Economic impact of venous thromboembolism management during primary ovarian cancer therapy

Objectives: Venous thromboembolism (VTE) is the second leading cause of death in patients with solid malignancies. The association of VTE in the setting of epithelial ovarian cancer (EOC) is well established. Following primary surgery (PS) for EOC, current postoperative recommendations include 28 days of low molecular weight heparin (LMWH). However, the VTE risk continues to increase throughout the full episode of primary EOC care, with 44% of VTEs occurring beyond the prophylaxis timeframe. Yet there are no guidelines supporting prophylaxis after 28 days. To expand the understanding on the role of long-term VTE prophylaxis, we sought to determine the cost of care associated with the management of VTE for women treated for EOC.

Methods: Women who underwent PS for EOC between January 1, 2003 and July 1, 2014 and completed adjuvant chemotherapy at Mayo Clinic (Rochester, MN) comprised the study cohort. Inclusion criteria included a primary debulking surgery and a diagnosis of EOC. Patients were excluded if they had recurrent EOC or non-epithelial ovarian cancer or if they were treated with neoadjuvant chemotherapy (NAC) and interval debulking. Standardized direct medical costs were estimated from billed services captured by the Mayo Clinic Rochester Cost Data Warehouse. Adjusted mean costs were calculated for the 6-month timeframe following PS. The study was approved by the Mayo Clinic Institutional Review Board.

Results: 307 patients were identified in the cohort. The rate of VTE following PS was 7% (21/307) at 30 days and 13% (40/307) at 6 months. The adjusted 30-day mean cost of care for patients without VTE was $32,552 vs. $47,817 for patients with a VTE ($15,265 increase; p<0.01). The difference in cost of care increased to $21,435 (p<0.01) and $22,325 (p<0.01) at three months and six months following PS, respectively.

Conclusions: Following PS, a VTE diagnosis increases the 30-day cost of care by 47% or approximately $15,000 and progressively increases, along with VTE rate, over the six-month course of primary EOC.
Michael Michalik  
Dr. Michael Schnaus;  
Dr. Paula Skarda

**Leveraging Electronic Medical Record Order Display to Improve Quality and Costs**

Background: Electronic medical records have had a profound effect on the medical community over the past two decades, influencing almost every aspect of patient-related care. In many ways, the transition to medical records has had a positive impact on the quality of services offered in the hospital and in the clinics. In other ways, EMRs have introduced errors in clinical practice, and dependence on the technology offered through electronic medical records remain a concerning weakness of the system. During an unplanned and unannounced change that occurred within our healthcare network’s electronic medical record during a 23-day period in July 2016, the pre-selected and highlighted option within the list of orders when searching for a “complete blood count” (CBC) changed from CBC without differential, as had been the norm for years prior, to CBC with differential. The reason for the switch of this pre-selected order was a computer-generated change within our electronic medical record during a system update. This natural change within our electronic medical records system allowed us to review and provide commentary on the benefits and pitfalls of pre-selected orders.

Methods: In July of 2016, our healthcare system experienced a change in the pre-selected and highlighted option amongst a list of orders when providers across all hospitals and clinics searched to order a CBC from CBC without differential, to CBC with differential. We retrospectively reviewed ordering-behavior of CBCs before, during, and after this change.

Results: The total population of patients before, during, and after the EMR preference change was 13,242, 14,017, and 13,238 respectively. Overall, providers ordered CBCs with a differential over CBCs without a differential 37.7% of the time before the change, 50.0% of the time during the change period, and 39.8% of the time after the change period. Significantly more CBCs with differentials were ordered during the change period compared to the time periods before and after the change (p<0.01). Significantly more complete blood counts with differential were ordered in both the outpatient and inpatient setting during the change time period (p<0.01).

Discussion: Our retrospective study of the natural change of the pre-selected CBC order from “CBC without differential” to “CBC with differential” showed a clear impact on provider ordering-behavior in both the inpatient and outpatient setting. We hypothesize that the reason for this is due to different heuristics and biases, and propose that pre-selection of orders, while potentially harmful, can also be leveraged to achieve a higher quality of care at a lower cost.

Anna Najor

**Developing a Practical Methodology for Integration of Training on Health Care for LGBT Patients in Medical Schools**

Background: Health outcome disparities among LGBT patients are...
egregious, but what is perhaps more upsetting is that much of the harm suffered to these patients is due to negative experiences at the hands of healthcare providers. To address this issue, the AAMC has outlined extensive guidelines for medical school education on LGBT patient care. However, merely five hours on average is devoted to this topic in US and Canadian MD and DO schools. Recognizing an opportunity for improving education on caring for LGBT patients in the Mayo Clinic School of Medicine (MCSOM) curriculum, we instigated the integration of a comprehensive curriculum on LGBT health into the MCSOM curriculum.

Objectives: (1) Implement an LGBT health care curriculum at the MCSOM that builds on the strengths of other published curricula, addresses weaknesses; and then evaluate efficacy of the approach, and optimize the approach. (2) Develop practical guidelines for achieving educational goals, thereby reducing the barrier for similar change in other institutions.

Methods: A hierarchical list of core competencies for medical education on LGBT patient care was developed using AAMC guidelines and Mayo Clinic Transgender and Intersex Specialty Care Clinic (TISCC) consultant expertise. From this, curriculum elements were developed and implemented. Surveys are being given to medical students before and after each curriculum element to assess the effectiveness of each session in reaching competency outcome goals. This approach is being standardized and implemented in the Scottsdale, AZ MCSOM site.

Results: Based on AAMC guidelines and TISCC expert advice, the most promising training domains include interactive implicit bias training, training on health disparities, training on proper terminology to discuss issues of gender and sexuality with patients, and practice caring for patients who belong to gender and sexual minority groups. Data gathered from the class before and after each new curriculum element support this hypothesis, and elucidate important pitfalls in particular approaches.

Discussion: There are many labor-intensive methodologies to ensuring medical student competency in caring for the needs of gender and sexual minorities. Measured changes in medical student competency in caring for this patient population suggest that our approach will prove to be an attractive option for other medical schools wishing to meet the AAMC guidelines with minimal disruption of their curriculum.

Andrew Pattock  
Erik Faber;  
Audrey Jiang

Medical Student and Health System Partnership in Rapid-Cycle Readmission Improvement Pilot Project

The University of Minnesota Medical Center (UMMC) has a 30-day readmission rate (16.4%) slightly higher than the national average (15.3%). One tactic identified to improve this rate was to initiate post-discharge phone calls for all patients. To develop this process, we needed to better understand our patient population and their needs, especially those at high risk for readmission and high healthcare utilization. A student-designed method of data collection and analysis was used to gather information regarding patients discharged from the hospital medicine service to inform system priorities.

Students worked with system leaders to design and implement data collection procedures and collected data over a span of 14 days for all
patients discharged from select hospitalists on the medicine floor at the University of Minnesota Medical Center. Information collected included: patient demographic information, patterns of care engagement, care coordination efforts, and number of previous hospital admissions. Following review of the data with hospital administrators, a full-time RN was hired to call all patients within 72 hours of discharge to ensure continuity of care and avoid loss of reimbursement for excess readmissions. Additionally, other phone call follow-up procedures have been previously shown to decrease readmissions.

Data was again collected over 14 days after the implementation of the follow-up phone call protocol. About 160 patients were discharged over each 14 day period. After the RN was hired to make follow-up phone calls, the percent of patients with no follow up decreased from 40% to <5%.

There were several notable additional findings from data collection. The most common admission diagnoses were pneumonia, sepsis, and chronic disease complications. The median number of specialists seen by patients in the past 12 months was 3. About 50% of patients’ primary care providers were within the affiliated health system (Fairview/UMMC/M Health). Patients with high utilization were more likely to have a PCP listed and been seen by more specialists, but they received follow-up phone calls at the same rate as patients who did not have high utilization.

With the information collected, we were able to better understand our inpatient population to tailor the our discharge follow-up strategies to our patients’ needs. Our patients have complex diseases that need thorough follow-up, and many areas for improved care coordination were identified. Future work includes using data to help limit number of redundant follow-up calls, measuring readmission rates to determine efficacy of phone call intervention in the long-term, and tracking percent of discharged patients with high utilization.

Garrett Welle
Sarah Koepp, RN; Dr. Michael Rhodes; Kevin Shores, MA, MEd

Optimizing Appropriate Utilization of Cardiac Telemetry on an Adult General Medicine Unit

Background: M Health in collaboration with the University of Minnesota Medical School delivered a quality improvement solution to reduce the cost of healthcare spending based on the ‘Choosing Wisely’ campaign, an initiative of the American Board of Internal Medicine (ABIM) Foundation. Within their top recommendations was the need to address and avoid continuous telemetry monitoring in non-ICU patients without using a protocol that governs continuation. Optimizing the utilization of cardiac telemetry was based on revising current guidelines to incorporate the indications put forth by the American Heart Association and American College of Cardiology. These modifications were made in an effort to guide the appropriate ordering and discontinuation of telemetry. The project’s aim was to reduce the non-indicated use of telemetry on an adult medical unit by 50% by February 1, 2017.
Methods: A process flow chart was devised using Six Sigma principles to show the procedure for a telemetry patient from admission to discharge. To avoid scope creep, three areas of opportunity were identified and used to elucidate the scope. The in-scope items included: registered nurses (RNs) unaware of the reason for telemetry, no set discontinuation protocol, and providers and RNs unknowledgeable about monitoring criteria. The interventions addressed these problems by creating a hard-stop indication line within the electronic medical record (EMR), revision of guidelines to evidence-based telemetry criteria, and increasing collaborative communication about telemetry criteria. We opted to monitor and implement changes on an adult general medicine unit at the University of Minnesota Medical Center. To analyze the pre- and post-intervention data, surveys were administered on a four-point Likert scale to guide RN perceptions of telemetry, a 2-sample T-test was used to evaluate the length of time patients were on telemetry, and a one sample T-test was used to explore the proportion of patients on telemetry daily.

Results: We found there was an increase in RN awareness of the telemetry criteria and increased collaborative communication. Additionally, there was a significant decrease of 18.5 hours ($p = 0.009$) in average time on telemetry and a significant decrease of 2.9% ($p = 0.025$) in the average number of patients on telemetry daily. Finally, we surpassed the original aim of reducing non-indicated telemetry by 50% to reducing non-indicated telemetry by 100% ($n= 34$), as all patients had a documented indication for telemetry.

Conclusion: Through focused education, revision of guidelines for ordering and discontinuation with evidence-based criteria, and amendment of the EMR order set, we were able to optimize the use of cardiac telemetry and eliminate non-indicated use by 100%.

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<td><strong>Crystal Donelan</strong></td>
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<td>Dr. A. Duran; Dr. Bowman Peterson; Dr. C. Roth; Dr. P. Weissmann; Dr. S. Gladding</td>
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*Making a “Case” for High Value: Educating Residents on High Value Care Communication*

Health costs are soaring in the United States. Physicians evaluate the value of the tests and therapeutics they order for patients balanced against potential harms and costs. The ACGME requires that graduating residents demonstrate the ability to practice and advocate for cost-conscious care. A survey of 600 internists in 2014 found that 53% would order an unnecessary test if the patient was insistent; 58% felt that health care providers are in the best position to address this issue.

To address this need, we piloted a high value care (HVC) communication skills curriculum for internal medicine residents based on the Four Habits Model (ref) for communication (invest in the beginning, elicit the patient’s perspective, demonstrate empathy and invest in the end). This model offers practical elements for structuring challenging patient conversations, such as disagreements about ordering low value tests. We anticipated residents would value this training and demonstrate improved value-based communication skills.
Thirty first-year residents were randomized to a standard (a single HVC standardized patient (SP) encounter with immediate faculty feedback) or high intensity HVC communication skills curriculum (an in-person SP with immediate faculty feedback plus a recorded SP phone encounter that was debriefed with peers. Residents’ attitudes surrounding HVC and impressions of the intervention were assessed in a post-intervention survey; SPs assessed resident skills in a simulation following the year long intervention with a CARE empathy checklist (reference).

Twenty-one residents completed the post-intervention survey. The high intensity group (n=12) reported greater interest in similar curricula in the future (50% vs 22%), and rated the SP encounters as beneficial to their communication skills (83% vs 44%). They were more likely to report incorporating feedback from SPs (83% vs 44%) and faculty observers (75% vs 55%) into subsequent patient encounters. After a year of interventions, SPs evaluated residents with the CARE empathy checklist following face-to-face encounters. SPs rated the high intensity group higher in: making the patient feel at ease, letting patients tell their story, providing clear explanations, and helping take control of the patient encounter. There was no difference in overall performance between the two groups on the CARE empathy checklist (high intensity mean 27 vs 28 for standard).

Our intervention provided focused training on the communication of value-based decision making. Learners reported benefits in their communication skills and increased likelihood of incorporating skills into future patient encounters. In patient simulations they utilized more patient-centric methods. However, there was no difference in overall performance on an empathy checklist. Future work will focus on validation of a four-habits based behavioral check list, training of video raters, and review and scoring of a second video encounters to assess difference in skills in the high intensity versus the standard curriculum groups.

**Correlation of Exhaled Nasal Nitric Oxide to Sinus CT and Sinonasal Outcome Test Scores: A Prospective Study**

**Background:** Computed tomography (CT) of the paranasal sinuses is the gold standard for diagnosis of chronic rhinosinusitis (CRS). Detection of nasal nitric oxide (nNO) levels has been investigated as a diagnostic tool in sinus disease, as sinus obstruction results in a lower level of nNO.

**Objective:** The primary aim was to determine the correlation of passive (baseline) and dynamic (humming) nNO to CT findings of sinus inflammation and symptoms as measured by the Sinonasal Outcome Test (SNOT-26).

**Methods:** Fourteen subjects were recruited. Baseline and humming nNO levels were measured using a Siever chemiluminescence NO analyzer. Each subject completed the SNOT-26 survey. The CT was scored using the Lund-Mackay (LM) system (positive CT defined by LM score > 3). Correlation was measured by linear regression analysis (LRA) comparing SNOT-26, LM, and nNO measurements.
Results: LM scores had a positive pairwise correlation with total SNOT-26 scores ($R^2 = 0.1457$, corr: 0.3817) and nasal-specific SNOT-26 scores ($R^2 = 0.4036$, corr: -0.6353). Baseline nasal nNO scores had a negative pairwise correlation with LM scores ($R^2 = 0.1172$, corr: -0.3424), total SNOT-26 scores ($R^2 = 0.1515$, corr: -0.3893), as well as nasal-specific SNOT-26 scores ($R^2 = 0.0805$, corr: -0.4343).

Detected nNO levels at baseline validate total LM scores from CT imaging, as well as increasing SNOT scores with higher total LM scores. However, the nNO detected levels while humming did not demonstrate the same correlation.

Conclusions: Nasal NO may be a useful tool for screening for CRS as an inexpensive point-of-service screening test and for longitudinal monitoring of treatment response.

Jessica Piche
Andrea Peterson;
Dr. Jill Tyler

Doctor's in Theater: An Evaluation of a Medical Improvisation Course

Objective: To implement a medical improvisation course to improve medical students’ patient centered communication skills.

Method: Nineteen second year medical students at the University of South Dakota Sanford School of Medicine (USD SSOM) were enrolled in the study over the span of two years. Students completed a 10-hour medical improvisation course created by Katie Watson, J.D. at Northwestern University Feinberg School of Medicine. Pre- and post-course self-assessments, pre- and post-course standardized patient encounter evaluations, and a course evaluation survey were used to assess how the medical improvisation course affected medical students’ patient-centered communication skills.

Results: Students self-identified significant improvement in the category of spontaneity, communication etiquette, interpreting body language, conveying sincerity, empathy, teamwork, communication of medical knowledge, and multitasking following five 2-hour classes of medical improv. Anxiety was found to be unchanged. Standardized patient encounter evaluations found an average increase of two points following the conclusion of the course. This improvement in patient-centered communication was found to be significant.

Conclusions: Improvement in patient-centered communication skills was observed in students who enrolled in the medical improvisation course. Self-identified improvements were also perceived in the category of spontaneity, communication etiquette, interpreting body language, conveying sincerity, empathy, teamwork, communication of medical knowledge, and multitasking. With positive results following this course, we are investigating ways to further incorporate medical improv into the USD SSOM curriculum.

Thomas Schmidt
Dr. Jessica Fiege;
Dr. Ryan Langlois

Influenza in Phagocytic Cells: A Story of Direct Infection or Exogenous Uptake of Antigen

Influenza A virus (IAV), is an eight segmented, negative sense RNA virus. IAV is an extremely infectious virus that infects 9.2-35.6 million people in the US each year. Understanding the viral kinetics in the host
is critical to developing an effective vaccination strategy. Phagocytic cells such as macrophages and dendritic cells (DCs) are a vital link between the innate and adaptive immune systems, trafficking antigen from the site of infection to the draining lymph node and presenting antigen to T cells. While phagocytic cells have been reported to contain IAV antigen, it is unclear if phagocytic cells obtain IAV antigen either through direct infection or phagocytosis. We developed an IAV expressing Cre recombinase (IAV_Cre) to specifically label infected cells in a Cre-inducible tdTomato (iRED) reporter mouse. Cre recombinase selectively removes a loxP flanked stop cassette and allows for the subsequent transcription of the fluorescent reporter tdTomato. This genetic alteration is irreversible and indelible, allowing us to track any cell that has ever been infected. Previous studies have characterized tdTomato expression in epithelial cells in the lung after IAV infection, while immune cells have not been studied in this system. We sought to 1) identify what subsets of phagocytic cells are reporter+ at various time points after IAV_Cre infection, 2) determine if phagocytic cells are directly infected or take up exogenous tdTomato after IAV Cre infection of iRED mice. To determine the peak of tdTomato+ phagocytic cells, we harvested lungs, draining LNs, and spleens on days 3, 5, 7, 10, and 21 post infection (d.p.i.). We observed reporter+ phagocytic cells in the lung and draining lymph nodes at multiple time points after infection. Loss of reporter+ cells in the lung corresponded with a concordant gain of reporter+ phagocytic cells in the lung draining lymph nodes over the time course. To directly assess if phagocytic immune cells can obtain tdTomato via phagocytosis, we used a bone marrow derived macrophage culture system. We were able to demonstrate that macrophages can successfully phagocytose tdTomato from IAV_Cre infected iRED fibroblasts. Using the bone marrow chimera mice, we were able to demonstrate macrophages and dendritic cells can phagocytose tdTomato in vivo. These data demonstrate that phagocytic immune cells can phagocytose IAV antigen and traffic to secondary lymphoid organs. These results have implications to IAV vaccination demonstrating that subunit vaccinations are a viable option for priming adaptive immune cells in secondary lymphoid organs.

Linnea Swanson
Dr. Justin Spanier; Tijana Martinov; Dr. Brian Fife

**A Novel Cross Reactive Monoclonal Antibody Recognizing Insulin Peptides in MHC Class II Blocks Diabetogenic CD4 T Cells**

Type 1 Diabetes (T1D) is caused by an autoimmune destruction of insulin-producing beta cells, mediated by MHC class II-restricted CD4 T cells. Insulin is one of the major targets in patients and in the non-obese diabetic (NOD) mouse model of spontaneous T1D. In NOD mice, CD4 T cell reactivity to the native insulin β-chain 9-23 (InsB) peptide presented by the NOD MHCII molecule (IAg7) is required for disease. An additional subset of CD4 T cells recognize and respond to a substitution of a glutamic acid for glycine at position 8 (p8) of the insulin peptide. We generated a monoclonal antibody (mAb) recognizing both native insulin peptide (InsB p8E) and a modified insulin peptide (InsB p8G) in the context of IAg7. We called this mAb cross-reactive insulin 1 (XR11). XR11 inhibits in vivo antigen specific CD4 T cell proliferation and prevents antigen recognition by insulin-specific CD4 T cells in response to peptide pulse and CFA priming. XR11 prevented expansion of both InsB p8E- and InsB p8G-specific CD4 T cells to
endogenous insulin peptides when NOD mice were treated weekly with XRI1 starting at 3 weeks of age. Interestingly, XRI1 also prevented the expansion of endogenous Chromogranin A specific-CD4 T cells, another important beta cell autoantigen in T1D. This effect was observed in vivo, but not in vitro, suggesting the potential of XRI1 in prevention of epitope spreading. Future experiments will test the therapeutic efficacy of XRI1 to prevent spontaneous T1D in NOD mice. Taken together, the ability to generate novel mAb specifically targeting peptide: MHCII represents an exciting antigen-specific therapeutic approach that could prevent autoimmunity.

**Christina Warner**  
Sam Carlson, MPH; Dr. Michael Ross  

*Sexual Health Knowledge, Education, and Practice in Undergraduate Medical Education*

Introduction: Only half of U.S. medical schools require formal instruction in sexuality and sexual health knowledge is severely underrepresented on formal licensing exams. Furthermore no comprehensive survey exists evaluating sexual health literacy amongst US medical students.

Methods: A 32 question online survey was distributed electronically to representatives from all MD and DO granting medical schools in the United States. Knowledge was evaluated across five domains: Sexual Function & Dysfunction, Fertility & Reproduction, Sexuality Across the Lifespan, Sexual Minority (LGBTQIA) Health, Society, Culture & Behavior, as well as Safety & Prevention. Respondents were asked to choose True, False, or Unsure to each question. The survey was developed in coordination with clinicians in the fields of infectious disease, women’s health, and human sexuality.

Results: Survey respondents (n=994) scored an average of 65.65% correct (19.7/30). Overall, students scored lowest on questions regarding safety and prevention (x=49.05%) and highest on questions regarding sexual function and dysfunction (x=72.96%). Higher knowledge scores were associated with medical school year (p=0.0001), race (p=0.0005), sexual orientation (p=0.0001), religion (p=0.0055), future medical specialty choice (p=0.0276), type of medical school program (MD vs. DO) (p=0.001), and medical school sexual health education courses (p=0.0137). The most common questions with incorrect answers were “Emergency contraception ends an established pregnancy if taken within 72 hours” (43.8% incorrect) and “With typical use, the birth control pill is 99% effective at preventing pregnancy” (45.1% incorrect). The most common questions with correct responses were “Men and women who receive the HPV vaccine will become sexually active earlier than their non-vaccinated peers” (95.4% correct) and “There are no treatments for postmenopausal women who experience pain during intercourse” (95.2% correct). The most common questions that students marked “unsure” were “Penile enlargement can be safely achieved through surgical intervention” (32.0% unsure) and “Individuals with intellectual disabilities cannot consent to sexual activity” (27% unsure).

Discussion: Significant advances must be made in medical school sexual health curricula to combat increasing rates of sexually transmitted disease, health disparities, and morbidity in America. Further
applications of this research include medical education curriculum development and health advocacy.

**Clinical Vignette - Medical Student**

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<th>Annica Alwine</th>
<th>Superior Mesenteric Artery Syndrome: Recognizing a Rare Cause of Duodenal Obstruction</th>
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<td>Dr. Abdelghani El Rafei</td>
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Introduction: Superior Mesenteric Artery (SMA) syndrome is a rare, but debilitating, cause of proximal small bowel obstruction. Reported incidence is 0.1-0.3% in the general population. The presumed pathology is a reduced aortomesenteric angle due to rapid loss of retroperitoneal fat. A few cases have been reported with anorexia nervosa, cardiac cachexia, diabetes mellitus, and spinal surgeries.

Case: A 33 year old woman with an 8 year history of anorexia nervosa presented to the emergency department with abdominal pain and intractable emesis. Physical exam showed a chronically ill-appearing, morbidly underweight female (BMI 13) with lower abdominal tenderness. CT scan of the abdomen revealed a markedly distended stomach and narrowing of the duodenum with a beaklike appearance at the level of superior mesenteric artery. Based on image findings and clinical presentation, she was diagnosed with SMA syndrome secondary to anorexia nervosa. Patient was managed conservatively with Naso-Gastric tube placement for decompression and Gastro-Jejunal enteral feeding tube placement. She was hospitalized for 8 days to monitor for refeeding syndrome. Since discharge, she was admitted twice to an eating disorder treatment center. Her most recent BMI reported was 17.2. She is tolerating oral feeds, and was able to be weaned off tube feeding.

Discussion: This case highlights the importance of correlating clinical and radiological findings in patients with suspected SMA syndrome. Diagnosis is based on clinical signs and high clinical suspicion along with radiological investigation. CT scan often shows abrupt narrowing of the third part of the duodenum along with narrowing of the aortomesenteric angle. Mainstay of treatment is conservative management with weight gain and resolution of underlying condition. Reporting this rare syndrome will help familiarize physicians with this condition and its actual incidence.

**Maria Bryan**  

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Introduction: Multiple Myeloma is a malignant neoplasm of plasma cells belonging to the family of plasma cell dyscrasias which produces a spike in monoclonal immunoglobulin or “M-protein.” While multiple myeloma has many classic clinical features, a malignant pleural effusion is a very uncommon manifestation of this illness.

Clinical presentation: A 61-year-old man with known multiple myeloma presented with shortness of breath and fatigue. On exam, he had decreased breath sounds on the left lower and mid-lung fields. CT showed diffuse lytic and sclerotic lesions throughout the skeleton and a large left sided pleural effusion. Sampling of the pleural fluid showed
very bloody fluid. Testing for infection was negative and cytology showed plasma cells and findings consistent with malignant pleural effusion.

Discussion:
This case highlights an uncommon manifestation of multiple myeloma. While malignant effusions may be seen in many cancers, in myeloma it suggest very advanced disease with an associated poor prognosis.

**Histologically-Proven Cardiac Sarcoidosis - A Case Series**

Learning objective: Recognizing various presentations of cardiac sarcoidosis.

Cases: From our institutional cardiac sarcoidosis registry, we identified six cases of endomyocardial biopsy (EMB)-proven cardiac sarcoidosis (CS). Of the six patients, 50% (3/6) were males, 83% (5/6) were Caucasian while 17% (1/6) were African-American. Their ages ranged from 41 to 81 years. Only 33% (2/6) had a history of histologically-proven extra-cardiac sarcoidosis. On presentation, 50% (3/6) had LVEF <50%. 33% (2/6) had complete heart block, 33% (2/6) had non-sustained VT, 17% (1/6) had sustained VT, and 17% (1/6) had sinus bradycardia with sinus arrest. With regard to symptoms, 83% (5/6) had palpitations, 50% (3/6) had chest pain, 50% (3/6) had pre-syncope, and 17% (1/6) had syncope. All patient had normal coronary angiograms and late gadolinium enhancement on cardiac MRI. All patients received implantable cardioverter-defibrillators (ICD) after the diagnosis of CS. During follow up, all patients had ventricular arrhythmias. Over a mean follow up of 2.8 years, 33% (2/6) received a left ventricular assist device but subsequently died of cardiac causes before they could get cardiac transplantation. 33% (2/6) successfully underwent cardiac transplantation and are alive.

Discussion: According to the 2014 Heart Rhythm Society Consensus Statement, a definitive diagnosis of CS is only made with histological diagnosis from cardiac tissue, with clinical diagnoses considered “probable”. We describe six cases of histologically-proven CS and highlight the varied patient demographics, clinical presentations and outcomes of this condition. Our case series illustrates that isolated CS - without another organ involvement - occurs often. Studies suggest that CS is becoming more prevalent, likely due to improvements in imaging and/or thorough investigation, rather than a true increase in the disease prevalence. Patients with CS have a poorer prognosis than sarcoidosis patients without cardiac involvement. Therefore, it is paramount for clinicians to consider CS in all patients presenting with heart failure, conduction abnormalities or ventricular arrhythmia, even without a previous diagnosis of extra-cardiac sarcoidosis. Timely diagnosis of CS could prevent or possibly delay adverse cardiac outcomes such as sudden cardiac death, left ventricular assist device placement and cardiac transplantation.

**Degrees of Weakness: Biases in an Unusual Presentation of Statin Naïve HMG CoA Reductase Myopathy in a Patient with Tetraplegia**

Emily Davis
Dr. Marc Nolan; Dr. Hollis Krug
A 64 year-old man with a history of C6 tetraplegia after motor vehicle accident six years prior presented to the emergency department with subacute progressive upper and lower extremity weakness. His medications included Oxycodone, Gabapentin, Ibuprofen, and multiple herbal supplements including red yeast rice. On exam, upper and lower extremity strength were decreased bilaterally below baseline. Labs were notable for an elevated creatine kinase (CK) of 3648 units/L and transaminitis. CT and MRI imaging showed no change from previous studies. CK continued to rise during his inpatient stay. While hospitalized, he was evaluated by neurology who felt his elevated CK was most likely secondary to prolonged immobilization. Despite a rising CK, he was discharged home.

One month later, he was evaluated by rheumatology who noted a continued increase in CK with an otherwise negative workup including negative ANA, RF, ENA panel, and anti-Jo1. EMG was obtained showing non-specific neurogenic changes and short duration high frequency discharges, which could be associated with spinal cord injury. His persistent weakness was attributed to disuse atrophy given his history of C6 tetraplegia, and physical therapy was recommended.

Six months later, he was admitted for worsening generalized weakness, shortness of breath and dysphagia requiring a PEG tube. Admission labs showed a CK of 6200. MRI of the pelvis revealed mild intramuscular edema of scattered muscles in bilateral thighs, and biopsy was recommended. Muscle biopsy demonstrated muscle damage with atrophic and necrotic myofibers with signs of inflammation, but a complete myositis panel and genetic testing for alpha-glucosidase deficiency were unrevealing. Repeat EMG testing showed new evidence of widespread myotonic discharges, indicative of irritative myopathy. HMG CoA reductase (HMGCR) autoantibody testing returned elevated at 45,000 units (negative < 20). Given his presentation, EMG findings, autoantibody levels, and biopsy findings, the patient was diagnosed with HMGCR autoimmune necrotizing myopathy, thought possibly related to his herbal supplement use. His treatment included Prednisone, Methotrexate, Cyclophosphamide, and Plasma Exchange, later with Rituximab and IVIG infusions, resulting in clinical improvement.

This case demonstrates an atypical presentation of HMGCR associated autoimmune myopathy (also called statin-associated autoimmune myopathy). Clinically, it is characterized by symmetric proximal muscle weakness, positive anti-HMG CoA reductase autoantibody, significant elevations in CK, and necrotizing myopathy on muscle biopsy. While 60% of patients are documented to have statin exposure, up to 40% of patients are statin naïve. Statin-like compounds may be found in dietary supplements like red yeast rice, possibly triggering a similar response – a potential explanation of this patient’s disease without known statin exposure. This case also illustrates the potential for cognitive bias interfering with accurate and timely diagnosis when co-morbid conditions like tetraplegia are present that may amplify presenting symptoms or mask a new and evolving process.
| Aakash Deshpande  
Dr. David McDonald | Eosinophilic Myocarditis in the setting of Tropical Travel |
|-------------------|----------------------------------------------------------|
| Shortness of breath is a common presenting complaint and the differential diagnosis is quite broad with cardiac, pulmonary, hematologic, rheumatologic, and immunologic etiologies. Given the range of etiologies, it is important to maintain a broad differential diagnosis when evaluating a patient with shortness of breath.

A 72-year-old gentleman with a history of hyperlipidemia, hypertension, benign prosthetic hypertrophy, and recurrent nephrolithiasis presented to the ED with a 2-month history of intermittent night sweats, shortness of breath, and chest pain. He had recently been admitted with bacteremia after a procedure and had been discharged on minocycline. He then departed on a cruise to the south pacific and additionally had spent many months in Mexico over the last year. He began having diaphoretic episodes while on the cruise; an initial chest X-ray and TTE were unremarkable. He was treated with Augmentin without improvement and due to worsening diaphoresis he was advised to return home from the cruise. While returning he began experiencing shortness of breath and recurrent episodes of substernal chest pressure and left arm pain that resolved without intervention.

In the ED, he was found to have an elevated troponin (0.327 ng/mL) and an EKG showed a left bundle branch block. Chest x-ray revealed cardiomegaly. He was admitted with suspicion for NSTEMI. Physical exam was notable for visible diaphoresis, irregular cardiac rate & rhythm, and mild crackles in both lung bases. He had a normocytic anemic with a hemoglobin of 10.8 and a mild eosinophilia (WBC 10.71, Eos 0.74) CRP was 101 and ESR 82. He was started on broad spectrum antibiotics due to his recent bacteremia. Given his extensive travel history an infectious and rheumatologic workup was undertaken and was negative other than elevated IgE (156 IU/mL). Repeat echocardiogram showed a newly decreased ejection fraction (35-40%) and eosinophil count continued to rise (0.74 to 1.78). Coronary angiography was normal. An endomyocardial biopsy was performed revealing numerous eosinophils infiltrating the myocardium, leading to a diagnosis of eosinophilic myocarditis. Steroid treatment was initiated with rapid improvement in eosinophilia and symptoms. A follow up echocardiogram and MRI revealed no sign of myocarditis. The suspected cause was chlorthalidone which had been initiated 2 weeks prior to onset of symptoms.

Eosinophilic myocarditis is a rare condition that can mimic a variety of cardiac pathologies. Etiologies include medication hypersensitivity, allergic disorder, vasculitis, idiopathic, and parasitic infections. Treatment involves high dose IV steroids and usual heart failure treatment followed by an oral steroid taper, and if relevant, removal of the offending agent. Chlorthalidone, and more commonly hydrochlorothiazide, have been implicated as causes of eosinophilic myocarditis. |

| Elizabeth Fairbairn  
Dr. Kyle Lehenbauer | Should recent vaccine administration be considered a cause of shoulder bursitis or inflammatory arthritis? |
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<td>Sticking Point: Severe Shoulder Pain Following Vaccine Administration.</td>
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A 76-year-old man with past medical history of ulcerative colitis treated with vedolizumab, mesalamine, and methotrexate presented to clinic with a swollen and painful right shoulder that he believed was due to a pneumococcal vaccination he received 10 days prior. He was on day 3 of a 10-day course of amoxicillin/clavulanate for a dental infection. His right shoulder is visibly swollen with a tender effusion on the anterior shoulder. Patient refuses range of motion testing due to pain. Ultrasound imaging of shoulder revealed a 6 cm mildly complex subdeltoid fluid collection communicating with the joint space.

There are case reports of shoulder injuries related to vaccine administration (SIRVA). It is believed to be caused by antigenic material improperly injected into shoulder bursas or synovial space.(1) Symptoms usually appear in 24 to 48 hours and can last for weeks to months. Risk factors include decreased subcutaneous fat or low muscle mass and underlying shoulder abnormalities. This patient’s BMI of 22.7, muscle atrophy, and x-ray findings indicative of a rotator cuff pathology put him at increased risk.

The fluid collection was aspirated with the following results: cloudy yellow fluid; 62,000 WBCs, 4000 RBCs; cultures were negative; crystals and a few intracellular calcium pyrophosphate (CPP) crystals were seen. One case report on SIRVA following pneumococcal vaccination included aspiration results with elevated WBC, but no crystals.(2) No cases of CPP deposition in possible SIRVA have been reported.

Treatment consisted of a course of prednisone for pseudo-gout. Recommended treatment for SIRVA is also prednisone. At time of submission, patient has been on prednisone for 5 days and reports continued symptoms with decreased pain.

SIRVA can be prevented by proper vaccine administration focusing on proper site, patient positioning, and needle length to ensure the injection is in the deltoid muscle.(3) Being at the same level as the patient; e.g., both sitting, can help ensure the injection isn’t given too high on the shoulder and that the needle is perpendicular to the skin.

References:
patients. However, PCP can also be seen in other conditions of T cell immunosuppression, including patients on chronic corticosteroids.

Case presentation: A 47-year-old woman with history of possible autoimmune hemolytic anemia (treated with 40 mg of prednisone daily for 2 months) and end stage liver disease secondary to alcoholic hepatitis was admitted to the hospital with anasarca and anemia. She was initially treated with albumin for her anasarca and a blood transfusion for the anemia.

She developed worsening dyspnea on hospital day 2 without productive cough, fever, or chills. Exam showed no fever or hypoxia, but did show increased work of breathing and crackles in the upper lung fields. She also had a holosystolic murmur at the left upper sternal border and 4+ pitting edema to her mid-chest.

Labs showed high-normal white blood cell count, chronically elevated bilirubin and LDH, and low albumin. Chest x-ray which showed diffuse opacities most prominent in the lung apices.

Echocardiogram did not show heart failure that could cause cardiogenic pulmonary edema. CT done later showed bilateral ground glass opacities with peribronchovascular distribution and interlobular septal thickening.

Because of the non-resolving infiltrates, an induced sputum was done which showed a direct fluorescence antibody (DFA) positive for PCP. She was started on trimethoprim-sulfamethoxazole therapy.

Discussion: This case reiterates that PCP, though well-studied in HIV patients, is poorly understudied in non-HIV (NH) infected patients, making its prevention, diagnosis, and management a precarious course for providers to navigate and a fertile field for further investigation. It is important to remember that the PCP presentation in NH-PCP varies drastically from that in HIV-PCP, and that significant history for immunosuppression and imaging consistent with PCP should prompt further investigation into PCP, even in a virtually asymptomatic patient. Moreover, this case illustrates the need to have a broad general approach to pulmonary edema, including both cardiogenic and non-cardiogenic causes. In this case, the patient’s alcoholic liver disease and steroid use both likely predisposed her to the infection. While some guidelines recommend PCP prophylaxis for patients on long term steroids, systematic reviews have not found a consistent dosing regimen or recommendation to guide treatment.

Fatal Nonuremic Calciphylaxis: A Case of Atypical Risk Factors

Calciphylaxis (calcific uremic arteriolopathy) is a rare and often lethal disorder characterized by calcification of the arterioles, leading to tissue ischemia and necrosis. While this disorder is usually seen in individuals with end-stage renal disease, it is not limited to this patient population. We report a case of fatal nonuremic calciphylaxis in a woman recently diagnosed with malignancy.
An 82-year-old woman was transferred to our hospital from an outside emergency department for acute encephalopathy, acute kidney injury, and a left leg wound refractory to outpatient treatment with cephalexin and levofloxacin. The patient’s medical history was significant for recently-diagnosed multiple myeloma, for which she decided against pursuing treatment, chronic warfarin anticoagulation for atrial fibrillation, previous cardioembolic strokes, and type 2 diabetes mellitus. Upon admission physical exam, the patient was disoriented, and skin exam revealed a large, tender, nonpurulent ulcer with overlying eschar and local noninflammatory retiform purpura on her left posterior calf. Laboratory studies were significant for macrocytic anemia with hemoglobin of 10.1 mg/dL, leukocytosis of 18.8 x 10^9/L, serum creatinine of 2.4 mg/dL, normal ionized calcium of 4.6 mg/dL, decreased serum albumin of 2.6 g/dL, and supratherapeutic INR of 6.5. She was initiated on broad spectrum antibiotics with vancomycin, piperacillin-tazobactam, and metronidazole. There was high suspicion for an underlying vascular process due to the painful nature of the wound, lack of healing, and surrounding retiform purpura. Doppler studies of the lower extremity were negative for arterial occlusive disease, and serum rheumatologic autoantibody levels were not suggestive of vasculitis. Calcium and phosphorus levels were normal, but parathyroid hormone level was elevated at 111 pg/mL. Ultimately, biopsy revealed necrotizing panniculitis with intravascular and extravascular calcification, consistent with calciphylaxis. Tissue culture grew coagulase-negative Staphylococcus (+1). The wound was treated with ultrasonic MIST therapy, topical sodium thiosulfate, and anticoagulation with apixaban. The patient and her family declined more aggressive treatments, such as surgical debridement and hyperbaric oxygen, and ultimately decided upon palliative therapy. She was discharged to hospice care and expired shortly thereafter.

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<th>Merry Huang</th>
<th>Dr. Ozra Esalampanah Nobari</th>
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<td>“Doctor, is my brain wired with excess copper?”: A Case of Neuropsychiatric Symptoms in Untreated Idiopathic Copper Toxicity</td>
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Introduction: Copper is a highly reactive metallic ion that is found in the liver and brain in high concentrations. Copper is a cofactor for enzymes involved in neurotransmitter and collagen synthesis. However, its excess causes oxidative damage and tissue deposition leads to global organ impairment. Copper excess is commonly recognized interchangeably as Wilson’s disease, however, isolated chronic copper toxicity can be an organic cause to psychiatric disturbances.
Case: Patient is a morbidly obese wheelchair-bound 43 years old Caucasian female with a complex medical and psychiatric history. She presented to the Emergency Department (ED) by police due to expressing Suicidal Ideation (SI) and reported behavioral disturbances including aggression and violation of multiple residential rules at her group home. Due to endorsing current SI with a plan and considering the patient’s extensive history of over 33 psychiatric hospitalizations, she was admitted to inpatient care.

During the hospitalization, patient was initially agitated and combative. After minor medication adjustment, her mood improved and anger outbursts decreased. However, her long-standing passive SI remained unchanged. Clozapine and lithium were not tried to target her suicidality during the current hospitalization due to past failed trials. Patient’s hospitalization prolonged due to complicated post discharge placement, which allowed the primary team to revisit the diagnosis and investigate the underlying cause of her clinical presentation.

Her past medical history was significant for a wide range of diagnoses including osteoarthritis, seizure disorder, Obstructive Sleep Apnea (OSA), recurrent Deep Vein Thrombosis (DVT) (on warfarin), chronic pain (on intrathecal morphine delivering pain pump), dermatitis, nonspecific thyroid disorder, anemia, endometriosis, numerous gastrointestinal problems (including dysphagia, cholecystitis post cholecystectomy, acute pancreatitis), and status post bariatric surgery with multiple revisions.

Chart review for psychiatric history revealed diagnosis of developmental delay and intellectual disability at a young age. Her psychosocial history was significant for emotional, physical, and sexual abuse. Patient experienced a new onset of seizures, migraines, and hallucinations at her 20s and was placed on SSDI for her disabilities. The inconsistent psychiatric diagnosis through her lifetime was puzzling and included Major Depressive Disorder (MDD) with psychotic features and suicidal tendencies to Bipolar Disorder (BP) with borderline personality traits.

In this poster, we present a case of a 43 years old female with significant medical and psychiatric history. We explore the role of idiopathic copper toxicity at explaining this patient’s diverse clinical symptoms.

Discussion: Micronutrients imbalance cause psychiatric disturbances. Chronic idiopathic copper toxicity should be considered as an organic cause to unexplainable neuropsychiatric symptoms especially in the setting of other seemingly unrelated medical problems. Medications used to treat excess copper are extremely inexpensive compared to the costs of inpatient hospitalizations. We suggest that detection of copper toxicity and early intervention can improve patient care outcomes.

Joseph Jude

Remarkably Elevated Lipase in the Absence of Pancreatitis

Introduction: Serum lipase elevation is seen most frequently in acute pancreatitis. Elevations can be seen in many other disorders, including intestinal and kidney diseases. However, very elevated lipase
measurements in the absence of pancreatitis have rarely been described.

Clinical presentation: A 29-year-old woman with inflammatory bowel disease (IBD) was admitted for acute left lower quadrant abdominal pain. She reported four weeks of diarrhea and no epigastric or pain radiating to her back. She had nausea and poor oral intake. On exam, she had focal tenderness to palpation in her left lower quadrant without rebound or guarding. She had normal bowel sounds, no masses, or organomegaly.

Her initial labs were notable for a normal complete blood count, normal metabolic panel and liver function tests and a serum lipase of 1,572 IU/L.

CT of the abdomen showed a normal appearance pancreas and also no inflammation of the intestine suggestive of an IBD flare. She was managed with pain control and bowel rest and slowly improved over two days.

After admission, upon further review of her chart, it was noted that she had two prior admissions with similar presentation and similarly elevated lipase (as high as 2,000 IU/L) with normal pancreas imaging.

Discussion: While elevated lipase is consider over 90% specific for acute pancreatitis, providers must be aware that even highly elevated levels are not a “slam dunk” for diagnosis. This could help providers not only recognize other causes of elevated lipase, but also avoid unnecessary additional testing based on a lab value without clinical correlation.

Christina Lee
Dr. David MacDonald

Back Pain: The Unusual Presentation of Diffuse Large B-cell Lymphoma (DLBCL)

Low back pain is among the most common reasons to seek medical attention and the total cost of low back pain exceeds $100 billion/year. The vast majority of patients improve without additional evaluation or intervention; however, low back pain can be a symptom of serious pathology. A 70 year old man presented to our hospital with subacute back pain, which had been progressively worsening for two months. The pain radiated from his lower back to the buttocks and bilateral ankles. He had no sensory or strength complaints and no changes in bowel or bladder function. At the time of admission, the pain had become unbearable and was awakening him at night. He had tried oxycodone, ibuprofen, and cyclobenzaprine without relief. He had obtained temporary relief with 2 courses of steroids. He denied any recent weight loss, night sweats, fevers, or trauma. He had no history of IV drug use.

On physical examination, he was obese, had severe pain and reduced lumbar spine range of motion. The remainder of the exam including his neurological exam was unremarkable. A CBC with differential was unremarkable; he had mild elevations in his c-reactive protein and erythrocyte sedimentation rate. A lumbar spine xray revealed a compression fracture of the L3 vertebral body. A subsequent lumbar spine MRI demonstrated abnormal T1 and T2 signal enhancement in the L3 vertebral body with extension of the abnormal enhancement into the L2-L3 foramina, and moderate to severe central canal stenosis at L2-L3 and L3-L4 due to the combination of degenerative changes and an epidural mass. Due to concern for malignancy, a CT
chest/abdomen/pelvis was obtained, which did not reveal any evidence of primary malignancy. A prostate specific antigen was normal. He then underwent L2-L4 laminectomy and biopsy of the L3 epidural mass and vertebral body, which demonstrated infiltrative large pleomorphic neoplastic lymphoid cells with sclerotic background, consistent with diffuse large B-cell lymphoma (DLBCL) which was confirmed with immunohistochemistry. A PET CT revealed evidence of likely metastatic spread to periaortic and periduodenal lymph nodes. He was at moderate risk for CNS metastasis; thus, a brain MRI and C1/C2 lumbar puncture were performed, which showed no evidence of intracranial disease. He will likely begin R-CHOP in the coming weeks. Non-Hodgkin's Lymphoma arising in the spine is rare; two recent case reports have documented three patients diagnosed with DLBCL in the thoracolumbar spine, two of whom presented with back pain and one who presented with paraparesis. Diagnosis of NHL localized to the spine can be delayed due to symptoms similar to benign causes of back pain and non-specific imaging characteristics. This case highlights the importance of maintaining a high clinical suspicion for more ominous causes of back pain.

Joy Liu
Dr. Wil Santivasi; Dr. Dagny Anderson

When Friend Becomes Foe: A Case of Piperacillin/Tazobactam-Induced Neutropenia

Introduction: Piperacillin/tazobactam (Zosyn) is often used for its extended coverage of gram negative and positive bacteria, including Pseudomonas aeruginosa. Though it is most classically associated with renal injury and dermatologic reactions in short-term inpatient use, myelosuppression can be seen with longer outpatient treatment courses.

Case Presentation: A 21-year-old man with a history of polymicrobial osteomyelitis secondary to open fractures from a motor vehicle accident was admitted to the hospital with a 7-day history of malaise, subjective fever, and chills. On admission, vital signs were HR 67, BP 138/82, temperature 36.9°C, RR 16, with oxygen saturation 98% on room air. Physical exam revealed well-healing surgical incisions over the right arm with healing grafted skin. Laboratory evaluation disclosed leukopenia and neutropenia (1,900 cells/mm³ and 390 cells/mm³, respectively). Of note, on admission, the patient was 23 days into a planned 36-day course of outpatient antibiotic therapy with piperacillin/tazobactam for his osteomyelitis. Routine outpatient laboratory monitoring had been within normal limits up until three days prior to admission, when he was noted to have mild neutropenia (1,250 cells/mm³). Due to concern for drug-induced neutropenia, piperacillin/tazobactam was discontinued in favor of meropenem. Peripherally- and PICC-drawn blood cultures obtained on admission were without growth. Though he had reported subjective fevers prior to admission, he remained afebrile throughout his hospitalization. Following transition to meropenem, his leukopenia and neutropenia gradually improved. Five days after discontinuation of piperacillin/tazobactam, laboratory monitoring showed resolution of his leukopenia and neutropenia (4,900 cells/mm³ and 2,040 cells/mm³, respectively). His blood counts remained stable through completion of his course of meropenem.
Discussion: This case demonstrates the importance of regular blood count monitoring in patients receiving outpatient antibiotics, such as piperacillin/tazobactam. While more common in children, piperacillin/tazobactam-induced neutropenia can be seen in adults, as was the case here. The risk of neutropenia increases with duration of exposure, with the majority of blood count abnormalities occurring after treatment day 14. In these patients, it is important to rule out recurrent or incompletely-treated infection and/or new PICC-associated bloodstream infection as a cause of the patient’s neutropenia. More serious systemic reactions to the drug, including Stevens-Johnson syndrome and DRESS, may also occur and must be ruled out when drug complications arise. The definitive treatment of drug-induced cytopenias is discontinuation of the offending agent with supportive care through count recovery.

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<th>Allison McCarter</th>
<th>Pain, Fever &amp; Erythema Nodosum: An Atypical Presentation of Q Fever</th>
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<td>Dr. Prowpanga Udompap; Dr. Robert Fell; Dr. Wei Duan-Porter</td>
<td>Introduction: The most common presenting symptoms of Q fever are non-specific headache (68-98%) and myalgias (47-69%); yet a majority are asymptomatic. We describe the case of a middle-aged gentleman who presented with 1.5 weeks of diffuse pain, fever, night sweats and erythema nodosum. Extensive work-up for rheumatologic, infectious and malignant etiologies led to the diagnosis of Q fever.</td>
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<td>Case description: A previously healthy 54-year old man from Northern Minnesota presented with diffuse pain, night sweats and fever for 10 days. Physical exam was notable for low-grade fever and erythema nodosum (EN) of the bilateral shins. Basic laboratory results were normal aside from diffusely elevated inflammatory markers: CRP 179 mg/L, ESR &gt;120 mmHg. Extensive laboratory testing and imaging studies were performed to further evaluate fever of unknown origin. Quantiferon gold, ASO titer, lyme titer, and all fungal, parasitic and microbiologic testing were negative. Testing for tularemia and Q fever was sent, but results were pending for 8 days. A chest CT revealed multifocal solid nodules greatest in right upper lobe and hilar lymphadenopathy. CT of the abdomen and pelvis was notable for numerous retroperitoneal lymph nodes. Rheumatologic studies were negative, including ANA, RF, CCP, ANCA and ACE levels. Pain and fever were controlled with scheduled acetaminophen and oxycodone throughout clinical course. The EN resolved without treatment. The remaining differential largely included vasculitis and malignancy, including lymphoma. While awaiting further results, PET CT was obtained to examine the retroperitoneal adenopathy. This revealed mild hypermetabolism of the large vessels, esophagus and hilar lymph nodes. EGD with EUS identified an irregular, 2.5 cm mass at the GE junction invading the pleura. Interestingly, the biopsy result was negative for malignancy. As there was no definite culprit, we planned to proceed with steroids in the treatment of vasculitis. Simultaneously, serology revealed 1:16 titer of Phase-II Q Fever IgG on day 20 of admission. The patient was treated with a 2-week course of doxycycline for Q fever and a low-dose steroid taper for vasculitis with complete resolution of symptoms.</td>
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Discussion: Fever of unknown origin (FUO) may represent a wide range of disease including autoimmune, malignancy or infection. Thus, a broad differential diagnosis for FUO is warranted. Though uncommon, erythema nodosum has been reported as the presenting feature of Q fever. C. burnetii serology may be a helpful diagnostic tool among patients with FUO with atypical presentation, especially in those with exposure to cows, goats, sheep, cats or dogs. Awareness of atypical presentation in FUO may prevent unnecessary and potentially invasive intervention.

Kevin Miller  
Dr. Shaji Kumar  

**New Onset of Lytic Lesions in a Patient with a Monoclonal Gammopathy: Keeping Multiple Possibilities Abreast**

A 69-year-old woman was incidentally found to have anemia while being worked up for a seizure-like episode in August 2004. Her past medical history was significant for hypothyroidism, as well as enlarged ovaries which were surgically removed and benign. Her evaluation included SPEP and immunofixation, which demonstrated an M-spike of approximately 2 g/dL with an elevated IgM of 3154 mg/dL. Consequently, she had a bone marrow biopsy which showed 7% plasma cells and 20% small lymphocytes. As this met the criteria for IgM monoclonal gammopathy of undetermined significance (MGUS), she was observed without treatment.

Seven years later in November 2011, she noticed a lump in her left breast. She was diagnosed with invasive ductal carcinoma, T1cN0M0, which was ER+, PR+, and HER-2 negative. This was treated with local surgery and partial breast irradiation totaling 3400 cGy. She was in remission for several years until she developed a new nodule in her left breast in May 2015. Biopsy revealed a 3.5 mm deep dermal recurrence. In addition, MRI showed an enhancing lesion in the right breast, which was biopsied and found to be grade 2 invasive lobular carcinoma, ER+, PR+, HER-2 negative. Therefore, she had a bilateral mastectomy. She declined the recommended adjuvant chemotherapy and radiation, but did take anastrozole, stopping after one year because of significant fatigue. In January 2016, CT and bone scans were negative for metastases, and there was no evidence of axillary or hilar adenopathy.

About one year later in May 2017, she developed lower back pain radiating to her extremities. MRI showed diffuse abnormalities throughout the spine, sacrum and iliac bones without cord compression. Her hemoglobin was 10.4 g/dL and serum IgM was 3970 mg/dL at that time. Subsequently, she was referred to Mayo Clinic for management of what was thought to be progression of her IgM MGUS to Waldenström macroglobulinemia. Her hemoglobin was found to have precipitously dropped in the interim to 8.6 g/dL. However, her M-spike remained stable. Further evaluation included a PET-CT, which demonstrated extensive hypermetabolic lytic lesions throughout the entire axial and appendicular skeleton. Moreover, she had a single FDG-avid lesion in the liver. She underwent a bone marrow biopsy, which clinched the unfortunate diagnosis: metastatic breast adenocarcinoma, with strong expression of GATA3, ER, and PR. In addition, 5% kappa-restricted plasma cells were noted, representing her underlying MGUS clone. She was started on denosumab, letrozole, and palbociclib, received palliative
radiation to the spine and pelvis, and returned to her home institution.

In summary, lytic lesions are common in multiple myeloma (present in about 67% of patients at the time of diagnosis), but can also occur in other malignancies, including metastatic carcinoma. In a patient with both possibilities, a thorough investigation is warranted.

**Hanna Nedrud**  
**Dr. Janewit Wongboonsi**

*The Pressure is On! Intracranial, Systemic, or Both?*

Introduction: We report a unique case of idiopathic intracranial hypertension, which occurred in a man and dually presented with hypertensive emergency.

Case description: A 39-year-old man with a history of pre-hypertension and obesity presented with six months of progressive left eye visual loss and headaches exacerbated by lying down relieved with ibuprofen, and one week of right eye “flashers”. Physical examination revealed blood pressure of 234/152 mmHg, pulse of 108/min, unremarkable cardiopulmonary examination, no focal neurologic deficits except for severe bilateral decreased vision more prominent on left side, and bilateral papilledema. Initial labs revealed creatinine of 1.56 mg/dl, hypokalemia, mild normocytic anemia and troponin of 0.281 µg/L. Peripheral blood smear revealed no schistocytes. Brain MRI revealed no masses, thrombosis or optic nerve enhancement, but demonstrated bilateral posterior scleral flattening with protrusion of optic discs. The patient was admitted to the ICU for tight blood pressure control. A lumbar puncture was performed, revealing an elevated opening pressure of 50 cm, 1 WBC, 0 RBCs, 65 mg/dL glucose, 45 mg/dL protein, negative for gram stain, cultures and oligoclonal bands. The patient was diagnosed with Idiopathic Intracranial Hypertension (IIH). Initial management included acetazolamide 500 mg twice daily and blood pressure control. The patient reported improvement of symptoms. However, upon repeat ophthalmologic evaluation, his visual field and acuity remained severely impaired on the left and worsened on the right. Thus, intravenous methylprednisolone was initiated as a bridge to optic nerve sheath fenestration. The patient also underwent a workup for secondary hypertension, which returned unremarkable. The patient’s acute kidney injury was attributed to hypertensive nephrosclerosis. Three weeks following discharge, the patient’s visual acuity had decreased bilaterally and his visual field in the right eye had worsened. The patient underwent an urgent right ventriculoperitoneal shunt placement the next day.

Discussion: This case illustrates a unique presentation of IIH. IIH, raised intracranial pressure in absence of a space-occupying lesion, usually occurs in women of childbearing age. It is associated with elevated systemic arterial hypertension, however an association with hypertensive emergency has not been reported. Although uncommon in men, male gender is associated with a more severe presentation and disease prognosis. Given the weak association between IIH and hypertensive emergency, we deemed these disorders as separate entities, requiring the patient to have a full work up of secondary hypertension. It is important to note that the patient’s ocular findings did not include common manifestations of systemic hypertension including retinal arteriolar...
attenuation, arteriovenous ‘nicking’, and cotton wool spots. This suggests a subacute, rather than chronic, hypertensive disorder. This case also illustrates the importance of aggressive management for IIH and the objective assessment of visual symptoms to identify appropriate treatment as the patient did report subjective improvement in vision.

**Joseph Novak**  
**Dr. Sam Ives**

*Brain and Spinal Cord Lesions in a Patient with Miliary Tuberculosis*

Healthcare providers have the unique privilege to evaluate and treat patients from around the globe. This often involves managing diseases less prevalent in US-born patients. In Minnesota, for instance, 615 of the 747 documented cases of tuberculosis between 2011 and 2015 occurred in patients not born in the United States. Described here is the case of an older man, originally from Somalia, with an unusual presentation of tuberculosis.

An 81-year-old Somali man with a past medical history of latent tuberculosis, hepatitis C, hypertension, and Meniere’s disease visited a neurology clinic for sharp pain and paresthesias wrapping from his right back to the periumbilical region. There was no sensorimotor deficit in any of his limbs. He also reported fatigue, weight gain, decreased appetite, diaphoresis, and hemoptysis. An MRI of the thoracic spine ordered for this trunk pain revealed intramedullary nodular enhancement within the cord at level T6-7, concerning for neoplasm. In light of these findings, a brain MRI was obtained. It showed innumerable brain lesions, some with cystic-appearing components, as well as subtle leptomeningeal enhancement in the right parieto-occipital lesions. Concern at that time was primarily for metastatic disease, though tuberculosis remained a consideration.

Following his appointment with neurology, the patient was found to have a positive AFB smear during a visit at his primary clinic. He was admitted to the hospital to treat a reactivation of latent tuberculosis. A chest CT at that time showed diffuse, tiny pulmonary nodules bilaterally, consistent with miliary tuberculosis. It was therefore presumed that his brain and spinal lesions were tuberculous, rather than metastatic, in nature. Treatment was initiated with rifampin, isoniazid, and pyrazinamide.

Unfortunately, the patient’s condition deteriorated early in his hospitalization. His mental status followed a downward trajectory and he became difficult to awaken, minimally responsive to pain, and unable to take medications by mouth. After several days, his neurological exam began to wax and wane, though it consistently remained below his baseline. Repeat MR imaging of his brain revealed areas of ischemia consistent with stroke in addition to the previous lesions. Throughout this process, his occasional inability to take oral medication necessitated frequent changes to his tuberculosis treatment.

Dissemination of tuberculosis to the central nervous system occurs in only 1% of cases of tuberculosis. Among these cases, intramedullary involvement is relatively uncommon, representing approximately 4% of all CNS tuberculosis. The prognosis of spinal intramedullary tuberculosis is favorable if it is found early and managed appropriately.
However, its similarity in presentation to intramedullary spinal cord tumors may delay its discovery and thereby, its treatment. The case described here demonstrates the importance of both treating latent tuberculosis and maintaining a high index of suspicion for tuberculosis among at-risk populations.

**Jeremie Oliver**  
Dr. Deanna Menapace; Dr. Shelagh Cofer

**Otorhinolaryngologic Manifestations of Hartsfield Syndrome**

Learning Objective: To elucidate otorhinolaryngologic manifestations and management strategies of Hartsfield syndrome.

Core Competencies Addressed: Medical knowledge, Patient care.

Clinical Vignette: The first case of familial Hartsfield syndrome was seen at our institution approximately three years ago. An established clinical diagnosis of Hartsfield syndrome is achieved through the recognition of three distinct pathologies: holoprosencephaly, ectrodactyly, and bilateral cleft lip-palate syndrome. Accurate diagnosis of this disease can be achieved through prenatal ultrasound to detect holoprosencephaly, ectrodactyly of the hands and feet, as well as cleft-lip and palate. Physical findings should be confirmed by genetic evaluation of the FGFR1 gene. This report focuses on the pertinent otorhinolaryngologic manifestations and management strategies that to our knowledge, have never been reported.

Design: Case series and review of literature.

Method: Patient medical record data collection. Review of literature performed using an electronic search using Ovid Medline and PubMed in August 2016 without timeframe limitations. Only English-language citations were considered. The following were used as either keywords or Medical Subject Headings (MeSH) in all combinations in the search strategy: “Hartsfield syndrome”, “Hartsfield holoprosencephaly and ectrodactyly” and “Hartsfield bilateral cleft-lip and palate”. The compiled reference lists were compared and reviewed for potential relevance and assessed using the inclusion and exclusion criteria.

Conclusions: Hartsfield syndrome is a very rare disorder with less than 20 cases reported in the literature. Manifestations of Hartsfield syndrome pertinent to otorhinolaryngologists include: congenital bilateral cleft-lip and palate, retrognathia, gastroesophageal reflux disease, ear deformities, eustachian tube dysfunction, midface abnormalities and craniosynostosis. Management should include multidisciplinary and longitudinal care coordination including: a pediatrician, geneticist, otorhinolaryngologist, plastic surgeon, endocrinologist, neurologist and speech and swallow specialist.

**Pierce Peters**  
Dr. Samuel Shabtaie

**Blinded by Korsakoff: Diplopia and Gait Ataxia in a Patient with Alcohol Use Disorder**

Wernicke’s encephalopathy is described clinically as encephalopathy, oculomotor dysfunction, and gait ataxia. Unfortunately, it rarely presents with this triad, making diagnosis difficult and empiric treatment necessary to prevent permanent damage.
A 55-year-old man with a past medical history of alcohol use disorder and hypertension presented to his local hospital three days after acute onset of diplopia and gait unsteadiness with associated nausea and vomiting. The patient had been sober for years until he relapsed three months before, averaging 8-10 beers per day since that time. His symptoms did not change over the following three days, leading him to present to his local emergency department for evaluation. The ED physician found limited extraocular movement. Exam was otherwise unremarkable. Serum ethanol level was negative and electrolytes/CBC were within normal limits. Vitals were notable for hypertension, ranging 161-211/92-128. Head CT was negative for any acute abnormalities. He was started on IV thiamine and was subsequently transferred to our institution for further evaluation. Neurology was consulted and confirmed extraocular muscle impairment, finding impaired abduction and adduction in the left eye, impaired abduction in the right eye, and horizontal nystagmus with right eye abduction. There was no vertical limitation in either eye. He walked with a wide-based, ataxic gait. Treatment proceeded under a working diagnosis of Wernicke’s encephalopathy. However, MRI the following day revealed a small acute infarction within the left dorsal pons and no mammillary body abnormality. Thiamine, folate, and B12 levels returned normal, though after 24 hours of replacement. CT angiogram was ordered at that time, finding significant carotid atherosclerosis bilaterally but an unremarkable vertebrobasilar system. Blood pressure was lowered to a goal of 160 systolic. Further investigation revealed that he had not followed his antihypertensive regimen for years, stating that it was too expensive to travel to retrieve his prescription. He was diagnosed with acute ischemia secondary to cerebral small vessel disease. His neurological symptoms improved over the course of his hospitalization. At discharge, a mild sixth cranial nerve palsy persisted.

Wernicke’s encephalopathy is primarily a clinical diagnosis and must be treated empirically to avoid Korsakoff’s syndrome. MRI can be useful but sensitivity is low and presentation varies with timing. Thiamine testing is also unreliable, as the sensitivity and specificity of blood levels in symptomatic patients are unclear. In the face of diagnostic ambiguity, mimickers must be ruled out. This case highlights the value in completing a thorough workup in suspected Wernicke’s encephalopathy.

Laura Piccolo Serafim
Dr. Jonas Paludo;
Dr. Narjust Duma;
Dr. Naseema Gangat;
Dr. Alice Gallo de Morales

A Hematological Emergency Announced by the Eyes

Introduction: Leukostasis is a medical emergency caused by white blood cell plugs obstructing the microvasculature, leading to hypoxia and end-organ damage. Symptoms can be varied and are reflective of the location where the tissue hypoxia is occurring. The most common manifestations involve the respiratory and neurological systems with a mortality rate that may reach 40%. As the first evaluator, it is important for the Internist to suspect and recognize this condition with high morbidity and mortality. We report a case of chronic myeloid leukemia (CML) in a young man presenting with visual disturbance.

Case: A 39-year-old man presented to the ophthalmology office with acute blurry vision. He denied any other symptoms. Past medical history
was significant for a hypertensive emergency with visual changes in 2014 that had resolved. He denied alcohol, tobacco or illicit drugs use. On funduscopic assessment, diffuse retinal hemorrhages in both eyes, macular edema and foveal hemorrhage in the left eye were noticed. A CBC was ordered which showed a WBC of 542x109/L with a predominance of myeloid cells at all stages of maturation. The patient was referred to the Emergency Department for evaluation. His physical examination was remarkable for splenomegaly, with the spleen edge approximately 10cm below the costal margin. Patient denied any constitutional symptoms, headache or other neurologic signs, shortness of breath, chest pain, obvious abnormal bleeding, muscle or joint pain or skin rashes. Laboratory studies demonstrated hemoglobin of 9g/dL, platelets of 370x109/L and creatinine of 2.2mg/dL with BUN of 38mg/dL. Review of peripheral smear was consistent with chronic-phase CML. He was managed with fluids, allopurinol and one dose of rasburicase. Urgent leukopheresis was performed and hydroxyurea was started. Visual symptoms improved within hours of leukopheresis. Bone marrow biopsy confirmed chronic phase CML, BCR/ABL-positive, with 2% circulating blasts and <5% marrow blasts. The WBC count decreased to 73,9x109/L six days later, hydroxyurea was discontinued and he was started on definitive therapy with imatinib.

Discussion: Leukostasis is a rare but life-threatening complication of hematological malignancies. The clinical presentation is non-specific therefore investigation of symptoms, such as blurry vision with characteristic funduscopic findings, should include leukostasis in the differential diagnosis. Reasonable suspicion leads to prompt treatment as delayed intervention significantly increases mortality. The initial management of leukostasis involves urgent leukopheresis, administration of IV fluids, and cytoreductive therapy with hydroxyurea. Supportive measures, such as allopurinol, should also be considered to prevent tumor lysis syndrome.

Paige Selvey
Dr. Michael Schnaus

Cranial Abscess Masquerading As Hyponatremia

A 63-year-old man with pertinent past medical history of two prior ischemic strokes presented to the Emergency Department with progressive “weakness, fatigue, aches, nausea, and chills” for one week. Further history revealed similar episodes recurring intermittently over the previous two months, in addition to a poor appetite, night sweats, and weight loss of about 5-6 pounds. He also endorsed watery diarrhea with no blood or abdominal pain for the past 24 hours. On admission, laboratory analysis was remarkable for leukocytosis with a count of 28.2 X 109/L, thrombocytosis with a platelet count of 559 X 109/L, hyponatremia with a sodium of 132 meq/L, and acute kidney injury with a serum creatinine of 1.8 mg/dL. He was treated with 2L of normal saline and Zofran for the nausea with a working diagnosis of viral gastroenteritis. The following day the patient’s diarrhea resolved, creatinine improved, and cultures returned negative. However, he remained febrile with an ongoing leukocytosis and hyponatremia. On hospital day four the patient appeared ill with worsening leukocytosis and return of diarrhea. Further investigation with CT of the abdomen and pelvis showed a small perirectal abscess.
He was given Ciprofloxacin and Metronidazole, with surgery planned for the following day. Overnight, however, the patient became delirious and was given another 2L of normal saline. The next morning, the patient remained delirious and ill appearing.

Laboratory analysis demonstrated a serum sodium of 124 X 10^9/L. Further work up regarding his hyponatremia revealed a serum osmolality of 269, urine osmolality of 804, and urine sodium of 139 meq/L, with the patient appearing euvolesmic. Syndrome of Inappropriate ADH (SIADH) was diagnosed.

Notably, SIADH is a complex disorder caused by a variety of different pathologies and with a fairly broad differential. The acronym SIADH can be used to remember the most common causes: Surgery, Intracranial infections or injuries, Alveolar diseases or malignancy, Drugs (antiepileptic, antipsychotics, antidepressants), and Hormone disturbances (desmopressin, oxytocin, hypopituitarism, hypothyroidism), HIV, or hereditary. Although broad, keeping such a differential in mind when SIADH is diagnosed can help focus and narrow clinical thinking when the underlying diagnosis is not clear.

Given the differential for SIADH as well as change in his mental status, further workup with brain MRI was obtained identifying a small ring-enhancing lesion in the left parietal occipital lobe extending into the left ventricle representing an abscess, likely secondary to hematogenous spread from the perirectal abscess or from a subsequently identified dental abscess. Neurosurgery and colorectal surgery were consulted and the patient was brought urgently to the operating room for definitive therapy. This unique case of a patient presenting with intracerebral abscess causing SIADH demonstrates the importance of recognizing and identifying hyponatremia in the inpatient setting on a timely matter.

Rebecca Spurr
Dr. Annie Jacobsen

A Late Presentation of Vogt-Koyanagi-Harada Syndrome and the Necessity of Early Intervention for Acute Vision Loss

A 27 year old Somali-speaking female, recently immigrated from Uganda, was brought in by her roommates for several days of paranoia, refusing to take her medications, and psychotic symptoms. Her history was significant for bilateral panuveitis resulting in blindness since 2012, and sensorineural hearing loss (right > left) that developed over the course of a few weeks, several months prior to admission. She had recently been seen in clinic by ophthalmology and ENT and had received oral prednisone and 4 intratympanic steroid injections with no effect on hearing. Her most recent prednisone burst had ended about a month prior to presentation. Additional complaints prior to admission included headaches, dysphagia, and diffuse myalgias and arthralgias. The patient had been evaluated by Neurology shortly before admission and given a tentative diagnosis of incomplete Vogt-Koyanagi-Harada (VKH) syndrome, with urgent referral to Rheumatology.

Laboratory workup was negative for HSV, HIV, ACE, SPEP, and RPR. ANA was weakly positive and toxoplasma IgG was positive, though toxoplasma IgM was negative. A brain MRI obtained shortly before hospitalization showed chronic changes of bilateral phthisis bulbi.
consistent with her history of panuveitis, but was otherwise unremarkable. A CT of the chest and abdomen showed no organ or vascular abnormalities. Lumbar puncture demonstrated mildly lymphocytic and monocytic CSF that was acellular, with protein and glucose WNL and a negative gram stain. After infectious, toxic and metabolic causes were ruled out, she received intravenous methylprednisolone, 500 mg daily for 3 days. Her mental status improved, but a satisfying etiology of her acute psychotic symptoms has yet to be identified. She was discharged on a prednisone burst and Bactrim for PJP prophylaxis.

Bilateral panuveitis has a limited differential, including VKH, Behcet’s disease, sarcoidosis, Cogan’s syndrome, Alport syndrome, tubulointerstitial nephritis and uveitis, congenital rubella, and syphilis. VKH is a multisystem granulomatous inflammatory disorder affecting the eyes, auditory system, meninges, and skin. It is thought to involve a T-lymphocyte-mediated autoimmune process against an antigen associated with melanocytes. Our patient’s bilateral uveitis, retinal detachments, hearing loss, and reported skin changes and alopecia make VKH her most likely diagnosis.

Patients who present early in the course of this disease often do so with prodromal neurologic symptoms (headache, tinnitus, and meningismus) that precede vision loss. If identified early, aggressive steroid or immunosuppressive therapy can actually preserve vision in these patients and drastically improve long-term outcomes. Unfortunately for our patient, she did not receive early intervention and her vision and hearing are unlikely to recover. This case highlights the urgency of evaluation by a specialist in the setting of acute vision or hearing loss.

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Andrew Stone
Dr. David McDonald

Through a Murky Lens: Diagnosis and Treatment of Fungal Pneumonia in an Immunosuppressed Patient

Our patient was a middle aged man with bilateral lung transplant 21 years ago due to idiopathic pulmonary fibrosis, followed by kidney transplant 8 years ago due to nephrotoxicity from chronic calcineurin inhibitor use. He was chronically immunosuppressed on mycophenolate, tacrolimus, and prednisone.

The patient initially presented to the ED with 1 week of dyspnea, productive cough, and fever that had not improved with azithromycin and home oxygen. A chest XR showed bilateral patchy interstitial infiltrates, consistent with atypical pneumonia and a chest CT revealed numerous scattered lung nodules. He was given vancomycin and Zosyn in the ED and switched to ceftriaxone on admission.

Pulmonology and infectious disease were consulted and soon became suspicious for fungal pneumonia given the patient’s immunosuppression, lack of improvement despite appropriate antimicrobials, and the fact that areas of Minnesota are endemic for both histoplasma and blastomyces. A thorough travel history also revealed possible coccidioides exposure, as the patient had recently visited Nevada. Despite staying in an urban resort the patient and his wife, who had also developed milder URI-like symptoms, had been exposed to dust from nearby construction.
Antibacterials were stopped and he was treated with fluconazole for presumed coccidioidomycosis with improvement.

This case illustrates several points that complicate the diagnosis and treatment of coccidioides in the immunocompromised. For one, a definitive diagnosis of coccidioidomycosis has to this date still not been established. Serum antigen testing is typically only positive in patients with extensive infections, and sputum samples in this case were of poor quality and did not show fungal elements. Usual laboratory tests include enzyme immunoassay (EIA) tests for IgM or IgG antibodies or complement fixation (CF) tests. Because these tests involve antibody detection, they are less reliable in immunocompromised hosts. One retrospective study of 62 patients suggested that both EIA and CF have a sensitivity of ~67% for detecting coccidioides infection in immunocompromised patients, which improved to 82% if both EIA and CF tests were performed. In our case CF testing returned negative, while EIA and fungal cultures were pending at time of discharge.

Fluconazole and other azoles inhibit the cyp450 system (cypA34) and lead to higher levels of common immunosuppressants, including cyclosporine and tacrolimus. Our patient’s tacrolimus dosage therefore needed to be reduced, with close lab follow-up to monitor his levels.

The mainstays of coccidioidomycosis diagnosis remain the key elements of any infectious disease diagnosis: 1) a thorough history and physical 2) detailed travel history (including domestic) 3) attention to plausible exposures. While laboratory tests can play an important role, they are often slow to return and not particularly reliable in immunocompromised patients – the very individuals who are most at risk from fungal infections.

Background: Disseminated Herpes Zoster (HZ) is a clinical syndrome that presents with a vesicular rash in a wide dermatomal distribution and is often associated with immunosuppression. Even though there have been limited reports of disseminated cutaneous zoster in the absence of known immunosuppression, diagnosis in these cases can be challenging and lead to delay in treatment. We report a case of Disseminated HZ in an immunocompetent patient with Chronic Kidney Disease.

Case Presentation: A 63-year old patient presented to the hospital with a two-month history of worsening shortness of breath, lower extremity swelling, and fatigue. His past medical history was significant for chronic kidney disease (stage V) secondary to diabetes. He demonstrated capacity and consistent wishes against dialysis. Temporizing measures were used to stabilize electrolytes and reduce volume overload with the use of IV Furosemide, sodium polystyrene sulfonate and Sodium Bicarbonate. On admission, physical examination revealed a right lower lumbar hemorrhagic vesicular rash that the patient reported had been pruritic for the past week. During his second day of hospitalization, the rash had spread in a non-contiguous fashion to both his lower extremities to involve four separate bilateral lower-extremity dermatomes (T12, L1, L4, and L5). Bilateral, Non-contiguous, or
multiple dermatomes are associated with dissemination and rarely occur in immunocompetent individuals. Dissemination occurs in 2-10% of immunocompromised individuals. Recent testing for HIV, malignancy, HBV, and other viral causes had been negative. Clinically, the appearance of the lesion was most consistent with Herpes Zoster Virus. Initial skin swab for HZ PCR was negative, but on repeat testing PCR was positive. He was started on 14 day course of PO Valacyclovir. After three days of Valacyclovir (1g/day), his rashes began crusting and did not develop any new lesions. He was discharged from the hospital on Day 7 once his electrolytes had stabilized with close outpatient follow-up.

Discussion: Disseminated HZ is diagnosed when patients develop more than 20 blisters within a week of typical skin symptoms or a vesicular rash that affects more than three dermatomes. It is a clinical diagnosis, but the appearance and location may be atypical and require laboratory confirmation. PCR is more sensitive than viral cultures, but the sensitivity varies by swab. Disseminated HZ is most commonly seen in patients with HIV, Malignancy, or immunosuppression. Studies have reported that End Stage Renal Disease, Uremia, and Diabetes may contribute to an impaired host-immune system that may contribute to reactivation. Dissemination has an increased risk of visceral involvement, including life-threatening pneumonitis, ophthalmicus, and bacterial superinfection. A high-level of clinical suspicion and prompt identification and treatment with Acyclovir or Valacyclovir is of crucial importance, given infectiveness and high risk of morbidity and mortality.

Residents

| Sayed Obaid Aseem |  |
| Denis Whalen; Dr. Andrew Majka; Dr. Sanjeev Nanda |  |

**Quality Improvement - Residents**

**Improving Medication Reconciliation in Primary Care by Obtaining Pharmacy Records of Prescription Refills**

The Joint Commission has made medication reconciliation a national priority to reduce the harm caused by patients taking medications inappropriately. Medication reconciliation is the process of comparing and correcting errors between the patient’s prescribed medications and what the patient may be actually taking. This process can be divided into two distinct steps: first, generating an accurate list of medications the patient is taking; and second, a qualified health care provider determining that the medications are appropriately indicated and of appropriate dosage. The process relies heavily on the first step, which in the primary care setting often depends on patients being able to recall their medications correctly. In addition to being error prone, this step is also time consuming making it often impractical in acute visits. Clinical assistants could accurately and expeditiously generate a medication list by obtaining patients’ pharmacy records. Using this approach, we aimed to reduce the error rate in patient medication lists by at least 50% over a 12-month period. In our baseline data gathering stage, only 35.8% (19/53) of pharmacies randomly contacted were able to provide a record of medication refills. When we compared the pharmacy records to the
medication lists generated by current methods, 52.6% (10/19) of medication lists had an error. Of these, 36.8% (7/19) were errors of omission, which may be the easiest to recognize. These observations indicate that a national electronic registry of pharmacy records accessible to primary care providers may significantly improve medication reconciliation.

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<tr>
<th>Janewit Wongboonsin</th>
<th>Real-life barriers for prescription of appropriate dose statin for patient with Established cardiovascular disease (CVD)</th>
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<tr>
<td>Dr. Reut Danieli</td>
<td>Introduction: Secondary prevention with high-intensity statin has been repetitively shown to reduce CV events and all-cause mortality. However, adherence to statin was limited by multiple factors and was reported to be as low as 41.6% at 2 years after an acute myocardial infarction. This QI project aimed at exploring barriers of this issue in primary care setting provided by the resident. In our study, statin use on patients with CVD was evaluated and initiated if appropriate. Various barriers to not being on statins on these patients were identified.</td>
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<td>Method: Patients with CVD and their statin use were identified from VAMC clinic patient-panel of 9 Internal Medicine residents. Using the pre-existing panel database in CPRS (VA electronic medical record), we were able to determine the cross-sectional prevalence of appropriate statin amongst each individual at two different time points. Residents were encouraged by emails, face-to-face or letters to contact the patients to initiate statin. 8 months after launching the project, residents then reported their experience on such practice and identified barriers to appropriate statin prescription that they encountered.</td>
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<td>Results: Out of 741 patients, 177 (23.8%) had CVD. We demonstrated a significant increase in the use of statin (36% to 51%, p-value 0.03) by using simple conversational spread amongst our resident in conjunction with the aforementioned population-based clinical outcome-base feedback. Three main categories of barriers include a perception of the need for statin to be weak (48%), patient-related factors (43%) and effect from logistics of care (33%). Only 13% was accounted for side effect related to statin medication.</td>
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<td>Discussion: This project exemplified to use of population-based clinical outcome information that was available with most electronic medical record as a tool for improvement of the implementation of clinical guideline. As we identified top three common barriers to such practice, we then plan to come up with a long-term strategy that could lead to an automatic feedback to improve clinical practice amongst residents. We also found out that the side effect profile, which was known to be the most common barrier, was not the main contributor of statin prescription. Limitation of this study included a limit of prevalence calculation based on computerized data generated from problem lists panel.</td>
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<th>Nicholas Zorko</th>
<th>University of Minnesota Resident Primary Care Continuity Clinic</th>
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<td>Dr. Briar Duffy;</td>
<td>Prompt Notification of Patient Results: Strategies for Improvement</td>
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<td>Dr. Tanya Melnik</td>
<td>Communication is key to the doctor-patient relationship and impacts patient safety and satisfaction. Studies have demonstrated no notification in 75% of normal and 33% of abnormal results. Similar</td>
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issues were identified in the Internal Medicine resident clinic.

Our goal was to improve total results with documented patient notification in Epic and time to patient notification from final result. Time of final result, presence of a result note, and time of result note were compiled using pre-intervention, pooled, and paired resident result systems over the course of 3 PDSA cycles.

Mean time to result note between the first and third PDSA cycles decreased from 9.78 days pre-intervention to 0.98 days with paired intervention strategy (p<0.0001, 1-way ANOVA). Percent of results with notes increased from a mean of 50% in the pre-intervention group to 100% in the paired intervention group (p<0.0001, 1-way ANOVA).

This study showed significant increase in numbers of results with notes and decreased time to result note when comparing pre-intervention to paired result reporting strategies. Modifications in result reporting had the desired results, which may lead to improved patient satisfaction and safety.

**Research Residents**

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<tr>
<th>Rachel LaNasa</th>
<th>Demographics and Viral Suppression Rates of Case Managed HIV Positive Youth</th>
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<td>Dr. Julia Stumpf; Dr. Qi Wang; Dr. Meghan Rothenberger</td>
<td>Background: Youth and adolescents account for an increasing percentage of new HIV/AIDS diagnoses in recent years; in particular, young nonwhite men who have sex with men (MSM) are disproportionately burdened by HIV. The Youth and AIDS Projects (YAP) program provides comprehensive case management for HIV positive youth in the Twin Cities, serving a population largely comprised of nonwhite young MSM.</td>
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<td>Purpose: To explore the demographic makeup and viral suppression rates of YAP’s case managed cohort, and to evaluate if demographic differences exist between virally suppressed and non-virally suppressed clients.</td>
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<td>Methods: Demographic data were collected from a survey completed by HIV positive youth within the first year of case management by YAP. HIV specific data including viral load, medication regimen and CD4 count were obtained with client permission from medical clinics.</td>
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<td>Results: 48 clients were enrolled in YAP case management between 2014-2017; 67% identified as MSM and 86% were nonwhite. All reported income below the state poverty level; 52% endorsed a mental health diagnosis. All were on antiretroviral therapy, and 65% were on single pill regimens. 75% achieved viral suppression within the year of case management initiation. Non-virally suppressed clients were more likely to identify as homeless (p = 0.03), and were less likely to have received counseling for substance abuse in the past (p = 0.04); no other statistically significant differences existed between virally suppressed and non-virally suppressed clients.</td>
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<td>Conclusions: 75% of HIV positive youth case managed by YAP</td>
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achieved viral suppression, compared to 44% of youth nationwide (per CDC data). This high rate of viral suppression was achieved despite YAP’s clients having multiple complex psychosocial needs that may hinder antiretroviral adherence. These results highlight the importance of age appropriate, culturally sensitive comprehensive case management for HIV infected youth because not only does viral suppression benefit clients, it also has implications in decreasing risk of additional virus transmission within a community disproportionately burdened by HIV. Non-virally suppressed clients were more likely to identify as homeless than virally suppressed clients, suggesting that meeting basic needs may increase medication compliance and eventual viral suppression.

Implications for Practice: This data suggests that comprehensive case management of HIV positive youth is associated with high rates of viral suppression amongst a vulnerable population.

Bryce Mikel
Dr. David Tierney;
Dr. Lisa Kirkland;
Dr. Larissa Stanberry
Dr. Michael Samara;
Dr. Joseph Huguelet;
Dr. Roman Melamed

Measured vs. Calculated Oxygen Consumption in Critically Ill Patients

Introduction: Accurate determination of the cardiac output (CO) in critically ill patients is a challenge. The Fick CO is calculated using the arteriovenous oxygen content difference and the oxygen consumption (VO2). The arterial and venous oxygen content are directly measured, while the VO2 is a constant calculated using the patient’s body surface area (BSA). However multiple clinical states exist that impact a patient’s basal metabolic rate and VO2. We hypothesize the measured VO2 will vary from the calculated VO2 in multiple clinical states.

Methods: This was a prospective observational study performed on critically ill patients. Inclusion criteria were admission to the ICU, intubation, and pulmonary artery catheterization. 10 patients were included, amongst these 5 were measured during therapeutic hypothermia. Fick CO was obtained per treatment team discretion using both BSA-based calculated VO2 values and measured VO2 via indirect calorimetry.

Results: 28 pairs of calculated and measured VO2 values (median 3 measurements per patient) were recorded and analyzed. BSA-based calculated VO2 values remained stable for each patient while the measured VO2 varied considerably. The mean absolute difference between the BSA-based calculated VO2 and measured VO2 was 55.1 +/- 40.8 mL/min, coefficient of variation = 74, 95% CI [39.3,70.9]. Measured VO2 was consistently higher than the calculated VO2 in patients who were agitated, shivering, or febrile. Amongst the hypothermic subset of patients, measured VO2 was consistently lower than the calculated VO2 with a gradual rise during rewarming.

Discussion: Our study demonstrates wide variability of measured VO2 as demonstrated by the large coefficient of variation. This variability suggests the BSA-based calculated VO2 may not yield an accurate CO in the ICU patient population, especially in the setting of therapeutic hypothermia, fever, shivering, agitation. Clinicians should interpret Fick CO values based on the calculated VO2 measurements with caution in these settings. Though indirect calorimetry may yield a more accurate VO2 measurement, it is time consuming and requires technical expertise. Larger studies investigating the clinical value of measured VO2 in hemodynamic management are warranted.
| Nasreen Quadri  
Dr. Ann Settgast | Elevated Blood Glucose Prevalence in Newly Arrived Refugees |
---|---|
**Background/Purpose:** Data currently suggests pre-departure prevalence of diabetes in refugee populations is roughly 2-3% and typically diagnosed in symptomatic individuals. Other studies of diabetes prevalence in this population has been estimated between 3-8%. The prevalence is important in recognizing future risk for cardiovascular disease and complications of diabetes. This risk increases over time as lifestyle changes after US arrival as early as one to five years after resettlement.

**Objective:** To describe the prevalence of elevated glucose in newly arrived refugee patients and correlate associations with demographic data.

**Methodology:** This was a retrospective chart review of the electronic medical record of 2,332 refugee patients from May 2009 to February 2016 looking at markers of elevated glucose (random glucose from basic metabolic panel, hemoglobin A1c) and demographic information. (age, sex, language as proxy for country of origin, BMI).

**Results:** Roughly half of the patients in this study were male (52%). Of the 2,332 newly arrived refugee patients undergoing new arrival screening, 1.8% screened positive for elevated glucose defined as glucose > 199 mg/dL. The average positive screen glucose was 313 mg/dL and the average hemoglobin A1c was 9.4%. About 95% of the positive screen group were under the age of 65. When compared to other refugee populations in the study, Karen/Burmese speakers had a lower prevalence of elevated glucose (1.1%, p = 0.01) and Bhutanese Nepali speakers had a higher prevalence (4.1%, p = 0.005).

**Conclusion:** Though the prevalence in the this study population (1.8%) was lower than other estimates (2.3%), it was in accordance with the varied prevalence from 2.5% to 14% based on country of origin as demonstrated in other studies. These data suggest universal blood glucose screening of new arrival refugees as part of the domestic medical examination can be important to diagnose and treat prediabetes and diabetes to prevent morbidity and mortality.

| Christina Thaler  
Reinie Thomas;  
Dr. Michael Miedema;  
Dr. Barry Maron | Clinical Recognition of Hypertrophic Cardiomyopathy in a Population-Based Study of New Ulm, Minnesota |
---|---|
**Background:** The prevalence of hypertrophic cardiomyopathy (HCM) is estimated to be 1:500 adults. However, there is little data detailing the
clinical recognition of HCM in a real-world population that has not undergone systematic imaging or genetic screening.

Methods: A cross-sectional analysis was performed using electronic health record (EHR) data from the rural Midwestern town of New Ulm, Minnesota (population ~14,000), a population served by a single health care system with a unified EHR. Data was extracted for individuals aged > 18 years who resided within the New Ulm zip code with clinical encounters from 2005-2014 and a documented ICD-9 code potentially consistent with HCM. A broad range of ICD-9 codes were examined, including codes for all cardiomyopathies, congestive heart failure, or left ventricular hypertrophy. Individual chart review was then performed on individuals with relevant ICD-9 codes to determine if a diagnosis of HCM could be made.

Results: Of the 14,540 individuals with clinical encounters 436 had one of the targeted ICD-9 codes, 143 of which had a ventricular septal thickness > 14mm on echocardiography or cardiovascular magnetic resonance imaging. Of those individuals 14 cases of probable or definite HCM were identified, age 60 +/-16 years; 10 were male. Only one patient < 30 years of age had clinically recognized HCM. Septal thickness was 18 +/- 5mm (range 1.2 - 2.4). Two patients had prophylactic implantable defibrillators for high risk of sudden cardiac death. Six patients had evidence of left ventricular outflow tract obstruction (gradient >30mmHg at rest), 2 of whom had undergone surgical myectomy. The rate of clinical recognition of HCM was approximately 1:1,000, one-half of the estimated prevalence of HCM in the general population.

Conclusions: In a population-based study utilizing EHR data, clinical recognition of HCM was about 50% of its estimated prevalence. These data underscore the principle that heightened awareness and population-based strategies to optimize identification of HCM are needed.

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**Clinical Vignette- Residents**

Mohamad Adada  
Dr. Gregory Stroh;  
Dr. Linh Vu;  
Dr. Jeffrey Geske

*A Heart Made of Stone: Rethinking the Cause of Ascites*

Constrictive pericarditis is a reversible cause of heart failure and can be missed in patients with lower extremity edema and ascites. Following diagnosis, an investigation into the underlying etiology should be undertaken. A high index of suspicion is required to recognize these patients and provide appropriate treatment.

A 55-year-old woman had a five-year history of recurrent ascites requiring increasingly frequent, large volume paracenteses and progressive dyspnea. Given normal baseline hepatic function, her symptoms were thought to be idiopathic. She was treated with diuretics and paracenteses as needed. Due to progressive symptoms, she presented for further evaluation.

Physical exam in the upright position revealed prominent jugular venous distension and distended vessels of the forehead, ascites, pitting lower extremity edema extending to the sacrum, and a pericardial knock. She had no history of significant infections or rheumatologic disease.
Cardiac imaging studies showed evidence of constrictive pericarditis. Transthoracic echocardiography showed a normal left ventricular ejection fraction and no significant valvular disease. There was evidence of ventricular interdependence, respiratory variation of tricuspid and mitral inflow, a plethoric inferior vena cava without inspiratory collapse, and expiratory diastolic hepatic flow reversals. Cardiac MRI confirmed ventricular interdependence and demonstrated diffuse pericardial thickening.

She subsequently underwent right and left heart catheterization which showed non-occlusive coronary disease, circumferential pericardial calcification, and equalization of diastolic pressures in all cardiac chambers (right atrial pressure 40 mmHg, right ventricular pressure 100/36 mmHg, pulmonary capillary wedge pressure 40 mmHg, and left ventricular pressure 100/40 mmHg).

Cardiovascular Surgery was consulted and she underwent a complete pericardiectomy. At the time of initial pericardial incision, the heart was seen to “leap” out of constraining pericardium. Post-operative right atrial and pulmonary artery systolic pressures dropped to 6 mmHg and 37 mmHg, respectively. Systemic systolic pressure increased from 90 mmHg to 130 mmHg. Thereafter, her symptoms were controlled with oral diuretics and she was discharged home on post-operative day 23.

There are many known etiologies of constrictive pericarditis, including post-cardiac surgery inflammation, radiation, infectious pericarditis, and connective tissue disease. However, the vast majority of cases are idiopathic, as seen in this patient.

Pericardiectomy is the definitive treatment for constrictive pericarditis, although carries significant perioperative morbidity and mortality. Complete removal of the calcified and inflamed pericardium is necessary but can be technically difficult, and outcomes have been shown to be significantly better at high volume centers.

This patient’s history, exam, echocardiogram, MRI, and catheterization were all classic for constrictive pericarditis, a potential cause of cardiac ascites. Constrictive pericarditis is a reversible cause of heart failure, and, therefore, it is critical to recognize this entity in order to provide patients definitive and potentially curative treatment.

**Hamna Ahmad**  
Dr. Mark Sprenkle

*It Can Always Be TB*

Bronchial anthracofibrosis (BAF) is a term of relatively recent origin which refers to “black discoloration of bronchial mucosa with bronchial narrowing” previously described only in the setting of pneumoconiosis and biomass fuel exposure. This was first reported in association with TB in 1998 and several cases have been reported since then.

70-year-old female, immigrant from Mexico, presented with worsening shortness of breath, intermittent productive cough and gradual weight loss for a few months. Patient had a remote history of minimal exposure to tobacco however significant history of biomass smoke exposure. She
also had a positive T spot and was treated with Isoniazid for latent TB previously. Crackles were heard on right lung base. CXR was significant for right basilar infiltrate with mild right hilar prominence. CT re-demonstrated the infiltrate and showed right middle lobe bronchus obstruction with right middle lobe atelectasis and narrowing of right lower lobe bronchus along with significant mediastinal fibrosis. Narrowing of right pulmonary artery and right pulmonary veins was also noted. Histoplasmosis serologies were negative- mediastinal fibrosis is a common manifestation. Anthracosis from biomass fuel exposure and BAF in correlation with TB were among the considerations and bronchoscopy with EBUS was performed that showed extensive anthracosis with significant airway narrowing consistent with BAF. AFB culture was positive on the bronchoalveolar lavage and lymph node.

While biomass fuel exposure and smoke inhalation can be a potential culprit, it does not explain the sub-acute onset and pulmonary infiltrates. Moreover, relationship of BAF with TB has been described in the literature and it is likely the case in our patient. RML bronchus tends to be the most commonly involved site, as seen in our patient. This case illustrates the importance of considering TB as a differential for lymphadenopathy and bronchostenosis in more than one lobe/segment. As our patient had no evidence of TB on CXR that does not completely rule out the possibility of having an active TB infection.

Hasan Albitar
Dr. Allison Ducharme-Smith;
Dr. Saira Farid;
Dr Rizwan Sohail

Rhabdomyolysis Associated with Anaplasma Phagocytophilum Infection in an Immunocompromised Patient

Rhabdomyolysis has been associated with multiple infections. However, it is an exceedingly rare complication of Anaplasma phagocytophilum infection. We report a case of Anaplasma phagocytophilum infection in an immunocompromised patient with severe rhabdomyolysis resulting in renal failure, and death.

A 51 year-old male patient from northern Minnesota presented to the emergency department last July with fever and acute onset left leg pain of 2 days duration. His past medical history was significant for type 2 diabetes mellitus, hypertrophic obstructive cardiomyopathy, and supraglottic laryngeal squamous cell carcinoma treated with cisplatin, dexamethasone and radiotherapy. Physical examination revealed an elevated heart rate of 144, respiratory rate of 33, and temperature of 39 Celsius. He was also noted to have swelling and tenderness to palpation of the left thigh. Patient reported multiple tick exposure in the week preceding the acute illness.

Initial investigation revealed leukocytosis of 20.7 x10^9/L with 82% neutrophils, hemoglobin of 9.6 g/dl, platelet count of 76x10^9/L, serum sodium of 118 mmol/L, potassium of 6.5 mmol/L, AST of 2336 U/L, ALT of 721 U/L, total bilirubin of 1.5 mg/dl, creatinine of 6.0 mg/dl, pH of 6.88 with an elevated anion gap of 36, bicarbonate level of 5 mmol/L, and lactate level of 13.22. CT scan of the lower extremities showed diffuse enlargement of the muscles in the adductor compartment with thickening of the superficial and interfascial planes with soft tissue stranding in the surrounding subcutaneous tissues. His creatinine kinase (CK) was elevated at 114570 U/L with an aldolase of 186.5 U/L, and myoglobin of >5000 ng/mL, all consistent with rhabdomyolysis.
complicated by multi-organ dysfunction. General surgery team was consulted and surgical pathologies including compartment syndrome and necrotizing fasciitis were ruled out.

Blood cultures and tick-borne PCR panel were ordered and the patient was empirically started on Vancomycin, Cefepime and intravenous Doxycycline. Tick-borne PCR panel was reported positive for Anaplasma phagocytophilum. Treatment with intravenous doxycycline was continued. Despite maximum supportive therapy and appropriate antimicrobial coverage, the patient continued to deteriorate. This precluded tissue biopsy and culture from being obtained. 1 day later, the patient expired due to progressive multi-organ failure.

Tick-borne infections should be considered in patients living in endemic areas presenting with fever, thrombocytopenia, and elevated liver enzymes during summer or early fall. Anaplasma phagocytophilum is the causative pathogen of human granulocytic anaplasmosis. It is transmitted by Ixodes scapularis. Patients presenting with severe sepsis with evidence of multi-organ dysfunction and soft tissue infection should always be evaluated for possible necrotizing fasciitis or myositis. Mortality secondary to Anaplasma phagocytophilum infections is rare, however; higher rates of complications and mortality are observed in immunocompromised patients. Given the potential for severe and fatal disease, a therapeutic trial is indicated once the disease is suspected.

Matthew Alcasid
Dr. Erica Lin;
Dr. Luke Law;
Dr. Paul Daniels

Gemcitabine-induced Radiation Recall Myositis

Radiation recall, a well-studied but poorly understood phenomenon, refers to an acute inflammatory reaction in previously irradiated tissue that develops after exposure to an inciting agent. Various medications have been implicated, including chemotherapeutic agents such as gemcitabine. An 80 year-old female with metastatic urothelial cancer status-post right nephro-ureterectomy, radiation and on current chemotherapy with gemcitabine presented with intractable left pelvic and hip pain with weakness. Regarding her oncologic history, she was diagnosed with right urothelial carcinoma two years prior to presentation with which she underwent radical right nephro-ureterectomy with subsequent radiation. Follow-up CT two years later showed recurrence and she subsequently underwent three cycles of gemcitabine with carboplatin. On presentation she was afebrile and hemodynamically stable. On physical exam, abdomen was tender in the left lower quadrant without peritoneal signs, there was tenderness in the left inguinal area and she had proximal weakness of the left lower extremity. Laboratory studies were notable for elevated CRP 95.4, ESR 62 and CK 247 in addition to mild leukocytosis. Broad-spectrum antibiotics were initiated empirically given concern for infection in the setting of an immunocompromised host. A CT of the abdomen and pelvis was performed, which demonstrated hypoenhancement of the left rectus abdominis, psoas, iliacus, iliopsoas, and sartorius muscles, consistent with myositis and/or myonecrosis as well as surrounding subcutaneous cellulitis initially concerning for possible necrotizing fasciitis. General surgery was consulted, however, given an LRINEC score of 2 it was felt this was unlikely and she ultimately did not undergo surgical intervention. Additionally, an MRI of the pelvis was performed, which confirmed the findings on CT. Patient remained stable throughout her hospital course, and so antibiotics were eventually discontinued. Given
lack of evidence of a rheumatologic condition, Medical Oncology was consulted, and they were concerned for gemcitabine-induced radiation recall myositis, given the localization of the myositis with regard to her previous radiation exposure. Of note, patient was on simvastatin, but this was thought less likely to be the causative agent given lack of widespread myositis. She was initiated on empiric IV steroids with eventual transition to oral prednisone; further gemcitabine was withheld. During her hospitalization, her pain and strength improved and she was eventually discharged to a skilled nursing facility. With the increasing use of certain chemotherapy agents, providers who routinely care for patients with malignancies should be familiar with uncommon complications including gemcitabine-induced radiation recall. Additionally, this case highlights that even in the setting of an immunocompromised host, it is important to keep drug reactions in the differential for inflammation in what would otherwise be assumed to be an infectious process. Awareness of the features of this condition will facilitate early diagnosis and appropriate management, as management is drastically different from other leading diagnoses.

Kamran Ali

*Plane as a Vector: A Case of Falciparum Malaria*

A previously healthy 42 y.o. male who came in to the hospital with fever, chills and general malaise for the past 7 days. Patient also reported headaches, dark urine and nausea. On examination patient had icteric sclera. He had mild tachycardia and fever of 38.1 degree Celsius. Laboratory investigation revealed normocytic anemia of 11, platelet of 46 and mildly elevated LFTs with Direct Bilirubin of 2.2. Urinalysis showed proteinuria without pyuria. Patient’s dropping hemoglobin and platelet count was investigated with peripheral smear which was positive for parasites. Rapid malaria detection test was done which was positive for plasmodium falciparum. This was further confirmed with thick and thin smear. His parasitemia count was 6%. Further investigation revealed patient is originally from west Africa and last travel there was 2 years ago. No history of blood exchange or IV drug use. He works at an international airport as a technician. 2 weeks ago he helped a lady arriving from Senegal who had fever and was persistently vomiting. Patient was treated with atovoquone-praguanil for 3 days and 2 weeks later, patient reported resolution of headache, fever and nausea. He had resolution of thrombocytopenia and his LFTs were improving. His repeat peripheral smear didn’t show any parasitemia.

In the US, rare cases of outbreaks of locally transmitted mosquito-borne malaria have occurred; in such cases, local mosquitoes bite infected person travelling from endemic areas and transmit to local residents. Other methods of transmission includes mosquitoes carried in the plane from endemic areas and infecting local residents. Multiple case studies have reported people being infected within the perimeter of international airports. This case highlights the importance of keeping a high level of suspicion of malaria in people with no recent travel history but living/working in close proximity to international airports. Timely diagnosis and appropriate anti-malarial therapy is key to reducing complications and mortality in falciparum malaria infection.
Pancytopenia Secondary to Zinc Induced Copper Deficiency from Vitamin Supplementation

Dietary vitamin supplementation has become increasingly common in children and adults. This case emphasizes the importance of reviewing over the counter dietary supplementation and keeping a broad differential in mind when approaching pancytopenia.

A 58 year old male presented to the emergency department with progressive shortness of breath. The patient noted that his exercise tolerance had been decreasing progressively over a 1 month period, to the point where his family noted he was unable to ambulate in his house, and eventually prompting a call to 911. The patient’s medical history included GERD and unintentional weight loss of 20 lbs. Medications noted by the patient included three vitamin supplements that he took daily. His family history was notable for the death of his father in his 50s due to myelodysplastic syndrome. He had no significant cardiac history or chest pain. Initial work up included a basic metabolic panel, and a complete blood count, which was notable for hemoglobin of 2.8, a white blood cell count of 2.0, and a platelet count of 141. He was emergently treated supportively including NC oxygen and transfusion of pRBC. Once symptoms were improving and he was stabilized, work up progressed including a peripheral blood smear and eventual bone marrow biopsy demonstrating possible multilineage dysplasia with concern for myelodysplastic syndrome. LFTs were check and noted to be elevated to the 400 range in a hepatocellular pattern. In work up of liver enzyme elevation a ceruloplasmin level was checked and came back at < 3.0. This triggered review of micronutrients including copper, Zinc, B12, and folate. Patient was noted to have a toxic level of Zinc at 257 ug/dl and a low level of copper at 7 ug/dl. After a two month period of stopping vitamin supplements and targeted repletion of copper the patient’s cytopenias resolved. Zinc induced copper deficiency leading to pancytopenia in case reports has been mostly defined in the pediatric population (given vitamin supplementation had been more prevalent in pediatrics). With the recent boom in the herbal and supplement industry this presentation is likely to become more prevalent as patients use vitamin loading to treat many ailments.

This case demonstrates the potential deleterious effects of over supplementation of vitamins and the importance of a complete history. Although Zinc induced copper deficiency from over supplementation is rare, adverse effects of over supplementation are becoming more common as the herbal and supplement industry grows. It is important for physicians to approach pancytopenias with a broad differential. This case also demonstrates the importance of a thorough medical history including the discussion of over the counter medications and supplements. Ultimately, like with many things, moderation is key.

An Unusual, Non-Gastrointestinal Cause of Abdominal Pain in a 44-Year Old Woman

CLINICAL CASE: A 44-year old woman presented to the Emergency Department (ED) with 12 hours of severe abdominal pain. Her medical history is notable for coronary artery disease status post two
percutaneous transluminal coronary angioplasties (6 and 8 years prior to admission, respectively; on clopidogrel and aspirin), and medically complicated obesity status post Roux-en-Y gastric bypass surgery complicated by gastrojejunal perforation requiring exploratory laparotomy (3 months prior to presentation). She had weight loss of >25% body weight in the past year with decreasing muscle mass.

On the morning of presentation, she felt an acute onset of severe left lower quadrant abdominal pain upon turning in her bed. The pain was preceded by a ‘popping’ sound originating deep in her abdomen/back with radiation to her left flank. In the ED, vitals were notable for a blood pressure of 77/59. Labs were notable for elevated lactate 4.6 mmol/L (normal 0.6-2.3 mmol/L) and acute anemia (Hgb 7.8 g/dL, down from Hgb 10.3 g/dL 2 days prior) and leukocytosis of 18.4 x10^9/L. Computed Tomography (CT) Renal Protocol (without IV contrast) did not reveal any acute pathology. She was admitted to the medical ICU for concern of septic shock secondary to a urinary tract infection.

In the presence of acute anemia, elevated lactate, hypotension and abdominal pain along with the history of chronic atherosclerotic disease, a CT abdomen with IV contrast was obtained to rule out mesenteric ischemia. Unexpectedly, a left rectus sheath hematoma was identified without signs of mesenteric ischemia. Clopidogrel was held and she was managed conservatively before being discharged home in stable condition on hospital day 7.

**DISCUSSION:** Rectus sheath hematoma (RSH) is a relatively rare condition caused by rupture of the epigastric arteries or damage to the rectus muscles. RSH can present with hypotension secondary to hemorrhage into the hematoma, abdominal pain, nausea, vomiting, fever and chills. The clinical presentation can mimic appendicitis, ovarian torsion, ureteral obstruction from nephrolithiasis, or urinary tract infection. While our patient did not report direct trauma, her risk factors for RSH included atherosclerotic disease, anticoagulation (dual antiplatelet therapy), and decreased muscle mass. This patient was treated conservatively as she was hemodynamically stable after fluid resuscitation. In cases of hemodynamic instability secondary to RSH, angiography with embolectomy or surgical ligation is often needed. This case illustrates the need for clinicians to maintain a broad differential for abdominal pain including gastrointestinal, renal, musculoskeletal, vascular, and gynecological etiologies. Furthermore the case demonstrates the utility of contrast-enhanced radiographic studies to assist with identification of vascular pathologies.

References: PMID: 25529279; PMID: 24267499

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<th>Tori Bahr</th>
<th>Present with Seizures, Leave with an Esophagectomy</th>
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<td>An 18 yo woman with documented history of poorly controlled asthma and recent laparoscopic Heller myotomy for achalasia now presents after first time seizure. The patient was in her usual state of health when she reported to her mother that she couldn’t “get air out” while breathing which didn’t respond to albuterol. This prompted her mother to start driving her to the Emergency Department and on the way she had a</td>
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seizure episode. She arrived to the OSH with unremarkable vital signs and was taken for an MRI of her brain. While there she had another seizure with bladder incontinence and was noted to be cyanotic requiring bagging for a short period of time. Given new onset seizures she was transferred to the University Hospital for further management on the neurology service.

On arrival the patient was hemodynamically stable and breathing comfortably on room air. Further history was obtained with parents reporting she had been having similar dyspneic episodes since the age of 18 months which were unresponsive to bronchodilators. In reviewing her spirometry she was found to have a fixed intrathoracic airway obstruction. Further chart review found CT chest imaging from prior to her myotomy showing diffuse wall thickening and dilatation of the esophagus, measuring up to 6.5cm in diameter at the level of the aortic arch. CT chest imaging was repeated showing stable dilation. Pathology was then contacted to review the previous myotomy pathology and after further staining and review the pathology report was changed from fragments of smooth muscle to leiomyomatosis. With this change a urinalysis was obtained showing hematuria and proteinuria, prompting further evaluation by genetics and subsequent diagnosis of Alport Syndrome.

This case highlights multiple levels of cognitive bias and the importance of taking a step back to look at the bigger picture when caring for patients with a long history of symptoms. Despite clearly having a fixed airway obstruction on spirometry flow volume loops for several years she was continually treated for asthma despite not responding to treatment. Further, this case emphasizes that pathology results, like all results in medicine, are not immutable and subsequent information can support a call to have the previous pathology slides reviewed.

Erin Batdorff
Dr. Christopher Kobe;
Dr. Janewit Wongboonsin

A Curious Case of Anchovy Paste

Introduction: Multi-organ abscesses are associated with high morbidity and can be clinically challenging to manage. We encountered a case of liver and lung abscesses in a healthy 24 year old presenting with right upper quadrant pain radiating to the shoulder who had an excellent clinical outcome due to treatment based on clinical suspicion.

Case description: A previously healthy 24 year old male with high risk sexual behavior and alcohol abuse presented with a 20lb weight loss, fevers, nightsweats, hemoptysis, right shoulder and abdominal pain. He presented to clinic a month prior to hospital admission with a benign physical exam. His labs were significant for CRP 140mg/L and ESR 88. CBC, CMP, and chest x-ray were otherwise unremarkable.

On hospital admission, patient was noted to be diaphoretic with a temperature of 38.6C, nontender hepatomegaly, and decreased breath sounds in right lower lobe. Initial labs were significant for Hgb 9.9g/dL and WBC 14.4 with neutrophilia. LFTs, lactate, and procalcitonin were within normal limits. CT of chest and abdomen demonstrated a 11.8x5.5cm complex gas fluid collection in the right lower lung and 10x7.9cm complex mass in the right lobe of the liver.
Patient underwent percutaneous aspiration of the liver mass which revealed thick pinkish-brown exudate. Gram stain was negative with numerous polymorphonuclear cells. Blood, sputum, urine, stool, and liver exudate cultures all came back negative.

Patient was treated empirically with parenteral ampicillin-sulbactam and metronidazole. He had a fast clinical response becoming afebrile within 24 hours. The patient was discharged with a plan for intravenous ertapenem and total 10-day course of metronidazole. Two days after discharge, serum antibody for Entamoeba histolytica returned elevated at 16 units.

Discussion: E. histolytica can cause intestinal or extra-intestinal disease. This pathogen is common in developing countries. However in the US, there has been a recent rise within people engaging in oral-anal sexual practices. Thus, it becomes especially important to attain sexual history in patients presenting with multi-organ abscesses. Stool testings, the most common routes of diagnosis, are usually negative. The best confirmatory test is the serum antibody against E. histolytica, which is positive >90% of the time during active disease. However, given that there is sufficient lag time to result, empiric antibiotics should be initiated solely based on clinical suspicion. The aspirate, if performed, is described as being acellular, proteinaceous “anchovy paste” like substance.

Conclusion: Maintaining a high level of suspicion for E. histolytica in high-risk sexual groups is important to making this diagnosis in a timely manner. Initiating immediate treatment with metronidazole to cover for possible E. histolytica infection is key to a quick recovery.

| Amanda Blanchard  | A Case of Amyloidosis Masquerading as Guillain- Barre Syndrome (GBS) |
| Dr. Amy Holbrook | |

Amyloidosis is a condition of abnormal extracellular protein deposition that can result in a wide range of clinical manifestations depending on type, location and quantity of deposition. Its wide range of presentations can make it a diagnostic challenge, particularly when it presents as another condition.

An 87-year-old female presented to her clinic with gradual onset of lower extremity (LE) burning pain/numbness and weakness that progressed over 4 months to include her upper extremities. On exam she was ambulatory with a walker and symmetrically weak in LE with decreased sensation bilaterally. A clinical diagnosis of neuropathy was made and she was referred to Neurology for further evaluation. Neurology preformed an EMG that revealed severe distal sensorimotor polyneuropathy with both axonal and demyelinating features consistent with Guillain- Barre Syndrome with motor axonal involvement. Further work up showed normal ANA, ANCA, TSH, A1C, lyme PCR, ACE level, paraneoplastic panel, and ESR/CRP. Treatment for GBS was initiated with physical therapy and gabapentin was prescribed for pain. A lumbar puncture was later obtained and CSF revealed a pleocytosis with oligoclonal banding. SPEP was obtained and showed monoclonal
gammopathy with urine immunofixation showing IgG Lambda monoclonal protein. She was referred to Oncology for further consideration of an occult neoplastic process. However, she continued to decline with minimal improvements from therapy and ultimately required a wheelchair due to severe weakness and pain. Eight months after initial presentation she developed severe LE edema and was hospitalized for anasarca with new diagnosis nephrotic syndrome; EKG showed low voltages, and an echocardiogram was obtained which revealed a severely thickened left ventricle with hyperdynamic systolic function and diastolic dysfunction. Her new constellation of findings was concerning for an infiltrative pathology. Oncology was consulted and a bone marrow biopsy was obtained which revealed amyloid deposition in vessel walls and 15% malignant plasma cells and the diagnosis of primary (AL) amyloidosis was made. Treatment options were considered, however, given her systemic involvement (cardiac, renal, neurologic), the damage was felt irreversible and treatment became comfort focused. She was started on oral steroids and diuretics and was discharged to a nursing facility. At the facility she declined further becoming bed bound and developed autonomic dysfunction with orthostasis. Ultimately, 2 months post diagnosis, she presented to the ED in cardiogenic shock (LVEF 10%); she was made comfort care and died.

This case illustrates amyloidosis’ insidious ability to present as another well-defined condition such as GBS. Recognition and treatment of amyloidosis early has the potential to delay systemic spread and prevent multi organ involvement with irreversible damage.

Kathryn Bratko
Dr. Anya Jamrozy

Ticks and Tickers: A Case of Lyme Carditis

Lyme carditis is a rare complication of Lyme disease that can present with variable and fluctuating arrhythmias due to disease effects on the AV node. Recognition of Lyme as the causative agent in an arrhythmogenic process can require a complex synthesis of history, clinical factors, and lab data but ultimately can prevent both unnecessary invasive intervention and further Lyme complications. An 87 year old female with history of AVNRT s/p slow pathway ablation 9 years prior presented with new weakness and falls to the emergency department at a small hospital and was found to be in atrial fibrillation with rapid ventricular response. She underwent TEE-guided cardioversion but reverted to atrial fibrillation within 24 hours so was started on amiodarone.

Thereafter she experienced witnessed syncope twice with prolonged sinus pause triggering brief CPR resuscitation. Amiodarone was stopped and she was transferred to a tertiary care hospital for consideration of pacemaker placement. On the day of transfer, she unexpectedly developed a fever to 102°F and was started on broad spectrum antibiotics. Additional history revealed that during the summer she resides in a cabin on Pelican Lake north of Brainerd, MN, and had been experiencing fevers, malaise and anorexia for 2 weeks prior to admission. She is an avid gardener and reported finding multiple unattached ticks on her skin though denied history of tick bite or rash. Her ELISA screen for Lyme was positive and the Western Blot showed multiple positive IgM bands to confirm the diagnosis. Anaplasma PCR
was also positive. She was started on IV doxycycline. She was monitored on telemetry showing 1st degree AV block and, due to concern for underlying conduction disease, she underwent pacemaker implantation. This was likely a case of Lyme carditis and illustrates the variable presentation of arrhythmia in this disease. Lyme carditis represents a unique overlap of two common clinical scenarios -- arrhythmia and tick-borne illness. Even in older patients with history of arrhythmia, as in this case, Lyme should remain on the differential especially as treatment is usually well-tolerated and curative. While there are no large controlled trials involving Lyme carditis, case reports and series show that it most commonly causes varying degrees of heart block including complete heart block and even rare cases of asystolic sinus pauses. Lyme carditis is usually mild and self-limited but should be treated with appropriate antibiotics. In general, complete heart block usually resolves in one week and other arrhythmias within 6 weeks; nearly all patients have complete recovery. Transvenous or permanent pacemakers should be limited to patients with significant hemodynamic instability or other indications.

Courtney Burnett
Dr. Camille Robichaux

Acute B-cell Lymphoblastic Leukemia Presenting as Chronic Back Pain in a Young Woman

Acute B-cell lymphoblastic leukemia (B-ALL) is a rare lymphoid neoplasm typically seen in childhood but occasionally diagnosed in adulthood, most commonly seen in males. Presentation usually includes pancytopenia and the B-symptoms of fever, night sweats, and weight loss. In up to 50% of adults, presentation includes lymphadenopathy, hepatomegaly, and splenomegaly. The unusual presentation of B-ALL as chronic back pain in a young adult female makes this case an interesting diagnostic challenge.

A 35-year old female with no known past medical history developed low back pain without inciting trauma. She presented to the emergency room and was diagnosed with musculoskeletal pain secondary to discomfort from sleeping on an old mattress. She was treated conservatively with ibuprofen, acetaminophen, cyclobenzaprine, and amitriptyline. One month later, she developed bilateral lower leg weakness and presented to a general medicine clinic. X-ray of the lumbar spine was normal. A few weeks later, she had an acute, self-limited episode of right upper arm numbness and pain. Her primary care provider referred her to specialty clinics in pain, neurology, and orthopaedic surgery. Neurontin, tapering steroids, and trazodone were added to her pain management regimen. At this time, laboratory analysis showed an elevated WBC count of 15, a low vitamin D level of <4, and an elevated HgA1C of 6.6. Other laboratory results, including hemoglobin, platelets, folate, B12, TSH, and lyme antibody were unremarkable. MRI brain was normal. Symptoms improved with prednisone, but recurred once prednisone was stopped. The patient developed new symptoms including headache and joint pains in knee, elbow, and shoulder. Rheumatoid factor and ESR were not elevated. Patient was referred to rheumatology and mental health for new depression and was recommended daily exercise and a healthy diet. One month later, patient returned to clinic with “throbbing, beating pain with episodes of electricity shooting throughout her whole body.” She was continued on prednisone. MRI of the lumbar spine and
sacrum/coccyx were performed. Findings included “diffuse marrow signal abnormality, homogenous with replacement of fatty marrow,” along with multiple bilateral bone lesions concerning for bone infarcts. This was initially concerning for multiple myeloma or infiltrative disease.

The patient presented to our hospital around 4 months after symptoms began with intractable pain that had acutely worsened overnight. There were minimal findings on CBC apart from slight thrombocytopenia. Blood smear revealed 3% blasts and rare Auer rods. Flow cytometry was suggestive of acute leukemia, but extensive cell death made it difficult to delineate cell lineage. Bone marrow biopsy was performed and was consistent with B-cell acute lymphoblastic leukemia: 100% cellularity, 70% effacement by blasts with significant necrosis. Blasts were positive for CD34, CD10, CD20, dim CD22, CD79, TdT. FISH was positive for Ph translocation. The final diagnosis was Ph+ B-ALL.

Fiorela Calderon Sandoval

_A Case of Asymptomatic Leukocytosis; A Silent Reminder of a Rare Biliary Disorder_

INTRODUCTION: Gallstone ileus is an infrequent disorder that complicates 2-3% of all cases of cholelithiasis. It results in relatively high rates of morbidity and mortality in those patients who evolved into a mechanical bowel obstruction. This disorder presents predominantly in older women and is secondary to the presence of a biliary enteric fistula that allows a gallstone into the ileum with resultant impaction. About 60% are cholecystoduodenal fistulas and the rest is divided between cholecystocolonic and gastric fistulas. CT of the abdomen is the diagnostic modality of choice and treatment is an enterolithotomy. Low risk patients may undergo a definitive biliary procedure at the same time but for those at high risk the complete surgical intervention is not generally the approach. The result is a persistent fistula, an open window to future events: such as cholecystitis, cholangitis among some others…

CASE DESCRIPTION: A 75 year old African American female with past medical history significant for hypertension, hyperlipidemia, multinodular goiter and osteoarthritis comes in with a three day history of emesis, diffuse abdominal pain and constipation. A mechanical SBO is suspected so a CT scan is obtained. Evidence of cholelithiasis, pneumobilia and a cholecystoduodenal fistula with an obstructive stone is seen, confirming the rare diagnosis of Gallstone Ileus. Patient underwent exploratory laparotomy with enterolithotomy only. After several months of close follow up secondary to post surgical complications (including an abdominal abscess and wound dehiscence) the patient is seen four years later at the Medicine Clinic. A total right knee arthroplasty is planned soon so basic pre operative labs are ordered. Review of symptoms is negative. No fever is present and the physical exam is unremarkable, but a slight WBC elevation is noticed. On repeat tests mild leukocytosis persists, now with new aminotransferases elevation and possible history of chills. No abdominal pain or jaundice is present. Considering the PMH a CT of abdomen is obtained: the common bile duct is dilated to 0.8 cm compared to 0.5 cm 4 years ago. ERCP confirms the presence of asymptomatic choledocolithiasis, sphincterectomy and stenting was performed.
DISCUSSION: This interesting case illustrates a rare biliary disorder that interposes both the surgical and medical settings. A female with previous history of Gallstone Ileus develops four years later a choledocholithiasis that is diagnosed after an incidental finding of asymptomatic leukocytosis. A high level of suspicion and curiosity is required in the absence of abdominal symptoms or initial altered liver function tests in those high-risk patients (ASA Class III or IV) in which a biliary surgery was not possible. Always keeping in mind that the real medicine extends further than what is described in the medical textbooks.

Joy Chen
Dr. Ashley Egan;
Dr. Eric Olson;
Dr. Robert Vassallo

A Rare Cause of Diffuse Alveolar Hemorrhage in a Middle-Aged Man

Introduction: Diffuse alveolar hemorrhage (DAH) is a medical emergency that can cause acute respiratory failure and death. DAH can result from a variety of conditions that affect the alveolar capillary surface resulting in bleeding into the alveolar space. Clinical presentation includes hemoptysis, anemia, and pulmonary infiltrates on imaging. Our case describes diffuse alveolar hemorrhage due to idiopathic pulmonary hemosiderosis in a middle-aged man.

Case: 57-year-old man with history of clear cell renal cell carcinoma status post right nephrectomy and a 25-pack-year smoking history presented to the hospital with progressive dyspnea, fatigue, and anemia over the past two years. He was recently hospitalized at an outside facility and empirically treated with antibiotics with poor clinical response. The patient underwent surgical lung biopsy and was treated for presumed histoplasmosis. His symptoms persisted, and he developed unintentional weight loss. He denied cough, hemoptysis, fevers, or chills. Over the preceding six months, he received 14 red blood cell transfusions and intravenous iron infusion for refractory anemia. Upon current admission, he required four liter oxygen by nasal cannula. Heart and lung exam were normal. Laboratory investigation was notable for microcytic anemia (hemoglobin 6.8 mg/dL). His chest CT showed diffuse basilar predominant ground-glass opacities with interlobular septal thickening. Bronchoscopy revealed a significant amount of hemorrhage coming from the entire tracheobronchial tree. Bronchoalveolar lavage showed marked increase in hemosiderin laden macrophage count (100%). Autoimmune workup including ANA (antinuclear antibody), anti-GBM (glomerular basement membrane) antibody, and ANCA (antineutrophil cytoplasmic antibody) were negative. Review of the prior surgical lung biopsy showed diffuse chronic alveolar hemorrhage without capillaritis or vasculitis. The patient was diagnosed with diffuse alveolar hemorrhage secondary to idiopathic pulmonary hemosiderosis. He was started on oral prednisone with successful weaning of supplemental oxygen therapy. His hemoglobin remained stable without subsequent need for transfusion.

Discussion: Idiopathic pulmonary hemosiderosis (IPH) is a rare condition that predominantly affects children. It is a diagnosis of exclusion established after ruling out other possible causes of diffuse alveolar hemorrhage. Histopathologic findings show alveolar hemorrhage with hemosiderin accumulation. The diagnosis is often delayed as in this case. Pathophysiology of IPH is unclear. First line
therapy for IPH is systemic corticosteroids. Approximately one quarter of patients will have recurrent disease that can lead to pulmonary fibrosis. Rarely, IPH presents with unexplained persistent iron deficiency anemia and lung infiltrates due to alveolar hemorrhage. Hemoptysis may be absent, as in this case, and bronchoscopy together with bronchoalveolar lavage, as well as surgical lung biopsy are needed for definitive diagnosis.

**Supavit Chesdachai**

**Dr. Joseph Thurn**

**Persistent Abdominal Pain for One Year after Appendectomy**

Crohn’s disease is a transmural inflammation of the gastrointestinal tract. Commonly affected areas include the distal ileum and proximal colon. Manifestations may vary, but abdominal pain, diarrhea and systemic symptoms such as fatigue are typical. Disease involving the appendix has usually been misdiagnosed as acute appendicitis. We report a case of Crohn’s disease involving the appendix.

A 24 year-old male with a medical history significant for traumatic brain injury presented to the ED 2 days after the acute onset of right lower quadrant (RLQ) pain. He was diagnosed with acute appendicitis. Abdominal CT scan demonstrated a perforated appendicitis with developing abscess in the RLQ. He was treated with a dose of intravenous ertapenem followed by oral ciprofloxacin and metronidazole for 10 days. Laparoscopic appendectomy was done 4 months after initial diagnosis. Pathology showed chronic appendiceal and peri-appendiceal inflammation with non-necrotizing granulomatous reaction. Starting two months after surgery, the patient returned to the ED 3 times due to persistent RLQ pain. The pain was described as being dull but severe and unaccompanied by diarrhea, nausea or vomiting. Abdominal x-ray, ultrasound and CT scan gave no definite diagnosis. One year after surgery, due to worsening abdominal pain the patient was hospitalized. Repeat abdominal CT scan done on admission revealed marked abnormal wall thickening involving a long segment of distal ileum with tethering of adjacent small bowel loops, and multiple entero-enteric and entero-colonic fistulas. Colonoscopy was performed and mucosal ulceration at the IC valve and cecum was found. Biopsy taken from a deformed ileo-cecal valve showed characteristics of Crohn’s disease. Retrospective review of the initial appendix pathology showed changes consistent with Crohn's disease.

Diagnosis of Crohn’s disease involving the appendix is challenging because the signs and symptoms of Crohn’s disease may mimic those of acute appendicitis. Pathology features of the appendix play an important role for the diagnosis of Crohn’s involving the appendix. The findings of discontinuous inflammation, cryptitis and granuloma formation suggest Crohn’s disease. The reported prevalence of appendiceal involvement has varied from 7 to 54 %. Kaplan et al. conducted a large population-based cohort study of the incidence of post-appendectomy Crohn’s disease and found that risk of developing Crohn’s disease is highest within six months post-appendectomy. However the authors concluded that the finding may be due to missed diagnosis of appendiceal Crohn’s disease at the time of surgery. Thorough history taking, physical and pathology exams are crucial for diagnosis of Crohn’s disease involved appendix.
### A Real Pain in the Neck: Early-Stage Takayasu Arteritis Presenting with Isolated Carotidynia

**Introduction:** Takayasu arteritis is a challenging diagnosis because it is infrequently encountered and symptoms can vary significantly based upon the stage of disease. Often there is a long latency to diagnosis after the appearance of symptoms, which is unfortunate because treatment during the pre-stenotic phase of illness may prevent vascular injury. Here, we report an unusual case where prompt recognition of isolated carotidynia led to early non-invasive vascular imaging and institution of disease-directed therapy.

**Case Description:** A 48 year old female with several chronic medical conditions including nicotine dependency, hypertension, hyperlipidemia, and type II diabetes mellitus presented to our clinic for evaluation of neck pain. She localized her discomfort to the left, anterior triangle of the neck with radiation to the ear. It had intensified over a period of two weeks and was refractory to over-the-counter analgesia. Associated symptoms included dizziness and arthralgia. On examination, vital signs were within normal limits. Peripheral pulses were equally palpable and no bruits were detected, but even gentle pressure over the left common carotid artery resulted in severe pain. The remainder of the examination was unremarkable. Limited laboratory studies were also unrevealing with a normal complete blood count, erythrocyte sedimentation rate 16 mm/hr (0-29 mm/hr), and CRP 3.9 mg/dL (<8 mg/dL). Suspicion for vasculitis remained high and vascular imaging was pursued. Computed tomography angiography showed long-segment, circumferential, inflammatory soft tissue thickening encasing the left common carotid artery, carotid bulb, and proximal internal carotid artery. Further imaging revealed similar inflammation of other vasculature, including the descending thoracic aorta. Fortunately, there was no evidence of vascular stenosis or dilatation, and her symptoms had a dramatic and prompt response to oral steroids initiated for Takayasu arteritis.

**Discussion:** Anterior neck pain is a common complaint encountered by the internist, and this case provides a couple of interesting teaching points. First, carotidynia is a rare but important cause of anterior triangle neck pain. It can be an early manifestation of Takayasu arteritis and has been reported in 10-30% of cases at the time of presentation, though not usually as an isolated symptom. Second, diagnosis during the early stages of Takayasu arteritis requires a high index of suspicion because current diagnostic criteria are less sensitive for cases, such as the one above, when the vascular lumen is fully preserved.

### Swollen Ears after Chemotherapy

**Purpose:** Cytarabine is known to cause delayed hypersensitivity reactions, termed palmoplantar erythema, which presents with pruritus and pain along with erythema, often in the hands and feet. We present a patient with acute myeloid leukemia who underwent induction chemotherapy with cytarabine and idarubicin who developed bilateral swelling, erythema, and tenderness of her pinnae 6 days after initiation of cytarabine.

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Case Presentation: A 51-year-old female with newly diagnosed acute myeloid leukemia was admitted for induction chemotherapy with cytarabine and idarubicin. She received cytarabine from days 1 to 7 and idarubicin from days 1 to 3. On day 7, after receiving 6 days of cytarabine, she developed swelling, erythema, and tenderness of her bilateral pinnae. There was no change in hearing and no discharge was present. She did not have a fever. She also developed erythema on her neck, chest, and palms. She was diagnosed with “Ara-C ears” and Dermatology was consulted. She received topical steroid therapy and vitamin B6 100 mg daily with resolution of swelling and pain in her ears within 5 days of starting treatment.

Discussion: This case illustrates an atypical presentation of a known hypersensitivity reaction, known as palmoplantar erythema that is associated with cytarabine therapy. As it only involves the pinnae of the ears, the condition is known as “Ara-C ears.” Fortunately, the erythema resolves with steroid therapy and in this case, vitamin B6 was added as there have been studies showing significant symptom improvement. Delayed hypersensitivity reaction can be seen in patients undergoing cytarabine therapy and management includes symptomatic relief.

Jennifer Clark  
Dr. Omar Abu Saleh

### A Rare Cause of Severe Hypercalcemia Masquerading as Lymphoma in a Young Man with Anorexia and Weight Loss

**INTRODUCTION:** Severe hypercalcemia presents with symptoms that may range from irritability and constipation to anorexia, dehydration, or coma. The most common causes of severe hypercalcemia are malignancy and primary hyperparathyroidism, which account for 80-90% of cases. However, granulomatous disease may also cause significant elevations in calcium levels through dysregulated calcitriol production by activated macrophages, leading to (PTH)-independent hypercalcemia. Determining the underlying etiology of severe hypercalcemia with a thorough history, examination, and the judicious use of imaging and laboratory testing is essential.

**CASE:** We present the case of a 19-year-old man with a history of global developmental delay who presented to the outpatient clinic with concerns regarding progressive irritability, intermittent fevers, violent behavior, poor oral intake, and recent weight loss over the course of many weeks. Laboratory evaluation performed at this visit revealed a total calcium of 15.2 with a normal albumin level. He was subsequently admitted to the hospital for management of severe hypercalcemia. Intravenous hydration was initiated; once euvolemic, furosemide was also given. Further serum studies suggested a PTH-independent hypercalcemia with elevated calcitriol (152 pg/mL) and ACE (77 U/L) levels. Erythrocyte sedimentation rate and C-reactive protein were found to be elevated to 75 and 29.1, respectively. Rugger-jersey spine was noted on chest x-ray. Given the elevated calcitriol level, PET scanning was pursued to investigate the possibility of granulomatous disease or malignancy. This revealed multilevel lymph nodal involvement in addition to splenic, pulmonary, and perihepatic increased metabolic activity; marked hepatomegaly was also noted concerning for lymphoma. Ultrasound-guided biopsy of an involved cervical lymph node was obtained, which revealed small, oval-shaped fungal organisms.
with narrow-based buds, consistent with histoplasmosis. Intravenous antifungal treatment was initiated, with subsequent improvement in hypercalcemia and other symptoms.

DISCUSSION: Disseminated granulomatous infections are rare, but important and curable causes of hypercalcemia. Histoplasmosis is the most prevalent endemic mycosis in the United States, but severe complications in immunocompetent individuals is uncommon. Initial infection is typically asymptomatic or presents as a self-limiting influenza-like syndrome. In immunocompromised individuals, acute illness can result from early dissemination, with high associated morbidity and mortality. However, in immunocompetent patients, dissemination may present as a more chronic wasting syndrome involving multiple organ systems. This syndrome can rarely be associated with hypercalcemia due to the presence of fungal granulomas, mimicking malignancy or sarcoidosis (as was seen in our patient). Therefore, the diagnosis of disseminated histoplasmosis requires a high index of suspicion in the evaluation of granulomatous disease, especially in patients with travel or residency in endemic areas, systemic symptoms, or unexplained hypercalcemia. A delayed or mistaken diagnosis can compromise outcomes and potentially lead to serious complications.

**Daniel Davies**
Dr. Iftikhar Kullo

**A 21-Year-Old with Hemolytic Anemia, Thrombocytopenia, and Atherosclerotic Cardiovascular Disease**

A previously healthy 21-year-old male presented from an outside facility for evaluation after newly diagnosed cirrhosis. At the time of presentation, he complained of substernal chest pain during minimal exertion, dyspnea, jaundice, abdominal distention, and hematochezia. His family history was positive for sudden cardiac death of a paternal uncle in his 20s. Physical examination was significant for scleral icterus, petechiae, abdominal distention, and left Achilles tendon thickening. Laboratory analysis revealed hemoglobin of 8.9 g/dL, platelet count of 55,000, elevated liver function tests, with an INR of 2.7, and normal albumin. A lipid panel showed total cholesterol of 152 mg/dL, LDL-C of 36 mg/dL, and HDL-C of 88 mg/dL. An abdominal CT displayed a cirrhotic liver, splenic vein thrombosis, and extensive splenic infarction. Dobutamine stress echocardiogram was positive for ischemia with inducible wall motion abnormalities and a subsequent coronary angiogram found total occlusion of the RCA and diffuse, subtotal occlusion of the mid-LAD. Measurement of plasma sterols revealed severely elevated campesterol and sitosterol (plant-derived sterols), diagnostic of sitosterolemia. Genetic testing revealed a homozygous nonsense mutation at c.1336C>T, in the ABCG5 gene on chromosome 2p21, one of two genes implicated in this disorder. The patient was started on ezetimibe, a low plant-fat diet, and was evaluated for heart, lung, and liver transplant.

Sitosterolemia is a very rare, autosomal recessive disorder characterized by excessive intestinal absorption and reduced biliary excretion of plant sterols due to mutation of the sterolin transport protein. Excess plasma sterols can affect cellular membrane integrity, platelet function, and promote atherosclerotic plaque formation leading to anemia,
A Healthy Heart? A Case of ACS Without Plaque Rupture

Introduction: Chest pain is the second most common reason for emergency medicine visits in the United states. A troponin assay, is typically performed during initial evaluation, looking for myocardial injury. Therefore, keeping a broad differential for chest pain and troponin elevation is critical for accurate diagnosis.

Case: We present a case of a 58 year-old postmenopausal Caucasian female who presented with sub-sternal chest pain radiating down her left arm. She has a past medical history of native mitral valve endocarditis, status post mechanical mitral valve replacement in 2001 on anticoagulation, paroxysmal supraventricular tachycardia, hypertension, and depression. She is a never smoker and has a father who died of a myocardial infarction at 66 years-old.

She was doing laundry when she suddenly developed sub-sternal chest pain radiating down her left arm, which improved with rest and was worse with exertion. She has never had similar symptoms. She called emergency medical services and was taken to the emergency department. In route, she received nitroglycerin and 324mg of aspirin which relieved her pain. Vital signs and physical exam were both normal. Initial labs were unrevealing except a therapeutic INR of 3.1 and initial troponin of 0.04 ng/mL. Chest X-ray was only significant for sternotomy wires and mitral valve replacement. Electrocardiogram was unremarkable revealing only mild T-wave inversions in lead aVL. The troponin steadily increased to 1.0 ng/mL over the next 6 hours, reaching a high of 1.7 ng/mL 12 hours after event.

She was started on intravenous heparin, high intensity statin, and loaded with clopidogrel for acute coronary syndrome (ACS). The following day she had a coronary angiogram demonstrating: normal three major coronary arteries, but, the distal right posterior descending artery, obtuse marginal, and ramus showed narrowing, with the overall appearances consistent with spontaneous coronary artery dissection (SCAD) with intramural hematoma. This was not amenable to intervention.

Conservative medical management was chosen as the best strategy for the patient. She was discharged within 48 hours of presentation with daily aspirin, metoprolol, statin, and as needed nitroglycerin. Clopidogrel was discontinued.

Discussion: This case illustrates the importance of keeping a broad differential in patients arriving with chest pain and troponin elevation, as not all ACS is secondary to atherosclerotic plaque rupture. SCAD primarily affects women, and though classically seen in younger women, increasing data suggests postmenopausal women may also be affected. Presenting with classic ACS symptoms, it can lead to intramural hematoma within the coronary artery, but conservative management is typically preferred due to high complication risks with these challenging interventions.
Tumor lysis syndrome is most commonly associated with hematologic malignancies following cytotoxic chemotherapy, but it can also been seen spontaneously in solid malignancies with high tumor burden. Furthermore, acute renal failure in these situations can be sustained even once electrolyte values have improved.

Mr. M is a 65 y/o previously healthy man who presented to his local physician for a 1-month history of lower abdominal pain, bloating, and fatigue. Initial evaluation showed elevated liver enzymes, and a CT abdomen and pelvis revealed extensive metastatic disease and lymphadenopathy. Liver biopsies showed poorly differentiated neoplasm with unknown primary. His symptoms worsened rapidly, prompting a therapeutic paracentesis 10 days later yielding bloody fluid. He then chose to be transferred to our facility.

Upon arrival, he was found to have oliguric acute kidney injury with electrolyte abnormalities consistent with spontaneous tumor lysis syndrome: Creatinine 3.1, BUN 69, Uric Acid 14.1, Phosphorus 6.5, Potassium 5.6, and Calcium 8.1. He received 1 dose of rasburicase, IV fluids, and furosemide but did not respond adequately. Thus, he was begun on urgent hemodialysis.

Meanwhile, biopsies done at the outside hospital were confirmed as metastatic melanoma. For approximately 10 days, Mr. M remained hospitalized requiring intermittent dialysis while awaiting further pathology testing to guide treatment – specifically, the BRAF mutation status of his tumor. If positive, he would be a candidate for dabrafenib, a BRAF-targeted therapy. Ultimately, BRAF mutation was negative. Despite gradual improvement in his metabolic derangements, his kidney function did not recover over the course of his hospitalization. He would therefore require indefinite dialysis support. Given this and the incredibly aggressive nature of his cancer, Mr. M clearly explained on multiple occasions that he wanted to prioritize quality over quantity of life and thus declined any cancer-directed treatment. He also refused further dialysis. He consequently passed away approximately 12 days following his presentation to our hospital.

Final autopsy results are still pending. However, preliminary results confirm metastatic melanoma with involvement of the following: lumbar spine, lungs, liver, adrenal glands, heart, pancreas, omentum (caking), and peritoneum. No primary site has (yet) been identified.

There are 3 key learning points from this case:
1. In a solid malignancy with large, aggressive tumor burden and AKI, have a clinical suspicion for tumor lysis syndrome.
2. Unfortunately, tumor lysis – or possibly aggressive metastatic malignancies themselves – can precipitate sustained renal failure even once electrolyte abnormalities are improving.
3. Patients’ goals of care must always be preeminent in guiding further treatment, especially in cases with incurable conditions. Mr. M had an incredible understanding of the terminal nature of his disease, and he remained explicitly clear in his wishes to not remain indefinitely dialysis-dependent to facilitate cancer-directed treatment.
### Brinda Desai

**Lost to Follow-Up**

Aortic disease is the most life-threatening complication in Marfan Syndrome (MFS). Improved medical management and surgical results of prophylactic aortic interventions on the aortic root have dramatically increased expectancy of life in Marfan syndrome patients. However, when follow-up is not arranged or sought, the results can be devastating.

A 29 year old male with known MFS but no follow-up for over 14 years presented with 3 weeks of generalized weakness, nausea, dyspnea on exertion, and bilateral lower extremity edema. In the ED, EKG showed sinus tachycardia. CXR showed biapical pleural thickening and left lung base infiltrate, BNP > 8000, BMP with hyponatremia, hyperkalemia, elevated BUN and Cr, LFTs > 1000s, INR 2.5. Patient continued to be tachycardic despite fluid resuscitation. CT abdomen/pelvis was done given concern for acute liver failure which demonstrated a right sided effusion, cardiomegaly, hepatosplenomegaly, and aortic root 8.3 cm. Patient was then placed on a esmolol drip and transferred to our facility.

At our facility, patient underwent TTE which showed moderate reduce LV function at 35-40%, moderate RV dysfunction with RA enlargement, severe central aortic insufficiency (AI), and severe aortic root dilatation of 9.2cm x 9.0cm. Patient underwent surgical repair; procedure included proximal ascending aorta replacement, AVR with mechanical valve, and coronary artery re-implantation. Procedure was complicated by post procedure diffuse bleeding thought to be secondary to thrombocytopenia and severe coagulopathy. Post-op complication also included low cardiac output syndrome with TTE revealing LV function 10-15% and moderately to severely depressed RV function. Why the RV/LV worsened post op is unclear. Patient’s post-extubation course was complicated by Enterobacter sepsis and a large intracranial hemorrhage with an ischemic component. Given his poor prognosis and high likelihood of mortality, he was not deemed to be a surgical candidate. Family decided to transition to comfort cares and patient was compassionately extubated and passed.

The case above demonstrates the importance of close follow-up for patients with chronic conditions such as MFS with serious complications such as cardiogenic shock 2/2 aortic insufficiency or aortic dissection. As illustrated in this case, failure to do so can have devastating consequences. The major cardiovascular manifestation in MFS is a progressive dilatation of the ascending aorta, leading to aortic aneurysm formation and eventually to fatal aortic rupture or dissection. Aortic dissection in early adult life is the leading cause of death in MFS. Early diagnosis of individuals at risk of the disease is extremely important as timely treatment of cardiovascular complications has greatly improved life expectancy in MFS. Despite progress in medical and surgical treatment of aortic aneurysms, MFS continues to be associated with significant morbidity and mortality.

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### Allison Ducharme-Smith

**Methotrexate Toxicity, TEN Times Worse than you Think**

Methotrexate is an antimetabolite used as a chemotherapy agent in acute lymphoblastic leukemia. It is highly effective treatment but associated
with numerous and severe toxicities if not treated and monitored appropriately.

A young 29 yo woman diagnosed with acute lymphoblastic leukemia (ALL) presented to our hospital as a transfer from an outside institution ICU unit for treatment of a diffuse, bullous, sloughing skin rash, pancytopenia, acute kidney injury, and mental status changes. She was diagnosed four months prior with ALL during routine workup of fatigue. She was undergoing HyperCVAD chemotherapy treatment and tolerated the first cycles of chemotherapy well. She received her second cycle of methotrexate and cytarabine on 8/29/2017. On 8/29/2017 she was hemodynamically stable, asymptomatic, and her laboratory evaluation was normal with a creatinine of 0.5. She received protocol doses of methotrexate and cytarabine. Her 48-hour level of methotrexate was elevated at 6 and creatinine increased to 2.5. She was discharged on 9/3/2017 without oral leucovorin therapy or further monitoring of her methotrexate serum level. She represented the next day with a spreading desquamative rash to her feet and lower extremities with profound pancytopenia, acute kidney injury with creatinine 2.0, and hepatotoxicity with total bilirubin of 15. Her rash progressively worsened and despite multiple transfusions her pancytopenia persisted. She was transferred to an ICU for higher level of care for sloughing rash covering over 90% of her body, uncontrollable pain, and altered mentation. She was intubated, and sedated. A methotrexate level on 9/7/2017 was elevated at 0.81. She was given leucovorin 100mg q6hours, and sodium bicarbonate to alkalize her urine and excrete any remaining methotrexate. She underwent skin biopsy, which was consistent with toxic epidermal necrolysis (TEN) syndrome. She is receiving supportive cares and remains critically ill in our ICU.

Methotrexate is a highly active chemotherapy agent, which works by inhibiting DNA synthesis via inhibition of the dihydrofolate reductase. In the treatment of cancer, methotrexate is used at high, lethal doses and if not used properly it is associated with severe toxicities including myelosuppression, hepatotoxicity, neurotoxicity, nephrotoxicity, mucositis, and rash including TEN syndrome. The safe administration of Methotrexate requires rescue leucovorin therapy and adequate renal elimination via aggressive hydration and urine alkalization. Leucovorin is the active form of folate, which bypasses the inhibited pathway and rescues cells from the toxic effects of methotrexate. Our unfortunate patient did not receive adequate leucovorin rescue and suffered acute kidney injury without appropriate urine alkalization leading to her clinical deterioration.

This case highlights the severe toxicities associated with methotrexate use, important considerations when using methotrexate, and supportive cares used for treatment of toxicity. Early diagnosis, initiation of appropriate therapy, and supportive care are paramount for patient outcomes.

Michael Eastman

A Striped Horse in a Herd of Zebras

Sweet Syndrome is an uncommon but not unheard of affliction which is associated with underlying malignancy, drug exposure, or idiopathic reasons. However, when a rare-animal taxidermist presents from an
outside hospital in respiratory failure and shock—and with a necrotic lesion on his face—the differential diagnosis becomes broad indeed. In a herd of possibilities, this case shows, once again, that uncommon presentations of (relatively) common conditions are more common than common presentations of rare conditions.

Ankle Arthralgia, More Than Just Pain

While the pathogenesis of sarcoidosis is not well understood, the classic clinical and chest imaging findings are well known. Unfortunately, the diagnosis can be missed in cases with less common presentations. This stresses the importance of awareness of other key manifestations of sarcoidosis.

A 44-year-old Caucasian gentleman was admitted to the hospital for pain control and further evaluation. Two years ago he had sinus surgery and subsequent fungal sinusitis treated with 6 months of voriconazole. Four weeks before admission he developed a green/yellow nasal discharge and accompanying sinus pressure. These symptoms promptly improved with Gentamycin nasal irrigation. However, night sweats and cough persisted. A week later he developed progressive bilateral ankle pain that migrated and became polyarticular. Several days prior to admission he became febrile with a maximum temperature of 38.5 C. He denied any sick contacts and had no abdominal or genitourinary symptoms. His medical history was notable for chronic rhinosinusitis, sinus surgery, fungal sinus infection, and previous hip surgeries. He never smoked cigarettes. His father was diagnosed with non-Hodgkin’s lymphoma at the age of 51. Labs were notable for a neutrophil predominate leukocytosis to 10.7 x 10^9/L, C-reactive protein of 46.9 mg/L and a normal ACE level. Rheumatoid factor, anti-CCP antibody, ANA, and ANCA assays were negative. Joint x-rays showed cam and pincer deformity of the right hip with previous osteotomy of the left hip; hands, knees, and ankles revealed only soft tissue swelling of the knees. Chest x-ray showed mediastinal and bilateral hilar adenopathy. Evaluation for leukemia/lymphoma and granulomatous infection was negative. Maxillofacial CT did not demonstrate active infection. Flexible bronchoscopy was conducted for transbronchial needle aspiration and the sampled lymph nodes demonstrated granulomas. A diagnosis of sarcoidosis was made. He was initiated on prednisone with initial improvement but was unable to taper prednisone because of disabling joint pain. Methotrexate and infliximab were subsequently added but unfortunately, arthralgia persists.

This case illustrates sarcoid arthropathy and highlights the association of bilateral ankle pain that has been found to be the most common pattern of sarcoid arthropathy. A recent population-based study found joint involvement, most often involving ankles, in 42/345 (12%) patients; most had joint symptoms before sarcoidosis was diagnosed (average 21 days before diagnosis). Although absent in this case, the presence of erythema nodosum, a common dermatologic manifestation of sarcoidosis, would have fulfilled the triad for Lofgren’s syndrome (erythema nodosum, bilateral hilar adenopathy, and arthralgia). While sarcoid arthropathy tends to have a favorable response this case demonstrates that chronic arthritis may develop.
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<th>Abdelghani El Rafei</th>
<th>Amanitin Mushroom Poisoning</th>
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<td>Dr. Karl Tjerandsen</td>
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Introduction: Amatoxin poisoning is a rare cause of acute liver failure. Once suspected it is a medical emergency characterized by a gastrointestinal and hepatotoxic phases, coma, and death. Toxicity is ascribed to several Amatoxin-containing species belonging to three genera: Amanita, Galerina, and Lepiota. To date there have been few case reports of amanita toxicity in Minnesota. We present a case of clinically diagnosed amanita toxicity.

Case: A 77 year old male with past medical history that is pertinent for DM II, HTN, and recent diagnosis of dementia, who presented to the emergency department with altered mental status. Patient was unable to provide history; history was obtained by family members. Around 36 hours prior to presentation patient ingested a wild mushroom while on family trip in northern Minnesota. About 12 hours after ingestion he developed intractable nausea and vomiting and was later brought by family members to the emergency department. Initial laboratory values were pertinent for ALT 242, AST 220 and ammonia levels <9. Repeat liver function tests showed the following trend: ALT 416 - 1109 – 1818, AST: 416 - 1127 - 1115, and ammonia 76. The Poison control center was contacted and patient was started on N-acetylcysteine and activated charcoal. Patient was admitted to the ICU and was started on Cimetidine, Vitamin C, milk thistle, octreotide, and aggressive fluid hydration 8L in the first 24 hours. Mitchell’s Santa Cruz lab was contacted and IV silibinin was ordered. Due to clinical improvement and downtrending LFTs on day four, IV silibinin was not administered. Patient mental status improved and he was discharged home on day 8. His liver function tests were near normal at the time of discharge, Alt 234 , AST 67.

Discussion: Amatoxin poisoning is characterized by a long asymptomatic incubation delay (from 6 to 12 hours) and three clinical phases. The first phase, or gastrointestinal phase (12–24 hours), hepatotoxic phase (24–48 hours), and irreversible coagulopathy phase (4-7 days). The mainstay of therapy is aggressive fluid hydration to replace fluid loss from the GI phase and prevent kidney damage by minimizing the Amatoxin renal transit time. Suspending enterohepatic circulation by making patients strict NPO and octreotide therapy prevents further release Amatoxin laden bile in the gut. Silibinin is an extract of Mediterranean milk thistle and inhibits Amatoxin uptake by organic anion transporting polypeptide. Our patient improved prior to administration to IV silibinin. Measures such as high dose vitamin C, cimetidine, and high dose penicillin G have limited efficacy and are poorly studied. Lactate and LDH levels are the most sensitive prognostic lab value. If Mushroom poisoning is suspected poison control should be contacted immediately.

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<tr>
<th>Kelly Gast</th>
<th>A Case of Bilateral Chylothorax and Diffuse Lymphadenopathy</th>
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<td>Dr. Hannah Nordhues; Dr. Ulrich Specks</td>
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Introduction: Chyle is a lymphatic fluid that is milky in appearance and contains a high content of triglycerides. A chylothorax develops when lymph fluid accumulates in the pleural space as a result of disruption of the thoracic duct or its branches. The etiology of chylothorax is often
categorized into traumatic and non-traumatic causes. Malignancy is the most common cause of non-traumatic chylothorax and includes lymphoma, chronic lymphocytic leukemia, and metastatic cancer.

Case description: A previously healthy 23-year-old male presented with insidious onset of fevers and shortness of breath. He was found to have bilateral pleural effusions and diffuse lymphadenopathy concerning for lymphoma. He underwent extensive work-up for infection, rheumatological conditions, and malignancy. Work-up consisted of multiple lymph node biopsies including inguinal, subcarinal, and mediastinal as well as bone marrow biopsy which were negative. Flow cytometry was also negative. He was treated empirically with high dose prednisone with improvement in his symptoms and discharged with a prednisone taper. He was re-admitted two weeks later due to increasing dyspnea, bilateral lower extremity edema, bilateral chyloous pleural effusions requiring chest tube placement, and a pericardial effusion with tamponade physiology requiring pericardiocentesis. During the hospitalization, he had peritoneal biopsy which demonstrated mesothelial proliferation with chronic inflammation. Due to ongoing symptoms and a still unclear diagnosis, the patient was transferred to our facility with a preliminary diagnosis of multicentric Castleman’s disease. Upon arrival, a pericardial drain and bilateral pleural drains were productive of milky white output. There was no palpable peripheral lymphadenopathy. Laboratory work up demonstrated significantly elevated inflammatory markers with an ESR of 52 and CRP of 100. Pleural fluid analysis was significant for a triglyceride level of 1,714 mg/dl and mesothelial cells with reactive lymphocytes were seen. PET scan was performed and demonstrated wide-spread FDG avid uptake including a pelvic mass that extended into the inguinal canal which was biopsied. Pathology revealed epithelioid malignant mesothelioma. The patient continued to have a complicated course which included lymphangiogram with embolization of the thoracic duct, decortication of his bilateral lungs, and eventually initiation of palliative chemotherapy with carboplatin and pemetrexed.

Discussion: The most common etiology for atraumatic chylothorax with associated diffuse lymphadenopathy is lymphoma. However, if work up for lymphoma is negative, practitioners must maintain a high index of clinical suspicion for another malignancy. Malignant mesothelioma is a rare diagnosis in young individuals. Yet, this case highlights that mesothelioma can occur in young individuals and in individuals without known asbestos exposure.

**Andre Genereux**  
Dr. Lisa Callies  
With an ACE High, the Odds Are Not Always in Your Favor

A 65 year old white male with a past medical history of hypertension, coronary artery disease and insulin dependent type II diabetes mellitus presented to his primary care physician with nausea and vomiting. A basic metabolic panel revealed an elevated calcium (13.1 mg/dL) and elevated creatinine (3.1 mg/dL). These labs were both normal eight months prior to that time. A PTH level was low (<7.0 pg/mL, normal 14.5-87.1). Serum 15-hydroxyvitamin D was decreased at 24.8 ng/mL (normal 30-100), and 1,25-dihydroxyvitamin D level was elevated at 127 pg/mL (normal 18-64). PTHrP was normal. SPEP revealed
increased light chains, non-monoclonal. An abdominal CT revealed splenomegaly, non-obstructing nephrolithiasis, no lytic or blastic bone lesions, and no lymphadenopathy. He was hospitalized, hydrated and given 200 U of Calcitonin subcutaneously. The patient had recently started Canagliflozin, which was discontinued (hypercalcemia is a known side effect). Four weeks later, despite halting Canagliflozin therapy, the hypercalcemia worsened (12.9 mg/dL) and the creatinine increased to 4.0 mg/dL. Although asymptomatic, he was hospitalized and treated with crystalloid fluids and Pamidronate, with modest improvement. An ACE level was elevated at 75 U/L (normal 8-53). Interferon-γ release assay was negative. PET CT was performed which showed an enlarged hypermetabolic spleen, hypermetabolic lymph nodes in the bilateral pelvis, as well as individual hypermetabolic lymph nodes in the peritracheal area and left hilar region. FNA CT-guided biopsy of the right external iliac lymph node revealed non-necrotizing granulomatous inflammation, negative for yeast, fungal hyphae and pneumocystis; benign soft tissue with nodular calcification was also present. The patient was diagnosed with extrapulmonary sarcoidosis and placed on Prednisone 60 mg daily. At discharge, the calcium was 12.1 mg/dL. In follow-up five days later, the calcium was 10.9 mg/dL. Upon recheck three weeks after hospitalization, the calcium had returned to normal at 8.7 mg/dL.

Sarcoidosis is a granulomatous disorder that can effect multiple organ systems. The cause is unknown. Extrapulmonary sarcoidosis is a rare manifestation of sarcoidosis that is more common in people presenting with sarcoid who are older, of European descent, and male. Up to 5% of patients who present with sarcoidosis over the age of 40 have hypercalcemia. Following the diagnosis, 10-20% of patients eventually develop hypercalcemia. Extrarenal production of 1,25-dihydroxyvitamin D in activated macrophages is the responsible culprit. Hypercalcemia may be the only clinical manifestation of sarcoidosis in these patients, or, more often, is seen in conjunction with more common signs of sarcoidosis, such bilateral hilar adenopathy, pulmonary reticular opacities or skin/joint or eye lesions. The treatment is similar to that of other sarcoid disease and is corticosteroid based. Ketoconazole, bisphosphonates, or dietary restriction of calcium can be used as additive therapy for severe cases.

Sophie Hapak
Dr. Reut Danieli

Varicella Zoster: An Elusive Virus with Unpredictable Complications
Presented here a case of a veteran who unfortunately developed two unlikely complications of shingles- post-herpetic neuralgia and paralysis of the surrounding muscles. One out of every three adults in the United States will develop shingles, a cutaneous manifestation of the varicella zoster virus marked by pain and pruritis along the dermatomes associated with the dorsal root ganglia in which the virus resides. Of those who develop shingles, 20% will develop neuralgia at the site of the prior shingles infection. An even smaller cohort of patients (0.3%) will develop muscle paralysis and/or inflammation, usually of those proximal to the initial shingles infection. The mechanism by which the virus affects the anterior horn motor neurons from its home in the dorsal root ganglia is unknown. Further, the incidence of one patient developing multiple complications of the shingles virus has never been measured. The modalities of diagnosis of these complications as well as treatment strategy will be reviewed.
| Michelle Herberts  
Dr. Lucinda Gruber | **Chief Complaint: Depression, An Unusual Presentation of Granulomatosis with Polyangiitis**  

Depression is a common chief complaint encountered by all physicians. Despite the fact that it is commonly encountered in inpatient and outpatient practice, it is sometimes important to rule out other life-threatening causes of a patient's presentation.

A 60 year old male with no prior psychiatric history was admitted to the hospital with a 3 week history of depression, anxiety, and auditory hallucinations. He had also been experiencing decreased appetite, headaches, and passive suicidal ideation. He was recently discharged from a local inpatient psychiatric hospital for these symptoms. His symptoms persisted despite anti-depressants, and his wife sought a second opinion.

Physical exam showed a disheveled man who was lying with his eyes closed. He answered questions by repeating his name and was not able to follow commands. He endorsed auditory hallucinations telling him to inflict self-harm. Laboratory findings on admission were significant for a leukocytosis of 13.7 g/dL, elevated ESR (57 mm/h, normal 0-29) and elevated CRP (37.1 mg/L, normal < 8.0). CT and MRI of the head showed diffuse dural thickening and opacification of the left mastoid sinus. A multitude of infectious and rheumatologic laboratory tests were performed and were within normal range. P-ANCA, C-ANCA, and PR3 levels were normal. MPO Ab was equivocal. A lumbar puncture was performed to rule out infectious etiologies with negative results. During his hospital stay, the patient experienced two episode of seizure like activity. EEG was performed and showed epileptogenic activity on bilateral temporal lobes and appropriate medications were initiated. Temporal artery biopsy was negative for vasculitis. Dural biopsy was ultimately performed and revealed pathologic features of granulomatosis with polyangiitis. The patient was initiated on IV methylprednisolone and Rituximab with improvement in his symptoms.

Granulomatous polyangiitis usually presents with end organ manifestations such as renal failure. Even in patients with meningeal involvement, the usual presentation is headache and cranial nerve deficits. While depression has been described in a few case reports, auditory hallucinations are rare. This case emphasizes the wide array of presentations which are possible with this rheumatologic disease. Although a relatively common chief complaint, it is important to keep a broad differential and use critical thinking when working up a patient with acute onset depression.  

| Brian Holmgren  
Dr. Amy Holbrook | **Don't Play Chicken with your Aorta**  

Salmonella aortitis is a condition characterized by infection and inflammation of the aorta which can result secondary to bacteremia. Given the vague symptom profile of aortitis, it is often a difficult diagnosis to make.

A 73 year old man with history of AAA status post repair with a straight Hemashield graft in 2008 presented to the emergency department with
four days of malaise, chills, and low back pain. He was afebrile with stable vitals. Labs were notable for a mild leukocytosis of 12,000. A CT scan of the abdomen was obtained and notable for new periaortic stranding when compared to CT from one month prior. The patient was admitted with concern for possible aortitis. Blood cultures were drawn. Further history was obtained concerning possible infectious sources. The patient had dental cleaning performed four days prior to admission as well as a vague diarrheal illness a few weeks prior. Inflammatory markers were obtained and elevated. Vascular surgery was consulted and recommended a PET scan. The patient had intermittent low-grade fevers during the hospital stay, but daily blood cultures were negative for three days. Antibiotics were not initiated given uncertainty of diagnosis and patient’s clinical stability. He was discharged with a scheduled outpatient PET scan and Vascular Surgery follow up. The patient was readmitted the day after discharge with mild confusion and continued low grade fevers. Blood cultures were again obtained. An inpatient PET scan was performed and noted increased metabolic uptake of the aortic graft consistent with aortitis. Infectious Disease was consulted and IV ceftriaxone was initiated. Blood cultures returned positive for Salmonella species. Vascular Surgery recommended three weeks of IV ceftriaxone prior to aortic graft removal and replacement. The patient was discharged with a midline IV and in-home antibiotics. He returned in three weeks and underwent uncomplicated graft removal and replacement. Cultures of the removed hardware again grew Salmonella species. The patient was discharged on three weeks of PO ciprofloxacin and outpatient follow up with Infectious Disease. The patient has since completed antibiotics and is recovering well.

Endovascular infections are a serious potential complication of salmonella bacteremia. Estimates of incidence are as high as 20% in cases of salmonella bacteremia with the infrarenal aorta being the most frequent site of infection. Although this is a rare condition, prompt recognition and diagnosis is essential given the potential for severe complications including aortic rupture. Symptoms to consider include fever, malaise, abdominal pain, low back pain, and pulsatile abdominal mass. Abdominal CT or MRI with contrast are the preferred initial diagnostic imaging modalities. Laboratory evaluation including blood cultures and inflammatory markers are also beneficial.

**Adam Hsu**
Dr. Daniel Reynolds

* A Profoundly Prolonged PT

Case Description: A 73-year-old male presented to the emergency department with the acute onset of non-traumatic severe right leg pain. His medical history was relevant for celiac disease and recurrent venous thromboembolism on chronic anticoagulation with apixaban. Physical exam was notable for cachexia with a BMI of 16, multiple ecchymoses over the upper extremities and chest, and limited range of motion of the right hip and knee joints due to pain. His right lower extremity sensorineural exam was otherwise unremarkable. Labs revealed a hemoglobin value of 8.1 and MCV value of 87.5, which were similar to his baseline, as well as an INR > 20. Radiographs obtained in the emergency room revealed diffuse osteopenia and a right subcapital femoral neck fracture. He was admitted to a medicine service due to his coagulopathy prohibiting any immediate orthopedic intervention.
Upon arrival to the floor, INR was confirmed to be > 20. aPTT was obtained which was also prolonged to 75. Liver tests were obtained and revealed AST 24, ALT 26, total bilirubin 0.2, alkaline phosphatase 290, and albumin 2.6. Further evaluation revealed decreased levels of factors II, VII, and X with normal levels of factors V, VIII, and XII. He was given 5 mg of IV vitamin K and upon recheck twelve hours later, his INR was 1.5. Levels of vitamin D, zinc, copper, and B6 were obtained and all found to be deficient.

His INR remained below 1.5 for the rest of his hospitalization and he was able to undergo cemented bipolar arthroplasty. He was dismissed with a multi-vitamin replacement regimen as well as follow-up with gastroenterology and a nutritionist.

Discussion: Some of the most obvious manifestations of celiac disease (CD) involve gastrointestinal symptoms such as abdominal pain and diarrhea. However, malabsorption can also lead to nutritional deficiencies which can present in a variety of different ways and may or may not be immediately obvious. The American College of Gastroenterology Clinical Guidelines on Celiac Disease recommends evaluation of nutritional deficiencies for all patients with CD. This includes, but is not limited to, evaluation of levels of iron, folic acid, vitamin D, and vitamin B12. Other nutrients, such as vitamin A, E, K, copper, and zinc may also be deficient. In the case of this patient, his vitamin D deficiency likely led to osteopenia, which made him susceptible to a non-traumatic femur fracture. Vitamin K deficiency led to a profoundly elevated INR with diffuse ecchymoses. Given the myriad possible complications of nutritional deficiencies in CD, it is essential that providers remember to assess and replete nutritional deficiencies in patients with celiac disease.

Marie Hu
Dr. Derek Ebner;
Dr. Thomas Poterucha

Pyoderma Gangrenosum: The Ultimate Mimicker

INTRODUCTION: Pyoderma gangrenosum is a rare, neutrophilic inflammatory dermatosis characterized by painful, necrotic ulceration. The diagnosis is challenging and often one of exclusion, as no specific laboratory or histopathologic findings confirm the diagnosis. However, several clinical features should raise clinicians’ suspicion for pyoderma gangrenosum.

CASE: A 29-year-old woman with history of polycystic ovarian syndrome presented to her PCP for evaluation of fever (39.3C), chills, and boil of the inner thighs. The previous day, she had noted induration there and lanced the area with a needle, but no fluid was expressed. On exam, a 4x2 cm tense lesion with surrounding erythema on the right thigh and a 2x2 cm similar lesion on the left thigh was noted. She was diagnosed with a bilateral skin and soft tissue infection and prescribed oral doxycycline. However, the next day the pain and erythema progressed, so she returned to the ED. Physical exam supported progression of erythema, with development of large bulla with central eschar bilaterally. She was tachycardic and tachypneic, and labs were significant for WBC 23 and CRP 209 mg/L. She was then admitted to the Medicine service and initiated on Vancomycin and piperacillin-tazobactam. Dermatology was consulted and obtained a punch biopsy of
the bullae. The following day, she had persistent pain and fever, with
new bullae, skin ecchymosis, and erythema. CT of the thighs showed
prominent cutaneous and subcutaneous inflammatory changes with fluid
along the myofascial plane. She was taken urgently to the operating
room due to concern for necrotizing fasciitis; although there was no
evidence of this, the superficial skin was necrotic and was debrided.
Within several days, there appeared to be new areas of necrosis along
the wound edges. Deep tissue cultures remained negative for bacteria or
fungi. Given the worsening necrosis following debridement suggesting
pathergy, pyoderma gangrenosum was then suspected and pathology of
the punch biopsy returned consistent with this. She was initiated on IV
methylprednisolone and later transitioned to prolonged oral prednisone
taper. She noted significant improvement in pain the day after steroids
were initiated, and the lesions appeared to stop progressing shortly
thereafter.

DISCUSSION: The diagnosis of pyoderma gangrenosum should be
strongly suspected in patients whose wounds are painful, rapidly
expanding, unresponsive to antibiotics, and worsening with debridement
or trauma (the phenomenon known as pathergy). Infection is always in
the differential and needs to be ruled out, especially since treatment for
pyoderma usually consists of high-dose systemic steroids. Additionally,
pyoderma gangrenosum is associated with an underlying systemic
disease in over 50% of patients, with the most common conditions being
inflammatory bowel disease, arthropathies, and hematologic disease or
malignancy. Thus, a thorough work-up for underlying conditions should
be taken in any patient newly diagnosed with pyoderma gangrenosum.

Brandon Huffman
Dr. Zelalem Temesgen

Resistant Pneumonia: Minnesota’s Lake Monster

Introduction: Pneumonias that do not respond to initial antibacterial
therapy should raise suspicion for fungal pneumonia, in particular
pulmonary blastomycosis in endemic areas. A thorough clinical
exposure history can often lead to earlier detection of atypical infections.

Case description: A 47 year old male from Northern Minnesota
presented to his primary care provider after seven days of nonproductive
cough and subjective fevers. He was prescribed albuterol and
amoxicillin-clavulanic acid. Two days later, he developed drenching
night sweats, chills, headache, dyspnea on exertion, and cough-induced
vomiting. After additional evaluation, he was febrile, hypoxic, and
tachycardic, so he was admitted to a local hospital. Initial outside
laboratory studies showed a leukocytosis (WBC 19.2) with extreme left
shift and hyponalbuminemia (2.7). Chest xray showed a left lower lobe
consolidation. A sputum gram stain was positive for gram positive
organisms. The patient was treated with ceftriaxone, azithromycin, and
eventually transitioned to vancomycin and levofloxacin. He did not
improve after 72 hours of therapy, so he was transferred to Mayo Clinic
for further evaluation and management. Upon initial presentation, his
temperature was 40.3 degrees Celsius and his oxygen saturation was
94% on 4 liters/min nasal cannula. He appeared mildly uncomfortable
with 4 word conversational dyspnea. He had a past medical history
significant for morbid obesity and hypertension. He was recently
excavating his lakeside resort, and one of his coworkers was
experiencing similar symptoms. On physical examination, he had bibasilar crackles, predominantly on the left. CT chest showed multiple consolidative masses in the bilateral lower lung fields, most predominant on the left. Sputum examination showed mixed flora with many WBCs and epithelial cells. On Calcofluor examination of the sputum, broad-based budding yeast forms were identified. Sputum PCR was positive for Blastomyces dermatitidis. The patient was treated with liposomal amphotericin B for two weeks with improvement. He was then transitioned to oral itraconazole for the remainder of one year.

Discussion: Fungal pneumonia should be suspected in patients who continue to be febrile for 48 hours despite adequate first-line pneumonia antimicrobial therapy. Particular attention should be paid to the exposure history as is the case in this situation. This patient was experiencing similar symptoms to his coworker who helped him excavate a lakeside resort in Northern Minnesota (an endemic area for Blastomyces). Diagnosis can be made by culture, detecting antigens or sputum PCR. In severe cases of pulmonary blastomycosis, induction with amphotericin B is necessary, and maintenance therapy with itraconazole yields excellent treatment efficacy.

Nick Huynh
Dr. Peter Lund

*Mycobacterium Arupense.... Colonizer or Pathogen*

Introduction: *Mycobacterium arupense* is a slow-growing, nonchromogenic, acid-fast bacillus first identified in 2006. Only nine cases of clinical disease have been reported. The rate of true infection vs colonization is unknown. Here we report a case of possible clinical significant *M. arupense*.

Case: A 66 year old Somali male presented with a 4 day history of dyspnea, rigors, cough and diffuse joint pain. He has a known positive screening Quantiferon TB Gold test, but no known previous TB exposures, is not immunocompromised, and has no known history of treatment for active or latent TB. He presented to the ED and was afebrile, BP 104/48, P 88, RR 28, O2 Sat 100% and BMI 17. A CT PE study revealed stable RUL scarring that was present on a previous CT, and a new Right Lower Lobe infiltrate with no PE. He was diagnosed with community-acquired pneumonia and discharged home with Moxifloxacin. His symptoms worsened and returned for hospital admission 4 days later.

On admission, exam revealed stable vital signs, clear lungs, polyarthralgias in knees, shoulders, and elbows without warmth or effusion and no lymphadenopathy. Labs were notable for WBC of 11.9, Hgb 11.1, Na of 129. Overall, given the presence of rigors, cough, and cachexia and pulmonary scarring typical of TB and new infiltrates, the diagnosis of reactivation pulmonary TB was considered highly probable. The patient was isolated, induced and bronchoscopic sputum specimens were obtained and mycobacterium TB therapy was initiated with directly observed 4 drug therapy (Isoniazid, Rifampin, Pyrazinamide and Ethambutol) while awaiting the results of AFB cultures. With the treatment regimen, the patient’s symptoms all resolved with three weeks of initiating therapy. Ultimately, sputum AFB cultures grew mycobacterium species further isolated as mycobacterium arupense.
Later a second AFB culture grew mycobacterium avium complex. All other mycobacterial, bacterial, fungal and viral cultures as well as PCR for m.tuberculosis remain negative at 3 months incubation. The patient self-discontinued therapy after six weeks secondary to medication intolerance and his symptoms have not returned.

Discussion: Since its isolation in 2006, there have been only 9 reports of clinical illness associated with recovery of mycobacterium arupense. 75% of the reported cases had prominent osteoarticular symptoms. This patient presented with respiratory, oligoarticular and systemic symptoms that worsened despite treatment with Moxifloxacin, and resolved with targeted mycobacterial therapy.

The limited available data supports the hypothesis that most m.arupense isolates from pulmonary sources represent colonization rather than infection. Nonetheless, this clinical vignette may represents true m.arupense vs mycobacterium avium complex pulmonary infection with associated transient polyarthritis. This case should serve as a reminder that m.tuberculosis reactivation should be considered in all patients born in TB endemic areas and that non-tuberculous mycobacteria can present similarly to TB.

| **Brenden Ingraham**  
| Dr. Michael Klajda;  
| Dr. Para Karmacharya;  
| Dr. Vaidehi Chowdhary |

**Drug-Induced Rhabdomyolysis with Tenofovir and Atorvastatin**

**INTRODUCTION:** Severe rhabdomyolysis resulting in debilitating proximal muscle weakness and acute kidney injury in a patient with hepatitis B on tenofovir and chronic statin use.

**CASE DESCRIPTION:** 70-year-old male with CLL and hepatitis B reactivation after treatment with a BTK inhibitor (subsequently stopped) presented with one week of progressive proximal muscle weakness. He takes diltiazem and aspirin for chronic atrial fibrillation, atorvastatin for hyperlipidemia, and tenofovir (nucleotide reverse transcriptase inhibitor; initiated 6 weeks prior) for hepatitis B. He was previously active but could not lift his arms above his head or rise from a chair at presentation. He could ambulate with a walker by taking small, shuffling steps. Mild pain in the pelvic/shoulder girdles. No distal weakness, diplopia, dysphagia, sensory abnormalities, or rashes. Review of systems otherwise unremarkable. Pertinent exam findings include jaundice, lower extremity proximal strength 3/5, upper extremity proximal strength 3-4/5, and distal strength 5/5. Pertinent admission labs include creatine kinase 27,500, aldolase 279, LDH 1360, creatinine 1.4 (baseline 1.3), AST 1942, ALT 943, total bilirubin 9.2, direct bilirubin 6.2, albumin 3.0, INR 1.0, and urinalysis revealed large amount of hemoglobin but < 3 RBCs. Anti-Jo-1, MyoMarker 3, anti-HMGCR, and ANA were negative. He was tentatively diagnosed with rhabdomyolysis secondary to drug-induced myopathy and acute kidney injury, and tenofovir and atorvastatin were discontinued. Despite this, his weakness worsened, CK rose to 112,000, and creatinine was 2.0 on hospital day 4. There was concern for immune-mediated necrotizing myopathy related to atorvastatin, so methylprednisolone 1000 mg daily for 5 days was started empirically. EMG showed severe proximal myopathy with features suggestive of necrosis. Triceps biopsy revealed rare necrotic fibers associated with a slight myopathy and no inflammatory changes.
making immune or auto-immune-mediated etiology less likely. He began to improve by hospital day 6 with a CK of 3,175 and a creatinine of 1.6. He was discharged home with an oral prednisone taper and physical therapy. He is doing well now off the tenofovir and atorvastatin.

DISCUSSION: The incidence of tenofovir-induced rhabdomyolysis is extremely rare only being described in case reports. The incidence of atorvastatin-induced rhabdomyolysis is 0.6/10,000 person-years. The risk of rhabdomyolysis increases with the concentration of the statin. Atorvastatin is metabolized through the CYP450/3A4 pathway in the liver, which is not affected by tenofovir. However, he had decreased liver function related to his hepatitis B, which would decrease the clearance of atorvastatin. It is unclear if his acute episode was the result of initiating tenofovir 6 weeks prior or if the statin was the culprit in the setting of deteriorating liver metabolism. Regardless, it is imperative to be mindful of the medication list, rare adverse reactions, and ever-changing physiology in complex patients.

Sae Jang  
Dr. Dayne Voelker;  
Dr. Thomas Beckman

Aortic Mural Thromboses may be Overlooked in Patients with Paraplegia and Limb Ischemia

Introduction: Aortic mural thrombi (AMT) are typically discovered when evaluating distal extremity emboli. However, AMT may be missed in patients with paraplegia and limb ischemia.

Case Description: A 57 year-old woman with past history of T10 paraplegia, multiple stage IV decubitus ulcers of bilateral lower extremities, osteomyelitis, neurogenic bladder, recurrent UTIs, and tobacco abuse presented with 4-days of fever, nausea, vomiting, and fatigue. In the emergency department she had a fever to 39.3 degrees Celsius and tachycardia to 104 BPM, so was initiated on broad spectrum antibiotics. On physical examination she appeared malnourished. She was found to have an ischemic left foot that was cool to palpation with purple discoloration up to her ankle, a diminished left posterior tibialis pulse, and an absent dorsalis pedis pulse. She had not inspected her feet in several days, and because of her paraplegia, had been unaware of pain or other changes in her extremities. Blood tests were remarkable for microcytic anemia with hemoglobin = 6.8 grams/dl, thrombocytosis with platelets = 597K, and Protein C level low at 60%. Hypercoagulability testing was otherwise negative. Imaging included lower extremity US and CT angiogram which revealed a mural thrombus of the infra-renal abdominal aorta resulting in 75% stenosis; occlusion of the left common iliac and popliteal arteries; infarcts involving the spleen and bilateral kidneys; and multiple bilateral lower extremity DVTs. A transthoracic echocardiogram was unremarkable. Review of systems revealed no diabetes, vascular disease, prior DVT, estrogen therapy, or family history of thromboses.

Extensive testing for infection was negative and antibiotics were discontinued. She was initiated on aspirin, atorvastatin, and intravenous heparin, and was transitioned to rivaroxaban upon discharge from the hospital.
Discussion: Thromboembolic disease should be included in the differential diagnosis for fever. A study found that most patients with AMT without underlying aortic disease have coagulation disorders. Therefore, patients with aortic mural thrombi and no underlying aortic disease should undergo a hypercoagulability evaluation. However, paralysis and associated complications may be overlooked as independent risk factors for AMT. The current patient had multiple predisposing factors for thrombosis including paraplegia, immobility, smoking, chronic inflammation from venous stasis wounds, and poor nutritional status leading to protein C deficiency. This case highlights the importance of performing careful extremity and vascular examinations in paretic patients with decreased sensation.

Jake Jasurda

Approach to the Heavy Heart: Cardiac Amyloidosis in a Patient with Underlying Plasma Cell Dyscrasia

Cardiac amyloidosis is a rare disorder characterized as an extracellular accumulation of amyloid fibrils resulting in a restrictive cardiomyopathy. It is often sub-classified as either light chain amyloidosis (AL) or transthyretin amyloidosis (ATTR), each of which has a distinct set of therapeutic options. A 57 year old male with a past medical history of pulmonary hypertension and obstructive sleep apnea presents from clinic with worsening dyspnea over the past 3 months, hypotension and bilateral lower extremity edema. Physical examination was notable for 4+ pitting edema of the bilateral lower extremities up to the waist, jugular venous pressure of 10 cm of water, and macroglossia. Initial laboratory findings were significant for NT-proBNP of 9323 and troponin of 0.116. Electrocardiogram was most notable for diffuse low voltage QRS complexes. Transthoracic echocardiogram was performed and demonstrated mild concentric wall thickening of the left ventricle, left ventricular hypertrophy with a left ventricular ejection fraction of 30-35%, a moderate sized pericardial effusion and elevated bilateral atrial pressures. Further investigation yielded an elevated monoclonal free kappa light chain on urine immunofixation and elevated free kappa light chains in serum. Bone marrow biopsy was then performed and demonstrated >10% monoclonal plasma cells, which in the setting of the aforementioned light chain findings were suggestive of a plasma cell myeloma. An abdominal fat pad biopsy was performed and found to be negative for the presence of amyloid. An endomyocardial biopsy was planned, but the patient suffered a PEA arrest prior to the procedure. He was resuscitated and return of spontaneous circulation was achieved, but he subsequently suffered a recurrent PEA arrest and expired shortly thereafter.

Cardiac amyloidosis, as seen in this patient, can present with the typical constellation of symptoms seen in congestive heart failure. Given the significant mortality risks associated with this disease, early recognition of low voltage QRS complexes, echocardiogram findings suggestive of an infiltrative myocardial process, or otherwise unexplained congestive heart failure symptoms are crucial for early and effective intervention.

Kimberly Johnson

A Different Kind of Tick

A 31 year old South African man with no significant past medical history presented to the ED with 1-week history of worsening headaches, myalgia, and high-grade fever. He had recently arrived from...
South Africa where he worked as a farmer tending sheep and cattle. He had noted a bug bite on his right calf of unknown duration shortly after arriving. Symptoms started with diffuse body aches, mild headache, chills and subjective fevers. He was evaluated at an urgent care where blood work revealed a CRP of 65.3 and negative malaria testing. He was treated with amoxicillin and tetanus shot. Symptoms progressed over the next 4 days with worsening of his headache, photophobia, severe fatigue, and a diffuse rash across his chest, abdomen, and extremities. He reported previous tick and mosquito bites, and multiple animal exposures during his work on the farm. He denied any ill contacts, although, he had recently been a long plane flight. He was unsure of his immunization record. On presentation to the ED, he had a noncontrast head CT, lumbar puncture (LP), and initiation of meningitis treatment before being admitted.

On admission, his physical examination revealed a diffuse macular rash involving his chest, back, and distal extremities, and a single 1cm eschar with surrounding erythema was present on the posterior aspect of right calf distal to the knee. His examination was otherwise negative. The results of the LP showed a total cell count 6, glucose 66 mg/dl, and protein 62 mg/dl. A presumptive diagnosis of African Tick Bite Fever was made and doxycycline treatment was initiated. The patient’s symptoms improved the following day and he was discharged with a 7-day course of doxycycline. Later immunoglobin testing revealed an elevated Spotted Fever Group IgG at 1:128 and IgM <1:64.

The diagnostic power of a detailed history and careful physical exam are highlighted in this case. The differential for headache and fever is broad; however, eschar and diffuse body rash in the setting of travel from South Africa was clinically enough to arrive at the proper diagnosis without further testing. African tick bite fever may be the most widespread spotted-fever group rickettsia causing human illness. Although reports in the United States are few, the illness has been reported multiple times in European literature, often in travelers returning home after visiting endemic areas. Although the illness is not known to be fatal, it is readily treatable and easily identifiable in the correct clinical context.

Additionally, this case highlights how the world’s growing interconnectedness necessitates the need for a complete differential to include diseases prevalent in all parts of the world.

Nycole Joseph
Dr. Amy Oxentenko

A Penny For Your Thought Process: The Neuropsychiatric Component of Wilson Disease

Wilson disease is an autosomal recessive condition resulting in progressive hepatolenticular and neurological deterioration. Patients with Wilson disease who present primarily with neuropsychiatric manifestations are at risk for a delayed diagnosis, leading to significant comorbidities that can be irreversible.

A 26-year-old man from Saudi Arabia, who was previously healthy, presented to his primary care provider with psychiatric symptoms. He was diagnosed with bipolar disorder and started on olanzapine. Four
months later, he developed rigidity, tremors, poor coordination, gait instability, ophthalmoplegia, dysphagia and dysautonomia. He was then hospitalized after a syncopal episode, with concerns for stroke. On investigation, MRI of the brain demonstrated T2 hyperintensities involving the basal ganglia. Additionally, abnormal liver biochemistries, decreased serum ceruloplasmin (7mg/dl), and elevated 24-hour urinary copper (228mcg/24 hr) were noted. Abdominal imaging showed a cirrhotic liver (MELD score of 13) and splenomegaly. Kayser-Fleischer rings were absent on slit-lamp exam. He was diagnosed with Wilson disease and started on penicillamine and zinc via nasogastric tube due to issues with oropharyngeal dysphagia. Of note, this patient’s brother had a known diagnosis of Wilson disease, having manifested with compensated cirrhosis.

Despite treatment, the patient’s neurological status continued to decline. He lost his ability to communicate and ambulate, became fully dependent on his family who had difficulty caring for him and he had to be hospitalized again. He was then referred for another opinion on management. Subsequently, he was switched to trientine, with the concern that his worsening neurological status was attributed to penicillamine use. While slight improvements were observed in his neurologic status with physiotherapy, these plateaued in such a way that precluded liver transplantation.

This case aims to highlight the rapid deterioration that can be seen in the neurological status in a patient secondary to Wilson disease. Many individuals with neuropsychiatric manifestations of Wilson disease may have cirrhosis, but frequently are not symptomatic from their liver disease, such as this patient who has compensated cirrhosis. Worsening of neurologic symptoms has also been reported in 10%-50% of patients treated with D-penicillamine during the initial phase of treatment. This case also illustrates the importance of screening first-degree relatives of any patient newly diagnosed with Wilson disease. While genetic testing can be done in relatives of index cases, it is expensive, not universally available and sometimes indeterminate. Therefore, a combination of clinical findings and biochemical testing is necessary to aid in the diagnosis. Assessment should include: history relating to liver disease and neuropsychiatric involvement, physical examination, serum free copper, serum ceruloplasmin, liver biochemistries, 24-hour urinary copper and slit-lamp examination of the eyes for Kayser-Fleischer rings. Significant morbidity and mortality could be prevented by early recognition and treatment.

Matthew Kalina

*Cat Scratch Fever*

This case is about the complications that may occur from cat scratches and bites, and management of a patient given a history of cat attack. This case shows the importance of obtaining an accurate history, and correctly diagnosing and treating an infection from cat scratches/bites.

Patient was a 41 year old female with a past medical history of attention deficit hyperactivity disorder, substance abuse, and bipolar disorder, who was admitted to the intensive care unit with diagnoses of acute encephalopathy and septic shock which was attributed to severe cat
scratches and bites on her right lower extremity. The patient’s right lower extremity had numerous scratches and puncture wounds from her boyfriend’s cat that had attacked her two days prior. The right lower extremity was warm with intact pulses and soft compartments, no crepitus or drainage at this time, and requiring no surgical procedures during this hospitalization. Patient was found to have an ejection fraction of 20% on admission with global hypokinesis and troponin elevation that peaked at 0.100. Required push doses of epinephrine in the emergency department, with a lactate slightly elevated to 2.3. Computerized tomography (CT) scan of the head was negative for acute processes, and CT chest, abdomen, pelvis showed atelectasis with superimposed aspiration. Patient became hypercapnic, and due to concerns about her ability to protect her airway secondary to encephalopathy, she was intubated. She was sent to the intensive care unit on Levophed, intravenous Unasyn, azithromycin, and Vancomycin. The patient was also given rabies vaccine and immunoglobulin, though the cat that attacked her was her boyfriend’s and was up to date on vaccines. The patient decided to leave against medical advice on the fifth day, declining penicillin and being sent home with oral Moxifloxacin. She returned to the hospital eight days later with worsening leg wounds and required surgical debridement and allografts.

Blood cultures were negative, though thoughts were this process was related to either Bartonelle Henslae, Capnocytophagia, or Pasteurella. Given the presentation, the latter 2 were thought to be most likely, and her condition improved Unasyn. Notably, she remained afebrile throughout her stay. Patient did require potassium and magnesium replacement during her stay and there was no clear cause. She ultimately left the hospital almost one month after initial presentation, requiring physical therapy and dressing changes for her grafts. This case shows a severe course from cat scratches and bites, and the multiple complications that can occur.

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An Unusual Case of Fever and Abdominal Pain

Introduction: Pyogenic liver abscesses (PLAs) are a common type of intraabdominal abscess and occur in approximately 3.6 per 100,000 individuals. Common presenting signs include fevers, malaise, abdominal pain, nausea, and emesis.

Case Description: A 63-year-old man presented with a 2-day history of nausea, multiple episodes of non-bloody, non-bilious emesis, generalized abdominal pain, malaise, chills, and fevers up to 103 degrees Fahrenheit. Physical exam was notable for right-sided abdominal tenderness to deep palpation without rebound and guarding. Laboratory workup revealed hemoglobin 12.9 g/dL, leukocytes 14.4 x 109/L, aspartate aminotransferase 266 U/L, alanine transferase 201 U/L, total bilirubin 0.9 mg/dL, direct bilirubin 0.7 mg/dL, INR 1.3, albumin 3.2 g/dL, and lactate 2.7 mmol/L. Upon admission, he was resuscitated with intravenous fluids and started empirically on vancomycin and piperocillin-tazobactam. Given the persistent abdominal pain, a computed tomography (CT) scan of the abdomen/pelvis was obtained, which revealed a low attenuating lesion within the right lobe of the liver measuring 5.2 x 5.0 x 5.8 cm, mild diffuse gallbladder wall thickening.
measuring 4-5 mm with pericholecystic fluid and mural edema, mild pericolonic fat stranding in the sigmoid colon, and mild focal colonic wall thickening involving a diverticulum. An abdominal ultrasound revealed a 5.6 cm hyperechoic area in the central liver, consistent with a hepatic abscess, and a thick walled gallbladder filled with stones. A drain was placed into the right hepatic abscess and 12 mL of bloody, opaque, purulent fluid was aspirated. The aspirated fluid and blood cultures returned positive for Streptococcus anginosus. Antibiotics were de-escalated to ceftriaxone and metronidazole, which were switched to ertapenem at the time of discharge and continued for four weeks from the time of drain placement. At discharge, the patient was doing well clinically and his fevers and abdominal pain had resolved.

Discussion: PLAs are associated with significant in-hospital mortality, with rates close to 5.6%, and even higher rates in patients with bacteremia or septicemia. PLAs can form in a variety of ways, including via the portal circulation after bowel leakage and peritonitis, contiguous spread from biliary infection, and hematogenous seeding. Right-sided liver involvement is more common due to its larger size and greater blood supply. CT and ultrasound are the imaging modalities most useful in the evaluation of PLAs. Streptococcus milleri infections (including Streptococcus anginosus), the pathogen present in our case, are classically associated with simultaneous infections at alternative sites than the liver. Treatment involves a combination of antibiotics and drainage.

**A Problem of Complements**

**Background:** Hemolytic Uremic Syndrome (HUS) presenting clinically with hemolytic anemia, thrombocytopenia, and acute renal failure is classically caused by Shiga-toxin producing Escherichia Coli (STEC). However, while rare, there are non-infectious presentations of atypical HUS (aHUS) related to loss-of-function mutations in alternative complement pathway proteins.

**Case Presentation:** A healthy 28-year-old woman presented for evaluation of profound lower extremity edema with worsening renal function including a creatinine of 5.9 and a urinalysis revealing 2+ protein and 3+ blood. On admission, she was afebrile without concern for infection. Hemoglobin was 8.2 and Doppler ultrasound demonstrated no vascular abnormalities. Hemolysis labs showed undetectable haptoglobin, elevated lactate dehydrogenase and normal bilirubin. A peripheral smear revealed schistocytes and helmet cells. C3 and C4 levels were normal and an ADAMSTS13 of 67%. During her hospital course, her creatinine worsened and her hemoglobin and platelets decreased, requiring transfusions and plasmapheresis. Renal biopsy showed subacute thrombotic microangiopathy concerning for aHUS. Subsequent rheumatologic, renal, and hematologic workups were largely negative except for genetic testing showing elevated SMAc with decreased complement factor H consistent with aHUS. Along with supportive care, eculizumab was initiated to control complement dysregulation leading to improvement in hemoglobin, platelet count, and creatinine. In light of the increased risk of life-threatening meningococcal infections with eculizumab, she received a host of
vaccinations and prophylactic antibiotics for two weeks. Following stabilization on eculizumab, she was discharged with close monitoring in the outpatient setting.

Discussion: Clinically, aHUS presents similarly to STEC-related HUS producing anemia, thrombocytopenia, and acute renal failure. However, genetic mutations in complement proteins and autoantibodies are implicated in the atypical presentation seen in both infancy and adulthood. Outcomes are dependent on which complement protein is mutated; Complement Factor H (CFH) is associated with a poor prognosis as many patients’ renal function deteriorates towards end stage renal disease commonly requiring transplant within 12 months whereas CD46 mutations causing aHUS are associated with a slower disease progression. Although initial treatment of HUS is supportive irrespective of cause, eculizumab, a monoclonal antibody with anti-C5 activity, is first-line treatment for aHUS. Important considerations for eculizumab include its prohibitive cost along with the major risk of meningococcal infections, which requires vaccination prior to initiation. Finally, there is an extensive risk evaluation and mitigation strategy (REMS) program to monitor its use, which should be started at the first suspicion of an aHUS diagnosis. By reporting this uncommon case of HUS, we highlight the importance of rare genetic abnormalities that contribute to an atypical presentation of this disease and reinforce life-saving treatment options available to patients.

Michael Klajda
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Polyarteritis Nodosa In A Patient With Pancytopenia

Introduction: Polyarteritis nodosa (PAN) is a necrotizing medium vessel vasculitis affecting vascular supply to critical organ systems such as renal and abdominal viscera and may rarely be associated with myelodysplastic syndrome (MDS). Recognizing this association and diagnosis at an early stage is critical as early management may prevent serious complications.

Case Description: 76-year-old male with a recent diagnosis of low-grade MDS presented to the hospital with complaints of severe fatigue, anorexia, abdominal pain and a 60 pound weight loss over six months. He also had a history of right foot drop about 2 months back of unclear etiology. PET scan at the time showed increased uptake in abdominal lymph nodes. He was subsequently hospitalized 1 month before presenting to our hospital with increasing abdominal pain with CT abdomen showing ileitis, mesenteric lymphadenopathy and colitis and treated with broad spectrum antibiotics with minimal improvement. Initial laboratory studies at our hospital showed pancytopenia with values near baseline, and elevated sedimentation rate of 82 mm/hr and C-reactive protein was 72.5. He had normal electrolytes, creatinine, copper, vitamin B 12, vitamin-D levels. Urinalysis was within normal limits and serum protein electrophoresis did not show any monoclonal proteins. Complement, cryoglobulin levels were within normal limits. His serologies including ANA, ANCA, antiphospholipid antibody levels, hepatitis B and C were within normal limits. EMG of the lower extremities showed involvement of right distal sciatic nerve or both tibial and peroneal nerves. CT enterography and colonoscopy revealed nonspecific inflammation of the colon and nonspecific biopsy findings.
CT angiography revealed aortoiliac atherosclerosis but no evidence of vasculitis. Despite negative work up so far, a mesenteric angiogram was ordered with high clinical suspicion for vasculitis which revealed multiple areas of narrowing along the superior & inferior mesenteric artery. Following this information patient was given pulse dose steroids for 2 days followed by a slow oral taper. Further evaluation of MDS with FISH and T-cell receptor analysis was recommended. Cytosan was not preferred in the setting of MDS with ongoing consideration of hypomethylating agents such as azacytidine.

Conclusion: PAN should be considered in patients with a constitutional symptoms and signs of organ involvement such as abdominal pain, renal failure, mononeuritis multiplex. In patients with MDS, symptoms concerning for PAN should prompt extensive evaluation for vasculitis. Treatment is based on severity. Involvement of the gastrointestinal, nervous, or cardiac system should warrant aggressive treatment with prednisone and immunosuppressive therapy such as cyclophosphamide.

In cases where PAN is associated with MDS, alternative options might have to be considered. Although rituximab could be a possible option, it has not been specifically studied in PAN. Primary therapies for MDS itself such as hypomethylating agents (azacytidine) might be helpful for associated vasculitis.

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Dr. Dennis Regan

Moo Point?

Background: Streptococcus dysgalactiae is traditionally known to cause infections in cattle. However, it has been increasingly identified as a source of both invasive and non-invasive infections in humans. Incidence varies from 1.4 to 6.3 per 100,000 people and mortality has been reported to be as high as 18% in severe infections.

Case Presentation: A 65-year-old male cattle farmer from Minnesota presented for evaluation of acute onset confusion, weakness, and fevers. His past medical history was significant for type 2 diabetes, diastolic heart failure, and hypertension. Initial evaluation revealed a right sided accessory nerve impairment with facial droop and right sided arm and leg weakness. NIH stroke scale was 8. A CT head was negative for hemorrhagic or ischemic stroke. Laboratory workup revealed a leukocytosis of 21.5x10^9 cells/L. A lumbar puncture was negative for pleocytosis, CSF cultures, gram stain, and HSV PCR. Further workup including a vasculitis panel, antiphospholipid antibodies, Lyme panel, and Cryptococcus antigen were negative. On hospital day 2, blood cultures revealed Streptococcus dysgalactiae for which he was started on ceftriaxone. Further evaluation with a MRI revealed multiple, bilateral punctate infarcts primarily involving the left MCA distribution, consistent with a central source of emboli. A transesophageal echocardiogram (TEE) was negative for vegetations and a carotid ultrasound was negative for hemodynamically significant stenosis. A multidisciplinary team of Internal Medicine, Neurology, and Infectious Diseases agreed that despite a negative TEE, his initial presentation was likely secondary to infective endocarditis with Streptococcus dysgalactiae resulting in multiple embolic infarcts. He was treated with IV ceftriaxone for 4 weeks and discharged on Aspirin for stroke prevention. He has not experienced any further recurrence of infection or stroke to this date.
Discussion: *Streptococcus dysgalactiae* (Lancefield groups C and G), part of normal human flora, is primarily known to infect cattle but recently has become more prevalent in the human population, particularly among cattle farmers. Elderly patients with multiple comorbidities and skin breakdowns are the most susceptible. It’s been described that this pathogen can cause illnesses ranging from mild skin and soft tissue infections to life-threatening conditions like necrotizing fasciitis, toxic shock syndrome, and infective endocarditis. *Streptococcus dysgalactiae* has been reported to be the cause of infective endocarditis in 1.1-3.3% of all endocarditis cases with mortality rates from invasive infections ranging as high as 18%. In cases where infective endocarditis and emboli to the brain are involved, ceftriaxone has been shown to be effective due to its ability to penetrate the blood brain barrier. By reporting this uncommon case of infective endocarditis leading to multiple embolic infarcts, we reinforce the increasing prevalence of *Streptococcus dysgalactiae* as a source of invasive infections, recognize the high mortality rate, and reinforce the treatment options available for patients.

**Alice Lehman**

*Thick Blood, Heavy Heart: A case of Hyperviscosity Syndrome*

Hyperviscosity syndrome (HVS) can significantly impact patient morbidity. This case demonstrates the difficulty of timely diagnosis for HVS, in addition to how etiology helps guide the direction of the definitive management.

A 54yr old female presented with fatigue, gum bleeding, and a hemoglobin of 6.5. Smear demonstrated rare blasts, initiating marrow biopsy on Day4. Day11, the patient developed dysarthria and focal extremity parathesias, where MRI demonstrated new hemorrhagic conversion of CVA. Emergent plasmapheresis (PLEX) was performed. Clinical course was further complicated by acute heart failure, PEAs, retroperitoneal hemorrhage, and AKI needing CRRT in addition to multiple emergent PLEX treatments. Viscosity index ranged from 1.7 to 9.5, with IGM 944, IGG 2520, and positive cryo-kappa, cryo-IGM. Marrow demonstrated atypical lymphoid and plasma infiltrates. Patient was diagnosed with Waldenström's macroglobulinemia (WM) and Bortezumab was initiated on day 9. Patient was found to be Hepatitis C positive with a high viral load. Harvoni was initiated on day 22. Patient now resides in a longterm care facility.

HVS is a clinical diagnosis consisting of a vague presentation found in conjunction with "viscous" blood. A delayed diagnosis can have profound impact on patient’s morbidity, making early diagnosis crucial. Incomplete understanding of laboratory measures contribute to delayed diagnoses. Laboratories use a capillary tube to measure the time required for a serum sample to flow through the tube under the influence of gravity, known as viscosity index(1). HVS is rare when viscosity indices are < 4.0. Cryoglobulinemia can result in a temperature dependent elevation of serum viscosity indices (2). IgG paraproteins increases the serum viscosity proportionally, while viscosity rises exponentially when IgM paraproteins are > 3g/L (3). There exists a consistent viscosity index at which HVS symptoms present in the same WM patients(4) thereby reflecting the utility of knowing protein composition. The
clinical value of viscosity indices lie in the patient specific viscosity threshold above which symptoms of HVS appear, thereby helping guide preemptive treatment with PLEX.
PLEX reduces plasma viscosity approximately 30% per session; however etiology of HVS guides definitive treatment. Our patient’s simultaneous presentation of HCV and WM questions the correlation and relative contribution of both components to the patient’s HVS. Our patient presented with mixed cryoglobulinemia, characterized by deposition of immune complexes containing Rheumatoid Factor (RF), IgG, and HCV RNA. Studies describe links between HCV and B cell dyscrasias through IgG bound HCV driving the clonal expansion of RF + B cells(5). HCV eradication is the most effective treatment for HCV mixed cryoglobulinemia(6). When active HCV and WM present concurrently, differentiating contributing factors is difficult, therefore often delaying necessary treatment.

This case highlights the importance of viscosity indices, paraprotein composition, and HCV status to help determine emergent and definitive treatment for patients with hyperviscosity syndrome.

**Shall We Thrombolyze?**

Acute hemiparesis should prompt immediate investigation. One of the most common reasons for acute onset hemiparesis is an ischemic stroke. Time is of the essence as early thrombolysis reduces morbidity and mortality. However, it is important for physicians to consider other causes based on careful history and physical examination.

An 82-year-old lady with a history of type II diabetes mellitus, hyperlipidemia and hypertension presents with a sudden onset of left arm and leg weakness. She was sitting in church when she developed left-sided neck and shoulder pain, followed shortly thereafter by left-sided hemiparesis. She had no trauma and was not taking any antithrombotic medications. On examination, she had 0/5 power in the left upper and left lower limbs. Sensation was preserved. Other neurological examinations, including cranial nerves was normal. Her calculated NIH Stroke Scale was 8. Blood tests revealed normal platelet counts, APTT and INR. A non-contrast CT scan of the head revealed no abnormalities.

A telestroke consultation was initiated to address whether IV thrombolytics should be administered. Because of the neurologist’s concern about the ongoing left neck and shoulder pain, a CT angiography of the neck and chest was performed, which excluded arterial dissection or intramural hematoma. Because of the ongoing clinical suspicion, an MRI of the cervical spine was performed, revealing a left epidural hematoma extending from C2-C5 with associated compression of the spinal cord. The patient began to spontaneously recover neurologic function after the MRI and was managed conservatively.

Epidural hematomas are usually seen in the setting of trauma, even minimal trauma. When it occurs spontaneously, it is seen usually in the setting of a bleeding predisposition, such as thrombocytopenia or
antithrombotic therapy. There are case reports in the literature of spontaneous cervical epidural hematoma with no identifiable cause. This case highlights the importance of good history taking and being suspicious when the history ‘does not make sense’. The presence of persistent neck pain in the setting of acute hemiparesis should prompt evaluation for etiologies other than ischemic stroke. If this patient had received IV thrombolytics, it certainly could have led to disastrous consequences. Primum non nocere.

**Angie Lobo Romero**  
**Dr. Peter Lund**  

**Spontaneous Coronary Artery Dissection: The Importance of Considering Non-atherosclerotic Acute Myocardial Infarction in Women**

Introduction: Spontaneous coronary artery dissection (SCAD) is an increasingly recognized etiology of non-atherosclerotic acute coronary syndrome.

Case description: A 59-year old woman with systemic lupus erythematosus and type 2 diabetes mellitus presented to the emergency department with right facial droop, headache, and right arm weakness. Physical exam and imaging were diagnostic of acute ischemic stroke. Electrocardiogram showed normal sinus rhythm without ischemic changes. Transthoracic echocardiography demonstrated normal left ventricular function (LV), akinetic apex and a large LV thrombus. Coronary computed tomography angiography (CCTA) revealed severe proximal left anterior descending (LAD) artery stenosis with possible distal LAD thrombus, and non-invasive CT-FFR of 0.63 suggestive of a flow-limiting lesion. CCTA 3 years prior had not shown coronary disease. Invasive coronary angiography demonstrated 90% stenosis the proximal LAD with severe distal disease. Intracoronary imaging using optical coherence tomography (OCT) confirmed diagnosis of spontaneous coronary artery dissection. Patient underwent successful stenting and was discharged on a statin, clopidogrel, warfarin, metoprolol and methylprednisolone.

Discussion: SCAD is increasingly recognized as an important etiology of non-atherosclerotic acute coronary syndrome, especially in middle-age women. It has been associated with other conditions, such as pregnancy and fibromuscular dysplasia, but it should also be considered in women with connective tissue diseases, such as SLE. SLE can lead to inflammation within the arterial wall, and promote atherosclerosis. Uncontrolled SLE may lead vasculitis and subsequent dissection of the intimal wall. This case illustrates an atypical presentation, in which the patient presented a ischemic stroke leading to a work-up that yielded the identification of an LV thrombus plus apical hypokinesis requiring further coronary evaluation. Coronary imaging was essential, first CCTA to establish the diagnosis of a severe proximal LAD stenosis, and second invasive angiography with OCT to confirm the diagnosis of SCAD. The treatment of SCAD remains a matter of debate. Some studies favor conservative management, whereas others favor percutaneous coronary intervention with stenting, mainly when associated with unstable symptoms and impaired coronary blood flow. This case illustrates several points. First, the importance of a meticulous evaluation in patients with an ischemic stroke, including the need for echocardiography to assess for cardioemboli. Second, the need to assess for coronary artery disease when LV thrombus is present. Lastly, the
nuances of coronary disease, not all due to atherosclerosis, with intracoronary imaging using OCT proving critical to diagnose SCAD.

Conclusion: SCAD is an etiology of ACS that occurs in the absence of atherosclerosis, mainly occurring in middle-age women. Its diagnosis requires a meticulous assessment and high degree of suspicion. For patients with suspected ACS, imaging techniques such as CCTA, intracoronary imaging such as OCT or intravascular ultrasound, are important tools to delineate the mechanism of ACS, mainly in women without evidence of overt atherosclerosis.

Luke Lundeen

**Ertapenem Induced Encephalopathy**

Case Description: A 74 year old female with history of renal transplant on immunosuppresives with normal post-transplant renal function presented to the hospital for evaluation of a 4 day history of gait instability and poor coordination, slurred speech, and hallucinations now using a walker to support ambulation when previously able to hike up inclines on rough terrain 3-4 miles per day. Denies headache, numbness to lower extremities or new bowel/bladder incontinence. She had completed a 10 day course of IV ertapenem for urinary tract infection the day prior to admission. Her physical exam was notable for wide based gait and dependence on a walker, but neurologic exam was otherwise nonfocal. Workup was unremarkable, including complete blood count, electrolytes, renal function, liver function, lumbar puncture, EEG, CT venogram of the head, and CT of head, thoracic, and lumbar spine. On the second day of hospitalization it was further understood that her gait instability was related to her significant visual hallucination burden, with the patient attempting to dodge out of the way of small children she was hallucinating in the hallway while walking. Patient progressively improved throughout the hospitalization without intervention and returned to near baseline function upon discharge following a six day hospitalization. Her hallucinations were presumed to be secondary to ertapenem toxicity given the negative workup and her improvement following discontinuation of the medication for 72 hours.

Discussion: Visual hallucinations are a known but very rare side effect of ertapenem, more commonly seen in renal failure patients, although there are reports in patients with normal renal function as well. Ertapenem should be considered as a possible cause of profound neurologic changes, especially in elderly patients and those with impaired renal function. Effects of ertapenem toxicity on the central nervous system have been demonstrated to last 3-14 days following discontinuation of the medication.

Teija Madhusoodanan

**Eosinophilia, Rash, and Substance Abuse: DRESS Syndrome without a Clear Offending Agent**

Drug reaction with eosinophilia and systemic symptoms (DRESS) syndrome is a rare, potentially life-threatening condition characterized by a diffuse rash with eosinophilia. The clinical presentation can include rash, facial edema, fever, lymphadenopathy, increased serum alanine aminotransferase, reactivation of viruses, and possible organ damage.
The patient is a 34 year old female with past medical history significant for type I diabetes mellitus, end stage renal disease on hemodialysis, and hypertension presenting with diffuse pruritic rash that began two days prior to admission along with facial swelling. Per the patient’s nursing home, the patient did not have any recent medication changes. The patient was vitally stable. The most notable physical exam findings were significant facial edema and diffuse rash. She had erythematous urticarial papules that coalesced into plaques on the entire body including extremities and trunk. WBC count was mildly elevated at 11.09 and peaked at 19.4. Absolute eosinophil count was 1.77 and peaked at 9.89. Infectious workup for parasites was negative. IgE was within normal limits, HIV was negative, T-spot was negative. Liver function tests were remarkable for elevated alkaline phosphatase 417, ALT 72, AST 95, direct bilirubin 0.4. Skin biopsy showed perivascular inflammation with abundant eosinophils. Bone marrow biopsy with cytogenetics and flow cytometry did not reveal a malignant cause of eosinophilia.

Using the DRESS scoring scale, she was categorized as possible DRESS syndrome and she was treated as DRESS syndrome to avoid the consequences of potential organ damage or death. There was stepwise discontinuation of offending agents as outlined by Dermatology. Systemic treatment was somewhat delayed as it was unclear if patient’s preexisting eosinophilia was masking new eosinophilia. It was not until her eosinophil count began rising that systemic treatment was initiated. Trial of cyclosporine was attempted, but eosinophil counts did not reduce, therefore requiring high dose steroids, which were hoped to be avoided given patient’s history of labile blood sugars. With adequate steroid dose and discontinuation of most prior to admission medications, patient began to have improvement in eosinophil count. Blood drug screen found to have unexplained oxycodone, methamphetamine, and diazepam though patient continued to deny any drug use, therefore no illicit drug could be pinpointed as the offending agent. Unfortunately no clear agent was specified in causing this drug eruption.

Use of the DRESS scoring scale can be helpful in determining whether to treat a suspected case of DRESS syndrome. When determining offending agents, sometimes none may be clearly identified. In these instances, treatment is still warranted. DRESS syndrome though rare, is important to consider in a patient with a combination of the above symptoms because if not properly diagnosed can be potentially fatal resulting in end-organ damage and possible death.

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Dr. Michele LeClaire

Delayed Metastasis of Primary CNS Lymphoma: Fact or Fiction?

A 41-year-old man with a long standing history of smoking was diagnosed with primary diffuse large B-cell lymphoma (DLBCL) of the CNS following biopsy of a right parietal lobe mass after he presented with a three to four-week history of progressive left sided weakness and dizziness. Staging included bone marrow biopsy, CT scan of the neck, chest, abdomen and pelvis, ocular exam and CSF analysis, none of which revealed the presence of lymphoma.

His treatment plan included high dose methotrexate, rituximab and
temozolomide as this regimen has been shown to lead to a significant clinical response in up to 80% of cases. After completing two cycles he refused further chemotherapy and instead completed a course of whole brain radiation therapy. The right parietal brain mass decreased in size over time based on serial brain MRI.

One year later, he presented with altered mental status and was found to have hypercalcemia (serum Calcium 17). An x-ray of the left shoulder obtained for shoulder pain revealed an incidental lung nodule in the left upper lobe. A follow-up CT chest confirmed the left upper lobe ground glass nodule, and additionally demonstrated an 8 cm lung mass in the medial right upper lobe with involvement of mediastinal structures. A bronchoscopy was performed and complete obstruction of the right upper lobe bronchus was detected.

The patient’s history of smoking, the central location of the mass, and the presence of hypercalcemia as a possible para-neoplastic syndrome all supported the notion that this could be a primary malignancy, likely small cell lung cancer. Endobronchial biopsy however, confirmed the mass to be DLBCL. Repeat bone marrow biopsy at this time again showed a normocellular bone marrow.

It is unclear whether this patient developed metastases from a primary lesion in the brain or whether the mass in his lung was a distinct lesion. Primary central nervous system lymphoma (PCNSL) almost always remains confined to the nervous system with systemic involvement being exceedingly rare. Per our review, only six such cases have been previously described. It is interesting to note that it can occur even in the event of significant local response to treatment. The development of subsequent histo-pathologically distinct malignancies after the diagnosis of PCNSL has also been described and one study found the incidence of these to be 8%.

Therefore, screening for recurrence and occurrence of second malignancies should be considered in long-term follow-up of patients with PCNSL.

**Cassedy Mahrer**

**Bill White**

*A Case of Cerebral Salt Wasting: Approaching Hyponatremia in the Setting of Brain Hemorrhage and Fanconi Syndrome*

Introduction: Cerebral salt wasting causes an ADH-mediated hyponatremia in the setting of central nervous system dysfunction (typically subarachnoid hemorrhage) resulting in inappropriate natriuresis with subsequent hypovolemia. Diagnosis is complicated by clinical similarity to the syndrome of inappropriate anti-diuretic hormone secretion (SIADH). Failure to distinguish between the two could cause adverse consequences due to the differences in management.

Case Description: A 42 yo woman was admitted after being found down at home. She was found to have a ruptured middle cerebral artery aneurysm leading to intraparenchymal hemorrhage and underwent surgical drainage and clipping with subsequent admission to the ICU. Aggressive IV hydration with normal saline was initiated. Over several days, the patient’s daily urine output (UOP) increased from 4 L, to 15 L,
to 26 L on POD12.

The patient’s volume status on examination was consistent with euvolemia, though on TTE the IVC was 1.4 – 1.6 cm, suggesting mild hypovolemia despite aggressive volume resuscitation. Other studies were significant for a drop in her sodium from 150 on admission to 128, urine sodium 202, and non-anion gap metabolic acidosis, and positive urine anion gap. Hypertonic saline was started, but did not affect the sodium.

Cerebral salt wasting was suspected as the cause for her natriuresis given her dependence on IV fluids (indicating hypovolemia) in the setting of intracranial bleed. On further inspection, her labs were consistent with Type II Renal Tubular acidosis and urinalysis early in admission demonstrated proteinuria and glucosuria, making Fanconi syndrome likely cause. Aggressive electrolyte supplementation was initiated, hypertonic saline was continued. The following day her serum sodium normalized and her urine output decreased. All saline infusion was stopped. Urine sodium and osmolarity were confirmed to have normalized. The patient discharged to rehabilitation on POD21 and has made a dramatic functional recovery.

Discussion: The most likely diagnosis in our patient was Cerebral salt wasting (CSW) because of the low serum sodium, high urine sodium and low IVC with a dependence on IV fluids to maintain euvolemia. The patient’s preexisting Fanconi syndrome (a cause of proximal RTA) likely contributed to hypovolemia through solute diuresis or reduced concentrating capacity.

CSW is a rare diagnosis. There is controversy over whether CSW exists at all. The diagnostic criteria for SIADH and CSW are nearly identical, and differ only in the volume status. CSW is associated with hypovolemia (via decreased sympathetic output to the kidney) and SIADH presents in euvolemia. The treatment of these conditions are fluid repletion and fluid restriction, respectively. Therefore it is worth the time to distinguish between the two, as mistreatment could result in increased patient morbidity.

**Anti-Histamines: A Risk Factor for Exertional Heat Stroke?**

Introduction: Exertional heat stroke (EHS) is a life-threatening disorder resulting from exercised induced hyperthermia. Multiple organ systems are affected. Encephalopathy, rhabdomyolysis, liver and kidney injury are characteristic. Unlike non-exertional heat stroke, which typically effects the geriatric population, exertional heat stroke is seen in otherwise healthy adults and adolescents in the setting of vigorous exercise.

Case Description: A 27 y/o female with no significant prior medical history was nearing the end of a 6 mile foot race when she became confused and collapsed. Bystanders on the scene noted that her skin was hot to the touch. She was covered in ice packs and was taken to a local emergency department. Soon after arrival she became agitated and combative. She required heavy sedation and was intubated for airway
protection before being transferred for further care. On arrival she was hemodynamically stable. Physical examination was unremarkable. Laboratory evaluation was notable for: sodium, 140 mmol/L; [135-145 mmol/L]; creatinine, 1.7 mg/dL [0.8-1.3 mg/dL]; creatine kinase, 5696 U/L [38-176 U/L], lactate, 9.82 mmol/L [0.6-2.3 mmol/L], pH, 7.18 [7.35-7.45], and troponin T, 0.74 ng/mL [<0.01 ng/mL]. ECG showed so ischemic changes. Urine drug screen was negative for controlled substances. CT scan of the head, chest, and abdomen showed no abnormality. Blood cultures, cerebrospinal fluid cultures, and viral studies were obtained and returned negative. She was treated aggressively with intravenous fluid hydration and improved rapidly. She was extubated within the first 24 hours. Following extubation, she reported severe muscle soreness and intermittent double vision that resolved after 12 hours of rest. She noted that she had taken over-the-counter Loratadine prior to the race, which she had never done before. After 48 hours of hospitalization, her laboratory abnormalities were markedly improved and she was dismissed home.

Discussion: Exertional heat stroke (EHS) is a life-threatening disorder resulting from exercised induced hyperthermia (typically a temperature >40.0°C). EHS occurs when the body’s thermoregulatory mechanisms are insufficient to dissipate excessive amounts of heat. Risk factors include environmental conditions, prescription medications, illicit substances, and patient factors such as level of physical fitness. Antihistamines may increase risk of EHS by inhibiting cholinergic sweat mechanisms. Treatment of EHS consists of rapid cooling, aggressive IV fluid hydration, and supportive care. Early recognition is critical to ensure good clinical outcomes.

Endocarditis is a rare condition that can present in a variety of ways including constitutional symptoms, splenomegaly, and stroke. However, despite advances in imaging and antibiotic therapy, the illness continues to have a significant risk of mortality and morbidity, especially when unrecognized on initial evaluation.

A 50-year-old man with a history of type 2 diabetes mellitus and mitral valve prolapse presented to an outside facility with subacute fevers, diarrhea, unintentional weight loss and acute onset right homonymous hemianopia. He was found to have infarcts in both the posterior and middle cerebral artery territories, as well as a sedimentation rate of 100 mm/hr. Transthoracic echocardiogram and CT angiogram were negative for causative etiology. He was discharged on atorvastatin and clopidogrel for secondary stroke prevention. Throughout the next month, the patient continued to have constitutional symptoms, as well as progressive, severe low back pain. He presented to the hospital for further evaluation which was revealing for anemia (hemoglobin 8.6 g/dL), leukocytosis (white blood cells 14.7 x 109/L), splenic infarction on abdominal ultrasound, hematuria and granular casts on urinalysis, new first-degree heart block on electrocardiogram (PR interval 210 milliseconds), and blood cultures positive for streptococcal species. Physical exam revealed a 3/6 holosystolic murmur at the apex with radiation to the axilla. Further work-up for presumed infective
endocarditis was pursued. Transesophageal echocardiogram revealed severe mitral regurgitation with two mitral vegetations on the A1 and P1 scallops measuring 9x5 millimeters and 13x9 millimeters, respectively. An MRI of the brain showed multiple new, small foci in the right caudate head, frontal lobes, left periventricular white matter, and left cerebellum. A spinal MRI demonstrated early osteomyelitis at the level of L1-L2. Speciation of blood cultures demonstrated S. mutans. A Panorex was unremarkable for oral etiology of this patient’s endocarditis; however, on further history he mentioned that several months prior, his dentist noted five untreated caries during a routine examination. Throughout the hospitalization, the patient was treated for his symptomatic pain and dehydration, and he was discharged with six weeks of outpatient antibiotic therapy with ceftriaxone with plans for subsequent mitral valve repair. Ultimately, the patient required a mechanical mitral valve replacement and tricuspid valve repair.

This case demonstrates the importance of early recognition of endocarditis as patients may face many complications throughout its course. As seen in this patient, complications include osteomyelitis, heart block, glomerulonephritis, and embolic phenomena such as stroke and splenic infarction. Thorough cardiac and infectious evaluation is critical in patients presenting with stroke to prevent the morbid sequelae of endocarditis.

Nicholas McDonald
Dr. John Ratelle

Zebras and Horses: Bleeding in a Patient with Neurofibromatosis

Acute anemia is an extremely common problem in the hospital, affecting as many as three-quarters of medical inpatients. Anemia can be defined as a hemoglobin <13-14 mg/dL in men or hemoglobin <12 mg/dL in women. Acute blood loss is an important and potentially life-threatening cause of anemia and should be promptly evaluated. However, there are many insidious causes of acute blood loss and a comprehensive differential diagnosis should be entertained.

A 40-year-old woman presented to an outside hospital with acute right flank pain and hematuria. Her past medical history was significant for Neurofibromatosis type-1, s/p anterior and posterior fusion, VP and peritoneal shunts. She underwent computed tomography scan (CT) and was found to have a right renal infarct due to a 1.7 cm renal artery aneurysm. She was started on Heparin to mitigate further thromboembolic phenomena. The following day, she developed worsening pain and acute anemia. A repeat abdominal CT showed a new, large, right-sided flank hematoma, possible right-sided paraspinal pseudoaneurysm and an incidental 3.2 cm paraspinal mass. She was transferred to our hospital for possible intervention.

Vital signs and neurologic exam were normal. Neurosurgery and interventional radiology were consulted and she underwent CT angiography (CTA) to evaluate for active bleeding. CTA revealed redistribution of the hematoma without active bleeding and resolution of the previously seen pseudoaneurysm. She was not a candidate for IR intervention due to the location of her aneurysm (distal third of the right main renal artery with segmental arteries arising from the aneurysm).
During her fifth hospital day, she continued to improve clinically, but her hemoglobin continued to downtrend from 9.3 mg/dL (previously 10.5 mg/dL at OSH) to 8.0 mg/dL. There was concern the patient may have developed re-bleeding into her hematoma so an urgent CT was obtained. Fortunately, her CT findings were stable and after further discussion with the patient, it was revealed she was on day 5 of her menstrual cycle. On recheck, her hemoglobin stabilized and she continued to clinically improve. She was discharged home on Aspirin with a plan to follow-up with vascular surgery in 4-6 weeks for repair of her renal artery aneurysm. Her paraspinal mass was thought to be a hamartomatous lesion which could be followed with MRI in 5 years.

This case emphasizes the importance of keeping a broad differential and revisiting alternative causes when the clinical picture does not match the data. As it applies to acute anemia in the hospital, consider the following insidious causes of blood loss; hematoma, menstruation, iatrogenic (blood loss from phlebotomy), hemolysis and gastrointestinal bleeding. Furthermore, this case highlights that patients with rare conditions can still have common causes for their presentation!

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**Claire McNeil**  
Dr. Elizabeth Rogers; Dr. Michael Aylward  

**Sex Chromosome Analysis for Infertility: A Case for Cultural Humility and Multidisciplinary Care**

With 10-15% of couples affected by infertility, general internists frequently encounter this issue. This personal and value-laden matter demands physicians practice cultural humility. While not unique to infertility, the work-up may present internists with both the opportunity and responsibility to explain genetic testing results.

A 28-year-old Somali female with schizoaffective disorder and possible cognitive delay presented with concern that despite having sexual intercourse 4-5 times monthly over one year of marriage, she had not conceived. Since menarche at 16 years old, she had irregular cycles every 2-3 months, lasting 2-3 days. She reported her husband’s infertility testing showed no abnormalities. Medications included metformin for diabetes mellitus and the atypical antipsychotic, lurasidone.

Physical exam revealed a 5-foot tall female in hijab with limited eye contact and flat affect, speaking mostly in Somali. No abnormalities of the skin, facial hair growth, or thyroid noted on exam. After initially declining genital exam, later exam showed Type III female circumcision, normal hair distribution, and pale vagina with loss of rugae and thin, white, non-odorous discharge and no other abnormalities of genital structures.

Initial laboratory work-up included: negative urine HCG, TSH 1.99 U/L, prolactin 8 ng/mL, Hemoglobin A1C 6.6%, LH 38 IU/L, and FSH 64.2 IU/L. LH and FSH levels were in the postmenopausal range, concerning for premature ovarian insufficiency (POI). Repeat lab testing showed: LH and FSH in similar range and estradiol level of 22 pg/mL, consistent with follicular phase, confirming diagnosis of POI. After consultation with Gynecology, additional work-up included: normal transvaginal ultrasound, negative Antibody testing (thyroid peroxidase enzyme, anti-thyroglobulin antibody, 21-hydroxylase antibody), and negative testing for fragile X. Sex chromosome analysis with FISH interphase testing revealed XXX female.
Trisomy X (47, XXX) is the most common female chromosomal abnormality, occurring in approximately 1 in 1,000 females. Most are only mildly affected and likely only the minority diagnosed. Psychiatric disorders and cognitive delays are seen more frequently in Trisomy X females. Thus, in addition to this patient’s POI diagnosis, her mental health diagnoses may also be associated with her karyotype, with exact mechanism yet unknown.

Medical and mental health treatment for this patient has focused on multiple attempts to give thorough information via a multi-specialty team regarding the facets of health impacted by this diagnosis: bone and cardiovascular health, need for IVF and oocyte donation if pregnancy is desired, and mental health treatment and supports. While she started supplemental calcium and vitamin D, she has not initiated hormonal therapy, indicating intermittently that she believes she may get pregnant if God wishes. This highlights the need for multi-disciplinary clinical care with culturally appropriate, transparent communication to best support the patient.

**Charles Meade**

*There Will Be Blood: Massive Hemoptysis in Primary Lung Neoplasm*

A 57 year old man with a 4 month history of squamous cell lung presented to an outside hospital with a 1-day history of hemoptysis and acute respiratory distress. The patient was intubated and sedated and transferred to our facility. Physical exam revealed frank blood visible from the ET-tube and admission labs showed a hemoglobin of 10.8 g/dl.

Emergent bronchoscopy visualized blood originating from the left lung. A cavitary lesion was seen extending into the lung parenchyma through which a possible vessel could be appreciated. No actively bleeding areas were visualized and an endobronchial blocker was advanced into the left mainstem bronchus. Following bronchoscopy no further frank hemoptysis occurred. Hemoglobin levels plateaued at 7.4 g/dl.

Subsequent cross-sectional imaging revealed a left lower lobe cavitary lesion communicating with the left lower lobe bronchus. Within the cavitary lesion a contrast-enhancing segmental branch of the left lower lobe pulmonary artery was seen without evidence of contrast extravasation.

In the interventional radiology suite angiography re-demonstrated a segmental left lower lobe pulmonary artery pseudoaneurysm. The contributing inferior and posterior pulmonary artery branches were embolized using platinum coils. Repeat arteriography demonstrated no filling of the pseudoaneurysm. The patient was discharged after three days with no further episodes of hemoptysis.

Pulmonary artery pseudoaneuerysm (PAPs) are an uncommon cause of hemoptysis and airway compromise. PAP rupture is an emergent, life-threatening event associated with significant mortality. PAPs are an important consideration in the differential for massive hemoptysis as a source arising from the pulmonary arterial circulation, in contrast to much more common sources arising from bronchial circulation.
PAPs complicating primary lung neoplasms remain rare. Amongst the described cases, the most common presenting symptom is massive hemoptysis. In 8/9 identified cases, the underlying primary malignancy was of squamous cell origin. In 4/9 cases the underlying primary lung cancer was undiagnosed at presentation. The remaining cases all occurred within 6 months of initial diagnosis.

The pathogenesis of PAPs in primary lung cancers is poorly understood. In PAPs associated with tuberculosis or pneumonia, contiguous spread of the infectious agent leads to damage which predisposes the involved vessel to pseudoaneurysm formation. The hypothesized mechanism for PAP formation in the setting of primary lung malignancy is similar, necessitating direct tumor invasion of the vessel wall. Central cavitation and subsequent arterial invasion appears to be pre-requisite for PAPs in primary lung neoplasms. On imaging PAPs associated with primary lung neoplasms are found either within cavitary lesions or filling former cavitary spaces.

This case highlights PAPs as a rare, early complication of lung neoplasms. There appears to be an association with squamous cell pathology, cavitary lesions, and male gender. The standard of care in management of PAPs is minimally invasive intravascular coiling and embolization.

Scratching the Surface: Cutaneous Clues to a Deadly Microbe

Mycobacterium tuberculosis (TB) is the number one cause of death worldwide due to a single infectious agent. Fortunately, screening and recognition of the classic manifestations and risk factors for TB have led to effective diagnosis and treatment. However, atypical presentations persist, and subsequent treatment can be delayed. Erythema Induratum (EI) is a rare, though significant, cutaneous manifestation of tuberculosis. Recognition of EI is critical to effective diagnosis of both active and latent TB infection (LTBI).

A 24-year-old Vietnamese woman immigrated to the United States in 2011. She was referred to the Infectious Disease clinic three months postpartum for a positive interferon-gamma release assay that was discovered during the first trimester of pregnancy. A chest X-ray was conducted and showed no signs of pulmonary TB. She denied cough, hemoptysis, and night sweats. She did endorse back pain and purplish skin nodules that developed four to six years prior. She elaborated that the skin lesions started on her legs but progressed to her arms over the last year. They were slightly tender and waxed and waned in number. Her family history was notable for a brother with extrapulmonary drug-susceptible TB disease treated several years ago. Physical examination revealed indurated, violaceous nodules measuring 0.5 to 1 cm in diameter on the posterior aspects of the legs and thighs. A few nodules were noted on her arms and the anterior aspects of her legs. Some nodules showed overlying desquamation, but none were ulcerated or suppurative. There were also separate areas of hyperpigmentation consistent with resolved lesions. Biopsy obtained from a right arm nodule showed perisepal lobular mixed granulomatous inflammation and vasculitis consistent with EI. PCR for TB and acid-fast stain of the tissue were both negative. One month later, prominent cervical
lymphadenopathy was noted. Subsequent PET-CT demonstrated a centrally cavitating, hypermetabolic lesion in the left iliacus with involvement of the left sacroiliac joint and lumbosacral bones, and multiple hypermetabolic cervical and axillary lymph nodes. The iliacus abscess was evacuated, and the peritoneal fluid was PCR and culture positive for drug-susceptible TB. She was treated with directly observed standard four-drug therapy for active TB.

This case demonstrates that in the absence of typical TB signs and symptoms, EI can be a clue to latent or extra-pulmonary TB infection. In non-endemic areas, EI can be mistaken for Erythema Nodosum (EN). While EN can also be associated with TB infection, other causes, including streptococcal infection and idiopathic cases, are more common. Moreover, EI is only rarely associated with non-tuberculous etiologies, and several retrospective studies suggest a strong association between EI and TB infection. EI can be distinguished from EN on the basis of distinct clinical and pathologic features of the lesions in question.

A Rare Complication of Adult-Onset Still's Disease

Introduction: Adult-Onset Still’s Disease (AOSD) is an inflammatory syndrome characterized by spiking fevers, evanescent rash, and arthritis. While rare, it should be recognized by the general internist as it can be accompanied by serious complications such as Macrophage Activation Syndrome (MAS).

Case Description: This is a case of a 27-year-old woman with history of Graves’ disease and AOSD who presented to hospital with one month of fevers, fatigue, diffuse arthralgias, and intermittent rash. Prior to presentation, she was treated with variable doses of prednisone for management of AOSD. She was recently prescribed methotrexate as a steroid-sparing agent but she had not started this yet.

On exam she was febrile, tachycardic, and tachypneic. She was ill appearing with a diffuse non-confluent maculopapular rash over her arms, legs and chest. Joint exam revealed diffuse tenderness to palpation without evidence of active synovitis.

Initial labs were significant for pancytopenia and markedly elevated inflammatory markers (including a ferritin of 47000 ng/mL) and triglycerides of 190 mg/dL. CT chest revealed numerous small mediastinal lymph nodes, right paratracheal adenopathy and a prominent spleen. Of note, initial exam and imaging did not reveal a clear etiology of underlying infection.

She was started on intravenous methylprednisolone 125 mg daily on admission for possible AOSD flare. On day 2, she remained persistently febrile with worsening neutropenia and a rising ferritin. Given some atypical features of her AOSD flare, such as neutropenia, thrombocytopenia, hypertriglyceridemia, as well as a lack of response to high-dose prednisone there was concern for concomitant MAS secondary to AOSD. Therefore, high-dose IL-1 inhibitor therapy with anakinra was initiated on day 2. Over the following 3-4 days, she
became afebrile and her objective markers such as WBC, inflammatory markers, and ferritin began improving. On day 7, she was discharged home with oral prednisone and daily anakinra until follow up with rheumatology.

Discussion: This case emphasizes the difficulty of diagnosing MAS in the setting of underlying rheumatologic disorder. In our case the patient already had AOSD, an uncommon disorder, making its complications even more rare. MAS can share features with an AOSD flare, but must be distinguished given the need for distinct therapy and the severe morbidity and mortality associated with this condition.

Samantha Morley

_A New Tumor Plop: Syncope as the Presenting Symptom of Renal Cell Carcinoma_

Case Description: A 69-year-old female with a history of chronic kidney disease stage 3, hypertension, and hyperlipidemia presented to the emergency department with recurrent syncope, instigated by leaning forward. Laboratory studies were notable for a thrombocytopenia with platelet count of 103,000 (normal range 150 – 450 x 10^9 / L) and an elevated serum creatinine of 1.3 (normal range 0.6 – 1.1 mg/dL). EKG showed normal sinus rhythm with right axis deviation. Transthoracic echocardiogram revealed a right atrial thrombus, prolapsing across the tricuspid valve (TV) into the right ventricle during diastole. Subsequent CT of the chest/abdomen/pelvis demonstrated a 10 cm right renal mass with tumor thrombus invading the inferior vena cava and extending into the right heart. MRI chest/abdomen with magnetic resonance venography again depicted the large right renal mass and the associated mobile tumor thrombus, extending from the right renal vein into the right ventricular apex. Preoperative transesophageal echocardiogram showed severe TV regurgitation from tumor prolapse across the native valve.

Discussion: Renal cell carcinoma (RCC) can present with a wide range of symptoms determined by the extent of disease. The classic triad of flank pain, hematuria, and a palpable abdominal mass occurs in less than 10% of patients. In patients with localized disease, common presenting symptoms include hematuria, abdominal pain, and weight loss. Even more common is the incidental detection of renal masses with abdominal imaging before symptoms arise. IVC involvement has been reported in 4-10% of patients with renal neoplasms. Clinically, this manifests as venous congestion with lower extremity edema and ascites or distal embolic disease. Tumor thrombi in RCC are classified into groups I through IV, based on the cephalad extent. Group I thrombi involve the renal vein, but do not extend into the IVC. Group II involve the infrahepatic IVC. Group III extend into the retro-hepatic or supra-hepatic IVC, but do not reach the right atrium. Group IV, as seen in this case, extend into the right atrium. Group IV thrombi occur in less than 1% of RCC cases. Even rarer, is the extension of the thrombus from the right atrium into the right ventricle. Few cases have been reported and of these cases, none presented with syncope. They presented with symptoms of right heart failure with or without hematuria. In this case, we suspect that intermittent prolapse of the tumor thrombus across the TV temporarily prevented flow across the valve, severely reducing right
ventricular preload and causing syncope.

Teaching Points: 1. RCC presents with associated tumor thrombus in 10% of cases. 2. Rarely, tumor thrombi extend into the right heart. 3. Patients such extensive thrombi typically present with symptoms of right heart failure. 4. When tumor thrombus affects the TV, patients may present with syncope.

Travis Mrkvicka  
Dr. Anya Jamrozy

Heart of Iron: Modern Diagnosis of Hereditary Hemochromatosis

Hereditary hemochromatosis is the most common genetic disorder in people of northern European descent. Many physicians still consider hemochromatosis to be a rare disorder, although reported prevalence is 1:200 in Caucasians. The classic triad of cirrhosis, bronze skin and diabetes is rare; the more common presentation is nonspecific fatigue and myalgias.

A 54 year old previously healthy Caucasian male presented with chest discomfort, dizziness, dyspnea and fatigue. He had rarely doctored prior to presentation and had no significant family history. He had 2 encounters throughout the previous year, one including an overnight observation stay, which failed to find any definitive cause for his symptoms. His third encounter was to the emergency room where an EKG displayed atrial fibrillation with rapid ventricular response. He was found to have a hemoglobin a1c of 6, mildly elevated LFTs and a TSH of 29. He was also found to be thrombocytopenic, mildly leukopenic and anemic. All were thought to be possible sequela of excessive alcohol use and primary hypothyroidism. He was started on a beta antagonist as well as levothyroxine and advised to abstain from alcohol. The patient was discharged with plans for an outpatient cardiac evaluation. Echocardiogram revealed a left ventricular ejection fraction of 35%. A nuclear medicine stress test displayed a medium sized area of antero-septal ischemia. Subsequent coronary angiography showed normal coronary arteries. A cardiac MRI then revealed evidence of cardiac siderosis. An MRI of the liver displayed evidence of hepatic iron deposition as well as a 2.7 cm liver mass. Iron studies confirmed hemochromatosis and genotyping found the patient to be homozygous for the C282Y mutation of the HFE gene.

An US guided needle biopsy was done of the liver mass, which was diagnostic for cholangiocarcinoma. The patient's subsequent course was complicated by worsening heart failure, worsening diabetes, and significant confusion. A brain MRI revealed iron deposition in the basal ganglia, red nuclei and substantia nigra. Iron chelation therapy was initiated. Eventually the patient was stabilized and able to be discharged to a transitional care unit. He underwent CT guided radiofrequency ablation and to date continues chemotherapy to treat his cholangiocarcinoma.

As evidenced in this patient's case, hereditary hemochromatosis can have significant effects on the liver, heart, pancreas, and pituitary. Considering the diagnosis early when subtle symptoms are present can
prevent significant morbidity. All patients with liver disease should be evaluated for hemochromatosis. Similarly, unexplained cardiomyopathy or conduction disturbances should prompt consideration of HH evaluation as this is the sole presentation in 15% of cases. Availability of cardiac MRI will likely increase our diagnosis of late stage HH; our goal should be increasing early identification.

Warda Niaz

Hooked on Prolactin

Introduction: A 33-year-old female presented to Endocrinology clinic for galactorrhea and 8 year history of infertility. Found to have normal prolactin levels on multiple checks. Extensive work up done per gynecology was unremarkable for definite cause of infertility. Underwent testing with serial dilution of prolactin levels to elevate for the ‘hook effect’, with confirmation of elevated prolactin levels.

Case Description: A 33-year-old female presents to Endocrinology clinic for galactorrhea and 8 year history of infertility. Initially presented to gynecology clinic for evaluation of infertility. She had regular predictable menstrual cycles and no history of sexually transmitted disease or abdominal surgeries. Prolactin levels were 13.8 ng/mL, 5.0 ng/mL and 14.4 ng/mL (normal range 6.0-29.9 ng/mL) on multiple checks. TSH levels were 1.2 uIU/ml (within normal range). Ultrasound pelvis showed normal endometrial strip and stable persistent right ovarian fibroma. Also underwent hysterosalpingogram which showed patient fallopian tubes and normal uterus. Mammogram showed BI-RADs category 2 and left breast ultrasound showed a 7 mm simple cyst. Work up was otherwise unremarkable. Upon presentation to endocrinology clinic she underwent serial dilution testing to test for falsely low prolactin levels. This phenomenon is otherwise known as the ‘hook effect’. Recheck of prolactin levels using this method showed elevated prolactin levels at 86.0 ng/mL. Pituitary MRI showed a 9 x 6 x 10 mm nodule in pituitary gland with subacute hemorrhage confirming diagnosis of prolactinoma. Patient started treatment with cabergoline twice weekly and within 1 month of starting therapy was able to conceive spontaneously. Pregnancy test was positive and patient was advised to stop taking cabergoline. Repeat TSH levels were found to be high and T3 levels were low so was started on low dose levothyroxine therapy for central hypothyroidism.

Discussion: Clinicians should be aware of this laboratory phenomenon when evaluating pituitary masses. When the hook effect is suspected, dilution testing of prolactin samples may prevent incorrect diagnosis and warrant correct treatment for patients with prolactinomas.

Edra Nordstrom

Dr. Miguel Ruiz; Dr. Paula Skarda

Cardiobacterium Hominis Endocarditis of a Bioprosthetic Aortic Valve

The HACEK organisms are a group of fastidious gram-negative bacteria that are a rare cause of infective endocarditis and only responsible for 1.4–3% of all cases of the disease. Prosthetic valve endocarditis treatment is more difficult than treatment of native valve endocarditis and may require surgical replacement of the prosthesis in addition to antibiotic therapy.
Our patient is a 64 year old male with a past medical history significant for aortic and tricuspid valve repair in 2007 secondary to MRSA endocarditis, SJS/TEN while on vancomycin, levofloxacin, and piperacillin-tazobactam, who presented with chest pain, malaise, nausea, vomiting, abdominal pain, and decreased appetite. Physical exam was remarkable for poor dentition, a III/VI holosystolic murmur best heard in the upper sternal border, and diffuse abdominal tenderness. He had a slightly elevated troponin at 0.18 (0.00 - 0.03), ESR was elevated at 87, and CRP was elevated at 25.5. CT abdomen and pelvis revealed an area of attenuation in the spleen thought to be a splenic infarct. Because of the CT findings and the murmur, patient underwent a work-up for endocarditis. The TTE showed severe aortic insufficiency but the valves were overall poorly visualized. A TEE was obtained which showed a vegetation on the bioprosthetic aortic valve. The blood cultures from two consecutive days were positive for gram variable organisms which the Minnesota Department of Health identified as Cardiobacterium hominis by 16S ribosomal sequencing. This organism is part of the HACEK group and it has been associated with endoscopies but our patient did not have endoscopic procedures prior to his presentation, therefore, he likely developed Cardiobacterium endocarditis due to poor oral hygiene. Because the organism is fastidious, the blood cultures took 12 days to finalize, during which time the patient was receiving different antibiotics. Although treatment of choice is a third-generation cephalosporin, we were unable to use it in our patient because he developed an allergic reaction to ceftriaxone. He also developed a morbilliform rash with meropenem and aztreonam. Ceftriaxone desensitization was attempted but failed as patient again developed a rash. He was treated with gentamicin, however he developed worsening renal function with new proteinuria. Levofloxacin desensitization was accomplished successfully and the patient was discharged on a six-week course of levofloxacin. He has follow-up with Dentistry for dental extractions and CV surgery for valve replacement of the aortic valve after clearance of his infection.

This case stresses the importance of considering the HACEK organisms as a rare cause of endocarditis not only of native valves but also of bioprosthetic valves. It also emphasizes the complexity of treatment when a patient has serious allergies to multiple antibiotics. In addition, it highlights the importance of good dental health in patients with a history of endocarditis.

John Ogden

Get Wellens' Soon

Case Description: An 81 year old female with a past medical history of breast cancer status-post surgery, chemo, and radiation, hypertension, and hyperlipidemia presented to the emergency department with a 2 day history of intermittent chest discomfort. The first episode came on when she was cleaning her horse stables. The pain was described as a squeezing/tightness that started in the mid upper back and radiated to the neck and anterior upper left chest. She also endorsed some dizziness and nausea with the pain. The pain lasted for approximately 10 minutes and was relieved by rest. She had a total of 5 episodes over two days, each coming on with exertion and resolving with rest. In the Emergency Department, she was pain free with no symptoms.
Initial troponin was negative and chest x-ray showed no acute findings. EKG showed a biphasic T wave in V2 and V3 with deep T wave inversions in leads V4-V6. Previous EKG from earlier in the month only showed T wave inversions in V2. Repeat EKG showed deep T wave inversions in V2-V6. Repeat troponin was slightly elevated at 0.02. This pattern was concerning for Wellens’ syndrome. She was loaded with aspirin and ticagrelor, started on heparin, and admitted for an urgent angiogram. In the cath lab, angiography revealed a tight 90% stenosis in the LAD. A drug eluting stent was placed and blood flow was restored. The patient did well post-operatively and was discharged home two days later.

Discussion: Wellens’ syndrome is a rare but serious presentation of impending myocardial infarction. It is characterized by deep T wave inversions in the anterior and septal leads, most commonly seen in V2-V3. Alternatively, it can also present with biphasic T waves in these leads. Usually the patient is pain free when the EKG is performed. Cardiac enzymes are normal or may be slightly elevated. Wellens’ syndrome implies a severe stenosis of the proximal LAD coronary artery. It is important that it is recognized soon, as 75% of untreated patients’ progress to an acute anterior wall MI within days to weeks.

Vignesh Palaniappan
Dr. Sally Mung Ting Yung;
Dr. Cuong Pham

A Tough NUT to Crack: Empyema in a Young Patient

NUT midline carcinoma is a rare, aggressive, undifferentiated carcinoma. It is defined by rearrangements of the NUT gene often with coding sequences fused with the BRD3 or BRD4 gene, leading to a fusion oncogene that inhibits cell differentiation. These cancers arise in the midline, occur in all age groups, and carry a poor prognosis. Pulmonary Actinomyces is a similarly rare diagnosis that often masquerades as malignancy and causes severe chest pain and suppurative pneumonia with a propensity to fistulize. Here we present a challenging case where these two infrequent findings coalesced.

A 29 y/o female initially presented to the hospital with dyspnea, severe chest pain, cough, and RML consolidation. She was treated for community-acquired pneumonia and discharged with opiates due to continued pleuritic chest pain. One month later, she presented to outside hospital with a persistent RML consolidation and hypoxia. CT scan and bronchoscopy did not show evidence of malignancy, and the patient was discharged with IV antibiotics via PICC line and narcotics for continued chest pain. Two weeks later, she presented to our hospital with worsening chest pain, hypoxia, hemoptysis, and new large right pleural effusion. Chest tube was placed with evidence of empyema growing polymicrobial flora including Actinomyces odontolyticus. Despite pleural drainage and IV antibiotics, the patient’s effusions and clinical picture did not improve significantly. Evaluation for acquired/inherited immunodeficiencies was begun due to recurrent pneumonia in a young patient, and bronchoscopy with EBUS was repeated. This showed evidence of extrinsic compression of the RML and RUL concerning for mediastinal mass. Biopsies confirmed NUT Midline Carcinoma with the BRD4-NUT rearrangement. The PET scan that followed showed evidence of metastatic disease to the mediastinum, ovary, and bone. The
patient received palliative radiation to the mediastinal mass that was causing recurrent pneumonia. Due to the extent of the empyema caused by her Actinomyces pneumonia, she required a prolonged course of antibiotics before experimental treatment for her NUT midline carcinoma could be considered.

Diagnosis and management of this patient were challenging for a number of reasons: 1) Our patient initially presented as a young healthy female, and the major concern was for inherited or acquired immunodeficiencies given recent CT and bronchoscopy findings. 2) Actinomyces’ species’ propensity for fistulization led to empyema, which obscured her diagnosis, and delayed her cancer therapy as she could not enroll in clinical trials until off antibiotics 3) Her chest pain due to Actinomyces and cancer complicated her workup due to the patient’s reticence to undergo further painful testing.

Overall, this case highlights the importance of considering malignancy in a young patient, the fulminant nature of NUT midline carcinoma, and the challenges of treating mediastinal malignancies when they present as post-obstructive pneumonia.

Samuel Philbrick  
Dr. Janelle Gyorffy;  
Dr. Adrian Bersabe;  
Dr. Richard Upton;  
Dr. Derek Mathis;  
Dr. Austin Peters;  
Dr. Alexander Brown

A Unique Case of Muscle-Invasive Metastatic Breast Cancer Mimicking Myositis

Introduction: Breast cancer is the most commonly diagnosed cancer in women with an estimated lifetime risk of about 12.5%. Metastases to muscles of the neck are almost unheard of, with only one previous case documented in medical literature. We present a unique case of a patient who presented with neck swelling and upper airway obstruction due to metastatic breast cancer invading the sternocleidomastoid muscles.

Case: An 84-year-old female with no known history of malignancy presented with progressive neck swelling and dysphagia leading to difficulty controlling secretions. Urgent evaluation with otolaryngology was arranged, during which the patient acutely developed respiratory failure requiring emergent fiber-optic nasotracheal intubation and transfer to the medical intensive care unit. CT was remarkable for moderate narrowing of the supraglottic airway and extensive inflammatory stranding of muscular and fascial planes in the neck, mediastinum, and anterior chest. A course of steroids failed to reduce swelling or improve symptoms. Inflammatory markers were normal and autoimmune workup was negative. Biopsy of the left sternocleidomastoid muscle revealed infiltrating carcinoma. Follow-up bilateral breast ultrasound showed hypoechoic masses in the left and right breast highly suggestive of malignancy (BI-RADS 5). Serum tumor markers were remarkable for elevated CA 15-3 and CA 27.29, consistent with a breast primary malignancy. The diagnosis was discussed with the patient and she elected for palliative tracheostomy, a single dose of anti-estrogen therapy with Fulvestrant, and palliative radiotherapy. The patient died in the care of home hospice services.

Discussion: This case demonstrates a rare instance of breast cancer metastases to the sternocleidomastoid muscles and resultant airway obstruction. Airway obstruction rarely occurs in cancers of the breast.
English-language case reports describing breast cancer metastases causing dysphagia and airway obstruction document masses in the nasopharynx and sinuses, not invasion of the neck musculature. Moreover, the nature of the muscular invasion makes this case truly unusual. Skeletal metastases in breast cancer are rare, and seem to occur as islands in an intrinsically hostile microenvironment created by skeletal muscle’s pH, ability to activate lymphocytes and natural killer cells, and mechanical motion. Such metastases have been described as well-defined masses with clear margins. In defiance of this trend, in this case neoplastic cells spread in the midst of skeletal muscle and fibro-connective tissue, even producing myositis. Instead of polymyositis or dermatomyositis comorbid with malignancy, biopsy confirmed that inflammatory changes seen on imaging were the direct result of cells invading skeletal muscle. The unusual cause of this patient’s airway obstruction highlights the value of biopsy for patients with progressive dyspnea, dysphagia, and signs of myositis on imaging. The origin of the patient’s myositis further emphasizes the need to maintain a high index of suspicion for neoplasm for any patient presenting with an indeterminate myositis.

Mitchell Pitlick  
Dr. Kenneth Warrington

**Microscopic Polyangiitis Misdiagnosed as Systemic Sclerosis**

Autoimmune conditions can be difficult to diagnose given the non-specific and overlapping symptoms that can occur. Serologic tests aid in the diagnosis of specific conditions, but are often given more weight than the patient’s overall clinical presentation, which has the potential to cause misdiagnosis.

A 75-year-old woman presented to her physician for chronic cough occasionally productive of yellow sputum. She also complained of reflux and Raynaud’s phenomenon. She had no skin thickening, telangiectasias, or calcinosis. A CT scan showed lower-lobe predominant interstitial fibrosis. Laboratory investigation revealed negative anti-nuclear and anti-centromere antibodies, a weakly positive SCL-70 antibody, and a positive p-ANCA with a corresponding strongly positive myeloperoxidase antibody. A clinical diagnosis of systemic sclerosis was made, and the patient was treated with azathioprine.

She presented to the emergency department five years later with worsening shortness of breath. A chest CT was concerning for pneumonia, so she was admitted for IV antibiotic therapy. Soon after admission, she developed swelling, pain, and erythema of her ankles. An aspiration of the right ankle showed non-inflammatory synovial fluid. She soon developed similar symptoms in her left elbow, metacarpophalangeal, and proximal interphalangeal joints. This was accompanied by painless hemorrhagic papules on her hands and fingers. A transthoracic echocardiogram did not show any valvular vegetations, and blood cultures were negative. Repeat testing revealed an erythrocyte sedimentation rate of 140 mm/h, negative SCL-70 and anti-centromere antibodies, and a positive p-ANCA with a corresponding positive myeloperoxidase antibody. A urinalysis showed minimal proteinuria with a bland urinary sediment. Kidney biopsy was offered, but the patient declined. Biopsy of a skin papule revealed occlusion of dermal vessels with neutrophilic leukocytoclasia. A clinical diagnosis of
microscopic polyangiitis was made. She was treated with 40 mg of prednisone daily and saw improvement in her skin and joint symptoms.

This case illustrates the pitfalls of relying too heavily on serologic tests for diagnosing autoimmune conditions. These tests must always be interpreted in the clinical context. In this case, a weakly positive SCL-70 antibody was used to support a diagnosis of systemic sclerosis, despite the fact that the patient lacked classic cutaneous findings of this illness. Microscopic polyangiitis was a unifying diagnosis that was supported by serology and the clinical presentation. The distinction is important, as patients with systemic sclerosis are precluded from receiving high-dose steroids given the risk of precipitating renal crisis. This patient showed a need and positive response to steroid treatment.

**A Case of Desquamation**

Identifying rashes in hospitalized patients can often present a unique challenge, especially when the clinical picture is complex. The differential diagnosis of desquamating rashes includes potentially life threatening conditions. Toxic shock syndromes can be characterized by fever, hypotension, multi-organ damage as well as diffuse rash. This can often overlap with the somewhat more benign drug rashes as well as the severe but more rare spectrum of Stevens-Johnson Syndrome. It is important to consider a broad differential and act quickly when presented with desquamating rash.

In this case, a 75 year-old man with history of CAD, cardiomyopathy, CKD, PAD, and 3 year history of leg wounds presented with foot pain. Over the preceding 2 months he noted increasing leg pain, swelling, drainage, and shaking chills. There had been no new medications within several months and no other apparent triggers. The day of presentation, he was seen in his primary care clinic where he was noted to be hypotensive and tachycardic with a red rash on his arms and stomach. Unable to walk due to foot pain, he was sent to the emergency room where he was noted to have bilateral bright erythema, foul-smelling drainage and desquamation of the legs to the mid-shin. Further exam revealed papular red rash across arms, chest, abdomen, palms, and thighs with no vesicles, desquamation, or other secondary skin changes. The mucous membranes were spared. Labs notable for moderate acute kidney injury and elevated CK. He was started on ceftriaxone and clindamycin. His statin was held due to previous adverse reaction. His vitals rapidly stabilized. The papular rash spread to flanks and back over the following days and he developed desquamation of the palms and arms. Biopsy showed subacute spongiotic dermatitis, which was nonspecific (immunofluorescence was negative). His cellulitis gradually improved, he had no further episodes of hypotension and his creatinine returned to baseline. His rash stabilized (never involving mucous membranes). He was discharged on cephalexin to complete his treatment for severe cellulitis with possible toxic shock.

This patient was initially hypotensive, but without documented fever, and with end-organ damage of 3 systems (AKI, altered mental status, and elevated CK). He had diffuse erythroderma and desquamation of his feet on presentation but proceeded to desquamate his hands and arms.
This case was initially treated as possible toxic shock syndrome but was also consistent with an erythematous drug reaction. SJS spectrum was considered but was less likely given lack of mucous membrane involvement. This case demonstrates that knowledge of the differential for desquamating rashes is critical in distinguishing between several severe conditions to allow the practitioner to act quickly and effectively by treating with antibiotics and considering steroids, IVIG or transfer to a higher level of care.

<table>
<thead>
<tr>
<th>Clay Reed</th>
<th>An Unexpected Cause of Dyspnea</th>
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<td>Dr. Meltiady Issa</td>
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Dyspnea is a common symptom in patients hospitalized with underlying malignancy. While the differential might be broad and challenging, it is important to recognize certain acute etiologies that could be life threatening if not diagnosed and treated in a timely manner.

A 67-year-old woman with recently diagnosed squamous cell carcinoma of the right upper lung presented to the emergency department with increased lower back pain and bilateral lower extremity weakness. She had a past medical history of COPD, atrial fibrillation, CHF, and complete heart block status post dual chamber pacemaker. Her home medications were continued including aspirin 81mg Qday and apixaban 5mg BID. On hospital day three, she began to develop progressive shortness of breath and right upper chest discomfort. She was afibrile with tachycardia and hypoxia. No jugular venous distention or lower extremity edema was present. An electrocardiogram showed no acute ST or T wave changes, but chest X-ray was concerning for obstructive pneumonia. She was started on broad-spectrum antibiotics, but she continued to deteriorate clinically and continued to be tachycardic and tachypneic. A chest CT angiogram demonstrated bilateral pulmonary emboli despite being anticoagulated with apixaban. Doppler ultrasound confirmed bilateral thrombi in the lower extremities. The apixaban was stopped, and she was started on a high dose heparin infusion. She was then transitioned to a low molecular weight heparin (LMWH) 1 mg/kg subcutaneously BID.

This case demonstrates the importance of keeping pulmonary embolus on the differential in hospitalized cancer patients even if they are anticoagulated with a novel oral agent. These agents are still not FDA approved for venous thromboembolism (VTE) prophylaxis in patients with malignancy given the absence of safety and efficacy data in cancer populations. Head-to-head randomized controlled trials or robust comparative effectiveness studies are needed and are currently underway. Until then, patients admitted with underlying malignancies should receive LMWH for first-line VTE prophylaxis treatment.

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<th>Muneeb Rehman</th>
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<td>Dr. Nikhil Kolluri; Dr. Matt Hardman; Dr. Guiherme Piovezani Ramos; Dr. Thomas Poterucha</td>
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True Soul?

Background: Thrombophlebitis migrans (TM) is characterized by recurrent, migratory episodes of segmental thrombosis that affect superficial veins. Although TM is associated with pancreatic tumors as Trousseau Sign, it is also seen with other solid tumors, most notably mucinous adenocarcinomas. Diagnosis should prompt an evaluation for underlying malignancy.
Case Presentation: A 65-year-old female presented with left-arm swelling and erythema. She reported a recent history of multiple episodes of superficial clots involving her lower extremities. On exam, she had spider angiomata of the chest with bilateral lower extremity lymphedema. Laboratory evaluation showed hemoglobin of 9.6 g/dL and albumin of 2.1 g/dL. Lower Extremity ultrasound was negative for deep venous thrombosis, but revealed thrombosis of great saphenous veins bilaterally. Computerized tomography of abdomen and pelvis showed liver cirrhosis with multiple hypoattenuating lesions and a cystic 9-cm mass on her right adnexa. Right Upper Quadrant Ultrasound confirmed cirrhotic nodules without malignant masses. Patient was started on warfarin and subsequently developed hematomas on the right-anterior abdominal wall and left chest wall. She was found to have Protein C deficiency and warfarin was discontinued. Pelvic ultrasound showed a stable 9-cm cystic mass that was biopsied with anatomopathological evaluation showing bland epithelioid cells consistent with benign serous cyst. Further evaluation for age appropriate screening including colonoscopies, pap smears, and mammographies were unrevealing for underlying malignancy. Hematologic evaluation confirmed genetic variant responsible for Protein C (PC) deficiency. She has not developed further episodes of superficial thrombosis.

Discussion: TM is an acquired coagulopathy strongly associated with malignancy. They appear to be triggered by the interaction of circulating mucin secreted by carcinoma cells with platelet and leukocyte selectins. Other diseases have also been linked to TM, including Buerger’s disease, inflammatory bowel disease, coagulation defects such as deficiencies of protein C or S, or lupus anticoagulant. Multiple factors could have promoted the development of TM in the present patient: ovarian mass and hypercoagulable state secondary to PC deficiency. The benign character of her mass speaks against this as the cause. Of note, PC deficiency has been previously reported in a case of TM. Thus, it seems the most likely cause of her TM. PC is a vitamin K-dependent anticoagulant protein synthesized in the liver. Its primary role is to inactivate coagulation factors Va and VIIIa. Deficiency of PC can lead to a hypercoagulable state. Occasionally, the diagnosis of thrombophlebitis precedes the development of neoplasm by months or even years. Therefore, it is possible that the patient may develop a neoplasm in the future. By reviewing a common presentation of TM in the setting of an unusual precipitant, we review common aspects of diagnosis, management and treatment of TM and Protein C deficiency.
showed extensive alveolar opacities and bilateral pleural effusions. Subsequent echocardiogram revealed severe mitral regurgitation with mitral valve lesions consistent with Libman-Sacks endocarditis. Further laboratory testing showed a positive ANA, anti-dsDNA, anti-phospholipid IgG, anti-beta 2 glycoprotein, and anti-cardiolipin antibodies. She was started on enoxaparin and aspirin for presumed antiphospholipid syndrome along with intravenous diuresis for congestive heart failure thought to be secondary to her valvular disease. She responded well, her respiratory status returned to baseline, and she was discharged on warfarin.

Two weeks after discharge, the patient returned to the ED with encephalopathy, fever, and acute renal failure. Family members reported a three day history of somnolence, hallucinations, and abnormal language. Brain MRI showed extensive cerebral and cerebellar infarcts secondary to microembolization. Laboratory evaluation revealed a hemoglobin of 6.9, platelets of 56,000, creatinine of 3.6 mg/dL, elevated fibrinogen, and elevated LDH. An ADAMTS13 assay was within normal limits. She was started on an unfractionated heparin drip, broad-spectrum antibiotics, and pulsed IV methylprednisolone for catastrophic antiphospholipid syndrome. Her renal function and respiratory status continued to worsen over the first 48 hours and she was started on hemodialysis and plasma exchange. Bronchoscopy also revealed diffuse alveolar hemorrhage. Her hospital course was further complicated by severe pseudomembranous colitis and a hemodialysis line infection. It was later discovered that three days after her first hospitalization, the patient stopped taking warfarin as prescribed due to doubts about its necessity. At the time of dismissal, the patient was on a steroid taper and was advised to continue anticoagulation indefinitely as discontinuation of anticoagulation was thought to incite catastrophic antiphospholipid syndrome (CAPS).

Discussion: Clinicians should be aware of antiphospholipid syndrome (APS) and the critical importance of anticoagulation. In rare cases, APS progresses to CAPS causing vascular occlusion of multiple organs. This condition can be difficult to recognize and is associated with a mortality rate as high as 50%. There have been several case reports of APS progressing to CAPS upon discontinuation of anticoagulation, which was thought to be the inciting event in this case.

Juan Ripoll
Dr. Mahrurk Rizvi; Dr. Danile Johnsrud; Dr. Zachary Yetmar; Dr. Rebecca King; Dr. Craig Daniels

Severe Babesia Microti Infection Presenting as Multi-Organ Failure in an Immunocompetent Host

Babesiosis is a tick-borne disease caused by protozoa of the genus Babesia. The severity of the infection ranges from an asymptomatic disease to fulminant illness, primarily depending on the immune status of the host. The infrequency of immunocompetent hosts presenting with multiorgan failure and shock makes Babesiosis a formidable diagnostic and therapeutic challenge.

A 67-year-old farmer with an intact immune system presented to an outside medical institution after a two-week history of fatigue, shortness of breath, non-productive cough, and intermittent fevers. Laboratory studies were remarkable for hyperglycemia (343 mg/dL), anemia...
(hemoglobin 11.4 g/dL), and thrombocytopenia (platelet count 49x10^9/L). A certain diagnosis was not initially apparent, and because of symptomatic dyspnea and a CT-pulmonary angiogram revealing signs of early emphysema, he was discharged home on a regimen for COPD exacerbation. Over the ensuing days, the patient’s symptoms persisted while he developed progressive worsening respiratory status. He represented to the emergency department with shock and hypoxemic respiratory failure requiring prompt intubation and fluid resuscitation. Laboratory studies were remarkable for worsening anemia (hemoglobin 8.2 g/dL), thrombocytopenia (platelet count 53x10^9/L), hyperglycemia (668 mg/dL), metabolic acidosis (lactate 6.7 mmol/L; bicarbonate 12 mmol/L; anion gap 29 mmol/L), and acute kidney injury (creatinine 5.66 mg/dL). The patient was initiated on vasopressors and antibiotics for septic shock, and insulin drip for presumed diabetic ketoacidosis. He was emergently transferred to our institution.

On arrival to the intensive care unit (ICU), he demonstrated refractory shock and anemia (hemoglobin 5.6 g/dL). A peripheral smear was ordered, which demonstrated absence of microangiopathic hemolytic anemia. There was no clinical evidence of bleeding. A closer review of the peripheral smear displayed red blood cell inclusion bodies consistent with Babesiosis. The patient was started on clindamycin and loaded with intravenous quinidine, and subsequently transitioned to quinine. Broad-spectrum antibiotics were discontinued. As a result of the significant burden of the disease (12.5% of red blood cells infected), a red cell exchange transfusion was pursued with improvement of the parasite load (1.9%). Babesia microti PCR was positive and the assessment for co-infection of tick-borne pathogens was negative. Two-days later, the patient was extubated and weaned-off of vasopressors. The ICU course was complicated by oligo-anuria requiring continuous veno-venous hemodialysis followed by intermittent hemodialysis. He was discharged on clindamycin/quinine and scheduled for outpatient intermittent hemodialysis due to persistent oligo-anuria.

This case illustrates the potential for severe presentation of Babesiosis in immunocompetent hosts. In addition, it highlights the value of exchange red blood cell transfusions among patients with severe parasite burden. Cases are most common in Northeastern and upper Midwestern United States, however, endemic areas of Babesia are expanding. A high degree of clinical suspicion of this entity is paramount for prompt recognition, early treatment, and avoidance of complications.

**Alexander Ryu**
Dr. Michael Toledano

*A Case of Lyme Neuroborreliosis Presenting with Constipation, Hyponatremia and Weakness*

Lyme disease is the most common vector-borne illness in the United States. Nervous system involvement is common and usually manifests as meningitis, cranial neuritis, and radiculoneuritis. Herein, we present an unusual case of Lyme radiculitis notable for severe constipation, hyponatremia, and leg weakness.

A 62-year-old woman presented with three weeks of low back pain later accompanied by constipation and progressive bilateral leg weakness. She initially experienced onset of lancinating lower back pain radiating
down her legs weeks after a brief diarrheal illness. Roughly two weeks later, she also developed worsening symmetric leg weakness and paresthesias. Additionally, she perceived difficulty supporting her torso while walking and reported absence of bowel movements for 15 days.

On admission, vital signs were unremarkable. Her sodium was 117 mmol/L. Exam revealed symmetric flaccid paraparesis and areflexia. Despite her paresthesias, she lacked identifiable sensory deficits. Her abdomen was mildly distended but soft, with normoactive bowel sounds. Laboratory findings were consistent with the syndrome of inappropriate antidiuretic hormone secretion (SIADH).

Given history and examination, Guillain-Barré syndrome (GBS) was suspected. Cerebrospinal fluid (CSF) however, revealed a protein of 649 mg/dL and a lymphocytic pleocytosis with 363 cells/mcL. Magnetic resonance imaging (MRI) showed thoracolumbar root enhancement without nodularity; nerve conduction studies and electromyography (NCS/EMG) demonstrated corresponding denervation with axonal damage but no demyelination. Further querying revealed frequent tick exposure without rash. Serum and CSF Lyme serology was positive, as was CSF Lyme polymerase chain reaction. The patient was started on intravenous ceftriaxone and fluid restriction. Following 12 days of inpatient rehabilitation, she was discharged home normonatremic, with improving strength and constipation.

Neuroborreliosis typically presents as meningitis and cranial neuropathies, with or without radiculitis. Painful radiculoneuritis in the absence of clinical meningitis has been described and is likely under-recognized. GBS-like presentations are very rare and can occur either due to direct infection or as post-infectious phenomena. Our patient’s history and clinical presentation were strongly suggestive of GBS. MRI was compatible but the extent of thoracic involvement was atypical. NCS/EMG did not show demyelination which is seen with most GBS variants. Importantly, the marked CSF pleocytosis strongly favored an alternative diagnosis. Hyponatremia due to SIADH is common in GBS but has only rarely been reported in neuroborreliosis. The etiology of this SIADH remains unclear. Additionally, cases of neuroborreliosis with severe constipation are rare but exist in the literature. In our case, this was initially attributed to GBS-associated autonomic dysfunction but our patient lacked other signs of dysautonomia. A likely contributor may have been significant abdominal muscle weakness in the setting of thoracic radiculitis.

Lyme neuroborreliosis can present with isolated radiculoneuritis mimicking GBS. Careful history taking and awareness can expedite diagnosis and initiation of treatment.

| Omid Salaami |
| Dr. Dennis Manning |

Lower Back Pain with a Twist of Lyme

A 62 year old woman with no significant past medical history presented to an outside emergency department for progressive lower back pain limiting ambulation. She initially experienced one week of sharp, shooting pain down the posterior aspect of her lower extremities and later developed a dull lower back ache that persisted for three weeks. She also had associated mild, bilateral lower extremity weakness. Four
days before presentation, her weakness suddenly progressed so that she could no longer ambulate independently and she developed lower extremity paresthesia that ascended to involve her trunk. In addition, she did not have a bowel movement for two weeks and endorsed urinary retention. She denied any fevers, night sweats, weight changes, rashes, arthralgia, myalgia, or headaches. There was no history of trauma, prior malignancy, sick contacts, or recent travel. She is an avid gardener but denied any tick bites. Four weeks previous, she was diagnosed with viral gastroenteritis that resolved with supportive care. In the outside emergency department, a CT lumbar spine was unremarkable and she was incidentally found to have hyponatremia on laboratory evaluation.

Upon admission, physical exam was remarkable for diminished strength in the bilateral iliopsoas, quadriceps, and hamstring muscles; diminished sensation to touch from the hip to ankles; and reduced patellar reflexes. Urinalysis showed inappropriately elevated sodium and osmolality consistent with SIADH. An MRI of the lumbar and sacral spine showed abnormal bilateral diffuse nerve root enhancement from at least C6 to the conus level, suggesting an inflammatory process. Lumbar puncture was remarkable for high protein (629 mg/dL), total nucleated cells (363 cells/mcL; predominately lymphocytic), and normal glucose. CSF flow cytometry was negative for malignancy. A comprehensive panel for infectious, rheumatologic, and other etiologies was positive for Lyme serology (IgM and IgG) and PCR in the CSF. EMG showed evidence of acute thoracolumbar polyradiculopathy affecting L2-L4. Antiganglioside antibodies were negative. The patient was started on a twenty eight day course of IV ceftriaxone for neuroborreliosis.

This case illustrates multiple important points. First, Lyme disease classically has three stages: early localized infection (hallmark of erythema migrans); early, disseminated (flu-like symptoms, myalgia, 15% develop neurologic involvement ranging from meningitis, cranial neuritis, to peripheral radiculoneuropathies); late, persistent (arthralgia, transverse myelitis, polyneuropathy). However, the presentation can be varied, such as in this case marked by Bannwarth Syndrome (lymphocytic meningoradiculitis), SIADH, and as a mimic of Guillain-Barre syndrome (GBS). Second, absence of an inciting tick bite and erythema migrans can be seen in Lyme disease. Third, although Lyme disease and GBS have significant clinical overlap, albuminocytologic dissociation is characteristic for GBS and can help differentiate the two conditions as it did in this case.

Wil Santivasi
Dr. John Lieske

*Not for Kids Only: New-Diagnosis Henoch-Schönlein Purpura in an Adult*

Introduction: While IgA vasculitis, or Henoch-Schönlein purpura, is most often considered a pediatric disease, up to 10% of cases occur in patients over the age of 18. Compared to children, adult patients with IgA vasculitis are more likely to present with palpable purpura, are more likely to demonstrate renal involvement, and are less likely to experience gastrointestinal effects.

Case Presentation: A 40-year-old man presented to the emergency department with a month-long history of “foamy” urine and two-week
history of a worsening rash on his hands and feet. On presentation, he was found to be hypertensive to 145/87, with heart rate 97 and temperature 36.8°C. Physical examination disclosed non-blanching, violaceous palpable purpura in a centripetal distribution. Initial laboratory evaluation revealed normal kidney function (serum creatinine 0.9), elevated blood glucose (281), serum albumin of 2.7, and total cholesterol of 241. Urinalysis revealed large occult blood, 3+ protein, 31-40 red blood cells (>25% dysmorphic), 4-10 white blood cells, hyaline casts, granular casts, fatty casts, free fat, and oval fat bodies. C-reactive protein was elevated to 16 (nl < 8.0). A 24-hour urine collection confirmed 3.3 grams of total protein. A biopsy of a skin lesion revealed leukoclastocytic vasculitis by light microscopy with IgA deposition within papillary dermal vessels by immunofluorescence. A kidney biopsy revealed mild segmental mesangial proliferative glomerulonephritis with significant mesangial IgA deposition. There were no crescents or diabetic changes. Thus the diagnosis of IgA vasculitis (Henoch-Schönlein purpura) was made. A tapering course of oral prednisone was initiated (40 mg to 10 mg over 4 weeks). On follow up at 3 weeks the rash had resolved. On follow up at 8 weeks, proteinuria had improved substantially (24-hour excretion 819 mg).

Discussion: This case illustrates a surprising, though not uncommon, diagnosis in an adult patient that presented with rash and heavy proteinuria. While the most common causes of nephrotic-range proteinuria in adults are diabetic nephropathy, focal segmental glomerulosclerosis, membranous nephropathy, and minimal change disease, IgA nephropathy is an important differential diagnosis to consider. These patients most often present with a palpable, purpuric rash and normal-to-mildly elevated serum IgA levels. Renal involvement in adults is usually manifest by mild proteinuria and hematuria without red cell casts, although heavy proteinuria and more active urine sediment-as was present in this case-can occur. Renal and skin biopsies are necessary to confirm the diagnosis. The prognosis is typically good, with complete remission not uncommon, with or without a short course of steroids. In other cases, stronger immunosuppression may be necessary using prolonged corticosteroids, mycophenolate mofetil, and/or calcineurin inhibitors.

Sara Jaskanwal Deep
Dr. Ryan Khodadadi;
Dylan Barth, PharmD;
Augustin Joseph, BS;
Dr. Caroline Burton

Amyloidosis - Unifying Heart Failure and Nephrotic Syndrome in a Headscratcher

Case Report: A 69-year old lady with a medical history of hypertension and hyperlipidemia was in good health until three months prior when she presented to an outside hospital with a one-week history of breathlessness, cough productive of green sputum and fevers. At the time a chest x-ray showed bilateral moderate sized pleural effusions with patchy consolidation in the right lower zone. She was treated for community acquired pneumonia with IV antibiotics and also received a Lasix drip as she was clinically fluid overload. An echocardiogram showed an ejection fraction of 55-60%, mild left ventricular hypertrophy and normal diastolic filling. She was also noted to have evidence of renal impairment with an admission creatinine of 1.64 mg/dL in the absence of a previous history of renal disease. She was discharged on oral Lasix but began to feel breathless again after one week with
worsening orthopnea and lower limb swelling. She re-presented to an outside facility and was again treated for pneumonia with antibiotics, and fluid overload with IV Lasix. Laboratory work-up demonstrated a BNP of 25,326 pg/dL, CRP 20 mg/L and a negative ANA and Rheumatoid factor and urinalysis demonstrated blood ++ and protein +++ with 24-hour total urine protein of 4602g. Her husband and her elected to transfer her care to Mayo Clinic, at which point she remained breathless at rest and had ongoing signs of significant fluid overload. IV Lasix was continued and she underwent an immunologic screen that was negative for HIV, hepatitis B and C, ANCA, PLA2 antibody, Rheumatoid factor, myeloperoxidase and proteinase PR3. However, serum immunofixation demonstrated a monoclonal lambda free light chain. A renal ultrasound scan showed increased parenchymal echogenicity and a repeat echocardiogram showed increased left ventricular filling pressure with restrictive filling pattern suspicious for amyloid. She underwent a renal biopsy that confirmed a diagnosis of AL amyloid, and was transferred to the Hematology service where she began treatment with chemotherapy.

Discussion: Diagnosing patients with heart failure and nephrotic syndrome in isolation is insufficient and clinicians should always search for the underlying causes of these syndromes. Amyloidosis represents a rare group of diseases in which abnormal protein, namely amyloid fibrils, build up in various organs. Presentation depends on which organ are involved and could include diarrhea and weight loss, suggestive of gastrointestinal involvement and breathlessness and fluid overload suggestive of cardiac and/or renal involvement. Clinicians should not hesitate in repeating laboratory testing in patients who present from outside hospitals, nor should the seemingly ‘low yield’ of an immunologic screen amongst patients with nephrotic syndrome act as a deterrent from providing comprehensive care to patients. These tests may after all provide the only clues to the underlying cause of a patient’s presentation.

<table>
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<tr>
<th>Brenton Schneider</th>
<th>Pneumocystis Pneumonia in an HIV-Negative Patient taking Corticosteroids</th>
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<td>Pneumocystis pneumonia (PCP) is a rare complication of immunosuppression in HIV-negative patients, but the incidence is increasing. Identifying and treating this insidious infection can be challenging.</td>
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<td>The patient in this case is a 47 year old woman with decompensated end-stage liver disease from alcohol use, and autoimmune hemolytic anemia, which was treated for several months with high dose corticosteroids. She presented with fatigue and abdominal pain from severe anasarca and ascites. In the days following admission she began complaining of mild shortness of breath without cough or fever, and her oxygen saturation gradually declined. Her exam was notable for jaundice, asterixis, diffuse crackles in all lung fields, and pitting edema extending from her feet to her upper abdomen. A chest x-ray revealed diffuse opacities throughout both lungs. Her respiratory symptoms were initially attributed to pulmonary edema or pneumonia, but broad spectrum antibiotics and potent diuretics had no effect. Her oxygen needs increased, up to 10 L</td>
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by nasal cannula. A chest CT showed crazy paving in both lungs with an upper lobe predominance. A beta-D-glucan was positive, as was a subsequent Pneumocystis direct fluorescent antibody test. She was diagnosed with PCP, and started on treatment with trimethoprim-sulfamethoxazole and PCP-dose corticosteroids. After several days of improvement, her hypoxia abruptly worsened, eventually requiring intermittent BiPAP, then intubation. She developed ARDS, as well as distributive shock from her liver disease, requiring several vasopressors. Eventually the patient’s family determined that continuing aggressive treatment was not in line with her wishes, and opted to withdraw life sustaining measures.

Corticosteroid use is a major risk factor for PCP, usually when combined with other immunosuppressing medications or conditions, as in this patient with advanced decompensated liver disease. Prophylactic treatment decreases infection risk. Compared to HIV-associated PCP, HIV-negative infections often have a more fulminant presentation, cause higher hospital and ICU admission rates, and carry a significantly higher mortality risk. Diagnosis can be challenging due to confounding symptoms and radiographic findings, but should be entertained in any patient with risk factors.

Jonathan Senkler

**Negative Anion Gap Metabolic Acidosis Secondary to Brominism from Dextromethorphan Overdose**

Bromide is an element with the same number of valence electrons as chloride. On lab testing bromide is interferes with the measurement of chloride and can cause a falsely elevated chloride level. Dextromethorphan contains bromide and has significant abuse potential. Metabolic acidosis is commonly described as being elevated or non-elevated anion gap metabolic acidosis. The anion gap can be calculated easily if the sodium, chloride and bicarbonate are known. Further the anion gap can be corrected for albumin, which accounts for a significant amount of unmeasured anions. Other unmeasured anions and cations can affect the anion gap.

A 31 year old male with no known past medical history presented to the Emergency Department for altered mental status. He was found down at the light rail station by bystanders. In the Emergency Department he was noted to become more somnolent, with occasional tonic movements. Administration of IV naloxone produced no response. The patient was not protecting his airway and required intubation. On subsequent labs he was found to have a negative anion gap metabolic acidosis with a chloride of 122. Urine drug screen was positive for dextromethorphan and guaifenesin. Poison control was consulted and the patient was started on IV lorazepam for sedation given concern of serotonin syndrome, a known complication of dextromethorphan overdose. The patient improved gradually over the next several days.

This case illustrates that overdose of dextromethorphan causes a negative anion gap metabolic acidosis and can cause serotonin syndrome. The bromide found in dextromethorphan interferes with the measurement of chloride, which consequently results in a falsely raised chloride level on the basic metabolic panel. Ultimately this contributes
Symptoms of brominism are non-specific but include headache, somnolence, memory loss, and ataxia. Elevated chloride levels with a negative anion gap metabolic acidosis should be recognized as possible dextromethorphan overdose, which should then prompt evaluation for life threatening serotonin syndrome.

An Unusual Cause of Embolic Stroke: Bicuspid Aortic Valve and Thoracic Aorta Thrombosis

Introduction: Bicuspid aortic valve (BAV) is a common cause of congenital heart disease, affecting roughly 1% of the population. BAV has been associated with aortic stenosis, aortic regurgitation and infective endocarditis. Further complications include ascending aortic aneurysms and aortic dissection, yet BAV has not been established as a risk for valvular thrombosis. We report a rare case of an embolic stroke secondary to a spontaneous bicuspid aortic valve thrombus in addition to thrombi identified in the proximal and mid descending thoracic aorta.

Case Description: A 41-year-old male with a past medical history for hypertension, tobacco use, and prior incarceration presented with acute onset confusion, global aphasia, right facial droop and right upper extremity weakness. On physical examination the patient was afebrile, found to have a grade 2/6 systolic murmur most prominent in the left 2nd intercostal space, with no evidence of splinter hemorrhages, Osler’s nodes or Janeway lesions. Brain imaging, including CT and MRI, revealed a large subacute infarct involving the left insular region and left frontal lobe, with smaller acute infarcts involving the left frontal and parietal lobes. Given suspicion for a cardioembolic etiology, a transesophageal echocardiogram (TEE) was obtained and revealed a bicuspid aortic valve with right-left cusp fusion and mobile echodensities on the ventricular aspect of the aortic valve, as well as hypermobile densities in the proximal and mid-descending thoracic aorta. Blood cultures, infectious serologies and inflammatory markers were negative and the patient did not meet Duke Criteria for endocarditis. The appearance of the masses was most consistent with thrombi, and anticoagulation with intravenous heparin was initiated, followed by warfarin recommended indefinitely. A repeat TEE performed two weeks after beginning anticoagulation demonstrated resolution of the masses, confirming the clinical suspicion of thrombus. Laboratory studies and CT of the chest, abdomen, and pelvis showed no evidence of non-bacterial thrombotic infective endocarditis, malignancy, thrombophilia or autoimmune disorders.

Discussion: The presence of a thrombus on a bicuspid aortic valve is an unusual phenomenon and in association with thoracic aorta thrombi and stroke is even more atypical. Diagnosis can be challenging, given imaging findings with resemblance to infective endocarditis. Management should include a workup for hypercoagulable state and ruling out other etiologies of valvular lesions. Acute and long term management strategies center on anticoagulation and in our case the valve and thoracic aortic thrombi resolved with two weeks of anticoagulation.
### Parvati Singh

*Interpreting Maladies before the Heart: Consequences of Mediastinal Masses*

Cancer can present in various ways, though weight loss is often used to help determine if malignancy is in the differential diagnosis. In this case, a 51 year old female with a past medical history of Type 2 DM and HTN presents to the hospital with severe chest pain radiating to her back, shortness of breath, and cough of one week duration. She did not have any weight loss in the past year. Imaging with CT chest showed an 8.6 cm anterior mediastinal mass with lymphadenopathy and compression/invasion of the superior vena cava (SVC). On further evaluation, the patient reported experiencing facial fullness for several weeks. Biopsy of the mass revealed an intermediate grade neuroendocrine tumor. Other complications that the patient suffered from this malignancy included pericarditis, pericardial effusion, diaphragmatic paralysis (phrenic nerve compression), and a right atrial thrombus. Thus, this case illustrates the classic presenting symptoms, such as SVC syndrome, of mediastinal masses.

### Brody Slostad

Dr. Rajiv Kumar; Rahul Maheshwari, BS

*A Case of Recurrent Weakness and Proteinuria*

A 38 year old male with a past medical history of hypertension presented to clinic with fatigue and cough. A bacterial URI was diagnosed and antibiotics were initiated. Three weeks later he presented to the emergency department with bilateral foot numbness, progressive weakness and frothy urine.

Physical examination revealed symmetric 2+ pitting edema in the lower extremities. Neurologic examination showed diffuse hyporeflexia throughout the upper extremities. Lower extremity reflexes were absent. Direct muscle strength testing demonstrated symmetric upper extremity weakness involving the triceps, biceps and deltoid. In the lower extremities, moderate symmetric iliopsoas, hamstring, anterior tibialis, peroneus, and posterior tibialis weakness was noted. Modest symmetric loss of vibratory sense was also noted.

Lumbar puncture revealed an albuminocytologic dissociation with an elevated total protein (52 mg/dL) and a normal cell count (3 nucleated cells/µL). Peak expiratory flow was normal 650 L/min (predicted 643 L/min). EMG was unremarkable. However, MRI of the lumbar spine showed enhancement of several lumbar nerve roots bilaterally compatible with demyelinating polyneuropathy. Serologic evaluation demonstrated a positive Mycoplasma pneumoniae IgM.

Renal investigation showed normal creatinine (0.9 mg/dL, eGFR >60 mL/min), abnormal urinalysis with occasional fatty casts, elevated 24 hour urine protein excretion (8.7 g/24 hours), low serum albumin (2.7 g/dL), and a normal lipid panel. Further workup revealed negative testing for hepatitis B, hepatitis C, HIV, syphilis, ANA, complement, and serum and urine protein electropheresis. Renal biopsy was consistent with the tip variant of focal segmental glomerulosclerosis (FSGS).

The patient was diagnosed with concomitant acute inflammatory demyelinating polyneuropathy (AIDP) and FSGS. Plasma exchange was initiated for five days for AIDP and high dose steroids were started.
The patient’s strength improved dramatically, renal function remained stable and he was discharged on an oral steroid taper. Initial follow up revealed that the patient’s weakness and proteinuria were improving.

The patient had three recurrences of concomitant weakness and proteinuria, suggesting a likely etiologic link between his now chronic inflammatory demyelinating polyneuropathy (CIDP) and FSGS. FSGS is a common cause of nephrotic syndrome in the United States. Characterizing FSGS as primary or secondary is important as this distinction affects management and prognosis. Primary FSGS tends to respond to immunosuppression, while treatment of secondary FSGS aims at reducing intra-glomerular pressure.

This case illustrates the association of CIDP and FSGS—an exceedingly rare association. The etiologic link between the conditions is supported by the concordance of flares and remissions of the two conditions with time and therapy. Additionally, this case highlights the importance of distinguishing histological subtypes of FSGS as this has an impact on management.

Jessica Slostad
Dr. Anjali Bhagra

The Importance of a Systematic Approach to Acute Kidney Injury

Acute kidney injury (AKI) is a common presentation in both the inpatient and outpatient setting. A systematic step-wise approach to diagnosis is critical in finding the underlying etiology of a condition that can lead to significant morbidity and mortality. Diagnostic evaluation includes a comprehensive history and physical examination, appropriate serum and urine laboratory tests, and as needed imaging.

A 68-year-old male with past medical history of neurofibromatosis type 1 (NF1) and splenectomy, who had not sought medical care for many years, presented with acute low-back pain to his local clinic. Additional history revealed urinary frequency, unintentional 25-lbs weight loss, and malaise. Medications included aspirin and naproxen. Laboratory tests showed elevated creatinine (2.7 mg/dL, baseline 0.7 mg/dL). Physical exam was notable for enlarged prostate. Urinalysis showed proteinuria. 24-hour urine collection confirmed 3.4g protein. Renal ultrasound showed a renal cyst without hydronephrosis. NSAIDs were discontinued and increased fluid intake was recommended. One week later, he presented to our institution with fever, poor oral intake, and nausea. He had worsening renal function with creatinine 4.1 mg/dL, BUN 44 mg/dL, leukocytosis 22.5 mg/dL, hemoglobin 10.9 mg/dL, and calcium 11.4 mg/dL. Physical exam revealed diffuse NF1 subcutaneous nodules and lower lumbar spinal tenderness. Infectious work up was unrevealing.

Further laboratory evaluation with serum protein electrophoresis revealed an M-spike. Serum kappa light chain was 1390mg/dL with kappa/lambda ratio >1000. He was diagnosed with kappa light chain Multiple Myeloma (MM) with light chain cast nephropathy. Worsening renal failure prompted urgent plasmapheresis. Bone marrow biopsy confirmed kappa light-chain multiple myeloma and minimal peripheral blood involvement. He was initiated on bortezomib, cyclophosphamide, and dexamethasone. His kappa/lambda ratio improved.
The patient unfortunately developed a lower gastrointestinal bleed on day 3 of plasmapheresis with worsening renal failure and kappa light chain ratio. He then required continuous renal replacement therapy. Despite aggressive management of multi-organ failure, he had worsening shock, severe metabolic acidosis, and acute respiratory distress and the patient unfortunately died. Autopsy confirmed kappa light-chain multiple myeloma as well as Staphylococcus aureus and Candida albicans bilateral pneumonia.

Our case describes an unfortunate outcome of a patient presenting in severe renal failure with newly diagnosed MM. Renal injury is the first presentation of MM in 50% of patients. The most common cause of severe renal injury in MM is cast nephropathy with 5% of patients requiring dialysis. Survival is improving in patients with cast nephropathy due to novel chemotherapeutic agents, and first line treatment is bortezomib/dexamethasone +/- third agent. Severe renal impairment in newly-diagnosed MM is a risk factor for high mortality and early death. This case outlines the importance of clinician awareness of a step-wise and quick diagnostic approach to AKI, especially in the setting of newly diagnosed Multiple Myeloma.

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<tr>
<th>Kevin Song</th>
<th>Metastatic Adenocarcinoma of the Lung Presenting as an Incidental Polyp on Colonoscopy</th>
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<tr>
<td>Dr. Brian Hanson</td>
<td>Introduction: we report a case of metastatic adenocarcinoma of the lung discovered in a patient who presented with incidental colonic polyp on colonoscopy.</td>
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<td>Case Presentation: a 64 year old African American male with thirty pack year smoking history presented to hospital with extensive unprovoked DVT in left lower extremity found on ultrasound. He had no recent travel, surgery, known cancer or other hormone replacement. Patient was discharged on rivaroxaban but presented to hospital two weeks later with hematochezia without abdominal pain, nausea, or vomiting. EGD and colonoscopy were performed but did not identify a source of bleeding. Two 8 mm polyps in transverse and sigmoid colon were resected; pathology report showed tubular adenoma with metastatic poorly differentiated adenocarcinoma into the lamina propria, consistent with pulmonary origin (TTF1 and CK7 positive; CK20, CDX2, GATA3 and S100 negative). A follow up CT chest/abdomen/pelvis demonstrated multiple spiculated pulmonary nodules with metastatic supraclavicular, cervical, mediastinal and hilar nodes. Based on mutation pattern (PD-L1 positive; EGFR/Alk/Ros unmutated), patient was started on pembrolizumab with a palliative intent.</td>
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<td>Discussion: lung cancer is one of the most common primary malignancies with nearly 50% distal metastasis at the time of diagnosis. However, intestinal metastasis is considered to be rare. Prevalence of metastasis to GI tract varies from 0.5-14% depending on the study. Most common histological found were SCC and large cell. Most common site of metastasis found were esophagus, small intestine, and stomach. Only 1/3 of colonic metastases from lung cancer are asymptomatic at the time of the diagnosis. Of the symptomatic cases, the most common symptoms described were abdominal pain, nausea, vomiting, anemia, and weight</td>
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loss. Metastases to the GI tract are most often diagnosed during workup for symptomatic cases or found incidentally on imaging or autopsy of asymptomatic cases. Distant metastasis in lung cancer confers poor prognosis. Diagnostic modalities such as colonoscopy likely do not confer mortality benefit to the patient. Less is known regarding morbidity and prevention of GI complications. In conclusion, we present an interesting case of metastatic lung presenting as an asymptomatic colon polyp. It is worthwhile to note that not all colon polyps are colon cancer, although an asymptomatic lung adenocarcinoma to the large intestine represents an extremely rare subset of an uncommon metastasis site.

**A Rare Cause of Acute Pancreatitis**

Introduction: Acute pancreatitis is the most common cause for hospitalization due to gastrointestinal disease in the United States. It has an overall mortality of 1 to 5 percent. While gallstones and alcohol abuse cause the majority of cases of acute pancreatitis, other etiologies must be considered and recognized to treat appropriately and to prevent recurrences.

Case Description: A 64 year old woman with a history of hypothyroidism and osteoporosis presented to the Emergency Department with epigastric abdominal pain and nausea after undergoing a colonoscopy earlier in the day. The colonoscopy was uncomplicated; she had three small polyps removed. On physical exam, she had tenderness over the epigastrium with no rebound or guarding. Lipase was elevated to 10,808 and ALT was normal. CT abdomen showed acute pancreatitis. Cholelithiasis was noted but there was no choledocolithiasis and no bile duct dilatation. Cholesterol panel showed normal triglycerides. She had no significant alcohol use history.

With a normal ALT and no bile duct dilatation, gallstone pancreatitis was unlikely. Given the timing of her symptoms in relation to her procedure, it was felt compression of the pancreatic tail during the colonoscopy lead to her pancreatitis. She was treated with intravenous fluids and pain medications with resolution of her symptoms.

Discussion: This case of pancreatitis after colonoscopy illustrates an uncommon cause of acute pancreatitis and complication of colonoscopy. Correctly identifying the cause of acute pancreatitis helps guide appropriate treatment and prevention. This case demonstrates how ALT is a helpful marker in identifying gallstone pancreatitis. It also emphasizes the importance of recognizing causes of pancreatitis other than gallstones and alcohol abuse. A detailed history, physical exam, laboratory, and imaging review is necessary in evaluating patients with acute pancreatitis.

**Integrase Inhibitors: We Need TB Careful!**

Introduction: The use of integrase inhibitors in antiretroviral therapy (ART) is a risk factor for immune reconstitution inflammatory syndrome (IRIS). We present the case of a man with HIV and TB infection who developed TB-IRIS 10 days after the initiation of abacavir/dolutegravir/lamivudine.
Case description: 46-year-old homeless man presented with fever and cough for 1 week. The patient had been living in a homeless shelter for the past 3 months with untreated HIV, diagnosed 17 months prior. Given his risk factors, TB was our greatest concern. CT of the chest showed 2.2 cm nodular, non-cavitary opacity in the right upper lung. One of 3 sputum samples had 4+ AFB smear and 2 of 3 were positive for mycobacterial culture. Empiric TB treatment, including isoniazid, rifabutin, pyrazinamide, and ethambutol, was started on day 6 of admission. Fever and cough gradually improved over the next 2 weeks. After 12 days of TB treatment, ART with abacavir/dolutegravir/lamivudine was initiated. At that time, his HIV viral load was 56,703, CD4+ was 381, and no drug resistance was noted. He initially tolerated both ART and TB therapy well. Following 2 weeks of TB treatment and 2 days of ART, sputum samples were AFB negative. One week after this, the patient developed fever, rigors, and tachycardia to 200 bpm. Repeat labs revealed HIV viral load of 2,940 and CD4 count of 517, while chest CT identified an increase of the right upper lung nodule. Infectious workup failed to identify a new source. Thus, his symptoms were attributed to paradoxical TB-IRIS and he was started on scheduled ibuprofen. Symptoms gradually improved without further management, leading to his discharge.

Discussion: Our patient had concurrent HIV and TB infections, requiring simultaneous management. The complicated issue in our patient was to determine the optimal timing to start ART while considering potential toxicities, drug-drug interactions, and TB-IRIS risk. Although the risk for TB-IRIS in our patient was relatively low given his CD4+ level of 381, it is possible to happen since his ART regimen consisted of an integrase inhibitor (INSTI), dolutegravir. Dolutegravir is highly effective in lowering the HIV RNA level and considered to be an appropriate choice to use with rifabutin-based TB regimen. However, several studies have recently shown that initiation of INSTI-based ART may double the risk of IRIS given its effectiveness in decreasing viral load, enhancing the immune system and together with the effect from TB treatment that destroying the bacilli, it could lead to an exuberant inflammatory response, and eventually the development of TB-IRIS. Additionally, the risk for TB-IRIS will be much higher among those with a low CD4+ count. Thus, further delay in the initiation of ART with INSTI may be required for those with HIV/TB co-infection.

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<tr>
<th>Jonathan Urbach</th>
<th>Dr. Amy Holbrook</th>
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<td>A (Non-Caseating) Eclipse of the Heart: Cardiac Sarcoidosis as a Cause of Acute Systolic Heart Failure</td>
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Introduction: Sarcoidosis is known as one of the "great imitators" of medicine, as it can present in countless ways given its often multisystem infiltration with non-caseating granulomas. Cardiac sarcoidosis occurs in a subset of patients with systemic sarcoidosis, though symptoms (e.g. heart block, sudden cardiac death, arrhythmias, heart failure) are similarly varied in timing and severity. The diagnosis of cardiac sarcoidosis is challenging, and treatment depends on strong clinical suspicion and early identification.

Case: 45-year-old man with no past medical history other than seasonal allergies initially presented with several months of progressive shortness...
of breath and activity intolerance. He saw his PCP, and was diagnosed with pneumonia and started on antibiotics, but did not improve. He then underwent a chest CT, which revealed bilateral hilar adenopathy, and he was referred to a pulmonologist. The patient was scheduled to undergo a lymph node biopsy and echocardiogram, but he continued to have worsening shortness of breath, cough, orthopnea and paroxysmal nocturnal dyspnea, which prompted him to seek emergency evaluation. In the hospital, he was found to be profoundly volume overloaded, and had marked improvement of his symptoms with diuresis. ECG showed a right bundle branch block. Echocardiogram confirmed acute systolic heart failure with a severely reduced ejection fraction of 10-15%, and cardiac MRI was suggestive of an infiltrative process. Hilar lymph node biopsy revealed non-caseating granulomas. Given these findings, a diagnosis of cardiac sarcoidosis was made, though no myocardial biopsy was obtained. Patient remained in cardiogenic shock during much of his hospitalization, and ultimately required a left ventricular assist device and careful titration of his heart failure medications. After inotropic support was weaned, he was discharged with close outpatient follow-up, which included a PET myocardial perfusion scan showing active cardiac sarcoidosis, despite ongoing immunotherapy. Patient remains asymptomatic, though is undergoing workup for cardiac transplant given his persistent disease activity.

Discussion: Myocardial infiltration with non-caseating granulomas underlies the pathophysiology of both systolic and diastolic heart failure in cardiac sarcoidosis. Cardiac involvement can occur at any point of the disease progression of systemic sarcoidosis, and heart failure can be the initial presenting symptom. In addition to EKG, echocardiography, and myocardial biopsy, cardiac MRI and PET have become helpful in the diagnosis of cardiac sarcoid. CMR can show acute inflammation (T2) as well as structural changes attributable to infiltration (T1), and classically involves the sub-epicardial tissue of non-vascular territories. PET has high sensitivity for detecting active cardiac sarcoidosis, though is relatively non-specific (with similar findings in other inflammatory processes). While there is no single set of diagnostic criteria, clinical suspicion for cardiac sarcoid should be high in young adults with new onset EKG abnormalities or symptoms concerning for heart failure.

Sravya Vinnakota
Dr. Sorin Pislaru

**Ballooning Heart: A Cautionary Tale of Epinephrine**

Background: To the general internist, epinephrine is the mainstay for management of several conditions including allergic reactions, hymenoptera stings, anaphylaxis, cardiogenic shock and cardiac arrest. It is often used as an adjunct with other local anesthetics for routine office procedures. Its property of vasoconstriction increases the duration of action of the anesthetic and decreases systemic absorption and toxicities. Although rare, a review of literature does highlight several instances of catecholamine-mediated cardiotoxicity associated with improper administration of epinephrine.

Case: A 20-year-old male with no past medical history presented to an outside hospital for an elective septoplasty. Patient inadvertently received 10 times the anticipated dose of epinephrine during the procedure. This error was immediately identified. The patient initially
became hypertensive to 180/130 and tachycardic to 150/minute for 40 minutes. He then became hypotensive with blood pressure 80/64, hypoxic with oxygen saturation of 89 percent on 15 liters oxygen through non-rebreather mask. His respiratory rate was 34 per minute. Lab results were significant for a new troponin leak of 0.47. A stat chest x-ray showed pulmonary vascular congestion. He received intravenous furosemide and was transferred to Mayo Clinic for further management. ECG showed sinus tachycardia with a heart rate of 115/min with no other significant changes. Further blood work demonstrated leukocytosis, elevated lactate (4.2 mmol/L) and BNP (774 pg/ml). Troponins were trended and resulted as 0.42, 0.43, 0.32 ng/ml. He underwent an emergent echocardiogram which showed basal left ventricular hypokinesis with apical sparing. He had a calculated LV ejection fraction of 35 percent. He was managed conservatively. His blood pressure improved with gentle fluid resuscitation. Beta blockade was initiated at low dose. Patient initially required respiratory support of up to 15 liters of oxygen through closed facemask. Orthodeoxia was also noted. By day 2, his hemodynamics improved, repeat echocardiogram showed normalization of EF at 56 percent. Patient was asymptomatic at the time of discharge and was discharged on a beta-blocker. At a follow-up visit after 4 weeks, patient was asymptomatic with echocardiogram showing LVEF of 64 percent. Beta-blocker was discontinued after 8 weeks.

Conclusion: Epinephrine can cause transient and minor side effects, even when used appropriately. Although major cardiac events are rare, they can be fatal if not recognized promptly. The catecholamine surge associated with epinephrine has been linked to Takotsubo and Reverse Takotsubo syndromes in multiple case reports. Most were associated with inadvertent intravenous administration of concentrated epinephrine. In severe cases, it could lead to refractory cardiogenic shock requiring ventricular assist devices or extracorporeal membrane oxygenation. Given how routinely it is used by internists for various office procedures, it is essential to develop and implement mechanisms to reduce the inadvertent administration of concentrated epinephrine.

Paul Viscuse
Dr. Arya Mohabbat

Dire Consequences of the “Steroid Stress Test”

Glucocorticoid therapy has been indicated in numerous medical conditions. Though effective, glucocorticoids can negatively impact the immune system, which may leave a patient vulnerable to potentially serious infections.

A 69-year-old woman presented to the hospital with lethargy, dyspnea on exertion, productive cough, right calf pain, and left thigh pain over the course of a few months. Her past medical history was notable for glomerulonephritis secondary to microscopic polyangiitis for which she had been started on high dose prednisone four months prior to presentation and had been tapered down to 20 mg daily. She had not been on pneumocystis prophylaxis due to a reported drug allergy. On presentation, she was stable though requiring 4 liters of oxygen. Exam was notable for coarse breath sounds with expiratory rhonchi bilaterally and a firm, tender 5 cm subcutaneous mass palpated in the right calf. Chest x-ray demonstrated diffuse interstitial opacities. Lower extremity
ultrasound revealed an occlusive deep vein thrombosis in the left soleal vein and a well-circumscribed mass in the right calf. Computed tomography (CT) noted ground-glass opacities throughout both lungs and a 4.0 x 2.6 cm consolidation in the right apex suspicious for cavitary necrosis. An indeterminate 1.6 cm subcutaneous mass in the anterior abdominal wall was incidentally noted. CT of the left hip showed diffuse intramuscular thickening with increased intermuscular fluid signal with multiple septations suspicious for multifocal intramuscular abscesses. She subsequently underwent bronchoscopy which initially returned positive for Pneumocystis pneumonia. However, sputum subsequently grew Nocardia which was also cultured from fluid aspirates from the left hip and abdominal fluid collections. She was initiated on sulfamethoxazole-trimethoprim following a rapid desensitization protocol, meropenem for disseminated Nocardiosis, and continued on prednisone. Magnetic resonance imaging of the head discovered three abscesses, the largest measuring 2.2 x 1.4 x 1.7cm with vasogenic edema. Linezolid was added due to cerebral involvement. Given the cerebral findings, therapeutic heparin was discontinued and an IVC filter was placed with prophylactic dose heparin. She was maintained on prednisone for her glomerulonephritis. She was discharged on sulfamethoxazole-trimethoprim, meropenem, and moxifloxacin with close follow-up arranged.

This case illustrates the potentially dire consequences of glucocorticoid use, specifically when prophylactic measures are not taken. It is essential for the internist to identify patients who are at risk for opportunistic infections due to steroid use (prednisone equivalent ≥20 mg daily for ≥1 month) and initiate appropriate prophylaxis. Diagnostically, this case demonstrates that it is important to obtain head imaging when evaluating an immunosuppressed patient who tests positive for Nocardia and that a false positive Pneumocystis result may occur in the setting of Nocardia infection.

**Dayne Voelker**
Dr. Sae Jang; Dr. Thomas Beckman

*Tortuous Ureters: A Roundabout Cause of Obstructive Nephropathy*

**Tortuous Ureters: A Roundabout Cause of Obstructive Nephropathy**

Introduction: Acute Kidney Injury (AKI) is commonly seen in the hospital setting. The three main categories of AKI are pre-renal, intrinsic, and post-renal, with pre-renal and intrinsic being the most common. However, when the clinical picture doesn’t fit the diagnosis, it is important to consider unusual post-renal causes of AKI. Obtaining a thorough history is essential in developing an effective diagnostic strategy.

Case Description: A 64-year-old woman presented to Emergency Department with 5-days of abdominal pain, nausea, vomiting, and decreased oral intake. Her past medical history was remarkable for Hodgkin's lymphoma treated 37 years earlier with chemotherapy, radiation, and splenectomy. Physical examination revealed only tachycardia to 102 BPM and decreased breath sounds at the lung bases. Laboratory evaluation showed a creatinine elevation of 4.0 mg/dl compared with a baseline of 0.8 mg/dl, and a BUN of 61 mg/dl. Urine microscopy showed no casts, WBCs, dysmorphic RBCs, or crystals. Chest x-ray showed bilateral plural effusions. Initial treatment was directed at pre-renal causes of AKI due to poor oral intake, as well as
intrinsic causes including contrast-induced nephropathy based on history of a recent CT scan. Treatment included intravenous fluid administration which was ineffective. Eventually, her kidney function worsened, she became anuric, she developed worsened nausea and vomiting, and she was dialyzed. Obstruction from post-radiation changes due to fibrosis was then considered. Although initial imaging did not show evidence of obstruction, a retrograde pyelogram was performed, demonstrating bilateral tortuous ureters. Ureteral stents were placed, resulting in brisk diuresis and normalization of her renal function.

Discussion: This patient’s bilateral tortuous ureters were likely caused by fibrotic changes secondary to radiation. Although tortuous ureters are uncommon, the clinical context suggested that this finding was the cause of her post-renal obstruction. This case highlights the importance of keeping retroperitoneal fibrosis in the differential diagnosis for tortuous ureters, and that radiation changes with resulting renal failure may take many years to develop. Additionally, this case is a reminder that Hodgkin’s lymphoma and radiation therapy places patients at risk for obstructive nephropathy.

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<th>Jessalyn Weaver</th>
<th>Intravascular Lymphoma Masquerading as Susac Syndrome</th>
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| A 47 y/o female with past medical history significant for type II DM, HTN and CAD initially presented in 2015 with subacute bilateral hearing loss and vertigo. MRI at the time showed lesions concerning for vasculitis. She subsequently developed memory loss and confusion, and the diagnosis of Susac Syndrome was made. Susac Syndrome is a very rare form of microangiopathy characterized by encephalopathy, branch retinal artery occlusions and hearing loss. She was treated with high dose steroids, IVIG and Rituxan. Later that same year she was referred to Hematology/Oncology for work up of new pancytopenia. Initial bone marrow biopsy showed a small clonal B cell population concerning for lymphoma, but the sample was suboptimal for morphologic analysis. Repeat bone marrow biopsy was also non-conclusive and she continued treatment for presumed Susac Syndrome. She then presented in 2017 with fatigue and abdominal pain shortly after tapering the dose of her steroids. She was found to have rising LFTs and a new, large hepatic mass. Biopsy of the mass was consistent with Diffuse Large B Cell Lymphoma. Bone marrow biopsy was performed and showed Large B-cell lymphoma involving 20% of the marrow space with clinical and morphologic features suggestive of intravascular B-cell lymphoma (IBCL) with prominent hemophagocytosis, concerning for HLH. Shortly after admission she developed severe pancytopenia and oliguric renal failure secondary to tumor lysis syndrome. Despite aggressive treatment of her metabolic abnormalities including intubation and CRRT she suffered a cardiac arrest and was unable to be resuscitated. Autopsy was deferred; however, based on opinion of the consulting oncologist and neurologist, it was thought that her initial neurologic symptoms were likely due to undiagnosed CNS lymphoma that was only partially treated with treatment of Susac Syndrome.
Recognition of intravascular Lymphoma is critical for prompt and adequate treatment. Because of its heterogeneity in presentation, Intravascular Lymphoma has been called the “Great Imitator”, making diagnosis particularly challenging. This case illustrates a misdiagnosis of Intravascular Large B cell Lymphoma as a rare CNS vasculitis.

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<th>Isaac Weeks</th>
<th>Cerebral Fat Emboli Syndrome in a Patient with Hemoglobin SC Disease</th>
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<td>A 26-year old female with hemoglobin SC disease presented with vasoocclusive crisis. After admission, the patient became febrile with acute hypoxemic respiratory failure and encephalopathy. She was found to have thrombocytopenia, new cor pulmonale and diffuse embolic strokes from paradoxical cerebral fat embolism in the setting of a patent foramen ovale. Vasoocclusive sickle cell crisis is a frequent cause of hospital admission while fat emboli syndrome is a rare consequence of vasoocclusive crisis. Rapid recognition of this syndrome is necessary to limit the consequence of this highly morbid syndrome.</td>
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<th>Kathleen Wilcox</th>
<th>Once Bitten, Thrice Tested: Fever and Fatigue after Travel to Mexico</th>
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<td>Dengue is the most prevalent mosquito-borne viral pathogen in humans. Though management is supportive care, proper diagnosis is important so patients can be counseled to avoid secondary infection, which, through antibody-dependent enhanced pathogenicity, carries a greater risk of severe disease. The patient is a middle-aged, previously healthy woman who presented to our clinic with a two week history of myalgia, fatigue, and subjective fevers. She had recently returned from Morelos, Mexico when she began to experience myalgia, fatigue, headache, sore throat, and subjective fevers. Four days prior to presentation, patient had a rash “like chicken pox” which started on her chest and torso, then spread to her face and extremities before resolving. Around the time of the rash, the patient also noted one episode of a large amount of bright blood with stool. The patient expressed concern that she might have chikungunya, which she knew to be endemic to Morelos. On physical exam the patient was afebrile with normal vitals and tired appearing. She had no rash, no signs of pallor, and her mucous membranes were moist. A digital rectal exam did not show blood. The rest of her exam was unremarkable. Lab work showed a hemoglobin of 11.2 g/dL. Leukocytes, platelets, and hematocrit were within reference ranges. Serologic testing was sent for chikungunya, dengue, and zika viruses. Additional testing was later sent for malaria and typhoid. One week after her initial presentation, patient’s serology returned positive for IgG antibodies to dengue virus (9.08 IV), and negative for IgM or IgG antibodies to chikungunya virus. Zika serology is still pending. When patient was notified of results, she reported ongoing fatigue and feeling light-headed, and was asked to return to clinic. On repeat exam she remained vitally stable, with no orthostatic hypotension, and unchanged physical exam. She was given 1 L normal saline for symptomatic treatment of subjective light-</td>
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headedness and counseled to continue aggressive oral rehydration at home.

Dengue is a neglected tropical disease caused by infection with the dengue virus. Like chikungunya virus and zika virus, dengue virus is transmitted by the Aedes aegypti mosquito, and thus all three share endemic territory. Clinical manifestations of dengue virus infection also overlap significantly with those of chikungunya and zika, including fever, myalgia, headache, eye pain, joint pain, and variable macular rash. Thus when either dengue, chikungunya, or zika virus infection is suspected, clinicians should strongly consider testing for all three.

**Zachary Yetmar**  
Dr. Cassandra Batzlaff

*Aspergillus Fumigatus as a Cause of Cavitary Lung Lesion*

Cavitary lung lesions present a diagnostic challenge due to the breadth of diseases with this presentation. Many of these conditions have a propensity to be acutely dangerous, communicable, or difficult to treat. Having a framework for a diagnostic approach is imperative to appropriately managing these patients. The causes of cavitary lung lesions include infectious, autoimmune, and neoplastic etiologies. Within these categories are mycobacterial or fungal infection, vasculitis such as granulomatosis with polyangiitis, or malignancy.

An 85 year-old male farmer with a history of chronic obstructive pulmonary disease (COPD) and benign pulmonary nodules presented to an outside medical institution with a one month history of weakness and cough productive of yellow-green sputum. He complained of weakness culminating in a fall from standing, productive cough, dyspnea, subjective fever, and chills. His travel history included station in Japan and Korea during the Korean War, Mexico in the early 2000s, and Arizona in 2012. He had been started on prednisone 20 mg daily and trimethoprim-sulfamethoxazole prophylaxis three weeks ago for dyspnea. He underwent chest CT with contrast which showed a right upper lobe apical thick walled cavity measuring 6.0 x 5.3 cm. Within the cavity a soft tissue structure measuring 3.6 x 3.4 cm was seen. A second thick walled gas containing cavity measuring 1.7 x 1.2 cm was seen in the anterior right upper lobe. These findings were new since his recent chest CT from 2 months prior. Laboratory studies were significant for leukocytosis (10.6 x 109/L), hyperglycemia (622 mg/dL), and acute kidney injury (creatinine 1.53 mg/dL).

He was admitted to the intensive care unit. On arrival he was alert, oriented, and in no acute distress but had episodes of rigors. Physical examination showed an ill-appearing man with diffuse wheezing on auscultation. He had an episode of hypotension that responded to a fluid bolus. An extensive infectious disease and autoimmune work-up was begun, including testing for mycobacterial, fungal, and rheumatologic etiologies. The subsequent laboratory results would include positive beta-D-glucan assay (341 pg/mL), negative aspergillus antigen (<0.500), and positive sputum culture with 3+ filamentous fungus speciated as Aspergillus fumigatus. The patient was started on voriconazole 200 mg twice a day, a prednisone taper, and discharged with follow-up with the Pulmonary and Infectious Disease clinics.
Cavitary lung lesions are difficult to manage due to the diversity of the diseases that cause them. Including infectious, autoimmune, and neoplastic etiologies, diagnosing these lesions requires insight in predisposing factors and establishing an individualized framework for work-up. Predisposing factors for pulmonary aspergillosis include prolonged corticosteroid use, neutropenia, malignancy, chemotherapy, HIV, immunosuppression, bone marrow or solid organ transplantation, and structural lung disease. In this case, corticosteroid use for a presumed COPD exacerbation likely predisposed the patient to infection with Aspergillus.

Margot Zarin-Pass

What's my type again?

A 29 year old man with history of type 2 diabetes mellitus presents to resident clinic to establish care and obtain refills of his medications. His diabetes history is notable for diagnosis several years ago with presumed type 2 diabetes based on “a blood test”. He presents on a regimen of metformin 1000mg bid, canagliflozin 300 qday, and insulin glargine 15 units qAM. He reports that he stopped his glargine recently as his blood sugars were in the 100-120 mg/dl range. He reports no hospitalizations for diabetes and no low blood sugars requiring treatment. He has never seen an endocrinologist. Physical exam is remarkable for a young, healthy appearing man with a BMI of 27.8. He is muscular and fit without significant adiposity. He does not have acanthosis nigricans. His hemoglobin A1c at this visit was 6.5%, down from 10% four months prior when he was initiated on canagliflozin therapy. Antibody panel was sent and remarkable for positive islet cell antibody, insulin autoantibody, GAD antibody, and IA-2 antibody.

This gentleman has an autoimmune diabetes (type 1) pathology that was mistakenly treated as a insulin resistance mediated (type 2) pathology. He never went into diabetic ketoacidosis (DKA), even after self stopping glargine, which was his only exogenous insulin. This is not unusual, as the older the patient is at presentation of type 1 diabetes, the longer the “honeymoon period” of residual beta cell function.1 His A1c drop is likely attributable to starting canagliflozin, which blocks renal tubule reabsorption of glucose, causing glycosuria. Theoretically, any hyperglycemic individual with normal kidney function will have lower blood sugar on this medication.

This case highlights two points for the general internist. First, consider type 1 diabetes in any young adult with new diagnosis of diabetes. One should be especially concerned, and send pancreatic autoantibodies, when hyperglycemia persists after 1 medication, the patient has no signs of metabolic syndrome, and the patient has no family history of diabetes.1,2 Second, it is important to keep appraised of the new anti-hyperglycemic medicines and their mechanisms of action. The fact that this gentleman’s A1c improved on canagliflozin does not point to an underlying pathophysiology of his diabetes nor does it mean that it should be first line treatment for him.

1. Harrison’s Internal Medicine, 19th Ed. 2014.
2. Type 1 Diabetes Through the Life Span: A Position Statement of the American Diabetes Association. 2014 PMID: 24935775
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<th>Zin Zhang</th>
<th>Eosinophilia in Ethiopian Immigrant: A Case of Strongyloides Infection</th>
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<td>Dr. Luke Hafdahl</td>
<td>Introduction: Eosinophilia is a common problem encountered by the general physician. We present the case of a young healthy Ethiopian woman who presented with abdominal pain and had evidence of eosinophilia on laboratory studies.</td>
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<td>Case description: A 38 year old healthy woman, originally from Ethiopia, presented to clinic with postprandial abdominal pain. She did not take any medications or supplements. Laboratory revealed hemoglobin 10.9 g/dL, MCV 85, eosinophils 890/microL, ferritin 9 mcg/L, and negative tissue transglutaminase antibody. Stool studies were negative for H pylori antigen; ova and parasite stool culture was negative. On review of previous labs, eosinophilia has been present for two years and has been as high as 1500/microL in the past. Strongyloides IgG antibody and trichinella IgG antibody were obtained for further evaluation. Strongyloides IgG antibody was positive. Despite negative subsequent strongyloides stool culture, patient was treated with ivermectin 200 mcg/kg orally once daily for two consecutive days given high clinical suspicion of chronic strongyloidiasis. At a follow-up visit 5 months later, abdominal pain has resolved. Repeat laboratory data demonstrated normal eosinophil count at 150/microL and improved ferritin level at 18 mcg/L.</td>
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<td>Discussion: Strongyloides stercoralis is endemic in tropical countries, with prevalence of up to 20% in Ethiopia {Schar, 2013 #1}. Gastrointestinal symptoms include upper abdominal pain associated with duodenitis, anorexia, nausea, vomiting, and diarrhea. Patients may also be asymptomatic and solely present with eosinophilia. Routine stool exam has less than 50% sensitivity for detecting strongyloides. Agar plate stool culture for Strongyloides is 3 to 5 times more sensitive and is preferred to identify current infection. Serology detecting IgG to filariform larva has sensitivity of 83-93%, but it cannot distinguish between active and past infection.</td>
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<td>In conclusion, strongyloides infection must be considered in a patient with possible exposure history and eosinophilia.</td>
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| Zhenmei Zhang | An Uncommon Presentation of Acute Coronary Syndrome |
| Dr. Rajiv Gulati | A previously healthy 33-year-old female with history of migraines presented to the hospital with 3 days of intermittent left sided chest, neck and arm pain. The pain resolved after rest on day 1 but returned after she walked 1/4 mile on the day of hospital presentation. Her physical exam was unremarkable except for tenderness to palpation over the left shoulder. Laboratory testing was significant for troponin of 0.14, 0.27 and 0.55 at 0, 3 and 6 hours respectively, and CRP of 8.9. Serial EKGS showed Q waves in leads II, III, aVF and later T wave changes in the anterior leads. Patient was started on heparin infusion and received a loading dose of aspirin and clopidogrel. She underwent coronary angiography on hospital day 2 and was found to have near total dissection of the left anterior coronary artery. No intervention was performed and patient was observed. She was discharged on hospital day 5 on aspirin and metoprolol. |
Spontaneous coronary artery dissection (SCAD) is an uncommon cause of acute coronary syndrome. It is commonly seen in younger, otherwise healthy patients. Data from SCAD case series that excluded existing atherosclerotic disease showed a female predominance and an association with peri- or post-partum state and fibromuscular dysplasia (FMD). The diagnosis of SCAD is typically made with coronary angiography. There is limited evidence to guide therapeutic management. Current consensus favors conservative treatment with aspirin and beta blockers, and based on expert opinion, dual antiplatelet therapy can be administered for one year. A retrospective analysis of 189 patients with SCAD showed no increase in early mortality when managed conservatively. PCI is particularly challenging given the frailty of the diseased vessel and the propagation of dissection with additional instrumentation. And at five year follow up, the rates of SCAD recurrence is similar in conservatively managed and revascularized groups. Fibromuscular dysplasia (FMD) also needs to be excluded in young patient without conventional risk factors presenting with SCAD. One case series study reported up to 86% of patients diagnosed with SCAD had underlying FMD.

In this clinical vignette, the patient is a young female without traditional atherosclerotic risk factors who presented with non-ST segment elevation myocardial infarction. Keeping spontaneous coronary artery dissection in the differential when encountering young patients presenting with acute coronary syndrome is important, and would guide additional diagnostic workup including investigation of FMD.

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<td><strong>A Case of Decompensated Hypothyroidism</strong></td>
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Hypothyroidism is a commonly diagnosed disorder in internal medicine. As illustrated in this case, patients occasionally present with uncommon symptoms making the diagnosis less apparent. Recognition of atypical presentations of hypothyroidism is important in preventing life threatening complications.

A 59-year- old female woman was admitted for evaluation of worsening lower extremity edema and shortness of breath. Review of systems was positive for worsening fatigue, weakness, constipation, cold intolerance, dyspnea on exertion, weight gain, memory loss, dry and flaking skin, hoarseness, myalgias, paresthesias, and depression. The patient’s past medical history is significant for hypothyroidism secondary to radioiodine ablation for Grave’s disease. On further questioning the patient admitted that she had not taken her synthroid in over two years. Her initial lab work up was notable for an elevated TSH (26.1) and undetectable free T4. The patient was vitally stable on admission.

Physical exam revealed a morbidly obese female with significant periorbital and lower extremity edema, dry skin, clear lungs and muffled heart sounds. Chest x-ray revealed markedly increased cardiomegaly compared to previous imaging so a transthoracic echocardiogram was obtained which showed a large pericardial effusion, estimated to be nearly a liter in size, with evidence of mild tamponade. Cardiology was consulted and
recommended against pericardiocentesis. Endocrinology was consulted and recommended restarting levothyroxine at prior dose. The patient was carefully monitored for signs of myxedema coma including hypothermia and hyponatremia, but thankfully she did not decompensate after treatment was initiated. After resuming thyroid hormone replacement, the patient’s symptoms markedly improved and there was a decrease in pericardial effusion on repeat echocardiographic evaluations.

This case illustrates classic signs and symptoms of decompensated hypothyroidism. Additionally, it highlights the importance of recognizing more atypical presentations of hypothyroidism including cardiac involvement like pericardial effusion. While rare, if patients go untreated patients they may develop life threatening complications of hypothyroidism including myxedema coma.