, TSH 4.12, flow cytometry: negative

**Differential Diagnosis:** On admission differential included primary parathyroid disease, malignancy associated hypercalcemia, endocrinopathy, and granulomatous disease.

**Discussion:** This patient had an appropriately suppressed PTH with elevated calcium and concerning symptoms. His 1,25 Vit D level indicated an upregulation of 1-alpha hydroxylase activity. He had no symptoms of sarcoidosis, which is the primary etiology in this setting. He also did not have any risk factors for TB. Marrow biopsy revealed diffuse B cell lymphoma after a month of diagnostic workup that was stage IVB with diffuse retroperitoneal spread.

**Conclusion:** In patients with symptomatic hypercalcemia and suppressed PTH with significantly elevated 1,25 Vit D level an aggressive approach must be taken to look for lymphoma even without lymphadenopathy on exam or peripheral symptoms of disease. This process can be quite advanced without significant physical findings and only persistent hypercalcemia as the cardinal finding. Furthermore, in a patient with suppressed PTH Vitamin D levels, both 25 and 1,25 are important in outlining further workup. Calcitriol mediated hypercalcemia is a rare cause of hypercalcemia<sup>1,2</sup> but should be suspected with suppressed 25 Vit D and elevated 1,25 Vit D levels<sup>4</sup>. Sarcoidosis is seen in greater than 50% of cases of calcitriol mediated hypercalcemia<sup>1</sup>. If calcitriol mediated hypercalcemia is found an aggressive search should be undertaken to assess for sarcoidosis at first<sup>3,4</sup>. If this workup is negative flow cytometry, imaging, and bone marrow biopsy should be pursued unless there are overt signs and symptoms of fungal or mycobacterial infection<sup>4,5,6</sup>, which compromise 8% of calcitriol mediated hypercalcemia<sup>1</sup>. Symptoms of calcitriol mediated hypercalcemia etiologies often overlap<sup>1,3</sup>. Therefore, lymphoma should be a serious diagnostic consideration as it compromises 17% of calcitriol mediated hypercalcemia<sup>1</sup>.

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CHAPTER WINNING ABSTRACT

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ACETAMINOPHEN'S UNPLEASANT SURPRISE: A CASE OF ACETAMINOPHEN-INDUCED ERYTHEMA MULTIFORME MAJOR

First Author: Christian Bergman, MD Kathleen J. McEntee, MD

**Introduction:** Acetaminophen remains the most commonly used over-the-counter (OTC) analgesic and antipyretic agent since its approval in 1951. Although well-tolerated with minimal side effects, a review of the FDA Adverse Event Reporting System (FAERS) in 2013 prompted the FDA to release a statement warning physicians about the potential of acetaminophen induced Stevens-Johnson syndrome (SJS), toxic epidermal necrolysis (TEN), and acute generalized exanthematous pustulosis. We present a case of erythema multiforme major likely induced by acetaminophen in an effort to raise awareness of this OTC product's lesser known side effect.

**Case Description:** A 21 year old African-American male without a significant past medical history was transferred to our hospital over a concern for Stevens-Johnson syndrome. The rash started on his left arm six days prior to presentation and progressed to mouth ulcers with odynophagia two days prior to admission. On the day of admission, he developed ocular edema which prompted the emergency room visit. The patient denied any new medications, vaccines, environmental exposures, or recent illness. He did endorse taking two tablets of acetaminophen three days prior to the development of the rash for a headache. On physical exam, vital signs were stable and he had bilateral eyelid edema, oral mucosal erosions with significant swelling and hyperemia with upper and lower lip erosions. On skin exam, he had multiple (>twenty) plaques consisting of concentric zones of color with a dusky center (targetoid lesions) scattered over the chest, abdomen, back, and bilateral upper and lower extremities. Laboratory studies, including a complete blood count, basic metabolic panel, liver function panel, hepatitis panel, CMV, herpes, EBV, HIV were unremarkable except for a slightly elevated sedimentation rate (ESR) at 25 mm/hr and C-reactive protein (CRP) at 8.8 mg/L. Flexible laryngoscopy revealed a small vesicle on the left lingual surface near the epiglottis. Dermatology agreed with the diagnosis of erythema multiforme major in the absence of systemic symptoms, dusky red/purpuric macules consistent with SJS, or significant desquamation. The patient improved with high dose methylprednisolone 20 mg/kd/day for three days and topical supportive care. He was ultimately discharged on hospital day seven.

**Discussion:** Acetaminophen's dermatological side effects are not well documented in the general medical literature despite the FDA warning issued in 2013. We present a case of erythema multiforme major diagnosed clinically on the basis of a lack of systemic symptoms, typical targetoid lesions, and lack of desquamation. Acetaminophen has been reported to cause erythema multiforme and SJS/TEN even in cases of known prior exposure and is thought to be immune-mediated. Although acetaminophen is not a commonly-encountered causative agent of these serious dermatological conditions, it must be added to every clinician's differential diagnosis to reduce iatrogenic injury.

## NOT YOUR AVERAGE UTI

First Author: Meghana R Bhatta, MD Cathleen Kenary, BS Suzanne Chang, MD

Xanthogranulomatous pyelonephritis (XGP) is an uncommon variant of chronic pyelonephritis in which the renal parenchyma becomes severely inflamed and fibrotic. It is thought to be caused by recurrent urinary tract infections in the setting of longstanding urinary obstruction, most often by staghorn calculi. XGP almost always requires nephrectomy; rarely, mild cases can be managed with antibiotics and surgical debridement.

A 60 year-old obese female with history of diabetes, coronary artery disease, and recurrent urinary tract infections presented to the emergency department with two weeks of severe abdominal pain, confusion, weakness, and unintentional weight loss. Three months earlier, a left perirenal mass measuring 14 x 13 x 17 cm had been discovered on CT scan. Renal biopsy revealed collections of foamy macrophages with mixed inflammatory cells and fibroadipose tissue. Patient was subsequently lost to follow up.

On arrival to our institution, physical exam was notable for tachycardia, hypotension and a tender left-sided palpable abdominal mass. CT scan showed that the mass had grown to 15 x 16 x 23 cm, now with extensive local invasion and adherence to surrounding structures. The patient was treated with meropenem for presumed sepsis, and she underwent an emergent radical nephrectomy. Per the operative report, the kidney appeared grossly purulent with neoangiogenesis, and inflammation extended from the spleen to the pelvis. A large amount of retroperitoneal pus and necrotic tissue was debrided. Surgical pathology showed lipid-laden macrophages, foamy giant cells, and areas of suppuration and sclerosis throughout the mass, consistent with Xanthogranulomatous Pyelonephritis.

Post-operatively, the patient became profoundly anemic due to hemorrhage. She required three operations for re-packing the wound, drain placement, and closure. In the intensive care unit, she required intermittent pressor support and mechanical ventilation. After a month-long hospital stay, the patient was discharged to a rehabilitation facility.

Our patient was diagnosed with Xanthogranulomatous Pyelonephritis, an unusual complication of recurrent urinary tract infections which predominantly affects middle-aged females. This case demonstrates the importance of timely treatment and vigilance for urinary tract infections, a common occurrence in the primary care setting, with CT imaging and prompt biopsy. Delays in management as well as medical comorbidities contributed to our patient's difficult post-operative course.

In advanced forms, XGP may invade local tissues and form fistulas with nearby organs including the gastrointestinal tract. It is generally unilateral and often misdiagnosed as renal carcinoma. Definitive therapy of XGP is radical nephrectomy, with high rates of conversion to open procedure. The potentially devastating consequences of XGP, though rare, warrant close observation and aggressive treatment in patients with recurrent urinary tract infections.

## GOING GOO GOO OVER GABAPENTIN

First Author: Vinay M Gidwani, MD Zachary Nealy, Jody Boggs, Eastern Virginia Medical School, Norfolk, VA

Background: Gabapentin is a widely used drug for a variety of uses including as an antiepileptic, for chronic pain, psychiatric disorders, and alcohol dependence. It is eliminated primarily by renal excretion(1) and it has been regarded as "free of unwanted side effects"(2). Herein we present two cases of Gabapentin toxicity.

Case Presentation: Case 1: A 47 Year old African American female with chronic systolic heart failure, diabetes mellitus, hypertension, and chronic pain presented to our service with chest pain and shortness of breath. She was felt to be in acute heart failure and started on Bumetanide and subsequently Milrinone. Several days into her hospitalization she was found to be somnolent and with asterixis of her upper extremities. Her medications included Gabapentin 1200mg three times a day. Her serum creatinine was found to be 2.4 mg/dL, up from 0.9 mg/dL on admission. Venous ammonia was normal, blood urea nitrogen was 55 mg/dL, arterial blood gas revealed a pH of 7.34, carbon dioxide of 65.5 mmHg, and oxygen of 53 mmHg. Computed tomography of the head revealed no acute intracranial pathology. We discontinued her sedating medications including Gabapentin. Supportive care with noninvasive ventilation was begun in addition to intravenous fluid replacement. Subsequently, she seemed improved.

Case 2: A 27 year old male with cystic fibrosis was admitted to our service with abdominal pain, nausea, vomiting and diarrhea for several days. He was previously started on Gabapentin prior to admission. We increased his Gabapentin and he complained of what seemed to be myoclonic jerks. The Gabapentin was discontinued and the movements were found to have abated.

Discussion: While both were on multiple other drugs, an increased effective serum concentration of Gabapentin was suspected to be a culprit in both cases. In the former, with decreasing renal function, there was perhaps decreased clearance and in the latter there was introduction of a higher dose. A brief review of literature reveals 36 reported cases of myoclonus developing in patients treated with Gabapentin for seizures and epilepsy(3-14). Finally, in examination of 40 symptomatic cases only in 16 of these cases was gabapentin toxicity considered as the provisional diagnosis indicating perhaps an under recognition of the condition(15).

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ROYAL FLUSH - A CASE OF METASTATIC CARCINOID

First Author: Tharini Gunapalan, MD

Carcinoid tumors, although rare, have been increasingly more prominent over recent decades given the emphasis on screening colonoscopies. In patients who are discovered to have rectal or colonic carcinoid, simple resection is adequate given tumor size of less than 1cm and no evidence of lymphovascular invasion; and post-treatment surveillance is typically not recommended. Recurrence in this setting has seldom been reported. We present a rare case of metastatic carcinoid in a patient with previously resected rectal carcinoid tumor.

Case: A 53 year old male with history of rectal carcinoid status-post resection in 2009, HIV with a CD4 count of 600 and coronary artery disease presented with three weeks of nausea, vomiting, diarrhea and flushing. CT of the abdomen showed multiple lesions in the liver concerning for metastasis. Despite a normal 24 hour urine 5-HIAA, an elevated chromogranin A and subsequent liver biopsy confirmed the diagnosis of metastatic carcinoid tumor. Upon initial diagnosis of rectal carcinoid, he underwent lower anterior resection with biopsy indicating no lymphovascular invasion and a tumor size of 1cm with negative margins. He was followed with surveillance colonoscopies one and five years out with no evidence of re-occurrence. Octreotide scan was done after the diagnosis of metastatic carcinoid was made which did not indicate any evidence of carcinoid within the GI tract. His hospitalization was complicated with small bowel obstruction requiring exploratory laparotomy which unfortunately resulted in a delay of treatment with somatostatin. Several weeks later, somostatin therapy was initiated and has since shown a favorable response with >20% reduction in tumor burden.

Discussion: Although quite rare this case sheds light on the possibility of metastatic disease in stage I disease. Although this patient previously did not have invasive disease, post-treatment surveillance with colonoscopy was employed and was unable to discover recurrence. This case presents quite a rare phenomenon in which carcinoid tumor with negative margins and no evidence of invasion with negative surveillance went on to develop metastatic lesions to the liver, only then manifesting itself clinically as serotonin syndrome since hepatic clearance was bypassed. Although rare, this case may raise the question of imaging surveillance in patients with carcinoid in addition to screening colonoscopy.

## STROKE IN THE YOUNG: HOMOZYGOUS C677T METHYLENETETRAHYDROFOLATE REDUCTASE GENE POLYMORPHISM

Bradley Icard, DO

**INTRODUCTION:** Stroke less commonly occurs in patients < 55 years of age. This clinical scenario often spurs additional evaluation and a higher index of suspicion for a specific underlying abnormality. We present a case of ischemic stroke in a patient whose only main stroke risk factor was homozygosity for the C677T methylenetetrahydrofolate reductase (MTHFR) gene polymorphism.

**CASE:** A 24-year-old Caucasian female presented to the hospital after being found minimally responsive at home. The patient was a daily smoker, took no medications, and denied drug abuse. Physical examination revealed a benign cardiopulmonary examination, normal motor strength, confusion, aphasia, and visual agnosia. Initial computed tomography demonstrated left temporal parietal involvement. Lumbar puncture was unremarkable and magnetic resonance imaging was suggestive of a subacute evolving stroke. Laboratory examination revealed a complete blood count, complete metabolic panel and HIV screening which were normal or negative. In addition she had a vitamin B12 of 170 (pg/ml), folic acid of 4.97 (ng/ml) and markedly elevated homocysteine of 44.1 (umol/L). Echocardiography revealed a small patent foramen ovale. The patient was treated with aspirin, folic acid, vitamin B6, and vitamin B12 with substantial improvement. Gene testing revealed homozygosity for the C677T MTHFR gene polymorphism. Her homocysteine level decreased to 6.8 (umol/L) 50 days later and she made an excellent recovery.

**DISCUSSION:** The differential diagnosis of stroke in the young is vast. Many categories must be considered: cardioembolic, vasculopathies, drug-induced, hypercoagulable states, and hereditary diseases. A skilled neuro-radiologist may be helpful in differentiating between a stroke, other brain pathologies, and suggesting a possible cause. The majority of strokes between the ages of 15-49 are caused by cardioembolic and carotid vascular disease. Elevated homocysteine levels are linked to premature vascular disease in patients with homocystinuria. Elevated homocysteine levels are also linked to an increased risk of atherosclerosis, myocardial infarction, stroke, peripheral artery disease, venous thrombosis, dementia, neural tube defects and other pregnancy complications. The MTHFR enzyme converts 5,10-methylenetetrahydrofolate to 5-methyltetrahydrofolate in the metabolism of vitamin B12 and folic acid. The presence of the MTHFR C677T allele is considered a risk factor for stroke, and when combined with classic risk factors, exerts a synergistic action in the development of ischemic stroke in the young. We believe this patient's MTHFR gene polymorphism and resultant very elevated homocysteine levels played a pivotal role in the development of her ischemic stroke. The potential role of hyperhomocysteinemia should still be considered an important and treatable cause of stroke in the young, especially when there is paucity of traditional vascular risk factors.

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IDIOPATHIC GETS A NAME: A RARE PRESENTATION OF SARCOIDOSIS

First Author: Derick N Jenkins, MD Katie Bean, MD

Introduction: Sarcoidosis has been reported as a rare cause of recurrent pleural and pericardial effusions. It is extremely uncommon for patients to present with both simultaneously.

Case Description: A 46 year old African American man with two years of idiopathic, symptomatic, and recurrent pleural and pericardial effusions, asthma, and chronic pericarditis was admitted with one month history of progressive exertional dyspnea. He was a nonsmoker, and he had not traveled outside of the state. Family history was unremarkable. His heart rate was 102, respiratory rate 18, and oxygen saturation 97% on room air. Physical exam was significant for muffled heart sounds and clear breath sounds. Labs were unremarkable. Chest x-ray showed cardiomegaly and an increased right pleural effusion. Two years prior, he had a thoracentesis which revealed an exudative, lymphocytic effusion. At that time, pleuroscopy with pleural biopsy showed caseating granulomas and chronic pleuritis. Pleural fluid GMS (fungal) stains and fungal cultures, Histoplasmosis and Blastomycosis urinary antigens, Cryptococcal serum antigen, and Nocardia testing were negative. Three AFB sputum stains and cultures, Ziehl-Neelsen stains of his pleural fluid, and AFB pleural fluid cultures were negative. Adenosine deaminase of the pleural fluid was low. Despite the caseating granulomas, it was felt that tuberculosis was adequately ruled out.

On this presentation, TTE showed a moderate pericardial effusion with mild hemodynamic compromise. Cardiac MRI revealed a moderate to large circumferential, simple-appearing pericardial effusion with hemodynamic compromise (dilated IVC and biatrial/right ventricular partial collapse) and small left and moderate right-sided pleural effusions. Pericardial window was performed and fluid analysis revealed 889,888 RBC/mm<sup>3</sup> and 6,235 total nucleated cells/mm<sup>3</sup> with lymphocytic predominance. Pericardial biopsy revealed chronic pericarditis and non-caseating granulomas. RPR was negative. GMS and AFB stains were negative, fungal culture was final negative, and AFB culture is negative to date. Culture of the pleural fluid revealed only one colony of *Corynebacterium* which was a skin contaminant. Due to the presence of non-caseating granulomas on pericardial biopsy with negative fungal and AFB testing and an extensive negative workup for other causes of recurrent pleural and pericardial effusions, he was diagnosed with sarcoidosis. He had no history of ocular, dermatologic, gastrointestinal/hepatic, joint, neurologic, or cardiac involvement. He was started on prednisone with improvement of his symptoms.

Discussion: Sarcoidosis is a rare cause of recurrent pleural and pericardial effusions, and presenting with both concurrently is extremely uncommon. Nonetheless, sarcoidosis should be considered in patients presenting with idiopathic pleural and pericardial effusions, even in the absence of other systemic symptoms of the disease.

THE UNEXPECTED PATHOGEN: A CASE OF LISTERIA (WINNING CHAPTER PRESENTATION-ACP 2015)

Erica McBride, MD PGY-3 Omar Siddique, MD PGY-3 Suzanne Chang, MD

*Listeria monocytogenes* is a common cause of central nervous system infection in the elderly and immune compromised[1]. Though typically causing bacterial meningitis, *Listeria* can also result in encephalitis or rhombencephalitis, which manifest as focal neurologic deficits in addition to altered mentation[2]. We present a rare case of *Listeria* rhombencephalitis in an immune competent host.

A 66 y/o Female, with no significant medical history, presented to the Emergency Room with three days of worsening headaches, malaise, nausea and vomiting. She also complained of left facial paresthesias and gait ataxia after returning from a trip to Cartagena, Columbia. Physical exam revealed normal vital signs, decreased sensation along the right cranial nerve V distribution, as well as gait ataxia. Cerebrospinal fluid (CSF) analysis revealed a lymphocytic pleocytosis (WBC: 248; N: 23; L: 66; M:11; RBC:221), with normal protein and glucose. CSF cultures were negative. MRI revealed enhancement of the right 5<sup>th</sup> cranial nerve. The patient was initiated on IV acyclovir for presumed HSV encephalitis. She clinically improved, and was discharged home on oral valcyclovir.

The patient returned to the emergency room five days later with worsening nausea and vomiting. Though the headaches on the left side of her face improved, she had persistent left sided paresthesias and had now developed new right-sided headaches and paresthesias, new photophobia and blurry vision, as well as worsening ataxia. A repeat Lumbar Puncture (LP) still showed a predominant lymphocytosis with no growth on CSF cultures.

A second MRI showed new enhancement of the left cerebellomedullary junction and left CN V, as well as bilateral enhancement of CN VII. The patient's neurological condition rapidly worsened despite treatment with broad spectrum anti-virals, antibiotics and anti-fungals. She developed double vision, aphasia, right Horner's pupil and loss of her gag reflex and required intubation due to concern for brain stem paralysis. A third LP now showed a predominantly neutrophilic pleocytosis (WBC:629; N:377; L: 94). In the setting of persistently sterile cultures, an MRA was obtained and found to be negative for vasculitis, Neurosurgery was consulted for brain biopsy of the right thalamic and brain stem lesions, which grew *Listeria monocytogenes*. The patient was treated with a prolonged course of ampicillin and trimethoprim/sulfamethoxazole and regained most of her neurological function, however still suffers with deficits including bilateral ptosis and vertigo.

This case illustrates the importance of maintaining a broad differential and broad antibiotic coverage in cases of encephalitis with negative cultures and worsening clinical picture even in the immune competent patient. Practitioners should not rely on negative CSF cultures to rule out bacterial CNS infections when the patient is clinically deteriorating. Early *Listeria* encephalitis frequently does not grow in CSF and often presents with a lymphocytic pleocytosis before neutrophilic pleocytosis in cell count studies[3]. When clinical suspicion for CNS infection persists despite negative CSF cultures, brain biopsy should be considered.

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## TERRIFYING TETANUS TOXIN

First Author: Sarah E Morgan, MD

A 26 year old man presented with muscle rigidity and spasm since stepping on a nail 9 days prior. Four days later he noticed bilateral lower extremity pain and cramping which progressed to rigidity and spasm of muscles in his arms and back. For two days he had the feeling of a rubber band holding his jaw closed and had difficulty eating and speaking which prompted his trip to the emergency department. His last tetanus shot was 10 years prior. On initial exam his vitals were within normal limits except a heart rate of 114, he was in moderate distress due to pain with limited movement of his jaw and neck stiffness. He had rigid, tense lower extremities from toes to hips with toes in flexion and intermittent spasm of muscles of bilateral arms with contracted hands. Abdominal exam with intermittent tense muscle spasm. His left foot had a well-healed puncture wound with no erythema or swelling. Initial labs were benign with exception of an elevated CK to 606 units. He was admitted directly to the intensive care unit (ICU) and was given the Tetanus immunization as well as 3500 units of tetanus immunoglobulin in divided doses over multiple muscle groups. Orthopedic surgery was consulted, and he had surgical debridement of the left foot for source toxin control. Given his presentation, there was concern for progression and respiratory failure which would have necessitated intubation with likely conversion to tracheostomy and gastrostomy tube. This was recommended to the patient who declined elective intubation and reported he wanted to be treated without intubation understanding that his diaphragm could be paralyzed and he could die. He was started on IV dilaudid for pain control as well as IV versed, and flexeril to control his severe spasms. He remained in the ICU for one month with spasms up to 15 times per day during which his entire body would contract and he would require up to 12mg IV lorazepam and 8mg IV Dilaudid per spasm. In between spasms he worked with physical therapy and rehabilitation medicine to maintain his ability to walk. He required a nasogastric tube for feeding. Despite his negative predictive factors, almost 2 months to the date of admission, he was discharged to home, walking independently and requiring only oral benzodiazepam and narcotic taper.

Tetanus is increasingly rare in developed countries; therefore, there is relatively limited clinical trial data for appropriate treatment and management; consensus being that symptomatic support in an ICU with early aggressive nutrition, therapy and respiratory protection is key. Case fatality rate in developed countries in adults is ~13% with the tetanus toxin induced effects lasting 4-6 weeks because recovery requires growth of new axonal nerve terminals.

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PRE-SYNCOPE: A LOADED CHIEF COMPLAINT

First Author: Manoela P Mota, MD Hemrajani, Reena Bedoya, Pablo

A 59 year old woman presented with 1 week history of worsening dizziness, orthostatic symptoms and chest pain with ambulation, relieved by rest. Two months prior, she was admitted with non ST elevation myocardial infarction and pericardial effusion. Blood Pressure was 142/78 mmHg and 120/76mmHg 93 pulse standing. She had lungs clear to auscultation, no jugular venous distension. The point of maximal impulse was forceful and laterally displaced. A loud systolic crescendo-decrescendo ejection murmur worse in left sternal border was increased with Valsalva and decreased with standing. EKG showed bi-atrial enlargement, left ventricular hypertrophy. Cardiac enzymes were unremarkable. Transthoracic echocardiogram revealed severe concentric left ventricular hypertrophy with normal systolic function, small stable pericardial effusion, severe left ventricular outflow tract obstruction with a gradient of 80-110 mmHg and systolic anterior motion of mitral valve with moderate regurgitation. Cardiac MRI showed end diastolic septal thickness enlarged at 27mm, left ventricular hypertrophy asymmetrically involving the left ventricular septum, trace pericardial fluid, , ejection fraction 80% with a near complete obliteration of left ventricular cavity during systole. The patient's anti-hypertensives were discontinued and she was given intravenous saline and started on verapamil. Her blood pressure remained stable and she experienced improvement of the dizziness and pre-syncope and no further episodes of chest pain. The patient was referred for an alcohol septal ablation.

Discussion: Pre-syncope is a common presentation in outpatient and inpatient settings. However, a diagnosis of hypertrophic cardiomyopathy in an older adult with no past medical history of syncope or exercise intolerance is seldom anticipated. Hypertrophic cardiomyopathy's (HCM) prevalence is circa 1 in 500 adults and is the most common genetic cardiovascular condition and most common cause of sudden cardiac death in young adults. The patients have a dynamic left ventricular outflow tract (LVOT) obstruction and as the septum thickens and pressures soar within the left ventricular cavity, a drag force anteriorly displaces the mitral valve which worsens cardiac output. Patients with HCM are pre-load dependent and mild dehydration or increase in demand (exercise) can cause orthostatic symptoms and syncope. Patients are also at higher risk for cardiac arrhythmias and reversible myocardial ischemia as the thickened myocardial mass outgrows proper blood supply especially under higher demand.

Generally, patients will have a strong family history of sudden deaths or HCM. Upon further questioning, our patient remembered her sister has had syncopal episodes and was diagnosed with mitral valve prolapse, but the patient had no family history of HCM or sudden deaths.

Patients with severe LVOT obstruction are sensitive to decrease in pre-load as evidenced by this patient's worsening pre-syncopal episodes after being started on a diuretic for blood pressure control.

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DECIPHERING THE HALF MASKED DIAGNOSIS

First Author: Mit Patel, MD Second Author: Dr. Mitch Goodman, MD

Introduction: Peripheral facial nerve palsy has largely been attributed to viral or idiopathic etiology, which have been implicated in 75% of such cases. However, alternate etiologies must be considered based on a patient's history and physical exam findings in the setting of the larger clinical context. We report a case of isolated facial nerve palsy secondary to acute ischemic pontine infarct.

Case: A 58yo female with history of poorly controlled diabetes, HTN, CAD, previous TIA on aspirin/statin and poor medication compliance presented to the ED feeling dizzy and nauseated. She reported seven episodes of non-bloody/non-biliary emesis. On exam, she was noted to have a blood pressure of 227/99, and was found to have lagophthalmos and right facial hemiparesis, along with loss of right nasolabial fold and reduced forehead crease. She was given IV labetalol for her blood pressure with good response. CT head without contrast was negative for any acute process. She was later found to be lethargic and hypertensive, at which time she was moved to the ICU for blood pressure management and an MRI was ordered to evaluate the patient for AMS secondary to ischemic insult. She was found to have 2 acute ischemic insults in the brainstem – a 5mm infarct in the posterior lateral lower pons and a 6mm infarct in the left medial pons. Her mental status improved with blood pressure control and she was transitioned to a PO regimen. The patient was started on Plavix and statin and artificial tears and carboxymethylcellulose were prescribed for her lagophthalmos, which markedly improved upon discharge.

Discussion: Although this patient presented with acute symptoms and a presentation fairly typical of a peripheral seventh nerve palsy, her past medical history of hypertension, diabetes and previous TIA warranted a more thorough evaluation to explore the possibility of a central lesion. Involvement of both the upper and lower portion of the face are most often attributed to peripheral lesions, versus lower facial paresis which is usually due to a central lesion. Upon questioning, the patient revealed that she was intermittently compliant with her medication regimen, increasing the likelihood for a stroke. Subsequently, a thorough physical exam coupled with knowledge and understanding of neuroanatomy were instrumental in localizing the lesion and developing an alternative, although rare, diagnosis of isolated seventh nerve palsy secondary to pontine insult. Review of the literature reveals that only 1% of all isolated peripheral nerve palsies have been attributed to pontine lesions, and only two such case reports have been published. In conclusion, this case reminds us that the clinician should consider all potential diagnostic etiologies as it pertains to the the clinical context of the patient's history and physical exam findings.

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STATUS IATROGENICUS: WHEN SHOULD PSYCHIATRY BE INVOLVED IN THE DIAGNOSTIC EVALUATION OF DELIRIUM?

First Author: Daniel Rausa, LT, MC, USN Additional Authors: Gregory Condos, LT, MC, USN (Associate), Justin P. Lafreniere, LCDR, MC, USN (Member)

A 52 year-old female with systemic lupus erythematosus (SLE), and associated nephritis requiring hemodialysis presented to the emergency department after 5 days of altered mental status and episodic unresponsiveness. Her family noted that she was making inappropriate “dance-like” movements. The patient’s medication history included 20 years of lithium therapy, which the family reported was prescribed following an episode of lupus cerebritis. They stated that 3 weeks prior to the onset of symptoms, the patient had discontinued this medication out of concern that she was receiving a different brand from her pharmacy. At the time of admission, the patient was delirious with a Glasgow Coma Scale of 12 and CAM-ICU positive. She was exhibiting choreiform movements, but was otherwise without focal neurological deficits. She had an undetectable serum lithium level and a procalcitonin of 3.43. The remainder of her basic serum and urinary studies were unremarkable. CSF studies were normal. Non-contrast head CT, and CXR were unremarkable. She was admitted to the internal medicine service for further evaluation of her delirium. A psychiatric evaluation was not requested at that time.

Upon admission she was started on empiric antibiotics out of concern for an occult infection. On the third hospital day nursing found her unresponsive soon after her intern performed a routine morning exam. Based on a GCS of 3, she was endotracheally intubated, and transferred to the ICU. During her ICU stay, she experienced several complications, including a central venous catheter-associated DVT and a large right thigh hematoma secondary to therapeutic anticoagulation. Inability to wean her from ventilator support along with a failed extubation eventually required placement of a tracheostomy tube.

After consultation with psychiatry, neurology and rheumatology, her treatment team felt that lupus cerebritis was an unlikely etiology of her delirium and no source of infection was ever identified. The patient’s mental status returned to normal after lithium therapy was restarted.

This case demonstrates delirium following the cessation of a chronic psychotropic medication. The differential diagnosis of delirium is broad, but for this patient it included infection, lupus cerebritis, and mania. The patient’s history of lupus cerebritis, elevated procalcitonin at admission, and lithium therapy without a reported history of bipolar disorder all served to funnel this patient towards a medical rather than psychiatric evaluation. Her hospital course and medical interventions resulted in a cascade of iatrogenic complications. We use this case to illustrate the importance of early engagement with psychiatric clinicians in medically complex patients presenting with delirium. Doing so may facilitate early recognition of underlying psychiatric disease and prevent unnecessary medical interventions, shorten length of stay, and most importantly, improve patient outcomes.

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STAPHYLOCOCCUS LUGDUNENSIS, AN EMERGING CAUSE OF ENDOCARDITIS

First Author: Bryant Self, DO Second Author: Michael Wiid, MD, FACP

Coagulase negative staphylococci are common skin flora; as such they are often disregarded as contaminants and left untreated. *Staphylococcus Lugdunensis* (*S. Lugdunensis*) is a lesser known coagulase negative staphylococcus. It tends to have characteristics and virulence similar to that of *Staphylococcus Aureus*. In this regard, it can cause a rare but destructive form of infective endocarditis. We report a case of native mitral valve *S. Lugdunensis* endocarditis in an immunocompetent patient that is unrelated to implantation of a medical device or procedure.

A 68 year old female with a past medical history significant for hypertension, diabetes, breast cancer treated with mastectomy and radiation presented with a three day history of progressive non-productive cough, fever, chills, dyspnea and generalized weakness. She had no history of recent procedures, dental work, indwelling vascular catheters or intravenous drug abuse. On admission, she had a temperature of 99.6 degrees Fahrenheit, pulse of 106 beats per minute, blood pressure of 97/56 mmHg, respiratory rate of 35 breaths per minute and oxygen saturation of 92%. She required intubation, ventilation and vasopressor support. All blood cultures grew *S. Lugdunensis*. Transesophageal echocardiogram revealed a 3.2 cm x 1.0 cm multilobar vegetation on the mitral valve. Despite appropriate antibiotic therapy with Nafcillin and Rifampin she continued to experience a poor response to medical therapy. Cardiothoracic surgery was consulted for mitral valve replacement; conservative management was recommended with a grim prognosis in the setting of shock, renal failure, and prior chest wall radiation. Her mental status limited attempts at extubation, and a MRI revealed evidence of multiple brain septic emboli. On the day of her death, she was noted to have an acute arterial occlusion of her right femoral artery. Embolectomy was planned because of concern for septic embolization to the leg; unfortunately, she coded and died during induction for surgery.

In a case review published in 2008 only 83 cases of *S. Lugdunensis* endocarditis have been described since it was first identified in 1988. *S. Lugdunensis* is an unusually virulent coagulase-negative staphylococcus that is found in normal skin flora. It has typically been described following a medical procedure such as dialysis catheter placement, cardiac catheterization, and pacemaker implantation. *S. Lugdunensis* endocarditis tends to produce large vegetations which are prone to embolization and local valve destruction resulting in an increase incidence of heart failure. When compared with early surgical intervention, aggressive medical care alone results in a three times higher mortality rate. The authors would like to draw the reader's attention to this emerging, virulent coagulase negative staphylococcus which cannot be ignored as a contaminant in blood cultures.

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SEVERE VANCOMYCIN-INDUCED IMMUNE THROMBOCYTOPENIA

First Author: Clinton J Thurber, MD

A 67-year-old man was admitted to the hospital for acute cellulitis of the right lower extremity. He was treated with piperacillin/tazobactam and vancomycin over the first five days of his hospital course, after which his regimen was changed to intravenous cefazolin. He was discharged after nine days to finish therapy at home. Approximately thirty-six hours after discharge, he returned complaining of symptom recurrence. He was started again on vancomycin and piperacillin/tazobactam, and the following morning he complained of oozing from his affected leg. At that time his vital signs were unremarkable and his exam was significant only for warm erythema surrounding a clean-based 2-centimeter shin ulceration; there was no bruising or gingival bleeding. His labwork showed zero platelets per  $\mu\text{L}$  on two consecutive draws, after his platelet count had been 242,000 per  $\mu\text{L}$  the previous night. Microscopy did not visualize any platelets or clumping. Heparin flushes and enoxaparin were held; heparin-induced-thrombocytopenia antibodies and serotonin release assays were sent, returning later with negative results. A panel for disseminated intravascular coagulation was also unremarkable. The patient was transfused twelve units of platelets, after which his serum count showed only 2,000 per  $\mu\text{L}$ . At that point, vancomycin and piperacillin/tazobactam were replaced again by cefazolin, serum samples were sent for vancomycin- and piperacillin/tazobactam-induced anti-platelet antibodies, and the patient was transferred to the ICU for hourly stroke checks. He subsequently received a dose of intravenous immune globulin prior to transfusion of eighteen more units, a regimen which had to be repeated again the following day before he sustained a platelet count  $>40,000$  per  $\mu\text{L}$ . The patient was ultimately positive for vancomycin-induced anti-platelet antibodies. Vancomycin was added to his allergy list, and he was discharged home in stable condition on intravenous antibiotic therapy.

Discussion: Vancomycin-induced immune thrombocytopenia is most famously described in a 2007 New England Journal of Medicine case series by Drygalski et al. In her antibody-positive subjects, the median nadir of platelets after exposure was 10,000 per  $\mu\text{L}$ , a 93% decrease from baseline. While this drop is significant, none reached zero platelets, as in our present case, highlighting the importance of immediate therapy and hourly monitoring for hemorrhagic complications. Further, it is important to note how different etiologies of thrombocytopenia present. For example, in the present case it was beneficial to realize that heparin-induced thrombocytopenia produces a more gradual decline in platelet count along with a higher nadir. Thus, while we still discontinued heparin flushes and enoxaparin, we did not delay our search for the more likely offending agent. This ultimately resulted in a more rapid diagnosis and fewer total days of severe thrombocytopenia for this patient.

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AN UNUSUAL CASE OF PERIORBITAL RASH

First Author: Clinton J Thurber, MD

A 69-year-old Caucasian man presented to clinic with a rash around his eyes which had progressively worsened for three days. Two and three months prior to presentation, he underwent uncomplicated cataract surgeries on each eye, ultimately completing a course of prednisolone eye drops five days prior to presentation. Three days prior to presentation, he developed a pruritic rash around his eyes. He denied vision changes or ocular discharge. He denied new exposures, cosmetic products, or recent changes in medication aside from the aforementioned steroid drops. He went on a hunting excursion in the western Virginia mountains six days prior to presentation, and he had recent tinea cruris, untreated. He denied fever, headaches, sinus congestion, sore throat, lymphadenopathy, cough, shortness of breath, nausea, and joint pains. Physical exam revealed stable vital signs and a patchy 6x7cm erythematous, blanching, non-raised, slightly scaly area around the right eye with sharply-demarcated, irregular borders and skip areas covering both lids and onto patient's face. Around the left eye, the same rash covered a 2x4cm area. The globes were spared bilaterally and there was no ocular discharge. Cranial nerves II-XII were intact, visual acuity was unaffected, and the retinal exam was normal. The remainder of the exam was unremarkable except for large erythematous patches in the inguinal regions bilaterally. Periorbital skin scrapings were obtained and examined microscopically, revealing many hyphae. A 30-day course of oral terbinafine and topical ketoconazole for both the orbital and genital regions was prescribed. 12-day follow-up demonstrated significantly improved erythema, as well as complete resolution of pruritus and scaling. On 30-day follow-up, all symptoms had completely resolved.

Discussion: The close temporal proximity of the salient historical elements rendered it difficult to pinpoint the etiology of this patient's rash. The recent ophthalmological work, post-operative steroid eye drops, trek to the mountains, and tinea cruris were all potential clues to diagnosis. The exam findings appeared most like a contact dermatitis versus a fungal process; however, isolated fungal dermatitis in the periorbital region is quite rare. The most likely diagnoses included a hypersensitivity reaction to the eye drops versus spread of fungal elements from tinea cruris. Upon visualizing numerous hyphae on microscopy, it was determined that the patient likely had rubbed his eyes, translocating hyphae from his tinea cruris to the periorbital region. This area exhibited a degree of localized immunosuppression from topical prednisolone therapy, rendering it an especially suitable nidus for infection. The findings from this case highlight the importance of considering interplay between multiple historical elements in the workup of an unusual rash.

## TIMELY RECOGNITION OF TRACHEOBRONCHOMEGALY

Van Taunay, J.; Munoz, M.

A 50 year-old woman presented to our hospital with a recrudescence of hemoptysis. That day, she expectorated 500cc of bright red sputum during an all-too-familiar coughing fit. Her course in the previous six months was notable for a series of MRSA pneumonias, chest tube decompression, wedge resection, and tracheostomy (re-cannulated after 2 months); after which she tolerated room air, and participated in physical rehabilitation. She returned home, ambulating with a walker over short distances, limited by weakness.

Her medical history was notable for a 20 year cough productive of clear-white sputum. She denied history of asthma, inhaler use, or recurrent infections. Her allergies included fish and sertraline. She was a 20-pack-year active cigarette smoker. She was employed for 30 years as a commercial building cleaner. Her sister worked at a shipyard, and died at age 48 secondary to respiratory failure of unclear etiology.

Examination revealed a Caucasian female of slight habitus, with muscle wasting. Right upper lung field breath sounds were absent, but auscultation was otherwise clear. She had no expiratory stridor, digital clubbing, cutis laxa, or chondritic craniofacial changes.

Computed tomography (CT) imaging revealed tracheobronchomegaly, tracheal diverticuli with scalloping, and bronchiectasis. Compared with serial CT imaging from the past six months, her tracheobronchomegaly developed progressively. Her sagittal trachea, right, and left main bronchi respectively measured: 1.45cm/1.2cm/1.1cm initially, and 2.8cm/1.5cm/1.4cm six months later. Tracheal measurements were taken 2cm above the aortic arch. CFTR mutation panel, alpha-1-antitrypsin mutation, and rheumatoid-factor assay were within normal ranges.

Her hospital course was uncomplicated. Her hemoptysis did not recur. She was discharged to a rehabilitation facility.

While our patient's presentation exhibits hallmark findings of tracheobronchomegaly, several classic associations were not observed. Her absence of digital clubbing, lifetime history of recurrent infections, and cutis laxa is not uncommon in tracheobronchomegaly patients, suggesting that phenotypic expression of this disease may likely have multiple etiologies. This consideration is essential in obtaining a targeted patient history, which should guide a timely diagnostic approach for early disease recognition, and ultimately an etiology-specific intervention. Tracheobronchomegaly has historically been synonymous with Mounier-Kuhn Syndrome. MKS is considered an autosomal recessive disease, characterized by connective tissue atrophy resulting in tracheobronchomalacia and bronchiectasis. Radiographic characterization lacks consensus, with tracheal cross-sections ranging from 2.3cm-3cm as the lower limits for diagnosis. Our patient's radiographic findings, along her intriguing family history, suggest MKS. Further investigation should include pulmonary function testing, inspiratory-expiratory CT imaging, and histology for definitive diagnosis. If confirmed, our patient will benefit from targeted treatment and improved preoperative risk stratification. Furthermore, this case would offer a unique documentation of the progressive radiographic changes associated with MKS.

## PERHAPS HISTORY REPEATS ITSELF...AN OLD DIAGNOSIS REVISITED.

First Author: Nathaniel C Warner, MD

A 71 year-old gentleman presented to the hospital with two weeks of progressive, crampy, right-sided abdominal pain and voluminous, watery diarrhea without fevers or chills. Five days prior to presentation he went to an outside hospital emergency department and an abdominal CT scan revealed distal colitis. His past medical history included alcoholic cirrhosis status-post liver transplantation, bladder cancer, chronic kidney disease and mild dementia. Home medications included sirolimus, tacrolimus, ciprofloxacin, and donepezil. He occasionally drank alcohol and lived with his wife and children, who were all in good health. He denied any recent travel, sick contacts, camping, well water, animal, or foodborne exposures.

Pertinent physical exam revealed unremarkable vital signs, dry mucous membranes and a soft, nontender, nondistended abdomen. There was no rebound or guarding and no masses detected. There was no hepatosplenomegaly. Admission labs were remarkable for acute on chronic renal failure (Cr 4.34 mg/dL, baseline 2.5 mg/dL), hypokalemia, and severe leukocytosis with left shift (WBC  $23 \times 10^9/L$ ). Tacrolimus and sirolimus levels were not elevated.

Given his presentation, antibiotic-associated *Clostridium difficile* colitis was suspected and oral vancomycin was started empirically while awaiting toxin PCR and stool cultures. After three doses of vancomycin, the patient's *C. difficile* toxin assay returned negative and he was switched to ciprofloxacin and metronidazole to treat infectious colitis. Over the next two days, the patient's diarrhea moderately improved but his leukocytosis rose to  $33 \times 10^9/L$ . His stool culture returned positive with pure *Staphylococcus aureus* on day three and he was switched to oral doxycycline for a seven day course with improvement in his diarrhea and no complaints at his follow-up visit one week after discharge.

Discussion: Enterocolitis due to *Staphylococcus aureus* was first described in the 1940s as an outbreak in a maternity unit. In the 1950s and 60s, *S. aureus* was thought to be a common cause of antibiotic-associated enterocolitis (AAE). With the advent of increasingly sensitive *C. difficile* detection methods, *S. aureus* fell out of favor as a cause of AAE except occasionally in the Japanese literature. Today, many question whether AAE due to *S. aureus* is a true disease. Many institutions do not permit stool cultures in hospitalized patients, making even the possibility of diagnosis difficult. This patient, with radiographic evidence of colitis, systemic evidence of infection, negative *C. difficile* toxin assay, and stool culture with pure *S. aureus* seems compatible with a diagnosis of antibiotic-associated enterocolitis due to *Staphylococcus aureus*. Perhaps the diagnosis should be considered if AAE is suspected and highly sensitive *C. difficile* testing is negative.

PANCREATIC MYCOBACTERIUM TUBERCULOSIS IN A PATIENT WITH END-STAGE RENAL DISEASE

Sarah H. Chung MD, Jennifer R. Lyden MD

Introduction: Pancreatic tuberculosis can mimic malignancy and should be considered in the differential diagnosis for focal pancreatic lesions with weight loss, particularly in patients with end-stage renal disease (ESRD) who represent an immunocompromised population.

Case: A 62 year-old man with ESRD on hemodialysis and a known pancreatic mass presented to the emergency room with severe abdominal pain. A 2 x 2 centimeter cystic mass in the pancreatic head had first been discovered on CT imaging eight months prior, when he presented to an outside hospital with abdominal discomfort. At that time, endoscopic ultrasound (EUS) with fine-needle aspiration (FNA) revealed bland necrosis with minimal lymphocytes and no malignant cells. Findings were thought to be due to cystic degeneration of an intraductal papillary mucinous neoplasm, and radiologic surveillance was planned. However, he continued to experience intermittent epigastric pain over the next several months, with an acute worsening of the pain one week prior to presentation, associated with diaphoresis, altered sensorium, and rigors. On exam, the patient was febrile, hypotensive, and cachectic. Abdominal exam revealed epigastric tenderness without rebound, guarding, or palpable masses. Complete blood count, basic metabolic panel, liver enzymes, and lipase were normal. Repeat CT imaging of the abdomen revealed an interval increase in the size of the cystic mass with fat stranding and free fluid. He was treated with intravenous fluids and meropenem for septic shock thought secondary to bacterial superinfection or rupture of the pancreatic cyst. His septic shock resolved after three days, EUS with FNA was repeated, and biopsies were sent for bacterial and fungal cultures as well as Mycobacterium tuberculosis (MTB) and nontuberculous mycobacterium (NTM) DNA polymerase chain reaction (PCR). Pathology revealed degenerative debris with granulomas. MTB PCR returned positive. Acid-fast bacillus (AFB) culture of the pancreatic mass grew MTB after seventeen days. Pulmonary MTB was ruled out with three negative AFB sputum cultures, and the patient was started on four-drug therapy with rifampin, isoniazid, pyrazinamide, and ethambutol. His treatment course was complicated by several medication-related toxicities, and he was ultimately transitioned to isoniazid and cycloserine for a total treatment course of eighteen months.

Discussion: Patients with ESRD have defects in cell-mediated immunity and demonstrate a higher incidence of extra-pulmonary TB infection. Clinicians should recognize that these patients represent an immunocompromised population, warranting a broad differential for weight loss and sepsis. Secondly, pancreatic TB can mimic malignancy, and there are no distinguishing radiographic findings on CT, PET, or EUS. Tissue diagnosis remains the gold standard and should be sought in high-risk patients presenting with pancreatic mass and constitutional symptoms.

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RIGHT ATRIAL WALL VEGETATION MIMICKING MYXOMA

First Author: Anish Brahmhatt, MD

Infective Endocarditis is the infection of the endothelial surfaces of the heart, with echocardiogram being the diagnostic modality of choice. Vegetation found at an atypical location can mimic a cardiac tumor. We report a case of infective endocarditis with vegetation in a rare location and discuss its diagnostic challenges.

This case is a 33-year-old female with history of intravenous drug abuse who was admitted to an outlying facility with pleuritic chest pain, cough and hypotension. The patient was started on broad-spectrum antibiotics and treated for sepsis. Initial diagnostic testing revealed pneumonia and possible infective endocarditis; transthoracic echocardiogram revealed an abnormal density in the right atrium and the patient was transferred to CAMC for further workup. Transesophageal echocardiogram showed an abnormal density measuring 3.5 x 1.0cm at the junction of superior vena cava and the right atrium suspicious of a myxoma. The mass was eventually excised with presumptive diagnosis of a myxoma but the pathology came back as infective endocarditis showing presence of gram-positive cocci (*Staphylococcus Aureus*)

Infective endocarditis can present in an atypical location and sole reliance on echocardiography can sometimes lead to incorrect diagnosis and treatment. This case was unique that there have been only 2 published case reports where a vegetation was found around the junction of superior vena cava and right atrium. In two previous case reports, cardiac MRI has been shown to accurately diagnose these atypical presentations of infective endocarditis. The case highlights not only an atypical presentation for intracardiac vegetation, but also using cardiac MRI as an adjunct to transesophageal echocardiography in the evaluation of cardiac masses.

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SHAPIRO SYNDROME AS AN UNUSUAL CASE OF PAROXYSMAL HYPOTHERMIA

First Author: Joseph D Caveney, MD Additional author: Joel Yednock, MD, Assistant Professor

Shapiro Syndrome is a rare condition of spontaneous periodic hypothermia, corpus callosum agenesis and hyperhidrosis. The pathogenic mechanism is not fully understood, however corpus callosum agenesis is the hallmark and only fifty-two cases of Shapiro Syndrome have been documented in literature.

A 63 yo male presented to an outpatient clinic with shivering and shaking. These episodes used to happen once or twice a year, but now had become more frequent in the last thirty days and particularly over the past four days. He endorsed excessive sweating to the point that he had to change shirts three or four times daily. The patient was admitted to the hospital due to vital sign instability of BP 90/54 and a recorded body temperature of 89.8 F. Inpatient laboratory work-up was largely unremarkable including CBC, BMP, hepatic functional panel, thyroid, and cortisol levels all within normal limits. MRI brain performed and read as, "Agenesis of corpus callosum is essentially complete. Small focus of adipose signal tissue is identified in the midline, a frequent accompaniment to callosal agenesis. Ventricular distortion is diffuse and reflects the congenital agenesis of corpus callosum." Treatment consisted of aggressive fluid resuscitation, intermittent active external rewarming and initiation of cyproheptadine. The patient's home clonidine was held at admission due to hypotension, however restarted at time of discharge. Home oxybutynin was also continued at discharge.

The case illustrates an exceedingly rare cause of paroxysmal hypothermia known as Shapiro Syndrome. The patient demonstrated no evidence of either thyroid or adrenal insufficiency in the setting of complete agenesis of the corpus callosum. The hypothalamus is considered as the main 'thermostat' of body temperature. The anterior center controls heat dissipation by inducing vasodilation and sweating. The posterior center conserves heat by inducing vasoconstriction and shivering. It is currently unclear whether structural hypothalamic lesions are responsible for the hypothermia demonstrated in Shapiro syndrome. Some autopsies in Shapiro Syndrome have shown severe neuronal loss and fibrillary gliosis in hypothalamic nuclei. However, surgical resection of the corpus callosum has not been shown to cause episodic hyperhidrosis and hypothermia. The episodic nature of attacks leads support to a possible neurochemical abnormality rather than a structural lesion. Studies have found dysregulation of dopamine and norepinephrine in Shapiro Syndrome. One study showed hyperthermia in a case of 'reverse Shapiro's' resolved with administration of a dopamine agonist. No definitive treatment is known, however therapy with dopamine agonists, dopamine antagonists, serotonin antagonists and alpha2-adrenergic agonists have all been attempted.

## SARCOIDOSIS MASQUERADING AS FEVER OF UNKNOWN ORIGIN

Jenna Sizemore, MD Joann Hornsby, MD

Sarcoidosis can be difficult to diagnose due to wide variability in initial presentations. This case report of a patient with fever of unknown origin, myositis, and sudden onset peripheral edema found to have sarcoidosis illustrates the heterogeneity of symptoms associated with the disease.

29 year old Caucasian female presented to an outside hospital with acute onset low back pain, gait abnormality, pedal edema, urinary retention, fevers, and migratory arthralgias with leukocytosis and elevated creatinine kinase. Past medical history was significant for post-traumatic stress disorder, anorexia nervosa with severe malnutrition, intravenous drug use, benzodiazepine abuse, and bipolar disorder. Magnetic resonance imaging was inconclusive for infectious source, including abscess or myelitis. No further source of infection was elucidated. She was subsequently transferred to our facility with concern for an inflammatory myopathy. She developed fever spikes every 48 hours with onset of right foot drop and ophthalmodynia. Laboratory studies continued to show leukocytosis and elevated liver enzymes. Patient underwent extensive infectious work up, including repeat MRI of the lumbosacral spine that revealed non-specific edema within subcutaneous tissues. An extensive evaluation of the cerebral spinal fluid was negative. HIV and evaluation for common variable immunodeficiency were negative. The patient continued to have fevers every 48 hours despite broad-spectrum antimicrobial coverage. Patient underwent rheumatologic evaluation that showed negative rheumatoid factor, anti-citric citrullinated antibody, anti-nuclear antibody, and anti-Jo antibody. However, this evaluation revealed an elevated C-reactive protein (28.0 mg/L), serum angiotensin-converting enzyme (60 U/L), and continued elevation of creatinine kinase (2018 U/L). Computerized tomography of the chest showed pulmonary infiltrates in right upper, lower, and left lower lobes with unilateral hilar lymphadenopathy. Patient underwent treatment for possible aspiration pneumonia without improvement in cyclical fevers. Given lack of clinical improvement with elevated serum angiotensin-converting enzyme, pulmonary infiltrates and hilar lymphadenopathy, the patient underwent bronchoscopy for evaluation of sarcoidosis. A trans-bronchial biopsy showed focal non-necrotizing granulomatous inflammation consistent with sarcoidosis. A bone marrow biopsy was obtained that was negative for granulomatous infiltration. The non-specific tissue edema found on MRI was presumed to be secondary to granulomatous infiltration. High dose steroids were recommended for initial therapy, however, the patient refused due to previous history of steroid-induced psychosis with exacerbation of bipolar disorder. Second line treatment with methotrexate and azathioprine was deferred as patient continued to have elevated liver enzymes, likely from granulomatous infiltration of the liver. The patient was therefore transferred for initiation of anti-tumor necrosis factor therapy.

This case report showcases the variability of presenting symptoms of sarcoidosis in addition to the pragmatic evaluation of a differential for fever of unknown origin.

MYXEDEMA ASCITES: A RARE PRESENTATION OF PRIMARY HYPOTHYROIDISM

First Author: Marc Atzenhoefer, MD Second Author: Jeanette Man, BS Third Author: Ehab Saad, MD

Introduction: Ascites as a presenting symptom of primary hypothyroidism is rare occurring in <1% of cases. An uncommon complication of hypothyroidism, myxedema ascites is often diagnosed after lengthy workups. Treatment with levothyroxine is a simple and inexpensive solution that leads to complete resolution of true cases. This is the first documented presentation of myxedema ascites in a solid organ transplant recipient.

Case Report: A 61 year old male presented initially to primary care with complaints of generalized weakness, dyspnea, and weight gain of twenty three pounds over five months. The patient was referred to transplant nephrology given his complicated medical history of end stage renal disease secondary to type 1 diabetes mellitus status post kidney-pancreas transplant in 1991 now with chronic kidney disease stage III as well as Non-Hodgkin's B-cell lymphoma treated in 1998.

On arrival, physical examination was notable for anasarca, bulging flanks, abdominal fluid wave, and severe pitting edema. Initial laboratory studies revealed elevated creatinine and positive cardiac enzymes. Thus, he was admitted to rule out acute coronary syndrome. Further workup revealed albumin of 2.6 g/dL, TSH of 186 uIU/mL, and free T4 of 0.1 ng/dL. Chest x-ray showed bibasilar pleural effusions and serial electrocardiograms were remarkable only for low voltage QRS. Transthoracic echocardiogram was normal and diagnostic paracentesis demonstrated fluid that was low in protein with a serum-ascites albumin gradient (SAAG) of 1.5g/dL. Aggressive diuresis with albumin and furosemide was begun along with immediate initiation of Levothyroxine. Given his history of lymphoma, a CT of the chest/abdomen/pelvis was ordered which showed a mass in the left lower quadrant (LLQ) along with moderate ascites and pneumobilia.

Transplant surgery and hepatology were consulted. They recommended a complete abdominal ultrasound with doppler which revealed normal liver morphology/hemodynamics and a benign LLQ peri-pancreatic cyst. Complete hepatitis serology for HBV, HAV, and HBE were negative along with normal liver function tests, ceruloplasmin, anti-nuclear/mitochondrial/smooth muscle antibodies, and serum protein/immunoglobulins. The patient was discharged with close follow up and plans to recheck thyroid function in the near future.

Discussion: Primary hypothyroidism rarely presents with ascites. However, this clinical finding in a patient with a history suggestive of hypothyroidism should prompt thyroid testing. Diagnostic workup should include a paracentesis and determination of the SAAG. Literature review revealed sixty cases of myxedema ascites many of which had SAAGs of >1.1g/dL and a total protein of >2.5 g/dL. Since the pathophysiology of myxedema ascites is incompletely understood and so few cases have been documented, other causes of ascites should be considered and ruled out. Treatment with levothyroxine remains an effective solution with excellent prognosis.

## A COCAINE COMPONENT'S COMPLICATIONS

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Introduction: Levamisole is a frequent additive to cocaine. Due to rising levamisole concentrations in cocaine and cumulative exposure, the incidence of levamisole-induced vasculitis (LIV) is increasing.

Case: A 51-year-old male with a history of recent pneumonia and cocaine use (most recently 4 weeks prior) presented to the emergency department for shortness of breath. Exam demonstrated decreased left sided breath sounds with x-ray confirming left lower lobe consolidation and effusion. Labs revealed neutropenia and a negative urine drug screen. Neutropenia was attributed to use of cocaine contaminated with levamisole, due to a similar findings one year prior, and treated with filgrastim. By hospital day (HD) five, neutropenia and pleural effusion had nearly resolved.

On HD six, the patient developed painful, purpuric skin nodules with central darkening on his ankles progressive to his bilateral legs, upper extremities, and ears. Testing yielded negative results for blood cultures, hepatitis B and C, HIV, RPR, anti-nuclear antibodies, and rheumatoid factor. Septic emboli and cryoglobulinemia were thought to be less likely due to normal echocardiogram and negative cryoglobulins. Rheumatology recommended checking anti-nuclear cytoplasmic antibodies (ANCA), myeloperoxidase antibody (MPO), proteinase 3 (PR3) and anti-phospholipid antibodies (APA) - all of which resulted positive except for pANCA. Skin biopsy revealed leukocytoclastic vasculitis.

The patient's exam, labs, and biopsy were consistent with levamisole-induced vasculitis. He was started on prednisone, with significant improvement of skin findings within 24 hours. He was discharged on a steroid taper with cocaine cessation counseling.

Discussion: Levamisole was historically used as an anti-helminthic drug in veterinary medicine, and recently as an immune-modulator in the treatment of rheumatoid arthritis. Severe adverse effects including severe neutropenia, vasculitis, pulmonary, and renal dysfunction prompted withdrawal from medical use. It is now popularly used as a cocaine adulterant due both to its mood-enhancing properties as well as drug potentiation effect.

LIV most commonly begins as purpura, progressing to bullae then necrosis. The time of onset is usually within 24 hours of ingestion. Lesions commonly involve the lower extremities, however ear involvement is considered pathognomonic. Levamisole's short half-life makes lab detection difficult. Labs are typically positive for ANCA, MPO, and PR3 antibodies, with variable APA results. Biopsies can range from no evidence of vasculitis (48%), thrombotic vasculitis (36%), to leukocytoclastic vasculitis (16%). Positive myeloperoxidase (MPO) and proteinase 3 (PR3) antibodies differentiate LIV from cocaine-induced vasculitis. Besides cessation of levamisole exposure and supportive care, limited literature supports benefit from systemic corticosteroids only in patients with leukocytoclastic vasculitis. Given the rising incidence, consideration of this diagnosis is essential to initiate appropriate diagnostic testing and treatment.

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AN NOVEL CASE OF IDIOPATHIC PANCREATITIS AND ATYPICAL HEMOLYTIC UREMIC SYNDROME

**Jennifer** Jamison MD, Chad Glisch MD, Sara Koller MD

**Introduction:** Atypical hemolytic uremic syndrome (aHUS) is a complement mediated disease characterized by microangiopathic hemolytic anemia, thrombocytopenia and renal impairment. It is a rare condition and requires a high index of suspicion for definitive diagnosis. Treatment requires plasma infusions, apheresis and or immune modulating therapies.

**Case:** A 39 year-old female with history of recurrent thrombotic events on chronic enoxaparin was admitted with severe abdominal pain and elevated lipase. CT scan was consistent acute idiopathic pancreatitis. Hematology was consulted on hospital day 2 for thrombocytopenia with platelet count decreasing from 243 to 67. Creatinine on admission was 1.69. A diagnosis of HIT was excluded with a low pretest probability (4T score =2) and negative PF4-heparin antibody ELISA. Over the next 24 hours, the patient had a 4 gram drop in hemoglobin with a 13mg/dL associated rise in total bilirubin and LDH of 2402. Haptoglobin was undetectable, and direct antiglobulin test was negative. Creatinine continued to rise despite aggressive fluid resuscitation for pancreatitis. D-dimer was elevated with normal PT. Peripheral smear at the time showed less than 1 schistocyte per hpf.

Over the next day renal function worsened with persistent hemolysis and worsening thrombocytopenia. Platelet count reached a nadir of 18. ADAMTS13 activity at this time was normal. Peripheral smear confirmed thrombocytopenia and identified increased schistocytes. With worsening blood counts the patient was transferred to the ICU and plasmapheresis was started 2 days after admission for presumed TTP. Patient required 4 units of PRBC for ongoing hemolysis. aHUS diagnosis was considered as the patient had microangiopathic hemolytic anemia, thrombocytopenia, renal impairment with no history of diarrhea and normal ADAMTS13 activity.

The patient received a total of 6 days of plasmapheresis with normalization of platelet count on discharge, normalized hemolysis labs and improving renal function. aHUS panel was performed was remarkable for deficiency of MCP (Membrane-cofactor protein, or CD46), consistent with heterozygous mutation confirming aHUS diagnosis. Factor H, I, other complement markers for aHUS were normal. Further genetic studies revealed the patient has a novel genetic variant of MCP.

**Discussion:** Atypical HUS is a rare condition with patients predisposed to developing thrombotic microangiopathy due to mutations in one or more complement factors. aHUS can follow a relapsing remitting course with high morbidity and mortality in the acute phase. A few case reports of pancreatitis induced aHUS have been described in the literature. Additionally our patient has a unique genetic variant of MCP not previously described in the literature. The mutations in the complement system lead to dysregulation and activation of complement; however it is unclear at this time the exact mechanism that lead to pancreatitis induced aHUS in our patient. Appropriate treatment of aHUS requires interdisciplinary team collaboration and timely diagnosis and discussion regarding ongoing immune modulating therapy to prevent reoccurrences.

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A RARE CASE OF THROMBOCYTOPENIA- EPSTEIN SYNDROME

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Introduction: Epstein syndrome is one of the four clinical manifestations of a group of disorders known as myosin heavy chain 9 [MYH9]-related platelet disorders (MRPD). Here, we present challenges faced to arrive at MRPD diagnosis.

Case Presentation: A 36 year old female G1P0 was referred to the hematology clinic for persistent thrombocytopenia during pregnancy. Complete blood count (CBC) showed a platelet count of <10K. The patient was previously diagnosed with idiopathic thrombocytopenic purpura (ITP) at age 16 when she presented with meno-metorrhagia, thrombocytopenia and giant platelets on peripheral smear. She was treated with steroids and IVIG with no response and underwent splenectomy and rituximab infusions despite which the platelets remained low. During this whole time she was on oral contraceptive pills for menorrhagia and therapeutic platelet transfusion support. 2 years later she presented with renal failure, hematuria and proteinuria and underwent renal transplant. Around that time, patient noted blurred vision and difficulty hearing and was diagnosed with cataracts and sensorineural hearing loss respectively. The diagnosis of her thrombocytopenia was then attributed to a congenital defect related to Alport's syndrome due to the constellation of findings noted above. No pertinent family history on her mother's side but the patient was unaware of the family history on her father's side. On the basis of refractory macrothrombocytopenia and syndromic features (renal failure, hearing loss, cataracts), the patient was offered genetic testing. Gene sequence analysis revealed that she had a mutation of myosin heavy chain-9 (MYH9) consistent with Epstein's syndrome.

Discussion: This case illustrates the challenges encountered in arriving at a diagnosis of MRPD. Genetic syndromes should be an important differential in younger persons presenting with thrombocytopenia. Early recognition of this disorder is vital to avoid erroneous interventions due to incorrect diagnoses.

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RENAL FAILURE & RASH WITH EOSINOPHILIA: A CASE OF ACUTE INTERSTITIAL NEPHRITIS  
SECONDARY TO CEPHALEXIN

Authors: Quinn, B. MD; Koerner, K. MD FACP

Introduction: Acute Interstitial Nephritis (AIN) is characterized by reduced creatinine clearance and inflammatory changes of renal interstitium. Most often, the condition is precipitated by drug therapy but may also be associated with systemic autoimmune disease or infections. The vast majority of AIN results from treatment with beta-lactam antibiotics but other medications including non-steroidal anti-inflammatories, antimicrobial sulfonamides, diuretics, quinolones, H-2 blockers, proton-pump inhibitors, xanthine oxidase inhibitors, 5-aminosalicylates, and protease inhibitors have been implicated in this condition. Patients with drug-induced AIN typically present with a clinical triad of symptoms of fever, rash, and eosinophilia.

Case: A 68-year-old female presented with fevers, diffuse macular rash, oliguria, and generalized weakness following multiple antibiotic courses for treatment of combined lung and brain infections with microaerophilic streptococci species. Notably, she had initially been treated with penicillin and had developed marked eosinophilia and generalized rash. This was transitioned to vancomycin, which was discontinued after precipitating acute tubular necrosis (ATN). After this renal injury subsided, she was briefly treated with cephalexin for refractory lung abscess. Once again, she developed worsening renal failure and oliguria, now accompanied by signs of systemic allergy (fever, diffuse maculopapular desquamating rash, peripheral eosinophilia, and eosinophiluria). As AIN was strongly suspected based on the clinical picture, no renal biopsy was attained. Once the offending cephalosporin was removed and she completed a course of intravenous steroids followed by an oral steroid taper, renal function normalized. Dialysis was not required.

Discussion: AIN should be suspected in patients with elevated serum creatinine and urinalysis revealing pyuria, white cell casts, hematuria, and eosinophiluria; particularly in the setting of exposure to drugs known to cause AIN. Proteinuria is common but typically 1L UOP / 24hrs). Other case reports suggest by utilizing steroids for AIN, one can prevent the need for hemodialysis, which was the case for our patient.

## HETEROPHILE NEGATIVE EBV-ASSOCIATED MONONUCLEOSIS

Authors: Quinn, B. MD; Koerner, K. MD FACP

**Introduction:** Epstein Barr virus (EBV) is the primary etiologic agent responsible for infectious mononucleosis. Approximately 90-95% of adults are EBV-seropositive, with the majority of primary infections being subclinical. EBV is associated with the development of B cell lymphomas, T cell lymphomas, Hodgkin lymphoma, and nasopharyngeal carcinomas and reactivation can precipitate lymphoproliferative disorders in patients on immunosuppressive therapy. Infectious mononucleosis initially begins with malaise, headache, and low-grade fever followed by pharyngitis and cervical lymph node enlargement. Peripheral blood demonstrates lymphocytosis, with significant portion of atypical lymphocytes.

**Case:** A 19-year-old male presented with a 1-month history of intermittent fevers and sore throat. He was initially seen by his PCP, who noted exudative pharyngitis. He was prescribed a course of amoxicillin-clavulanate with marginal response. Later, he was re-evaluated and treated with an additional antibiotic course suboptimal response. Rapid strep test was negative at both visits. His symptoms persisted and investigatory labs were notable for reactive lymphocytosis on peripheral blood smear and a negative heterophile antibody test. Due to refractory symptoms and unclear etiology of complaints, he was evaluated by infectious disease consult. His fevers worsened and were accompanied by drenching night sweats. He was admitted for in-patient evaluation after being found to have progressive thrombocytopenia, transaminitis, elevated LDH and inflammatory markers including ferritin, and neutropenia with monocytosis and lymphocytosis. Manual blood smear was notable for reactive lymphocytes and spherocytes but no schistocytes to suggest hemolysis and direct antiglobulin testing showed complement. Presenting chest x-ray did not reveal any mediastinal mass and CT chest/abdomen/pelvis with contrast noted mild splenomegaly did not reveal any lymphadenopathy. CT neck with contrast demonstrated non-specific cervical lymphadenopathy. Flow immunophenotype panel showed no evidence of lymphoproliferative disorder so hematology consult opted not to attain bone marrow biopsy. Infectious labs were negative for infectious mononucleosis screen, HIV screen, tuberculosis quantiferon, acute hepatitis panel, EBV panel IgM/IgG, CMV NAAT, gonorrhea/chlamydia NAAT, group A strep NAAT, malaria smear, cat scratch antibody, babesia serology, toxoplasmosis serology, and extended viral respiratory NAAT. Prior to attaining lymph node biopsy of enlarged cervical nodes, EBV NAAT resulted with >17,000 copies so procedure was aborted. Patient was discharged in stable condition with supportive care for prolonged EBV infection and follow-up monitoring of liver function tests and EBV titer.

**Discussion:** Primary EBV infections are frequently asymptomatic but may manifest with fever, painful pharyngitis, and cervical lymphadenopathy. Laboratory studies typically show reactive lymphocytosis, commonly being atypical, and abnormal liver function tests. In most cases, serology is positive. Our case represented a serology negative infection with positive EBV DNA. In cases where clinical suspicion is high and serology is negative, one may consider EBV DNA versus serial EBV serology.

MYCOBACTERIUM BOVIS ASSOCIATED MYCOTIC GIANT CORONARY ARTERY ANEURYSM

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Background: Intravesical administration of live attenuated Mycobacterium bovis (M. bovis), Bacillus Calmette-Guerin (BCG), is an effective therapy for superficial bladder cancer with rare reported complications. As part of these rare complications, mycotic vascular aneurysms have been reported and have involved various arteries including the aorta, carotid, popliteal, iliac, femoral, and tibial arteries. Less than 30 cases of BCG-associated mycotic aneurysms have been reported, and therefore it is not frequently suspected on initial presentation and appropriate therapy is often delayed. To our knowledge, our patient represents one of the first case reports of a BCG-associated mycotic giant coronary aneurysm.

Case: A 63 year old male with hypertension, 50 pack-year history of smoking, and grade 1 transitional cell bladder cancer treated with 16 instillations of intravesical BCG immunotherapy (last dose 7 months prior to current presentation) initially presented with chief complaint of pain and swelling of the left posterior knee. He also endorsed a 6-12 month history of 80-pound weight loss and intermittent, mild fevers. On examination, there was severe swelling, pulsation, and tenderness to his posterior knee. He also appeared somewhat cachectic. Vitals, routine blood chemistry, and CBC were within normal limits. Given concern for vascular pathology, computed tomography angiogram (CTA) of abdomen and pelvis with lower extremity run-off was obtained. He subsequently underwent emergent repair of a ruptured left popliteal artery aneurysm (6.5 x 5.5 cm). No culture or pathology specimen was sent given lack of clinical suspicion for infectious etiology of the aneurysm. The CTA also identified multiple aneurysms in peripheral sites including bilateral iliac (1.7 cm each), bilateral femoral (L: 2.9 x 2.6 cm – R: 1.8 x 2.9 cm) and right popliteal (2.9 x 3.1 cm) arteries. No other intervention was performed, and he was discharged with 5 days of surgical repair with strict follow-up and physical rehabilitation arranged.

Approximately two months later, he presented again with similar complaints but to his right posterior knee. Further evaluation revealed that he had ruptured his right popliteal aneurysm which required emergent repair. Repeat CTA revealed mild expansion of other aneurysms prompting elective repair of his bilateral femoral aneurysms. At this point, an infectious etiology was suspected given history of severe weight loss and BCG immunotherapy. A biopsy of the femoral vasculature was sent for pathology and blood cultures were drawn. A total of three months had elapsed since initial left popliteal artery rupture when both blood cultures and tissue pathology were positive for M. bovis infection. Despite appropriate triple therapy (Isoniazid, Rifampin and Ethambutol), he exhibited failure to thrive and repeat rupture of his left popliteal aneurysm which prompted further surgical repair.

The patient had a prolonged hospital course for intravenous antimycobacterial therapy, rehabilitation, and nutritional optimization. Most of his prolonged stay was uneventful until he was noted to be hypotensive and with chest pain during rehabilitation one morning. CTA of his chest was obtained given concern for possible aortic aneurysm and this revealed a giant left anterior descending (LAD) coronary aneurysm measuring 6.5 x 5.9 cm.

Decision Making: Coronary angiography confirmed a mid-LAD giant coronary artery aneurysm (GCAA) with distal occlusion and collateral flow from the right coronary artery. While most GCAA's are treated surgically, his active infection, repeated aneurysmal ruptures and cachexia limited surgical candidacy. Interventional options considered were coiling, polytetrafluoroethylene (PTFE) covered stent deployment and thrombin injection. Coiling

would unfortunately not occlude distal small vessel blood supply to the aneurysm which would allow continued growth. Additionally, our cardiovascular interventionalists felt that the distal-LAD could not be accessed via endovascular approach so a PTFE covered stent placement was not feasible either. Lastly, the reflux of dye during angiographic injection suggested thrombin would disseminate to other arteries. Because of these conclusions, we chose to optimize surgical candidacy by adding amikacin and moxifloxacin along with intensifying nutritional support over the next several months. He has clinically improved with no further aneurysmal extension through 3 months of treatment. Surgical intervention is planned but not until completion of a prolonged 9-month antimycobacterial regimen (April, 2016).

**Conclusion:** In patients with a history of BCG treatment, mycotic aneurysms should be considered early in the right clinical context so aggressive treatment can be promptly implemented and surgical candidacy maximized.

