In celebration of the excellence demonstrated by the 133 poster presenters from the ACP Arizona Chapter Medical Student and Associate members at the 2012 Annual Scientific Meeting held on November 16-18, 2012 at Midwestern University in Glendale, Arizona, and as part of the ongoing commitment to a healthy future, ACP AZ is honoring the work of these Medical Student and Associate members and their educators and mentors in this e-publication, ¡Salud! The winner of the Oral Vignette competition and the Research Poster competition travel to the national ACP meeting to present their work and represent the Arizona Chapter. Each will receive a travel stipend from the Chapter. Internal Medicine 2013 will be held April 11-13, 2013 in San Francisco, California. We hope to see you there, and also hope you will plan to attend the 2013 ACP Arizona Chapter Annual Scientific Meeting at the University of Arizona in Tucson on November 15-17, 2013.

(Photos above: Poster presentation made to ACP Arizona Chapter Judges Panel at the 2012 Annual Scientific Meeting)

Congratulations to the ACP AZ 2012 Medical Student and Associate Poster and Oral Vignette winners, set forth here by category.

Medical Student Posters. First Place: Megan Hamre, Creighton College of Medicine, “Adrenocortical Carcinoma, A Unique Presentation and Metastasis: A Case Report Creighton College of Medicine”; Second Place: Jonathan Kang, Midwestern University Arizona College of Osteopathic Medicine, “The
ACP AZ 2012 Medical Student and Associate Poster and Oral Vignette winners, set forth here by category (continued from page 1).
Ob/Ob Mouse Jejunum Has Distinctly Different Characteristics Than Its Lean Counterparts”; Third Place: Salma Patel, University of Arizona College of Medicine Tucson/Phoenix, “Sudden Cardiac Death and Hypertrophic Cardiomyopathy.”

PGY-1 Posters. First Place: Colin Fitterer MD, Mayo Clinic Arizona, “Pancreatitis and a Renal Incidentaloma; Big and Benign”; Second Place: Qi Yu DO, University of Arizona College of Medicine at South Campus, “An Interesting Case of Hiccups and the Blues”; Third Place: Aaron Fernandes MD, University of Arizona College of Medicine at South Campus, “Tachycardia Induced Cardiomyopathy.”

Associate Research Posters. First Place: Robyn Emanuel MD MPH, Mayo Clinic Arizona, “Essential Thrombocythemia (ET) and Polycythemia Vera (PV) Symptom Burden: Phenotypic Cluster Analysis Among an international Sample of 1,141 Persons with ET and PV”; Second Place: Andrew Chung MD, Mayo Clinic Arizona, “Predictive Value of Nasal Meticillin-Resistant Staphylococcus Aureus PCR Swab for MRSA Pneumonia”; Third Place: Natalya Azadeh MD, Mayo Clinic Arizona, “The Impact of Early and Short-Term Corticosteroids on the Clinical Course of Patients with Primary Pulmonary Coccidioidomycosis – A retrospective Case Control Study.”

Associate Case Report Posters. First Place: Mohan Ashok Kumar MD, St. Joseph’s Hospital & Medical Center, “An Uncommon Cause of Cholestatic Hepatits – Epstein Barr Virus”; Second Place: Anju Nair MD, University of Arizona College of Medicine at South Campus, “A Rare Intestinal Transformation”; Third Place: Preethi William MBBS, University of Arizona College of Medicine at University Campus, “Calcific Uremic Arteriolopathy - An Atherosclerotic Masquerade.”


Fellow Poster Presenter. Bijin Thajudeen, MD, University of Arizona College of Medicine Renal Subspecialty Fellowship Program, “Morbidity And Mortality In Non Proteinuric Type 2 Diabetes Mellitus Patients With Chronic Kidney Disease-A Retrospective Study.”


The abstracts from the 2012 ACP Arizona Chapter Annual Scientific Meeting are set forth here, organized by Medical Student, PGY-1, PGY-2, PGY-3, Fellow, and the Oral Vignette abstracts, each section in alphabetical order by author’s last name. To these Medical Students and Associates, as the physicians whom we entrust with our ¡Salud!, we celebrate your accomplishments and contributions. Thank you,
ACP Arizona Chapter
MEDICAL STUDENT SUBMISSIONS

FIRST REPORT OF SPLENIC ABCESES DUE TO COCCIDIOIDOMYCOSIS
Shabnam Assar, Abstract 81

Introduction: Involvement of the spleen by Coccidioidomycosis is common with disseminated disease. However, clinically significant splenic abscesses have not been reported. Most often only granulomas are found in the spleen at autopsy in patients with disseminated infection. We report an immunosuppressed patient with splenic abscesses due to Coccidioides that required splenectomy. Case Presentation: 33-year-old Hispanic male with dermatomyositis for five years and coccidioidomycosis for two years, presented to the emergency room because of left upper quadrant abdominal pain, fever and chills. Treatment of his dermatomyositis over the years included prednisone, imuran and intravenous gamma globulin (IVIG). In 2010 the patient was treated with intravenous liposomal amphotericin B for acute pulmonary coccidioidomycosis followed by oral fluconazole. Admission vital signs were temperature: 38.4°C, blood pressure: 147/81 mmHg, heart rate: 106 bpm, respiratory rate: 18 breaths/min, and pulse oximetry: 99% on room air. Physical exam was significant for pigmentation on his face consistent with dermatomyositis, tenderness in the left upper quadrant region, and significant weakness in upper and lower extremities. His medications on admission included fluconazole and prednisone. An admission urine culture grew > 100,000 Klebsiella pneumoniae. CT scan of the abdomen revealed multiple splenic abscesses. It was initially believed that the splenic abscesses were due to Klebsiella. The prednisone was stopped, and fluconazole was continued. Piperacillin-tazobactam and IVIG were started. After two days, the patient’s abdominal pain and fevers did not improve. To avoid a splenectomy, a splenic biopsy was performed. The biopsy was consistent with a Coccidioides infection. A laparoscopic splenectomy was then performed, and the pathology on the removed spleen showed multiple coalescing necrotizing granulomas with numerous Coccidioides spherules. Post operatively, fluconazole was replaced by voriconazole, and the patient was restarted on prednisone for his dermatomyositis. The patient’s symptoms improved and he was discharged home. He would be followed up as an outpatient. Discussion: This patient illustrates an unusual complication of disseminated coccidioidomycosis. We could not find any reports in the English literature of true splenic abscesses complicating Coccidioidomycosis. We investigated the potential reasons for developing splenic abscesses in our patient. He received IVIG to control his dermatomyositis after he developed his Coccidioides infection. It is known that fungemia occurs in immunosuppressed patients with significant Coccidioidomycosis. It could be theorized that Coccidioides organisms were coated by gamma globulins when the patient received IVIG treatments for his dermatomyositis. He received IVIG to control his dermatomyositis after he developed his Coccidioides infection. It is known that fungemia occurs in immunosuppressed patients with significant Coccidioidomycosis. It could be theorized that Coccidioides organisms were coated by gamma globulins when the patient received IVIG treatments for his dermatomyositis. The opsonization of the organisms by the IVIG allowed the spleen to take up viable organisms into the reticuloendothelial system, which in turn resulted in the localization and abscess formation within the spleen. We suggest that these unusual circumstances resulted in splenic abscesses which previously had not been reported in coccidioidomycosis.
MUCORMYCOSIS PRESENTING AS NASAL ABSCESS IN DIABETIC PATIENT
Steven Brown, Abstract 80

Introduction: Mucormycosis is a rare fungal infection seen in diabetic and immunocompromised individuals. Over 50% of cases of mucormycosis are associated with Diabetes Mellitus (DM) and most commonly involves the genera Rhizopus, Mucor, Cunninghamella, Apophysomyces, Absidia, Rhizomucor and other species. Most mucormycosis infections are life threatening, and risk factors such as diabetic ketoacidosis and neutropenia are present in most cases. Severe infection of the facial sinuses, which may extend to the brain, is the most common presentation. There is limited literature available regarding mucormycosis presenting as a nasal abscess. 53 year old poorly controlled diabetic female presented to the hospital with a ten-day complaint of facial and nasal pain and swelling. She initially presented to her primary care physician twice and urgent care once and was subsequently diagnosed with sinusitis. She was prescribed a ten-day course of Cephalexin and then Levaquin, but continued to have worsening pain and swelling. She then presented to the emergency department on request from a family member for continued pain and impaired nasal breathing. In-hospital CT scan of the sinuses showed a large, lobular, mixed-attenuation soft tissue mass in the left nasal turbinate and cavity consistent with an abscess, and therefore patient was started on empiric antibiotics and corticosteroids. Physical exam at this time was remarkable for erythema, tenderness, and swelling in nasal area, no black eschar. The rest of exam was within normal limits. Laboratory data revealed mild leukocytosis and elevated blood sugars with HbA1c of 16.5. She was transferred to our facility for ENT evaluation and surgical management. Culture from tissue and wound samples demonstrated growth of Rhizopus. Based on these results, patient underwent several procedures for incision, drainage, and debridement of inflamed and necrotic tissue, aggressive glycemic control, and systemic amphotericin during hospital stay. Was discharged with posaconazole and received a total of 6 weeks of antifungal treatment. As of the submission of this abstract, she continues to follow ENT specialist with no signs of recurrence of disease after 10 weeks of initial presentation. Discussion: Our case represents an unusual presentation of nasal mucormycosis. It was recognized early and the disease was limited to soft tissues of the nose. Poorly controlled diabetics and immunocompromised are at greatest risk. Mainstay of treatment is aggressive surgical and medical management. This case illustrates potential presentation for early atypical mucormycosis in a patient unresponsive to broad-spectrum antibiotics and poorly controlled Diabetes Mellitus. Increased awareness and early recognition are critical to institution of an appropriate therapy and prevention of progression of disease.

A CONCERNING RENAL MASS
George Chesteen, Abstract 51

Introduction: In clinical practice, we constantly weigh the risks and benefits of diagnostic and therapeutic procedures in a patient’s care. In this case we were faced with the dilemma of performing a risky diagnostic procedure in order to rule out a potentially fatal disease. Case Presentation: A.M. is a 24 year old female with a past medical history of tuberous sclerosis (TS) who presented to our service with a chief complaint of constant abdominal pain. The pain was located in the left upper and
lower quadrant, radiated to the left flank, dull, severe and started about 2 months ago. In addition, a history of seizures and developmental delay consistent with TS was described. On physical exam she demonstrated many of the typical findings in TS, including facial angiofibromas, hypomelanotic macules, cardiac rhabdomyoma, and renal angiomyolipoma. A computed tomography (CT) of the abdomen showed multiple lesions consistent with bilateral angiomyolipomas as well as an enlarging solid mass in the left kidney with sub-capsular hemorrhage and no evidence of fat attenuation. This mass was concerning for renal cell carcinoma (RCC). Discussion was held with the patient and her family about the bleeding risk associated with CT-guided percutaneous fine needle aspiration of a renal mass. The biopsy was performed without incident, and the patient’s hemoglobin remained stable afterward. Pathology was consistent with an angiomyolipoma and the patient was discharged home. Discussion: Tuberous sclerosis (TS) complex is a rare condition characterized by the formation of hamartomas in multiple organ systems. The incidence of renal involvement in these patients is very high, reported at over sixty percent, and largely consists of benign angiomyolipomas (AML). Additionally, these patients are at an increased risk for renal clear cell carcinoma (RCC)(1). Currently there is no reliable non-invasive study available to differentiate an angiomyolipoma from a renal cell carcinoma(2). The combination of renal involvement among TS patients, along with the inability to differentiate RCC from AML via imaging makes this case significant. The most common complication of biopsy is bleeding risk ranging from 0.1-12% (major complications from 0.2-4%) associated with AMLs further complicates the management and diagnosis of these patients. Due to the (95.2) of percutaneous renal mass core biopsy, this modality is preferred for ruling out RCC(3). However, AMLs are highly vascularized, making renal mass core biopsy relatively contraindicated. We were therefore faced with the dilemma of performing a procedure to rule out RCC that could cause life-threatening bleeding if the mass were an AML. An alternative diagnostic procedure is fine needle aspiration (FNA) that has not matched the benefits of image guided biopsy. Techniques involving CT/US guided have become safer; the risk of seeding RCC during sampling is considered to be negligible and bleeding is minimal(4).

COMPARISON OF THE EFFECTS OF HALOTHANE AND ISOFLURANE WITH ION CHANNELS

Winnie Cheung, Abstract 101

Comparison of the Effects of Halothane and Isoflurane with Ion Channels  Winnie Cheung1; Trevor Batty1; J Ryan Jackson1; Joel P. Goodman1; Krista Pearman1; Michael J. Murray2, MD, PhD; Gerald B. Call1, PhD  1. Arizona College of Osteopathic Medicine, Midwestern University, Glendale, AZ; 2. Department of Anesthesiology, Mayo Clinic, Scottsdale, AZ  Introduction: For more than 160 years, volatile anesthetics (VAs) have been used, yet the action of VAs upon the central nervous system is unknown. Recent evidence suggests that VA effects occur through multiple targets as opposed to a single common mechanism. Previous studies have indicated that ion channels may be VA targets. Our previous study was the first comprehensive analysis of ion channels in the response to isoflurane. We tested 359 ion channel genes within Drosophila melanogaster in an RNA interference (RNAi) screen to determine
potential candidates for VA action. A large percentage of the flies with ion channel genes reduced were found to have resistance to isoflurane. In the previous study, a small halothane screen was performed for data verification on a few ion channels; however, this revealed differences in their responses to the two VAs. As isoflurane and halothane have overall similar chemical structures, their responses were expected to be similar. This study explores the effects of halothane on ion channels and compares them with the isoflurane data. Methods: Drosophila melanogaster is a useful species to study the effects of VAs. In addition to the genetic benefits, it passes through each stage of anesthesia and its central nervous system is more complex than other invertebrates. RNAi constructs were used for gene-specific silencing of ion channels throughout the genome. An inebriometer was used to quantitatively study the effects of halothane on the flies. The data were analyzed using regression analysis and then compared with the previous isoflurane data. Results: Of the 340 genes tested, only 4 showed sensitivity to halothane, while almost 40% exhibited resistance. These results are very similar to those seen with isoflurane, with only 1 sensitive and 33% resistant. Of all the genes that showed resistance to VAs, 69 were resistant to halothane only, 77 had resistance to isoflurane only, and 64 showed resistance to both VAs. In this shared resistance category, we found that 11 genes were either resistant or strongly resistant to both VAs. A more detailed analysis will be presented at the meeting. Conclusion: Analysis of the data revealed that there are significant differences between the actions of isoflurane and halothane on ion channels. These differences indicate that there are many potential pathways for VA action, even among those with similar chemical structures. The 11 mutually and highly resistant genes will be further investigated, as they represent a possible common mechanism for VAs.

EARLY-STAGE MYCOsis FUNGOIDES WITH A DISAPPEARING T-CELL CLONE IN THE PERIPHERAL BLOOD

Samuel Ecker, Abstract 59

The clinical significance of gene rearrangement studies in the staging and prognosis of mycosis fungoides is evaluated in a 45 year-old woman presenting with early-stage disease. Initial laboratory testing identified a T-cell clone in a skin biopsy and peripheral blood sample. Blood samples taken after three weeks of treatment with phototherapy and topical corticosteroids did not detect the presence of a circulating clone by flow cytometry or repeat polymerase chain reaction. Although current guidelines include serum clonality in the diagnostic algorithm, the prognostic significance of circulating T-cell clones remains poorly defined. The presence of a skin and serum clone in early-stage mycosis fungoides is uncommon and there is considerable evidence that this may be associated with a worse prognosis in the event that the skin and blood clones are identical.1 This case appears to be the first report of early-stage mycosis fungoides with a disappearing T-cell clone in the peripheral blood following three weeks of skin directed therapy. Future research is needed to investigate the potential use of gene rearrangement studies as a means to determine prognosis and monitor response to treatment in early stage disease.
ADRENOCORTICAL CARCINOMA, A UNIQUE PRESENTATION AND METASTASIS: A CASE REPORT

Megan Hamre, Abstract 52

Adrenocortical Carcinoma, A Unique Presentation and Metastasis: A Case Report.
Megan Hamre, Yousef Usta MD Creighton University School of Medicine, Omaha, Nebraska St. Joseph’s Hospital and Medical Center, Phoenix, Arizona

Introduction: Adrenocortical carcinoma (ACC) is a rare disease, with an incidence of approximately 1-2 cases per million. The majority of ACC patients present with signs and symptoms of adrenocorticoid excess such as Cushing’s syndrome, virilization, hirsutism and hypertension. Approximately one-third of patients present initially with distant metastases of the lung, liver and bone. We present an atypical case of ACC who presented with symptoms of a bowel obstruction. Case: A 60 year-old male with a history of severe cardiomyopathy and methamphetamine abuse presented to the emergency department complaining of increasing abdominal pain, nausea and vomiting. The patient was afebrile, normotensive and diffusely tender on abdominal exam with no rebound tenderness. Patient appeared cachectic with temporal wasting on examination. He denied a prior history of colon cancer screening. Initial abdominal computed tomography (CT) showed a dilated small bowel, ascites and an atrophied liver as well as a left adrenal mass measuring 5.9 x 5.8 cm. Paracentesis revealed a nonmalignant transudative ascitic fluid with no signs of spontaneous bacterial peritonitis. A CT-guided biopsy of the adrenal lesion yielded a diagnosis of primary adrenocortical neoplasm.

Radiology found evidence of omental metastatic lesions with omental caking and mesenteric adenopathy. There was also evidence of peritoneal carcinomatosis. Oncology and Surgery were consulted, and both recommended no further medical or surgical therapy appropriate for this patient secondary to severe dilated cardiomyopathy. Patient was transferred to home palliative care.

Conclusion: Adrenocortical carcinoma usually presents with symptoms of adrenocorticoid excess; however, there is a small subset of patients presenting with non-functioning ACC. These individuals often have initial symptoms of abdominal discomfort, including nausea, vomiting, abdominal fullness or back pain caused by the tumor mass, similar to our patient’s presentation of bowel obstruction. This case is an example of a non-functioning ACC with a very rare presentation of peritoneal and omental metastasis in the absence of metastasis to lung, liver or bone. Primary colon cancer metastasizing to the adrenals and omentum is common, and was initially thought to be the diagnosis in our case. Patients with nonfunctioning ACC are often difficult to diagnose until late sequelae of the disease appear. Based on the revised TNM staging proposed by the European Network for the Study of Adrenal Tumors (ENSAT), 5-year survival for stage IV ACC is 15%. It has been shown that patients in stage IV without distant metastases have an improved survival compared to those with distant metastases.
Second Place – Medical Student Submissions:

THE OB/OB MOUSE JEJUNUM HAS DISTINCTLY DIFFERENT CHARACTERISTICS THAN ITS LEAN COUNTERPARTS

Jonathan Kang, Abstract 102


It is widely acknowledged that insulin resistance leads to hyperglycemia in the ob/ob mouse, however the role of intestinal glucose uptake and intestinal secretion is unclear. The ob/ob mouse lacks functional leptin and presents as obese, as well as hyperglycemic. We used ob/ob and lean mice aged 13-14 weeks, and the ob/ob mice had a significantly increased weight (49.3±1.6 g, n=5) compared to the lean mice (25.1±0.5 g, n=5). The goal of this study was to characterize jejunal function in this clinically relevant mouse model. We measured transepithelial short circuit current (Isc), across freshly isolated segments of jejunum from the ob/ob and lean mice. Basal Isc was significantly increased, by almost 2-fold, in the lean mice (95.2±20.3 µA/cm², n=4, P<0.05) compared to the ob/ob mice (50.5±9.5 µA/cm², n=5). The Isc in response to the adenylate cyclase activator, forskolin (10 µM, bilateral) was similar in both groups of mice, indicating no change in the cAMP-dependent Isc. The Isc in response to the Na+/K+/2Cl- co-transporter inhibitor, bumetanide (100 µM, basolateral), was similar for ob/ob and lean mice, indicating a similar Cl- secretory component. The Isc in response to acetazolamide (100 µM, bilateral), to block the HCO3- secretory component was also analogous in both groups. A comparison of jejunum morphology (i.e. villi length, number of goblet cells/villi, crypt depth, number of goblet cells/crypt) is currently in progress. Expression of the glucose transporter protein, Glut5 (normalized to GAPDH), was significantly increased in ob/ob mice 0.74 ±0.06 (n=14, P<0.05), compared to lean counterparts (0.37±0.06, n=11). Plasma glucose levels were almost 2-fold greater in the ob/ob mice (242±26 mg/dL, n=5, P< 0.05) compared to lean mice (124±8 mg/dL, n=5). These data suggest that basal jejunal Isc in lean mice is ~2-fold that in ob/ob mice, and may reflect a slower transit time in the gastrointestinal tract in the ob/ob mice which may contribute towards increased nutrient absorption (specifically increased glucose uptake via Glut5), an increased weight gain and the associated diabetic phenotype. This leads to speculation as to whether or not increased Glut5 expression in ob/ob mice is an effect of diabetes, or if Glut5 abundance results in the diabetic state. Jonathan Kang, Ashesh Bhakta, and Leslie Tamura were supported by the Midwestern University Summer DO Fellowship Program. Layla Al-Nakkash was supported by the Soy Health Research Fund.

A CASE OF LEFT SIDED POLAND’S SYNDROME ASSOCIATED WITH CHARCOT’S ARTHROPATHY

Mario Mitkov MD, Abstract 93

Article: Case Report  Title: A case of Left sided Poland’s Syndrome associated with Charcot’s arthropathy Author: Mario Mitkov1 BS, Yousef Usta MD2, Rashmi Kumar MD2, Priya Radhakrishnan MD2 Creighton University School of Medicine 2500 California Plaza Omaha, NE 68178 Department of Internal Medicine, St Joseph’s Hospital and Medical Center, 350 West Thomas Road, Phoenix, Arizona, 850132 Intro: Poland’s syndrome is a rare congenital musculoskeletal defect which is
described as an absence or underdevelopment of the pectoralis major muscle on one side of the body with various other ipsilateral musculoskeletal deformities. It involves the right side more often than the left, however, bilateral and contralateral variants have also been noted. It is a diagnosis that is first suspected clinically in the early years of life and later confirmed with radiographic imaging. Case: We present a case of a 45 year old male with a past medical history of left sided Poland’s syndrome previously confirmed by CT scan, which showed left pectoralis major aplasia. Additionally, he has a concurrent diagnosis of hepatitis C and has positive findings for severe left sided arm and leg atrophy, joint instability, bilateral decreased foot sensation, inability to bear weight, and erythematous, swollen ankle joints with decreased deep tendon reflexes. Clinical findings were suggestive of charcot’s neuroarthropathy which was later confirmed with X-Ray imaging. To the authors’ knowledge this is the first reported case of charcot’s arthropathy associated with left sided Poland’s syndrome. Conclusion: All known causes of charcot’s arthropathy were ruled out including the three most common; diabetes mellitis, syringomyelia, tertiary syphilis. Given his past medical history of joint and limb instability due to atrophy secondary to Poland’s syndrome, we conclude that his arthropathy is associated with his Poland’s syndrome. We recommend that children with Poland’s syndrome to be screened for charcot’s arthropathy so that proper measures can be taken before the advancement of the disease.

Third Place – Medical Student Submissions:

SUDDEN CARDIAC DEATH AND HYPERTROPHIC CARDIOMYOPATHY
Salma Patel MD MPH, Abstract 18

Hypertrophic cardiomyopathy (HCM) is a genetically determined disease, characterized phenotypically by unexplained thickening or hypertrophy of the left and sometimes right ventricle. The feared and devastating complication of HCM is sudden cardiac death. This 60 year old gentleman has a history of HCM. He has a concentric increase in left ventricular wall thickness with the septum measuring 20mm. There is systolic anterior motion (SAM) of the mitral valve apparatus with a resultant resting left ventricular outflow tract gradient of 121mmHg and mild SAM associated mitral regurgitation. The left ventricular ejection fraction is 50% with no regional wall motion abnormalities. He had NYHA class II functional status limited by symptoms of shortness of breath. He was treated with Disopyramide CR 150mg bid and Toprol XL 100mg bid. There is no family history of HCM, or personal history of unexplained syncope, or nonsustained ventricular tachycardia (NSVT) on repeated Holter monitor evaluations. His father died at the age of 57 in his sleep, but was too noted to have cancer and was a heavy smoker. The blood pressure response to exercise is unknown. He was transferred from the Philippines one month following an out of hospital cardiac arrest complicated by respiratory failure requiring tracheostomy, sepsis, acute kidney injury requiring temporary dialysis, and quadriplegia felt secondary to critical illness myoneuropathy. On initial examination: HR 100bpm, BP 102/67mmHg, RR 15 and SPO2 99%. He was in no acute distress. Tracheostomy was in place.
and he was on a ventilator. Neurological exam was significant for only being able to wiggle his toes bilaterally. The carotid pulse was bifid. The PMI was at the apex, located in the SICS just lateral to the midclavicular line. The first and second heart sounds were normal in intensity. There was a fourth heart sound. A 3/6 systolic murmur was noted loudest at the LLSB. Coarse breath sounds were noted bilaterally. Abdomen was soft, nontender, nondistended. Extremities had 2+ pitting edema. Pulses were palpable and symmetric. Patient was weaned off the ventilator, resumed beta blocker therapy and underwent a dual chamber pacemaker and ICD implantation. He was discharged to a long term care facility for rehabilitation. This case highlights the imprecision of risk stratification for sudden cardiac death in those with HCM. Current indications for ICD therapy (primary prevention) are based on discussion, disclosure and informed consent and risk is stratified based primarily on the “big 5” independent risk factors (family history of SCD in a first degree relative, LV wall thickness >30mm, recent unexplained syncope, NSVT, and an abnormal BP response to exercise).

LIPIDIC NANOCARRIERS TO ENHANCE ANTICANCER PRO-APOPTOTIC ACTIVITY OF GENISTEIN

Jimmy Pham DO, Abstract 103

Lipidic Nanocarriers to Enhance Anticancer Pro-apoptotic Activity of Genistein  JIMMY TAM HUY PHAM 1, VU M. PHAN 1, TAMER ELBAYOUMI 2* 1 Midwestern University, College of Osteopathic Medicine-Glendale, Glendale, AZ 2 Midwestern University, College of Pharmacy-Glendale, Glendale, AZ (*corresponding author) OBJECTIVES: Our work focuses on the development and evaluation of lipid-based nanocarriers (NCs), namely liposomes (LIPs), nano-emulsions (NEs) and polymeric phospholipid micelles (MIC), as vehicles for improved oral and parenteral delivery of genistein, the major soy isoflavone best known for its ability to markedly inhibit cancer progression, angiogenesis, and metastasis. METHODS Prototype LIPs were prepared using egg phosphatidylcholine and Cholesterol (60-90:40-10 M%), mixed with increasing amount of genistein, via modified lipid-hydration technique. Polymeric MICs with different ratios of vitamin E-TPGS and PEG2000-phosphatidylethanolamine (PEG-PE) were stirred with genistein in acetone/methanol mixture, followed by solvent evaporation. Tocopherol-rich NE, were prepared by ultra-sonication. Physico-chemically-screened LIPs, NEs and MICs loaded with genistein were tested in vitro against murine breast carcinoma (4T1), human prostate carcinoma (PC3), and resilient colon cancer (C26). RESULTS Genistein-loaded vesicles showed high drug solubilization capacity (NE>MIC>LIP) and favorable nano-scale properties, leading to improved delivery of genistein, and superior cytotoxicity in cancers of different origins. Significant induction of morphological apoptosis in 4T1 with genistein-loaded LIP, was evident microscopically, compared to free drug and empty vehicle controls. Superior cytotoxicity was demonstrated for genistein MIC and NE, against 4T1, C26 and PC3 cancer cells, vs. treatment controls. IC50 values for genistein NCs were at least 6-15 folds < conventional drug solution. IMPLICATIONS Genistein-loaded NCs showed high solubilization capacity and favorable nano-scale properties, leading to improved delivery of genistein to various cancer cells. These genistein NCs can lead to therapeutic strategies using this potent pro-
apoptotic nutraceutical, as active adjuvant to augment current cancer chemotherapeutics.

GENISTEIN AND R117H CF MOUSE JEJUNUM: A LACK OF EFFECT IRRESPECTIVE OF ROUTE OF ADMINISTRATION
Esa Rayyan DO, Abstract 104

GENISTEIN AND R117H CF MOUSE JEJUNUM: A Lack of Effect Irrespective of Route of Administration
E. Rayyan, S. Polito, B. Barrett, A. Bhakta, J. Kang, L. Leung and L. Al-Nakkash

Cystic fibrosis (CF) results from the loss or reduction in function of the CFTR (cystic fibrosis transmembrane conductance regulatory protein) chloride channel. One CFTR mutation seen clinically is R117H, which has partial CFTR function. Genistein, a naturally occurring phytoestrogen, is known to stimulate CFTR function in vitro. We have previously shown that female wild-type mice (Wt, C57BL/6J) fed a genistein-containing diet (600 mg/kg food, 600Gd) for 4-weeks have significantly elevated basal levels of jejunal chloride (Cl) secretion (measured as transepithelial short circuit current, Isc) compared to genistein-free fed mice (0Gd). This was a gender-specific response to 600G diet; with no response in males. Interestingly, both male and female Wt mice injected with genistein (600 mg genistein/kg body weight, 600Gi) daily for 1-week have significantly elevated basal levels of Isc compared to DMSO treated controls (0Gi). We aimed to determine whether route of administration of genistein mediated differential effects in male and female R117H CF mice. In those R117H mice fed 600Gd or 0Gd diet for 4-weeks, basal Isc was unchanged in 600Gd male mice (39.2±11.7 µA/cm², n=9) compared to 0Gd controls (34.9±7.7 µA/cm², n=9), and unchanged in 600Gd females (36.7±8.6 µA/cm², n=10) compared to 0Gd controls (29.3±6.5 µA/cm², n=7). In those R117H mice injected with 600 Gi or 0Gi for 1-week, basal Isc was unchanged in 600Gi male mice (28.5±2.3 µA/cm², n=5) compared to 0Gi controls (27.5±5.5 µA/cm², n=4), and unchanged in 600Gi females (31.3±7.7 µA/cm², n=5) compared to 0Gi controls (38.5±5.2 µA/cm², n=5). We have measured the effects of Isc in response to the following: the adenylate cyclase activator, forskolin (10 µM, bilateral) the Na+/K+/2Cl- co-transporter, bumetanide (100 µM, basolateral) to indicate the Cl- secretory component and acetazolamide (100 µM, bilateral) to indicate the HCO3- secretory component. Morphology analysis (i.e. villi length, number of goblet cells/villi, crypt depth, number of goblet cells/crypt) for the R117H mice is currently underway. These data suggest that, in both male and female R117H mice, neither a 4-week genistein diet treatment nor 1-week of daily genistein injections, provide any benefit towards the overall magnitude of basal secretion, indicating that partial CFTR function is not sufficient for genistein’s action. These effects are in contrast to the stimulatory effects of genistein on Wt mice. Sarah Polito, Ashesh Bhakta and Jonathan Kang were supported by Midwestern University Summer DO Fellowships. Layla Al-Nakkash was supported by the Soy Health Research Fund. R117H mice were generously provided from Dr. M. L. Drumm (Case Western Reserve University).

HYPERPARATHYROID CRISIS SECONDARY TO PARATHYROID ADENOMA
Muhammad Omar Salim DO, Abstract 22

Introduction: Hyperparathyroid crisis secondary to a parathyroid adenoma is a rare
and critical disease that requires immediate surgery to avoid fatality. Case Study: A 50 year old female with PMHx of HTN and GERD presented to ED complaining of lethargy, HA, and balance problems. She was found to be severely hypercalcemic at 26.1 mg/dL (nl: 8.4-10.2) and in acute renal failure with a creatinine of 1.8 mg/dL (nl: .6-1.2) and albumin of 3.5 g/dL (nl: 3.5-5.5). She was aggressively hydrated and given furosemide, calcitonin, and pamidronate. Her calcium did not improve, so she underwent hemodialysis. PTH level was 2,090 pg/mL (nl: 230-630). The patient received a CT / thyroid US revealing left sided parathyroid and thyroid nodules. Surgery consult performed a neck exploration with left inferior parathyroidectomy and left thyroid lobectomy. Pathology reveals a parathyroid adenoma and adenomatous and hyperplastic nodularity of thyroid with no evidence of malignancy. Postoperatively, the patient recovers, but noted with worsening nonoliguric acute renal failure with creatinine of 4.7 mg/dL. Calcium had improved drastically, and actually found low at 7 mg/dL. Patient was started on calcium supplements. PTH levels improved and normalized. Repeat TSH / Free T4 were unremarkable. Patient was later discharged after a 13 day hospital course. Discussion: The prevalence of PHPT is approximately 1:1000, most commonly manifested as an asymptomatic disease. Acute PHPT is rare and can precipitate a parathyroid crisis, characterized by life-threatening hypercalcemia that can cause muscle weakness, headache, neurological symptoms, and renal failure as described in the patient. Elective parathyroidectomy is the most appropriate treatment for PHPT. In surgical series there is an estimated reported prevalence of intrathyroidal parathyroid adenoma that varies from 1.4-6%. Hyperparathyroidism due to parathyroid abnormalities are classified into three forms: primary, secondary, and tertiary. Parathyroid adenoma accounts for about 85% of all cases of hyperparathyroidism, considering all the forms: primary, secondary and tertiary. Other less prevalent causes include parathyroid hyperplasia (15%) and carcinoma (3-4%). Conclusion: The hypercalcemic crisis of hyperparathyroidism due to a parathyroid adenoma is a rare endocrine emergency that is fatal without surgical intervention. Despite emergency parathyroidectomies, mortality rates remain high. Even postoperatively, the danger of a rapid decline of serum calcium contributes to complications. Patients should be hydrated and have their calcium consistently monitored to allow gradual, stable decline. Citations: Case Report Med. 2010;2010:596185. Epub 2010 Dec 20. J Emerg Trauma Shock. 2012 Apr-Jun; 5(2): 196–198. doi: 10.4103/0974-2700.96497

LACTIC ACIDOSIS AND HYPOGLYCEMIA SECONDARY TO MALIGNANCY Muhammad Osman Salim DO, Abstract 23

Introduction: Lactic acidosis in the absence of hypoxia or tissue hypoperfusion (type B) is rare and associated with drug usage or malignancy. Hypoglycemia complicating lymphoma/leukemia is an unusual event and indicates grave prognosis. Case Study: 37 year old female with PMHx significant for NK/T-cell lymphoma diagnosed 1 year ago began chemotherapy 3 months after diagnosis. She was admitted in January 2012 for her fifth cycle of chemotherapy. During that admission, the patient left AMA before chemotherapy completion. She returned in February complaining of epistaxis and rectal bleeding for days. She was febrile at 100.4°F and pancytopenic. Filgrastim was given for 3 days as
well as empiric antibiotics and antifungals. The patient was also found to have acute renal failure with a creatinine level of 4.5 mg/dL (nl: .6-1.2), lactic acid of 3.5 meq/L (nl: .7-2.1), and metabolic acidosis with hyperuricemia. She was hydrated and given allopurinol. Due to worsening acidosis, a bicarbonate drip was initiated. The patient’s WBC count recovered, however, she continued to have a leukemoid reaction with WBC elevation up to 79,000/mm3 (differential of 13% lymphocytes, 13% myelocytes, 5% premylocytes, 13% bands, and 40% neutrophils). An elevated creatinine persisted, contributing to development of pedal edema and anasarca. Lactic acid level continued to rise in parallel to the white cell count up to 24meq/L in absence of hypotension and negative microbial cultures. Serum glucose was low as 9 mg/dL (nl: 70-110) with patient only mildly symptomatic. Lactic acidosis was resistant to sodium bicarbonate, and the patient’s profound hypoglycemia required glucose infusions of 20% dextrose. Due to a grim prognosis, the patient wished for DNR and no further treatment with discharge to a hospice facility. Discussion: Natural Killer/T-cell lymphoma can cause metabolic deviations, including lactic acidosis and hypoglycemia. This is because of excessive metabolism due to the carcinogenic virulence, though exact mechanisms are not clearly understood. True hypoglycemia complicating lymphoma is a rare event, attributed to overproduction of lactic acid as well as to an impaired hepatic gluconeogenesis in the presence of leukemic cells. Most reported cases of hypoglycemia with leukemia have been ascribed to in vitro glycolysis by abnormal leukocytes and have been labeled "artificial" hypoglycemia. And those were only reported with high WBC counts (200,000/ mm3). Conclusion: Malignancy causing type B lactic acidosis and hypoglycemia is an exceedingly rare and poorly understood manifestation associated with a high mortality rate. This combination may lead to considerable morbidity and mortality. Regular monitoring for this complication should be part of lymphoma management. Emergency blood glucose analysis should be performed on any patient with malignant disease who presents with confusion, drowsiness, or loss of consciousness. Special consideration should be paid to potential artificial hypoglycemia secondary to in vitro glycolysis.

VOLUNTARY EXERCISE TRAINING IMPAIRS THE EXPRESSION OF CARDIAC NATRIURETIC PEPTIDES IN THE OB/OB MOUSE

Leslie Tamura, Abstract 9

Regular exercise is generally recommended for the treatment of obesity and type 2 diabetes. Exercise reduces body weight, improves glycemic control and cardiovascular function. This study was designed to determine the impact of voluntary wheel running on the metabolic state and cardiac oxytocin-natriuretic peptide system in the ob/ob mouse, a model of insulin resistance and obesity. We hypothesize that voluntary exercise training improves the metabolic status of ob/ob mice, resulting in a corresponding improvement in the oxytocin-natriuretic peptide system. Five-week-old male ob/ob mice and non-obese heterozygote control littersmates were assigned to sedentary or voluntary running groups for 8 weeks. Body weight and blood profile were not altered by running in control mice, despite a 4-to-6-fold greater running activity. In obese mice, despite reduced running activity, running decreased body weight but failed to improve blood glucose, glycated hemoglobin, insulin and triglyceride levels. Expression of the cardiac oxytocin receptor, A-type, B-type, and C-type
natriuretic peptides remained unchanged after running in control mice. In obese mice, however, voluntary running induced a down regulation in the expression of all three natriuretic peptides. In conclusion, while voluntary exercise running is not associated with an improvement in cardiovascular markers and natriuretic peptides in non-obese mice, this form of training is associated with a reduced expression of key cardioprotective peptides.

THE EFFECTS OF RESVERATROL ON THE HEART AND PULMONARY ARTERIES IN A MODEL OF MONOCROTALINE-INDUCED PULMONARY HYPERTENSION

David Wilson DO, Abstract 123

Pulmonary hypertension (PH) is a disease characterized by irregular vascular remodeling, upregulation of inflammatory cytokines, and a decrease in vasoactive agents that eventually lead to an increased resistance in the pulmonary arterial circuit and increased stress on the right ventricle. If untreated this leads to RV hypertrophy, arrhythmias, and heart failure. Monocrotaline (MCT), a pyrrolizidine alkaloid, has been used widely to induce and study pulmonary hypertension in rats. Resveratrol (3,5,4''-trihydroxy-trans-stilbene), a dietary polyphenol found in the skins and seeds of grapes, nuts, and some berries has been found to have antioxidant, anti-inflammatory, and other beneficial effects on the heart. The purpose of this study was to examine the effects of monocrotaline on heart and pulmonary artery structure of rats treated with resveratrol (RES). Pulmonary hypertension was induced in male Sprague-Dawley rats by a single subcutaneous injection of monocrotaline (50 mg/kg of body weight) after which they were given resveratrol (25 mg/kg of body weight) by oral gavage over a 21-day period before being sacrificed. PH was confirmed by seeing significant: decreases in body weight of 9% in the MCT group and 11% in the MCT + RES group, increases in wet lung weight of 40.44% in the MCT group and 43.16% in the MCT + RES group, and increases in right ventricle lumen area of 195.76% in the MCT group and 131.74% in the MCT + RES group compared to control. Right ventricle lumen area increased without increases in total muscle area indicating a dilation of the lumen without noticeable hypertrophy of the cardiac muscle tissue. Resveratrol appeared to have no significant treatment effect on the cardiac or pulmonary vascular muscle.

A DIFFUSE CLOSTRIDIUM SEPTICUM INFECTION: CASE REPORT AND LITERATURE REVIEW

Saba Ali MBBS, Abstract 84

Intro: Clostridium Septicum is a gram-positive, spore forming, anaerobic bacterium that causes gas gangrene, an opportunistic infection that can result in devastating tissue necrosis. Unlike other Clostridium species, C. Septicum occurs spontaneously, in that no trauma is necessary at the site of infection and it can cause rapidly metastatic myonecrosis. It has been hypothesized that the infection is established by hematogenous spread from the gastrointestinal tract and has been associated with colorectal cancer and other defects of the bowel. Case: We present a case of a 63 year old male with past medical history of gastric cancer s/p gastrectomy, uncontrolled diabetes, and ETOH dependence who presented with epigastric pain of 1 week duration. Initial investigations revealed pneumobilia on abdominal imaging and sepsis with
polymicrobial bacteremia secondary to ascending cholangitis. The patient died 12 hours after admission following unsuccessful attempts at resuscitation for respiratory arrest. An autopsy revealed multifocal necrosis with associated gas pockets of multiple visceral organs including the heart, liver, bowels, and spleen due to C. septicum bacteremia.

Conclusion: The lack of colorectal malignancy or other bowel pathology, along with the incidental renal cell carcinoma discovered on autopsy, may create a new link between C. Septicum and renal malignancy that has never been reported before. The severity of this case and the multiorgan involvement make it a rare presentation for C. Septicum bacteremia. In addition, the route of entry into the bloodstream in this case was ascending cholangitis which is different from the previously described hypothesis of hematogenous spread from bowel malignancy or injury. Anaerobic sepsis is uncommon, but given this patient’s comorbidities, he was vulnerable to this overwhelming infection.

HYPONATREMIA AND ADRENAL FAILURE AS PRESENTING FEATURES OF BILATERAL PRIMARY ADRENAL LYMPHOMA

Saifuldeen Al-Qaisi MD, Abstract 24

Introduction: Primary adrenal lymphoma (PAL) is a rare clinical entity, and it presents usually with adrenal insufficiency (AI). So it is always important to put in the differential workup PAL as the cause behind AI and to consider it especially if Adrenal masses or enlargement is found in the Imaging studies like CT. Case presentation: 74 year old man without significant past medical history, presented to the hospital with progressive weakness and nausea for 6 weeks. He denies vomiting, abdominal pain and diarrhea. His review of system was negative other than the history of present illness. Physical exam was unremarkable with no skin pigmentation, no LAD or HSM. The rest of the physical examination was normal. BP was 105/50 with heart rate in the 80’s. Initial workup found sodium of 116mEq/L potassium 5.1mEq/L, creat 1.6mg/dl, white count 3.4, HB 12.3, platelets 120000, calcium 9.1mg/dl, LDH 161IU/L, Glucose 92mg/dl, normal Liver function test and normal thyroid function tests. Work up of hyponatremia showed high urine osmolality with low serum osmolality raising suspicion of SIADH. Further work up of low sodium revealed low serum cortisol of 2.3 Mg/dl. Serum cortisol remained low at 2.5 one hour -post cosyntropin stimulation test. Baseline ACTH level came back later significantly elevated 1135 (6-50 pg/mL) confirming primary adrenal insufficiency. CT abdomen pelvis showed enlarged adrenal glands, right adrenal glands measured 6 cm in length and left adrenal glands 4 cm in length. Adrenal lesions demonstrate intermediate to high signal on the T2 weighted MRI. Chest and brain CT scans were negative for any mass. Because of pancytopenia and probability of malignancy, bone marrow aspiration was done and was negative for malignancy. Biopsy of adrenal gland showed high grade B - cell non Hodgkin lymphoma. Discussion: Primary adrenal lymphoma (PAL) is an extremely rare entity. It is characterized by a high incidence of bilateral adrenal involvement of diffuse large B-cell lymphoma. The poor outcome of primary adrenal lymphoma was speculated to might be related to the bulky tumor size at presentation, non-germinal center B-cell phenotype, and frequent BCL-6 gene rearrangement (1). Hyponatremia and subtle clinical signs of adrenal insufficiency led to a diagnosis of Addison’s disease, a common complication of primary adrenal lymphoma. Thus, bilateral
adrenal masses associated with Addison’s disease should raise the suspicion of possible primary adrenal lymphoma. In cases of suspicious primary adrenal lymphoma, percutaneous computerized tomography or ultrasonography-guided needle biopsy can help to avoid unnecessary surgeries. References:

THE AXIAL SUPPORT BALLOON ANCHORING TECHNIQUE IN THE INTERVENTION OF CHRONIC TOTAL OCCLUSIONS

Seth Assar MD, Abstract 11

Introduction: Innovative techniques using dedicated systems are safely advancing and making more accessible chronic total occlusions by way of percutaneous coronary intervention. The axial anchoring technique involves deploying an over-the-wire balloon catheter proximal to the target CTO and utilizing the system for both the generation of directed support, as well as for wire exchange. We present a case utilizing the axial support anchoring balloon technique in treatment of chronic total occlusions in a patient who has failed medical therapy.

Case Description: A 78 year old male with a past medical history significant for coronary artery disease status-post coronary artery bypass graft surgery presented with fatigue on minimal exertion as well as persistent pain radiating into his left shoulder. The patient also demonstrated resting ECG ST-segment depressions in the inferior leads. Angiography 22-years prior had diagnosed a chronic total occlusion of the right coronary artery. After failing maximal medical therapy, the choice was made to attempt percutaneous revascularization of his occluded artery. The patient underwent percutaneous coronary intervention via the radial artery approach. An over-the-wire (OTW) balloon using the anchoring balloon technique was applied proximally to the lesion which was able to advance an initial hydrophobic wire half way across the CTO. This wire was then exchanged for a hydrophilic wire. With the anchoring balloon in place, the swapped wire was able to easily navigate the lesion. A total of four everolimus-eluting stents were placed in the artery, beginning in the ostium. Angiogram following this showed improvement in TIMI flow from 0 to 3. The patient tolerated the procedure well and there were no complications. At follow-up, the patient reported that his shoulder pain and fatigue had completely resolved.

Discussion: CTO recanalization can result in improved ventricular function, reduction in symptomatology, and ultimate need for surgery. Furthermore, upon PCI, recanalization rate is an independent predictor associated with reduction in 12-month incidences of sudden cardiac death and myocardial infarction. Chronic total occlusions can be successfully treated with specified systems that have proven superior in terms of procedural success and patient outcomes compared to conventional mechanical wires. In this example, an axial support balloon was chosen because conventional guide wire crossing had failed - a point at which many operators would have aborted the procedure. Further restructuring and specifying of medical devices intended for chronic total occlusions are evidently not only needful, but foreseeable. While there is much work ahead to advance access to a once inevitable cardiothoracic surgery, the convergence to technique, exposure, and subsequent experience of interventional cardiologists may pave the way for greater outcomes under less risk.
AUTOIMMUNE POLYGLANDULAR SYNDROME IN A 19 YEAR OLD MALE

Anjuli Brighton MD, Abstract 96

Introduction Autoimmune polyglandular syndrome type II (APS-2), also known as Schmidt’s syndrome, is a rare endocrine disorder characterized by the presence of adrenal insufficiency with autoimmune thyroid disease and/or type 1 diabetes mellitus (1). Hypogonadism, pernicious anemia, and vitiligo may also accompany these disorders (1).

Case Report A 19 year-old Caucasian male presented to our institution with lightheadedness upon standing from a seated position, fatigue, and generalized weakness for one month. He had experienced salt cravings over the preceding two weeks. Four days prior to presentation, he developed nausea, vomiting, and low systolic blood pressures measured at home. He had no previous medical conditions, did not take any regular medications, had no allergies, and is a nonsmoker and nondrinker. Family history was significant for hypothyroidism in a brother.

On initial evaluation, the patient was noted to be hypotensive at 81/39. Physical exam was remarkable for an enlarged thyroid gland, slight hyperpigmentation and reduced secondary sex characteristics. Labs were significant for the following: sodium 124 mmol/L (135-145), potassium 5.6 mmol/L (3.8-5.0), thyroid stimulating hormone (TSH) 12.75 mIU/L (0.30-5.00), free T4 0.6 ng/dL (0.6-1.6), thyroglobulin antibody 370 IU/L (<116), adrenocorticotrophic hormone (ACTH) 867 pg/dL (10-60), prolactin 26 ng/dL (3-13), testosterone 171 ng/dL (240-950), plasma glucose 91 mg/dL (70-100). A morning cortisol level was <1.3 mcg/dL. Cosyntropin stimulation test reported undetectable cortisol levels at 0, 30 minutes, and 60 minutes. The patient was volume resuscitated with 3 liters of normal saline and an Endocrinology consult was obtained. The patient was started on hydrocortisone, 20mg daily at 0800 and 10 mg daily at 1700 (20-0-10-0). On hospital day 2, the patient’s blood pressure normalized and his symptoms improved. The patient was discharged on hospital day 3 with a prescription for levothyroxine and a scheduled Endocrinology outpatient follow-up.

Discussion This case demonstrates the simultaneous presentation of two endocrinopathies in a young male, leading to a diagnosis of APS-2. Alongside a classic presentation of adrenal insufficiency with hypotension, hyponatremia and hyperkalemia confirmed with Cosyntropin stimulation testing, initial testing revealed a concomitant hypothyroidism. Existing literature notes that the prevalence of adrenal insufficiency in APS-2 is 100% and the prevalence of thyroid disease in APS-2 is 69% (1). The patient’s prolactin and testosterone will be rechecked on follow-up to ensure that they normalize on correction of his thyroid function. While the endocrine disorders constituting APS-2 may present many years apart (2), this case demonstrates the value of a complete initial workup to ensure initiation of appropriate therapy, since hydrocortisone should be commenced before levothyroxine to avoid precipitating adrenal crisis. (1) Neufeld M, Maclaren NI, Blizzard RM. Medicine 60: 355-362 (1981). (2) Michels, AW & Gottlieb, PA. Nat Rev Endocrinol. 6: 270-277 (2010).

ANXIETY: CAN IT BE LIFE-THREATENING?

Kathleen Casamiquela MD, Abstract 105

Introduction: Severe hypophosphatemia is often life threatening and has several etiologies. We describe a very rare presentation
of an otherwise healthy young man who presented with symptomatic and severe hypophosphatemia. Case Report: A 24 year old man with no past medical history presented to the emergency room with chest pain, shortness of breath, weakness, palpitations, and generalized paresthesia. He had similar presentation at an outside hospital and underwent an extensive medical workup to rule out life threatening causes of such symptoms. Physical examination during this visit was unremarkable except for tachycardia and tachypnea. Laboratory investigations showed undetectable serum phosphorus levels. A CT angiogram of the chest showed no evidence of pulmonary embolism or any acute pulmonary findings. It was concluded that the presentation was most likely as result of hypophosphatemia. Repletion of phosphorous resulted in rapid correction. Subsequently, work up for several etiologies for hypophosphatemia was negative. ABG showed respiratory alkalosis. Fractional excretion of phosphorous was consistent with renal wasting. Based on the entire evaluation, it was concluded that the most likely etiology for hypophosphatemia was patient’s anxiety disorder with panic attacks. A formal psychiatric evaluation confirmed this diagnosis. The patient was placed on SSRI and as needed benzodiazepine and phosphorous levels checked on a weekly basis were in normal range Discussion: Symptoms of hypophosphatemia are hard to recognize since they can mimic more serious medical problems such myocardial infarction and pulmonary embolus. The causes of hypophosphatemia include malabsorption and chronic diarrhea, vitamin D deficiency; poor intestinal absorption associated with antacid abuse, alcoholism, starvation due to anorexia, primary hyperparathyroidism, and increased urine loss of phosphorous in fanconi syndrome, and oncogenic osteomalacia. Our patient denied chronic diarrhea, his Vitamin D level was in normal range and he denied the use of antacids. He had a mildly elevated PTH - 69 (normal 7-53) and normal calcium levels. A sestamibi scan was performed which showed no adenoma. His bicarbonate was mildly low throughout the hospitalization. His mild acidosis was most likely due to chronic respiratory alkalosis and not RTA or Fanconi syndrome. Finally, fibroblast growth factor was 52 (normal <180) which eliminates tumor-induced osteomalacia. It was concluded that the patient’s anxiety was causing him to hyperventilate which led to respiratory alkalosis. Respiratory alkalosis due to hyperventilation causes transient intracellular shift of phosphorous stores. Hence, although the serum phosphorus levels can be profoundly low, there is no true phosphate depletion. Conclusion: Although uncommon, anxiety is an important cause of transient hypophosphatemia. It is a diagnosis of exclusion but needs high level of suspicion since severe hypophosphatemia can be life threatening.

DOUBLE TROUBLE: HYPERCALCEMIA AND PRIMARY ADRENAL INSUFFICIENCY ASSOCIATED WITH COCCIDIOIDOMYCOSIS AND HIGH DOSE FLUCONAZOLE
Nam Chan MD, Abstract 97

Introduction Although uncommon, both coccidioidomycosis and adrenal insufficiency can cause hypercalcemia. We report a case of severe hypercalcemia in the setting of severe coccidioidomycosis with newly diagnosed primary adrenal insufficiency. Case description The patient is a 68-year-old African American male who moved to Arizona 3 years ago, presented with a one-year history of fatigue,
weight loss, and cough. He was diagnosed with pulmonary coccidioidomycosis in March with a titer of 1:64. Fluconazole 400mg daily was initiated with symptom improvement. At his two-month follow-up, the fluconazole dose was doubled due to worsening titer (1:256). Over the next two months, his health declined and was admitted for failure to thrive. Exam was significant for lethargy, joint tenderness, generalized weakness, dehydration, hyperpigmentation and nodular lesions on the shins bilaterally. Labs were notable for severe hypercalcemia, 17.2 and renal failure, BUN of 44 and creatinine of 2.2. He had suppressed levels of PTH, 25-hydroxyvitamin D, calcitriol, and negative PTHrP. Coccidioides titer was 1:256, despite 4 months of fluconazole. Morning cortisol level was low at 4.2 and unresponsive to ACTH stimulation. MRI of the abdomen showed new bilateral adrenal hyperplasia. He was treated with AmBisome, hydration, pamidronate and hydrocortisone. His lethargy and anorexia resolved in two days. Calcium level was normalized by day 5. AmBisome was replaced with voriconazole prior to discharge on day seven.

Discussion
The severe hypercalcemia in our patient was likely multifactorial. Disseminated coccidioidomycosis is an uncommon but reported cause of non-PTH-mediated hypercalcemia. In sarcoidosis, macrophages produce calcitriol independent of PTH and the elevated calcitriol level increases intestinal calcium absorption. In coccidioidomycosis, the role of calcitriol is unclear. Most reported cases with calcitriol measurement showed suppression, as in our patient. However, three cases have documented calcitriol levels that were inappropriately high in the presence of hypercalcemia. One case of disseminated coccidioidomycosis and hypercalcemia with elevated serum PTHrP has been reported. Another theory for hypercalcemia in coccidioidomycosis involves circulating unidentified osteolytic or osteotropic substances. Hypercalcemia is a rare complication of adrenal insufficiency, usually associated with renal insufficiency. Interestingly, clinical deterioration in our patient temporally correlated with the increased titer and subsequently increased fluconazole dose. Five cases of reversible adrenal insufficiency associated with fluconazole have been reported, either with high doses or in critically ill patients. None of these patients had adrenal abnormalities on imaging. Four cases of adrenal coccidioidomycosis resulting in primary adrenal insufficiency have been reported. Two of these had extensive tissue destruction on autopsy - one with marked bilateral adrenal enlargement and the other without. A third patient had bilateral adrenal masses on imaging studies. Given the variable appearance of the glands in these reports, bilateral adrenal hyperplasia in our patient may represent adrenal involvement of coccidioidomycosis.

BACTEREMIA AND COLON CANCER: A ‘STREP’ IN THE RIGHT DIRECTION
Molly Disbrow MD, Abstract 77

Introduction: Streptococcus gallolyticus subspecies gallolyticus (formerly S. bovis) bacteremia of undetermined etiology is often associated with an underlying colonic neoplasia. Case Presentation: An 87-year-old male with prior mitral and aortic valve replacement, permanent pacemaker, and Barrett’s esophagitis, presented to the emergency room with generalized weakness and disorientation. Further questioning identified recent onset of chills, cough, chest pain, and dyspnea. He was found to be
Two sets of blood cultures revealed growth of Streptococcus bovis, susceptible to penicillin. Intravenous vancomycin was initiated. Transthoracic echocardiogram did not reveal any vegetations. Repeat blood cultures were negative. He requested transfer to Mayo Clinic for further evaluation. A transesophageal echocardiogram did not identify valvular or pacemaker lead vegetations. Given the well-described association between S. bovis bacteremia and colonic neoplasia, upper and lower gastrointestinal endoscopies were performed. Esophagogastroduodenoscopy revealed chronic inflammation and Barrett’s epithelium. A colonoscopy was remarkable for an ulcerated, partially obstructing, large mass in the distal transverse colon. The mass involved one-half of the luminal circumference. A colonoscopy four years prior had been unremarkable, aside from diverticulosis. The colonic mass was biopsied and demonstrated moderate-to-poorly differentiated invasive adenocarcinoma. No evidence of metastasis was present on staging computed tomography and CEA was within normal limits. The patient has since consulted a colorectal surgeon and plans are pending for surgical intervention. In the interim, he is completing a six-week course of intravenous ceftriaxone. Discussion: Streptococcus galolyticus subspecies galloyticus (SGG) bacteremia has been well described in patients with underlying colonic neoplasia. It was formerly referred to as S. bovis. It can lead to various clinical syndromes, including bacteremia, infective endocarditis, diskitis, septic arthritis, and abscesses. SGG is usually susceptible to penicillins and cephalosporins. In the absence of a clear source of SGG bacteremia, it is imperative that patients undergo a diagnostic colonoscopy to exclude underlying colonic neoplasia.

HYPERTROPHIC OBSTRUCTIVE CARDIOMYOPATHY (HOCM) AND CONCURRENT ACUTE PULMONARY EDEMA WITH CARDIOGENIC SHOCK: WALKING AROUND LANDMINES

Carlos Echevarria MD, Abstract 4

Introduction: Hypertrophic cardiomyopathy is a common inherited condition present in 1 in 500 of the general population. Historical misconceptions about high mortality remain due to skewed referral patterns to large tertiary specialty centers, but mortality rates in such patients are, in fact, not significantly different from the general population. Nevertheless, complications attributable to HOCM may dramatically worsen the prognosis of these patients. We report a case of HOCM complicated by pulmonary edema and cardiogenic shock, and bring to light the challenges faced in the medical management of concurrent conditions that warrant contraindicated therapy. Case Description: A 37 year old male with past medical history of a syncopal episode presented to the hospital in acute respiratory distress. Physical exam was pertinent for hypoxia, tachypnea, pulmonary crackles and lower extremity edema. Chest x-ray confirmed pulmonary edema. He was started on IV furosemide and BIPAP for acute respiratory failure. He failed to improve and was transferred to the ICU for mechanical ventilation. Transthoracic echocardiography showed marked septal hypertrophy with systolic anterior motion of mitral valve, causing significant obstruction of the left ventricle outflow tract and severe mitral regurgitation. The consultant cardiologist recommended stopping diuretics and starting gentle hydration along with beta-blockers. A dose of IV metoprolol immediately caused bradycardia,
hypotension and subsequent cardiac arrest. He was successfully resuscitated and started on phenylephrine drip, as positive inotropes were deemed prohibited. Beta-blockers were placed on hold to avoid further decompensation. In light of his hemodynamic instability, a diagnostic cardiac catheterization along with an intra-aortic balloon pump placement (IABP) was undertaken by cardiology, which revealed normal coronaries and a LV systolic gradient of 70mmHg. He continued to require vasopressors despite IABP placement. Labs during the hospital course showed worsening acidemia and hyperkalemia for which he was given calcium gluconate and bicarbonate. Unfortunately, he developed pulseless electrical activity and all attempts at resuscitation failed. He was pronounced dead within 18 hours of his initial presentation.

Discussion: This case highlights the challenges encountered when managing HOCM with concurrent congestive heart failure and shock. Rapid diuresis likely resulted in deterioration of the condition. Recent studies about HOCM have shown that the use of IABP may have questionable results. With a wide gamut of relative to absolute contraindications, from diuretics to positive inotropes, the question about ideal treatment of such patients remains unanswered. We propose a protocol for the development of an ED screening tool that would capture this type of patients prior to blind diuretic therapy and for urgent transfer to a quaternary medical center for extracorporeal membrane oxygenation, myomectomy, and possible transplant.

First Place – Associate Research Poster:

ESSENTIAL THROMBOCYTHEMIA (ET) AND POLYCYTHEMIA VERA (PV) SYMPTOM BURDEN: PHENOTYPIC CLUSTER ANALYSIS AMONG AN INTERNATIONAL SAMPLE OF 1,141 PERSONS WITH ET AND PV

Robyn Emanuel MD MPH, Abstract 56

PURPOSE: Symptom burden among persons with ET and PV can be severe and adversely affect QOL and considerable symptom heterogeneity exists between patients. No studies to date have empirically evaluated whether disease characteristics can be grouped in related symptom clusters. METHODS: Using the Myeloproliferative Neoplasm Assessment Form (MPN-SAF) (Blood 2011;118:401-408) given in conjunction with the Brief Fatigue Inventory (Cancer 1999;85:1186-1196), data regarding disease burden was collected from an international cohort of MPN patients. Symptom cluster development was based on r-squared in hierarchical clustering using Ward linkage. ANOVA and chi-squared tests were used for comparisons between clusters. RESULTS: Subject Demographic and Disease Characteristics: Data from 1,141 subjects with PV (N=519) and ET (N=622) was prospectively collected (Chinese 236, French 305, German 45, Italian 114, Dutch 191, English 56, Spanish 109, Swedish 85). Age (mean 59, range, 26–87) and gender (54% F) were typical of this disease. Five clusters were selected to represent the whole. Frequencies of prior bleeding, spleen size, anemia, presence of any lab abnormality, language, gender, and MPN type varied significantly between clusters (P<0.05). Cluster 1: The “Reduced Symptom” Profile (n=421 (37%; 60% ET, 40% PV) The largest cluster, subjects had increased sexual difficulties and fatigue. There was a slightly
higher proportion of subjects with ET (60%) versus PV. Few lab abnormalities (28% prevalence) and less prior bleeding (3%) were present. Spleen size was smallest of any clusters (1 cm below costal margin). Cluster 2: The “Fatigue-dominant” Group (n=286 (25%; 56% ET, 44% PV)). Subjects in this cluster were predominantly female and had relatively few laboratory abnormalities (19%) than other cohorts. Complaints are characterized by high severity of fatigue compared to end-organ symptoms. Symptom profiles emphasize fatigue, QOL and insomnia with some end-organ complaints. Cluster 3: The “End-Organ Complaints” Group (n=210 (18%; 49% ET, 51% PV)). Male predominant (56%), subjects had mainly macro-vascular symptom complaints including sexual difficulties, insomnia, and overall QOL, with few microvascular related symptoms (low itching/night sweats). Cluster 4: “Cognitive Complaints” Cluster (n=110 (10%; 53% ET, 47% PV)). The smallest cluster and female predominant (64%), main complaints include fatigue, insomnia, concentration, numbness, and sad mood. Cluster 5: The “Highly Symptomatic” Cluster (n=114 (10%; 44% ET, 56% PV)). Subjects had many cognitive complaints and symptoms correlated with severe micro-vascular abnormalities (pruritus) and or splenomegaly. This cluster had the largest spleen sizes (mean 3 cm), the highest prevalence of prior thrombosis (29%), and frequency of lab abnormalities (43%).

CONCLUSION: This analysis offers new means of evaluating persons with PV and ET utilizing symptom clusters. Laboratory and physical abnormalities differed significantly between symptom clusters indicating that our groupings likely result from biological alterations present in specific disease phenotypes. Future studies should investigate correlations between clusters’ prognosis and genotype.

ACUTE EOSINOPHILIC PNEUMONIA MASQUERADING AS COMMUNITY ACQUIRED PNEUMONIA Natalie Ertz, Abstract 121

Introduction Community acquired pneumonia (CAP) is a common medical problem in both the outpatient and inpatient settings. Patients presenting with symptoms of pneumonia and an infiltrate on chest x-ray (CXR) in the setting of immunocompetence are treated by standard guidelines. Occasionally, rare pulmonary diseases, such as acute eosinophilic pneumonia (AEP), can manifest signs and symptoms that mimic infectious causes of pneumonia. Case Report A 69 year-old female, immunocompetent, and an Arizona resident with a history of atrial fibrillation on amiodarone for the past 17 months, was admitted with worsening shortness of breath, cough, and pleuritic chest pain. One week prior to presentation, she was diagnosed with CAP and completed a course of azithromycin. On admission she was afebrile, tachypneic, and mildly hypoxic, with diffuse crackles on auscultation. Pertinent laboratory values included a neutrophil predominant leukocytosis and mild hyponatremia. A CXR performed on admission, when compared to her previous study, revealed worsening of a heterogeneous infiltrate involving the right lower lung and new multifocal infiltrate of the right upper, left middle, and lower lobes. She was started on broad spectrum antibiotics and supplemental oxygen. She continued to worsen and thus her coverage was broadened to include fluconazole given findings of multifocal extensive infiltrates with bilateral mediastinal adenopathy on computed tomography of her chest. A bronchoalveolar lavage (BAL) of the right lower lobe was performed revealing yeast and pseudohyphae. All additional cultures and
studies were negative. On hospital day 4, her amiodarone was discontinued due to suspicion of amiodarone-induced lung toxicity. She subsequently required mechanical ventilation for hypoxic respiratory failure, and underwent an open lung biopsy, which revealed acute lung injury with features of eosinophilic pneumonia, favoring a drug reaction. High doses of steroids were initiated. Ultimately, a tracheostomy was placed and her steroids were tapered in parallel with signs of clinical improvement. Four weeks following admit, she was tracheostomy-dependent and discharged to a rehabilitation facility. Discussion Rare pulmonary diseases such as AEP can present similarly to CAP. In our patient’s case, evidence of a rapidly evolving multilobar pneumonia despite standard CAP therapy in an otherwise healthy 69 year-old female required a reassessment of differential diagnoses. Bronchoscopy with BAL is required to confirm the diagnosis. However, occasionally a lung biopsy may be necessary. In AEP, eosinophils collect in the alveoli either from drugs, environmental triggers, parasitic infections, malignancy, autoimmune, or idiopathic causes. The clinical presentation includes fever, dyspnea, and non-productive cough with progressive dyspnea that may evolve into acute respiratory distress syndrome. AEP is treated with high doses of steroids and respiratory support. Therefore, early identification of AEP is important in order to initiate the appropriate management, as the standard therapeutic therapies of both CAP and AEP are dramatically different.

Third Place – PGY-1 Submissions:

TACHYCARDIA INDUCED CARDIOMYOPATHY
Aaron Fernandes MD, Abstract 12

Introduction: Tachycardia induced cardiomyopathy (TIC) is one of the reversible causes of ventricular dysfunction, and is most commonly induced by a long standing supraventricular or ventricular arrhythmia. Prolonged tachycardia is an established cause of non-ischemic cardiomyopathy. Multiple neurohormonal and electrophysiological abnormalities have been hypothesized as the mechanism by which tachycardia leads to structural cardiac changes, however none have been confirmed. TIC has been linked with various tachycardias including atrial fibrillation, atrial flutter, ectopic atrial tachycardia, atrioventricular nodal reentrant tachycardia, atrioventricular reciprocating tachycardia and ventricular tachycardia. We report a rare case of TIC induced by atrial flutter. Case Description: A 64 year old male with history of atrial flutter, successfully cardioverted thirteen years ago, presented with worsening fatigue and lower extremity edema. During initial presentation, the patient complained of overall fatigue, but denied any chest pain or shortness of breath. Physical examination showed elevated jugular venous pressure, tachycardia, and only trace bilateral lower extremity edema. During initial presentation, the patient complained of overall fatigue, but denied any chest pain or shortness of breath. Physical examination showed elevated jugular venous pressure, tachycardia, and only trace bilateral lower extremity edema. A 12-Lead EKG demonstrated atrial flutter at a rate of 135 with a 2:1 conduction. Trans-thoracic echocardiogram showed severe global hypokinesis of left ventricular contractility with an impaired ejection fraction (EF) less than 20%, dilated right ventricle, and moderate left ventricular hypertrophy. Since the patient had a recent normal coronary angiogram, TIC became a possible diagnosis. The cardiology service was promptly consulted, and the patient immediately received a single 150 joule biphasic synchronized shock from which he successfully converted to normal sinus rhythm. He was provided rate control and anticoagulation acutely, and was discharged on...
these medications. Six weeks later, the patient presented for follow up. Repeat trans-thoracic echocardiogram showed resolution of the cardiomyopathy, with a normal left ventricular size, overall normal left ventricular systolic function with EF 60-65%. Discussion: Tachycardia induced cardiomyopathy is a rare complication of extended periods of rapid ventricular heart rates. The mechanism by which prolonged tachycardia causes structural changes in the heart remains unclear. Clinical presentation may vary from asymptomatic to symptomatic congestive heart failure (CHF). Although initial treatments for TIC and CHF are the same, in TIC additional aggressive early efforts should be made to restore sinus rhythm. After sinus rhythm is achieved, it is possible to obtain complete reversal of cardiac structural changes and left ventricular systolic dysfunction within 24 hours to several months. Risk for recurrent tachyarrythmias may exist, so routine surveillance with echocardiography and ambulatory monitoring for one to two years after initial clinical improvement is recommended, although exact guidelines currently do not exist. Prognosis for left ventricular disease in TIC depends on the rate and the duration of the tachyarrythmia that influenced the cardiac dysfunction. When the tachyarrythmia is treated appropriately, the systolic function will improve.

First Place – PGY-1 Submissions:

PANCREATITIS AND A RENAL INCIDENTALOMA; BIG AND BENIGN
Colin Fitterer MD, Abstract 37

Renal oncocytomas are benign tumors that represent 3-7% of all renal neoplasms and have a reported average size of 4.9 +/- 2.7 cm. Based on clinical presentation and abdominal imaging, pre-operative diagnosis and differentiation from renal cell carcinomas proves to be difficult. A 59 year-old male presented to our institution with symptoms of acute onset abdominal pain, nausea, and vomiting. He was diagnosed with acute pancreatitis with a significantly elevated serum lipase and mild to moderate peri-pancreatic fluid collections on computed tomography of his abdomen and pelvis. A more ominous radiographic finding at that time included a large, approximately 10 centimeter right renal mass with heterogeneous enhancement and a central low density area of necrosis versus scarring, highly suspicious for renal cell carcinoma (RCC). The etiology of the patient’s pancreatitis was unclear as he denied alcohol use, had no evidence of gallstones, hypertriglyceridemia, hypercalcemia, or other known risk factors. He was managed conservatively and after complete resolution of his pancreatitis, he underwent a laparoscopic radical right nephrectomy 7 weeks later. Gross inspection of the tumor revealed a mahogany brown, focally hemorrhagic mass with a central stellate-appearing area of fibrosis. The histologic diagnosis was renal oncocytoma. A number of radiologic features, including homogeneous enhancement and the presence of a characteristic central scar, have been associated with oncocytomas. However, these findings are not highly specific. This case report reviews the proposed radiologic features of oncocytomas versus RCC and the appropriateness of consideration for preoperative core biopsy or watchful waiting. Ultimately, aggressive surgical resection and tissue diagnosis of solid renal masses remains necessary in most instances.
A “MULTIPLE” PRE-TRANSPLANT EVALUATION
Diana L Franco MD, Abstract 99

Introduction: Hepatic involvement in Multiple Myeloma (MM) is commonly reported as a post-mortem finding, ranging from 26 to 46% of the cases. MM presenting with a predominant hepatic involvement is exceedingly rare. Several reports have suggested that Hepatitis C Virus (HCV) infection is associated with B-cell lymphoproliferative disorder; however, a direct causative role of HCV in MM has yet to be established. We report a case of a 56-year-old male who presented to our facility for a combined liver and kidney transplant evaluation which was thought to be caused by HCV infection and chronic alcohol abuse (in remission). Case Report: A 56-year-old male with a 20-year history of HCV infection, genotype 1a, confounded by chronic alcoholism was referred to our institution for an evaluation for liver/kidney transplantation due to decompensated liver disease. Complications of cirrhosis included esophageal varices, ascites, encephalopathy and presumed hepatorenal syndrome. During his evaluation he reported chronic tongue ulcers. Laboratory analysis revealed pancytopenia, hypoalbuminemia, prolonged prothrombin time, hypercalcemia, and stage 5 chronic kidney disease for which he had already started hemodialysis. Computed tomography of his abdomen did not reveal evidence of hepatocellular carcinoma. A transthoracic echocardiogram showed interventricular septum thickening (15 mm) with grade II diastolic dysfunction. His tongue ulcers were biopsied given his tobacco and alcohol abuse. Pathology revealed positive birefringence under polarized light after Congo red staining consistent with lambda-type amyloid deposition (AL). Serum protein electrophoresis showed IgA lambda M-spires. A bone marrow biopsy was performed which showed atypical plasma cells with lambda light chain restriction, confirming the diagnosis of MM. Given the clinical picture, active treatment for his MM was deferred, transplantation denied and palliative care measures were instituted. The patient died shortly thereafter. Discussion: MM as a primary cause of cirrhosis is exceptionally rare. It is known that MM alone can cause amyloid type deposit that leads to liver failure, but the mixed pathophysiology that we see in this case highlights important diagnostic and management nuances. From the diagnostic perspective, this case illustrates the need to view all transplantation candidates with a very careful eye to eliminate all potential comorbidities that can complicate or limit the usefulness of solid organ transplantation. Furthermore, a rapid assumption that there can only be one unifying diagnosis, in this case the progression of portal hypertension to hepatorenal syndrome, can quickly lead to ignoring renal involvement of MM. A thorough, multi-disciplinary approach with an open mind to unusual mitigating circumstances can prevent expensive and potentially futile care. This case also leaves open the question of the possible role of HCV in the development of B-cell malignancies.

TREATMENT AND SURVIVAL TRENDS OF PATIENTS WITH BRAIN METASTASIS WITHIN THE MARICOPA INTEGRATED HEALTH SYSTEM
Carlo Guerrero MD, Abstract 57

Purpose: To determine whether gains in survival time after a diagnosis of brain metastasis were seen between the periods of 1997-1999 and 2007-2009 in patients within the Maricopa Integrated Health System (MIHS). Methods: Sample populations from the aforementioned time periods were compiled by
mining the electronic medical records for patients who were given a diagnosis of brain metastasis within each time period. In this manner, two discrete study populations were established: the Pre-period (7/1/1997-12/31/1999) and Post-period (7/1/2007-12/31/2009). These time frames were chosen to allow a reasonable interval of time for outcome comparison (10 years), as well as to generate appropriately-sized patient pools. The initial search was conducted using ICD-9 codes for any brain malignancy, so a review of each patient chart was necessary to identify and remove primary brain tumors from the study populations. The end result yielded a total of 26 patients in the Pre-period, and 42 in the Post-period. Survival time was deemed to be the period between diagnosis and death. In the event that a date of death was unknown (or if the patient was still alive), the date of the last clinic/hospital note was used as a surrogate. Additional data was gathered including patient demographics, method of diagnosis, histopathologic exam findings, number and location of metastases, performance status at diagnosis, treatments offered, and compliance with treatment. Compliance with recommended treatment was especially salient for this study population, which generally includes patients in low socioeconomic status. An inability to pay for recommended therapies represents an additional challenge to these patients, which may also impact their clinical outcomes. For the statistical analysis, a Mann-Whitney U test was utilized to determine the existence of any significant difference in survival time between the Pre- and Post-periods. This test was selected due to the variance of survival times within and between periods. For the same reason, survival time for each period was reported as a median value rather than a mean. Results: Median survival time was found to be 51 days in the Pre-period and 175 days in the Post-period. Despite the greatly improved survival in the latter period, the difference observed was not statistically significant (p = 0.27). Conclusions: This is a preliminary result in an ongoing study to evaluate the survival time of patients with brain metastasis in the MIHS. The most significant limitations of the study included the low sample sizes, as well as the need to estimate the date of death in cases where this information was not included in the medical record. Planned changes to the study include expanding the time periods to increase the sample sizes, as well as collaborating with Hospice of the Valley to obtain accurate dates of death.

CONNECTING THE DOTS: ISCHEMIC STROKE AS A COMPLICATION OF PNEUMOCYSTIS PNEUMONIA AND AIDS
Juxiang (Jennifer) Huang DO, Abstract 87

Cerebral vascular accident is not a common phenomenon in the younger population; however HIV associated stroke tends to affect the younger population. Typical risk factors such as hypertension, diabetes, hyperlipidemia or smoking are not commonly seen in younger stroke patients with HIV. The exact mechanism by which HIV and stroke are connected is unknown, however, the burden of concurrent opportunistic infection (such as pneumocystis jiroveii pneumonia) is suspected to be associated with increased risk. Risk factors for stroke in the young HIV population tends to be associated with infections, vasculitis, coagulopathy and HIV associated vasculopathy. A 48 year-old male with no significant PMH presented to the Emergency Department with complaints of progressive shortness of breath and worsening nonproductive cough for
approximately one month. He also reported subjective fevers, chills, and night sweats with approximately 10 lb weight loss over the month prior to presentation. He completed a course of oral antibiotics and steroids approximately 10 days prior to admission without resolution of symptoms. On further questioning, he revealed homosexual intercourse with 10 different partners over a 10 year period without the use of condoms. He also reported having thrush intermittently for the past year. Physical examination revealed a cachectic male with white plaques in the posterior pharynx and on the uvula. Bibasilar crackles were auscultated on exam and chest X-ray showed evidence of bilateral infiltrates. Laboratory studies revealed an elevated LDH, positive HIV antibody assay, and CD4 count of 25. Bronchoalveolar lavage was positive for Pneumocystis jiroveii. He was started on Bactrim and IV steroids for treatment of PCP pneumonia. On hospital day 8, he developed a large right MCA infarct. TPA was initiated; however, there was no clinical improvement. As a consequence he required endotracheal intubation to maintain airway. On hospital day 10, while intubated and in the face of difficult oxygenation, he developed bilateral pneumothoraces. He never regained consciousness after his stroke and after discussion with his family, care was withdrawn and he died on day 13 of hospitalization. This case illustrates the importance of history taking and appropriate HIV screening in patients with high-risk behaviors. Pneumonia in young adults without medical comorbidities should raise the suspicion for HIV immunosuppression and opportunistic infection as a differential diagnosis. Furthermore, timely identification of HIV will result in early intervention and prevention of opportunistic infections such as PCP pneumonia. Our case also demonstrates the occurrence of ischemic stroke in a young AIDS patient without the traditional risk factors for thromboembolic stroke such as hypertension, diabetes, hyperlipidemia or atrial fibrillation.

ACUTE HEPATITIS: AN UNCOMMON PRESENTATION OF EPSTEIN-BARR VIRUS INFECTION

Yunhee Im MD, Abstract 42

Intro: Epstein-Barr virus (EBV) is a herpes virus that is the causative agent of infectious mononucleosis through the infection of B Lymphocytes. More than 90% of the world’s population has serologic evidence of past infection, most of which is subclinical. Infectious mononucleosis is the classic presentation of EBV infection and is characterized by fever, oropharyngitis, and bilateral lymphadenitis in adolescents or adults. Acute hepatitis without signs or symptoms of infectious mononucleosis is a very uncommon presentation of EBV infection. Case: We present a case of a 47-year-old previously healthy Latino female who presented to emergency department with a urinary tract infection (UTI) with jaundice. The patient did not report any complaint other than UTI symptoms and denied any other symptoms. The patient’s physical exam was unremarkable except for jaundice. Vital signs were normal. Patient’s lab showed AST 1120 u/L (5-40u/L), ALT 689 u/L (7-56u/L), Alkaline Phosphatase 171 IU/L (20-140 IU/L), total bilirubin 19.1 mg/dL (0.3-1.9 mg/dL), direct bilirubin 14.6 mg/dL (0-0.3mg/dL), iron 217 mcg/dL (60-170 mcg/dL), transferrin 232 mg/dL (204-360 mg/dL), percent saturation 66.8% (15-55%), ferritin 2036 ng/mL (12-150 ng/mL in females). The patient’s differential diagnosis included hemochromatosis and viral hepatitis. Genetic
testing for hemochromatosis was negative. The patient’s Hepatitis panel was negative for Hepatitis A, B, C, and E. Serum Acetaminophen was negative. C3 and C4 levels were within normal limit and smooth muscle antibody was negative. Herpes simplex virus and Cytomegalovirus were also negative. Abdominal ultrasound showed an enlarged liver at 15.6 cm but showed no evidence of biliary obstruction. However, the patient’s CBC and blood smear showed lymphocytosis, target cells, and Howell Jolly bodies and the patient was tested for EBV infection. The patient remained asymptomatic and was sent home on meropenem for her UTI the next day. Subsequently the patient’s EBV PCR result came back positive. Patient lost to follow up.

Conclusion: In our case, an immunocompetent adult patient had isolated severe cholestatic hepatitis from EBV infection without symptoms of mononucleosis. EBV is a rare causative agent of acute hepatitis and isolated EBV cholestatic hepatitis is rarely reported. Treatment for EBV hepatitis is usually supportive as it is generally self-limiting. However, severity of EBV hepatitis varies and it can rarely result in hepatic failure. As in our case, patients with asymptomatic hepatitis with evidence of splenic involvement and a pattern of elevated alkaline phosphatase, EBV hepatitis needs to be considered.

SELECTING TARGETED THERAPIES FOR METASTATIC CLEAR CELL RENAL CELL CARCINOMA (MRCC)
Andrew Kovoor MD, Abstract 68

Introduction: Guidelines for the treatment of metastatic renal cell carcinoma (mRCC) have been rapidly evolving in the new era of new molecular targeted therapies. Systemic treatment was previously limited to cytokine therapy with interleukin-2 (IL-2) or interferon-α (IFN-α). The development of newer agents that target tumor growth pathways such as vascular endothelial growth factor (VEGF) and mammalian target of rapamycin (mTOR), have provided more effective alternatives. These targeted agents include: VEGF tyrosine kinase inhibitors (TKI), VEGF monoclonal Ab, and mTOR inhibitors. These therapies offer more effective treatment alternatives limiting the role of single agent IFN-α or IL-2 in the management of mRCC. Despite these advances in therapies, it remains unclear how to optimize these treatment modalities and what sequence of agents to administer. Case Report: We present a 68-year old woman with mRCC involving the lungs, liver, and spine who has been treated successfully for the past 5-years with sequential mTOR inhibitors and VEGF-directed therapies. The patient initially presented with pulmonary metastasis and was started on sunitinib (TKI) and maintained disease stability for 20 months. Therapy was transitioned to everolimus (mTOR inhibitor) after routine scans revealed an approximately 24% increase in tumor size. Eleven months later, the patient was placed on another TKI, pazopanib, after further disease progression was noted, however limited clinical benefit was demonstrated with this therapy. Single agent temsirolimus therapy was initiated and continued for 12 months until she developed liver and bone metastasis. The patient was most recently started on axitinib therapy and is doing well with stable disease. Discussion: The targeting of molecular pathways has expanded therapeutic modalities and improved Progression Free Survival (PFS) and Overall Survival (OS) in mRCC. Our patient’s prognosis at presentation was poor with a predicted OS of 1 year with immunotherapy. Fortunately, her tumor has responded well to targeted therapies with
sequential TKIs and mTOR inhibitors and she has maintained stable disease for greater than 5 years. Based on our review of the literature, we propose an algorithm of sequential and combination therapies for the effective management of mRCC, based on patient characteristics and performance status. Our review of the AVOREN and CALBG trials (with each including over a 600 patients) suggests that first-line combination therapy with bevacizumab plus IFN-α (PFS 8.5-10 months and OS 38.6 months) is superior to single agent TKIs (PFS 11 months and OS 26.4 months) or single agent mTOR inhibitors (PFS 3.8 months and OS 10.9 months). However, this combination therapy is associated with greater toxicities including grade 3/4 hypotension, fever, and decline in performance status. According to the TARGET and RECORD-1 trials, they suggest that single agent mTor inhibitor and TKI are effective second line therapies.

PARENTERAL NUTRITION: BEWARE OF WHAT PHYSICIANS DO NOT KNOW
Rashmi Kumar MBBS, Abstract 125

Introduction: Although total parenteral nutrition (TPN) improves survival of patients with short bowel syndrome; it is vital for physicians to be aware of its potential complications which are not limited to septicemia, hyperglycemia, venous thrombosis and liver disease. While TPN induced cholestasis has been extensively studied, this is the first case of parenteral nutrition associated pancreatitis being reported to the author’s knowledge. Case Description: A 21 year-old-female in persistent vegetative state secondary to intraventricular and intraparenchymal bleed and subsequent bowel necrosis with short bowel syndrome, who has maintained on TPN for 5 years presented with sepsis secondary to PICC line bacteremia. On physical exam she had severe jaundice and multiple spider angioma. Lab showed total bilirubin- 24.7 mg/dl, direct bilirubin- 16.9 mg/dl, AST-145 u/l, ALT- 119 u/l, Albumin- 1.6 g/dl; PT-22.1 sec, INR-2, PTT-45.1 sec, TG- 174 mg/dl, Cholesterol- 100 mg/dl, lipase- 179 u/l. Ultrasound abdomen showed enlarged liver without focal mass, splenomegaly, mild to moderate ascites suggestive of portal HTN. CT abdomen showed right hepatic lobe subcapsular collection, marked splenomegaly, no extrahepatic or intrahepatic biliary duct dilatation. Pancreas normal but poor visualization. Patient was started on peripheral parenteral nutrition (PPN) and over the course of 2 days her lipase jumped to 851 u/l from 179 u/l. After ruling out other causes of elevated lipase a diagnosis of chemical pancreatitis was made secondary to intralipid in PPN. Conclusion: In short bowel syndrome where major source of nutrition is parenteral, diagnosing complications early may reduce the morbidity and mortality in these patients. So, it is important for physicians to be aware of the association of pancreatitis before initiating intralipid treatment as a part of PPN in patients with compromised gastrointestinal reserve.

INTERNAL CAROTID DISSECTION IN A TYPE I DIABETIC PATIENT WITH SEPTIC ABORTION: THERAPEUTIC DILEMMA
Sotiris Mitropanopoulos MD, Abstract 5

Sotiris Mitropanopoulos MD (Associate), Kareem Ahmad MD (Associate), Richard W Carlson MD PhD, (Fellow) Depts. Medicine, Maricopa Integrated Health System, Phoenix AZ, University of Arizona and Mayo Clinic Colleges of Medicine, Phoenix, Scottsdale, AZ
Introduction: The pathophysiology of internal carotid artery dissection (ICAD) involves separation of the intimal or sub-adventitial layer of a vessel, allowing blood to enter the wall of the artery creating an obstructive hematoma. Although traumatic dissections tend to be more common in young patients, individuals with connective tissue disease, hypertension and tobacco use are more prone to spontaneous dissections. Unfortunately, management is frequently suboptimal with patients having chronic neurologic deficits. We describe a case of a type I diabetic woman that developed an ICAD after a septic abortion complicated by diabetic ketoacidosis. Case: A 36 year-old Hispanic woman with type I diabetes presented to the emergency department with fever, chills, abdominal pain, and vaginal bleeding. She was tachycardic and hypotensive with a blood glucose of 414 mg/dL, an arterial pH of 7.28 and a serum bicarbonate of 16 mEq/L. She was admitted for incomplete septic abortion. Dilation and curettage was performed and was subsequently admitted to the MICU for severe sepsis and diabetic ketoacidosis. While in the ICU, the patient was discovered to have Horner’s syndrome and right hemiplegia. CT demonstrated a left hemispheric ischemic stroke estimated to have occurred more than 12 hours earlier. CT angiogram of the neck revealed a left ICAD with a hematoma that occluded 100% of the lumen. Neurology and Neurosurgery consultants did not recommend surgical intervention because of the 100% occlusion. Thrombolysis was contraindicated due to the time of the initial stroke. She was placed on aspirin and simvastatin for anticoagulation. Patient continued to have ischemia with a subsequent MRI indicating a new ischemic lesion in the occipital region. After stabilization she was discharged with follow-up to neurology.

Discussion: There is no consensus on the effectiveness and safety of thrombolysis for ICAD. The prevailing theory is that thrombolysis would cause the hematoma to enlarge. However, a recent meta-analysis of 180 patients treated with thrombolysis concluded that there was no difference in safety or outcome from ICAD and all causes of stroke when using thrombolysis. Although this patient had contraindications that precluded her from thrombolysis, physicians should consider the option of thrombolysis in similar scenarios.

HEPATITIS B REACTIVATION AFTER 2 MONTHS OF RITUXIMAB-BASED CHEMOTHERAPY IN A PATIENT WITH NHL
Zohreh Movahedi Smith MD, Abstract 34

Introduction: Hepatitis B reactivation and progression to fatal hepatic failure has been increasingly observed in patients with non-Hodgkin’s lymphoma (NHL) who received rituximab-based chemotherapy. Early recognition and prompt antiviral treatment is crucial to prevent further progression of hepatitis B. Case report: We present a case of hepatitis B reactivation with rituximab in a 69-year-old with past medical history of hypertension and NHL in remission after chemotherapy who presented with painless jaundice, a coagulopathy, and markedly elevated transaminases and bilirubin. The patient was first diagnosed with low grade NHL in January 2012, based on abdominal mass biopsy which was positive for CD20. She received six cycles of R-CHOP chemotherapy over four months leading to a complete remission. Serologic testing for hepatitis B was not done prior to starting chemotherapy. There was no elevation of serum aminotransferase levels throughout the course of chemotherapy. However, an isolated elevation of ALT was
noted in May 2012. Two months later she presented with painless jaundice, nausea and vomiting. Serum aminotransferase levels were significantly elevated. Laboratory tests revealed ALT 2532 U/L, AST 2436 U/L, total bilirubin 14.3 mg/dl and INR 1.5. Serum was positive for total hepatitis B core Ab and hepatitis B surface Ag and HBe Ab, but HBe Ag was negative with high HBV viral load >170,000,000 copies/mL. Hepatitis A and C, HIV, CMV, EBV and autoimmune hepatitis studies were negative and the diagnosis of reactivation of hepatitis B was made and antiviral therapy with tenofovir 300 daily was started. Aminotransferase levels declined on tenofovir and no hepatic decompensation developed thereafter. HBV DNA one month after tenofovir therapy came down to 170,000 copies/mL. Discussion: Reactivation of hepatitis B refers to the abrupt increase in hepatitis B virus (HBV) replication in a patient with inactive or resolved hepatitis B. The importance of reactivation of hepatitis B rests on its potential severity of flare of disease leading to progressive liver injury. Available oral antiviral therapy against hepatitis B are relatively potent and are well tolerated, so HBV reactivation can be prevented by prophylactic use of antiviral agents, so high risk patients should be screened for evidence of hepatitis B before starting immune suppression or chemotherapy. One third of the world’s population has evidence of previous infection with the hepatitis B and 350 million people have chronic infection. Current AASLD (American Association for the Study of Liver Diseases) and APASL (Asian Pacific Association for the Study of the Liver) guidelines recommend routine HBsAg testing before the initiation of chemotherapy, and prophylactic antiviral therapy should be administered to hepatitis B carriers at the onset of chemotherapy and maintained for three to six months afterwards.

LEFT LEG PAIN: AN UNEXPECTED OUTCOME
Soyoung Park MD, Abstract 116

Streptococcal toxic shock syndrome (TSS) presenting with necrotizing fasciitis is a rare but fatal medical condition. Thus far, only nine cases have been documented – of which five were adult patients – in the English-language literature, which were all from the 1980s-90s. We describe a case in which a patient with a previous history of deep venous thromboses (DVT) and pulmonary embolism (PE) presented with leg pain that turned out to be TSS with necrotizing fasciitis. The patient was a 40-year-old woman who had bilateral leg pain on presentation accompanied by swelling progressing over the course of several months. The patient had a previous ultrasound about a week prior to that was positive for bilateral DVTs for which patient was started on therapeutic anticoagulation with warfarin. Despite anticoagulation, patient’s bilateral leg pain became progressively worse, limiting her ability to work, and on exam, patient was extremely sensitive touch. Suspecting necrotizing fasciitis, patient was taken to surgery for debridement where her left leg showed necrotic skin and questionable muscle necrosis with serous fluid in all areas. Three days after initial presentation, patient’s blood cultures demonstrated growth of Streptococcus mitis. By then, patient had already been started on IV vancomycin, meropenem, clindamycin, and ciprofloxacin. Patient ultimately underwent severe sepsis with multiple organ failure and did not survive. Despite how fatal it is, the presenting symptoms of TSS are difficult to distinguish – fevers, chills, myalgias, diarrhea – and can easily lead physicians down a path of
dismissing it as a more commonly seen condition. Those with necrotizing fasciitis will tend to initially present with limb pain along with other physical exam findings that can cause physicians to misdiagnose patients with food poisoning, influenza, muscle strain, or DVT. In patients without a defined portal of entry, proliferation occurs deep within the strained muscle. Even if physicians have a low suspicion for a rapidly developing necrotizing fasciitis due to streptococcal TSS, it is extremely crucial to have a low threshold for recognizing and treating this infection as most patients don’t survive it because of severe sepsis and multiple organ failure.

OCCAM’S RAZOR OR HICKAM’S DICTUM IN THE ERA OF IMAGING.

Krunal Patel MBBS, Abstract 110

Introduction: Despite dramatic improvements in technology, the importance of a thorough physical exam and an astute clinical acumen cannot be underestimated. The following case elicits the importance of coming up with the most logical line of thought and adhering to it despite negative test results. Case report: A 68-year-old female presented with an acute onset of vertigo with numbness and pain on the left side of her face, left facial weakness and difficulty in speaking of a few hours duration. Physical exam was significant for tachycardia, dysconjugate lateral gaze, lower motor neuron type left facial weakness involving the entire left face and reduced sensation to light touch over left facial area. A computed tomographic scan of the head ruled out hemorrhage and a magnetic resonance imaging/angiography was requested which showed no acute stroke. As the above testing was negative an initial diagnosis of benign paroxysmal positional vertigo (BPPV) and Bell’s palsy were made in the emergency room and the patient was treated with meclizine with recommendations to follow up as an outpatient with neurology. Because of unsteadiness, however, the patient was admitted for observation. Since all of the patient’s symptoms could not be explained on the basis of the above diagnoses and also since a combination of two unrelated disease processes starting at the exact same time is highly unlikely, we sought to repeat the magnetic resonance imaging as the most logical explanation would be a brain stem stroke. Repeat magnetic resonance imaging done on the next day of hospitalization showed a left posterior brainstem infarct. She was then started on clopidogrel as she was allergic to aspirin. Further testing for evaluating the possible etiology of stroke was undertaken. Discussion: Of the symptoms this patient had, the vertigo could be explained by BPPV and the left sided facial weakness could be explained by Bell’s palsy. However, even if she had the misfortune of having two separate disorders, they could not explain her other symptoms. Her facial numbness, dysconjugate and left facial weakness all point to cranial nerve involvement. Based on the above, a clinical diagnosis of acute stroke can be made and the lesion can be localized to pons as the involved cranial nerves are V, VI and VII. Although DWI MRI can detect majority of ischemic strokes in acute stage, the sensitivity is still 80-90% and it should be noted that 10-20% of strokes might not be reported on initial MRI. This case report emphasizes the importance of clinical examination in the era of diagnostic test driven medicine, as early diagnosis of ischemic strokes in acute stage will facilitate definitive treatment and better outcomes.
A MEGA ESOPHAGUS – LOST TO FOLLOW-UP!
Ioan Puscas DO, Abstract 35

A Mega Esophagus – Lost to Follow-Up! Ioan Mircea Puscas DO (Associate), Mariya Balon MD (Associate), Pedro Quiroga MD Maricopa Integrated Health System, Phoenix, Arizona

Introduction: Achalasia, first recognized more than 300 years ago, is characterized by failure of relaxation of the lower esophageal sphincter. The physiologic alterations in achalasia result from neuronal loss/dysfunction leading to a spectrum of symptoms that develop over time. We describe a case of end-stage achalasia in a young patient who had learned compensatory techniques over the years to aid with swallowing of food such as bending his neck and extending his back as well as drinking plenty of water with every meal. Case Report: The patient is a 33 year old African American male who originally had signs of esophageal achalasia on a chest x-ray in 2002 after having surgery for an eye injury. The patient remained asymptomatic until 2010 when he started developing symptoms of GERD and regurgitation and was evaluated at another facility. At that time the patient had an EGD which showed esophageal achalasia and he was scheduled to undergo surgery. However, the patient was lost to follow-up secondary to financial reasons and lack of healthcare resources. The patient presented to our facility from prison for infrequent hemoptysis, a 150 pound weight loss and chronic nocturnal cough. As a part of the work-up to rule out active tuberculosis, a chest x-ray was done that showed diffuse large right lower lobe consolidation. The x-ray finding turned out to be a massively distended esophagus occupying the right chest cavity with resultant tracheal compression and narrowing. Subsequent EGD showed extreme achalasia with copious amount of food which was removed in multiple EGD sessions. The patient was evaluated for possible esophagectomy with colonic interposition. He finally underwent laparoscopic Heller myotomy and jejunostomy tube placement while awaiting shrinkage of esophagus prior to esophagectomy.

Discussion: Esophageal achalasia is caused by failure of relaxation of the lower esophageal sphincter (LES) with swallowing, along with loss of peristalsis in the lower esophagus. The symptoms of achalasia can be subtle early in the course becoming more obvious with progression. The initial symptoms include dysphagia for solids and liquids, heartburn and chest pain leading to regurgitation and weight loss once esophageal dilation develops. The diagnostic modalities include functional studies (fluoroscopy and manometry) and EGD. Unfortunately, no therapy reverses the aperistalsis and treatment modalities focus on relieving the LES pressure. The above patient has had clinical signs suggestive of disease for more than a decade but unfortunately ended up developing a sigmoid esophagus secondary to lack of access to medical care.

ADDISON’S DISEASE, AN INSIDIOUS DISEASE THAT CAN CHALLENGE CLINICIANS
Sirisha Rao MD, Abstract 26

Intro : Adrenocortical deficiency also known as Addison’s disease (AD), is a rare condition that affects approximately 40-60 people per one million. However, failure to recognize and treat the condition can result in significant morbidity and mortality. The symptoms of AD can be vague and difficult to recognize. They can include nonspecific symptoms such as fatigue, lightheadedness, weakness, skin hyper or hypo pigmentation, and abdominal discomfort.
Case: We present a 47-year old female with a past medical history of gastroesophageal reflux disease (GERD), gallstones and dyslipidemia who presented to the hospital with a history of epigastric pain, lightheadedness, and extreme fatigue of one month duration. The pain was described as sharp, intermittent, and associated with shortness of breath. She denied any weight loss, melena or hematochezia. Due to the patient’s chronic history of GERD and increasing abdominal discomfort, the patient was scheduled for an esophagogastroduodenoscopy (EGD). On arrival to the endoscopy suite, the patient was extremely lethargic, bradycardic with a heart rate of 36 bpm (nl: 60-100 bpm). A 12 lead electrocardiogram (ECG) was done and showed sinus bradycardia with a prolonged QT interval of 452 ms (nl: <450 ms). Due to the extreme nature of her symptoms of fatigue, lightheadedness, bradycardia and subtle accentuation of normal skin pigmentation in non-sun exposed areas (in contrast to typical hyperpigmentation in palmar creases and sun-exposed surfaces), a concern for adrenal insufficiency was raised and the EGD was cancelled by the GI team. Subsequently, the next morning, two ACTH stimulation tests were done and the patient was found to have a baseline cortisol level at time 0 = 3.5 µg/dl (nl: 20-30 µg/dl), at 30 minutes 9.3 µg/dl, and at 60 minutes 13.3 µg/dl (nl: 40-60 µg/dl), consistent with primary adrenal insufficiency. She was then treated with Hydrocortisone 50 mg PO each morning for three days and a plan to continue with Hydrocortisone 5 mg PO each day at 2pm after discharge. This regimen resulted in full resolution of her symptoms.

Conclusion: This case illustrates the insidious constellation of symptoms that can be produced by Addison’s disease, and the need to have a high index of suspicion in diagnosing this serious and illusive condition. A patient who presents with bradycardia, fatigue, abdominal symptoms, or shortness of breath can easily be misdiagnosed. Had the patient undergone the EGD with monitored anesthesia care (MAC) sedation, an adrenal crisis could have been precipitated with grave consequences including death. Clinicians are urged to consider this diagnosis in any patient exhibiting autonomic dysfunction, extreme fatigue, subtle but typical skin findings or screening lab studies suggestive of Addison’s disease.

Carcinoid, Cushing’s, and Confusion
Karen Sapienza MD, Abstract 21

Introduction: Carcinoids are rare neuroendocrine tumors with an incidence in the United States of 1 to 4 cases per 100,000 population. These tumors may produce various hormones, with the most common being serotonin and substance P. Less than 5% secrete other hormones such as gastrin, glucagon, calcitonin or adrenocorticotropic hormone (ACTH). Case Presentation: A 52-year old male with known metastatic carcinoid tumors to liver and nonfunctioning pituitary microadenoma presented to his oncologist with fatigue, weakness and diarrhea. Routine labs revealed a critical serum potassium level of 1.5 mmol/L that led to his hospitalization. Physical exam was notable for a blood pressure of 179/104, pulse of 96, and no obvious Cushingoid features such as atrophic thin skin, bruising or striae. EKG demonstrated a prolonged QTc of 725 ms. New-onset diabetes was noted with a serum glucose of 328 mg/dL and HgbA1c of 6.5%. Random cortisol was 108.6 mcg/dL, A.M. cortisol was 105.9 mcg/dL (reference: 7.0 – 25.0 mcg/dL), ACTH was 1126 pg/mL (reference: 10-60 pg/mL), and 24-hour urine cortisol was 10,056 mcg/24h. Gastrin was...
4,136 pg/mL, chromogranin A was elevated at 3,450 ng/mL (reference &8804; 225) and 5-HIAA was slightly elevated at 8.3 mg/24h (reference &8804; 8.0). CT abdomen and pelvis demonstrated a slight increase in size of hepatic metastases, stable pancreatic lesion, and unchanged adrenal thickening. CT chest showed no new or progressive lung nodules or masses with unchanged appearance of the thyroid. The ACTH-dependent Cushing’s syndrome in this patient was attributed to ectopic production of ACTH from his known carcinoid tumors, as his pituitary adenoma was non-functional. Emergent management of severe hypokalemia required both IV and PO potassium. Initial treatment of hyperglycemia was challenging, as insulin would lead to intracellular shift of serum potassium and further exacerbate his hypokalemia. For long term management of his ectopic ACTH production, bilateral adrenalectomy was undertaken on hospital day 7. He responded well to post-operative steroid administration. Prior to discharge, his EKG showed normalization of QTc interval, his glucose was better controlled, and a home insulin and steroid regimen including fludrocortisone and hydrocortisone were initiated. Discussion: While carcinoid tumors themselves are rare, only a small fraction demonstrates ectopic ACTH production. Although cases of Cushing’s syndrome due to ectopic ACTH from carcinoid tumors have been described, it remains a rare occurrence in this uncommon tumor. In this case, the result was an ill patient with profound metabolic disturbances. The acute onset of ectopic ACTH production and resulting clinical manifestations are exemplified by the notable absence of the usual Cushingoid features.

AN UNUSUAL PRESENTATION OF LEUKOCYTOSIS AND HYPERCALCEMIA IN UROTHELIAL CELL CARCINOMA
Natasha Sharda MD, Abstract 66

INTRODUCTION: Hypercalcemia and leukocytosis are always concerning developments in any patient. Leukocytosis is classically a marker of infection while high calcium levels can be the first clue to diagnosing cancer. Although hypercalcemia is a common finding in cancer, it is not generally seen in the absence of bony metastasis or paraneoplastic syndromes. Here we discuss an unusual case of leukomoid reaction and hypercalcemia in a patient with urothelial cell carcinoma. CASE PRESENTATION: A 68 year old male presented to the emergency department with a 3 day history of nonbloody diarrhea. The patient’s past medical history was significant for diabetes, coronary artery disease, heart disease on anticoagulation, and urothelial cell carcinoma status post radical cystoprostatectomy with an ileal conduit formation 5-6 months prior to admission. The tumor was staged at T4 N2. The patient did not receive any adjuvant chemotherapy or radiation. Review of systems revealed generalized weakness and fatigue but no fevers or chills. On physical exam, the patient was afebrile with relatively dry mucosal membranes and a normal abdominal exam. Initial labs revealed a white blood cell count of 62.6/1000uL with 85% neutrophils and a metabolic panel significant for hypercalcemia at 13.8mg/dL, albumin of 2.6g/dL, INR of 1.2, and creatinine of 2.1mg/dL. A CT of the abdomen on 07/27/12 showed an 8cm x 8cm mass consistent with a hematoma in the right lower abdomen. Given his leukocytosis and loose stools, broad spectrum antibiotics were initiated. An infectious work up ensued, and all
cultures including blood, urine and clostridium difficile PCR were negative. Consequently, antibiotic therapy was discontinued without any deterioration in status. Evaluation for hematologic malignancy was also investigated, with negative flow cytometry, undetectable urine M protein, JAK2 mutations and BCR-ABL mRNA. Nuclear bone scan showed no osseous metastatic disease. As the extreme leukocytosis persisted, a repeat abdominal and pelvis CT was done (08/06/12) and showed an increase in the previously seen abdominal mass, which was no longer suspected to be a hematoma. A CT guided biopsy confirmed high grade urothelial cell carcinoma. Further lab work revealed appropriately suppressed PTH at 10pg/mL and PTHrP elevated at 4.3pmol/L. It was concluded that this patient had leukocytosis-hypercalcemia paraneoplastic syndrome from his urothelial cell carcinoma. Due to the extensive nature of his disease, the patient chose palliative treatment with only radiation. CONCLUSION: Leukocytosis and hypercalcemia are paraneoplastic syndromes which have rarely been associated with high grade urothelial tumors. There have been 5 documented cases of this association reported in the literature. The mechanism of the hypercalcemia results from tumor secretion of PTHrP and the leukocytosis stems from tumor production of GM-CSF. Thus tumor reoccurrence should be suspected in patients with past medical history of urothelial cell carcinoma presenting with idiopathic leukocytosis and hypercalcemia.

ATYPICAL CASE OF NECROTIZING ENTEROCOLITIS WITH PRIMARY SMALL BOWEL INVOLVEMENT AND ABDOMINAL COMPARTMENT SYNDROME TREATED USING CONSERVATIVE MANAGEMENT

Samir Sultan DO, Abstract 33

Introduction: Necrotizing enterocolitis is a condition diagnosed in neutropenic patients with fever, abdominal pain, and bowel wall thickening of greater than 4 mm. Although the pathogenesis of this condition is not well understood, it has been suggested that inflammation of the bowel wall by chemotherapeutics is followed by bacterial invasion into the damaged mucosa with rapid proliferation. The classic triad of necrotizing enterocolitis is described as fever, abdominal pain and diarrhea. This condition has also been documented to lead to bacteremia with gram negative organisms. Case Presentation: This is a 64 yo male with history of stage IA esophageal adenocarcinoma treated with chemotherapy (docetaxel) who presented initially with neutropenic fever, and later developed the symptoms of abdominal pain, diarrhea and abdominal distension. Abdominal compartment syndrome was diagnosed with a bladder pressure of 19 mm Hg at this time. Bacteremia with a gram negative organism was an additional complication of Necrotizing enterocolitis encountered during this admission. The diagnosis was further confirmed using CT abdomen with oral and IV contrast; which showed pronounced cecal wall thickening with predominant small bowel wall thickening. The patient was treated successfully using conservative management as he was deemed a poor surgical candidate. He was given bowel rest by ordering NPO and low maintenance IVF to decrease any progression of the abdominal distension, which was followed using serial bladder pressures. A paracentesis was also performed with 2L of fluid drained to further alleviate abdominal pressure. Tobramycin and Cefepime were used for treatment of bacteremia and neutropenic fever. His fever, bacteremia, abdominal pain
and distension, and diarrhea gradually resolved. Discussion: Necrotizing enterocolitis is rare condition that has been associated with chemotherapeutics in some patients. A Medline search revealed zero cases of necrotizing enterocolitis with primary small intestine involvement. In this atypical presentation of necrotizing enterocolitis we were able to demonstrate successful treatment using conservative therapy.

RECURRENT CHRONIC PANCREATITIS:
PANCREATIC DIVISUM
Kevin Tozer MD, Abstract 43

Intro: The most common risk factor for chronic pancreatitis is alcohol use, accounting for between 55-80% of all cases. Pancreatic divisum is an embryological variant that results in the duct of Santorini, normally a minor duct obliterated in adulthood, becoming the major duct of the pancreas and the only route of communication to the duodenum. Case: We present a 40 y.o. female who presents to the emergency room with a five-day history of severe mid-abdominal pain, intractable nausea, and diarrhea. She describes the pain as constant, most severe over the mid-abdomen, with occasional radiation to her back. The diarrhea was “oily and always floating in the toilet” but she denies any melena, hematochezia, or coffee-ground emesis. She says the symptoms were originally associated with food intake but are now constant and not associated with any alleviating factors. She endorses a 20lb weight loss over the last month. She has had six similar presentations each lasting between 4-10 days and with varying levels of severity. All previous hospital admissions and emergency room visits results were inconclusive. She denies any drug use, alcohol intake, or sick contacts. Her history is significant for asthma and chronic leg pain secondary to a gun-shot wound several years ago. Her medications are Albuterol, Gabapentin, Cyclobenzaprine, and extended release morphine. The review of symptoms is non-contributory. At presentation, her vital signs were normal and initial labs were significant for an elevated amylase 445 U/L (25-125) and lipase 1581 U/L (18-180). The patient’s CBC and BMP were within normal limits. The patient had a normal lipid panel and normal LFTs. The RUQ U/S revealed no stones or biliary duct dilation. Immunologic studies were ordered, including an ANA, dsDNA, anti-Sm, anti-SSA, anti-SSB, SCL-70, and anti JO-1 all of which were negative. IgG, IgA, and IgM levels were within normal limits. A stool culture was negative. Secondary to not having a cause for her recurrent pancreatitis, an MRCP was performed which revealed the main pancreatic duct draining through the Santorini duct was indicative of pancreatic divisum. Conclusion: Although pancreatic divisum is present in 2-3% of the population, it is found in 5-28% of patients with otherwise idiopathic pancreatitis. Invasive procedures like MRCP and imaging are usually not warranted in mild pancreatitis, but they may be helpful in determining less common causes of recurrent chronic pancreatitis presentations as in our patient. Recognizing pancreatic divisum as a potential cause of chronic pancreatitis in a previously undiagnosed patient with recurrent admissions is imperative because it allows us to develop appropriate treatment plans including surgery. Early diagnosis would have decreased our patient’s pain, morbidity, and would help prevent more recurrent emergency visits and long term complications.
TUMOR OR THROMBUS? AN ELUSIVE DIAGNOSIS FOR AN ATRIAL MASS.
Maja Udovcic MD, Abstract 8

A 45-year-old man with a history of premature ventricular contractions (PVCs) and tachycardia-induced cardiomyopathy status post ablation presented to our hospital after a left atrial mass was found on a routine outpatient echocardiogram. The patient had an electrophysiology study and ablation in May 2011 for symptomatic PVCs. As part of pre-operative evaluation, a transthoracic echocardiogram (TEE) and a cardiac Magnetic Resonance Imaging study (MRI) demonstrated focal dyskinetic bulging of the right ventricular free wall. This was absent on imaging subsequent to the ablation. The procedure resulted in symptomatic improvement and he was scheduled for follow-up in one year with repeat ECG, Holter monitor and TTE. The patient remained well during the year post-ablation and returned for a TTE, which was normal. Another TTE was performed at 18 months post-operatively which revealed an unexpected finding: a mass attached to left atrial septum measuring 2 x 2.2 cm. The appearance of the mass was consistent with a myxoma, as it was a mobile, round density with irregular edges attached to the atrial septum on the left atrial side. However, development of this mass in the 14-month interim between imaging was most suggestive of a thrombus. In contrast, a cardiac MRI showed a mass lesion most suggestive of adherent thrombus as the lesion did not enhance with contrast material. As different imaging modalities were not conclusive in determining whether the mass was a myxoma or thrombus, the patient was empirically anticoagulated with heparin. After several days of heparin therapy, the size of the mass remained unchanged. With an inconclusive diagnosis, a transseptal excision was performed, with closures of an atrial septal defect and patent foramen ovale. The gross appearance of the mass was compatible with a myxoma, which was confirmed peri-operatively by frozen section with the presence of myxoid cells, well developed vessels, stromal hemorrhage, and hemosiderin laden macrophages. The patient tolerated the surgery without complications and continues to do well. This case is remarkable for several reasons. First, standard imaging modalities (TTE and cardiac MRI) were unhelpful in identifying whether the mass was an organized thrombus or myxoma. While the TTE suggested the mass was a myxoma, the characteristics on cardiac MRI were consistent with an adherent thrombus. As the treatment for a thrombus differs from treatment for a myxoma, it is of great importance that the diagnosis is certain before a patient undergoes cardiac surgery or a trial course of anticoagulation. There are several case reports that revealed the limitations of standard imaging in accurately discriminating an atrial thrombus from a myxoma (Kale, Jang, Kodali). It would be interesting to study the predictive value of each modality of imaging in correctly diagnosing a mass after the etiology has been concluded.

A PAIN IN THE NECK
Elizabeth Ulliman MD, Abstract 112

A Pain in the Neck Liz Ulliman, MD University of Arizona College of Medicine at South Campus Introduction Dystonic Storm is a medical emergency consists of acute onset of persistent dystonic contractions that can be life threatening. This disease process is rare, but its severity requires quick clinical recognition and treatment. Case Presentation: A 37 male with past history of Hepatitis C, depressive disorder,
schizoaffective disorder, polysubstance abuse and cervical dystonia presented with worsening of his dystonia over 7 days. He complained of repetitive cervical muscle contraction with anterior movement of the head. Each contraction lasted for a few seconds and was associated with sharp 10/10 neck pain, radiating to the occipital region and upper chest. The pain was so severe that the patient experienced difficulty swallowing, and sleeping. Insomnia combined with unrelenting pain, caused the patient to have suicidal ideation. On exam he was in considerable discomfort with tachycardia, tachypnea and fever of 101.5 F. The patient had uncontrollable repetitive anterocollis. He had tenderness of the anterior cervical neck muscles, but no palpable masses. The patient had a depressed mood and suicidal thoughts and ideation. He was admitted and treated with diazepam, hydromorphone, and trihexyphenidyl for dystonia. With treatment, the anterocollis decreased slightly in severity and his vital signs normalized. On the third day, however, the patient developed new tachypnea, fever and tachycardia with acute hoarseness and cough productive of yellow phlegm. He had bilateral inspiratory crackles and chest x ray showed consolidation of the right upper and middle lobe, and left upper lobe consistent with pneumonia. His CPK was elevated at 3579 and a diagnosis of dystonic storm with rhabdomyolysis and aspiration pneumonia was made. The patient was treated with intravenous normal saline, antibiotics and increased diazepam. Lorazepam was added. His CK was monitored. The storm gradually improved and his CK trended down. On day 8 he was at baseline level of dystonia and pain. He was discharged and referred to botox clinic. Discussion Dystonic storm (Status Dystonicus) is a rare potentially fatal complication of dystonia. Etiologies vary, but include response to stress situation in patients with preexisting dystonia, medication withdrawal, DYT1 dystonia, and baclofen pump failure. It’s characterized by acute worsening of symptoms with generalized and severe muscle contractions that can involve the vocal cords or laryngeal muscles, leading to airway obstruction. Patients may develop rhabdomyolysis and renal failure. Patients should be managed in an ICU and treated with one or a combination of anti-cholinergic’s, diphendydramine, baclofen, benzodiazepines, or dopamine blockers. Anesthesia with muscle paralysis may be required to control the spasms. Conclusion: With early recognition and appropriate aggressive management, adverse events associated with dystonic storm such as, rhabdomyolysis, renal failure and respiratory failure can be avoided.

THE ADDED VALUE OF POSITRON EMISSION MAMMOGRAPHY IN THE EVALUATION OF BREAST CANCER
Dane Van Tassel MD, Abstract 61

Background: Although breast MRI is currently considered the standard of care in breast cancer lesion detection1, there are limitations in regards to its sensitivity and specificity. Reported sensitivity of MRI is approximately 85-95%, while specificity is much lower, at approximately 65-75%2-4. Call-back rates for additional imaging range from 8% to 17% in MRI screening studies, and biopsy rates range from 3% to 15%5-10. Although special MRI sequences can assist in reducing the false positive rates, it is often difficult to identify a breast cancer11. These limitations may lead to unnecessary breast biopsies in cases where MRI is unclear. Positron emission mammography (PEM) is a high-resolution imaging technique performed with a dedicated breast PET device.
Rather than relying on morphology or anatomic appearance, PEM relies on a lesion’s metabolism to make a malignant diagnosis\textsuperscript{12}. As such, PEM may provide unique information about the biology and metabolism of a lesion that is not yet available through other imaging techniques. Methods: A retrospective analysis of the imaging records of 75 subjects who underwent both PEM and MRI evaluation for known or suspected malignancy was evaluated. The presence of malignancy was confirmed by biopsy or surgery. Statistical analysis was performed and sensitivity, specificity and the added value of combined modality detection rates compared to individual analysis was determined. Results: A total of 73 subjects were eligible for review and 142 lesions were identified. A total of 124 lesions (88\%) had known pathologic correlation. 78 demonstrated confirmed carcinoma while 46 were characterized as non-malignant. The sensitivity of MRI and PEM individually was found to be 97\% (95\%CI.938-1.010) and 92\% (95\%CI.862-.982) respectively (p>0.05), while the specificity of MRI and PEM were 33\% (95\%CI.200-.467) and 58\% (95\%CI.444-.723) respectively (p>0.05). The specificity for the combined exams was calculated to be 73\% (95\%CI.603-.855) which was statistically significant over MRI alone (P<0.05). Additionally, using statistical analysis to determine the added value of PEM combined with MRI revealed that using PEM yields a positive likelihood ratio that is significantly better than using MRI or PEM alone(p<0.05). Conclusions: The results of this study indicate that PEM provides complementary information to MRI in the evaluation of breast cancer, and when combined with MRI, can improve the performance of breast cancer detection. The increased specificity of PEM allows for more confident diagnosis of malignant carcinoma when compared to MRI. Used in combination with MRI, in the presence of a malignant lesion, a positive PEM result has a greater likelihood of being confirmed malignant on subsequent pathology than MRI or PEM alone. This suggests that PEM with MRI might help reduce unnecessary breast biopsies as patients with confirmed PEM and MRI positive lesions are extremely likely to be malignant at the time of surgery.

Second Place – PGY-1 Submissions:

AN INTERESTING CASE OF HICCUPS AND THE BLUES
Qi Yu DO, Abstract 46

Introduction: The Blue Rubber Bleb Nevus Syndrome (BRBNS), also known as “bean syndrome,” is a rare condition of systemic venous malformation. It can involve the skin, bone, and any visceral organs. Affected individuals have cutaneous violaceous to dark blue colored flat or raised lesions. When present in the gastrointestinal tract, these lesions are friable and can spontaneously rupture causing chronic blood loss leading to iron deficiency anemia or acute hemorrhage leading to death. Other gastrointestinal related complications include intussusceptions, volvulus, and bowel infarction. Case Presentation: A 47 year-old male with known history of BRBNS presented with intractable hiccups associated with nausea and vomiting for past 6 days. Significant history includes recurrent GI bleeds, iron-deficiency anemia, and partial colectomy for colon perforation during colonoscopy. Physical exam revealed multiple scattered small flat blue nevi on his face, oral mucosa, chest, extremities, and soles. His initial Hgb was 12.5 and remained stable. A CT chest demonstrated a long segment of...
nodular circumferential esophageal thickening, which was thought to be irritating the phrenic nerve and causing his hiccups. MRI of abdomen revealed benign hemangiomas and biliary ductal hamartomas. GI and CT surgery was consulted and a subsequent EGD and EUS depicted hypertrophied thickened vascular esophageal mucous with vascular blebs visualized throughout the duodenum. Fine needle aspiration of the area of esophageal thickening was inconclusive for malignancy. The patient was treated symptomatically with chlorpromazine, baclofen, and reglan during his hospital stay. Chlorpromazine was subsequently held secondary to pancytopenia, however his hiccups eventually resolved on reglan and baclofen and he was discharged without other interventions. Conclusion: BRBNS, first described by William Bean in 1958, is a venous malformation that can lead to fatal gastrointestinal bleeding if not recognized. Approximately only 200 cases are reported in the literature making the recognition of this condition that much more significant. Due to the lack of standard of care for patients with this condition, periodic surveillance studies needs to be instituted.

HEPATOSPLENIC T CELL LYMPHOMA: A RARE CAUSE OF NEUTROPENIC FEVER
Ming Zhang MD, Abstract 54

Introduction Hepatosplenic T cell lymphoma is a rare and aggressive peripheral T cell lymphoma characterized by T cells that infiltrate the sinusoids of the spleen, liver, and bone marrow. It is difficult to diagnosis due to the infiltrative manner of its growth and high false negative biopsy rate. Case A 55-year-old African-American female with an unremarkable past history presented with fever, chills, night sweats, nausea, and vomiting of two days duration. She had noted chronic right upper quadrant abdominal pain, malaise, fatigue, and weight loss for the previous six months. Previous examination had yielded neutropenia, hepatosplenomegalgy, and weight loss with no specific diagnosis found despite an extensive work-up including bone marrow and liver biopsies. On admission, she was febrile with hepatosplenomegalgy and mild right upper quadrant abdominal tenderness. Laboratory studies demonstrated pancytopenia and severe neutropenia. The patient was started on vancomycin and cefepime for neutropenic fever with no improvement. Infectious workup was unremarkable including blood cultures. Imaging revealed a 1.6 cm right hepatic lobe lesion that could not be biopsied. A peripheral smear was benign. Repeat bone marrow biopsy was performed and the marrow sinusoids were infiltrated by atypical lymphoreticular cells with an abnormally high nuclear cytoplasmic ratio. Immunoperoxidase stains of these cells were positive for leukocyte common antigen (LCA) and CD45RO (T cell), negative for CD20 (B cell). Flow cytometry analysis confirmed an abnormal population of double negative (CD4-/CD8-) Gamma/Delta T cells, consistent with hepatosplenic T cell lymphoma. The patient was promptly begun on therapy with cyclophosphamide, doxorubicin, vincristine, and prednisone. Discussion Hepatosplenic T cell lymphoma is a rare mature T cell lymphoma, accounting for less than 1 percent of the non-Hodgkin lymphomas. Median age at onset is 35, and it is associated with chronic immunosuppression. Patients present with non-specific symptoms, such as fever, malaise and weight loss. Evaluation demonstrates hepatosplenomagalgy, without lymphadenopathy, and pancytopenia. Although T cells can be identified in the spleen, liver, and bone marrow, these cells diffusely infiltrate in
the organ sinusoids. Diagnosis can be challenging, even with a biopsy. In this case, she had previously undergone unrevealing bone marrow and liver biopsies. The prognosis of this disease is very poor, with a median overall survival around one year. The current therapeutic strategies include CHOP (cyclophosphamide, doxorubicin, vincristine, and prednisone), splenectomy, and allogeneic bone marrow or peripheral blood stem cell transplantation. As this disease is rare and its pathogenesis is still unclear, more insight as to the biology and pathology of this disease may provide a more targeted and successful therapeutic modality. Conclusion Hepatosplenic T cell lymphoma is a rare and aggressive disorder that is challenging to diagnose and carries a very poor prognosis.

MULTILOBAR GROUND GLASS OPACITIES FROM INVASIVE ADENOCARCINOMA
Kareem Ahmad MD, Abstract 53

Introduction: We present a case of rapidly progressing pulmonary adenocarcinoma that masqueraded as a multi-lobar infectious or inflammatory process, resistant to antibiotic or steroid therapy. Case: The patient is a 59-year-old homeless Caucasian male with extensive past medical history of intravenous drug abuse, hepatitis C, chronic obstructive pulmonary disease secondary to tobacco abuse, disseminated Histoplasmosis as a child, and latent tuberculosis which was previously treated. The patient was admitted after outpatient failure of oral antibiotics (course of trimethoprim-sulfamethoxazole and azithromycin) for suspected pneumonia. A trial of steroids also failed to relieve patient’s complaints. The patient noted a nearly two month history of cough productive of yellow sputum and worsening shortness of breath. He admitted to subjective fever, chills, night sweats, weight loss of 30 pounds during this period, and pleuritic chest pain. Chest imaging found bilateral consolidations indicating acute process versus rapid progression of underlying chronic interstitial disease, but no focal nodules. Chest CT showed lesions consistent with acute multi-lobar ground glass opacities with bilateral pleural effusions. CT also demonstrated calcified mediastinal lymph nodes and pleural thickening which were unchanged from examination done three years prior. Bronchoscopy was performed for further diagnosis as sputum and blood testing failed to find an etiology. Bronchoalveolar lavage found possible actinomycosis, but no fungal infection, acid fast bacilli, or malignancy were seen. A CT-guided biopsy was recommended but the patient initially refused. He was discharged on oxygen to a local nursing home with doxycycline and steroid therapy. He returned a few days later with hypoxic respiratory failure. Again biopsy was recommended either via bronchoscopy or CT-guided. Due to the possibility of requiring intubation if he was to acutely decompensate, the patient refused and requested hospice care to where he was transferred. He again returned later seeking a diagnosis. Repeat CT scan found worsening of ground- glass opacities bilaterally involving original right middle and left lower lobes. CT-guided biopsy was performed and the tissue diagnosis of invasive pulmonary adenocarcinoma was made. Given poor prognosis, the patient was placed on comfort care and passed away 48 hours after biopsy. Conclusion: Following the invention of low-tar filtered cigarettes in the 1960’s, adenocarcinoma of the lung is now the most
common form of lung cancer. Challenges in diagnosis still remain due to its ability to present is multiple different patterns radiographically and histologically. Though rare as it may be, our case presents the importance of considering adenocarcinoma in the differential of a diffuse parenchymal process especially if it does not respond to empiric antibiotics or steroids.

**HYPOCALCEMIA CAUSED BY DEFECTIVE 1,25 VITAMIN D: A NEW CLINICAL ENTITY.**

**Aswani Kumar Alavala MBBS, Abstract 27**

Hypocalcemia caused by defective 1,25 Vitamin D: A new clinical entity. Aswani Alavala, MBBS, Harold M. Szerlip, MD and Hussein Yassine, MD, University of Arizona South Campus.

Introduction: Calcium deficiency can occur due to multitude of causes including hypoparathyroidism, vitamin D deficiency and rarely vitamin D resistance. We describe a case of symptomatic hypocalcemia which appeared unresponsive to endogenous 1,25-OH vitamin D but responsive to exogenous administration of small doses of calcitriol. Case report: A 44 year old Asian woman with short stature (height: 4’10”) and slightly bowed legs presented with positional vertigo and spontaneous muscle spasms. Labs reveal: serum calcium 6.5mg/dl, phosphorous 4.8mg/dl, parathyroid hormone (PTH) level 129 pg/ml (15-65pg/ml) and 24 hour urine calcium 28mg. A diagnosis of pseudohypoparathyroidism type 1b was made and calcitriol 2.25 mcg twice daily and calcium carbonate 500 mg oral daily were initiated. Her serum calcium improved to 9.8mg/dl and PTH level improved to 33 pg/ml. Evaluation of urinary cyclic-AMP in response to administration of PTH was not done. All symptoms resolved. Calcitriol was discontinued because of cost concerns and the patient was started on vitamin D3 400 units daily and calcium carbonate 500 mg daily. Doses were gradually increased to 2000 units daily and 1000 mg daily respectively; during this time she had several low calcium levels ranging from 7.4-8.6mg/dl and elevated PTH levels ranging from 114-147pg/ml. At this time her care was transferred to us. On initial evaluation her lab values were calcium 8.6 mg/dl, 25-OH vitamin D 59 ng/ml (30-80mg/ml), 1,25-OH vitamin D 44pg/ml (15-75pg/ml), PTH 147pg/ml, phosphorous 3.4 mg/dl. Calcitriol 0.5 mcg twice daily was restarted and the patient’s labs were recked in 4 weeks at which time her calcium was 10.1mg/dl, vitamin D level 53ng/ml and PTH 20pg/ml . Discussion: The initial differential in this case included lack of calcium intake, pseudohypoparathyroidism type 1b, vitamin D dependant rickets and vitamin D resistant rickets. Her hyperparathyroidism was felt to be secondary to her low calcium. As soon as her calcium normalized her PTH returned to normal. There was no history of low calcium intake to explain her hypocalcemia and both 25- and 1,25- vitamin D levels were normal suggesting end-organ insensitivity to 1-25 vitamin D. In both pseudohypoparathyroidism and vitamin D dependent rickets 1,25-OH vitamin D level should be low. In this patient, relatively low dose calcitriol and minimal dose calcium supplementation resulted in normalization of calcium and PTH. We suggest that the patient’s endogenous 1,25 Vitamin D was in some way partially inactive. This entity has not previously been described in the literature. In order to confirm this hypothesis it would be necessary to see if her 1,25 vitamin D bound appropriately to the 1,25 vitamin D receptor.
CLOT OR NOT: THE CASE OF THE VANISHING THROMBUS
Bisi Alli DO, Abstract 1

Bisi Alli, DO, MS, Joseph Orme, DO, MPH, Victor Sein, DO, Geetha Kolli, MD, Banner Good Samaritan Medical Center, Departments of Internal Medicine, Cardiology, & Hepatology and Liver Transplant, Phoenix, AZ

Introduction: Hepatic cirrhosis is a condition associated with many complications including ascites. Treatment of refractory ascites may include placement of a TIPS stent, which may become infected or thrombosed. A thrombus may occlude the stent, but rarely is it so large as to cause confusion in identity or location.

Case Report: A 38-year-old female with known cirrhosis, MELD 18, listed for liver transplant, and recurrent cholangitis, was admitted for complaints of persistent fever, chills and right upper quadrant abdominal pain with duration of one month. Her cirrhosis is the consequence of a cholecystectomy with unfortunate complications of secondary biliary cirrhosis, hepatojjunostomy, large ventral abdominal hernia, and long term biliary drainage catheter. She is status post transjugular intrahepatic portosystemic shunt (TIPS) with revision and known TIPS stent occlusion. Physical exam was notable for right upper quadrant abdominal tenderness to palpation, ascites, stable biliary drain catheter as well as additional clinical stigmata of cirrhosis. A clinical diagnosis of Streptococcus anginosus bacteremia was made from several blood cultures with likely gastrointestinal source. Broad-spectrum antibiotics were continued and transesophageal echocardiogram (TEE) was performed to rule out endocarditis. TEE demonstrated an incidental 1.2 cm, highly mobile, pedunculated, non-obstructing mass, which was visualized diving into and out of the right atrium with cardiac motion and notably, passing into the tricuspid annulus in diastole. It appeared to extend from the cephalad aspect of the inferior vena cava with likely adherence to the cephalad aspect of the TIPS stent. Systolic function was preserved and there was no evidence of vegetation, right heart strain, or significant valvular dysfunction. There were no clinical sequelae of venothromboembolism. The differential diagnosis for the mass was thrombus, vegetation, or malignancy. One week later, repeat TEE and inferior venocavogram with follow through to pulmonary angiography no longer demonstrated the mass, but instead a very small, residual echodense lesion at the edge of the hepatic TIPS stent, which was of identical echodensity to the previously visualized mass. This led to the conclusion that it was a thrombus that had spontaneously resolved in the setting of coagulopathy secondary to end stage liver disease.

Discussion: While spontaneous resolution of a thrombus can occur, the rapid disappearance of a 1.2 cm mass is surprising in the absence of common clinical sequelae. This case presentation illustrates the unusual clinical course of a known complication of the TIPS stent.

CANCER + NEUTROPENIA + FEVER = HEPARIN DRIP
Hammam Alquadan MBBS, Abstract 62

Introduction: Pulmonary Embolism (PE) is a common and often fatal disease. Mortality can be reduced by a high index of suspicion for the diagnosis and early therapy. Unfortunately, the clinical presentation of PE is not always obvious and often asymptomatic and nonspecific, making accurate diagnosis difficult. Case: A 60 year old man recently diagnosed with a
recurrent squamous cell carcinoma of the right tonsil with lung metastases, presented with 2 days of fever. Ten days prior to admission he was started on chemotherapy with Taxotere. Several days later because of severe fatigue and diarrhea he became bed bound. These symptoms resolved just prior to admission. The patient had a chronic non-productive cough, but denied any dyspnea or chest pain. On physical exam the patient looked comfortable saturating 98% on room air. His temperature was 102.4°F, Pulse (98), BP (105/74) and RR (18). His cardiovascular, respiratory, abdominal and lower limbs examinations were all unremarkable. Labs revealed a WBC of 600 and absolute Neutrophil count (ANC) of 12. His hemoglobin, CMP and urinalysis were normal. Chest X-ray showed a lung nodule with no evidence of pneumonia. The patient was admitted for neutropenic fever and begun on cefepime and filgrastim. Blood and urine cultures were negative. ABG was never done. He continued to be febrile at night while on cefepime. Vancomycin was added on day 3. Despite the antibiotics and an increase in his ANC to greater than 3000 his fever persisted. A CT scan of the neck and chest was ordered looking for a hidden abscess and it revealed large pulmonary emboli of both the right and left main pulmonary arteries with no evidence of infarction. Anticoagulation was begun. Fever resolved one day after the anticoagulation was started, and the patient discharged home with a scheduled follow up. Discussion: There have been an increasing number of reports of incidental asymptomatic pulmonary emboli (PE) detected in patients undergoing chest CT for reasons other than suspected PE. The prevalence of asymptomatic PE found incidentally is close to 2.6%. The natural history of unsuspected PE is unclear. Whether the diagnosis of an unsuspected PE is associated with increased morbidity and mortality, especially in patients with cancer, remains to be determined. Furthermore, the optimal therapeutic strategy when an asymptomatic PE is incidentally diagnosed is uncertain. In the absence of evidence of the risk and benefits of treatment, it is currently recommended that these patients be treated similarly to symptomatic patients. It is also important to recognize that large PE may occur in asymptomatic patients. In a neutropenic cancer patient who remains febrile despite antibiotics and despite the improvement in white cell count the astute clinician needs to consider a PE in the differential diagnosis.

SEVERE HYPERCALCEMIA SECONDARY TO MILK-ALKALI SYNDROME
Huthayfa Ateeli, Abstract 29

ABSTRACT INTRODUCTION: Hypercalcemia is a metabolic disorder which has been known to cause altered mental status. Literature review reveals plenty of cases of moderate to severe hypercalcemia, but rarely with calcium levels more than 18mEq/dL. Here we present a case of profound hypercalcemia in a patient with serious decline in the level of consciousness secondary to Milk Alkali Syndrome. CASE PRESENTATION This is a 56-year-old gentleman with history of recurrent seizures and gastroesophageal reflux disease, who presented with altered mental status. The patient was found down by a friend, for an unknown period of time. At the time he was brought to the emergency department, he was agitated, disoriented and uncooperative. His pupils were constricted, but equal and reactive. He was moving all four extremities non-purposely. His deep tendon reflexes were 1+ bilaterally. His fingerstick blood sugar in the field was 103, and his vital signs were within
normal limits except for slightly elevated blood pressure. HOSPITAL COURSE: A head CT scan was obtained immediately and was unremarkable. His initial laboratory serum workup showed creatinine of 2.3 (baseline 0.6), urea nitrogen of 34, Calcium level of 21.2 meq/dL corrected to albumin, Phosphorous of 3.8, and serum bicarbonate of 31. Other serum tests including alcohol, ammonia, sodium, and thyroid stimulating hormone (TSH) were reported normal. The patient was started on generous amounts of intravenous fluids for hypercalcemia and acute kidney injury. He was also given calcitonin and pamidronate. Over 3 days his calcium dropped to 7.8 corrected to albumin. His mental status improved back to his baseline and his creatinine dropped to 0.6. The patient underwent an extensive workup for hypercalcemia. His parathyroid hormone (PTH) was 6 pg/mL (Reference range: 15 to 65) and parathyroid hormone related peptide (PTHrP) was also low <0.2. His 25-hydroxyvitamin D3, 1,25-dihydroxyvitamin D3, 25-hydroxyvitamin D2, TSH, urine protein electrophoresis (UPEP) & serum protein electrophoresis (SPEP) were normal. After recovery patient admitted that he was taking around 6-8 grams of Tums daily for worsening heartburn with poor hydration for about 2 weeks prior to admission. CONCLUSION: Milk-alkali syndrome, which was once considered to be of only historical interest as a cause of hypercalcemia, is still occurring today in the face of life threatening serum levels of calcium. Patients and physicians should be aware that even though these medications are considered over-the-counter, they can still be dangerous.

Third Place – Associate Research Poster:

THE IMPACT OF EARLY AND SHORT-TERM CORTICOSTEROIDS ON THE CLINICAL COURSE OF PATIENTS WITH PRIMARY PULMONARY COCCIDIOIDOMYCOSIS – A RETROSPECTIVE CASE CONTROL STUDY

Natalya Azadeh MD, Abstract 75

Aim: To characterize the effects of short-term corticosteroids on the clinical outcome of primary pulmonary Coccidioidomycosis (CM). Background: Coccidioidomycosis is an endemic fungal infection of the desert southwestern United States. Primary pulmonary CM manifests as a febrile respiratory syndrome of varying degrees of severity, often associated with symptoms (such as rash or wheeze) which prompt clinicians to prescribe brief courses of corticosteroids. Immune-suppression, including the use of corticosteroids, is a known risk factor for severe or disseminated CM infections. Previous studies investigating the impact of corticosteroid use on the course of CM have focused on immune-suppressed patients who were chronic recipients of corticosteroids. The effect of short-term corticosteroids on the clinical course of immunocompetent patients with CM infection is not known. Methods: A retrospective review was conducted of all immununocompetent patients with acute pulmonary CM who received systemic corticosteroids for symptomatic relief of CM-related symptoms or signs. Age and sex matched controls were also reviewed. Pre-determined endpoints were: death due to CM, hospitalization, extra-pulmonary dissemination, relapse, symptom duration, and pulmonary manifestations. This study was approved by the Mayo Clinic Institutional Review Board. Results: 74 patients met criteria for inclusion to the steroid group. An equal number of age and
sex-matched subjects who did not receive steroids were reviewed as our control group. Cumulative doses of steroids ranged between 10-3600 mg (mean 206, median 120 mg). Steroids were prescribed most commonly for rash (58%) or asthma/wheezing (18%). In the steroid group, 81% received antifungal treatment compared to 77% in the control group (P = 0.6). CM-related hospitalization occurred in 19 patients (26%) in the steroid group, compared to 18 patients (32%) in the control group (p =0.7). CM-related symptoms in the steroid group resolved within a mean of 19 weeks (median 8 weeks, range 2 – 156 weeks) vs. a mean of 16.9 weeks (median 8 weeks, range 1-168 weeks) in the control group. There were no deaths due to CM in either group. Relapse of CM symptoms occurred in 12% of patients in the steroid group vs. 10.9 % in the control group (p=1.0). Extra-pulmonary dissemination occurred in 8% vs. 6.7% (p=1.0) in the steroid and control group respectively. The mean duration of anti-fungal therapy in the steroid group was 42 weeks (range 1-286 weeks) vs. a mean of 35.5 weeks (range 3-209 weeks) in the control group (p=0.49).

Conclusion: This retrospective case-control study found no adverse effects of short-term corticosteroids on the clinical course of acute pulmonary CM. There were no significant differences in outcomes between the steroid and the control groups: All of the endpoints assessed were unaffected by short-term corticosteroid therapy for early symptomatic treatment in acute pulmonary CM.

MIRIZZI SYNDROME
Devang Butani DO, Abstract 38

Pt is a 73 year old Hispanic female who presented to a surgeons office for abdominal pain. She stated that she has been having the pain for 2 weeks with accompanied nausea and vomiting. She has been “turning yellow” since her pain started. She has lost 145 lbs over 1 year and has been progressively been feeling weak. She denies any fevers, chills, night sweats, shortness of breath, diarrhea, dysuria. A 14 point of review of system was done and negative besides what was listed above. The surgeon was concerned and decided to admit the patient for further work up of her abdominal pain and jaundice. Patient had CT of the abdomen and pelvis with contrast, an ultrasound of the gallbladder, and MRI of the abdomen without contrast. CT which showed no calcified gallstones, however, the gallbladder wall appeared to be moderately thickened to 6 mm. CT also showed intrahepatic biliary dilatation with focal nodular opacity within the left lower lobe of liver. There was also an incarcerated umbilical hernia seen. US showed similar findings as the CT but also revealed common bile duct dilatation of 1.2 cm. MRI showed small cholelithiasis with intrahepatic biliary dilatation. Based off the radiological findings, the surgeon operative intervention for acute cholecystitis along with an exploratory laparoscopy. At one point it was elected to become a laparotomy due to complexity of the adhesion seen on the laparoscope. The gallbladder was removed with multiple gallstones. No stones were identified in the common bile duct. Where the gallbladder had been attached, it was assumed that had been the area where the compression had resulted in impedance of the bile causing the obstructive jaundice. At this time the T-tube was placed. Then the surgeon proceeded to go remove the incarcerated hernia which turned out to be a strangulated hernia. After all was repaired, a JP drain was placed. All the tissue removed was sent to pathology. Patient’s current state was expected to improve. Her
pain did but her jaundiced worsened. It was unsure why she did not get better until the pathology report came back, showing cholangiocarcinoma with metastasis to liver, omentum, and umbilical hernia. At this point different treatment options were discussed including aggressive chemotherapy or palliative care. After careful consideration, it was decided by the family that the patient go to hospice care. Mirizzi Syndrome refers to common hepatic duct obstruction caused by an extrinsic compression from an impacted stone in the cystic duct or Hartmann’s pouch of the gallbladder. It has been estimated to occur in 0.7 to 1.8 percent of all cholecystectomies. It is often not recognized preoperatively, which can lead to significant morbidity and biliary injury, particularly with laparoscopic surgery.

**DENGUE FEVER COMES TO THE DESERT**  
**Dino Cekro MD, Abstract 73**

Introduction: We present a case of Dengue fever that made its way to Phoenix, Arizona. Our young patient was traveling throughout South America participating in Frisbee golf competitions. The patient had just arrived in the United States when he began to develop an acute febrile illness requiring hospitalization. Case: This patient is a 25 year old Danish male with no significant past medical history who had been traveling around the world. He most recently traveled through South and Central America visiting Belize and Yucatan, Mexico. Upon arrival in the United States, he started to experience fever with night sweats, nausea, vomiting and a severe headache for two days. The patient claimed adherence with malaria prophylaxis, denied eating uncooked food, and reportedly only drank bottled water while being abroad. He also denied having any unprotected sexual intercourse. However, he did recall having been bitten by an unknown type of insect while in Belize. Upon initial evaluation the patient’s temperature was 39.2°C, he continued with undulating fevers. Physical exam was benign except for a petechial rash present on both shoulders. Initial labs revealed an elevated PT of 17.5 seconds and INR of 1.6, mild transaminitis, leukocytosis of 16.7x103/# L and thrombocytopenia of 72,000 K/# L with giant platelets. An extensive workup for an infectious etiology was initiated; he had a negative chest x-ray, normal urinalysis, peripheral blood smear found no plasmodia, blood cultures failed to grow any organisms, and stool samples revealed no ova or parasites. Lumbar puncture showed mildly increased neutrophils, however, cultures were negative. While our initial work up for acute febrile illness failed to reveal any significant cause, Dengue fever was suspected due to the patients recent travel history. At this time Dengue fever work-up was initiated. Public health confirmed the diagnosis of Dengue fever with antibody testing. Supportive care was continued with gradual improvement in thrombocytopenia and resolution of fever, the patient was discharged. Conclusion: Dengue Fever is a very important mosquito-borne disease worldwide; however it is very rare in the USA. It is mostly found in the sub-tropical climates. Today 3-8% of all febrile travelers who visit endemic area are diagnosed with Dengue Fever. Dengue infection is acquired through the bit of Aedes aegypti and Aedes albopictus mosquitoes. Even though currently Dengue infections are still rare in US our case proves that Dengue virus infection should be considered in the differential diagnosis of an acute febrile illness in a patient who has been living in or traveled to an endemic region in the last two weeks prior to presentation.
Second Place – Associate Research Poster:

PREDICTIVE VALUE OF NASAL METHICILLIN-RESISTANT STAPHYLOCOCCUS AUREUS (MRSA) PCR SWAB FOR MRSA PNEUMONIA

Andrew Chung MD, Abstract 76

RATIONALE: Methicillin-resistant Staphylococcus aureus (MRSA) has emerged as an increasingly important pathogen in pulmonary infection, particularly in those with a history of significant health-care contact. S. aureus, including MRSA, colonizes the nares, and colonization has been shown to be a predictor of future clinical infection. MRSA colonization can be reliably detected in the nasopharynx using the nasal swab polymerase chain reaction (PCR) test. It has been postulated that this test may have diagnostic value in patients with culture-negative MRSA infections. In this study we evaluated the performance of the nasal swab MRSA PCR in predicting MRSA pneumonia.

METHODS: A retrospective analysis was performed in an academic tertiary care facility from January 2009 to July 2011. Inclusion criteria included patients with a nasal MRSA PCR swab performed during the study time period, a diagnosis of pneumonia, and a clinical culture (sputum, BAL or blood). Exclusion criteria included age <18, an alternative diagnosis more likely than pneumonia, and the MRSA swab being performed more than one month prior to culture for patients presenting with pneumonia and more than 7 days prior to culture in hospital-acquired cases. Data was then used to calculate sensitivity, specificity, positive predictive value, and negative predictive value.

RESULTS: A total of 435 patients met criteria for analysis. MRSA nasal swabs were positive in 14.3% and the prevalence of MRSA pneumonia was 5.7%. Analysis resulted in a sensitivity of 88.0%, specificity of 90.1%, a positive predictive value of 35.4%, and a negative predictive value of 99.2%.

CONCLUSION: The results of this retrospective analysis suggest that in patients with pneumonia, the MRSA PCR nasal swab has a poor positive predictive value, but has an excellent negative predictive value for MRSA pneumonia in a population with low MRSA pneumonia prevalence. The most clinically relevant implication of these results is that in patients treated empirically with MRSA-active antibiotics, a negative MRSA PCR swab can be reasonably used to guide antibiotic de-escalation provided that the pre-test probability of MRSA pneumonia is not extremely high. A prospective trial is needed in order to confirm these findings.

A CASE OF URTICARIAL VASCULITIS WITH A UNIQUE UNDERLYING DIAGNOSIS

Corinne Cox MD, Abstract 114

Introduction: Urticarial vasculitis is a condition, often systemic, characterized by urticarial lesions and skin biopsy consistent with histopathologic evidence of leukocytoclastic vasculitis. The condition is usually idiopathic but has been associated with a number of underlying diseases. Case description: A 65 year old Caucasian male with a three month history of pruritic rash not responsive to topical corticosteroids, arthralgias, fevers and night sweats with recent diagnosis of pernicious anemia is transferred from a community hospital to a tertiary care center for further evaluation. Skin biopsy of erythematous, urticarial macules and patches most prominent on upper extremities and abdomen reveal histopathology consistent with urticarial vasculitis. On laboratory analysis erythrocyte sedimentation rate is markedly elevated, complement levels are normal and ANA is
negative. Urinalysis reveals proteinuria and CT scan reveals splenomegaly. CBC was remarkable for a leukocytosis of 27,000 and a normocytic normochromic anemia with a hematocrit of 27%. Bone marrow biopsy was undertaken. Hematopathology revealed markedly hypercellular marrow and dyserythropoiesis consistent with an underlying diagnosis of myelodysplastic syndrome. Discussion: Urticarial vasculitis is usually idiopathic. Systemic involvement can overshadow underlying diagnosis such as a hematologic malignancy. A literature review was undertaken using Pub Med with the keywords urticarial vasculitis, myelodysplastic syndrome, myelodysplastic disorder, and myeloproliferative disorder. Only a few cases are described. A retrospective review from the Mayo Clinic, Rochester found that 7/162 patients diagnosed with myelodysplastic syndrome had cutaneous vasculitis, only 1 with urticarial vasculitis. A retrospective case series from the University of Florida noted a correlation wherein from 1970-1987 8/1,730 cases had vasculitis associated with lympho/myeloproliferative malignancy compared with 0/13,160 cases in all other malignancies. Conclusion: Urticarial vasculitis is usually idiopathic or associated with rheumatologic, infectious, or allergic diseases. If clinically warranted, a complete evaluation should be considered to rule out underlying causes of urticarial vasculitis including this unique association with myelodysplastic syndrome.

THE MANY FACES OF HYPERTHYROIDISM
Chirag Desai MD, Abstract 28

Chirag K Desai, MD; Rosemary Browne, MD, University of Arizona Medical Center, South Campus

Introduction: The association between hyperthyroidism and atrial fibrillation has been well established. Less common in the literature are associations with right ventricular overload and tricuspid regurgitation. We report the case of a middle-aged female who presented with all three findings in the context of a new diagnosis of Graves disease. Case Presentation: The patient was a 46 year old female with no known past medical history who presented with 3 weeks of gradually worsening dyspnea on exertion and lower extremity swelling. On presentation she was found to have an irregularly-irregular tachycardia, jugular venous distension to the mandibular angle with prominent c-v wave, a high-pitched 3/6 holosystolic murmur at the left lower sternal border, right upper quadrant abdominal fullness, and 3+ pitting bilateral lower extremity edema. She had a diffusely enlarged thyroid without discrete nodularity or tenderness. Lab data was notable for low TSH, high free T3, and positive thyroid stimulating immune globulin. An ECG demonstrated atrial fibrillation with rapid ventricular response between 110-125 bpm without evidence of right ventricular hypertrophy. A 2D-echocardiogram demonstrated preserved systolic function with ejection fraction (EF) of 55-59%, normal RV systolic function but severe tricuspid and mitral regurgitation, dilated left and right atria, and flattened interventricular septum during diastole consistent with RV volume overload. All valves were structurally intact. The patient was started on methimazole, propranolol, and furosemide, and she had gradual improvement of her leg swelling and shortness of breath. Follow-up 2D-echocardiogram four months later showed complete resolution of valvular dysfunction and normalization of all chamber sizes. Discussion: The mechanisms underlying
the various cardiovascular manifestations of Graves disease are varied and likely interrelated. Congestive heart failure is thought to result from the ‘hyperdynamic state,’ which shifts the heart to a mechanically unfavorable portion of the Frank-Starling curve. Pulmonary hypertension and frank ventricular failure with low EF and/or tricuspid annular plane systolic excursion (not seen in our patient) have also been identified in hyperthyroid patients without any other identifiable cause. Similarly, atrial fibrillation and severe tricuspid regurgitation are both independently associated and frequently observed in those patients who go on to develop some degree of heart failure. Controversy exists as to whether or not there is a pure ‘thyrotoxic cardiomyopathy.’ But most if not all studies are in agreement that treatment of the underlying hyperthyroid state leads to resolution of cardiac manifestations. This case illustrates a less common presentation of Graves hyperthyroidism in the form of clinical findings of congestive failure with right ventricular overload and severe tricuspid regurgitation as well as atrial fibrillation. It also reinforces the value of searching for a unifying, reversible cause.

A RARE CAUSE OF DYSPHAGIA - ESOPHAGEAL SQUAMOUS PAPILLOMATOSIS
Anjali Dixit MD, Abstract 47

Introduction – Squamous papillomatosis of the esophagus is a rare condition which in severe cases can cause dysphagia, and may also progress to squamous cell carcinoma. Case Description – 76 Y M was admitted secondary to chronic progressive dysphagia for one year. Initially he had difficulty swallowing solids, but over the last two weeks liquids as well. He lost 30 lbs over one year but denied changes in bowel habits, melena, hematemesis, or abdominal pain. He had intermittent nausea, odynophagia, sore throat, cough and food regurgitation. He took NSAIDs intermittently and drank alcohol chronically. He had a history of intermittent dysphagia to solids for the last 10 years, and had multiple EGDs which showed chronic candida esophagitis and distal benign appearing (likely esophagitis related) esophageal stricture s/p dilations. He was treated with daily PPI and multiple courses of Fluconazole. EGDs performed over the last year showed diffuse edematous and friable mucosa from mid to distal esophagus along with diffuse and progressive multiple nodular to papillomatous growths of varying size causing luminal narrowing and obstruction in the lower half of esophagus. Biopsies on many occasions showed acute on chronic esophagitis, reactive squamous epithelial hyperplasia consistent with papilloma, negative viral cytopathic effect and candida esophagitis. HIV, Hepatitis B and Hepatitis C screens were negative. CT of the chest showed a thick distal esophageal wall. Currently, patient is treated with twice daily PPI, Sucralfate and Fluconazole and being fed by nasogastric tube. He is scheduled for EGD with EUS for deep esophageal tissue biopsy and possible endoscopic mucosal resection of large papillomatous growths.

Discussion - Development of extensive esophageal squamous papillomas is known as squamous papillomatosis of the esophagus. It is a rare condition and English literature review shows about a dozen case reports only. The individual papilloma lesions may be of varying sizes and appear as wart-like exophytic growths. Histology demonstrates the characteristic finger-like projections of hyperplastic squamous epithelium with underlying fibro-vascular connective tissue core. The pathogenesis of esophageal
papillomatosis is hypothesized to be secondary to infection with HPV and/or repetitive mucosal chemical injury followed by regeneration causing continued squamous hyperplasia and papilloma formation. Due to the rarity of reported cases, little is known about the etiology, natural course and best clinical management of this disease. Reports of spontaneous regression, esophageal stricture and development of squamous cell carcinoma have been found. Extensive lesions with dysphagia symptoms such as the one described above would likely require the surgical resection for concurrent diagnosis and treatment.

Conclusion – Esophageal squamous papillomatosis is an incompletely understood and rare condition which requires further evaluation to monitor for development of squamous cell carcinoma.

LEUKEMIA IS ONLY SKIN DEEP: A CURIOUS CASE OF PANCYTOPENIA
Laura Durling MD, Abstract 50

Introduction: Pancytopenia is a fairly common condition seen in the hospital and the workup can be extensive. Etiologies include infection, medication side effects, and malignancy. One of the causes is AML which rarely has an extramedullary presentation. Case Presentation: This is a case of a 44 year-old African American man who presented to hospital with three week history of nonproductive cough, fatigue, decreased appetite, and URI symptoms. He denied any recent weight loss, easy bruising/bleeding, alcohol or drug use. He did not have any recent travel or history of occupational exposure to toxic chemicals. He had labs done 9 days prior to admission and at that time his WBC was within normal limits, with mild microcytic anemia, and normal platelet count. However, on day of admission the patient was pancytopenic with WBC of 3.47, hemoglobin of 8.3 and platelet count of 62. Physical exam revealed hypotension, fever of 38 degrees, and no obvious signs of petechiae. He had a 5-6 cm circumscribed indurated, mildly erythematous lesion on medial upper thigh a few weeks in duration. A skin biopsy was obtained and sent for infectious studies and pathology. During his hospitalization the patient experienced several neutropenic fevers so an infectious workup was started. The working differential diagnosis included coccidioidomycosis, tick borne diseases, and atypical infections and he was started on Doxycycline, Levaquin, and Fluconazole. However, there was no significant improvement in his symptoms. Initial diagnostic labs were done and revealed B12 deficiency with B12 level of 196. Iron studies were consistent with anemia of chronic disease. Other labs that were obtained and were negative or within normal ranges were HIV, EBV, CMV Coccidioidomycosis, Rickettsia, Histoplasma, Blastomyces, HHV8, and folate level. Bone marrow biopsy “findings are highly unusual. They include erythroid hyperplasia with maturational abnormalities, megakaryotic hyperplasia, myeloid left shift and zonal necrosis in the pattern suspicious for tumor.” The role of Neupogen therapy, which he received early in his hospitalization, was uncertain. When the results of the skin biopsy revealed atypical cellular infiltrate most consistent with myeloid sarcoma, the final diagnosis was AML with myeloid sarcoma.

Discussion: This case demonstrates the extensive workup for pancytopenia with neutropenic fevers. At first, an coccidioidomycosis or a different infection was the prevailing diagnosis. B12 deficiency was considered to be contributing, however it
seemed less likely as the primary cause given the rapid development of his pancytopenia. This case demonstrates that although AML is a common acute leukemia in adults, extramedullary signs are not commonly seen especially prior to finding acute leukemia in bone marrow.

GASTROINTESTINAL BASIDIODOBOLOMYOSIS, ANOTHER EMERGING FUNGAL INFECTION IN ARIZONA
Razan El Ramahi MBBS, Abstract 89

A 34 year old female presented with two-week history of intermittent abdominal pain and fever. Pain was described as right lower quadrant, progressively worsening and with no radiation. She has a past medical history of D.M., developmental delay and GERD. Upon admission she was febrile, had RLQ tenderness and was found to have elevated WBC count of around 14,000, 17% eosinophils. An abdomen/pelvic CT scan showed perforated appendicitis, with abscess formation, measuring up to 5.7 cm. IR drainage attempted but no aspiration or drainage performed. Biopsy specimens came back with no growth. She failed to improve with recurrent fever and persistent leukocytosis. Repeat CT scan showed stable right lower quadrant mass and abscess. Repeat IR drainage grew CONS, Lactobacillus and a mold. Patient was continued on IV antibiotics and discharged home on oral antibiotics with the drain in place. Follow up at the surgery clinic with an abscessogram revealed a colocutaneous fistula. Exploratory laparotomy, ileocecectomy and intra-abdominal abscess drainage were done. Specimens were sent for pathology. Pathology showed necrotizing fungal (extensive) granulomaticus inflammation and fungal hyphae consistent with Basidiobolus. Fungal culture from her previous admission was identified as Basidiobolus spp. She was placed on oral itraconazole, improved and discharged home. Basidiobolus ranarum is an environmental saprophytic fungus found worldwide in soil, decaying organic matter and as a commensal in the intestinal tract of amphibians, fish and reptiles. Infections usually present with subcutaneous nodules and commonly found in tropical areas of South America, Africa and Asia. Gastrointestinal involvement with invasive Basidiobolus infection has been rarely described in the literature. It was noted to be an emerging infection in Southwestern United States. A recent review examined 44 reported cases from around the world. Nineteen (43%) were from the United States, seventeen were residents of Arizona, one was from a Southern Utah and one was from Florida. No patients were immunocompromized. The most common presenting symptom was abdominal pain. Peripheral eosinophilia was noted in 26 patients (76%) and the most common imaging finding was a mass followed by bowel wall thickening. The initial provisional diagnosis in most of those patients was intraabdominal malignancy or inflammatory bowel disease. The typical histopathological feature is necrotizing granulomatous inflammation containing thin-walled, broad, aseptate hyphae with a surrounding eosinophilic sheath, known as Splendore-Hoepppli phenomenon. Confirmatory fungal cultures and fungal staining should always be done on biopsy specimens from unusual gastrointestinal masses. Sabouraud agar is an adequate medium, and visible growth is usually present within 2–3 days. Visceral Basidiobolomycoses are rare and still not well understood but should be suspected when a patient presents with abdominal complaint(s) along with elevated WBC count, eosinophilia.
and mass or thickening in the gastrointestinal system and fail to improve with conventional therapy.

AL AMYLOIDOSIS PRESENTING WITH XEROSTOMIA
Sarah Essary (Durnbaugh) MD, Abstract 91

Introduction: Amyloidosis is a rare disease characterized by the deposition of fibrils from monoclonal light chains which result in organ dysfunction. The clinical presentation of this disease can be quite varied and often mimics other more common diseases, often leading to late presentations and poor outcomes. Light-chain (AL) amyloidosis, or primary amyloidosis, is the most common variant and is associated with plasma cell dyscrasias such as multiple myeloma. A better understanding of the varied presentations of this disease will lead to earlier diagnoses and better outcomes. Case Presentation: The patient is a 54 year-old African American male with hypertension and ESRD on hemodialysis, who presented with progressive shortness of breath and weakness, as well as a ten month history of dysphagia secondary to xerostomia, gastroparesis and 100lb weight loss. Physical examination showed mild scleral icterus, JVD, hepatosplenomegaly, and 2+ pretibial pitting edema. Lab work was remarkable for elevated BNP, troponin I, and obstructive liver pattern. CT revealed cardiomegaly with pericardial effusion, diffusely heterogeneous liver, and hepatosplenomegaly. Signs of portal hypertension were present on a Doppler study. Echocardiogram illustrated EF 30-35%, increased RV wall thickness, and severely dilated atria, findings consistent with cardiac amyloidosis. At this point, subcutaneous fat pad biopsy was performed, showing green birefringence with Congo Red staining. These findings as well as SPEP and UPEP positive for monoclonal gammopathy were consistent with AL amyloidosis. Discussion: Amyloidosis often presents with non-specific complaints and therefore may be under diagnosed. Incidence is uncertain, but AL amyloidosis is suspected to occur in 6-10 cases per million in the United States. This diagnosis should be considered in patients with heart failure, non-diabetic nephropathy, and isolated hepatomegaly with elevated alkaline phosphatase. Screening of patients with these symptoms by serum and urine immunofixation is a sensitive, non-invasive test, and diagnosis must be confirmed by tissue biopsy, usually fat or marrow. The standard treatments of alkylating agents and corticosteroids have had moderate success, and more effective treatments are either still in clinical trials or limited to a small-subset of patients who qualify. Treatments become much less effective with extensive end-organ involvement, therefore early diagnosis is key. Despite current treatment, 30% of patients die within one year of diagnosis. Cardiac and hepatic involvement are poor prognostic factors in amyloid, both of which were present in our patient. It is possible his long-standing renal disease, previously attributed to hypertension, was a result of his amyloidosis, and he may have been a candidate for treatment years before his eventual diagnosis. Our patient decided to forego chemotherapy as the benefits would have been minimal, and he died one week after diagnosis. Internal medicine physicians need to keep rare disorders such as amyloidosis in the differential.
POLYMYALGIA RHEUMATICA: IT CAN HAPPEN TO AFRICAN AMERICANS TOO!
Meka Ezeume MD, Abstract 94

Meka Ezeume MD (Associate), Chelsi Scull MD (Associate), Bertram Hurowitz MD, Maricopa Integrated Health System, Phoenix, Arizona

Introduction: Polymyalgia Rheumatica (PMR) is an inflammatory rheumatic condition with both environmental and genetic factors playing a role where affected patients present with achiness and stiffness in the shoulders, hip girdle and neck. It is mainly a disease of the elderly and Northern European Caucasian females. We present a case of PMR in an elderly African American (AA) female. Case: An 89-year-old AA female with past medical history of hypertension, hypothyroidism, CKD stage 2, osteoarthritis, and pre-diabetes presented with severe generalized muscle aches, weakness, and urinary retention. On arrival to the ED, her vital signs were significant for pulse of 105 and temperature of 38°C. Her exam was significant only for muscle tenderness, especially in the extremities. She had a WBC count of 17x10³/µL with bandemia of 6%, mild anemia, creatinine of 1.32 mg/dL (slightly above her baseline of 1.1 mg/dL), AST 243 U/L, AST 66 U/L, and total bilirubin of 1.9 mg/dL. ESR was >140 mm/hour. ANA and C-ANCA were negative. She was started on 20 mg prednisone with suspicion for PMR and by evening, she had significant improvement in her symptoms. She also was found to have a urinary tract infection that was treated with sulfamethoxazole-trimethoprim. Rheumatology recommended tapering steroids over time and she was discharged home. Less than one week after discharge, after the dosage of her prednisone was decreased, she presented again to the ED with a return of all previous symptoms. Plain radiographs of her joints and CT scan of her neck showed only osteoarthritis. Once she was restarted on 20 mg of prednisone, her symptoms dramatically improved. She was discharged home on non-tapering dose of prednisone and told to follow up with rheumatology. Discussion: This case illustrates PMR in an elderly AA female with a classic presentation, usually seen in those of Northern European ethnicity. The actual incidence of PMR in AA population is unknown but is estimated to be up to seven times lower than that in Caucasians and is consistently published as rare. It has been proposed that though the prevalence of disease is likely similar among AA and Caucasians, the under-diagnosis in AA may be due to variable diagnostic criteria and differences in socioeconomic status. Recognizing that PMR may be more prevalent in AA than currently presumed may raise the differential diagnosis of PMR in AA with typical symptoms and may help provide the appropriate therapy.

Third Place – Associate Patient Safety/Quality Improvement Poster:

A RETROSPECTIVE DESCRIPTIVE REVIEW OF A PILOT ELECTIVE IN BEDSIDE ULTRASOUND IN EMERGENCY DEPARTMENT PATIENTS
Heather Fields MD, Abstract 131

Heather Fields MD, Joseph Wood MD, Janis Blair MD, Mayo Clinic Arizona

Aim: To describe the training received in a one month elective in bedside ultrasound. Background: Bedside Ultrasound (US) has been used by emergency medicine physicians and trauma surgeons for years when presented with patients in whom certain life threatening disorders (such as pneumothorax, myocardial...
infarction, cardiac tamponade, aortic dissection, and intra-abdominal hemorrhage) must be identified quickly. However, internal medicine residency programs are now finding great utility in ultrasonography and are developing curriculum for diagnosis and management of patients on hospital admission (Kimura et al). One variation of the bedside US limited exam known as “CLUE” (cardiovascular limited ultrasound exam) has been shown to improve diagnostic accuracy and change medical decisions even when performed by novices. We created a 4 week elective in bedside ultrasound for internal medicine residents similar to the above curriculum. Method: We retrospectively reviewed the experience of a single medicine resident during a one month pilot elective in bedside ultrasound in the Emergency Department (ED) setting. The resident underwent 3 two-hour sessions of small group training prior to the elective, reviewed online instructional videos, and attended a four hour ultrasound workshop provided by Sonosite at the completion. Potential US candidates were identified by the chief complaints of chest pain, dyspnea with or without hypoxia, shock, cardiac arrhythmia, syncope, abdominal pain, or flank pain. Findings were obtained and recorded with a M-Turbo (Sonosite Inc. Bothel, Washington). The US findings of 31 patients with chest pain and 17 patients with dyspnea were tabulated, and evaluated for correlation with formal echocardiography. Results: From August 6 - August 31, 2012 18 ED shifts and 153 hours were staffed by a single resident, and 165 ultrasounds were performed. There was a subjective, but overall positive correlation between findings and assessments of the formal echo and the bedside ultrasound. We found that the basic bedside ultrasound techniques can be used by the internal medicine resident to evaluate for overall heart contractility, pericardial effusions and tamponade, inferior vena cava (IVC) evaluation of fluid status, and pneumothorax. At the conclusion of the elective, the resident had great increase in confidence in her ability to use bedside ultrasound to help guide diagnoses in the future. One case of Takotsubo Cardiomyopathy was first identified by bedside US, and subsequently presented at Medicine Grand Rounds. Conclusion: Bedside ultrasound is an emerging diagnostic tool in internal medicine. The basics of the cardiac exam and IVC assessment can be taught and learned by mentorship within a four week period after attendance of 4-6 hours of small group introductory courses.

ALL BECAUSE OF A RING: AZOLE-CONTAINING MEDICATIONS CAUSING RECURRENT CASES OF TOXIC EPIDERMAL NECROLYSIS

Erica Flores MD, Abstract 92

Introduction: Imidazole and triazole are five member ring organic compounds which vary by the placement of a nitrogen versus a carbon at the four position. They are incorporated into a variety of pharmaceutical agents exhibiting a broad spectrum of medicinal properties. They both have the propensity to inhibit cytochrome P450, however, the triazole ring has a higher specificity for cytochrome P450, imparting on it a more potent adverse reaction profile. Case Presentation A 47-year-old male presented to his primary care physician with fatigue, fevers, and arthralgias. Serologic testing for coccidiomycosis was positive and fluconazole therapy was initiated. He subsequently developed a severe, desquamative full body skin rash with concurrent oral and ocular sloughing as well as mucosal erosion on endoscopy. Fluconazole was discontinued and
he was discharged on a prednisone taper for possible drug rash. The rash recurred with cessation of prednisone and he was readmitted. Biopsy of the skin lesion at that time was consistent with a drug eruption versus erythema multiforme. Steroids were restarted with improvement of his symptoms. In an effort to treat his coccidiodal infection, he was started on itraconazole, given the potential allergy to fluconazole. Itraconazole resulted in eruption of his rash once again. Given his critical illness after use of itraconazole, he was admitted to the ICU and suffered numerous complications. He was then stabilized and transferred to the medical floor. He developed diarrhea and tested positive for clostridium difficile infection for which metronidazole was started. He shortly thereafter developed a severe blistering skin rash. Skin biopsy was again consistent with a drug eruption. Cessation of metronidazole and IVIG therapy resulted in rash improvement. Unfortunately, prior to his anticipated discharge, the patient was transferred back to the ICU and required intubation for severe pneumonia and MRSA bacteremia. Lansoprazole was started for GI prophylaxis, resulting in recurrence of his severe, blistering rash. Given the correlation of his rash with the use of azole-type medications, including fluconazole, itraconazole, metronidazole, and lansoprazole, it was determined he had a severe drug allergy to medications containing azole rings with the devastating effect of toxic epidermal necrolysis. Discussion: Imidazole and triazole rings are five membered planar rings with nitrogen-containing polycyclic structures. This unique structure allows them to have a wide range of biological activities. Their therapeutic properties include anti-neoplastic, anti-fungal, anti-inflammatory, analgesic, and anti-helminthic. Many pharmacologic agents contain such rings thus creating an avenue for allergic reaction. While the imidazole and triazole rings are advantageous in many respects given the wide range of therapeutic benefits, they have the propensity to result in devastating effects, including severe drug allergies, namely toxic epidermal necrolysis. The case was notable for severe medical complications that can be seen with two similar biological components.

LACTIC ACIDOSIS AND THE ROLE OF GLUCOSE THERAPY IN THE SETTING OF CHRONIC LIVER DISEASE AND HYPOGLYCEMIA
Rene Franco Jr. MD, Abstract 44

Rene Franco Jr. MD, Harold Szerlip MD, Farhad Sahebjam MD, Anju Nair MD University of Arizona – South Campus

Lactic acidosis is associated with increased morbidity and mortality in hospitalized patients. Lactate accumulates when pyruvate produced during glycolysis cannot enter the tricarboxylic acid cycle because of either inadequate oxygen delivery to tissues or an inability to use oxygen. In addition, in the setting of acute or chronic liver disease, impaired hepatic gluconeogenesis decreases lactate metabolism and further exacerbates lactic acidosis. When assessing a patient with lactic acidosis, it is important to recognize the cause and whenever possible correct the abnormality. This, however, is not always straightforward.

A 70 y/o male with a past medical history including coronary artery disease, ischemic cardiomyopathy and alcohol abuse presented with severe lactic acidosis (19.8 mmol/l) with blood pH of 6.91 and glucose of 16mg/dl in the setting of acute decompensated heart failure, transaminasemia with AST 157 IU/L and ALT 67 IU/L and acute renal failure. Initial blood
pressure on arrival to the emergency department was 93/64 mmHg, which improved without fluids and remained greater than 100 mmHg systolic. While in the emergency department, the patient was given 50% dextrose, furosemide and antibiotics. Repeat labs obtained on arrival to the Intensive Care Unit six hours later showed a pH of 7.31, lactate of 8.3 mmol/l which improved to 2.6 mmol/l on morning labs only eight hours later. Although the lactic acidosis may have been secondary to hypotension, it is more likely that the extremely elevated levels were secondary to impaired hepatic gluconeogenesis in the setting of liver disease exacerbated by his hypoglycemia. There have been several previous case reports describing the association of hypoglycemia with lactic acidosis. In these cases, the administration of glucose promptly improved the acidosis. The role of glucose therapy in these situations is not well defined and is poorly understood. Whether correction of the hypoglycemia improves hepatic gluconeogenesis or oxidative metabolism is unclear. For this reason, further studies are needed to assess the role of glucose therapy in patients presenting with lactic acidosis and concurrent hypoglycemia.

WHEN IN DOUBT, TALK TO THE RADIOLOGIST AND REVIEW THE IMAGES
Lisa Fujima DO, Abstract 117

Lisa Fujima DO, Associate, Verde Valley Medical Center, Cottonwood, AZ

The etiology of acute symptomatic anemia may not always be clear, even with the availability of CT imaging. This is a case of trusting your medical instincts and if necessary, re-checking the radiographic imaging.

A 104 year old woman with a history of essential thrombocytosis presented to the ER at Verde Valley Medical Center from a nursing home with a one day history of left hip pain. There was no fall or trauma. Pain was severe enough that she was unable to walk on it. On Day one of admission, vital signs were pertinent for a blood pressure of 190/70. Physical exam showed no swelling or deformities in the hips and X ray of the femur showed normal alignment, no fracture, and normal soft tissues. CBC showed hemoglobin 9.8, hematocrit 28.2, WBC 10.8, and platelets 813.

On the second day of admission, the patient became hypotensive with a systolic blood pressure in the 80s and she was complaining of abdominal pain. Hemoglobin dropped to 8.4 g/dl. Rectal exam showed no gross deformities or lesions and stool occult blood was negative. CT scan of the abdomen and pelvis was ordered with the indication written as LLQ abdominal pain. The CT was read as mild thickening of the descending colon and diverticulosis of the rectosigmoid colon.

By the forth day of admission, the patient’s blood pressures had stablized, however, the patient’s hemoglobin dropped to 7.4 g/dl. Secondary to a high suspicion of internal hemorrhage, the Radiologist was approached to review the imaging studies. In the last two slides of the CT of the pelvis, a significant hematoma was found in the left thigh. A CT of the left thigh was ordered which showed an extensive intramuscular hematoma posterior to the femur estimated to be 6 x 7 x 22 cm. At discharge, the patient’s blood pressures and hemoglobin had returned to baseline. Thrombosis and hemorrhage are common complications of essential thrombocytosis with the incidences of hemorrhage greater than thrombosis. With a history of essential thrombocytosis and a presentation of acute
symptomatic anemia and hypotension, it is important to keep a high suspicion for unusual hemorrhages. This case emphasizes the necessity of providing the Radiologist with a relevant case presentation, trusting one's medical instincts, and re-checking the radiographic images when the clinical picture does not add up.

LIFELONG URTICARIA: THE ITCH THAT COULDN'T BE SCRATCHED
Jennifer Hill MD, Abstract 115

Cryopyrin-associated periodic syndromes (CAPS) are a rare group of clinically overlapping autoinflammatory disorders associated with aberrant cytokine regulation as a consequence of mutations in the NLRP3 gene. A 27-year-old Caucasian female presented for evaluation of recurrent urticarial rash, present since early childhood, and refractory to antihistamine and corticosteroid therapies. She described periodic, migratory, erythematous, raised eruptions located primarily on her trunk and thighs precipitated by generalized cold exposure. Episodes were associated with arthralgias, conjunctivitis, chills, and fatigue that lasted for days. Previous cold provocation testing produced delayed onset of urticarial rash that is precipitated by cold exposure and is associated with flu-like symptoms, arthralgias, fevers, and conjunctivitis. Disease occurs as a result of a gain of function mutation in the NLRP3 gene encoding for cryopyrin, which forms intracellular protein complexes known as inflammasomes important for interleukin-1β production. Defects lead to overproduction of interleukin-1β; with development of inflammatory symptoms. Clinical diagnosis is often delayed given overlap of symptoms with connective tissue diseases, vasculitides, and urticarial syndromes including acquired cold urticaria and systemic mastocytosis. Acute phase reactant elevations are common and skin biopsy generally demonstrates a neutrophilic predominant, perivascular inflammatory infiltrate without increase in mast cell numbers or tissue histamine levels. Ice cube test is initially negative but may precipitate symptoms several hours after contact. Genetic sequencing of NLRP3 is available; however, mutations are detected in only half of patients. Therapeutic agents targeting interleukin-1β overproduction including: Anakinra, Rilonacept,
and Canakinumab, provide relief of symptoms and sustained clinical remission in majority of patients. CAPS should be considered in any patient with chronic urticaria as symptoms are often erroneously attributed to a variety of conditions. Recognition of this syndrome and institution of targeted therapies to interleukin-1β can significantly improve quality of life in these patients.

ANGINAL PAIN DUE TO AN ANOMALOUS RIGHT CORONARY ARTERY: AN EXTREMELY RARE AND LIKELY DANGEROUS CONDITION

Christian Hourani MD, Abstract 2

Introduction: Anomalous coronary artery systems arising from a single vessel are extremely rare, found in only 0.03% of catheterized patients. A coronary system in which the right coronary artery (RCA) arises off of the left main trunk (termed L-IIP morphology), is the least common single-vessel system. In a study of 126,600 patients, L-IIP morphology was only present in eleven patients (0.009%). To date, there have been only four case reports of anginal pain in patients with L-IIP morphology who lack underlying coronary artery disease. Case Report: A 57-year-old man with past medical history of hypertension, hyperlipidemia, and tobacco abuse presented to his primary care physician with complaints of episodic chest tightness that radiated to his left arm which was exacerbated by exertion and alleviated with rest. He underwent an outpatient exercise treadmill test which elicited chest pain and ST and T-wave changes on electrocardiogram consistent with ischemia. Coronary angiography revealed a large caliber left main coronary artery that trifurcated into the right coronary artery, circumflex artery, and left anterior descending artery. Despite his positive stress test, no atherosclerotic disease was present throughout the patient’s entire coronary anatomy. The patient underwent cardiac MRI which revealed the RCA coursing between the aorta and the pulmonary trunk. Discussion: Non-atherosclerotic L-IIP coronary anatomy in the presence of signs and symptoms of myocardial ischemia has only been reported in four other patients. Three separate underlying mechanisms for ischemia in these patients have been postulated. Most often in L-IIP anatomy, the RCA courses between the great vessels, as was the case in all four previously described cases. External compression of the RCA as it courses between the aorta and pulmonary artery, leading to insufficient blood flow when myocardial oxygen demand is increased with exertion is one suspected cause. Separately, obstruction caused by engorgement of the aorta and pulmonary artery during exertion may induce dynamic external compression of the RCA leading to exertional angina. Thirdly, the underlying cause may be poor flow caused by the radical angulation necessary for the anomalous RCA to traverse from its left-sided origin to the right side of the heart. Given the rarity of this anomaly, the prognostic significance of L-IIP coronary anatomy remains unclear and is controversial. In the presence of other causes for angina, it is often considered an incidental finding and no further management is pursued. However, an autopsy study that included twenty-one L-IIP patients suggested increased risk of sudden cardiac death in these patients. In three of the four prior described cases in which patients had no other cause for ischemia, surgical re-routing of the RCA has been chosen. In this patient, surgical management will be strongly considered as it may alleviate symptoms and reduce mortality risk.
DYSPNEA FROM COMPLICATIONS OF SAPHENOUS VEIN GRAFT ANEURYSMS
Alexander Hu DO, Abstract 19

Dyspnea from Complications of Saphenous Vein Graft Aneurysms Alexander Hu, D.O. OGME-2, VVMC, Cottonwood, AZ

Significant functional deterioration with severe dyspnea in the setting of stable coronary atherosclerotic heart disease and negative cardiac work up can be a diagnostic challenge. A 69 year old male with an extensive history of atherosclerotic heart disease presented with increasing shortness of breath and deteriorating functional status for three months. Just two months prior, the patient was able to walk 300 yards on flat ground, whereas he now is unable to walk in his house without significant shortness of breath. The patient had classic angina starting in 1987 and continued for more than 15+ yrs even after a 4 vessel bypass. The patient had undergone multiple cardiac catherizations due to the chronicity of his chest symptoms up to 2003 without intervention. Recent history revealed a cardiac catherization in 2007 without need for intervention, multiple cardiolite studies in 2009, 2010 and more recently August 2011, all which showed no clear evidence for ischemia. Other evaluations for anemia, embolic disease and recurrent coronary events turned out to be negative. Consult was then obtained with a pulmonologist, given multiple negative cardiac studies, and as part of the pulmonary work up a CT scan of the chest with findings suggestive of a “4.7cm CABG pseudoaneurysm”. Completed work up did not show any evidence of lung disease. In defining the anatomy of the aneurysms, the most recent cardiac catherization revealed SVG to OM diffusely aneurysmal with poor flow and high grade stenosis at the distal anastamosis. SVG to RCA also diffusely aneurysmal with a filling defect distally that appears to be a clot or plaque also having poor flow characteristics. Left ventriculography showed a mildly enlarged ventricle, moderate diffuse dysfunction and an EF 35%. This progression of left ventricular dysfunction was not thought to be a major contributor to his symptoms. Saphenous vein graft aneurysms are exceedingly rare with complications related to slow blood flow, rupture and embolization. Case reports have shown wide variations in the development of SVG aneurysms even as early as four months post bypass. The etiologies for development of aneurysms include inherent vein graft deficiencies, vessel trauma during harvest and progression of atherosclerotic heart disease with a vein graft subject to arterial pressures. In this patient, the slow blood flow in multiple diffuse saphenous vein grafts are the major contributors to his significant dyspnea. Aortocoronary SVG aneurysms should be part of the differential diagnosis in patients with a medical history which includes coronary arterial bypass grafting regardless of the time interval between intervention and onset of chest symptoms. Recognition of progressive symptoms warrants additional investigation despite extensive past work ups is crucial to appropriate therapy.

WHEN A STROKE IS NOT A STROKE
Imam Imam MD, Abstract 108

Introduction: Patients with the acute onset of hemiplegia represent a medical emergency that requires prompt evaluation and treatment. The administration of thrombolytics within 4.5 hours of onset of clearly defined neurologic symptoms is associated with improved outcomes. It is important, however, to recognize that not all patients presenting with hemiplegia have an ischemic stroke. Conditions
that may mimic a stroke are migraines, seizures and conversion disorders. Case Presentation: 54-year-old Hispanic female with extensive past medical history including coronary artery disease, migraines and a questionable history of 3 previous CVAs presented with atypical chest pain. On day 2, she had an acute sudden onset of left sided weakness of upper and lower extremities, facial drop and slurred speech without aphasia. There was no loss of consciousness, no incontinence or tongue biting. The symptoms suddenly occurred at 3 AM. On examination, she had decreased pinprick and light touch on left face, left facial droop, and decreased strength on the left with normal deep tendon reflexes bilaterally. Head CT revealed no evidence of hemorrhage. Neurology consultation agreed with the administration of thrombolytic therapy for presumed ischemic stroke. MRA/I subsequently showed no signs of new or residual ischemic changes. Prior to this hospitalization she had two similar episodes at outside hospitals. For one of these she received thrombolytic therapy. Given her presentation and strong family history of migraine headaches, relevant genetic markers were sent for familial complex migraine. Discussion: Hemiplegic migraine is a rare type of migraine with aura characterized by recurrent episodes of varying degrees of focal weakness associated with migraine. According to International Headache Society criteria, hemiplegic migraine is diagnosed clinically on the basis of an accurate history, while routine biochemical and hematologic examinations, as well as lumbar puncture and imaging findings, help to exclude other more common disorders. There are two forms of hemiplegic migraine: the familial form and the sporadic form. Familial hemiplegic migraine is divided into two subtypes according to the genetic mutation. The first type, there is a point mutation in the CACNA1A gene on chromosome 19 and second type, there is a mutation in the ATP1A2 gene on chromosome 1. For sporadic hemiplegic migraine, only the first mutation is reported. It is important to recognize that acute hemiplegia may not always represent an ischemic stroke. In fact up to 13% of patient who receive thrombolytics for a stroke do not have a stroke. Because it is extremely difficult, however, to properly diagnose these stroke mimics and because the use of thrombolytics in this population is not associated with adverse outcomes, thrombolytics should not be withheld in these patients.

THE GREAT IMITATOR OF THE SOUTHWEST
Benjamin Johansen DO, Abstract 71

The Great Imitator of the Southwest Authors: Benjamin Johansen DO, Leonor Echevarria MD Institution: Banner Good Samaritan Medical Center Introduction: Disseminated coccidioidomycosis is associated with high mortality. Although coccidioidomycosis is a great imitator, the diagnosis can usually be made readily if high level of suspicion is maintained and appropriate diagnostic testing is performed. We present a case of severe bone involvement with widespread bony lytic lesions that appear as malignancy or multiple myeloma but which is actually disseminated coccidioidomycosis. Case Presentation: A 53 year old immunocompetent African-American male was transferred from an outside hospital with four months of low back pain, weight loss of 50 pounds and fever. Computed tomographic scan of the lumbar spine revealed no acute bony injury. MRI of the lumbar spine revealed extensive scattered bony lesions throughout the cervical, thoracic, lumbar spine, as well as the sacrum and ilium suggestive of
diffuse metastatic disease versus multiple myeloma. The patient was febrile with max temperature 39.4°C and developed pulmonary infiltrates that led to oxygen desaturation and eventual transfer to the ICU. Laboratory workup revealed a positive cocci screen, Hgb 8.2 g/dl, T protein 9.2 g/dl, albumin 2.3 g/dl. Further workup to investigate the protein albumin gap revealed an IgG gammopathy at 3,549 mg/dl, T protein PES 8.7g/dl, and urine protein 30mg/dl. Initial cocci serology by ELISA was IgM positive and confirmed by IDF (Immunodiffusion). Cocci comp fixation titers were <1:2. HIV test was negative. A CT guided biopsy of a lytic lesion in L2 revealed necrotizing granulomas and multiple spherules consistent with disseminated coccidioidomycosis. He was started on empiric fluconazole 800 mg daily. Due to transaminitis and severe disease, the patient was switched to lipid formulation of amphotericin, with good response. A month after admission Cocci comp fixation was >1:256. Before discharge he developed a rash due to the fluconazole and was successfully transitioned toitraconazole and tolerated the medication well. Discussion: Less than one percent of pulmonary cocci disseminates. African-Americans are ten times more likely to have dissemination occur. One of the manifestations of coccidioidomycosis can be lytic bone lesions. At initial presentation low levels of complement fixation titers may be misleading. This case emphasizes the importance of keeping coccidioidomycosis in the differential especially among African-American populations as it can present in ways that are least expected.

QTC PROLONGATION IN IL2 CHEMOTHERAPY
Christine Klassen MD, Abstract 7

BACKGROUND: Interleukin-2 (IL-2) is an immunologic chemotherapy that is approved for the treatment of stage IV renal cell carcinoma (RCC) and malignant melanoma (MM). IL-2 is known to be cardio-toxic, with initial Phase I/II trials showing 64% of patients with hypotension, 17% with supra-ventricular arrhythmias, and 1% with potentially lethal ventricular tachycardia (VT). QTc prolongation has not previously been noted in IL-2 therapy, but is a known risk factor for VT. Current FDA guidelines recommend further evaluation of any drug that demonstrates prolongation of the QTc interval greater than 10 msec. We evaluated QTc interval prolongation in patients treated with IL-2, and whether QTc prolongation correlates with the development of cardiac arrhythmias. HYPOTHESIS: IL-2 chemotherapy induces a prolongation of the QTc interval, and patients with significantly prolonged QTc (>50msec) have a higher risk for developing cardiac arrhythmias during therapy. SPECIFIC AIMS: To determine average QTc prolongation of patients receiving IL2 chemotherapy and any correlation with development of arrhythmia. METHODS: We conducted a retrospective review of patients receiving IL-2 for stage IV RCC or MM at the Mayo Clinic-Arizona or Mayo Clinic-Jacksonville from January 2007 until May 2012. We evaluated QTc interval on a daily basis based on ECGs and rhythm strips, interrogated all other medications administered for potential QT prolongation, and adverse events including hypotension, oliguria, metabolic acidosis, acute kidney injury, increased bilirubin, or altered mental status as well as dysrhythmia or elevated cardiac enzymes. RESULTS: A total of 16 patients underwent a total of 39 cycles of IL-
2 with an average of 7.1 doses per cycle. The average baseline QTc interval was 415 msec. The average change was 52.6 msec, with 76% of patients experiencing QTc prolongation >10msec and 51% experiencing a prolongation of >50 msec. 20% of patients experienced cardiac arrhythmia, all supra-ventricular. Two patients developed elevated cardiac enzymes requiring cardiovascular consultation. The average QTc of those experiencing arrhythmia was 55.8 msec, which was not statistically significant compared to an average of 47 msec in those without arrhythmia. No other medication interactions were evident.

DISCUSSION: QT prolongation with IL-2 has not been previously reported. We did not see any correlation between QTc prolongation and cardiac arrhythmias; however no ventricular arrhythmias were seen. This may reflect our small sample size or careful patient selection. Nevertheless, the data support studying QTc in larger cohorts of IL-2 patients to investigate if daily QT tracking or excluding patients with an elevated baseline QTc can decrease morbidity and mortality from IL-2.

CONCLUSION: There is significant QTc prolongation seen in our patient population. However, because of the small number of patients included, there were no incidences of ventricular arrhythmia and therefore a significant correlation could not be established.

PSEUDOMYXOMA PERITONEI: AN UNCOMMON MUCOUS PRODUCING ABDOMINAL TUMOR

Donald Lefevre DO, Abstract 60

The purpose of this presentation is to discuss a case of Pseudomyxoma peritonei – a relatively uncommon finding of abdominal mucous produced by appendiceal adenocarcinoma cells, the current treatments, and possible prognoses. A 56 year old Hispanic male presented with an extensive ascites type of abdominal distention and diffuse pain, described as increasing over the previous year. Paracentesis was unsuccessful, and CT displayed several suprarenal cysts and extensive ascites. Exploratory laparotomy was performed, removing cystic material and copious mucous from the abdominal cavity, which were consistent with the finding of Pseudomyxoma peritonei (PMP). Pathology reports were consistent with appendiceal mucinous adenocarcinoma and the patient was referred to oncology for further assessment and treatment. Accepted standards of treatment generally include debulking or cytoreductive surgery, followed by either heated intraperitoneal chemotherapy, or continued monitoring which the patient has currently selected.

ADDITIONAL CONSIDERATIONS IN THE WORKUP OF THROMBOPHILIA

Keri Maher DO, Abstract 69

Primary Myelofibrosis (PMF) is one of the three recognized (along with essential thrombocytosis and polycythemia vera) BCR-ABL negative chronic myeloproliferative neoplasms. This case report describes a 64 year old Hispanic female presenting with abdominal pain and melena, found to have multiple thromboses including: right atrial, portal, splenic, and superior mesenteric veins. Initial hypercoagulability investigation was significant for heterozygosity of prothrombin gene mutation. However, additional workup ultimately revealed JAK2 V617F mutation-positive myelofibrosis. Since its description in 2005, the JAK2 V617F mutation has received a great deal of attention as a potential pathophysiologic mechanism for the development of myeloproliferative neoplasms.
However, rates of positivity in confirmed cases of myeloproliferative neoplasms range from 23-97%, depending on underlying sub-type, with approximately 30-50% of patients with myelofibrosis being JAK2 V617F positive. Moreover, retrospective analyses have discovered patients presenting with splanchnic neoplasms (as described in the case) are more likely to be positive for the mutation than individuals presenting with thrombus in other areas (such as DVT’s). Interestingly, other known risk factors for thrombosis are not infrequently uncovered in these patients as well, signifying that they may be concomitant or incidental and not necessarily the sole cause of a patient’s apparent thrombophilia, further underscoring the need for complete workup of those presenting with abdominal thrombosis which may include JAK2 V617F screening as well as bone marrow biopsy.

A CLINICAL DILEMMA IN A PATIENT WITH CUSHING’S DISEASE
Varun Mehta MD, Abstract 25

Introduction: Hypopituitarism is a rare condition with a reported incidence of 4 cases per 100,000 individuals. Most cases are due to pituitary tumors or their treatment such as surgery or radiation. The increasing use of hormone assays and imaging has led to greater detection of pituitary tumors requiring some form of intervention. While prolactinomas show an impressive response to medical management, tumors secreting other hormones are more likely to require surgical intervention. Case Report: A 19 year old female presented with amenorrhea, acne and weight gain. She was diagnosed as having Cushing’s disease based on an elevated 24 hour urinary free cortisol, elevated ACTH and marked reduction in serum cortisol on the dexamethasone suppression test. MRI brain showed a 5x6 mm, left sided lesion compatible with a pituitary microadenoma. She underwent an endoscopic, endonasal transsphenoidal exploration of her pituitary but a definite adenoma could not be identified and a left hemihypophysectomy was performed. The patient continued to be symptomatic and had persistently elevated cortisol biomarkers. It is likely that the focus of ACTH production lies in the right pituitary which would need to be resected resulting in a state of panhypopituitarism. The patient is unsure if she would desire children at some point in the future. Discussion: Untreated Cushing’s disease has many associated comorbidities and is often fatal. In the absence of any reliable medical therapy for the same, surgical resection of the pituitary remains the only viable option. However, the timing of such a surgery is of paramount importance. There are few studies that elucidate fertility outcomes in women with panhypopituitarism. One such study showed that while fertility treatment can induce ovulation in about 95% of such women, pregnancy is achieved in about 47% of women with about 42% of them progressing to a live birth. Delaying surgery until after childbearing can leave patients susceptible to complications of unchecked Cushing’s disease.

THE CLINICAL SPECIFICITY OF THE ENZYME IMMUNOASSAY TEST FOR COCCIDIOIDOMYCOSIS VARIES ACCORDING TO THE REASON FOR ITS PERFORMANCE
Neil Mendoza MD, Abstract 78

Introduction: The diagnosis of coccidioidomycosis relies heavily on the results of serological tests in addition to clinical history, physical exam, and radiographic findings. Use of the enzyme immunoassay (EIA)
has increased because it is objective, rapidly performed, and does not require referral to a reference laboratory. The interpretation of immunoglobulin M (IgM) reactivity by EIA in the absence of immunoglobulin G reactivity has been problematic. Purpose of study: We sought to identify situations where IgM only EIA reactivity is more likely to be clinically specific for coccidioidal infection. Methods: Patients were identified by reviewing the records of all patients with reactive coccidioidal serology performed at our institution from January 1, 2004 through December 31, 2008. All patients with IgM only EIA reactivity were compiled into a data set. The records of such patients were reviewed for demographics, symptoms at the time of the serological testing, reason for the serological test, comorbid illnesses, details of the suspected coccidioidal illness, and the strength of the coccidioidal diagnosis ranging from confirmed to unconfirmed. Results: From January 1, 2004, through December 31, 2008, 1163 patients had positive coccidioidal serology. Of these 1163 patients, 1117 patients (96%) had EIA serology performed with multiple instances of testing in some patients, which resulted in 2950 pairs of IgM and IgG by EIA. 102 (9% of the 1117) patients had EIA IgM only reactivity, of which 78% had positive serological findings by other methods or had positive culture or histology. For all patients who initially had EIA IgM only reactivity, 13% later seroconverted to IgG and IgM reactivity. In the subgroup of patients in whom the EIA test was performed to evaluate symptomatic illness, 54 (90%) of 60 had probable or confirmed coccidioidal infection. When the EIA test was performed for screening purposes, 13 (45%) of 29 patients had coccidioidal infection. Conclusion: The use of the EIA for screening asymptomatic persons was associated with unconfirmable results in roughly half of those tested. Although the majority of patients in our study with isolated IgM reactivity by EIA had probable or confirmed coccidioidomycosis, this result must be interpreted with caution in asymptomatic patients.

EMPHYSEMATOUS CYSTITIS IN AN ALCOHOLIC WOMAN
Sadia Moinuddin MD, Abstract 106

Introduction Emphysematous Cystitis (EC) is a potentially life threatening condition characterized by air in the bladder wall caused by gas producing microorganisms. EC is most common in middle aged women with diabetes and immunosuppression. Due to its subtle presentation, a high index of suspicion is required as this disease can rapidly progress to bladder necrosis, emphysematous pyelonephritis, bacteremia, and death. Case We present a 47 year old female with emphysematous cystitis. Her past medical history was significant for alcoholic liver disease, asthma, and reflux esophagitis. She complained of diffuse abdominal pain, jaundice, and hematuria, but denied weight loss, fevers, chills, or dysuria. On physical examination, patient was afebrile and tachycardic (pulse 111bpm) with scleral icterus and jaundiced skin. Her abdomen was soft and distended with mild suprapubic tenderness. Initial laboratory data showed normal complete blood count and normal glucose; AST 814 U/L (5-34 U/L), ammonia level 82 umol/l (3-35 umol/l), and total bilirubin 15.2mg/dl (0.2-1.2 mg/dl). Urinalysis was leukocyte esterase positive with greater than fifty white blood cells. Urine and blood cultures were positive for Escherichia Coli. She was admitted to the intensive care unit for sepsis and alcoholic encephalopathy and Pipercillin/tazobactam...
3.375g IV Q 6hours was empirically started. When patient failed to improve, a subsequent computed tomography of the pelvis was ordered which showed emphysematous cystitis with surrounding extraperitoneal air and hemorrhage within the posterior bladder. Patient was started on continuous bladder irrigation. Her hospital course was further complicated by an acute abdomen with small bowel obstruction and pneumoperitoneum. General surgery opted for non surgical conservative management given patient’s comorbidities. The family decided to withdraw care. Conclusion EC is not often diagnosed by a routine approach due to its subtle presentation as it can range from incidental diagnosis on abdominal imaging, abdominal pain, or severe sepsis. Risk factors include female sex, poorly controlled diabetes, recurrent UTI, indwelling urinary bladder catheters, and bladder outlet obstruction. Diagnosis is usually made by abdominal imaging. Overall death rate of EC is 7%. In non-diabetic patients, impaired host response with impaired vascular supply may be responsible for the development of EC. It has only been reported in two non diabetic alcoholics in two previous case reports. We should have tailored our diagnostic approach to consider EC in the differential diagnosis of hematuria and abdominal pain in our female patient with alcoholic liver disease. Hence, we should have consulted urology course and started continuous bladder irrigation and perhaps could have prevented acute abdomen. Earlier detection may have altered the disease course and prevented this fatal outcome.

Second Place – Associate Case Report Poster:

A RARE INTESTINAL TRANSFORMATION
Anju Nair MD, Abstract 64

Introduction: Richter’s Transformation (RT) is characterized by the development of high-grade diffuse large B-cell lymphoma (DLBCL) in a patient with chronic lymphocytic leukemia (CLL) or small lymphocytic lymphoma. By acquiring new cytogenic abnormalities, existing CLL cells are able to histologically transform into DLBCL cells. RT occurs in 2-9% of patients with CLL. Most commonly, the transformation arises in lymph nodes, and fever, weight loss, and adenopathy are presenting symptoms. Primary intestinal RT is a rarely reported complication of CLL. We present a unique case of small intestinal DLBCL that transformed from CLL. Case Presentation: A 72-year-old female with a history of CLL in remission, presented to the emergency department with abdominal pain. The patient reported a four-day history of progressive symptoms including constipation, abdominal distension, the recent onset of nausea and mild, dull, generalized abdominal pain. On physical examination, the patient demonstrated normal bowel sounds, diffuse abdominal tenderness without guarding or rigidity, and guiac positive stool, with no palpable lymph nodes or hepato-splenomegaly. A CT with oral and IV contrast of the abdomen revealed a pneumo-peritoneum. The Surgery service was consulted and patient underwent an emergent exploratory laparotomy. The Surgeons found three areas of gross abnormality within the proximal ileum. Two areas of gross involvement were perforated, and one area of perforation showed transmural necrosis. The third area of involvement showed no signs of perforation. Surgeons resected a 10.5 X 10.0 X 5.0 cm region of small bowel and sent it for biopsy. The incision was closed after
re-anastomosis of small intestine. Surgical pathology reported DLBCL, arising from CLL. The biopsied sample revealed diffuse infiltration with large atypical lymphoid cells with prominent nucleoli consistent with DLBCL with the presence of many CLL cells. After an uneventful post operative course, the patient was treated for DLBCL with R-CHOP (rituximab-cyclophosphamide, doxorubicin, vincristine, and prednisone) therapy. She is currently in remission. Conclusion: This case highlights the rare clinical findings of intestinal perforation as a presenting complication of RT. CLL typically transforms into an aggressive lymphoma, usually DLBCL, within 23 to 47 months after the initial CLL diagnosis. No genetic or environmental factors have been confirmed to predict whether CLL will transform. Extraneous involvement of RT is a rare event, as seen in this case. Intestinal RT invades the digestive mucosa and so can appear clinically as recurrent gastric ulcer disease, upper or lower gastrointestinal bleeding, intestinal obstruction or rarely acute perforation. It is diagnosed with histopathology of the digestive mucosa. Treatment involves surgical resection of the lymphoma and chemotherapy, and oftentimes may even lead to remission. Primary digestive RT has a better prognosis than nodal RT. In cases that have been studied, median survival after chemotherapy is 22 months, ranging from 5-48 months.

CORRECTED ANION GAP (CAG) AND SERUM BICARBONATE, IMPROVED GUIDELINES FOR ACID-BASE/KETOSIS MANAGEMENT IN DIABETIC KETOACIDOSIS (DKA
Ali Omranian MD, Abstract 20

Background: An average of five liters of saline solution is utilized in early resuscitation of DKA, which leads to hyperchloremia and dilution of serum albumin. Accordingly, the anion gap (AG), to guide correction of ketoacidosis, is altered. Study Aim: We sought to determine if serum bicarbonate, together with closure of the cAG (anion gap corrected for albumin) are better predictors for correction of ketosis. Hypothesis: The cAG provides a more accurate assessment of acid-base status during acute management of DKA. Methods: We reviewed 60 consecutive patients with DKA. Demographics were recorded, together with metabolic data on admission (T0) and at termination of insulin infusion (T1). Values of AG [Sodium – (Chloride + Bicarbonate)] and cAG [AG + 2.5 (4.4-Albumin g/dL) at both T0 and T1 were compared. Results: This study included 37 males (62%) and 23 females (38%) with overall mean age of 41 years (19-75 y). Mean values at T0 were: glucose 619±337 mg/dL; sodium 134±7.2 mEq/L; bicarbonate 10.3±5.6 mEq/L; chloride 99±8.4 mEq/L; albumin 4.0±1.16 g/dL; pH 7.2±0.15; AG 25.2±7.6 mEq/L. Mean values at T1 were: glucose 167±138 mg/dL, sodium 138±3.5 mEq/L; bicarbonate 19.5±3.5 mEq/L; chloride 112±5.2 mEq/L; albumin 2.9±0.65 g/dL; AG 6.8±3.3 mEq/L; cAG 10.8±3.2 mEq/L. During a mean interval of 23 hours (T0 to T1) a mean of 4.8 liters of saline solution were infused. At T1 40/60 patients (66%) were hypoalbuminemic (<3.4 g/dL) and 47/60 (78%) were hyperchloremic (Cl/Na>0.79). Although AG was corrected (<12 mEq/L) for 100% of patients at T1, 15 patients (25%) had cAG >12 mEq/L (p value <0.05), and for 18 patients (30%) bicarbonate was <18 mEq/L, consistent with incomplete repair of acidosis. Conclusions: Hypoalbuminemia and hyperchloremia commonly develop during DKA management. We suggest that cAG and bicarbonate values be
used to assess acid-base and ketosis disturbance.

LEGIONELLA SKIN INFECTION IN BONE MARROW TRANSPLANT: CASE REPORT AND REVIEW OF THE MEDICAL LITERATURE

Leslie Padrnos MD, Abstract 79

Background: Hematopoietic stem cell transplantation (SCT) recipients can have a variety of skin abnormalities caused by medication side effects, graft versus host disease, and infection. Correct identification of the etiology is imperative as treatment can vary from steroids to antimicrobial therapy. We recently identified cutaneous legionella infection in a SCT patient, and due to its unusual occurrence, we sought to review and summarize clinical characteristics of this unusual cause of skin infection. Methods: The patient’s electronic medical record was reviewed. A comprehensive review of the pertinent literature on OVID Medline and PUBMED was conducted, using the search terms “cutaneous” or “soft tissue” or “stem cell transplant” and “legionella”. Relevant references identified in the initial articles identified were also examined. Results: Case Report: A 27 year old women diagnosed with pre-B cell acute lymphoblastic leukemia underwent a mismatched double cord blood allogeneic stem cell transplantation in May 2011. On post SCT day +172, she was hospitalized with right lower extremity pain and erythematous subcutaneous nodules. She was empirically treated with broad spectrum antibiotics. She developed hypoxia and underwent bronchoscopy, the cultures of which were negative. Her skin lesions progressed, and a repeat skin biopsy demonstrated subcutaneous neutrophilic microabscesses and cultures grew Legionella pneumophila. Azithromycin was added, but she became increasingly hypoxic and died on post SCT day +210. A literature search revealed a total of 16 published cases of skin or soft tissue infections due to Legionella species dating back to 1980. Immunosuppression was present in 6 of 16, and 3 had a predisposing condition to abscess formation. Skin manifestations included erythematos macular rashes (n=7), abscesses (n=3) and one had rapidly expanding erythema following thoracentesis. 10 of 16 cases had concurrent respiratory symptoms; 2 cases had asymptomatic bilateral pulmonary infiltrates. The etiologic agent was identified from surgical exploration of an abscess or progressive cellulitis in 9 cases, whereas the other 7 described skin changes in patients with concomitant Legionella pulmonary infection identified by urinary antigen or sputum culture without tissue confirmation of the extrapulmonary manifestation; for such cases, the rash was attributed to Legionella when the lung and skin findings improved following antibiotic administration. Nine cases utilized a macrolide as sole treatment, two employed fluoroquinolones, and 4 used a combination of macrolide and fluoroquinolone treatment. For 11 cases, the skin eruptions improved with antibiotic treatment, and one case improved after the infected hip prosthesis was removed. Three patients died from multiorgan system failure. Conclusion: Legionella skin and soft tissue infections are rare, and may manifest in both immunosuppressed or immunocompetent patients, with or without respiratory complaints. A review of the literature revealed varied presentations and criteria for diagnosis, but reinforced the importance of an accurate diagnosis to ensure appropriate treatment.
EXTENSIVE PACEMAKER WIRE VEGETATION WITH AN UNUSUAL BACTERIA
Tanmay Patwa MD, Abstract 10

INTRODUCTION Infection of intra-cardiac devices is a rare complication, unlike infective endocarditis which is fairly common. The HACEK group of organisms is known to cause 5-10% of bacterial endocarditis however their contribution to pacemaker or defibrillator wire infections is under-reported. CASE 45 years old male with history of Hepatitis C from intravenous drug abuse, permanent pacemaker implantation 8 years ago for Sick Sinus Syndrome (SSS) presented to the ED with fevers, pleuritic chest pain and bilateral leg edema of 10 days duration. Patient was hypotensive, tachycardic and found to have a new onset 2/6 systolic murmur at the mitral area. Patient was started on empiric broad spectrum antibiotics and transferred to the critical care unit for septic shock. Blood cultures drawn in ED on admission were positive for pan-sensitive Haemophilus para-influenzae. Trans-esophageal echocardiogram showed extensive vegetations involving both pacemaker wires and possibly the tricuspid valve as well. Cardiac surgery was involved for immediate removal of the patient’s pacemaker and for trans-venous temporary pacemaker support. His antibiotic regimen was tailored to the sensitivity panel and ceftriaxone was started. Subsequent surveillance blood cultures were negative for bacterial growth. Permanent pacemaker re-implantation was done after blood cultures had been negative for 72 hours and patient was discharged on ceftriaxone intravenous therapy for 6 weeks. DISCUSSION With the recent increased use of implanted cardiac devices, the rate of intra-cardiac lead infection is expected to rise in coming years. While Staphylococcus aureus are the major organisms, Haemophilus parainfluenzae and the rest of the bacteria in the HACEK family are rarer. Haemophilus para-influenzae, a gram negative cocccobacilli, is a normal oral and upper respiratory tract commensal organism that can cause pacemaker wire vegetation from blood stream infections after oral or airway instrumentation. Transesophageal echocardiogram is still the gold standard for detection of intra-cardiac vegetations. Infective foci control with pacemaker wire removal and re-implantation at a different site after negative surveillance blood cultures is standard therapy. HACEK organisms are usually sensitive to third generation cephalosporins and floroquinolones, however detecting them on blood cultures might require more than 6 days of incubation, during which time the physician has to carefully cover these bugs with empiric antibiotics. References: PAI, R. (2004). Pacemaker lead infection secondary to haemophilus parainfluenza. PACING AND CLINICAL ELECTROPHYSIOLOGY, 27(7), 1008-10.

SUPINE RESPIRATORY FUNCTION TESTING VERSUS PERCUTANEOUS NOCTURNAL OXIMETRY AS PREDICTORS OF SURVIVAL IN AMYOTROPHIC LATERAL SCLEROSIS
Emanuel Pauwels MD, Abstract 122

Patients with Amyotrophic Lateral Sclerosis (ALS) develop progressive weakness of respiratory muscles and death is generally caused by respiratory failure. Clinical use of respiratory function studies (RFTs) and, more recently, percutaneous nocturnal oximetry (PNO) have been proposed to guide disease management and to assess prognosis in these patients. The supine challenge, measuring the change in forced vital capacity (FVC) between upright and supine positions, is a sensitive test
to detect diaphragmatic weakness. The objective of this study was to compare survival in ALS patients identified to have a change in upright to supine FVC of > 10% with survival in a cohort of patients with abnormal PNO (mean SaO2 of < 90%). A retrospective chart review of sixty-one patients diagnosed with ALS was performed from 2004 until 2010. All patients underwent both supine respiratory function testing and percutaneous nocturnal oximetry. Survival was calculated from onset of a decline in supine FVC of greater than -10% compared with upright FVC, or the development of mean PNO saturation less than 90%. Ten patients were identified with a mean overnight oxygen saturation < 90% while thirty-three patients had a supine change of >-10%. Mean three-year survival in the PNO group was 16.9 months (508 days) compared with 8 months (241 days) in patients with abnormal supine FVC (p < 0.02). These findings suggest changes in supine FVC are more predictive of limited survival in patients with ALS than the presence of reduced saturations on PNO and may be more useful in the routine management of these patients.

CONTROLLING MULTIDRUG-RESISTANT ACINETOBACTER BAUMANNII IN A LONG-TERM ACUTE CARE HOSPITAL
Iram Qureshi DO, Abstract 83

Controlling Multidrug-resistant Acinetobacter baumannii in a long-term acute care hospital Iram Qureshi, DO, Lee Steininger, RN, CIC Background: Between December 2008 to May 2010 there were 63 cases of multidrug-resistant Acinetobacter baumannii at Kindred Hospital, a long-term acute care hospital in Tucson, AZ. We describe the steps taken to terminate the outbreak. Methods: The infection prevention team at Kindred identified a cluster of 8 multidrug-resistant Acinetobacter cases in April of 2009. Lab reports and admissions were reviewed and an index patient was indentified that was admitted to the hospital in December of 2008 and who remained at the facility for over 60 days. Mode of transmission was discussed by the team but a specific source of transmission was not identified. New guidelines for contact precautions were issued to staff to prevent further spread. When this failed to eradicate the organism, a bleach out of the entire facility was initiated in May 2010. All shared equipment was bleached, including but not limited to pumps, vents, IV poles, medication boxes, portable x-ray machine, bronchoscopy and endoscopy carts and towers, and pharmacy and wound care carts. Results: There were 3 main cluster of outbreaks throughout the 18 month period and despite a through review of data, a specific source of spread could not be found. The first cluster was identified as 8 cases between April and May of 2009, the second cluster was 13 patients between August and September 2009, and the largest outbreak was between February and May 2010 with 24 patients. Universal contact precautions protocol was reviewed and letters were sent out to the staff to educate them on the importance of adhering to the protocol. Each patient was issued their own BP cuff and stethoscope that was kept in the patient’s room for the duration of their stay and then discarded. However, these measures did not control the spread of Acinetobacter amongst the inhabitants of Kindred. Eventually, it was found that a through cleaning of the entire building and all the equipment with bleach was the only way to control the spread of this bacteria. Conclusion: Multidrug-resistant Acinetobacter baumannii has been a growing problem around the country. Bleach is the
most effective way of eradicating Acinetobacter baumannii from a facility.

THE PERFECT STORM
Shivani Ruben MD, Abstract 128

Introduction: A hemothorax is defined as blood within the pleural cavity. It is seen most commonly as the consequence of trauma, but also can occur rarely in a variety of other clinical situations. Prognosis is very poor as it implies grave illness, causing both respiratory and hemodynamic compromise. Here, we discuss the case of a perfect storm - the development of a hemothorax in a patient with multiple predisposing factors for this unusual complication. Case Presentation: A 68 year-old male smoker with a history of prostate cancer S/P prostatectomy and coronary artery disease S/P PCI, on both aspirin and clopidogrel, presented to the emergency department with chest pain and shortness of breath. He had been experiencing these symptoms for approximately 1 month, and had failed two outpatient courses of antibiotics for a presumed pneumonia. In the emergency department, a CT angiogram showed extensive bilateral pulmonary emboli as well as multifocal airspace consolidation. Per radiology interpretation, the consolidation was consistent with an “infectious process.” Pulmonary service was consulted for antibiotic-resistant pulmonary infiltrates, and he underwent a bronchoalveolar lavage, which was negative for any fungal, bacterial, or malignant cells. He was discharged on enoxaparin and warfarin for the pulmonary emboli, and advised to follow up with an outpatient pulmonologist for further diagnostic evaluation. The INR, checked 8 days after discharge, and was 7.7. He was instructed to stop both enoxaparin and warfarin, and to establish care with a PCP. At his PCP’s office the next day, he was found to have significant shortness of breath and tachycardia. He was sent to the ED, where imaging revealed persistent extensive bilateral pulmonary artery emboli and a new large left sided pleural effusion. There was near complete consolidation of the left lower lobe, and the multifocal areas of airspace consolidation were now interpreted by radiology as consistent with “malignant involvement.” A diagnostic thoracentesis was performed, which established the presence of a hemothorax. Cytologic analysis revealed adenocarcinoma; further cell marker testing was consistent with a primary lung tumor. Due to the terminal nature of his disease, the patient chose palliative care, and was admitted to Hospice. Discussion: A hemothorax, defined as hemorrhagic fluid in the pleural space with a hematocrit greater than 50%, usually occurs because of direct trauma. However, although extremely rare it can also be seen with lung malignancy, supratherapeutic anticoagulation, or with pulmonary emboli, particularly in the setting of pulmonary infarcts. Our patient had malignancy, VTE, and was on both antiplatelet as well as anticoagulation therapy with a supratherapeutic INR. A perfect storm had been created, and the patient suffered this rare complication as a result.

BACK PAIN IN A 28Y/O MALE: WHEN YOU HEAR HOOF BEATS IT MAY WELL BE ZEBRA
Victor Sanders MD, Abstract 65

Introduction: Multiple myeloma (MM) is a neoplastic process characterized by the proliferation of a single clone of plasma cells. Myeloma has an incidence of 4 to 5 per 100,000 and the mean age at diagnosis is 66. Although more cases are being reported in younger adults, only 2% of cases occur in
individuals younger than 40. Case Presentation: A 28 y/o male with a history of type 2 diabetes presented with intermittent lower back pain for 8 months which progressively worsened one week prior to admission. The pain was sharp, originated from beneath his scapulae, and was not alleviated or aggravated by any specific factors. He additionally noted shortness of breath and chest discomfort for the past 2 days. Review of systems revealed fatigue, weight loss, generalized weakness and night sweats. He denied fevers, lower extremity edema, bowel or bladder incontinence or paresthesias. Labs and x-rays obtained at an outside facility one week previously were “normal.” On examination, the patient appeared in mild distress. Vital signs were remarkable for a temperature of 98.6, BP 144/96mmHg, pulse 122, respiratory rate 22 and pulse oximetry 88% on room air. He had significant midline tenderness to palpation at T7 to T10 and L1-L2. Neurological exam was non-focal and the remainder of the physical exam was unremarkable. A lumbar x-ray showed T8 and L3 compression fractures and subsequent MRI of the thoracic and lumbar spine showed a soft-tissue mass at the L2 spinous process with possible marrow infiltration and severe spinal stenosis with thecal sac compression. Initial blood samples could not be analyzed due to high viscosity but after plasmapheresis, they revealed calcium 8.6, serum IgG 9287, LDH 253, haptoglobin 134, beta-2 microglobulin 3 and total protein 13.9 with albumin 1.7. He had a normocytic anemia with hemoglobin of 9.4, serum protein electrophoresis showed a 61.3% M component and serum immunoelectrophoresis showed an IgG kappa monoclonal protein. Bone marrow biopsy showed 51% plasma cells and 100% cellularity. The peripheral smear showed rouleaux. Fluorescent in situ hybridization (FISH) was positive for t(11;14). FNA biopsy of the L2 mass revealed sheets of kappa light chain restricted CD138(+) plasma cells; a diagnosis of MM was made. He was treated with radiation, pamidronate, dexamethasone and bortezomib and is currently undergoing evaluation for stem cell transplant. The patient has an International Staging System score of 2, translating into a median survival time of 44 months. Discussion: Although still extremely uncommon, this case highlights the importance of recognizing that MM is increasingly being recognized in younger adults. Younger patients exhibit more favorable features and less frequent adverse prognostic factors, translating into a significantly better survival compared to older patients. Therefore early awareness and treatment may be warranted for younger patients.

**ANOTHER GREAT MIMICKER: A CASE OF BILATERAL ATYPICAL PNEUMONIA**

**Jeff Schenk DO, Abstract 129**

Another Great Mimicker: A case of Bilateral atypical Pneumonia Jeffrey Schenk D.O, David Byun D.O Midwestern University, Verde Valley Medical Center Lungs are limited in the way they can respond to insults, and it can be very difficult to determine the exact cause based on radiologic presentation. Here I present a case of atypical lung infection with pneumothorax 38 year old Hispanic male presents with worsening shortness of breath and spontaneous pneumothorax. Patient reports having intermittent shortness of breath for 1 year. The shortness of breath became progressively worse three days prior to arrival in the ER. Patient also had pleuritic, left sided severe chest pain for the last 3 days that has been worsening as well. There is no radiation of the pain and no association with activity or
rest. Patient denies cough or hemoptysis, night sweats, fevers, chills, or prior TB infection. Patient is a native of Mexico and travels back and forth. He reports being exposed to a person with known TB infection. The Patient worked as a stone mason for many years and now manages a trailer park. No other past medical history, surgical history or prior medications. Initially, a chest x-ray showed a pneumothorax and extensive interstitial and nodular opacities suspicious of military Tuberculosis. A chest tube was placed with improvement of the pneumothorax. The patient had a negative PPD test followed by three negative AFB tests. Sputum cultures also remained negative. Despite chest tube being in place, pneumothorax began to worsen and a 28-French thoracostomy tube was placed. Pneumothorax continued with slow improvements. HIV, alpha I antitrypsin, ACE enzyme, and even Cocci IgG were all negative. A bronchoscopy was performed and tissue biopsy finally revealed Hyalinized granuloma with organisms consistent with coccidioides. Patient was eventually discharged on diflucan. Coccidioidomycosis is a fungal disease caused by Coccidioides immitis or C. posadasii. It resides in the soil the desert southwest and its spores are spread when the soil is disturbed. A person is infected by breathing in the spores. Usually the patient presents with symptoms similar to a community acquired pneumonia. Radiologic findings typically show unilateral infiltrate, hilar adenopathy, or parapneumonic effusion. Rarely thin-walled pulmonary cavities or nodules can be seen. This case shows the wide variability of response to cocci. The findings on the chest x-ray were not typical of cocci and the patient was having recurrent pneumothoraces. The case also shows the unreliability of negative serologic testing for Cocci. As we see many times in medicine things are not always as they seem and have to be further investigated.

DYSPHAGIA
Ryan Sefcik DO, Abstract 39

An atypical presentation of esophageal dysmotility. Chest pain is among the most common symptoms that patients present with in the emergency department. Physicians probe further into the patients history to decide whether this is an atypical or typical cardiac chest pain to risk stratify. When a 46 year-old male presents with intermittent retro-sternal chest pain, nausea, and pre-syncope one must wonder if this is truly cardiac chest pain. This patient underwent a cardiac workup that was found to be negative and a full gastrointestinal evaluation that had positive findings. A barium swallow was obtained first which showed no positive findings and appeared benign. Patient was placed on Dexilant 60 mg PO Q daily which mostly resolved the majority of symptoms, however they continued on mildly and further workup was completed. A high-resolution esophageal motility study showed an upper esophageal sphincter mean basal pressure of 14.6 mmHg, which was well below the normal range of 34-104. This patient is currently being worked up for primary vs secondary dysmotility and pending results of videofluoroscopy and neurologic evaluation for parkinsonism. All results will be available and included in poster presentation of esophageal dysmotility: an atypical presentation. Very commonly, chest pain can be non-cardiac and physicians must be able to diagnose these conditions beyond the cardiac standpoint. Atypical presentations of gastrointestinal and musculoskeletal conditions that include chest pain are very important in the patient evaluation process.
A NOT SO SILENT CASE OF SILENT MYOCARDIAL ISCHEMIA
Glenn Stokken MD, Abstract 16

Silent Myocardial Ischemia (SMI) is defined as the absence of angina or its equivalent in association with inducible EKG changes (on stress or daily life), reversible wall-motion abnormality on stress echo, or nuclear perfusion defects. SMI is an independent risk for morbidity and mortality, but the best way to screen in a cost effective manner remains unclear. A 66M with a PMH of poorly controlled HTN and tobacco use presented to the ED asymptomatically after a routine Veteran’s Affairs health-maintenance EKG demonstrated 7-8mm ST Elevation anterior leads. On the way to his appointment he states he ran up the stairs and felt mild shortness of breath for 2 minutes, but denied chest pain, palpitations, recent infections, or dizziness. He has no history of angina and no exercise intolerance. Cardiac Risk factors include HTN and tobacco use. He is not obese, not diabetic, has no family history of early CAD, not obese, no history of CVA or other vascular disease, no Alcohol abuse, and no autoimmune disease or chronic inflammation. Physical exam was normal, including cardiac exam. Review of EKG in 2009 revealed Normal Sinus Rhythm, no Q waves or ischemic changes. Emergency cardiac catheterization showed 100% Occlusion of LAD, TIMI Flow=0, Fresh Thrombus staining with contrast, and Lesion Length of 54mm. Three 2.5mm Zotarolimus (Endeavor) Stents (18, 12, and 30mm length) were placed with good angiographic result. Echocardiogram revealed LVEF: 40-45% Dyskinetic Apex, Hypokinetic Anterior Wall, No LV Aneurysm or Apical Hypertrophy. The patient was discharged on dual antiplatelet therapy, anti-anginal medications, and anti-hypertensive medications. At 6 week follow-up, our patient’s course was complicated by LV aneurysm diagnosed at a 1 month post-MI. Again, this was not present on initial echo. His LVEF had decreased to 30-35% at 6 weeks from 40-45%. His NYHA Heart Failure Status was Class 2 while he had no exercise intolerance in the past. This case brings to light that screening for silent ischemia may indeed have prevented this patient’s MI and resultant heart failure. Old and new data indicates silent ischemia not only is an independent risk factor for CV morbidity and mortality, increasing risk ~3-4x, but PCI is superior to medical management. Most notable is the ACIP trial. However this data is from patients with known CAD. New Data (April 2012, American Journal of Cardiology) from a Post-Hoc analysis of the COURAGE Trial shows that PCI reduces morbidity, and appears as though it will reduce mortality of patients with SMI and no known CAD. Further research is needed to identify these asymptomatic patients at highest risk who may benefit from screening.

LEPTOMENINGEAL CARCINOMATOSIS FROM UROTHELIAL CELL CANCER: AN RARE CAUSE OF ALTERED MENTAL STATUS
Varun Takkyar MD, Abstract 67

Introduction: Altered mental status (AMS) in cancer patients undergoing chemotherapy is especially concerning given their immunocompromised state. The broadest differential for infectious causes should be promptly considered and thoroughly investigated. The etiology of AMS, however, sometimes lies beyond infection, and other possible processes must also be considered. Here we present a rare cause of altered mental status in the setting of urogenital malignancy.
Case Report: 67 year old man with a recent history of locally advanced T4N3MO urothelial cell carcinoma (UCC) status post radical cystoprostatectomy with ileal conduit presented to UMC with altered mental status after a fall. He was currently receiving Methotrexate, Vinblastine, Doxorubicin and Cisplatin (MVAC) chemotherapy with no progression in disease. His wife reported that the patient had unsteady gait and vision abnormalities including diplopia for the past week. Physical examination revealed an afebrile, confused male with right sided ptosis and facial droop but no nuchal rigidity. CT scan of the head showed no evidence of intracranial bleed or mass effect. MRI of brain done on Day 2 was negative. Lumbar puncture revealed straw colored fluid with a WBC count of 91 cells (89% lymphocytes, 10% monocytes and 1% RBCs). CSF glucose was 22 mg/dL and protein was 105 mg/dL. Opening pressure was not recorded. Given the CSF findings, acyclovir and fluconazole treatment was initiated but discontinued after CSF HSV PCR and cryptococcus returned negative. Other infectious etiologies including bacterial, enterococcus, coccoidomycosis and CMV were also ruled out. Over the next 2 days, the patient’s CNS symptoms progressed with development of right upper extremity weakness. A repeat MRI head and brainstem with FLAIR signaling showed diffusely increased FLAIR signal abnormalities in the subarachnoid spaces with cranial nerve spread. CSF cytology returned and was demonstrated carcinoma cells. The patient was diagnosed with leptomeningeal carcinomatosis secondary to metastatic UCC. Because of the patient’s poor performance status he was offered palliative radiation. He was treated with whole brain radiation with for one week. Over the next two weeks, his upper extremity weakness and right-sided facial paralysis improved. Currently, patient is undergoing surveillance imaging to assess the need for more radiation and/or possibly intrathecal chemotherapy. Discussion: Leptomeningeal metastasis from solid tumors is a common complication occurring in advanced malignancies. However, its appearance in UCC is rare, with only 25 documented cases appearing in the literature. This case emphasizes the need for broadening the differential beyond infection when evaluating a cancer patient with altered mental status so that a prompt diagnosis is made and appropriate treatment provided.

UNUSUAL CAUSE OF A COMMON COMPLAINT
Gina Wu DO, Abstract 118

Unusual cause of a common complaint Gina Wu PGY-2 Acute pancreatitis is an acute inflammatory process of the pancreas. The etiologies of gallstones or alcoholism comprise the majority of cases. Other causes of acute pancreatitis comprise a small percentage including rare etiologies that should prompt further investigation. A 74 year old male presented to the emergency department with a chief complaint of sharp, epigastric pain that started earlier that day without radiation. He also complained of fever/chills, nausea, and vomiting. The vomiting was nonbloody, nonbilious with multiple episodes throughout the day. He states that nothing made better/worse including food. He was recently treated for pneumonia 10 days ago for which he completed a course of Levaquin. Recent medication information he provided to us was that he was scheduled to go overseas and received a course of oral typhoid vaccine 2 days prior to coming to the emergency room (total of 4 doses). In regards to his alcohol consumption, he states he has an occasional
glass of wine. The initial physical exam revealed moderate tenderness in the epigastric area with no guarding or rebound. Laboratory evaluation included normal liver function tests and lipase > 2800 and amylase 5907. US of the abdomen showed a normal appearing gallbladder. CT of the abdomen showed acute pancreatitis. Patient’s Ranson score was calculated and showed a 40% mortality. The patient’s hospital course consisted of multiple complications. This included pleural effusion, biliary sludge and pseudocyst that measured 6 x 13 mm in size. The patient eventually was discharged after an almost 3 week hospital course. This case illustrates the importance of further investigation including rare causes of diseases. Regarding the etiology of the patient’s acute pancreatitis, the common causes of alcoholism and gallstones were ruled out. The less common causes including hypertriglyceridemia, post ERCP, smoking, hypercalcemia, genetic mutations were excluded. In 30% of the cases, no obvious etiology is identified. When the medications were further evaluated, Levaquin and oral typhoid vaccine had rare instances of causing pancreatitis. Since the patient had completed a course of Levaquin therapy ten days prior to admission, it’s unlikely the pancreatitis was attributable to this medication. Oral typhoid vaccine was administered 2 days prior to admission and more likely the culprit. This vaccine is a live vaccine given to individuals traveling to Africa, Asia and Latin American. The oral vaccine is administered in four doses on day 1, day 3, day 5, and day 7 with the last dose given at least 1 week prior to travel. Adverse reactions associated with the vaccine are fever, headache, abdominal pain, nausea, diarrhea, vomiting and rash. Due to the high mortality associated with pancreatitis, it is important to evaluate all potential causes and treat appropriately.

UNEXPLAINED FEVER IN HUNTINGTON’S DISEASE
Mohamed Zghouzi MD, Abstract 85

Introduction: Neuroleptic malignant syndrome in patients with Huntington’s disease is a rare occurrence. Only three people with Huntington’s disease have been reported in the literature with neuroleptic malignant syndrome. Several risk factors associated with the risk of developing hyperthermia in Huntington’s disease including psychopharmacological drugs, infectious diseases, and dehydration, especially in the summer. Case report: A 47 year old male with history of Huntington’s disease, vascular dementia, hypertension, weight loss, dysphagia, right eye blindness, on tetrabenazine and haloperidol presented to Emergency Department from a long term care facility with fever. The patient was non-verbal at the time of admission with a clinical examination significant for increased rigidity of both lower extremities. The white cell count was 14.6, 73% neutrophils; a work up for infectious etiologies including chest X-ray, urinalysis, blood cultures, and CSF studies revealed no abnormalities. Of note, the patient’s CPK level on admission was 6006. Liver enzymes were mildly elevated at AST 63 and ALT 56 with normal bilirubin. The patient was started on empiric broad spectrum antibiotics Vancomycin, Ampicillin, Ceftriaxone, and Acyclovir along with aggressive volume resuscitation. Haloperidol was held initially, and his CPK came down to 2399. Within 48 hours, the patient became afebrile, his white cell count decreased down to 10, and his antibiotics were discontinued. His mental status returned.

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to baseline. Discussion: Neuroleptic Malignant Syndrome consists of extra-pyramidal rigidity and a change in mental status, associated with hyperthermia and leukocytosis that makes it difficult to differentiate from an infectious etiology. However, elevated CPK levels can be a clue to the diagnosis. Unexplained hyperthermia in patients with Huntington’s disease could result from neuroleptic medications combined with dehydration, especially in summer months, and should always be considered with such a presentation.

**PGY-3 SUBMISSIONS**

**GPA: DOCTOR, ARE YOU TALKING ABOUT MY GRADE POINT AVERAGE?**

Sohail Abdi-Moradi MD, Abstract 95

A 52 year old male with a past medical history of positive TB test as a child with subsequent treatment presented to the emergency department with three months of pleuritic chest pain, persistent cough, and hemoptysis. He also experienced subjective fevers, night sweats, fatigue, diffuse myalgias, and 15 pounds of weight loss within the previous three weeks. The patient’s history is also notable for inflammatory eye disease and non-healing leg ulcers that were treated with steroids and methotrexate 10 years ago. When his current symptoms first occurred, he was placed on 5 mg prednisone daily which was later escalated to 20 mg daily due to lack of response. Additional therapies including methotrexate 15 mg weekly and a course of azithromycin did not provide relief. On admission, his temperature was 37.1 degrees Celsius with a pulse oximetry of 100 percent while on 2 liters of oxygen supplementation via nasal cannula. Lung examination revealed few scattered inspiratory crackles only. He had normocytic anemia with a hemoglobin value of 12.3 g/dL. There was no leukocytosis. Urinalysis and creatinine were unremarkable. ESR and CRP were both elevated at 39 mm/hr and 63.9 mg/L respectively. CT chest with contrast demonstrated various pulmonary nodules with peripheral halo and cavitary appearance. The patient was subsequently placed in airborne isolation due to concern for possible active TB. His MTB quantiferon was negative. Coccidiomycosis IgM enzyme immunoassay was indeterminate; however, the complement fixation and immunodiffusion were negative. His cANCA was positive at 1:512 with anti-PR3 antibodies. Bronchoscopy identified blood in the major airways but no active bleeding. Cytology from the procedure showed no evidence of malignancy. The patient was ultimately started on 60 mg daily prednisone and rituximab for a diagnosis of granulomatosis with polyangiitis. He was also placed on trimethoprim-sulfamethoxazole for Pneumocystis prophylaxis. Granulomatosis with polyangiitis (GPA) is a small vessel necrotizing vasculitis with an incidence of 10 cases per 1 million. Extrapulmonary manifestations include acute renal failure, conduction hearing loss, scleritis, and myocarditis/pericarditis. Nasal ulcerations, sinusitis, airway stenosis, diffuse alveolar hemorrhage, and lung nodules can also occur. Diagnosis can be made with renal biopsy or lung biopsy via VATS. Anti-PR3 cANCA is present in 85 percent of cases. Treatment involves glucocorticoid and cyclophosphamide combination therapy. Rituximab is an alternative to cyclophosphamide. Methotrexate can be used for mild disease or maintenance. Plasma exchange can be used in those who have severe pulmonary hemorrhage or severe renal failure. It is essential to recognize and treat GPA in a timely fashion as mortality is 90 percent at two years without treatment.
DISSEMINATED SPOROTHRIX SCHENCKII IN AN IMMUNOCOMPETENT MALE
Kristin Anderson MD, Abstract 88

Sporothrix schenckii is a ubiquitous fungus found throughout the world that often causes subacute or chronic infection in humans. A lymphocutaneous form of infection is the most common presentation. Disseminated sporotrichosis is an uncommon entity that is almost strictly associated with an immunosuppressed state, often underlying HIV infection. When extracutaneous spread occurs, osteoarticular spread represents 80% of cases. Here we present a case of disseminated sporotrichosis in an immunocompetent male. The patient is a 57 year old male with a history of type two diabetes mellitus who initially presented with pain and swelling in his bilateral knees, right elbow, and multiple small joints of his hands. Fluid culture from his right elbow and tissue culture from his left index finger grew out Sporothrix schenckii. He was started on therapy with oral itraconazole. Despite initiation of therapy, he developed blurry vision and dyspnea, raising concern for fungal involvement of the ocular and pulmonary systems. Therapy was therefore switched to IV amphotericin B. He also underwent pars plana vitrectomy, along with intravitreal injection of vancomycin, cefazolin, amphotericin, and clindamycin. While on amphotericin B therapy he reported mild improvement in his ocular symptoms. He also experienced side effects including renal tubular acidosis. He underwent an extensive immunosuppressive work-up that was negative. He will require a minimum of one year of anti-fungal therapy. This case illustrates that although it is extremely uncommon, disseminated sporotrichosis can occur in immunocompetent patients. Secondly, if ocular spread occurs, the prognosis is particularly poor. Although intraocular injections of amphotericin B have been recommended as treatment, successfully treated cases are rare in published literature. Over the last decade no new medication has been tested in large, randomized clinical trials in comparison with standard or alternative treatment regimens. When tried, amphotericin B has not shown an acceptable efficacy in treatment of ocular, meningeal, or systemic sporotrichosis and often results in toxicity. This emphasizes the need for exploring more potent and less toxic treatment options when therapy with itraconazole fails.

CONGENITALLY CORRECTED TRANSPOSITION OF THE GREAT ARTERIES
Majid Asawaeer MBBS, Abstract 13

A 31 year old male who presented with progressive worsening of SOB, orthopnea, paroxysmal nocturnal dyspnea, and leg swelling over 3 months. His CXR revealed enlarged cardiac silhouette. Echocardiogram; was limited because of the patient body habitus; suggested that the left sided ventricle is morphologically a right ventricle with a tricuspid valve, and the right-sided ventricle is morphologically a left ventricle with a mitral valve. A moderator band-like structure was seen in the apex of the left-sided ventricle. Electrocardiogram revealed Atrial fibrillation with complete heart block and junctional escape rhythm. Comprehensive Cardiac MRI without contrast revealed that The anterior ventricle is consistent with the anatomical left ventricle and the more posterior and leftward ventricle is consistent with the anatomical right ventricle. The patient was discharged after a prolonged course of hospitalization, which was complicated by: 1- Hypercapnic respiratory failure, requiring
intubation and mechanical ventilation, secondary to metabolic alkalosis and decrease in the respiratory drive secondary to aggressive diuresis. 2-Ventilator-associated pneumonia. And 3- Atrial fibrillation/Complete heart block, treated with biventricular pacemaker and warfarin. Congenitally corrected TGA occurs in less than 1% of all forms of congenital heart disease. Late complications include systemic ventricular dysfunction, progressive systemic atrioventricular valvular regurgitation, congestive heart failure, infective endocarditis, and conduction abnormalities such as complete heart block, and supraventricular tachyarrhythmias such as atrial fibrillation and atrial flutter (as in our case). Complete heart block can develop at a rate of 2% per year. Patients undiagnosed until adulthood usually present due to an abnormal chest radiograph or ECG. These patients are asymptomatic until right ventricular dysfunction, tricuspid regurgitation, or complete heart block. Van Praagh et al described Three main anatomic types of corrected TGA: (1) TGA with solitus atria (S), L-loop ventricles (L), and L-TGA (L), that is, TGA [S,L,L] (in 94% of the cases); as in our case; (2) TGA with solitus atria (S), L-loop ventricles (L), and D-TGA (D), that is, TGA [S,L,D] (in 3% of the cases); and (3) TGA with inverted atria (I), D-loop ventricles (D), and D-TGA (D), that is, TGA [I,D,D] (in 3% of the cases). Nearly 25 years ago, Huhta et al reported the long-term follow-up of 107 patients examined at the Mayo Clinic over a 30-year period between 1951 and 1981. Overall survival from the date of Mayo Clinic diagnosis was 70% at 5 years and 64% at 10 years. The only variable that consistently correlated with decreased survival was left atrioventricular valve. Therefore, Cardiac MRI is a valuable and non invasive modality used in the evaluation of congenital heart disease, since the morphological details of chambers, septum, defects and anomalous connections are depicted accurately. Additionally, flow information across valves, chambers, outflow tracts and shunts are also provided.

First Place – Associate Case Report Poster:

AN UNCOMMON CAUSE OF CHOLESTATIC HEPATITIS – EPSTEIN BARR VIRUS
Mohan Ashok Kumar MD, Abstract 41

Introduction: Epstein–Barr virus (EBV) causes infectious mononucleosis, with fever, pharyngitis, lymphadenopathy, and hepatosplenomegaly. Sometimes it causes a self limiting hepatitis, with mildly elevated transaminase levels. Cholestatic jaundice is rare and in a recent, retrospective analysis, only 0.85% of jaundiced patients had active EBV infections. Here, we report a case of acute cholestatic hepatitis secondary to EBV infection without mononucleosis. Case Description: A 26 year-old caucasian female with a history of bronchial asthma, presented with a four day history of right upper quadrant abdominal pain, nausea, fever and constipation. Immediately preceding the abdominal pain, she reported experiencing “a cold” that had lasted for 3 weeks. She had completed a 2 week course of augmentin prior to presentation. She denied having any rash, vomiting or sick contacts. She was afebrile and icteric on examination with a heart rate of 118. Her abdomen was soft, but tender to palpation in the right upper quadrant, without guarding, rebound or discernible organomegaly. All other systems were normal on exam. Blood tests revealed AST:317, ALT:296, ALP:282, Total Bilirubin 8.4, Direct Bilirubin 5.3. A right upper quadrant ultrasound showed slight hepatosplenomegaly with no evidence of biliary stones or biliary dilation.
Viral Hepatitis panel was negative. A vasculitis panel including ANA, anti-dsDNA, anti-microsomal, anti-MPO, anti-smooth muscle antibodies, anti-Ro, anti-La, anti-Jo and antiproteinase 3 were all negative. Ceruloplasmin levels were normal. A viral panel was negative for all viruses except EBV anti-capsid IgM antibody which was positive with a titer of 200 copies/ml by PCR. The patient was treated conservatively, improving clinically and biochemically with almost complete normalization of liver function after a week of hospitalization. Discussion: EBV is a ubiquitous virus of the herpes family and it is estimated that up to 90% of the population are asymptomatic carriers. Transmission by blood transfusion and transplanted organs in previously seronegative recipients have been documented. Relatively few cases of EBV related cholestatic hepatitis have been reported. In the largest series identified by the retrospective analysis of 1995 patients with hepatitis, only 17 (0.85%) had EBV hepatitis. The median age was 40 years (range 18–68 years). Ten of 17 (59%) patients were aged >30 years, and seven of 17 (41%) patients were aged ≥60 years. Fifteen of 17 (88%) patients presented with clinical/biochemical evidence of jaundice. It is notable that our patient did not have the mononucleosis syndrome with lymphocytosis. The clues to the diagnosis included fever, splenomegaly and symptoms suggestive of a preceding viral prodrome. EBV hepatitis should be considered in the differential diagnosis of all patients with hepatitis, without a clearly identified etiology.

A RARE PRESENTATION OF POST INFLUENZA BILATERAL DIAPHRAGMATIC PALSY
Deepti Boddupalli MD, Abstract 124

Introduction: Existing literature reports a few instances of post-influenza unilateral diaphragmatic palsy due to underlying phrenic nerve involvement. We present a very rare case of a young man diagnosed of bilateral diaphragmatic paralysis after suffering from influenza infection. Case Report: A 43 year old otherwise healthy male presented to the emergency room with complaints of orthopnea, exertional dyspnea that was progressively worsening over the last one month. His activity was markedly limited by these new symptoms. On physical examination, he only appeared in some distress in the supine position and appeared to use his abdominal muscles during his periods of respiratory distress. EKG showed atrial fibrillation with rapid ventricular rate. A subsequent cardiology work up was negative. A CXR and CT-Angiogram of chest showed no acute pathology with the exception of bibasilar atelectasis. However, pulmonary function tests were consistent with severe restrictive pattern. A Sniff test showed no overt downward diaphragmatic movement on either side and phrenic nerve conduction study confirmed diagnosis of bilateral diaphragmatic palsy. He was referred to cardio-thoracic surgery for further evaluation and management. Discussion: Diaphragmatic palsy in adults presents with severe dyspnea especially when bending or in supine position. It is usually unilateral and is caused by phrenic nerve dysfunction. Bilateral diaphragmatic paralysis is a severe form of respiratory muscle weakness and needs prompt attention. One of the causes described in literature appears to be neuropathy including post-polio syndrome, Guillain Barre syndrome and post-viral neuropathy. It is postulated that it could likely be due to a neuropathological process called neurological amyotrophy. Often, patients complain of severe neck pain preceding the shortness of breath. Our patient
exactly described severe neck pain that was followed with severe and worsening shortness of breath. Diagnosis is often confirmed with pulmonary function tests, presence of paradoxical diaphragm movement in a Sniff test, nerve conduction studies, diaphragmatic electromyography and measurement of Transdiaphragmatic pressure. While self-recovery after prolonged symptom persistence remains unlikely, techniques such as diaphragm plication have shown a lot of promise for unilateral paralysis. Bilateral paralysis is often managed with ventilator support and diaphragmatic pacing when the phrenic nerve is intact. Our patient unfortunately had phrenic nerve dysfunction. Conclusion: This is a rare presentation of bilateral diaphragmatic paralysis in a patient presenting with atrial fibrillation shortly after influenza illness.

WE'RE NOT IN THE COLON ANYMORE TOTO...
Kathryn Boyle MD, Abstract 36

Introduction: Pseudomembranous tracheobronchitis (PMTB) is a rare entity that is generally associated with aspergillosis. Pseudomembrane formation in the trachea has a short list of differential diagnoses and is often divided into infectious and non-infectious causes. A majority of the infectious causes are found in immunocompromised hosts with Aspergillus as the predominant organism. Herein, we report of a case of PMTB caused by an organism that has never been described in the literature. Case: A 59 year-old male with a history of high risk CLL and a recent allogeneic stem cell transplant was admitted for intractable nausea and emesis. During his hospitalization evaluation of his intractable nausea revealed grade 4 graft-versus-host disease of his stomach and thus he received high doses of steroids and alemtuzumab. He subsequently developed a fever and worsening of his baseline cough on hospital day 10. A CT chest showed a new left upper lobe infiltrate and therefore a bronchoscopy was requested. Irregular erythematous endobronchial mucosa was noted in the distal left main stem bronchus and left upper lobe, particularly the lingula. Bronchoalveolar lavage from the left lingula was sent for culture and eventually grew Rhizopus. The patient was started on IV and inhaled ambisome as well as IV caspofungin. Unfortunately, he developed hypoxic respiratory failure and was intubated approximately 7 days after antimicrobial treatment was started. A second bronchoscopy was performed after mechanical ventilation was initiated and revealed denuded and desquamated appearing mucosa separating from the submucosal layer in the distal left main stem and proximal left upper and lower lobes. There appeared to be bloody secretions emanating from only the left lobes. Bronchoalveolar lavage of both upper and lower lobes returned blood tinged fluid that did not clear with repeated aliquots. He was diagnosed with diffuse pulmonary hemorrhage. Two days following the bronchoscopy the patient’s family opted for comfort care and he expired that evening. Case discussion: To our knowledge, this is the first reported case of PMTB caused by Rhizopus. Stem cell transplant patients are at an extremely high risk of invasive fungal infections, especially those treated for graft versus host disease. Mortality from Rhizopus infection is exceedingly high in this vulnerable population. PMTB described in the setting of invasive Aspergillus infection has similar mortality rates. Increased mortality has been seen in cases where the denuded mucosa was pulled away from the submucosa resulting in massive pulmonary hemorrhage. Even without biopsy
the patient had generalized pulmonary hemorrhage due to the friable mucosa.

INFECTIVE ENDOCARDITIS: SERPIGINOUS IN NATURE
Peter Cherian MD, Abstract 86

Infective endocarditis serpiginous in nature
Case: Peter Cherian, MD. University of Arizona South Campus
Introduction: In patients with a history of intravenous drug abuse, the evaluation of fever often raises the possibility of endocarditis. Although commonly caused by staphylococcus, enterococcus and streptococcus, it is important recognize that there are a growing number of more unusual organisms being recognized in this population. We report a case of endocarditis caused by Lactococcus garvieae. A 29-year-old with a history of active intravenous use of cocaine and heroin presented to the hospital with fever, chills, myalgias, malaise and chest discomfort. She has been using intravenous drugs for the past 7 months. She stated that the day prior to admission she injected a total of 1 gram of heroin and 20 ounces of cocaine. She claimed to use only bottled water for the injection and explicitly denied sharing needles. On exam she was febrile with a temperature of 102.2, 3/6 systolic heart murmur, white count 15.2, 96.3% neutrophils, aspartate aminotransferase of 833 and alanine aminotransferase of 446. Blood cultures were obtained and vancomycin and gentamicin started empirically. A transthoracic echocardiogram was done and positive for possible vegetation. Transesophageal echo showed a long serpiginous mass measuring 3 cm that was attached to the right atria and IVC junction. Blood cultures were positive for Lactococcus garvieae. Antibiotics were changed to ampicillin sulbactam and gentamicin. The serpiginous mass was not seen on a repeat TEE done 10 days later. She was discharged to complete a 4-week course of antibiotics.
Discussion: Lactococcus garvieae is a facultative anaerobic catalase-negative Gram-positive cocci. Lactococcus is a new genus of gram-positive cocci, previously known as the lactic group of the Streptococcus genus, from which it was separated in 1985. Of the eight known species of Lactococcus, both Lactococcus garvieae and Lactococcus lactis have been reported to be pathogenic in humans. Reported infections include endocarditis, liver abscess, sepsis, osteomyelitis and prosthetic joints. Endocarditis appears to involve native and prosthetic valves equally. The majority of subjects are immunocompetent. This is the 12th case report of Lactococcus garvieae endocarditis. Lactococcus species were thought to be low virulent opportunistic organisms but given the multiple case reports of endocarditis occurring in an immunocompetent host Lactococcus garvieae may be an emerging zoonotic pathogen. The incidence of Lactococcus might be underestimated because 1) its morphologic similarity with enterococcus can lead to misdiagnosis and 2) the lack of availability of PCR to confirm the diagnosis. Of note Lactococcus species has been isolated from dairy products and has been found in Italian cheese. Interestingly, Italian baby milk powder is one of the substances used to cut cocaine and maybe a reservoir for this bacterium.

ALLOGENEIC STEM CELL TRANSPLANTATION FOR MYELOPROLIFERATIVE NEOPLASM IN BLAST PHASE
Chad Cherington MD, Abstract 55

Purpose: The myeloproliferative neoplasms (MPN) have the potential to transform into an acute myeloid leukemia (AML) also known as
MPN-blast phase (BP). There is very little information available in the literature regarding the role for stem cell transplantation in this setting. We performed a retrospective literature review of patients at the Mayo Clinic in Arizona who had an MPN-BP transformation to AML of which 8 of 13 went on to receive allogeneic stem cell transplantation (allo-SCT). The primary purpose of this report was to analyze the success rate of allo-SCT in patients with MPN-BP, and to evaluate factors potentially associated with favorable outcome. As such, the primary endpoint was progression-free survival of all patients who underwent allo-SCT, defined as the time from allo-SCT to death or relapse. Secondary endpoints included overall survival (time from allo-SCT to death from any cause), engraftment, rates of acute and chronic GVHD, and relapse. Method: This study was approved by the Mayo Clinic Institutional Review Board (10-004882). A chart review was performed using patients listed in the Bone Marrow Transplant database as well as the Cancer Registry database. We identified 13 patients with Philadelphia-negative myeloproliferative neoplasm transformation to blast phase (MPN-BP). Cases of mixed myeloproliferative/myelodysplastic neoplasms (MPN/MDS), such as chronic myelomonocytic leukemia (CMML) or other overlap syndromes, were excluded. Results: The distribution of MPN disorders was essential thrombocythemia (ET) (2), post-ET myelofibrosis (MF) (2), polycythemia vera (PV) (4), post-PV MF (1), and primary myelofibrosis (4). The transformation from MPN diagnosis to AML occurred after a median of 9 years (range 5 months - 30 years). Induction chemotherapy cleared blood/marrow blasts in 60% (6/10) (2 declined therapy, 1 had early death). Of these 8 of 13 patients continued to allo-SCT. Their median age was 55 years. All 8 patients received allo-SCT within 6 months of diagnosis of AML. At the time of allo-SCT, 5/8 patients were in complete remission (CR) of their leukemia or had returned to MPN chronic phase (CP), 2 had residual blood blasts and 1 refractory with >5% marrow blasts. At follow-up (median 20.3 months), 6 patients are alive in CR of both their leukemia/MPN. All 5 patients in CR/CP at pre-allo-SCT remain alive in remission, while 2/3 with persistent blood/marrow blasts relapsed and expired. Conclusions: We conclude that MPN-BP can be cured by allo-SCT in a significant percentage of patients, but that adequate leukemic clearance prior to allo-SCT offers an optimal outcome.

ADVENTURES IN FLY FISHING: AN UNEXPECTED CATCH
Vanessa Costilla MD, Abstract 98

Introduction: Histoplasma capsulatum is a dimorphic fungus endemic to the Ohio Mississippi River Valley. We present a unique case of disseminated H. capsulatum presenting cutaneously in an immunocompromised patient with no exposure to the endemic areas. Case Description: A 63-year-old woman presented to the emergency department reporting fever and progressive redness of the right arm and thigh. The redness began after electromyography and biopsies for evaluation of dermatomyositis. Two weeks prior, she was treated with antibiotics at another hospital for presumed cellulitis. Medications on admission included prednisone and methotrexate. She reported a remote history of coccidioidomycosis, and was an expert fly fisherwoman with a travel history to Mexico, California, Colorado, Montana, Wyoming, and Idaho. On examination, her medial right thigh had a 12-cm, erythematous, nodular eruption with a smaller ulcerated area and overlying eschar, and the medial right arm had a nodular, erythematous,
tender eruption. Laboratory studies revealed mild anemia and normal white blood cell count (9.6 & 61620; 109/L). Peripheral blood cultures, urine culture, and coccidioidomycosis serologies were obtained. Coccidioidomycosis antibody by complement fixation was positive at 1:4. Erythema and induration of the right arm and thigh progressed, despite initiation of levofloxacin and vancomycin. Fluconazole was added without apparent improvement and thus, she underwent surgical debridement of the wounds on hospital day 5. Gomori methenamine silver stain demonstrated small yeast-like organisms in the tissue. Histoplasma urinary antigen positive. Fungal culture of the debrided tissue ultimately produced a slow-growing mold positive for Histoplasma capsulatum. Liposomal amphotericin B was administered and she had rapid improvement of her lesions. She was subsequently transitioned to itraconazole and completed a 1-year course. Discussion: Our patient had no obvious exposure to H capsulatum. The source of the infection was unproven. She had not lived in nor traveled to an endemic area and had no direct exposure to bats or birds. However, she crafted fishing flies from chicken feathers, and we speculate that she inhaled aerosolized fungal spores that were on the feathers before harvest. Patients with no prior exposure to endemic areas can acquire fungal infections through microfoci, such as commercially sold bird feathers. In addition, antihistoplasma antibodies may produce false-positive results for coccidioidomycosis or blastomycosis in complement fixation assays, resulting in delayed diagnosis and treatment of the true infection. Conclusions: This case illustrates several points. First, disseminated fungal infections must be considered as a possible cause of skin lesions in an immunosuppressed patient. Second, consideration must be given to fungi that are not endemic to the area. Third, results of apparently positive diagnostic tests may be misleading. This patient had multiple positive diagnostic tests, but it was not until the true origin was identified and treated that her lesions improved.

NOT YOUR TYPICAL PNEUMONIA
Vincent Duenas DO, Abstract 82

Not Your Typical Pneumonia. Vincent Duenas, OGME III, Sierra Vista Regional Health Center

Varicella pneumonia is a rare but serious complication of Varicella Zoster Viral infection with a very high mortality rate in immunocompetent adults. Despite vaccination, “breakthrough disease” may still occur. Individuals often experience progressive dyspnea, tachypnea, dry cough, and occasionally hemoptysis. The symptoms are insidious, typically presenting one to six days after the onset of the classic vesicular rash. A 22 year old female was evaluated in the emergency room for fever, rash, and dizziness of 3 days duration. At the time of presentation, she also complained of progressive shortness of breath and a nonproductive cough. A diffuse maculopapular rash was observed on physical exam with increased concentration in the abdomen, chest, and back. A chest x-ray was obtained and revealed bilateral interstitial infiltrates. The patient was a health care worker at a local skilled nursing unit and was exposed to an individual with herpes zoster just one week prior. She did report being vaccinated for Varicella within the recent years. Pneumonia with cutaneous involvement has a wide differential: Coccidioidomycosis, Legionella, Mycoplasma, and Coxiella, just to name a few. Early recognition and management is critical. Prompt administration
of intravenous Acyclovir in Varicella pneumonia is associated with improvement in clinical outcomes.

RIGIDITY IN THE PSYCHIATRIC PATIENT
Lisa Graham MD, Abstract 126

Rigidity in the Psychiatric Patient By Lisa Graham & Harold Szerlip
Rigidity in a psychiatric patient is akin to chest pain in a medicine patient. It can be caused by multiple life threatening conditions including: Neuroleptic Malignant Syndrome, Serotonin Syndrome and Malignant Catatonia. Often complicating the picture are co-morbid medical conditions, drug ingestions and withdrawal states. Mr. RM was a 55 year old male with history of paranoid schizophrenia, depression and hypothyroidism, who presented to an outside hospital after ingesting a month’s supply of levothyroxine, lisinopril, mirtazapine, and baclofen. He was found unresponsive at home and intubated. The following day he was extubated and transferred to our facility for psychiatric evaluation. At the outside hospital he was described as withdrawn, uncooperative and minimally responsive. Upon arrival to our psychiatric ward, he was diffusely rigid and unable to move his mouth to speak. He had received intravenous haloperidol prior to extubation but no other psychiatric or serotonergic medications. He had no history of catatonia. Physical exam was significant for BP of 170/90, clonus and diffuse rigidity, but he was afebrile, alert and able to follow simple commands. Labs revealed: TSH 0.03, T4 1.50, CRP 77.9 but Utox was negative. VBG, CPK, ammonia level, serum iron, LDH, CMP, CBC, and lactate were all within normal limits. EKG and CT Head were also normal. Despite lack of fever, dantrolene therapy was started but stopped due to induction of frequent PVCs. Lorazepam challenge negative. With re-institution of his baclofen, however, his symptoms resolved within 2 days. When approaching rigidity, life threatening causes should be ruled out first. Although many consider fever a necessary component in NMS, altered mental status and rigidity typically appear first and atypical cases without fever have been reported. However, his CK and serum iron were normal and his clonus was more consistent with serotonin syndrome. Serotonin syndrome can occur without exposure to the traditional antidepressant agents. For instance, it has been reported with MDMA, valproic acid and MAO inhibitors. Even though this patient had no known exposure to any of the associated drugs, keeping this in the differential helps avoid initiation of medication that will worsen the phenomenon, such as fentanyl, linezolid or ondansetron. Finally, a lorazepam challenge is a relatively easy and benign step to ruling out the life threatening disease process of Malignant Catatonia. In this patient, after the more common causes of rigidity were discounted, consideration was given to the possibility of baclofen withdrawal. Intrathecal baclofen withdrawal is a well known phenomenon; however, oral baclofen withdrawal is not a well-acknowledged complication. This unusual circumstance of oral baclofen overdose followed by therapy cessation reminds us how to approach rigidity in the psychiatric patient.

PERITONEAL COCCIDIOIDOMYCOSIS PRESENTING AS ABDOMINAL PAIN WITH EOSINOPHILIC PERITONITIS
John Horne MD, Abstract 70

Introduction: Peritonitis is an unusual extrapulmonary manifestation of coccidiodomycosis. As for other diseases that
are not reportable the true incidence of this entity may be underestimated. We present a case of peritoneal coccidioidomycosis that presented as eosinophilic peritonitis and was initially diagnosed as eosinophilic gastritis. Case Presentation: A 46 year old previously healthy Caucasian man presented to the emergency department with acute worsening of abdominal pain which began 5 weeks before presentation with constipation, no weight loss and no changes in appetite. He frequently travels all over North America, and is currently living in Arizona. Physical exam was pertinent for generalized abdominal tenderness without rebound tenderness. Computed tomographic examination of abdomen and pelvis showed mesenteric stranding with free fluid. An ultrasound-guided diagnostic paracentesis yielded 1.5 Liters of amber colored fluid with total nucleated cell count of 15,844; eosinophils 75%; lymphocytes 16%; neutrophils 2%; basophils 1%; mesothelial cells 4%; and red count of 15,486. The ascitic fluid had reactive mesothelial cells and numerous mature eosinophils. Additional blood tests: Hgb 13.1 g/dL; platelets 473 x 10^9 /L; WBC 12.4 x 10^9 cells/L; neutrophils 61%; lymphocytes 12%; monocytes 5%; eosinophils 22%; basophil 0.3%. Stool for ova and parasites was negative. Underwent endoscopy and gastric biopsy demonstrated mild chronic gastritis and duodenal biopsy had focal villous blunting and increased lamina propria eosinophils. Colonoscopy with colonic biopsy was consistent with lymphocytic colitis. Bone Marrow biopsy was unremarkable. He received a diagnosis of eosinophilic gastritis. However, clinical presentation had unusual features such as constipation; hence patient was sent for evaluation for a diagnostic laparoscopy, which found peritoneal studding of the wall of the abdomen. A peritoneal biopsy demonstrated necrotizing granulomatous inflammation with fungal spherules which are pathognomonic of Coccidioides. Cocci complement fixation titers were 1:64. Discussion: Coccidiomycosis peritonitis can present as eosinophilic peritonitis. It is very important to have a high index of suspicion to provide adequate treatment. Demonstration of the spherules of Coccidioides immitis in biopsy specimens is the most frequent means of diagnosis. Optimal treatment for peritoneal coccidioidomycosis is difficult to determine due to paucity of reported cases. Our patient was treated with fluconazole with good response to treatment at least 2 months after diagnosis.

CMV COLITIS IN THE SETTING OF INFLAMMATORY BOWEL DISEASE: A GREAT MIMICKER
Nasibo Kadir MD, Abstract 48

Abstract Title: CMV Colitis in the Setting of Inflammatory Bowel Disease: A Great Mimicker
Abstract Text: Authors: Nasibo Kadir, MD; Miriam Grigor, MD, University of Arizona College of Medicine
Introduction: The following is a case of Cytomegalovirus (CMV) colitis that was mimicking an Ulcerative Colitis (UC) flare in an immunosuppressed patient previously on chronic treatment for UC. CMV is a common virus found in the human population that causes complications in immunocompromised hosts. CMV colitis is increasingly being found in patients with UC who present with severe, unremitting UC flares. Case Description: 37 year-old Caucasian female with a history of UC diagnosed four years ago presents with a 3-day history of severe abdominal pain, bloody diarrhea, and nausea/vomiting. Pertinent medications include Colazal and Prednisone. Physical exam showed low grade fever, tachycardia, mild
tenderness to palpation of the lower abdominal quadrants bilaterally. Stool studies and CMV serologies were negative. CT of abdomen/pelvis showed colonic wall thickening from the splenic flexure to the rectum compatible with infectious or inflammatory colitis. The patient was initially diagnosed with a UC flare and was treated with IVF hydration, antibiotics, IV methylprednisolone, and resumption of colazal. She initially improved mildly then began to develop worsening abdominal pain, diarrhea, and high grade fevers several days into her hospitalization. A flexible sigmoidoscopy was performed and showed ulcerated mucosa and scattered pseudopolyps in the rectosigmoid colon. Multiple biopsies were performed and subsequently showed multiple enlarged epithelial cells with intranuclear inclusions diagnostic of CMV. Induction therapy with Ganciclovir IV was begun for a total of 2 weeks, and patient was switched to po Valganciclovir for a planned treatment course of 6-8 weeks followed by repeat sigmoidoscopy and biopsy. Patient subsequently improved on this course.

Discussion: CMV colitis is rare in immunocompetent hosts, but in patients with chronic Inflammatory Bowel disease, especially Ulcerative Colitis, CMV colitis is becoming increasingly common and often mimics UC flares. It remains unclear whether this CMV co-infection occurs because of existing structural disease or because UC patients tend to be immunocompromised from treatment of UC. In patients who are treated for UC flares and who fail to improve despite appropriate treatment, there should be a high clinical suspicion for CMV colitis and further investigation should be initiated.

BILATERAL HEARING LOSS, A VERY RARE CLINICAL PRESENTATION OF PSEUDOTUMOR CEREBRI

Rostam Khoubyari MD, Abstract 109

Pseudotumor cerebri may present with headache, nausea, vomiting, pulsatile tinnitus, diplopia, papilledema, visual loss and abducens nerve palsy. Hearing loss, however, is a very rare presentation of this disease that to best of our knowledge has only been reported in two previous cases. A 36-year-old Hispanic male with diabetes, ESRD on HD and HTN presented with nausea; non-bloody, non-bilious vomiting, intermittent headache and bilateral hearing loss for 2 days. He also complained of chronic intermittent headaches. He had no previous problems with hearing. He denied any loud noise exposure, fever, earache, ear discharge, or recent visual changes. He had been treated 2 months ago with cephalxin for a diabetic foot ulcer but otherwise there had been no change in his medications including gabapentin, glyburide, lisinopril, metoclopramide, omeprazole, ranitidine, ASA, metoprolol and calcium acetate. Vital signs showed T:37o C, BP:181/97, PR:78, RR:14, Sat:96%, BMI:32.7. He had new onset impaired lateral gaze bilaterally, bilateral diabetic retinopathy but no papilledema. There was complete bilateral hearing loss and decreased sensation of lower extremities. The remainder of the exam was unremarkable. BMP showed Na:132, K:4.2, CI:91, CO2:29, BUN:26, Cr:5.6. TSH:2.52, FT4:1.32. Head CT and MRI showed no ventricular dilation. LP was significant for elevated pressure of 430 mm of water in the first attempt and 500, 430, and 380 mm of water in the subsequent LPs with CSF removal of 18-20ml per each LP. CSF was clear and the analysis showed glucose:60, protein:24, WBC<1, RBC:15, negative gram stain/culture,
the patient was put on acetazolamide. Within 24h he started to regain his hearing. Almost full recovery resulted within 3 days. Later he was referred to neurosurgery for placement of a lumboperitoneal shunt. The annual incidence of pseudotumor cerebri is 1 to 2 per 100,000 with a higher incidence in obese women. The exact pathogenesis is unknown but to diagnose pseudotumor cerebri, other causes of increased intracranial pressure (e.g. CNS infections, intracranial tumors and cerebral venous thrombosis) should be ruled out. Pseudotumor cerebri can be associated with hypothyroidism, anemia, uremia, OSA, SLE, obesity, PCOS and medications (OCPs, NSAIDs, etc). The most common signs and symptoms are headache, nausea, vomiting, pulsatile tinnitus, diplopia, papilledema, visual loss, and abducens nerve palsy. The common recommended treatments are acetazolamide, corticosteroids, serial LPs, weight loss, and lumboperitoneal shunt. Our patient presented with hearing loss that is extremely rare, along with no evidence of ventricular dilation or papilledema that are very uncommon in Pseudotumor cerebri. However the diagnosis was accomplished by high clinical suspicious and effective multidisciplinary communication among internal medicine, neurology and radiology services. This reminds us that patients never read the textbooks to present necessitating of having the competency of recognition of uncommon manifestations of diseases.

DISSEMINATED COCCIDIOIDOMYCOSIS
CAUSING SEPTIC SHOCK WITH
HYPOCOMPLEMENTEMIC ACUTE KIDNEY INJURY
Jennifer Kim MD, Abstract 72

Jennifer H. Kim, MD; Richard Guthrie, DO;
Kashif Yaqub, MD; Karen Alonso, MD; Jay Blum, MD Internal Medicine Residency Program
Banner Good Samaritan Medical Center/Carl T. Hayden Phoenix Veterans Association Medical Center

Introduction: Disseminated coccidioidomycosis is associated with significant morbidity and mortality and tends to occur in small percentage (<5%) of cases. The clinical manifestations vary widely and can occur weeks, months, or years after primary infection. Early recognition and treatment can reduce morbidity and mortality, therefore understanding the presentation of the disease is of utmost importance. Septic shock from disseminated coccidioidomycosis continues to be under-recognized. Patient outcomes may improve if systemic coccidioidomycosis is recognized early as the etiology for septic shock. Case presentation: Fifty year old African American male with a past medical history significant for hypertension and pulmonary coccidioidomycosis which had been treated with six months of fluconazole, presented one month following completion of treatment. Upon current presentation, the patient reported three days of fever, dyspnea, and abdominal pain associated with nausea, vomiting, and diarrhea. Of note, he also complained of hip pain for which he had taken twelve grams of acetaminophen, daily, for three days prior to admission. The patient was found to be septic requiring intubation, and the source was thought to be pulmonary. After thorough investigation and consultation involvement, the patient was found to have multi-organ failure including cardiovascular, pulmonary, and renal compromise. The diagnosis of disseminated coccidioidomycosis was confirmed by bone marrow biopsy of a
right iliac crest lesion that showed dense infiltration with coccidiomycosis spherules. He also had hypocomplementemic renal failure.

Conclusion: Coccidioidomycosis is a fairly common, and sometimes asymptomatic, disease endemic to the southwestern United States. It is often subclinical and can be self-limited. Coccidioidomycosis infection is most frequently associated with pulmonary manifestations, but can rarely disseminate to other organs including the skin, lymphatics, skeleton, brain, retina, and solid organs. Even more rarely, disseminated coccidioidomycosis can cause septic shock and multi-organ failure. With our patient, septic shock manifested six months after being diagnosed with primary coccidioidomycosis. His clinical manifestations included altered mental status, respiratory failure requiring emergent intubation, and hepatic failure. Our patient also developed a rare case of hypocomplementemic acute kidney injury with suspected rapidly progressive glomerulonephritis. No cases were found in modern medical literature of hypocomplementemic acute kidney injury with rapidly progressive glomerulonephritis due to disseminated coccidioidomycosis.

Second Place – Associate Patient Safety/Quality Improvement Poster:

DOES THE ADAGE ‘YOU CAN LEAD A HORSE TO WATER...’ APPLY TO POST-HOSPITALIZATION PRIMARY CARE FOLLOW-UP?

Juliana (Jewel) Kling MD, Abstract 132

Introduction The transition from hospital to home is a vulnerable time for most patients. Many studies show an association between rates of readmission and scheduled outpatient follow-up after discharge. Our aim was to test the hypothesis that pre-scheduling a follow up appointment with primary care providers (PCP) prior to discharge decreased emergency department (ED) reevaluations in the 2 weeks following hospital discharge. Methods The charts of 200 randomly selected patients admitted to the Internal Medicine teaching service at our institution from July 2010 through December 2011 were reviewed, half during the pre-intervention period and half post-intervention. The intervention consisted of targeted education to interns regarding the discharge process and the importance of scheduling appointments with PCPs prior to discharge. We collected the following information: patient age, length of stay, PCP name & institution, presence of a discharge summary to and appointment with the PCP, any ED visit within 2 weeks and whether the patient kept the scheduled PCP appointment. Results Age (66.6 ± 18.2 vs. 65.6 ± 17.8, p = 0.65), study institution PCP (51.6% and 59%, p = 0.28), and length of stay (3.5 ± 3.5 and 3.3 ± 3.8, p = 0.70) were not statistically different between the pre- and post-intervention groups. Following the intervention, patients were not more likely to keep the PCP appointment (66.3% and 60%, p = 0.35) nor was there a difference in the number of ED visits (19.8% and 17%, p = 0.60) between the groups. A multivariate analysis was performed, demonstrating that patients were more likely to follow-up if they had a PCP at the study institution and less likely to follow-up if they returned to the ED within 2 weeks of their initial discharge. Conclusion Pre-scheduling outpatient PCP appointments prior to discharge did not improve post-hospitalization follow-up nor did it reduce the number of ED visits within 2 weeks. Further studies are needed to identify effective strategies for decreasing the number of post discharge ED visits and improving the post hospital transition.
A “FIB”, THE COLDHEARTED TRUTH
Mohamad Lazkani MD, Abstract 3

Atrial fibrillation (AF) is characterized by rapid atrial rate from 350 BPM to as fast as 600 BPM with irregularly irregular ventricular conduction. There are several etiologies for persistent AF, that can be exacerbated by acute hypoxia, hypercapnia, metabolic or hemodynamic abnormalities. It may also complicate existing heart disease or may occur in the absence of heart disease, considered lone fibrillation. Lone fibrillation may be seen during exercise, acute alcoholic intoxication, emotional stress, following surgery, or with a surge of vagal tone as during a vasovagal response. There are also documented some less frequent causes of atrial fibrillation. We present a case of atrial fibrillation induced by drinking a frozen smoothie. A 55 y.o. male was drinking a iced, blended fruit drink, when he noted palpitations. After presenting to the emergency department, he was diagnosed with AF with rapid ventricular rate. The same event occurred five years previous while drinking a similar beverage. At that time, it lasted less than one hour and resolved spontaneously. On presentation, his physical examination was unremarkable except for an irregularly irregular rapid heart rate. His blood count, electrolytes and TSH were normal. His EKG, showed AF with ventricular rate of 90-150 bpm. As his previous episode resolved spontaneously and his current episode was less than 24 hours in duration, he decided to postpone cardioversion, expecting the rhythm to self-convert. He returned to the emergency room the next morning and was still in AF. His vital signs were: blood pressure 112/85, pulse 87 and irregular, temperature 98.3, and respiration rate of 12. While waiting for his cardiologist to perform cardioversion, his rhythm converted spontaneously. There are a few reports about the development of supraventricular dysrhythmias after the ingestion of cold substances. Two etiologies were suggested as mechanisms: direct cooling of the left atrium through the wall of the esophagus and autonomic stimulation by the cold substance. Parasympathetic effects are usually preceded by progressive bradycardia and rarely lead to permanent AF. On the other hand, sympathetically induced AF tends to occur in those with heart disease and typically occur earlier in the day or during times of exercise or stress. The initial effect following heart disease is withdrawal of vagal influences and a subsequent increase in sympathetic influence, leading to micro-reentry, automatic and triggered automaticity phenomena. AF induced by ingestion of an iced fruit drink in an otherwise healthy male occurs via parasympathetic innervation. The parasympathetic response is mediated by esophageal nociceptors located in two distinct ganglia, the inferior ganglion and the jugular or superior ganglion, both located in the cervical region, as provided by research on guinea-pigs. These fibers transmit to the thalamus and cerebrum for further processing and thus mediate the autonomic nervous system.

PARADOXICAL EMBOLISM: MYTH OR TRUTH?
Naser Mahmoud MBBS, Abstract 14

Introduction: With the increasing number of patients diagnosed with thromboembolism, more cases of paradoxical emboli are being identified. Identification of a Patent Foramen Ovale (PFO) in a patient with an embolic event has been associated with many cases of organ infarctions. Case Presentation: A 45-year-old
male with chronic back pain presented with abdominal pain of 3 days duration, on his left upper quadrant (LUQ), sharp, increases with inspiration, radiating to the left shoulder, associated with anorexia and fever of 102F. He denies any diarrhea, vomiting or trauma.

Physical Examinations: The temperature was 104F and he has LUQ tenderness on palpation, otherwise the physical examination was unremarkable. Labs and imaging: WBC 10.3, hemoglobin 14.4, platelets 222. Sodium 136, potassium 3.7, BUN 10, Cr 0.8, ALT 19, ALP 58, ALB 3.8, PT/INR 15/1.2. CT scan of the abdomen with contrast showed enlarged spleen, the anterolateral and posteroinferior portions of the spleen do not enhance with contrast, distal branches the splenic artery supplying these regions do not enhance and these findings were consistent with early splenic infarction. The rest of the aorta and its branches were normal. A trans-thoracic echocardiogram didn’t show any vegetations or thrombi that can explain the splenic infarctions. Due to the possibility of endocarditis in this patient who is presenting with fever and splenic infarctions a transesophageal echocardiogram was done and it showed no evidence of a masses, clots or vegetations, a PFO with moderate Rt to Lt shunt was present with prominent Chiari network and interatrial septal aneurysm. A duplex ultrasound of all extremities showed large occlusive thrombus in the right saphenous vein. Testing for protein C/S deficiency, antithrombin 3 deficiency, antiphospholipid testing, prothrombin mutation and factor 5 leiden mutation was negative. Final Diagnosis: Paradoxical embolism through PFO causing 2 splenic infarctions. Management: The patient was anticoagulated with unfractionated heparin and warfarin for 5 days and continued on warfarin with a target IN of 2-3, 2 months later he underwent a device closure of his PFO.

Discussion: PFO occurs in 25 to 30 percent of the population. The prevalence is higher in patients with cryptogenic strokes. Most individuals are asymptomatic although some have clinical manifestations such as cryptogenic infarctions. Identification of PFO in a patient with an embolic event does not prove a causal relationship and the evaluation of patients with PFO with an embolic event should include assessment of other potential causes of thromboembolism. Agitated saline contrast with echocardiogram enables shunt identification. There are no studies supporting primary prevention but secondary prevention with aspirin, anticoagulation or device closure was shown in some studies to be helpful.


HEPATOCELLULAR CARCINOMA PRESENTING WITH BLOODY ASCITES
Ashish Mathur MD, Abstract 31

Introduction: Hepatocellular carcinoma (HCC) is a primary tumor of the liver usually due to chronic hepatitis B or C. Although patients may remain asymptomatic, clinical features include new onset ascites, jaundice, variceal bleeding, or encephalopathy from extension of the tumor into hepatic or portal veins or arteriovenous shunting. Diagnosis involves imaging, serum alpha-fetoprotein (AFP) measurement, and liver biopsy. Our case involves new onset bloody ascites as a clinical presentation of newly diagnosed hepatocellular carcinoma. Case Report: A 64 year old male with a history of cirrhosis due to chronic hepatitis C and alcohol abuse presented to the hospital from Gastroenterology clinic with increased abdominal girth, weight gain, and right sided
abdominal pain. A lesion on the right lobe of liver was biopsied three years ago without evidence of malignancy. Last paracentesis was five years ago. Physical exam revealed temperature 99.1, pulse 140, respiratory rate 13, blood pressure 97/63, pulse oximetry 95% on room air. Abdomen was soft, distended, caput medusa present, dullness to percussion right lower flank, nontender to palpation, bowel sounds hypoactive, no rebound or guarding. Pertinent lab data included leukocytosis of 23,400 with left shift, hemoglobin 12.8, platelet 367,000, alkaline phosphatase 132, AST 51, ALT 17, INR 1.3 and AFP 309.75 with baseline less than 10 in the previous six years. CT of the abdomen and pelvis revealed interval development of multiple new masses within the right lobe of the liver with increased prominence of the previously noted mass. Paracentesis revealed grossly bloody peritoneal fluid. Fluid studies showed a WBC of 2647, RBC of 567,760, serum ascites albumin gradient of 1.7, and no evidence of spontaneous bacterial peritonitis. Cytology showed metastatic carcinoma consistent with HCC or pancreatobiliary origin. A diagnosis of HCC was made based on history of cirrhosis, elevated AFP, liver lesions on imaging, and cytology of peritoneal fluid. A pleurx catheter was placed. Sorafenib was initiated. The patient developed renal failure, presumably due to hepatorenal syndrome. He entered hospice after discussion of his poor prognosis. He died two days after discharge. Discussion: Five percent of patients with cirrhosis have grossly bloody ascites, while approximately thirty-four percent with bloody ascites are diagnosed with hepatocellular carcinoma. Twenty percent of patients with malignant ascites is bloody. Fifty percent of these cases have HCC. The pathophysiology involves mass effect with erosion into small vessels or high shear stress over small vessels and lymphatics. Imaging with ultrasound or CT should be obtained. Management involves treatment of the underlying cause. In patients with bloody ascites from HCC, embolization of bleeding tumor vessel may be beneficial. The role of surgery is not well established. Prognosis is poor and therefore palliative systemic therapy is usually sought.  

EVOLVING MANAGEMENT OF ALCOHOL WITHDRAWAL SYNDROME (AWS): IMPACT ON INTENSIVE CARE UNIT (ICU) COURSE AND COMPLICATIONS

Luis Medina-Garcia MD, Abstract 120

Study Aim: To compare the course of AWS in 2010 at MMC with our previous study of 2005-7 (CCM 2008; 36 #12:S546) to assess the effects of enhanced admission criteria and changes in sedation practice. Hypothesis: Enhanced admission guidelines and use of poly-vs-mono sedation therapy reduce ICU complications and length of stay. Methods: A retrospective review of patients with AWS admitted in 2010, compared to a similar group reviewed in our previous study. Background: Patients with severe AWS who require ICU admission are prone to respiratory failure and pneumonia, with prolonged ICU and hospital length of stay (LOS). Although benzodiazepines remain the major class of agents used for sedation management, the use of supplemental agents such as propofol, dexmedetomidine or others is evolving. Results: The number of hospital admissions in 2010 was 0.83/day (306/361) vs 0.28/day (279/974) in the prior study (p<0.05). There were 80 ICU admissions among 306 episodes of AWS (26%) in 2010, compared to 87 patients requiring ICU care among 279 episodes of AWS from January 2005-September 2007 (p=ns). Demographics were similar: 94%
male in 2010 vs 91% male in 2005-7; mean age 45 years in 2010 vs 46 years in 2005-7, respectively (all p=ns). For ICU patients, ICU and hospital LOS were 5.5 +/- 4.7 days and 9.8 +/- 5.7 days in 2010, respectively; vs 5.4 +/- 6 days and 12.5 +/- 9.2 days in 2005-7, respectively (all p-ns). However, in 2010, 30/80 (37.5%) of ICU patients required intubation and mechanical ventilation (I/MV) vs 46/87 (52.8%) in the prior review (p<0.05). In addition, for ICU patients there were fewer episodes of pneumonia [29/80 (26%)] in 2010 vs 44/87 (50.6%) in 2005-7 (p<0.05). There was a similar requirement for I/MV among ICU patients with pneumonia in 2010 and in 2005-7 [22/29 (75.8%) vs 36/46 (78.3%) p=ns]. During the first interval, lorazepam was the primary sedative; 83% was given by IV infusion. In 2010, more than two agents were utilized for 59 of the 80 ICU patients (74%); including a benzodiazepine, propofol, dexmedetomidine, and/or haloperidol. Conclusions: Enhanced admission criteria and evolution to multi-drug therapy was associated with a higher hospital admission rate, as well as reduced requirement for I/MV and fewer instances of pneumonia among ICU patients. However, the proportion of those requiring ICU admission, as well as the ICU/hospital LOS of ICU patients were little affected.

ESOPHAGEAL PAPILLOMA IN NEWLY DIAGNOSED HIV/AIDS
Dalia Mikhael MD, Abstract 49

Esophageal papillomas are rare benign epithelial lesions characterized histologically by fingerlike projections consisting of a squamous cell lining with a core of connective tissue. The pathogenesis has been linked to underlying inflammatory conditions and the human papilloma virus (HPV). A 42-year-old previously healthy Hispanic male presented to the emergency department after two month of dysphagia, dyspepsia and 47 pounds of unintentional weight-loss. The patient underwent an esophagogastroduodenoscopy (EGD) in Mexico and was told he had a mass, likely cancer, but the diagnosis was not confirmed. The patient denies any significant medical history, social history, or high risk behaviors. During his hospital stay the patient subsequently underwent two more EGDS that showed esophageal inflammation and ulcerations, but no malignancy on biopsy. CT scan with contrast was suggestive of a distal esophageal neoplasm with prominent subcarinal and epigastric lymph nodes along with a lung nodule. The patient’s condition deteriorated with him spiking fevers daily, labs showing leucopenia and inability to tolerate oral intake. At this point the patient was tested for HIV and results showed an absolute CD4 count of 122 (19%) and a viral load of 85,200, confirming a diagnosis of AIDS. CT-guided biopsy of the lung nodule was positive for spherules consistent with coccidioidomycosis. The patient continued to minimally tolerate a liquid diet, and the etiology of the esophageal pathology was still unknown; therefore the patient underwent surgical EGD with biopsies and video-assisted cervical mediastinoscopy. Pathology resulted in a polypoid portion of squamous mucosa of the esophagus, suggestive of squamous papilloma. The patient was discharged home on Bactrim for PCP prophylaxis, Fluconazole, Morphine oral solution, liquid multivitamin, Pantoprazole and an infectious disease follow up appointment for HIV/AIDS treatment and monitoring. This case illustrates the importance of correctly diagnosing esophageal masses; despite signs and symptoms consistent with malignancy the final diagnoses was ultimately a benign lesion.
It is also important to appreciate that although esophageal papillomas are generally benign and usually found incidentally on EGD, in rare cases they can become extremely large and have a profound effect on morbidity. The case also demonstrates the importance of HIV screening as this patient denies any history that would indicate high risk for HIV infection yet at diagnosis he had a low CD4 count and a high viral load. Due to the low incidence and prevalence the link between HIV and esophageal papillomas are unclear. HIV/AIDS puts the host in a chronic inflammatory state that may predispose one to developing or exacerbating esophageal papillomas.

CARDIOVASCULAR COLLAPSE AFTER INITIATION OF BETA BLOCKERS - A CASE REPORT
Sangeetha Murugapandian MD, Abstract 15

INTRODUCTION Pheochromocytomas are rare neuroendocrine tumors arising from chromaffin cells of adrenal medulla and are often diagnosed incidentally. Cardiovascular complications of pheochromocytoma include malignant arrhythmias, left ventricular hypertrophy, hypertensive crisis and dilated cardiomyopathy. Pheochromocytomas can also present with acute coronary syndromes and cardiogenic shock necessitating early recognition of these tumors for appropriate therapy. CASE REPORT We report a case of a middle aged woman with no significant medical history who presented with chest pain, palpitations and diaphoresis for 1 month. Patient was hypertensive (186/100) and tachycardic with an EKG demonstrating ST-elevations in anterior leads. ACS protocol was immediately initiated which included metoprolol. Patient subsequently developed acute shortness of breath, worsening chest pain and hypotension. Emergent cardiac catheterization showed angiographically normal coronaries. A 2D-echocardiography revealed left ventricular ejection fraction (LVEF) <20% with Takotsubo ejection fraction. Patient began to decompensate, requiring inotropic agents, endotracheal intubation and was subsequently transferred to University of Arizona Medical center for possible mechanical circulatory support. CT scan of the abdomen incidentally described a 10x 8 cm right sided adrenal mass. Appropriate biochemical studies to identify the mass including urinary and plasma metanephrines could not be done secondary to patient being anuric and on vasopressors. She improved slowly with supportive measures, was weaned off pressors and extubated. Urine metanephrines and normetanephrines checked thereafter were highly elevated (21075, and 9554 nmol/dl respectively). Alpha blockade was started and clinical improvement followed as was evident from a repeat echocardiogram that showed LVEF >50 %. Complete resolution ensued with surgical excision of the adrenal mass, later definitively diagnosed as malignant pheochromocytoma. DISCUSSION Cardiovascular effects of Pheochromocytomas are partially due to excess catecholamine induced sympathetic stimulation. High index of suspicion is needed before initiating beta blockade therapy in patients presenting with chest pain and accelerated hypertension because unopposed alpha agonistic action may lead to worsening cardiac function and shock. Sympathetic crisis can exacerbate cardiac failure therefore early detection and treatment is essential. Adequate management and optimal recovery of cardiac function is essential before surgery for minimal peri-operative morbidity and mortality. Prompt alpha blockade prior to surgery can improve cardiac function thus increasing chances of successful recovery. Alternatively, emergent
adrenalectomy is sometimes necessary when patient is resistant to standard medical therapy. CONCLUSION This case illustrates the importance of considering pheochromocytoma high in the differential diagnosis of patients who worsen after beta blockade therapy in the absence of other clinical explanation.

EMBOLIC STROKE: A CASE OF MULTIFOCAL CEREBRAL INFARCTION CAUSED BY CARDIAC INVASION OF SMALL CELL LUNG CANCER
Ryan Nahapetian MD MPH, Abstract 63

Secondary tumors of the heart are a relatively common phenomenon found in approximately 10-15% of post-mortem examinations. Cardiac metastases can occur by hematologic spread or direct invasion. The majority of cardiac metastases arise from pulmonary malignancies. Small cell lung cancer (SCLC) has the distinct characteristic of rapid growth and higher likelihood of direct invasion of local structures. Cardiac tumors rarely produce overt symptoms; the most common manifestation is electrical conduction abnormalities evidenced by EKG changes. A rare but devastating complication involves embolization of tumor causing multifocal cerebral infarction. A 73 year-old female smoker with a history of HTN and hypothyroidism was transferred from an outside facility for care of recurrent cerebrovascular accident. Several weeks earlier she was seen at a local hospital for new weakness and visual disturbance, and was diagnosed with an ischemic stroke. A right-sided lung mass was incidentally discovered. She was placed on antiplatelet therapy and discharged home for follow-up evaluation of the lung mass. Prior to follow-up she again presented with acute onset of confusion, deviation of gaze, global weakness, and falls. She was again diagnosed with acute/subacute infarction and was transferred. On admission she was hemodynamically stable although tachycardic, oriented only to self, and unable to give medical history. Neurological exam revealed left-sided facial droop, left-sided neglect, inconsistent ability to follow commands, equal but reduced motor strength in distal extremities, positive Babinski response bilaterally, and brisk motor reflexes on the left compared to the right. Her speech demonstrated word-finding disturbance and decreased word output. Respiratory exam was notable for dullness to percussion and diminished breath-sounds over the right hemithorax. Except for a platelet count of 60k, laboratory analysis was unremarkable. EKG showed normal sinus rhythm, left atrial enlargement and intra-atrial conduction delay. Brain MRI showed acute right middle cerebral artery territory infarction as well as multiple enhancing foci in both frontal lobes, right parietal lobe, left occipital lobe, and left cerebellar hemisphere. CT scan of the chest revealed a 12x15x12 cm heterogeneous mass in the upper right hemithorax with extension of the mass into the left atrium and ventricle via the right pulmonary vein. Echocardiogram was remarkable for a mobile mass with the left atrium. CT guided biopsy of the lung mass was consistent with SCLC. After careful evaluation it was determined that no viable medical, radiological, or surgical options existed. This case demonstrates a rare but known complication of SCLC. Tumor invasion of the left atrium resulted in the showering of emboli to the brain. While cerebral infarct is a common entity, multifocal infarcts and recurrent events should always raise suspicion for occult embolic sources.
REDEFINING RASMUSSEN’S ANEURYSM: A CASE REPORT OF A RARE CAUSE OF PULMONARY PSEUDOANEURYSM

Bhupinder Natt MD, Abstract 127

Redefining Rasmussen’s Aneurysm: A case report of a rare cause of Pulmonary Pseudoaneurysm  Bhupinder Natt MD (1), Varun Takyar MD (1), Eric Chase MD (2), Yuval Raz MD (2) 1. Department of Internal Medicine, University of Arizona Medicine at South Campus. 2. Department of Pulmonology and Critical Care, University of Arizona.

Introduction: Pseudoaneurysm of the Pulmonary Artery (PAP) is an uncommon entity associated with trauma, pulmonary artery catheter malpositioning, vasculitis, congenital defects, infections and, very infrequently cancer. The association of a primary lung carcinoma and pseudoaneurysm formation is exceedingly unusual. Case Report: A 73 year old male was admitted to the hospital with complaints of fever, cough and hemoptysis of six day duration. Intravenous antibiotics were started for presumed community acquired pneumonia. Because of increasing respiratory distress he was intubated and mechanically ventilated. He had copious bloody secretions through the endotracheal (ET) tube. Bronchoscopy showed large amount of blood in the left lower lobe. Post bronchoscopy, the patient had further hemoptysis blocking the ET Tube leading to a pulseless electrical activity (PEA) arrest. He was resuscitated and the ET tube cleared with repeat bronchoscopy and vigorous suctioning. CT angiogram of the chest showed a 1.5 cm x 1.2 cm pseudoaneurysm of one of the branches of the left lower lobe pulmonary artery. The vessel feeding the pseudoaneurysm was embolized using coils. The hemoptysis resolved. Unfortunately because of his poor neurological status, care was withdrawn as per the family’s wishes and the patient expired. Broncho-alveolar lavage and brush biopsy from the initial bronchoscopy showed invasive squamous cell carcinoma.

Discussion: Pseudoaneurysms of the pulmonary artery are rare but life-threatening conditions. Malignancy as a cause of pulmonary artery aneurysm is unusual. The usual presentation in these cases is hemoptysis. Because these aneurysms do not involve all three layers of the vessel wall they are prone to rupture with resulting massive hemoptysis. Therefore prompt recognition and treatment are of paramount importance. Although often treated with surgery, there are increasing care reports of successful embolization of pseudoaneurysms of the pulmonary artery. Rasmussen’s aneurysm is classically a pseudoaneurysm associated with pulmonary tuberculosis. The entity is rare, especially in the western world. The pathophysiology of the pseudoaneurysm formation is due to direct invasion of the infection into the vessel. Similarly in malignancy, the pseudoaneurysm is formed by invasion of tumor into the vessel. The definition of Rasmussen’s aneurysm may therefore be expanded to included malignancy associated pseudoaneurysm formation.

THE CASE OF THE SWOLLEN TONGUE

Chad Nelson MD, Abstract 100

Case Report: A 44 year-old male with a history of L1-L2 fusion and erectile dysfunction on testosterone supplementation presented complaining of a swollen tongue and intense mouth pain for seven months. Initially his symptoms began with swelling and blistering of his tongue and buccal mucosa. These subsequently ruptured and were intensely painful. This oral pain was described as a “burning” sensation. He described difficulty
articulating his words and difficulty eating solid foods secondary to pain. This resulted in a 30 pound weight loss over the ensuing 7 months. He denied any rashes or other skin involvement. He denied any new medications. Evaluation prior to his current presentation included: Otolaryngology with biopsy of affected mucosa, and Gastroenterology with colonoscopy and esophagogastroduodenoscopy. A detailed evaluation was performed. Laboratory work up included negative studies for: HIV, Syphilis, HSV, Coccidiodomycosis, TB, ANCA, MPO, PR3, and ANA. His ESR and CRP were normal. Testing for an immunodeficiency included normal cbc, immunoglobulin levels, and complement levels. A biopsy was performed of the tongue and buccal mucosa revealing mucosal erosion with suprabasilar acantholysis, felt to be consistent with pemphigus. Subsequent testing revealed an elevated anti-desmoglein 3 antibody at 207.89 (normal < 9.0). He had negative antibody tests for anti-desmoglein 1, Bullous Pemphigoid 180 IgG, and Bullous Pemphigoid 230 IgG. Direct immunofluorescence was not possible due to the extremely denuded epithelium. Indirect immunofluorescence was positive for cell surface IgG to monkey esophagus, and negative for rat bladder. This finding is consistent with pemphigus vulgaris, and in combination with his antibody profile nearly rules out paraneoplastic pemphigus. The patient was initiated on prednisone 1 mg/kg daily, and after only 2 weeks reported significant improvement and weight gain. He was subsequently transitioned to a steroid sparing regimen including mycophenolate mofetil and is continuing to do well. Discussion: Pemphigus Vulgaris is a member of rare autoimmune bullous diseases. It results from auto-antibody formation against cell surface adhesion molecules including various elements of the desmosome, especially desmoglein 1 and desmoglein 3. These molecules are concentrated in the superficial layers of the epidermis. This interrupts intercellular adhesion and results in flaccid bullae that easily rupture. Thus, at presentation, patients typically present with ulcerations only. Nikolsky’s sign may be present, but is not specific for Pemphigus Vulgaris. Patients that present with positive anti-desmoglein 3 typically have limited mucosal involvement. However, patients with combined anti-desmoglein 1 and anti-desmoglein 3 have diffuse mucosal and cutaneous involvement. Therapy is initiated with glucocorticoids and transitioned to steroid sparing regimens for long-term management.

First Place – Associate Patient Safety/Quality Improvement Poster:

THE PROVIDER APPROVAL QUEUE AND PATIENT DIAGNOSTIC DATA: HOW RESIDENTS COMMUNICATE IMPORTANT PATIENT INFORMATION
Jonathan Olsen DO, Abstract 130

Purpose for Study: The Internal Medicine Center at Banner Good Samaritan Medical Center serves as the primary continuity clinic site for 60 Categorical Internal Medicine and Medicine Pediatric residents. The residents utilize the Provider Approval Queue (PAQ) in the electronic health record to review patient diagnostic information, consultation reports and to notify patients of results. Each resident is responsible for reviewing this information for their resident clinic partner in addition to their own patient panel. While attempts have been made to standardize this process with resident clinic partners it was not known how residents...
process these data and if patients were consistently receiving results and how the practice partner was kept informed about their own patients. Methods: In order to clarify current processes a survey was distributed to all residents that have an outpatient clinic panel reviewing key areas including frequency the PAQ is checked, how residents communicate with their partners, procedure for updating diagnostic information received, and how residents communicate information to patients. A total of 58 residents were included which included 18 Internal Medicine Pediatric and 40 Internal Medicine residents. A response rate of 48% was achieved. Summary of Results: Several areas of improvement were identified as a result of this survey. When reviewing new imaging or procedure results for patients 58% of residents accepted the task in the PAQ without updating necessary areas of the chart to reflect the result. When asked if the residents had an intern practice partner 75% of residents reported they did not and of those 73% reported they did not know who was checking their PAQ. When asked how often residents communicate important patient information to their practice partner 65% responded, "as needed". Conclusions: This survey demonstrated that important patient diagnostic information was not appropriately being communicated to the patient’s resident provider and not being updated in the patient’s chart. As an immediate result, the clinic attendings organized interns and residents into teams to ensure that all PAQs were being reviewed on a regular basis. Residents were instructed to standardize communication of important patient updates by sending a message to their resident clinic partner through the electronic health record ensuring notification and documentation in the medical record. It is expected that these changes will ensure that patient diagnostic information is communicated to the healthcare team and updated in the chart to facilitate quality care and up to date annual screenings. Future studies could investigate how often patients are receiving test results and their preferred method of notification.

WHATS EATING YOU?: RARE CASE OF ERDHEIM-CHESTER DISEASE
Jabraan Pasha MD, Abstract 58

WHATS EATING YOU?: A rare case of Erdheim-Chester disease Jabraan Pasha M.D. PGY3 Mayo clinic Scottsdale INTRODUCTION: Erdheim-Chester disease is a rare non-langerhans histiocytic disorder that causes osteosclerotic bone lesions with or without extra-skeletal involvement. CASE REPORT: 61 yo male in previously good health who has been experiencing increased fatigue, dyspnea, lower extremity swelling and lower leg pain over the last 6 months. He also endorses a 15 lb weight gain over the last 3 months. Patient denies any cough, orthopnea or fever. On exam, all vitals proved to be within normal limits. Pertinent physical exam findings included a cardiac exam without abnormalities, diminished breath sounds at lung bases bilaterally and 3+ pitting edema of the lower extremities to the mid-calf. Labs revealed a Hgb of 11.3 g/dL, and a WBC count of 12.5 g/dL. Basic metabolic panel was within normal limits. C reactive protein was elevated at 21.1 and antinuclear antibody was positive. Chest x-ray was significant for bilateral pleural effusions. Lower extremity plain films showed a “moth-eaten” appearance of the proximal and distal tibias. This was followed up by a bone scan that showed osteoblastic activity of several long bones and the maxilla. A diagnostic thoracentesis was performed and resulted in a hemothorax the
required open drainage. During the procedure, biopsies of pleura and pericardium were performed. Pathology showed histiocytic infiltration that was consistent with Erdheim-Chester disease. He was placed on long-term corticosteroid therapy and showed significant improvement on follow-up. DISCUSSION: Erdheim-Chester disease is a rare non-langerhans histiocytic disorder that involves bones, with or without extracellular involvement. Common extra-skeletal sites of involvement include the lungs, heart, CNS, eyes and skin. Erdheim-Chester has proven to be a very rare disorder, with less than 500 cases in the literature. It has been seen in all age groups but affects adults predominantly. There is a slight male predominance and the mean age of onset is 54. Treatment options include: interferon, systemic chemotherapy, glucocorticoids and palliative radiation. Disease severity and outcomes vary greatly, ranging from mild disease without need for treatment to severe disease with fatal complications.

DUSTING OFF THE HEART: A CASE PRESENTATION OF COMPRESSED AIR DUSTER CAUSING SEVERE CARDIAC TOXICITY WITH RECURRENT VENTRICULAR FIBRILLATION

Reena Patel MD, Abstract 6

Introduction: Compressed air duster spray is used to clean electronic equipment. It contains 1,1-Difluoroethane (DFE), a hydrocarbon gas, which when inhaled causes euphoria. When abused, DFE may lead to devastating consequences. Symptoms from intoxication with DFE are not well described in literature. Case report 40-year-old man was brought to Emergency Department after being found unresponsive in a store parking lot with eight canisters of UltraDusterTM in his vehicle. Emergency Medical Services personnel diagnosed Torsade De Pointe which was followed by ventricular fibrillation. The patient underwent defibrillation and Advanced Cardiac Life Support measures were started on scene. Upon arrival to the Emergency Department the patient had 3 more episodes of ventricular fibrillation each of which resolved after bi-phasic defibrillation. The patient was transferred to the Medical Intensive Care Unit where therapeutic hypothermia was started two hours after his last episode of ventricular fibrillation. Unfortunately, about twenty minutes into the therapeutic hypothermia protocol, the patient’s condition worsened and he had recurrence of his ventricular fibrillation after which the hypothermia was halted. In all, the patient had 120 episodes of ventricular fibrillation and required >120 defibrillations within the first 12 hours of his ICU stay. His ventricular fibrillation was refractory to the anti-arrhythmic medications amiodarone, lidocaine, and magnesium. It only responded to bi-phasic defibrillation. His electrocardiogram was significant for a prolonged QTc. Subsequently the patient developed cardiogenic shock requiring the use of an intra-aortic balloon pump and vasopressors, rhabdomyolysis, acute renal failure, encephalopathy, severe metabolic acidosis, disseminated intravascular coagulation, intestinal ischemia and a gastrointestinal bleed. Cardiac catheterization revealed patent and clean coronary arteries. Amazingly, the patient’s condition stabilized and despite the multiple complications he improved. He was discharged from the hospital 11 days after his admit. Discussion: DFE stimulates GABA and NMDA receptors in the brain causing a sensation of euphoria. It also sensitizes the myocardium to catecholamines, which can cause difficult to treat arrhythmias as
demonstrated in this case. Prolonged QT and death have been described after using DFE in what is known as Sudden Sniffing Death Syndrome. In one study on dogs Epinephrine after DFE inhalation caused immediate death from malignant cardiac arrhythmias. DFE has a short half-life but reaches very high concentration in heart and kidney thus causing direct myocardial and renal damage.

THE DOWNSIDE OF BEING TOO FLEXIBLE
Ali Raoof MD, Abstract 111

Ali Raoof, MD, University of Arizona South Campus; Harold Szerlip, MD, University of Arizona South Campus

Ehlers-Danlos Syndrome (EDS), is clinically a heterogeneous group of conditions generally characterized by hyperelasticity, fragility of the skin and hypermobility of the joints. It comprises of more than 10 different inherited disorders; all involve a genetic defect in collagen and connective-tissue synthesis and structure that can affect the skin, joints, and blood vessels. Oftentimes it goes unrecognized. A correct diagnosis requires a thorough history and physical. A 30 year old female with no significant past medical history presented to the hospital with complain of sudden onset right lower extremity pain while walking. She recalled noticing a “snapping” sensation immediately prior to the onset of her symptoms. The pain was localized and was limiting her ability to ambulate. On examination there was diffuse tenderness involving the entire right lower extremity below the knee with decreased dorsiflexion and plantarflexion. Sensation to light touch and pain were intact and there were good peripheral pulses. An MRI was obtained which showed partial-thickness tears of the extensor digitorum longus, extensor hallucis longus, proximal tibialis anterior and tibialis posterior muscles with associated superficial and deep soft tissue edema. The etiology of spontaneous muscle tear in an otherwise healthy young female was not entirely clear. A careful review of her history revealed that she had been double jointed since she was a child and had previous history of joint dislocations and muscle tears. She denied any history of syncope or visual disturbances. However, she did report a history of easy bruising without major bleeding complication. On examination, she had soft skin with increased extensibility; she was hypermobile in the small joints of her hands as well as her elbows and knees meeting eight points out of the nine-point Beighton scale. She had a normal arched palate and a normal eye exam. Based on the above findings a diagnosis of EDS Type III (hypermobility type) was made. The diagnosis was formulated on the basis of two major criteria (skin hyperextensibility and joint hypermobility) and two minor criteria (recurrent joint dislocations and easy bruising). Ehlers-Danlos Type III syndrome has autosomal dominant inheritance. Joint hypermobility is the main symptom which affects the large as well as the smaller joints like the fingers. Dislocations are common, joint pains can be chronic and severe. Even though, spontaneous muscle rupture is not classically associated with EDS Type III, more than one third of persons with EDS do not fit exactly into a single type and overlap is common. Diagnosis is based entirely on clinical evaluation and family history. Management focuses on symptom prevention and treatment of disease manifestations.
A ZEBRA HIDING IN PLAIN SIGHT: A CASE OF PML
Michael Richins DO, Abstract 90

A 78-year-old male with chronic lymphocytic leukemia (CLL) and IgG-deficiency presented to his primary care provider’s office for increased confusion. His treatment included intravenous Ofatumumab and monthly IgG therapy. Six months prior to this visit, the patient had abruptly lost the ability to tell time. Then, four months later, he lost the ability to remember dates or time, and could not read. It progressed to where the patient would get lost in his own house. His speech was fluent, but nonsensical. His wife believed he had right-sided weakness, and because of this, he was sent to a tertiary neurological center. His symptoms were felt to be related to his CLL, and an open MRI of the brain was performed. The findings were suspicious for a mass-effect in the occipital area of the left lateral ventrical. There was enhancement presumed to be due to an evolving subacute left posterior cerebral-artery territory infarct, although diffusion-weighted imaging was not available due to the open MRI. A small amount of edema was seen, and an early tumor, mass, or abscess was not ruled out. When the patient followed up with his primary physician, a right homonymous hemianopsia was discovered, but the patient did not have any right-sided symptoms. At this point, the patient had difficulty with word finding and comprehension, and oriented to name only. Because of the significant change spanning two months, he was admitted to the nearby community hospital. Vital signs were stable. The exam was difficult due to the inability to follow commands, but the right homonymous hemianopsia was still present. He had no facial droop, and his Babinski tests were up-going on the left and down-going on the right. Blood work and cultures showed no obvious metabolic cause of his symptoms. A repeat brain MRI was performed, and Neurology was consulted. This MRI showed somewhat restricted diffusion involving the cortical gray matter of the left occipital lobe, the adjacent subcortical and deeper white matter. Also involved was the deep white matter in the left frontal lobe extending slightly into the internal capsule and the subcortical right posterior parietal white matter at multiple adjacent locations. At that point, Progressive Multifocal Leukoencephalopathy (PML) was suspected due to the slow progression of symptoms, and his immunocompromised state. Cerebral spinal fluid was obtained and tested positive for the JC virus. The patient’s family was made aware of the results, and was given the treatment options. They decided to take the patient home to comfort care, and he expired three months later. This case reiterates the importance of keeping an open differential diagnosis, and that one can find rare diseases even in small towns.

HEMORRHAGIC PANCREATITIS WITH MINIMAL SYMPTOMS: CASE REPORT
Farhad Sahebjam MD, Abstract 45

Introduction Hemorrhagic pancreatitis is a rare condition in patients with acute and chronic pancreatitis. These patients usually complain of severe abdominal pain, nausea and vomiting. Sepsis and septic shock may complicate the disease course, requiring prolonged admissions to ICU. We present an unusual case of hemorrhagic pancreatitis with surprisingly no symptoms except for jaundice. Case Report A 46 years old male with history of alcoholism and chronic pancreatitis was referred to our hospital for evaluation of new onset jaundice and mild ascites. He was afebrile and in general...
was not in acute distress. Total bilirubin was 19 with direct bilirubin of 13.4. Ultrasound of abdomen showed extrahepatic biliary duct dilatation and a possible pseudocyst in the head of the pancreas as well as moderate ascites. A diagnostic paracentesis was performed which showed grossly bloody fluid, with a serum albumin ascites gradient of 0.8 mg/dl. Lipase in the ascitic fluid was 250 mg/dl, significantly higher compared to serum lipase of 52 mg/dl, ascites fluid RBC count was 911 per microliter. MRI of the abdomen showed a diffuse heterogeneous pancreatic signal abnormality seen on dynamic contrast enhanced T1 sequences with increased scattered foci of unenhanced T1 signal throughout the entirety of the pancreas consistent with acute/subacute hemorrhagic pancreatitis. Broad spectrum antibiotics were started and conservative management was pursued. Surprisingly patient remained relatively asymptomatic. His jaundice and ascites resolved spontaneously after 12 days and patient was discharged home.

Unfortunately, he did not return for his follow-up appointment. Discussion Although patients with hemorrhagic pancreatitis usually present severely ill with sepsis and septic shock, some patients may present with less severe symptoms. This patient was relatively asymptomatic and only had new onset jaundice and ascites. Paracentesis revealing bloody fluid and elevated lipase led to the correct diagnosis. MRI which is the diagnostic test of choice confirmed hemorrhagic pancreatitis. The astute clinician needs to recognize that even severe disease may present with relatively benign complaints.

WHEN ACTING DRUNK ISN’T FROM ALCOHOL  
Pratik Shah MD, Abstract 107

Case Report  A 70 year old female presented with a one day history of slurred speech, dizziness and ataxia. The patient’s family reported that she had a history of recurrent episodes of dizziness, chronic diarrhea and frequent falls for least one year and underwent an extensive work-up with no clear etiology identified. The patient’s multiple falls had resulted in bilateral wrist fractures and a right shoulder fracture which were surgically repaired. The patient’s past medical history was significant for diet-controlled type 2 diabetes mellitus, benign hypertension, asthma, sick sinus syndrome status post permanent pacemaker placement, and gastric bypass for morbid obesity. Her home medication included ferrous sulphate, lomotil, lisinopril, flonase, fosamax, calcium, vitamin D, aspirin, albuterol, advair, prilosec and percocet. No history of alcohol, tobacco, illicit drug use or toxic ingestion was reported. On physical examination vital signs were normal. The patient had slurred speech but no focal neurological deficits. The rest of the physical examination was unremarkable. Complete blood count was within normal limits.

Comprehensive metabolic profile showed sodium 143, potassium 3.9, Chloride 120, bicarbonate 6, BUN 9, creatinine 0.5, anion gap 17, glucose 75, AST 14, ALT 27. ABG showed pH 7.16, pCO2 14.7, pO2 128, HCO3 5, base excess -23. Lactic acid was 2.3 Urine toxicology screen was negative. Beta hydroxybutyrate was 0.8. Acetaminophen and aspirin levels were unremarkable. Urine study showed pH 5.5, specific gravity 1.010, trace protein, negative ketones. Serum ethanol, methanol and ethylene glycol level were negative. CT head without contrast showed no acute intracranial abnormality. MRI brain couldn’t be done because of pacemaker placement. Although D lactic acid was sent after starting intravenous
bicarbonate, it was found to be elevated at 3.7 mmol/L (normal <0.2 mmol/L). The patient was diagnosed with D-lactic acidosis secondary to gastric bypass done for morbid obesity. She was started on a low carbohydrate diet, intravenous bicarbonate and metronidazole which resulted in improvement in her neurological symptoms within 24 hours. A repeat ABG on day 2 showed pH 7.39, pCO2 32, pO2 98.2, HCO3 19.1, base excess -4. At the time of discharge her neurological symptoms had completely resolved and she was transitioned to sodium citrate, oral metronidazole and low carbohydrate diet.

Key Points: 1. D Lactic acidosis should be suspected in cases of metabolic acidosis where there is no apparent etiology of acidosis and patient has short bowel syndrome or chronic exocrine pancreatic insufficiency with high levels of D lactate. 2. Clinical presentation is characterized by recurrent episodes of unusual neurological manifestations and severe metabolic acidosis. 3. Low carbohydrate diet, bicarbonate therapy and poorly absorbed antibiotics help to control the symptoms.

INCORPORATION OF MEDICAL SIMULATION TRAINING INTO MORNING REPORT FOR INTERNAL MEDICINE RESIDENTS

Mutende Sikuyayenga MD, Abstract 133

Purpose: Each year medical errors claims up to 98,000 lives in the U.S. Following its wide acceptance in non-medical fields, studies have demonstrated that simulation can be applied in medicine to improve practitioner skills, develop better teamwork, enhance safety, and decrease medical errors. Mayo Clinic Internal Medicine Residency program trains more than 30 residents in a mid-size multi-specialty hospital and outpatient centers in the Phoenix metropolitan area. Given the complexity and multi-organ nature of pathology that residents are expected to manage from day one, the potential for errors is high. As such, our high-volume simulation center has been utilized for the ongoing training of residents in the most common medical scenarios they are likely to encounter. The purpose of this article is to outline the implementation of this program and its overall success in the ongoing training of post-graduate resident physicians. Methods: After identifying the common sources of errors and near-misses, medical simulations addressing the most common medical mistakes and learning points have been incorporated into weekly morning reports for internal medicine physician on hospital rounds. The outcome of the each case depends on the trainees’ efficiency in collecting pertinent information, prompt ordering of appropriate diagnostic tests, and initiation of therapeutic intervention in timely manner. Debriefing is provided after each session giving residents a chance for self-critique, identification of shortcomings and potential remedies. Results: Overall, the addition of medical simulation training to morning reports for hospital internal medicine residents has been well accepted and efficacious in providing ongoing training. Numerous resident surveys using the Likert scale show increase in overall resident satisfaction and confidence level. A study conducted by the chief residents in 2011 demonstrated the value of simulation training in improving resident core knowledge and skills, level of confidence, and team communication. About a third of residents took part in the study which tested core knowledge and confidence in managing atrial fibrillation and status epilepticus. Traditional didactic training resulted in significant expansion in core knowledge and boosting of confidence level by, respectively, 37% and 19.8% for atrial
similar results were achieved in status epilepticus as well, 23.3% increase in core knowledge and 15.9% rise in confidence level. There was however substantial additional improvement noted following simulation-based training using high-fidelity medical mannequin for both conditions. Conclusion: Clinical teaching on rounds and didactic lectures will certainly remain the cornerstone of physician training. However, with the advance in simulation technology, we offer this approach as a valuable adjunct to boast knowledge, confidence, procedural skills, team-work, and safety in order to decrease medical errors.

PANCREATICOBILIARY ADENOCARCINOMA PRESENTING AS LEFT URETERAL OBSTRUCTION
Jasmine Smith MD MBA, Abstract 32

INTRODUCTION  Pancreatic cancer is the fourth leading cause of cancer related death in the United States. The most common sites of metastases include the liver, peritoneum, lungs and bone. Metastases to the bladder wall are uncommon and ureteral obstruction has rarely been reported. A case of pancreaticobiliary carcinoma with bladder metastases and left ureteral obstruction is described. CASE REPORT We present a case of a 69 year old man with history of presumed congenital UPJ obstruction causing an obstructive uropathy status post bilateral ureteral stent placement, who presented to the Emergency Department (ED) with nausea, vomiting and weakness. The patient had been diagnosed with a methicillin resistant staphylococcal aureus (MRSA) urinary tract infection (UTI) after ureteral stent placement three months prior to hospital presentation. A colonoscopy performed one and a half years prior to presentation demonstrated no adenomatous lesions. Initial ED evaluation demonstrated an acute on chronic kidney injury. Subsequent blood and urine cultures demonstrated growth of MRSA. A renal ultrasound and subsequent Computed Tomography scan of the abdomen and pelvis without contrast (given the kidney injury) was performed and was notable for bilateral hydronephrosis and omental nodular lesions concerning for carcinomatosis. Given concern for worsening ureteral obstruction and the potential of the ureteral stent as a nidus of infection, a cystoscopy with plan for ureteral stent exchange was performed. This demonstrated significant extrinsic compression of left ureter with necrotic appearing tissue at the left ureteral orifice. The compression appeared to compromise the patient’s ureteral opening preventing successful stent exchange. Pathology report of the biopsied lesion was an invasive gland forming carcinoma, differential including a primary urothelial source, prostate metastases or metastases from an unknown primary. A flexible sigmoidoscopy was performed, though advancement of the scope was hindered by significant diverticulosis. Four days later, the patient developed peritonitis. Exploratory laparotomy demonstrated a large sigmoid mass with evidence of intestinal perforation. Omental and liver lesions concerning for metastases were also identified. Final pathology of resected specimens was consistent with poorly differentiated adenocarcinoma of pancreatobiliary origin. Given the lack of painless jaundice at presentation, it is felt that the patient’s carcinoma was more likely pancreatic in origin. The patient was discharged to hospice care and died 73 days after initial presentation. DISCUSSION The cure rate of pancreatic cancer remains poor secondary to late presentation of disease. This case illustrates the extent of metastatic spread of pancreatic tumors, and reminds clinicians not to limit the range of
neoplasms. Additionally, while the United States Preventative Task Force provides guidelines for colorectal cancer screening, adenomatous lesions can be missed (as in this case). Clinicians must be prudent in pursuing diagnostic modalities when necessary, especially when a clinical picture does not fit the norm.

THE GASP THAT KEEPS ON GASPING: NEGATIVE PRESSURE PULMONARY EDEMA, A CASE SERIES AND REVIEW OF THE LITERATURE

Nicholas Sparacino DO, Abstract 119

Introduction. Negative-pressure pulmonary edema (NPPE) is a rare (0.05 to 0.1% of general anesthetic cases) but potentially lethal entity most commonly encountered as a result of postoperative laryngospasm; however it can develop in a variety of disease states. The following series describes two cases of NPPE in disparate patients, followed by a review of the literature. Case 1. MB is a 32 year old male with a history of mild asthma, Graves disease status post radioiodine ablation and recently diagnosed seminoma, metastatic to lung and peritoneum. A radical orchiectomy was planned as the initial treatment. Pre- and intra-operatively, MB oxygenated well and the procedure was performed without complication. Upon extubation, the patient was noted to gasp violently, after which he rapidly desaturated to 80% by pulse oximetry on 100% oxygen, requiring re-intubation. Chest radiography revealed the interval development of pulmonary edema; no other investigations revealed a cause of his acute respiratory distress. Negative-pressure pulmonary edema was diagnosed. Furosemide was administered with excellent diuretic result, and the patient was rapidly weaned and extubated without complication the following day. Case 2. VD was a 49 year old male with a history of diabetes, hypertension, dyslipidemia, and obstructive sleep apnea treated with positive pressure ventilation nocturnally. He was taken to the operating room for an elective knee arthroscopy. The procedure was performed without complication, until upon extubation the patient’s pulse oximetry dropped to 78%. VD was then treated with bronchodilators, and was able to maintain his oxygen saturations at adequate levels with supplemental oxygen of 6 liters per minute, despite having no need for oxygen prior to this. A chest x-ray demonstrated pulmonary vascular congestion, and further investigations failed to yield a cause for his respiratory decompensation. NPPE was diagnosed, and the patient was treated with diuretics, similarly recovering rapidly with diuresis. Discussion. NPPE was first proposed in the early 1900’s, but not demonstrated until 1927 in a canine model. The pathophysiology is complex, and involves a strong inspiratory effort against a resistive load, creating a negative intrathoracic pressure of up to 140 centimeters of water (normal -3 to 0 cm H2O). This creates a dramatically increased preload and subsequent intrathoracic vascular volume. Thus a situation in which pulmonary microvascular engorgement and an extreme capillary transmural pressure gradient co-exist, resulting in rapid development of pulmonary vascular congestion from the mismatch in Starling forces. While this entity is well described in the anesthesia literature, it is rarely described in the general medical literature. With a continuing trend toward a “hospitalist with consultants” model, facility with this disease state is essential among internists. Furthermore, NPPE tends to develop in more robust patients in whom fewer complications are expected, adding to the need for vigilance.
SUCCESSFUL TREATMENT OF A LIFE THREATENING FUNGAL INFECTION OF THE STOMACH

Charles Stauffer MD, Abstract 74

Introduction: We report a patient with a life threatening infection of the stomach due to one of two unusual fungi. Mucor and Basidiobolus are two morphologically similar fungi. Infection by either of these two fungi is rare and identifying which fungus is causing the infection is difficult. Infections by Mucor are rapidly progressive and life threatening. In contrast, Basidiobolus infections tend to be indolent and unlikely fatal. Correctly identifying which of these two fungi is the cause of an infection is important because they are treated differently. We present a patient with life threatening hematemesis and stomach infection caused by an uncertain fungal agent. Case Report: A 25-year-old female with diabetes mellitus was admitted to the hospital with sepsis and diabetic ketoacidosis. She was critically ill and her course was complicated by renal failure, Clostridium difficile colitis, cardiomyopathy and gastrointestinal bleeding. Work-up included a gastric biopsy which showed “mucormycosis” on pathology. Amphotericin was initiated. However, she had a recurrence of significant hematemesis and hypotension requiring blood transfusion. Amphotericin was discontinued. Upon review of the stomach biopsy results, considering the patient’s history of diabetes, clinical course and fungal epidemiology, the infectious disease team felt that she most likely had a Basidiobolus infection rather than mucormycosis. The patient was started on posaconazole and within 48 hours her symptoms resolved and she was discharged a few days later. Discussion: The treatment dilemma was not having a specific fungal diagnosis which differentiated between the two fungal agents, Mucor and Basidiobolus. Both organisms have nonseptate hyphae and are morphologically similar. Mucor infections tend to be rapidly destructive and infections of the gastrointestinal tract are very rare. In contrast, Basidiobolus infections of the gastrointestinal tract are endemic to Arizona, but the infection is more indolent and less likely fatal. Epidemiological studies on Basidiobolus suggest that the risks for this infection include exposure to Southwestern desert soil, diabetes and medications that suppress stomach acids. Our patient had these risk factors making Basidiobolus the leading consideration. However, the treatment of Basidiobolus is different from Mucor. Basidiobolus is resistant to amphotericin and the preferred treatment is itraconazole. Our patient was initially started on amphotericin because of the reported mucormycosis on biopsy. Amphotericin is the drug of choice for Mucor, but is not effective for Basidiobolus. The treatment decision was made to use posaconazole as it is the only agent effective for Mucor, when amphotericin cannot be used. It is a broad spectrum azole antifungal that should have activity against both Basidiobolus and Mucor. This patient was treated effectively with posaconazole based on decisions made while the patient was clinically ill.

PROBABLE ENDOTIPSITIS FOLLOWING TRANS-JUGULAR INTRAHEPATIC PORTOSYSTEMIC SHUNTING (TIPS)

Thomas Taylor DO, Abstract 40

Introduction: Endotipsitis is a rare but serious complication of Transjugular intrahepatic
portosystemic shunting (TIPS). Though the term endotipsitis lacks a uniform definition, it has been initially described as 1) the presence of persistent bacteremia indicating an infectious focus in the systemic circulation. 2) Failure to find an alternate source of infection despite an extensive search. Though postoperative fever and transient bacteremia have been identified in as many as 25% of patients receiving TIPS, the overall incidence of endotipsitis is unknown due to difficulty of diagnoses and lack of a uniform definition.

Case Presentation: A 49 y/o Mexican American male presented to our hospital s/p transjugular intrahepatic portosystemic shunt (TIPS) secondary to gastroesophageal variceal bleeding as a complication of portal hypertension from years of alcoholic liver disease. He developed a persistent bacteremia with staphylococcus epidermidis secondary to suspected central line placement. The central line was removed and repeat blood cultures were again positive x2 with the same organism after a two week course of high dose antibiotics. The patient was again started on antibiotics x 4 weeks and a transthoracic echocardiogram was ordered and negative for signs of endocarditis. Though an abdominal ultrasound revealed no vegetations, the TIPS was patent but with slow flow. Conclusion: Endotipsitis is a rare but potentially fatal complications of TIPS procedures. The diagnosis is difficult and requires a high index of suspicion as multiple imaging modalities are needed to rule out other sources of endovascular infection. Endotipsitis should be considered in the differential diagnosis in TIPS patients who develop persistent bacteremia and unremitting fever with no other obvious source of infection. Once a probable diagnosis of endotipsitis is reached, the a long course of antibiotics is started as it is impractical to remove the infected stent. An alternative option is liver transplant. This case report demonstrates an unremitting fever, persistent bacteremia, and no other obvious source of infection, as a probable diagnosis of endotipsitis.

Third Place – Associate Case Report Poster:

CALCIFIC UREMIC ARTERIOLOPATHY - AN Atherosclerotic Masquerade
Preethi William MBBS, Abstract 17

Calcific Uremic Arteriolopathy(CUA)- an atherosclerotic masquerade  Preethi William,MD; Bijin Thajudeen,MD; Eva Maria Anwer,MD; Mordecai Popovtzer ,MD.
University of Arizona, Tucson  Introduction: We discuss a case with two diagnostic challenges - A case of Calcific Uremic Arteriolopathy masqueraded by atherosclerotic disease. CUA is a poorly understood disease causing vascular calcification and skin necrosis resulting in high mortality from sepsis and cardiovascular events. It occurs in 1-4% of ESRD patients annually. Case description: 65 year old Hispanic male with end stage renal disease on peritoneal dialysis for 2 years presenting with excruciating painful lesions involving extremities. He was diagnosed with atherosclerotic disease one year ago and underwent endovascular intervention with progressive lesions despite intervention. Significant past medical history include type 2 DM, hypertension and ESRD on PD (2 yrs) ; Medications included calcium acetate, calcitriol. Patient was on Warfarin 1 year ago for DVT for 6 months. Physical exam showed dry gangrene of fingers and toes including multiple ulcers covered with dark eschar involving hands, forearm, legs , dorsum of foot and heel with feeble peripheral pulses in upper and lower extremities. Diagnostics over the last
few months showed calcium 10-11 mg/dL, phosphorus 5.5-6.5 mg/dL, PTH 350-400 pg/ml. CT Angiography left upper extremity showed diffuse atherosclerotic calcifications and stenosis of brachial, ulnar and radial arteries for which he underwent ulnar artery exploration, patch angioplasty and finger amputations. At the onset, patient was treated as atherosclerotic disease. However due to predisposing factors and distribution of ulcers, possibility of CUA was raised and skin biopsy showed gangrenous necrosis of soft tissue with calcification of the subcutaneous arterioles consistent with CUA. Calcium supplements and calcitriol were stopped. Peritoneal dialysis was replaced by hemodialysis with low calcium bath, non calcium based phosphate binders, thrice a week intravenous sodium thiosulphate and wound care were initiated with some improvement in the skin lesions. Patient eventually died of sepsis. Discussion: Diagnosis of calciphylaxis could be missed in presence of atherosclerotic peripheral vascular disease. This case highlights formidable diagnostic challenge either due to underreporting of cases or lack of recognition and misdiagnosis of atherosclerotic disease. A well defined natural course of the disease has still not been established and no randomised controlled trial for treatment options is available to prevent morbidity and mortality associated with the course of this disease. A high index of suspicion is required especially in the background of predisposing factors. Just as risk stratification has been emphasized in atherosclerosis, simultaneous reduction of risk factors like bone mineral metabolism, sensitizing and challenging agents in CUA is essential.

“A NOT SO OBVIOUS CAUSE OF RASH” – A CASE OF DELAYED ONSET COUMADIN HYPERSENSITIVITY

Panagiotis Zervogiannis MD, Abstract 113

Coumadin is an oral Vitamin K antagonist used in the prevention and management of thrombosis and thromboembolism. Several reactions associated with its use have been described– the most serious of which are hemorrhage and anaphylaxis. Although, rash and urticaria has been documented these skin reactions are relatively uncommon occurring in less than 1% of patients over their lifetime. Given the widespread use of Coumadin, such hypersensitivity reactions while rare, pose a significant risk and morbidity to the population taking these medications. We describe a case report of an 83y/o Hispanic male with a delayed onset urticarial reaction to Coumadin. The patient had a past medical history hypertension and paroxysmal atrial fibrillation on Coumadin since April of 2007. The patient began noticing an urticarial pruritic rash in July of 2011 that began under his armpits and spread caudally involving his arms, trunk, groin, and lower extremities. The patient was seen by three dermatologists with two punch biopsies performed of the lesions, and medication changes made gradually on multiple clinic visits. The initial pathology of the skin biopsy revealed superficial perivascular dermatitis consistent with urticarial/hypersensitivity reaction. The patient was subsequently treated with Prednisone and antihistamines with moderate resolution of his symptoms. However, shortly after discontinuation of prednisone the patient’s rash and pruritus reemerged. Repeat biopsy revealed eosinophilic spongiosis, dermal edema, superficial dermal lymphoepithelioid infiltrate which was PAS negative and most consistent with urticarial/hypersensitivity
reaction as seen in the initial biopsy. The patient was then managed with topical triamcinolone ointment, hypo allergic soaps, and gradual medication changes to determine the causative agent involved in the hypersensitivity reaction. Because the patient had been on Coumadin for over 5 years, this medication was never suspected as a possible causative agent. In anticipation of a screening colonoscopy the patient was taken off Coumadin. After being off the medication for only three the patient noted a dramatic decrease in his symptoms of pruritus and a receding of his urticarial rash. Following the colonoscopy Dabigatran was substituted for Coumadin with complete resolution of his symptoms. This case illustrates that delayed type hypersensitivity reactions can occur with medications not commonly known to cause such reactions. The effects on quality of life and overall morbidity are significant enough that recognition that such reactions may be caused by medications the patient has been on long before the reaction had ever occurred.

MORBIDITY AND MORTALITY IN NON PROTEINURIC TYPE 2 DIABETES MELLITUS PATIENTS WITH CHRONIC KIDNEY DISEASE-A RETROSPECTIVE STUDY
Bijn Thajudeen MD, Abstract 30

Background: Diabetic nephropathy (DN) is the most common etiology for end stage renal disease (ESRD) in the developed world. Twenty percent of the diabetic’s progress to chronic kidney disease without albuminuria. Determinants of chronic kidney disease, natural course of renal dysfunction and mortality in this subgroup of diabetics has not been studied in great detail. Methods: In a retrospective study design we followed medical records of 121 patients (all males) above the age of 40 years with Type 2 DM and chronic kidney disease in the absence of proteinuria for a mean follow up period of 5.1 years. Urine dipstick protein should be negative for three consecutive years. CKD is defined as eGFR less than 60 ml/min/1.73m2 for three consecutive years. Average of three eGFR was taken as the baseline eGFR. Variables like age sex, presence of retinopathy, systolic blood pressure, diastolic blood pressure, Hba1c, serum potassium, smoking, lipid panel, duration of diabetes, Hemoglobin, potassium, MI history of smoking, exposure to contrast, hypertension, use of Angiotensin converting enzymes, Angiotensin receptor blocker, Statins and NSAIDS, at time of diagnosis of chronic kidney disease were looked into. Primary outcome measured were all cause mortality and requirement for hemodialysis. Secondary outcomes include 1) appearance of proteinuria 2) trend in the kidney function expressed as improvement or worsening of eGFR. Results: All-cause mortality was 33% with mean age of death of 75.9. 63% of the patients had improvement in eGFR at end of follow up period. Mortality was found to be higher in patients who had worsening eGFR over time compared to those who had improvement in eGFR (61.3 % vs. 38.7 %, p value 0.045).Most common cause of death was cardiovascular disease.5.8% of the patients ended up in hemodialysis.16% of the patients developed proteinuria at end of follow up and theses patients showed higher tendency for progression of renal failure. Multivariate logistic regression for improving versus worsening eGFR trend revealed no statistically significant predictors: blood pressures, BMI, HbA1c, Smoking status, the use of NSAIDS, and use of ACE inhibitors or ARB. Conclusions: This
observation study suggests that in Type 2 diabetic patients with chronic kidney disease, progression of renal failure may be a marker of increased mortality irrespective of proteinuria status. A substantial number of patients will have improvement in eGFR over time which could mean that careful search for kidney damaging causes that are potentially reversible is recommended. This could subsequently improve mortality.

**ORAL VIGNETTES**

First Place – Oral Clinical Vignette:

BAMBOOZLED BY SYNCOPE
Jonathan Olsen, DO, Banner Good Samaritan Regional Medical Center, Abstract 134

**Introduction**
Amyloidosis refers to the group of disorders that share the common feature of insoluble fibrillar protein deposition into organs and tissue. Though a rare condition, unless considered, it may go undiagnosed. Properly categorizing amyloidosis into one of three major categories can help with selecting an appropriate diagnostic approach.

**Case Presentation**
A 57 year-old female with no significant past medical history presented for expedited work-up of her neuropathy and severe orthostatic hypotension. She endorsed a one-year history increasingly severe bilateral lower extremity pain, burning and numbness, starting in her toes and extending proximally to her mid-calf. Additionally, she complained of lightheadedness on standing and occasional syncope, becoming more frequent and debilitating over the past few months. Home blood pressure readings were as low as 40/30 mmHg standing. She had been started on gabapentin for the neuropathy and fludrocortisone and midodrine for the orthostatic hypotension. She underwent extensive outside work-up prior to admission to our facility. Neurologic work-up, including MRI and MRA of the head and neck, electromyography, tilt table testing and sural nerve biopsy failed to achieve a diagnosis. Extensive endocrine and cardiac work-up were unrevealing. On presentation, physical exam was significant for orthostatic hypotension without a change in her heart rate. Neurologic exam revealed bilateral ptosis, decreased pinprick sensation in her feet bilaterally and absent vibratory sense in the toes. The differential diagnosis of a combined peripheral and autonomic neuropathy remained broad, including multi-system atrophy, multiple sclerosis, paraneoplastic syndrome and amyloidosis, among other etiologies. Additional work-up, including a fat pad biopsy in search of amyloidosis, was negative. However, when the transthyreitin antibody testing resulted as positive, additional investigation ensued. She underwent endomyocardial biopsy, which was confirmatory and she was diagnosed with transthyreitin amyloidosis, a hereditary disease.

**Discussion**
Though fat pad biopsy is often employed in the initial work-up of amyloidosis, a negative result and high clinical suspicion should warrant further investigation. Subsequent testing should be chosen based on the category of amyloidosis suspected. Primary (AL) amyloidosis, which results in monoclonal immunoglobulin light chain deposition, is related to multiple myeloma and fat pad aspirate is positive in 90% of patients with AL amyloidosis. Additional key diagnostic testing includes serum and protein electrophoresis and bone marrow biopsy with staining for alpha and kappa light chains. Secondary (AA) amyloidosis,
often associated with an underlying autoimmune disorder, can be diagnosed by fat pad biopsy and further staining for serum amyloid A protein. Familial (ATTR) amyloidosis, distinguished by deposition of the transthyreitin protein, can be diagnosed by serum transthyreitin testing or DNA-based testing for a mutant transthyreitin gene. Notably, fat pad biopsy is only 73% sensitive in this group and a negative fat pad biopsy and lack of a diagnosis should prompt further investigation.

Second Place – Oral Clinical Vignette:

A CHILD’S RASH CAUSING AN ADULT CHEST PAIN
Nickey R. O’Coyne MD, Mayo Clinic Arizona, Abstract 135

Case Description: 68 year-old man with a history of colon cancer and enlarged prostate recently treated for a urinary tract infection with antibiotics, presented complaining of rash and mid-sternal chest pain. Physical exam revealed palpable purpura. An electrocardiogram did not show any signs of ischemia. Initial laboratory results including cardiac enzymes, blood counts, and coagulation studies were largely normal, with the only abnormality being an elevated creatinine. A skin biopsy was performed which showed leukocytoclastic vasculitis with IgA present, supporting a diagnosis of Henoch-Schönlein Purpura (HSP). Imaging of the chest showed a thickened esophagus and subsequent endoscopy revealed advanced esophagitis with ulceration of the esophageal, gastric, and duodenal mucosa.

Discussion: HSP is a small vessel vasculitis that is most commonly associated with the tetrad of palpable purpura, arthralgia, renal disease, and abdominal pain. About ninety percent of HSP cases occur in children. A recent review of the GI manifestations of HSP stated that “the esophagus is usually spared.” What is unique in this case is the esophageal involvement of an older patient with HSP leading to a presentation of chest discomfort.

Third Place – Oral Clinical Vignette:

MYSTERY OF THE UNCONSCIOUS PATIENT
Divya R. Pati MBBS, University of Arizona College of Medicine at South Campus, Abstract 136

In patients who are found unresponsive, a thorough evaluation to rule out central nervous system disorders, metabolic diseases and drug overdoses needs to be promptly initiated. Occasionally, despite an extensive workup, an etiology to explain the cause of the decreased level of consciousness is not immediately forthcoming.

A 45-year-old male patient with a history of Klinefelters syndrome, depression and schizophrenia, was brought to the hospital from the department of corrections after being found unresponsive in his cell. The patient was unresponsive to painful stimuli and did not have any evidence of seizure activity. The patient’s breathing was spontaneous and his vitals were in the normal range. No improvement occurred after administration of Naloxone. The patient was therefore intubated for airway protection. The patient’s eye lids were closed and difficult to open. Because the pupils were rotated upward, a complete eye exam was not possible. A complete neurological examination could not be done as patient was intubated and sedated. Reflexes were normal, and there was no muscle rigidity.
The remainder of the physical examination was unremarkable. Laboratory investigations that included CBC, CMP, CK, ammonia and ABG were with normal. Blood alcohol, salicylate, acetaminophen, carbamazepine and lithium were negative. Urine drug screen was positive for benzodiazepines. EKG, Chest X-ray and CT head were unrevealing.

Despite the discontinuation of all sedation the patient continued to be unresponsive. Analysis of the CSF revealed a mildly elevated protein of 71mg/dl. Viral studies, blood, urine, CSF and sputum cultures were negative. MRI and an EEG did not reveal any abnormalities. Despite being off sedation for 2 days, the patient was unresponsive but showed spontaneous breathing. On day 3 a psychiatry consult raised the possibility of catatonia and a benzodiazepine challenge test was recommended. Within 1 hour of receiving 2 mg IV Lorazepam, the patient started moving his limbs and following commands. He was begun on 2 mg IV Lorazepam three times a day and was transferred to a psychiatric inpatient unit the next day.

Decreased level of consciousness is not typical for primary psychiatric illness. In any patient with a depressed sensorium, medical causes such as infection, drug overdose or CNS disorders should be ruled out. In the absence of a medical cause, especially in patients with a psychiatric history, catatonia should be considered. Catatonia is a poorly understood disorder that can be associated with metabolic conditions, neurological illnesses such as encephalitis and epilepsy, psychiatric conditions like psychosis and mood disorders and illicit drug related states to name a few. Catatonia is unique in its response to benzodiazepines and electro-convulsive therapy. When catatonia is suspected benzodiazepine challenge is invaluable in confirming the diagnosis.

“MY THROAT HURTS!”

Mohammad Abu Zaid, MBBS, University of Arizona College of Medicine at University Campus, Abstract 137

Purpose of the presentation: to present a case of possible Levamisole-induced neutropenia.

Case description: a 24 year old Hispanic male was referred to UMC for management of a peritonsillar abscess. On routine CBC at admission, he was found to be severely neutropenic with an absolute neutrophil count (ANC) of 220 and anemic with hemoglobin of 10.2. A workup for neutropenia was initiated.

Summary of results: Aside from severe neutropenia and anemia on CBC, and the finding of a peritonsillar abscess on neck CT scan, a detailed history, physical exam, and basic labs including TSH, ANA, hepatitis panel and HIV screen were inconclusive. A peripheral blood smear did not show any blasts or other clues to the cause of the neutropenia. The hematology attending, given recent case reports of neutropenia secondary to levamisole tainted-cocaine use, suspected the diagnosis and asked again about drug use, which the patient completely denied on initial History and Physical. His family members volunteered the information that the patient used cocaine on average every other day.

Conclusion: Levamisole tainted-cocaine is a recently acknowledged cause of bone marrow suppression and neutropenia. There has been a rise in the percentage of cocaine samples seized by the DEA and found to be cut with levamisole from around 2% in 2005 to over 80% in 2010. Although the diagnosis was not
confirmed by a plasma levamisole level (unavailable at the facility at the time) or a bone marrow biopsy (due to patient refusal), the hematology and medical oncology team felt very confident with the diagnosis and decided to follow the patient in the clinic.

“A CASE OF THE BLUES”

James Gee, MD, St. Joseph’s Hospital and Medical Center, Abstract 138
Second Author Yousef Usta, MD

Introduction. Acquired methemoglobinemia is a potentially life threatening clinical entity causing a range of signs and symptoms such as cyanosis, dyspnea, lethargy, seizures, and even death. These symptoms are caused by the accumulation of abnormally high methemoglobin levels in the blood which impairs the transfer of oxygen to tissues, leading to hypoperfusion of the organs. It is essential to recognize this condition early as it can be induced by commonly used medications and is readily treatable, but it is potentially fatal in severe cases if the diagnosis is delayed.

Case Presentation. A 68 year-old male was admitted to the medical floor with complaints of malaise, fatigue, fevers, and night sweats for the past few days. Labs were significant for a leukocytosis of 18.3 cells/ul (4.5-10) and a UA suggestive of a UTI urine WBC 25-50 (<5). Urine and blood cultures were both positive for Enterococcus within 24 hours of admission. The patient continued to have recurrent febrile episodes, raising the suspicion of endocarditis. A transesophageal echocardiogram (TEE) was scheduled. A topical benzocaine spray was used for oropharyngeal anesthesia prior to introduction of the probe and the TEE was performed. Within minutes of arriving back to the medical floor from the TEE, the patient developed cyanosis, confusion, increasing lethargy, and low oxygen saturation. Oxygen saturation was 80% on 15 L/min of oxygen via face mask. Examination revealed a patient in no respiratory distress but notably confused and increasingly somnolent. There was prominent facial, hand, and foot cyanosis despite good pulses. The chest exam was normal and the lungs were clear. The cardiac exam remained normal. A chest radiograph did not show any acute processes. An EKG showed sinus rhythm with no segment changes. Cardiac enzymes were normal. An ABG was done and was normal, so a methemoglobin level was checked and was markedly elevated >31% (≤1.5%). The diagnosis of acquired methemoglobinemia was established and the patient was transferred to the ICU for emergent treatment with intravenous methylene blue. Within 1 hour of treatment the patient’s symptoms resolved completely and he recovered uneventfully.

Conclusion. Acquired methemoglobinemia due to benzocaine exposure is a complication that has been well described in the literature, but due to the relative infrequency at which it occurs, some practitioners may not immediately consider the diagnosis despite how often these medications are used. Given the frequent use of these anesthetics in various endoscopies and procedures, it is imperative for the internist to consider methemoglobinemia in the differential diagnosis of acute hypoxia and cyanosis after suspected or known exposure.
AN INCIDENTAL FINDING ASSOCIATED WITH MECHANICAL VENTILATION IN PRONE POSITION

Hossein Ghofrani MD (Associate), Maricopa Medical Center, Abstract 139
Additional Authors: Hasan Chaudhry MD (Associate), Michelle Kauffman DO, Thomas Ardiles MD, Pedro Quiroga MD

Introduction
Prone-position mechanical ventilation has been used in patients with acute respiratory distress syndrome (ARDS). A number of randomized controlled trials and meta-analyses have shown improvement in oxygenation, and some survival benefit, in patients with ARDS and very severe hypoxemia. Pressure sores, accidental extubation, obstruction of endotracheal tubes, injuries to personnel, accidental removal of lines, need for increased sedation, hypotension and arrhythmias are some of the known previously-reported complications of prone position ventilation. Here, we report splenic laceration as a new potential complication associated with prone positioning; this was an incidental finding.

Case Report
A 39 year old male with history of hypertension, congestive heart failure (CHF), non-ischemic dilated cardiomyopathy, morbid obesity and obstructive sleep apnea presented with mixed respiratory failure with severe hypoxemia, CHF exacerbation with significant pulmonary edema, and anasarca. He had a prolonged and complicated hospital stay, requiring aggressive diuresis, inotropic agents, and treatment for pneumonia and NSTEMI. On hospital day 3, his mechanical ventilation was set on BiVent mode, requiring very high pressures to correct hypoxemia, which was continued until day 14. Due to his morbid obesity, severe cardiomegaly and associated mechanical disadvantages, patient was placed in prone position on day 5 (6hr-1hr ratio prone-supine) from which he was gradually weaned off until day 10.

On hospital day 17, a CT scan of the chest with IV contrast was done to evaluate airspace disease. There was an incidental finding of “Large splenic laceration extending to the splenic hilum without perisplenic fluid, and no active extravasation; likely subacute in nature.” This was confirmed again with a CT abdomen/pelvis. There was no evidence of blood loss in blood cell count. No surgical intervention was required as laceration was well-encapsulated.

Discussion
We report a case of splenic laceration that was most likely caused by placing the patient in prone position. Morbid obesity and severe volume overload with severe venous congestion were the likely underlying risk factors. Patient had significant cardiomegaly and chest wall restriction, and even though this was not a case of ARDS, the mechanical advantages of prone positioning allowed us to effectively oxygenate and ventilate this patient with more acceptable levels of oxygen. Unfortunately, we do not have any prior imaging to compare this finding with; however there was no evidence of any previous trauma or injury in the history taken from the patient and family, or in the physical exam. To our knowledge, this is the first case report of such complication in English literature. With the current epidemic of obesity and cardiac disease, the use of rescue ventilation modes like prone positioning will continue. This is an
important complication to monitor when using prone ventilation with similar patients.

“CAN YOU BE ALLERGIC TO YOURSELF?”

Rahul Rishi, DO, Sierra Vista Regional Medical Center, Abstract 140

Second Author: Duane Wong

A 44 y/o Caucasian female presented with acute diffuse hives in 2009. She had known allergic rhinitis, but had no triggers for her hives including food allergens, medications, contact allergens, or recent illnesses. Before presentation, she had taken 2 courses of medrol dose packs with temporary relief. Her hives were not controlled with H1 and H2 antihistamines, and she required 2 more courses of medrol over the next 2-3 weeks. At this time her hives had lasted >6 weeks without control with multiple H1 and H2 antihistamines and she was diagnosed with chronic urticaria. She was placed on a long taper of medrol to prevent rebound, and she did not tolerate decreasing the dosing below 6-8 mg per day. Between 10/2009 and 12/2010, she was steroid dependent for control of her hives with doses of medrol between 2-12 mg daily. She failed trials with montelukast, sulfasalazine, and doxepin. By 9/2012 she began having steroid side effects with significant weight gain, anxiety and high blood pressure. In 1/2011, she began a trial of omalizumab 300 mg monthly off label for her urticaria, and within 3 doses she tapered off her medrol and antihistamines. She has continued omalizumab for the last 1½ years with controlled symptoms. Her initial physical exam on presentation was normal except for diffuse urticaria lesions on her trunk and extremities with excoriation and moderate dermatographism. Urticaria is a skin condition in which transient pruritic wheals and flare develop on the skin. It is a common skin disorder with multiple etiologies, but it is considered chronic when it occurs for >6 weeks. About 40-50% of patients with chronic urticaria have an autoimmune etiology. The most common etiology is an IgG antibody directed towards the α-subunits of the IgE receptor. Binding of the antibody causing degranulation of basophils and mast cells, and subsequent histamine release and urticaria.

The mainstays of treatment include H1 and H2 antihistamines and leukotriene inhibitors. Systemic steroids and cyclosporine are frequently used in refractory disease, but chronically can lead to significant adverse effects. Omalizumab is a recombinant humanized monoclonal antibody against the IgE receptor. It inhibits the binding of IgE the high affinity IgE receptor (FCeRI) on mast cells and basophils and reduces the number of FCeRI on basophils. In chronic autoimmune urticaria it may be that omalizumab is able to reduce FCERI expression enough to prevent the IgG autoantibody from causing cross-linking and degranulation. Although omalizumab use hasn’t been approved for use of chronic autoimmune urticaria, there are multiple case reports of successful treatment of chronic autoimmune urticaria in patients. This case emphasizes that most chronic urticaria is autoimmune, and underscores the potential option of omalizumab therapy in the future for refractory patients.

“SIR, WHAT LARGE HANDS YOU HAVE!”

Andrew Sacks, DO, Verde Valley Medical Center, Abstract 141

Joint pain and swelling of the extremities are common complaints in primary care clinics, particularly from middle-aged and geriatric
patients. Likewise, weakness and fatigue occur in these populations with some frequency. However, when all occur acutely and simultaneously, the differential diagnosis can be quite broad. The following is a case of remitting seronegative symmetrical synovitis with pitting edema, or RS3PE.

A 68 year old male presented to his primary physician complaining of marked swelling in both hands and forearms, worsening over the past 2 months. This was associated with throbbing in both hands. Furthermore, he had pain in bilateral hips and shoulders with weakness in his upper arms and buttocks, finding it difficult to raise his arms and get up from a chair. These symptoms had been present for approximately 1 year. He experienced no relief with various NSAIDs. His symptoms resolved with an oral steroid dose pack, but returned immediately after stopping them. He was referred to a rheumatologist who noted tenderness and swelling in bilateral metacarpophalangeal, as well as proximal and distal interphalangeal joints of the fingers. The severe pitting edema in both hands was still present. The remainder of the history and physical exam was unremarkable. Labs were significant for an elevated erythrocyte sedimentation rate of 95. Rheumatoid factor and anti-cyclic citrullinated peptide antibody were both negative. All other findings were within normal limits. RS3PE was strongly suspected, with likely differentials being polymyalgia rheumatica (PMR) and late onset seronegative rheumatoid arthritis. The patient was started on prednisone at 20mg daily and returned for followup 1 month later. By that point, all symptoms had resolved. RS3PE is an important differential diagnosis in the workup of joint pain, weakness, and upper extremity swelling, particularly in men whom it affects twice as often as women. However, the diagnosis is often overlooked or delayed due to its striking similarity to far more common conditions including those mentioned above. Indeed, many consider PMR and RS3PE to be members of the same spectrum of disease.

Making matters even more difficult, no confirmatory or highly indicative test presently exists. While the condition itself is relatively benign, a percentage of RS3PE incidence has been shown to be paraneoplastic, usually to lung malignancy. This should be considered whenever risk factors or constitutional symptoms accompany the usual syndrome. Otherwise, the significance primarily lies in the treatment. There is no role for immune suppression or modulation beyond corticosteroids, as opposed to rheumatoid arthritis. Moreover, the typical effective dose and duration of steroid therapy are both lower for RS3PE than for PMR, with patients usually remaining asymptomatic years after a few months of treatment.

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