VAC Associated Gossypiboma: Not a Scenario to Gloss Over

Vacuum-assisted closure (VAC) devices provide negative pressure wound therapy (NPWT) and are used in the management of complex wounds. The VAC promotes rapid wound closure by encouraging perfusion, fibroblast migration, and proliferation. This however also presents with an underreported inherent risk for gossypiboma, defined as retained surgical pieces, acting as a nidus for infection.

Here we present two cases of retained sponges, leading to an abscess formation, persistent drainage and infection. A 76-year-old female post a femoral-popliteal bypass subsequently developed a chronic hematoma of the left leg and was placed on a wound VAC after multiple washouts. Follow-ups over 4 months showed a sinus tract with persistent purulent foul discharge. MRI showed a large fluid collection in the calf, with surgical material and surrounding myonecrosis.

A 20-year-old male post thoracic spinal fusion, epidural hematoma evacuation, and wound VAC, presented with persistent drainage for 5 months. CT of T-spine reflected an intramuscular abscess with packing material and a myocutaneous fistula. Delayed wound healing and drainage resolved in both cases after the retained foam was removed.

Retained sponges result in a broad spectrum of clinical presentations with time to diagnosis varying from days to years. In literature, diagnostic CT scans reflect a low-density heterogeneous spongiform mass with gas bubbles, however altering sponges with radiopaque material may allow earlier detection and treatment. Communication and proper recordkeeping between providers post discharge are vital for avoiding retained foam dressings. Failure to recognize this avoidable complication may have dire consequences.
Kindling Syndrome Exacerbated by Benzodiazepine Therapy in a Case of Alcohol Withdrawal

Introduction:
Kindling syndrome entails worsening hyperexcitability following repeated, intermittent exposure to a sensitizing agent. It relates to alcohol withdrawal (AW), as chronic alcoholism leads to an upregulation of excitatory neurotransmitters and a downregulation of inhibitory neurotransmitters, thereby overcoming alcohol's depressant effect. However, during sobriety, these adaptations cause hyperexcitability manifesting as worsening AW.

Case:
A 59 year old patient with past history of chronic alcoholism, and schizophrenia presented to the ER intoxicated. Owing to history of severe AW he was admitted on his home medications of risperidone and mirtazapine and started on CIWA protocol with lorazepam therapy. Following admission, the patient evidenced progressive agitation and worsening CIWA scores culminating in the need for restraints, despite graduation to a lorazepam infusion. His symptoms worsened to include tremors and hallucinations despite multiple pharmacotherapeutics including dexmedetomidine, propofol, and various antipsychotics.

Finally, psychiatry assessed the patient with kindling syndrome, secondary to chronic alcohol abuse, compounded by long-term intermittent benzodiazepine administration. The patient was subsequently started on valproate and olanzapine and evidenced resolution of agitation with decreasing need for sedatives and improvement in orientation.

Discussion:
Kindling syndrome should be considered in recalcitrant cases of AW, especially in patients with long histories of frequent episodes. Recurrent AW predisposes patients to a progressively “kindled” hyperexcitable state and escalates risk for eventual seizures. Benzodiazepine therapy should be judiciously used in these cases owing to similar receptor targeting and possible potentiation of the kindling process. Anticonvulsants would be more appropriate in these patients to avoid such complications.
An Apparently Puzzling Case with a Simple Treatment

INTRODUCTION: The development of fever and altered mental status in an individual often leads to work-up for infectious causes of encephalopathy. Gabapentin withdrawal is rarely considered a cause for these symptoms in patients who are on chronic gabapentin therapy. We present a case of gabapentin withdrawal syndrome presenting with fever and altered mental status.

CASE: A 58 year old male with history of chronic back pain on morphine, oxycodone and gabapentin presented to the emergency department with complaints of altered mental status over the last three days. The physical examination was notable for confusion with intermittent agitation and miotic pupils. The patient was started on Naloxone drip for possible opioid overdose, but he remained encephalopathic. After 48 hours of admission, the patient started developing fever up to 101.2 F. He was started on antibiotics and antivirals, which didn’t improve his condition. The patient’s family was re-questioned about drug use and it subsequently mentioned that he had stopped taking gabapentin a few days ago. Thus, a case of gabapentin withdrawal was considered. Since the patient was still confused, a nasogastric tube was placed to replace gabapentin. The next morning, the patient recovered fully.

DISCUSSION: With 57 million prescriptions written every year, gabapentin withdrawal syndrome is more important than ever. Several case reports have described agitation, confusion and disorientation as part of gabapentin withdrawal syndrome, but fever is rare. It is important to educate patients not to stop gabapentin abruptly to minimize development of gabapentin withdrawal syndrome.
Hepatitis C virus (HCV) is a well known etiology for chronic hepatitis, cirrhosis and hepatocellular carcinoma; furthermore HCV has also been implicated in extra-hepatic manifestations. A 51-year-old male, with a history of prior IV drug abuse and hepatitis C, presented with abrupt purpura, uncontrolled hypertension and anasarca. His hepatitis C had never been treated due to financial reasons. He has been diagnosed with marginal zone BCNHL in 2012, which represents the most common histological type of HCV-associated lymphoma. He was treated with Rituximab leading to a complete response. He developed acute kidney failure with proteinuria although his liver function tests were normal. His HCV load was significantly increased comparing to six months ago. Cryoglobulinemia was confirmed in his serum as well as low C4, normal C3 and positive rheumatoid factor. Kidney biopsy was performed and revealed cryoglobulinemic MPGN with 35% global glomerulosclerosis. Patient was started on Harvoni after 2-week treatment of Rituximab and steroid. Unfortunately, patient did not improve and died of kidney failure 2 months later.

Chronic HCV infection results in persistent B cell stimulation and proliferation. Epidemiological studies have demonstrated frequent associations of hepatitis C with B cell lymphoproliferative disorders. However, very few cases of HCV-induced BCNHL and cryoglobulinemic MPGN presenting in the same patient have been reported. Early implement of anti-HCV treatment is crucial for better prognosis. While eradication of HCV was proven to induce complete remission of indolent BCNHL, end stage kidney disease will not respond well to antiviral therapy.
A Rare Case of Refractory Hypocalcemia in Malignancy

Hypercalcemia of malignancy is commonly seen in practice, however refractory life threatening hypocalcemia is seldom reported. Here, we present an interesting case of hypocalcemia, its challenges in management, and also prevention strategies.

A 72 year old gentleman with history of diabetic stage 3 chronic kidney disease presented with severe low back pain. Examination revealed tenderness over T9 vertebra and a 3 cm left inguinal node. CT demonstrated sclerotic T8-T9 lesions and retroperitoneal lymphadenopathy, which led to a bone scan showing diffuse bony metastasis. Both prostate specific antigen (216 ng/mL) and alkaline phosphatase (933 IU) were markedly elevated. Inguinal lymph node biopsy confirmed metastatic prostate cancer. Patient was initiated on Bicalutamide, Leuprolide and Denosumab injection (Xgeva), to prevent skeletal related event. S.calcium and vitamin D levels were normal on discharge.

Patient returned 2 weeks later with weakness, and a S.calcium of 4.8 mg/dL. PTH was elevated at 250.6 pg/mL. Cause was attributed to Denosumab injection. He was treated with IV calcium gluconate (4-8 g/day), calcitriol 0.5mg QID and oral calcium. Calcium levels took more than a month to return to near baseline.

This case highlights identification of crucial risk factors for precipitating dangerous hypocalcemia, like pre-existing renal insufficiency in patients with extensive bony metastasis (indicated by elevated alkaline phosphatase). Other factors to be considered are vitamin D deficiency and a pre-existing low normal calcium level with corrected albumin. Careful decision making in these subset of patients is the key to prevent prolonged hypocalcemia and morbid hospitalization from Denosumab injection use.
Acquired Hemophilia: An Uncommon Case of Factor VIII Antibodies

Case description: A 74 year-old male presented to the hospital with complaints of nausea, shortness of breath, and productive cough with white sputum.

The patient had a history of chronic hematuria and had a recent cystoscopy which did not identify a specific source of the bleeding and the hematuria self-resolved.

During his hospital stay, he began having frequent maroon-colored stools and developed severe acute on chronic anemia requiring multiple blood transfusions. Coagulation studies revealed a PTT of greater than 200 with a normal prothrombin time. A clotting factor assay showed significantly elevated levels of Factor VIII antibody with low levels of Factor VIII, IX, XI, and XII activity.

Treatment was started and he had several nuclear medicine bleeding scans which revealed active bleeding in the ascending and transverse colon. The patient underwent angiography with successful embolization of several branches of the cecal artery. The patient ultimately had a laparoscopic right hemicolectomy due to his persistent gastrointestinal bleeding.

Discussion: This case demonstrates the need for the medical community to be aware of some of the less common causes of hemophilia. Acquired hemophilia is likely underdiagnosed or misdiagnosed due to its rarity, however, it can present with a somewhat unique pattern of persistent bleeding, frequently involving the genitourinary and gastrointestinal anatomy. This is in contrast to the typical joint bleeding seen in congenital hemophilia. The treatment of acquired hemophilia also differs from that of congenital hemophilia and early diagnosis may help improve overall mortality in this group of patients.
When the Heart Attacks: Haemophilus Influenzae Bacteremia with Myopericarditis Mimicking an STEMI

Pericarditis with involvement of the myocardium is termed myopericarditis. Underlying etiologies overlap those of pericarditis. In developed countries, viruses are the most common cause. Non-tuberculosis bacterial causes are rare. Most reported cases of Haemophilus influenzae-related pericarditis are in children. Here, we report a case in an adult male who had H. influenzae bacteremia with myopericarditis and cardiac tamponade. A 49-year old male with HIV (compliant with his anti-retroviral therapy) presented with generalized fatigue, nausea and non-bloody, non-bilious emesis for one day. He denied fever/chills, change in bowel/bladder habits, chest/abdominal pain, and shortness of breath. He was up-to-date on his immunizations. Multiple household members were recently sick with gastroenteritis-like symptoms that resolved a few days ago. Labs were remarkable for leukocytosis (>35.0 x 10⁹ per liter) with a left shift. Blood cultures grew H. influenzae and his antibiotics were de-escalated to ceftriaxone. Three days later, he complained of acute chest pain with evidence of an STEMI. Cardiac catheterization revealed no occlusive disease. An ECHO showed an EF of 55-60% with a moderate size pericardial effusion. He was started on aspirin and colchicine for myopericarditis. A follow-up ECHO showed worsening of the pericardial effusion with concerns for developing tamponade. He underwent a pericardial window without any complications. Although fluid and tissue cultures were sterile, fluid analysis suggested an exudative process – possibly secondary to H. influenzae. Myopericarditis is usually masked by systemic infection or inflammation and can mimic acute myocardial infarction. When the “heart attacks,” myopericarditis should be part of the differential.
Role of Eculizumab in Atypical Hemolytic Uremic Syndrome (aHUS)

INTRODUCTION:

aHUS is a rare genetic disease that affects different age groups. It is caused by inherited mutations causing complement system dysregulation which triggers an inflammatory reaction & systemic thrombotic microangiopathic events. Patients usually present with fatigue and oliguria.

CASE PRESENTATION:

A 19 y.o F presented with fatigue & anuria for four days. Workup showed thrombocytopenia, AKI & anemia. Stool cultures were negative; ADAMTS13 was normal & kidney biopsy revealed microangiopathy. She was diagnosed with aHUS & received four sessions of Plasma Exchange without improvement. The decision to start Eculizumab was made and patient responded dramatically within three weeks. Genetic testing was positive although not required for diagnosis.

DISCUSSION:

Our abstract will focus on the role and efficacy of Eculizumab in patients with aHUS. Eculizumab is a humanized monoclonal IgG antibody that works by inhibiting the complement system. Although Eculizumab is not a FDA approved, it is a promising new treatment that has been studied and trials has proven its efficacy & revealed clinically significant normalization of hematological measures, kidney function, morbidity & mortality, as was the case with our patient. The current standard treatment is plasma exchange does not treat the underlying process but transiently maintain normal hematological values.

CONCLUSION:

aHUS is diagnosed when workup is negative for TTP, typical HUS & immunological diseases. Eculizumab is effective in treating aHUS. In the setting of aHUS rarity, we don’t have enough studies to compare with standard therapy or to assess the best duration and dosage. Further studies are warranted.
Are Breast Cancer Guidelines Failing Our Patients?

Introduction:
The US National Cancer Institute Surveillance, Epidemiology, and End Results (SEER) has found a significant increase in breast cancer with metastasis in women below 50 years of age from 1976-2009. The American Society of Clinical Oncology (ASCO) recommends against imaging studies in patients with Stage I or II Breast Cancer for the identification of occult metastatic disease, unless worrisome signs or symptoms are present, because the risk of metastasis at diagnosis is low. This is extrapolated, however, based primarily on cancers diagnosed by screening women above the age of 50.

Case Presentation:
A 47-year-old female presented with left sided, positional chest pain. Physical exam identified a tender and fixed mass in the left breast that was later diagnosed as Stage IIa invasive ductal breast adenocarcinoma T2 N0, ER/PR positive and HER II negative. Per ASCO, no imaging for staging was warranted, because the patient was asymptomatic. Within one month of the lumpectomy, prior to start of therapy, she presented with worsening shortness of breath. Workup diagnosed metastasis with a malignant left sided pleural effusion, subcentimeter pulmonary nodules, and spinal lytic lesions.

Discussion:
We challenge the ASCO guidelines recommending against CT, PET, or bone scan for staging patients with Stage II breast cancer, which do not comment specifically on patients that have been diagnosed with breast cancer outside the screening criteria. The SEER database from 2005-2014 shows that women below 50 years of age are more likely to have regional metastasis at diagnosis than those above 50.
**Factor V Leiden Mutation and Arterial Thromboembolism: An Evidence Based Review**

**Introduction:** About 3% of the world population is heterozygous for factor V Leiden (FVL) mutation. It is also found in 25% of patients with recurrent VTE, but its contribution to arterial embolism is less clear. This case will illustrate the futility of testing for FVL Mutation in arterial embolism compared to the inappropriate frequency with which it is checked in the American health care system.

**Case Description:** A 37-year-old Caucasian male presented with diplopia secondary to simultaneously basilar artery thrombosis and NSTEMI. Cardiac catheterization revealed triple vessel disease with an EF of 25% and apical hypokinesis. Echocardiogram revealed no mural thrombus. LDL measured 92 mg/dl and total cholesterol was 138 mg/dl. His only risk factor was a smoking history. Hematology study revealed one normal FVL gene and one R506Q mutation. The Prothrombin G20210A was not detected. Did the patient’s FVL mutation contribute to his ischemic stroke?

**Discussion:** A 2002 Copenhagen City Heart Study meta-analysis of FVL mutation and ischemic stroke demonstrates no clear association. Even the current guideline literature does not support a change in stroke prevention management knowing the FVL mutation status. Despite this lack of relationship, our analysis of our own hospital system revealed that 81% of patients between the age of 31 and 40 had an FVL mutation workup. Hospital systems continue to injudiciously seek FVL mutations in young patients with ischemic stroke at a cost of nearly $300,000,000 a year, seizing significant resources.
Pregnancy and Lactation Associated Osteoporosis

Introduction: Pregnancy and lactation associated osteoporosis (PLO) is a rare disease of late pregnancy, puerperium and lactation. It occurs with the first pregnancy but can recur with subsequent pregnancies. PLO typically presents with back pain.

Case presentation: 30 years old woman presented with acute onset back pain two months after the delivery of her first child. She was exclusively breast feeding her newborn. Imaging showed acute multiple vertebral compression fractures. The patient underwent kyphoplasty. A variety of work up ruled out secondary causes of osteoporosis including metabolic bone disease, genetic disorders and malignancy. Diagnosis of PLO was made. Breast feeding was discontinued and treatment was started with Calcium, Vitamin D supplements and Calcitonin nasally. Bone density scan in 6 months demonstrated minimal improvement. Accordingly, decision was made to change treatment to Forteo.

Discussion: The main focus of our discussion will point towards diagnosis and treatment of PLO. Our initial work up would aim for explaining why a premenopausal woman would develop osteoporotic compression fractures. Overall, we have limited data on efficacy, long term outcomes and safety profile for most of the anti-resorptive medications in premenopausal women. Alternative medication, namely Forteo, has been highly recommended in numerous case reports and studies with very good outcomes.

Conclusion: PLO is a diagnosis of exclusion. Therefore, it requires having high suspicion index especially in premenopausal women with acute back pain during pregnancy or lactation. Timely diagnosis and treatment is essential because of the severe morbidity associated with PLO.
Case Review of Metronidazole-induced Pancreatitis

Introduction: Metronidazole is a popular antibiotic that was reported as a rare cause for acute pancreatitis. Here, we discuss a case of recurrent metronidazole-induced pancreatitis and provide a literature review. Case: A 60 year-old female presented with severe epigastric pain radiating to the back, nausea, and vomiting, started 3 days after starting metronidazole for C. difficile colitis. She recalled similar symptoms after taking metronidazole for 4 days for C. diff 2 years ago. Metronidazole was changed to vancomycin at that time. Vitals stable. Exam: severe epigastric tenderness. WBCs 16,000/uL; lipase 396 U/L, and amylase and LFTs WNL. CT abdomen: peripancreatic fat stranding at the pancreatic tail suggestive of acute pancreatitis. Symptoms and lipase improved 2 days after discontinuation of metronidazole and supportive care. She was discharged home without complications. Discussion: We identified 14 similar cases in the literature. 70% of patients were females; 50% of patients were <30 years old. Most patients presented with typical epigastric pain, nausea, and vomiting. All patients had stable vitals. Moderate-severe abdominal tenderness was the most common physical finding and only 2 patients had peritoneal signs. Abdominal CT and/or pancreatic enzymes levels were used to make the diagnosis. The levels of lipase varied from few hundreds to few thousands. The symptoms onset varied from <24 hours (3/15) to 2-7 days (12/15) from metronidazole initiation. 8/15 patients had recurrent symptoms after re-challenging with metronidazole. All patients had benign clinical course. Conclusion: Metronidazole is a rare but an important cause of drug-induced pancreatitis with a frequently benign clinical course.
Acute cholestatic jaundice is typically caused by either extrinsic or intrinsic obstruction of the bile ducts with a differential diagnosis including cholelithiasis, malignancy, or immune mediated etiologies including primary sclerosing cholangitis. A rare case is reported of overlap-autoimmune hepatitis presenting with cholestatic jaundice.

A 45-year-old women presented to the emergency room with scleral icterus. She was undergoing evaluation for elevated LFTs discovered on routine laboratory work three months prior. Physical examination included scleral icterus and excoriations of the skin. Laboratory evaluation demonstrated: AST=118, ALT=19, total bilirubin=5.3, alkaline phosphatase=2.3, albumin=2.3. A chest X-ray demonstrated prominent hilar adenopathy. Viral hepatitis panel, AMA, ASMA were negative. IgG4 and ACE level were elevated. Ultrasonography demonstrated heterogenous echotexture with contracted gallbladder with patent vasculature. Biopsy demonstrated active hepatitis with cholangiopathy, portal fibrosis with a paucity of plasma-cell-rich centrilobular inflammation and no clear granulomas. She was diagnosed with overlap-autoimmune hepatitis with concern for sarcoidosis. She was treated successfully with corticosteroids and ursodiol. She was transitioned to azathioprine and has been asymptomatic at one year follow-up.

Overlap-autoimmune hepatitis is considered a combination of autoimmune hepatitis and primary biliary cirrhosis or primary sclerosing cholangitis. Generally, patients present with elevation in liver function tests in both hepatocellular and cholestatic patterns. The presented patient is different in that she presented with jaundice and LFTs consistent with a primarily cholestatic pattern. The current case demonstrates the importance of maintaining a broad differential when evaluating patient with cholestatic jaundice.
Buschke-Lowenstein tumor of anorectal and perianal area is a rare but highly aggressive tumor frequently associated with Human Papillomavirus (HPV) type 6 and 11. It is often slow growing but highly destructive to local tissue.

A 61-year-old male with HIV presented with foul smelling mass and worsening pain in the anorectal area. He had history of anal squamous cell carcinoma (SCC) of rectum 10 years back treated with chemo-radio therapy and was on complete remission. Physical examination revealed approximately 15 cm x 10cm fungating mass involving entire right buttock and perineum. MRI of pelvis revealed extension of mass through the right greater sciatic foramen into the gluteal area posteriorly with sciatic nerve involvement. Biopsy showed fragments of squamous epithelium with positive P16 suggestive of HPV infection, compatible with diagnosis of condyloma. Treponema pallidum immunostains were negative. A diagnosis of Buschke-Lowenstein tumor was made due to the huge size of the mass and histological findings consistent with condyloma.

Buschke-Lowentstein tumor, often called giant condyloma accuminatum, is considered by some authors as intermediate between condylomas and squamous cell carcinoma. Histologically, they appear benign with hyperpapilomatosis, epithelial hyperplasia and koilocytosis but clinically they behave aggressively with extensive infiltration. Typically, slow growing in immune-competent individuals, it can grow rapidly in immunocompromised individuals as in our patient. Small fraction of these tumors can transform into invasive carcinoma hence early diagnosis and treatment is crucial.

Common treatment approach includes radical surgical resection with tumor free resection margins.
Showers From the Heart

Caseous calcification is an uncommon condition that affects the mitral valve; one study estimated its prevalence to be 0.06-0.07% of the population. It is usually discovered incidentally and is benign. However, it can rarely lead to dangerous consequences such as embolic stroke and mitral stenosis. A 67-year-old female presented to the hospital for intermittent right-sided vision loss. She was afebrile and wbc was normal. Electrocardiogram revealed normal sinus rhythm. Transthoracic Echocardiography demonstrated a mass located on the posterior mitral annulus. Cardiac Magnetic Resonance Imaging (MRI) better elucidated the mass—its size was 2.7x2.3cm and had unique characteristics of caseous calcification. The patient continued to have visual disturbances and MRI of the brain showed several foci of embolic stroke. No etiology other than the mitral valve mass was discovered as the causative etiology for the stroke. Therefore, minimally invasive surgery with mitral valve mass removal was performed. During excision of the mass, “milky fluid” was noted to be aspirated. Two sets of blood and all pathologic cultures were negative; nevertheless, the patient was treated for culture-negative endocarditis with intravenous antibiotics. The patient recovered well and was discharged home after 1 month.

Advanced cardiac imaging techniques, especially MRI, help diagnose caseous calcification accurately. This helps avoid misdiagnoses, for example labeling the abnormality as tumor or abscess, and subsequently preventing unnecessary testing or treatment. Although prognosis is good in majority of cases considering its benign nature, any sign of valve dysfunction, conduction abnormality or systemic embolization should prompt urgent surgical management.
Patient is a 60 year old lawyer who travels back and forth to Lebanon with a history of alcohol abuse who presented with generalized weakness and jaundice that began a few days prior admission. He was found to be in septic shock with multiorgan failure and taken directly to the ICU for vasopressors, mechanical ventilation for acute respiratory distress, and CRRT for acute renal failure. He was placed on broad spectrum antibiotics. The etiology for his sepsis remained unclear at that time as the full infectious work up remained negative. Due to pulmonary, hepatic and renal dysfunction within one month of traveling to Lebanon, rare infectious etiologies were sought. Further testing revealed IgM positive for Leptospirosis. Infectious disease treated him with doxycycline for 7 days. Although the patient denied any high risk exposure, he did mention that garbage disposal in Lebanon was halted due to a worker strike, leaving an unsanitary condition on the streets. He only drank bottled water but did regularly bathe with tap water.

Leptospirosis, also known as Weil disease, is a rare spirochete infection transmitted via contact with urine, blood, or tissue of infected animal (such as rats) that breaches the skin or contacts the mucosal membrane. Seldom seen in the United States, most infections are asymptomatic or cause a mild febrile illness, which was the presenting symptom in our patient. During the second immune phase many complications can occur such as the multi-organ dysfunction observed in our patient.
Stress Induced Stress - A Case of Mast Cell Activation Syndrome

Immune system dysregulation can result in downstream activation of multiple pathways. We describe an interesting case of mast cell activation syndrome presenting as acute respiratory failure and angioedema.

A 70 year-old-male presented to the emergency department with difficulty breathing, generalized rash and muffled voice for 2 days. He developed shock and angioedema within 2 hours of presentation. He was treated with Epinephrine intramuscularly and intubated. Initial consideration was lisinopril induced angioedema worsened by increased bradykinin release related with urinary tract infection. On day 4 of admission patient successfully passed parameters for extubation and was liberated from the ventilator. However, an hour after extubation, he developed hypotension and worsening stridor requiring re-intubation. Initial allergy work up revealed elevated serum tryptase levels of 90 ng/mL and normal C1 esterase antigen, C1 esterase functional assay and C4 levels. Patient was started on solumedrol 40 mg, diphenhydramine 50 mg IV, monteleukast 10 mg and famotidine 10 mg with improvement in blood pressure and airway edema. Repeat tryptase level decreased to 20 ng/mL. He was successfully extubated the second time after 6 days of mechanical ventilation.

Mast cell activation syndrome can have varying severity and involvement of different organs. Unlike lisinopril induced angioedema, which is due to increased bradykinin, mast cell activation leads to massive efflux of histamine. The specific laboratory finding is elevated tryptase and usually the conversion time is within 4 hours. Our case illustrates the high index of suspicion for mast cell activation in a patient presenting with angioedema.
Eosinophilic Myocarditis: A Rare Hypersensitivity Reaction to Mesalamine

Introduction:
Mesalamine, a locally-active anti-inflammatory medication, has been used for treating inflammatory bowel disease for more than 30 years. Life-threatening hypersensitivity reactions to mesalamine have rarely been reported.

Case presentation:
A 19-year-old woman with newly-diagnosed ulcerative colitis (diagnosed by colonoscopy) was started on oral mesalamine and budesonide. Her pre-treatment blood count was normal. Five days later she began experiencing intermittent high fever and pleuritic chest pain. Fourteen days after the initiation of treatment, she presented to the emergency department in cardiogenic shock. Initial labs revealed a WBC of 16.6 bil/L with an eosinophil count of 1.5 bil/L, troponin-I 2.7, and brain natriuretic peptide 881. Cardiac MRI demonstrated myocarditis with a reduced left ventricular ejection fraction (47%). Mesalamine was discontinued along with budesonide. Prednisone, lisinopril and carvedilol were initiated. Her symptoms improved and the eosinophilia resolved over the next four days. Prednisone was tapered over the course of 8 weeks. Repeat echocardiography five months later demonstrated normal diastolic and systolic function, and normal wall motion.

Conclusion:
Although viral myocarditis is the most common cause of acute heart failure in young patients, the presence of eosinophilia suggested an alternative etiology. Eosinophilic myocarditis has been associated with drug allergies, idiopathic hypereosinophilic syndrome, parasitic infections, malignancy, and various other causes of eosinophilia. Mesalamine-induced allergic eosinophilia has been associated with pneumonia, bronchiectasis, and gastritis, whereas myocarditis was rarely reported. Previous case reports and our case demonstrate that corticosteroids, beta-blockers, and angiotensin-converting-enzyme inhibitors, along with discontinuing the offending agents, can effectively treat allergic eosinophilic myocarditis.
Not Your Average CRAB: An Atypical Presentation of Multiple Myeloma

The typical manifestations of multiple myeloma are well known. Hypercalcemia, renal dysfunction, anemia, and bone pain characterize the most common presentation of disease. This can create a diagnostic dilemma when a patient with multiple myeloma presents without these signs. A 70-year-old female without significant past medical history presented with a 5 day history of fatigue and generalized weakness. On physical examination, pallor of the skin and conjunctiva were observed. Stool was negative for occult blood. Initial workup revealed hemoglobin = 6.5 g/dL (baseline ~ 16.0), platelets = 30 bil/L (baseline ~ 250s), schistocytes on peripheral smear, normal renal function, indirect hyperbilirubinemia, LDH = 805 U/L, undetectable haptoglobin, and a negative Coombs test. PNH screen, ANA, and cold agglutinin titers were also negative. Reticulocyte count was elevated. D-Dimer was elevated at 687 ng/mL, but fibrinogen was normal. PT/PTT were within normal limits. Serum IgA was elevated at 1.5 g/dL. Due to her age and severe bicytopenia, a bone marrow biopsy was performed and revealed a plasma cell myeloma comprising 90% of marrow cellularity consistent with multiple myeloma. Skeletal survey was negative for bone lesions. She was discharged home to begin treatment with dexamethasone, lenalidomide, and bortezomib, which resulted in normalization of blood counts.

Coombs negative hemolytic anemia with thrombocytopenia is rarely seen as the sole presentation of multiple myeloma. Furthermore, this patient's lack of hypercalcemia, renal dysfunction, or bone lesions illustrate the importance of having a high index of suspicion for multiple myeloma in the patient that presents with unexplained cytopenias.
Peritonsillitis to Mediastinitis: Be Aware of the Deadly Highway

Peritonsillar abscess is usually localized but has potential to spread to mediastinum, pleural cavities and pericardium leading to life-threatening pericarditis or mediastinitis. Despite the decreasing incidence in the post-antibiotic era; the risk of this deadly spread is still significant especially in immunocompromised individuals.

A 66-year-old man presented with right facial swelling and pain of 3 days. On examination he had dental caries, trismus and right tonsillar enlargement. CT neck revealed right peritonsillar abscess. Needle aspiration was done and IV ampicillin-sulbactum with IV steroid was started. Chest x ray done on second day revealed loculated right sided effusion for which thoracocentesis and chest-tube placement was done. He subsequently developed Atrial fibrillation with hypotension that required cardioversion. He continued to decline despite above management. Repeat CT chest and Neck with IV contrast was done which revealed multiple abscess within right parapharyngeal, retropharyngeal and superior mediastinum. Right video-assisted thorascoscopic surgery with pulmonary decortication and mediastinal exploration was done. Broad spectrum antibiotics coverage with cefepime, clindamycin and micafungin was continued. He gradually improved with this management. During his work up he was also found to be HIV positive.

Descending Necrotizing Mediastinitis is rare but possible complication of peritonsillar abscess. The Alar space in the retropharynx provides route for the spread. Immunocompromised persons are at higher risk of spread. Our case highlights the potential life threatening complication of commonly encountered peritonsillar abscess and the role of early detection and aggressive management for better outcome.
Unusual Viral Myositis Presentation

We present a rare case of viral co-infection presenting as rhabdomyolysis. An 18 year old male was transferred to our hospital with acute kidney injury. Two weeks prior he had noticed myalgia, odynophagia, and abdominal pain. He noted proximal muscle weakness and pain without any history of trauma. He had no significant prior medical or surgical history. He did admit to marijuana use and high-risk sexual behavior. Physical exam revealed cervical lymphadenopathy, diffuse muscle pain with palpation, and 4/5 muscle power in proximal muscle groups. There was no additional focal neurologic deficit and physical exam was otherwise benign. Laboratory tests revealed blood urea of 31 mg/dl, and creatinine of 2.50 mg/dl. Urinalysis revealed 3+ blood and 51-100 RBC. Creatine Kinase was 10,709 IU/L. Urine drug screen was negative. Liver enzymes were 324 (aspartate aminotransferase) and 280 (alanine aminotransferase) U/L. The total bilirubin, alkaline phosphatase, total protein, and albumin levels were within normal limits. ANA screen negative, complements C3/C4, anti-JO 1Ab, and Anti-dsDNA were negative. His HIV-1 antigen was positive and viral RNA Quantitation was at 2,320,000 copies/mL. Acute Ebstein-Barr Viral panel was positive. Additional viral panels were negative. Chest X-ray was normal. The patients muscle weakness and myalgias improved over the next couple days, as did the renal function with IV hydration. It was felt that the patient had acute HIV and EBV viral infections resulting in rhabdomyolysis and acute kidney injury. Clinical search has not revealed any other reports of HIV/EBV co-infection presenting as rhabdomyolysis.
Association Between APC Gene, FAP, and Hypertriglyceridemia

Background
Familial adenomatous polyposis (FAP) is caused by a mutation in the adenomatous polyposis coli (APC) tumor-suppressor gene. The APC gene is considered a “gatekeeper” gene for the progression of benign adenomatous polyps to malignancy. Laboratory studies in mice note that APC mutations can be associated with hyperlipidemia with a particular elevation in triglyceride levels.

Case
A 46 year old female patient with hypertriglyceridemia, DM type II, FAP s/p colectomy, and recurrent pancreatitis presented to the ER for evaluation of epigastric pain. Labs revealed a triglyceride level of 4510mg/dl. Lipase was 160U/L. Nephrology was consulted for consideration of plasmapheresis. However, this was held due to the patient’s clinical stability, normal calcium, normal lactate, and lack of findings suggesting sequelae of pancreatitis on imaging. Conservative management with NPO diet, insulin therapy, and resumption of home fibrate and statin was pursued. At discharge, the patient was counseled about the importance of dietary and medication compliance.

Discussion
This patient demonstrated both FAP and hypertriglyceridemia, possibly related to the pleomorphic effects of the APC gene. Furthermore, obesity and diabetes mellitus as well as elevated levels of insulin-like growth factor, hyperinsulinemia, and hypertriglyceridemia are related to increased incidence of colorectal cancer. In the laboratory, the use of PPAR inhibitors to reduce triglyceride levels have been shown to decrease colon cancer incidence in APC knock-out mice. The association between hypertriglyceridemia and the progression of colon cancer offers an important opportunity for intervention and directed therapies.
Bone Marrow Necrosis: A Rare and Fatal Manifestation of Essential Thrombocytosis

Bone marrow necrosis is a rare entity, with less than 500 reported cases to date. The vast majority of these are related to hematologic malignancies, solid tissue malignancies or sickle cell anemia. Treatment and recovery is based upon identification and treatment of the underlying systemic process. This is the very rare case of a bone marrow necrosis associated with essential thrombocytopenia.

An 85 year old male with a two year history of essential thrombocytosis and a known history of coronary artery disease, peripheral vascular disease and alcohol abuse presented with 1 month of history of lightheadedness and fatigue. He was found to be pancytopenic and to have melena on presentation. His hydroxyurea was held, he was transfused with packed red blood cells, and upper endoscopy showed no obvious source of bleeding. His symptoms initially improved with hydration and symptomatic treatment. Bone marrow biopsy was performed which showed bone marrow necrosis. Contralateral bone marrow biopsy was confirmatory. Further workup revealed peripheral blasts of 7%, but no definitive diagnosis of acute myeloid leukemia could be made. The patient was in constant pain with multiple readmissions. Over the next 2 months his pancytopenia progressed despite multiple transfusions, and the he ultimately expired.

This case highlights the importance of early bone marrow biopsy for patients with a history of myeloproliferative disorders and pancytopenia. Life-prolonging measures and potential cures are possible with early recognition of an underlying mechanism. At the very least, improved comfort care can be provided sooner with earlier recognition.
One Nocardia Short of a Full Deck

In primary cellulitis, the natural bacterial skin flora of Staph aureus and S. pyogenes are the usual causative agents. However, in immunocompromised patients, conditions surrounding the occurrence of cellulitis may dictate the pathogen. A wide variety of atypical pathogens can invade the skin of immunocompromised patients. Nocardiosis is an uncommon opportunistic infection caused by several species of soil and water-borne aerobic bacteria belonging to the genus Nocardia.

A 50-year-old male with a history of kidney and pancreas transplants on immunosuppressants presented with redness and tenderness to his finger. He denied trauma or injury to the area or recent sick contacts. An incision and drainage was preformed and cultures were sent. He was started on IV Vancomycin. Cellulitis improved with one dose of Vancomycin and he was discharged on oral Linezolid. Patient returned one week later with no improvement. The wound gram stain revealed a "rare gram positive bacilli with branching." Culture grew Nocardia species. CT head and chest were ordered and no disseminated infection was found. Linezolid was stopped and patient was started on Minocycline for cutaneous Nocardiosis.

This case demonstrates an interesting finding of an opportunistic infection in an immunocompromised patient. Although we expect these types of infections to occur, patient denied any trauma and it was assumed that the patient had S. aureus given that the patient seemingly improved with vancomycin. It is important for clinicians to follow patient’s cultures and treat patients according to the final results as both organisms can lead to different disseminated infections.
A 28 year old African American male presented with abdominal pain, nausea, vomiting, dark colored urine, for three days prior to admission. He denied having fever, chills, sore throat or neck swelling. Patient was working in a nursing home, so did have some healthcare exposure. Vitals were found to be unremarkable. Physical examination revealed scleral icterus and mild diffuse tenderness of the abdomen. Bowel sounds were present upon auscultation. CBC revealed atypical lymphocytosis. Serum Lipase was elevated to 594. RUQ ultrasound of abdomen was negative for gallstones or CBD dilation. CT abdomen without contrast showed fatty liver. Hepatitis panel was negative. Monospot test was positive. During the hospital course, there was persistent elevation of serum transaminases, ALKP, Bilirubin, GGT, ferritin and LDH levels. Peripheral smear revealed atypical cell morphology. Differential diagnoses included Acute hepatitis, HIV, autoimmune hepatitis, subclinical hepatitis with cholestasis from EBV, hemochromatosis and lymphoma. MRCP and CT chest/abdomen/pelvis with IV contrast were done, and were unremarkable. Anti LKM & Anti-smooth muscle antibodies, HIV were negative. Patient underwent liver biopsy that revealed viral hepatitis secondary to EBV. Patient received conservative management and was discharged home.

Discussion:
This case represents atypical presentation of Acute Infectious Mononucleosis (AIM) hepatitis with hyperbilirubinemia. About 12% of the cases have liver involvement in AIM. Jaundice is apparent in 5% to 9% of patients. Bilirubin and ALKP elevations are less frequent. Detection of IgM antibody to EBV viral capsid antigen is most reliable. Liver biopsy reveals vacuolization of hepatocytes.
An Outpatient Diagnostic Delay of Epiphrenic Diverticulum Presenting as Chronic Leukocytosis

Esophageal diverticula can occur in the upper-, mid-, or lower esophagus (referred to as Zenker’s, traction and epiphrenic diverticulum, respectively). This case represents a delayed diagnosis of epiphrenic diverticulum associated with chronic leukocytosis.

A 53 year-old male with past medical history of Diabetes, Hypertension, Hyperlipidemia, COPD, and Chronic Kidney Disease was seen in clinic for right flank pain. On examination, vitals were stable with scattered rhonchi bilaterally. Labs revealed neutrophilic leukocytosis (seen on prior labs) of 17,000, unremarkable UA and a baseline creatinine of 2.4. CT scan of the abdomen/pelvis showed a mildly abnormal distal esophagus. Esophagogastroduodenoscopy revealed an inflamed gastroesophageal junction with an undigested food bolus emanating from a large pulsion diverticulum at 35 cm in the right lower esophageal lumen. Looking back in his records, patient had a prior admission for right-sided pneumonia along with pleural effusion, without resolution of the leukocytosis after treatment. He was seen in the clinic several times for productive cough and noted to have neutrophilic leukocytosis on several occasions which could be explained by regurgitation and aspiration of food particles from the diverticulum.

Esophageal diverticula account for <1% findings on barium gastrointestinal radiographs and <5% of all cases of dysphagia. Epiphrenic diverticula, though uncommon, are diagnosed late because most patients are asymptomatic. Late diagnosis can lead to severe and life-threatening complications like aspiration pneumonia. Recognition of this as cause of delayed diagnosis, especially when the etiology of leukocytosis is unknown, may help reduce the high prevalence of diagnostic errors.
Cerebrovascular diseases are the fifth leading cause of death in US. Transient ischemic attacks are the warning signs of stroke. Cardio-embolism accounts for 10-30% of TIA and stroke, with atrial fibrillation being the most common cause. Here we report a case of TIA caused by atrial myxoma where early intervention prevented severe neurological insult in a patient with multiple cardiovascular risk factors.

A 70-year-old female with history of hypertension, hyperlipidemia, chronic kidney disease stage 3, left sided ductal carcinoma in situ status post mastectomy, obesity (BMI 33), presented to clinic with intermittent poor balance and confusion since 2 weeks. She had similar episodes 2 years ago which were attributed to vertigo. No loss of sensation, focal weakness, seizures, loss of consciousness or headache was reported. Neurological exam was within normal limits. MRI head without contrast showed early to subacute and chronic infarcts. Holter monitoring only recorded rare PVCs and PACs. Carotid US duplex was significant for 50% LICA occlusion only. TEE showed moderate sized fixed mass attached to fossa ovalis/atrial septum of left atrium concerning for Atrial Myxoma for which patient underwent resection. Repeat Echo was unremarkable. Patient denied any new neurological symptoms afterwards.

Atrial myxoma is a rare primary cardiac tumour with prevalence of 0.01%-0.3%. It commonly presents with cardiac and pulmonary symptoms. However, it can also present with neurological signs and symptoms, more commonly in young females. Studies have shown that the mobility of the tumour plays a role in embolization than the size. Treatment includes surgical excision.
A New Face to the Squamous Cell Carcinoma of the Lung

Introduction:
Metastatic breast carcinoma (MBC) from extra-mammary malignancy is extremely rare phenomenon with incidence of 0.4%-1.3%. Studies have reported an association of MBC with lymphoma, leukemia, and melanoma. Here in we present an interesting case of MBC from squamous cell carcinoma of the lung.

Case Discussion:
58 YO F who was recently diagnosed with stage IV metastatic-squamous-cell-carcinoma (SCC) of the lung with metastasis to the brain currently on palliative whole-brain radiation presented with chief-complaint of right breast mass. Physical exam revealed a palpable well-circumscribed nodule in the right breast periareolar region, with no skin or nipple retraction. Following a B/L mammogram was performed which showed 7.2 mm nodule in right breast extending into periareolar subcutaneous tissue. Ultrasound-guided core-needle biopsy showed histologically similar appearance to the original SCC of lung which was confirmed by immunostaining. At this-point patient was recommended for palliative brain, lungs and breast radiation followed by chemotherapy.

Discussions:
MBC is a very rare occurrence and is associated with poor patient prognosis. It usually presents as a rapidly growing, painless, well-circumscribed superficial palpable mass. Radiologically MBC can cause wide range of imaging manifestations, however unlike primary malignancy; MBC doesn’t demonstrate skin or nipple retraction. Hence it is important for clinicians to differentiate primary malignancy from MBC, as the treatment choices different significantly and only with the awareness of such a possibility can prompt diagnosis and optimal treatment be achieved.
A Case of Spontaneously Resolved Pituitary Apoplexy

69-year-old male presented to the emergency department with headaches, nausea and vomiting for 1 week. Physical exam revealed bitemporal hemianopia. His sodium was 123mmol/L. Head CT revealed a mass in the pituitary region measuring 1.6cm x 1.9cm x 3.3cm. MRI revealed a sellar and suprasellar mass consistent with a pituitary macroadenoma compressing the optic chiasm. Further labs revealed a morning cortisol 2.6ug/dl, TSH 0.25mIU/ml, FSH 2.3mIU/ml, GH <0.1ng/ml, LH 0.4mIU/ml, prolactin 2.6ng/ml. He was admitted for management of SIADH. He became more somnolent and his sodium decreased with normal saline infusion. He was started on 2% NaCl infusion, his mental status continued to decline so he was intubated. Repeat MRI revealed the same mass with heterogeneous signals changed from prior MRI, suggestive of internal hemorrhagic necrosis and pituitary apoplexy.

He was started on intravenous levothyroxine and hydrocortisone, with improvement in his sodium and mentation. He was extubated 3 days later. He was discharged several days later on levothyroxine and hydrocortisone, with plans for a transphenoidal hypophysectomy as outpatient. Preoperative MRI 3 months later revealed a significant decrease in size but persistent heterogenous, partially cystic, enhancing pituitary adenoma now measuring 1.4 x 0.9cm. The previously seen suprasellar extension was no longer present. The surgery was therefore cancelled. An MRI 6 months later revealed continued decrease in size of the residual cystic sellar lesion.

Conclusions:

Pituitary apoplexy may be managed conservatively in select stable patients. Tumor resolution may occur in up to half of patients treated conservatively with variable improvement in symptoms.
Introduction: Hyponatremia is the most common electrolyte abnormality and is associated with increased morbidity and mortality. Urea has demonstrated effectiveness in hyponatremia associated with the Syndrome of Inappropriate Antidiuretic Hormone Secretion (SIADH), heart failure, cirrhosis, and psychogenic polydipsia. Yet urea therapy remains rare. We describe two cases of urea therapy for hyponatremia.

Methods: Participants with chronic, moderate hyponatremia (serum sodium [SNa] 120–129 mmol/l) from nephrology clinics were enrolled in a prospective, non-randomized trial. Participants ingested 15–60 grams of urea powder dissolved in water; serum and urine sodium, potassium, and osmolality were monitored at pre-specified follow-up visits.

Results: Case 1: A patient with cirrhosis secondary to non-alcoholic steatohepatitis presented with a SNa of 125.3 mmol/l (SD=4.12, n=39) during the prior 42 days. Urea treatment resulted in SNa correction to 138 mmol/l after 4 days. Hepatic encephalopathy developed after inadvertent lactulose discontinuation, and she was disenrolled from the study after listing for orthotopic liver transplant.

Case 2: A patient with SIADH and SNa of 128.8 mmol/l (SD=3.7, n=9) during the prior 633 days improved her SNa to 132 mmol/l after 7 days' treatment with persistent correction afterward, without adverse events. Electrolyte-free water clearance, volume of plasma cleared of solute-free water per unit time, during urea therapy increased from 9% to 19%, with an increase in urine osmolality.

Conclusion: Urea is an oral, less expensive, effective alternative treatment for chronic hyponatremia.
Breaking Addison’s Heart: Takotsubo Cardiomyopathy Following Adrenal Crisis in Addison’s Disease

62-year-old woman with a history of Addison’s disease, ulcerative colitis, and colon cancer status post colectomy, radiation, and chemotherapy presented with fever, tachycardia, and hypotension refractory to intravenous fluids. There was concern for adrenal crisis and she was started on vasopressors and stress-dose steroids. The patient subsequently developed acute hypoxic respiratory failure requiring intubation. Infectious workup was negative. Chest radiograph revealed flash pulmonary edema. Troponin peaked at 1.81 with new q waves in antero-septal leads. Echocardiogram revealed an ejection fraction of 18% with severe septal and lateral hypokinesis, and apical ballooning consistent with ischemic heart disease. Patient was diuresed, weaned and successfully extubated. She underwent a left heart catheterization that showed non-obstructive coronary arteries. Repeat chest radiograph showed improvement in pulmonary edema. Repeat echocardiogram eleven days later revealed an EF 65%. She remained hemodynamically stable and was discharged. We present the third reported case of stress induced cardiomyopathy in a patient with Addison’s disease. Patients with Addison’s disease are inherently in a low glucocorticoid state, which results in an insufficient amount of cortisol and catecholamines to respond to physiological stress. Despite this fact, this is the third reported case of Takotsubo in an Addison’s patient – suggesting that there may be another mechanism (perhaps sepsis, cytokine induced, or iatrogenic) that may contribute to this cardiomyopathy. We suspect that the combination of genetic predisposition and sudden exposure to a high steroid/catecholamine dose in an Addison’s patient may have induced Takotsubo.
Yellow Nail Syndrome: It’s All About the Physical Exam

A 68 year old woman with a PMHx of smoking, chronic LE lymphedema, alcoholic liver disease, HTN and recurrent pleural effusions presented to us with chief complaint of shortness of breath. She was found to have another pleural effusion. Thoracentesis was performed and revealed a transudative effusion. Upon further examination, she was found to have yellow discoloration of her nail beds. She stated that her nails grew very slowly and attributed her nail discoloration to smoking. Interestingly, the discoloration was congruent on all of her nail beds. She did not complain of pain in her fingers or hands, she did not have any dryness of her fingertips. Upon literature review, she was thought to have a significantly rare disease of Yellow Nail Syndrome (YNS). Our patient exhibited the classical triad of YNS, characterized by lower extremity lymphedema, pleural effusions and yellow discoloration of nail beds.

The etiology of YNS has remained medically unknown. It has been thought that lymphatic dysfunction or defective lymph drainage has been hypothesized as the cause of the abnormal coloration of the nail beds as well as contribute to the chronic pleural effusions.

This case is was especially interesting to me in regards to the physical examination findings. I find it very interesting that we as physicians can diagnose many different diseases and syndromes based on our physical examination skills. I believe this is a skill that we can continue to build upon and hone.
Serous Carcinoma of the Peritoneal

Introduction:
Primary peritoneal serous carcinoma (PPSC) and ovarian cancer both have rare occurrence with the ratio of 7 to 120 per million cases. They are difficult to differentiate due to their similar histology and clinical symptoms.

Case presentation:
80-year-old healthy female who was on estrogen replacement therapy for the past 5 years and had ovary preserved-hysterectomy 40+ years ago presented with bloating. CT chest abdomen pelvis showed new ascites, a sheet like peritoneum mass with omentum nodules and mediastinal adenopathy. Both ovaries were not seen on the CT scan. Open-biopsy showed metastatic high-grade serous carcinoma to the omentum. It was diagnosed as metastatic stage IV ovarian cancer or PPSC. Neo-adjuvant chemo with Paclitaxel/Carboplatin was planned before surgical de-bulking.

Discussion:
Base on diagnostic criteria of PPSC, this patient likely has PPSC base on its absence of ovarian involvement and its significant extra-ovarian involvement. The clinical significance of differentiating PPSC from ovarian cancer is minimal because the treatment is the same. Epithelial layer of the ovary and the peritoneum both derive from coelomic epithelium during embryonic development. The same treatment will target both cancers. The etiology of PPSC is not as well studied as ovarian cancer due to its rarity. Evidence showed that BRCA1 increased the risk of developing PPSC by 5%. Studies have demonstrated that estrogen use increases the risk of serous type ovarian cancer by 30%. Because the peritoneal and ovary share a similar cell origin, it is safe to suspect estrogen can play a role in PPSC.
Atypical Diagnosis of Dry BeriBeri

Introduction: Beriberi is a manifestation of thiamine deficiency that can present with symptoms of polyneuropathy. With the increasing epidemic of morbid obesity, the events of bariatric surgical intervention is also on the rise, leading to increased risks of micro-nutritional deficiency complications such as beriberi. Presenting symptoms of beriberi can be nonspecific like fatigue with eventual progression to ataxia, heart failure, and encephalopathy. With thiamine being an essential cofactor in several cellular metabolism processes, its deficiency is related to altered mitochondrial function, and selective neuronal death.

Case: This is a case of beriberi in a 36-year-old female with an extensive history of gastric surgeries. She presents to the hospital with encephalopathy and ataxia worsening in the week prior to admission. The patient presented with a normal thiamine level, however, her symptoms of ataxia and encephalopathy had promptly resolved with the administration of thiamine. After TPN and vitamin repletion, her mentation returned to baseline, and she was on her way to full recovery.

Conclusion: Thiamin is an essential micronutrient that is not produced intrinsically. Its deficiency can lead to altered neurological function due to its involvement in glucose metabolism within the central nervous system. Thiamine storage is usually depleted after about 2 weeks without supplementation, but symptoms of its deficiency can be delayed in onset. Patients can also be resistant to thiamine if a mutation exists within a specific thiamine transporter gene. Further analysis of these carrier genes would be required for a definitive explanation of our patient’s atypical presentation of beriberi.
An Unusual Case of Diarrhea: Losartan Induced Sprue-Like Enteropathy

Severe, chronic diarrhea with substantial weight loss can occur after long term Losartan use. However, the infrequency with which this occurs makes the diagnosis challenging. The mechanism of action is still being studied, although, it has been hypothesized that cell mediated immunity plays a role. The importance of considering drug induced enteropathy in the differential of chronic diarrhea is highlighted in this case, as prompt diagnosis results in less unnecessary testing and better outcomes.

A 67-year-old male with hypertension on Losartan 50 mg for three years presented with loose stools for a week and unintentional weight loss of 20 pounds. Bowel movements were loose and 10-12 liters daily. He had tried antimotility agents, Ciprofloxacin and Metronidazole without change in symptoms. Extensive workup was completed to rule out infectious, inflammatory, absorptive and autoimmune causes. Enteroscopy with biopsies revealed marked villous blunting of the duodenum and lymphocytosis. The patient’s diarrhea resolved after 2 weeks of supportive treatment and discontinuation of Losartan. Follow up enteroscopy after 3 months showed complete resolution of villous blunting and atrophy.

Sartan induced enteropathy is one of the reversible causes of enteropathy with nonspecific features suggestive of infectious or autoimmune conditions yet negative serology. One should maintain a high index of suspicion in the right clinical context to ensure timely treatment and prevent several unnecessary tests.
The Troubling Trilogy of the Triphasic Response

Introduction
Dramatic shifts in water balance are common following neurosurgery. One extremely rare pattern is characterized by a progression from hypernatremia/polyuria to hyponatremia/oliguria and ultimately back to hypernatremia/polyuria as presented below.

Case Description
62-year-old female who underwent transsphenoidal resection of craniopharyngioma. Approximately 48 hours postoperatively, patient developed polyuria with 6.1L/day of urine output (UOP). She was started on 100 mcg of Desmopressin with improvement in UOP. One week later, the patient developed hyponatremia of 129 mmol/L associated with reduced UOP of 1.2L/day. With discontinuation of Desmopressin and fluid restriction of 1000 cc/day, the patient’s sodium improved. The patient’s sodium began climbing again to 144 mmol/L with urinary output of 6.9L/day on the several weeks later. Patient was again started on Desmopressin (titrated to 200 mcg/day) and discharged with plans to continue treatment for suspected DI indefinitely.

Discussion
This uncommon complication following hypothalamic damage is known as the triphasic response. The initial polyuric phase of the syndrome is frequently seen with hypothalamic stalk injury; whether due to neurosurgical intervention or otherwise. The following antidiuretic phase is characterized by hyponatremia in transient DI. Excessive water intake carried from initial polyuric phase combined with the release of stored ADH from the degenerating posterior pituitary causes the hyponatremia. The final phase is characterized by polyuria and development of suspected permanent DI. The etiology of this last phase phenomenon remains unclear. One suspected mechanism, as in this case, is dipsogenic DI where learned, psychogenic polydipsia preludes polyuria.
Duodenal Bezoar Complicating Presentation of DKA in a Patient with Diabetic Gastroparesis

Introduction
Gastrointestinal bezoars are aggregates of edible or inedible undigested or partially digested material that is often found as a concretion in the stomach, sometimes in the small intestine and rarely in the large intestine. Patients with altered gastrointestinal anatomy or motility disorders are at increased risk.

Case presentation
27-year-old female was brought to the emergency department for lethargy, drowsiness and confusion. Clinical and laboratory evaluation revealed severe DKA. She had a history of peptic ulcer disease and uncontrolled type 1 diabetes mellitus complicated by gastroparesis. DKA was treated appropriately and her mental status improved. However she continued to experience epigastric pain. Hemoglobin decreased from 12.3 mg/dL to 6.6 mg/dL by day 5 of admission with no evidence of blood loss or hemolysis. Given history of peptic ulcer disease and persisting epigastric pain in the setting of acute anemia, she underwent an upper endoscopy. This revealed a large bezoar in the duodenal bulb. It was fragmented using rat tooth forceps, disimpacted and allowed to pass distally. There was mild mucosal erythema in the duodenum but no ulceration. Patient’s epigastric pain resolved and her hemoglobin subsequently improved with iron supplementation.

Discussion
Gastrointestinal bezoars are rare but can present with abdominal pain and rarely cause intestinal obstruction. Clinicians should be aware of this differential diagnosis especially when managing patients with altered gastrointestinal anatomy or motility disorders. Endoscopic treatment of gastric bezoars is fragmentation and retrieval but intestinal bezoars can be difficult to retrieve and can be allowed to pass distally following fragmentation.
Allergic Agina: A New Differential Diagnosis and Pathophysiology of Chest Pain

Kounis syndrome is a novel diagnosis as a cause of Acute Coronary Syndrome that is based on allergic cell mediators released causing localized coronary vasospasm. In some cases the vasospasm can directly cause established plaques to erode and cause coronary artery occlusion. Diagnosis and treatment of Kounis syndrome remains unclear at the moment. The half life of tryptase is 90 minutes and is a marker of mast cell activity and has a sensitivity of 73% and specificity of 98% in detection. Treatments include corticosteroids and mast cell stabilizers once a conventional myocardial infarction has been ruled out.

Our patient is man with diabetes, OSA, hyperlipidemia, mastocytosis, obesity and NSTEMI. He developed chest pain, diaphoresis and shortness of breath. The patient had serial cardiac enzymes which were indeterminate, a normal EKG, and a normal echocardiogram. The patient had coronary catheterization that showed no coronary arterial disease. Given the history of mastocytosis and symptoms, Kounis Syndrome was investigated. An ESR was elevated at 24 and tryptase was 9.2, but was checked almost 24 hours after initial presentation. The patient was diagnosed with Kunis Syndrome and was discharged home with montelukast.

Our may bring a novel approach to the entity of chest pain. Because tryptase levels can be elevated within 90 minutes of an episode, it should be checked on initial presentation. Our case may help educate some in this diagnosis as well as establish some foundation in possible new pathophysiological approach to chest pain, coronary artery disease and stent thrombosis.
A Twist of Fate - A Rare Etiology of Dyspnea: Congenitally Corrected Transposition of the Great Arteries with Dextrocardia

Congenitally Corrected Transposition of the Great Arteries (CCTGA) is a congenital anomaly of the heart accounting for less than 1% of all heart defects. In CCTGA the looping is done in a leftward fashion resulting in the anatomical left and right ventricles being functionally switched. The right ventricle takes on the role of systemically delivering blood to the body while the left ventricle takes the role of delivering blood to the lungs for oxygenation. The myocardial differences in the left and right ventricles follow their anatomy and the stronger left ventricle takes the role of supplying blood to the low pressure pulmonic system while the weaker right ventricle is tasked with the role of providing pressure for the systemic system. This provides the potential for pulmonary hypertension. Our patient is a 52 year old man who presented with a five month history of progressive shortness of breath, initially diagnosed and treated as COPD. The patient underwent right and left sided cardiac catheterization for further assessment of his pulmonary and cardiac function, and the diagnosis of CCTGA with dextrocardia was made. Ejection fraction was difficult to ascertain on surface echocardiogram due to his dextrocardia, but intracatheterization pressures revealed an ejection fraction of 20% performed by the right ventricle and pressures consistent with pulmonary hypertension with marked valvular dysfunction. The patient was optimized medically and discharged to follow up at a tertiary center. Due to his advanced cardiomyopathy, he will require evaluation for heart transplantation.
A Rare Case of Fibrillary Glomerulonephritis in a Patient with Gastric Adenocarcinoma

Introduction: Fibrillary glomerulonephritis (GN) is a rare glomerular disease associated with malignancies, chronic infections and autoimmune disorders. To our knowledge, since its report in 1977, this is only the second case of fibrillary GN in a patient with gastric adenocarcinoma.

Case Summary: A 63-year-old Caucasian female with a history of hypertension and anemia presented with new onset ascites, hematemesis, and epigastric pain. Upper endoscopy revealed a large ulcerative gastric mass with irregular borders. Biopsies showed poorly-differentiated adenocarcinoma with signet ring cell features. Patient received chemotherapy with 5-FU, docetaxel and cisplatin. Creatinine was increased (2.27 mg/dL) and did not respond to hydration. Urine analysis showed proteinuria and microscopic hematuria. ANA, C3, C4 were within normal limits. Kidney biopsy showed focal sclerosing GN and thioflavin-T stain was negative for amyloid. Cisplatin was changed to oxaliplatin. Kidney function worsened but patient refused hemodialysis, and opted for palliative care.

Discussion: Fibrillary GN was first described by Rosenmann and Eliakim. This is a rare disease found in <1% of native kidney biopsies; approximately 150 cases have been reported. Clinically, it is indistinguishable from other causes of proteinuria or nephrotic syndrome. Diagnosis is by kidney biopsy showing glomerular deposition of non-branching, randomly-arranged fibrils, which are less than 30 nm in size, and are Congo-red negative. It has been associated with autoimmune diseases, cryoglobulinemia, lymphoma, and multiple myeloma. Pathogenesis is unclear but immunofluorescent findings suggest an immunologic origin. To date, there is no cure. About 50% of patients develop end-stage renal disease within 2-4 years of diagnosis.
Metastasis to Prostatic Fossa from Adenocarcinoma of the Duodenum - A Rare Case Report

Metastases to prostatic fossa (PF) from duodenal adenocarcinoma (DA) is rare. As per our knowledge, there has been no reported case. In 2014, a 76-year-old African-American male with a history of prostate cancer status-post surgery and radiotherapy presented with small bowel obstruction. CT abdomen showed an obstructing duodenal mass with lymphadenopathy. CEA and CA 19-9 were 7.7 ng/mL and 431.6 U/mL, respectively. Exploratory laparotomy showed a mass adherent to the jejunum and colon for which en-bloc duodenojejunal resection and left hemicolectomy was performed. Histopathology showed well-differentiated adenocarcinoma and adjuvant chemotherapy FOLFOX was administered. PET-CT and tumor markers were negative after treatment. In May 2016, tumor markers were elevated while PET-CT showed increased metabolism in the PF. Repeat PET-CT in August 2016 showed increased metabolism with the involvement of the bladder neck. Cystoscopy with biopsy showed invasive adenocarcinoma while positive immunostaining with CK20 and CDX2 and negative for PSA and PSAP confirmed its origin from DA. The patient opted for chemotherapy and FOLFIRI and bevacizumab was started.

DA is uncommon with a prevalence of 0.35% and 30-40% among gastrointestinal and small intestinal tumors, respectively. 40-65% of DA have nodal metastasis at diagnosis. It metastasizes most commonly to the liver, lungs and rarely to superior vena cava. PET-CT is useful for detecting both primary DA and metastasis. DA is treated with pancreaticoduodenectomy with lymph node dissection if localized, while adjuvant chemotherapy is indicated in metastasis. Patients with stage III and IV disease have decreased survival compared with stage 0, I and II.
Elephantiasis Nostras Verrucosa: A Rare and Deforming Complication of Chronic Lymphedema

Introduction:
Elephantiasis Nostras Verrucosa (ENV) is a rare complication of chronic non-filarial lymphedema characterized by significant deformation secondary to dermal fibrosis, hyperkeratosis, verrucous and papillomatous eruptions and fetid odor due to chronic bacterial and fungal colonization.

Case Summary:
A 53-year-old morbidly obese male presented with progressive, non-pitting, bilateral lower extremity edema for two years, complicated by the presence of non-tender, nodular and papillomatous lesions and associated with serosanguinous secretions, crust formation and severe pungent odor. He reported maggot infestation of the left leg; however, he had never sought medical attention. Vital signs and labs were normal. He was diagnosed with ENV secondary to chronic venous insufficiency (CVI). Maggot suctioning was done and compression stockings were placed. He was treated with oral clindamycin, topical deodorants and keratolytics. Surgical debridement was planned for the future.

Discussion:
ENV is caused by recurrent staphylococcal or streptococcal infections in the setting of chronic lymphedema. Etiology of lymphedema includes bacterial infections, obesity, congestive heart failure, CVI, radiation-induced fibrosis and malignant infiltration of the lymphatics. Pathogenesis involves excess protein-rich exudate causing fibroblastic proliferation and local immune system suppression resulting in fibrosis and recurrent lymphangitis with a perpetuating course. In rare instances, ENV can lead to septic arthritis and osteomyelitis necessitating amputation. The literature on management is limited; it includes conservative, mechanical, medical or surgical options. Eventual choice should be based on the patient’s presentation, co-morbid conditions and preferences. Management can be discouraging due to recurrences.
A Rare Case Report of Propionibacterium Acnes Splenic Abscess

INTRODUCTION: Propionibacterium acnes (P. acnes) is a slow growing, anaerobic, gram positive bacteria which is among the normal human flora. It rarely causes invasive infections. To our knowledge it has been associated with splenic abscesses in only four cases.

CASE PRESENTATION: A 64 year old Caucasian female with a past medical history significant for morbid obesity, hepatitis C, hypertension, multiple episodes of abdominal wall cellulitis, diabetes mellitus type 2 on insulin, intravenous and intradermal drug abuse presented to the emergency department complaining of intermittent left lower chest and shoulder pain for the past 6 weeks along with multiple episodes of vomiting. Her initial laboratory data revealed leukocytosis of 17,200 with a left shift and she was found to have multiple fever spikes. Abdominal and pelvic computerized tomography showed a splenic hypodense lesion, likely an abscess. Specimens from interventional radiology aspiration and splenectomy grew Propionibacterium acnes. Following splenectomy, patient’s symptoms resolved.

DISCUSSION: The exact cause for splenic abscess in our patient remains unclear. One case was reported in a diabetic where it was suggested that P. acnes was introduced during insulin administration. In another report, patient had a history of drug abuse. In our patient, intradermal drug abuse, multiple episodes of abdominal wall cellulitis along with insulin administration and intravenous drug abuse were the risk factors.

CONCLUSION: P. acnes has rarely been associated with splenic abscesses. Our patient represents the fifth case to our knowledge.
Severe Septicemia Due to Listeria Monocytogenes in an Immunocompetent Patient: A Rare Case Report

INTRODUCTION: Listeria monocytogenes (Lm), a gram-positive bacillus, is occasionally implicated in foodborne illnesses and presents as a self-limited, febrile gastroenteritis in immunocompetent hosts. In contrast, immunosuppressed individuals are susceptible to invasive infection causing septicemia and/or meningoencephalitis.

CASE PRESENTATION: A 44-year-old African-American male presented with vomiting, non-bloody, watery diarrhea and lethargy. He denied fevers, chills, abdominal pain, neck stiffness, photophobia, eating contaminated food or sick contacts. Exam showed fever, hypotension, tachycardia and mild abdominal tenderness but was negative for meningeal signs. Labs showed leukocytosis and elevated creatinine. CT abdomen showed enteritis and sigmoid colitis. The patient was treated for severe sepsis secondary to infective gastroenteritis with aggressive hydration, ceftriaxone and metronidazole. Hemodynamic parameters continued to deteriorate and he developed respiratory failure. Blood cultures grew gram-positive bacillus prompting antibiotic adjustment. Repeat labs demonstrated pancytopenia accompanied by progressive hyperlactatemia. HIV and C. difficile were negative. The patient required vasopressors and treatment for acidosis, but succumbed within 36 hours. Blood cultures were positive for Lm.

DISCUSSION: Lm leads to 1460 US hospitalizations annually; 260 result in death. It is usually a self-limited, food-borne illness in the immunocompetent, but individuals with suppressed cellular immunity are at risk for invasive disease. The diagnosis is made by a positive culture from an otherwise sterile location and by polymerase chain reaction if antibiotics have been administered. Lm responds well to ampicillin and gentamicin.

CONCLUSION: Although self-limited in immunocompetent individuals, in rare instances as in our patient, severe life-threatening infection may result; therefore prompt recognition and treatment is necessary.
Incidental Finding of Two Different Cancers in Separate Lungs

Introduction:
Lung adenocarcinoma (ADC) and squamous cell carcinoma (SCC) are two distinct subtypes of non-small-cell lung carcinoma. It is very rare for them to present at the same time in separate lungs. We present a case of pulmonary adenocarcinoma in one lung and squamous cell carcinoma in another.

Case:
74 year old female who is a smoker with a past history significant for chronic lymphocytic leukemia (CLL) presented with erythema and drainage from bilateral nasal vestibules. It was associated with diffuse swelling of the nares. Biopsy of the nasal lesion showed Methicillin-sensitive Staphylococcus aureus (MSSA) and herpes virus and CLL. She received cephalexin and valacyclovir for the infections.

Bilateral lung masses were found incidentally on the chest x ray. Computed tomography (CT) chest showed right upper lobe and left lower lobe spiculated lung masses with diffuse mediastinal, hilar, axillary, supraclavicular and infraclavicular adenopathy and splenomegaly. CT guided biopsy of the left lung mass showed adenocarcinoma (pT1B pN0 M0) and that of the right lung mass showed squamous cell carcinoma (pT1A pN0 M0). Endobronchial ultrasound (EBUS) guided biopsy of mediastinal lymph nodes showed chronic lymphocytic leukemia/small lymphocytic lymphoma (CLL/SLL). CT abdomen/pelvis did not show any evidence of metastatic lung cancer. She was started on Stereotactic Body Radiation Therapy.

Discussion:
The above picture could be due to either transdifferentiation of ADC to SCC or concomittent presentation. Mutational analysis could provide further clarification. Management varied significantly since the two lesions were the primary tumors rather than metastatic lesions from a single primary tumor.
Profound Hyponatremia Secondary to Excessive Intake of Coke Zero

Introduction:
Psychogenic polydipsia or “compulsive water drinking” refers to the excessive fluid intake (>3L/day) leading to hyponatremia. We describe a case of hyponatremia-induced seizures secondary to excessive intake of Coke Zero.

Case description:
A 74 year old male with past medical history of Bipolar disorder and Depression was brought to the ED after he was found lying on the floor in a puddle of urine with his hands twitching. At baseline, patient lives independently and takes Buproprion, Risperidone, and Mirtazapine. In the ED, patient was alert, confused, and denied excessive water intake. Labs were significant for sodium: 114 meq/L, Creatinine: 0.89 mg/dL, CPK: >20,000 U/L, Lactate: 1.7 mmol/L, Prolactin: 18.1 ng/mL, Serum osmolality: 245 mosm/kg, Urine Osmolality: 146 mOsm/kg, TSH: 1.43 u/mL, Morning cortisol: 15.2 mcg/dL. Electrocardiogram, CXR, & CT head were negative. Patient was started on normal saline @150 cc/hr. Sodium and CPK normalized over the following few days. Further history taking revealed that he had been drinking at least 12 cans of Coke Zero over the past year. Patient was educated on risks of excessive fluid intake and remained seizure free.

Discussion:
Psychogenic polydipsia or “compulsive water drinking” is often misunderstood as “excessive water intake” rather than excessive intake of fluids. Coke Zero consists of mainly carbonated water, which explains why our patient developed hyponatremia. The high amounts aspartame and caffeine may have decreased seizure threshold and potentiated the epileptogenic potential of hyponatremia. Careful history taking was crucial in diagnosing, treating, and preventing recurrence of life-threatening hyponatremia and seizures.
A Rare and Life-Threatening Cause of Gross Hematuria

Introduction: Uretero-iliac artery fistula (AUF) is a rare, life-threatening cause of intermittent gross hematuria in patients with risk factors for fistula development. We present such a case in an elderly patient.

Case: A 79 year old female with bladder cancer s/p cystectomy with ileal conduit, radiation therapy, bilateral ureteral stents (exchanged two weeks ago), and urostomy 13 years ago presented with altered mental status. The patient was noted to have large amount of red urine with blood clots in her bag. Laboratories were notable for hemoglobin 6.0g/dl, creatinine 1.8mg/dl, and urine microscopy with numerous RBCs and WBCs. CT abdomen/pelvis revealed subtle increased attenuation within the right renal pelvis, grossly stable appearance of bilateral ureteral stents, bilateral hydronephrosis. The patient was started on meropenem. She underwent bilateral ureteropyeloscopy with removal of ileal conduit clots and ureteral stent exchange, resulting in resolution of hematuria. On day three she bled massively, developed hemorrhagic shock, and was transferred to the ICU where she received vasopressors and 6U PRBCs. CT showed arterial hemorrhage from the left common iliac artery into the left ureter with a focal arterial pseudoaneurysm and extravasation into the ureter. The patient underwent endovascular placement of a covered stent to repair the fistula.

Discussion: The risk factors for AUF include prolonged use of ureteral stents, pelvic irradiation, and pelvic surgery. Diagnosis is challenging because bleeding is intermittent; imaging studies are often negative. Pyelogram, angiography, and exploratory surgery are the most useful diagnostic approaches. Repair is either surgical bypass or endovascular repair.
Introduction:
Pseudogout is a crystal arthropathy caused by calcium pyrophosphate crystal deposition (CPPD). Although pathophysiology of this disease is still not well understood, it is related with certain metabolic disorders including hyperparathyroidism. Paradoxically, parathyroidectomy can lead to acute flare of pseudogout, which is unusual sequela.

Case Report:
57 year old male with past medical history of hyperparathyroidism presented with 4 days of acute right wrist pain associated with swelling and erythema. Patient underwent parathyroidectomy 6 days prior to presentation. Leukocytosis (17,800) and elevated CRP was found upon admission. Also, significant hypocalcemia (Ca 7.3) was revealed. As the patient spiked a fever (101.3) and blood culture started coming back positive with gram positive cocci in cluster, broad spectrum antibiotics were started. X-ray was obtained, which revealed possible chondrocalcinosis. He was started on oral prednisone which led to significant improvement of the symptom. Positive blood culture turned out to be contamination, and other work ups including uric acid, anti-ccp Ab, and rheumatoid factor came back negative. Patient left the hospital against medical advice, but was given taper course of oral steroid which led to complete resolution of the symptom.

Discussion:
Pseudogout flare is an unusual sequela of parathyroidectomy and the presentation can mimic other causes. Fall in calcium level after parathyroidectomy can lead to CPP crystal shedding into the synovial fluid, which can cause pseudogout flare. Treatment consist of NSAIDs, colchicine, or local/systemic steroids. Early recognition of this phenomenon can help avoiding unnecessary empiric treatment and evaluation.
A Healthy Young Female with Severe Coronary Heart Disease

Introduction: Acute Myocardial Infarction (AMI) occurs at a frequency of 1/1000 person years in African American women aged 35-44. The risk of death is higher in young women after AMI. We present a case of severe CHD in a young female without significant risk factors.

Case Report: A 36-year-old athletic female with a history of well controlled hypertension without medications, and occasional cigarette smoking presented with acute severe chest pain. EKG showed ST elevation in V2, V3 and initial troponin was normal. She underwent defibrillation 8 times for ventricular fibrillation. Three drug eluting stents were placed during emergency cardiac catheterization, 2 in LAD (100% proximal occlusion) and 1 in RCA (100% mid segment occlusion). ECHO showed an LVEF of 20%. She had LDL cholesterol of 69, HDL of 55 and triglyceride of 49, done on presentation. TSH, homocysteine, and HbA1C were normal. There was no hypercoagulable state, endocarditis, oral contraceptive use, family history of premature CHD, or connective tissue disease. After 16 days of hospitalization, the patient was discharged on optimal medical treatment of CHD and CHF and lifevest.

Discussion: Although not common, this case shows a low risk young female with severe CHD. Risk factors in young women include hypertension, smoking, dyslipidemia, type 2 diabetes, obesity, depression, spontaneous coronary artery dissection, hypercoagulable states, connective tissue diseases and psychosocial stressors. Despite the low incidence, AMI should always be excluded in a patient with chest pain. The quick, simple and cost effective EKG should be done in such patients.
A Rare Case of Candida Albicans and Candida Glabrata Pneumonia in a Patient with Lung Adenocarcinoma

Introduction: Candida species rarely causes primary pneumonia. It accounts for only 0.7% of the patients with pneumonia in the intensive care unit. Candida albicans is the most commonly identified pathogen. However, Candida glabrata has been implicated. We report a case of C.albicans and C.glabrata pneumonia in a patient with newly diagnosed lung adenocarcinoma.

Case Description: A 72 year-old male smoker with Diabetes Mellitus (DM) and chronic obstructive pulmonary disease, presented to the hospital with progressively worsening shortness of breath. He was found to be hypoxic secondary to multifocal pneumonia demonstrated on chest X-ray. A computed-tomography (CT) of the thorax three months earlier demonstrated bilateral lower lobe ground glass opacities with mediastinal and hilar lymphadenopathy. Since interstitial lung disease was in the differential diagnoses, a high resolution CT-Thorax was performed which demonstrated worsening lymphadenopathy and was concerning for malignancy. Bronchoscopic biopsy revealed poorly differentiated adenocarcinoma. Sputum culture grew rare yeast. Fungal culture from bronchoalveolar lavage fluid grew C.albicans and C.glabrata. Since the patient did not respond to empiric antibiotic therapy for pneumonia and the histopathology and fungal culture revealed Candida, the patient was started on high dose Fluconazole with significant improvement.

Discussion: Candida is more often a colonizer of the respiratory tract; a primary pneumonia with Candida is very rare, but maybe seen in immunocompromised states such as human immunodeficiency virus infection, DM, neutropenia and malignancies. Although rare, Candida should be considered as a primary cause of pneumonia in immunocompromised patients. When suspected, it should be investigated and treated.
Sjogren’s Syndrome (SS) with Very Rare Yet Challenging Complication at Diagnosis, Lymphocytic Interstitial Pneumonia (LIP)

Introduction: Sjogren’s syndrome (SS) is defined as a chronic autoimmune inflammatory rheumatic disorder that manifests with exocrine and extraglandular symptoms. Being more common in Europe and Asia, the incidence is 7/1,00,000 in US. Owing to greater prognosis, it’s important to identify concerning complications such as Lymphocytic Interstitial Pneumonitis (LIP) for effective management and follow up.

Case Description: A 52 year-old female with SS presented with hemoptysis for one month duration. She denied any fever, weight-loss or upper respiratory tract infection. She was found to have dry crackles in bilateral lower lung fields and leucopenia. She had similar complaints in the past and was treated for pneumonia and extensive work-up were done including serology, serial CT-thorax scans (ground glass opacities with intermittent improvement and deterioration), bronchoalveolar lavage, lung biopsies (areas of chronic inflammation with foci of fibrosis) over the span of 3 years. These results raised the concern for usual interstitial pneumonia with cryptogenic organizing pneumonia. She was treated with Corticosteroids and Rituximab intermittently. On this presentation, CT-thorax showed right subpleural nodules, bilateral diffuse patchy ground-glass and subsegmental atelectasis with scarring which are cardinal findings in LIP. She was treated with Rituximab with complete resolution of her symptoms.

Conclusions: SS has higher propensity of developing extraglandular manifestations which later is diagnosed as secondary SS; 9-20% of cases experience interstitial lung disease. However, LIP should not be overlooked, due to the strong association with primary SS and possible progression to bronchus-associated lymphoid tissue lymphoma and the availability of effective treatment.
Disseminated Kaposi Sarcoma in the Era of Highly Active Antiretroviral Therapy (HAART)

Introduction: Kaposi sarcoma (KS) is a vascular tumor caused by Human Herpes Virus 8 (HHV-8). Types: classic, endemic, iatrogenic and AIDS-associated (epidemic KS). Initially, KS was a prominent clinical feature of the first cases of AIDS, and occurred in 79% of the patients diagnosed in 1981. By 1997 the number was <1%. The decline in the number of cases of KS was attributed to the success of antiretroviral (ART).

Case Report: A 31 year old man with HIV/AIDS and multiple dark raised lesions on the face, extremities, cervical and axillary lymphadenopathy was admitted for cellulitis on the left thigh. A big dark raised lesion was noted in the area of the cellulitis. The patient was treated with Vancomycin. Concerns for KS and Bacillary Angiomatosis (patient had a cat) were raised. Biopsy from the skin, including the area of the cellulitis and lymphnodes, was positive for KS. The patient was diagnosed with disseminated KS, and started on liposomal Doxorubicin chemotherapy. He was also started on highly active antiretroviral therapy (HAART).

Conclusions: Even in the era of HAART, KS in HIV patients is still diagnosed. Rarely disseminated visceral involvement can occur as the initial event. Low CD4 count and visceral involvement predict poor prognosis. Reason for this is noncompliance on the part of patient and/or failure of our system to get these patients into HIV care. Treatment can be local, topical and chemotherapy and it only works with HAART. Cost range 20 to 6000 dollars and depends on the health insurance.
Left Atrioesophageal Fistula with Cerebral Air Embolism – A Devastating Complication of Atrial Fibrillation Ablation!

Introduction:
Left atrioesophageal fistula occurs in 0.03-0.04% of patients undergoing radiofrequency ablation for atrial fibrillation, days to weeks postoperatively. Clinical presentation is nonspecific, including septicemia, GI bleed and Stroke. It is associated with 71-83% mortality.

Case Description:
A 42-year-old male dilated cardiomyopathy presented to the ED with chest pain, syncope and encephalopathy, one month after extensive radiofrequency ablation for atrial fibrillation. Examination revealed Paraparesis and blindness. CT-Head showed numerous cerebral air emboli. Cardiac troponin was elevated. Patient was transferred to the ICU where his neurological status deteriorated. Repeat CT demonstrated intracranial hemorrhage with multiple cerebral infarcts. CT-Thorax did not report any obvious pathology. The patient worsened hemodynamically, requiring vasopressors and intubation. Overnight, he had a massive GI bleed and hematuria requiring transfusion. The following day, echocardiography showed air in the left atrium with a transmural defect. He had multiple episodes of cardiac arrest and expired. Autopsy revealed a 2 cm x 0.8 cm transmural defect of the esophagus communicating with a defect in the left atrium.

Conclusions:
Although rare, atrioesophageal fistula is due to thermal injury to esophagus during pulmonary venous isolation, should be considered in patients presenting with new neurological symptoms post-ablation. Prompt diagnosis followed by immediate surgical repair is vital to improve survival.
Intravenous Dipyridamole as an Inciting Factor for Hemorrhagic Stroke in a Patient Presenting with Ischemic Stroke

Introduction: Dipyridamole is used as an antiplatelet agent for secondary stroke prevention in combination with aspirin and intravenously for pharmacological stress testing. Common adverse events related to intravenous dipyridamole are headache, bronchospasm, and rarely acute myocardial infarction, arrhythmias and ischemic stroke. We present a case of hemorrhagic stroke following dipyridamole stress test.

Case Description: A 50 year old female presented to our hospital with sudden onset right sided weakness. The patient was not a candidate for tissue plasminogen activator due to prior intracranial bleed. CT head showed old medullary hemorrhage and no new hemorrhage or ischemia. Echocardiogram done as part of stroke work up showed new wall motion abnormalities. She underwent dipyridamole cardiolyte stress test. A few minutes later, she developed weakness in her right leg and left arm. Repeat CT head showed increase of old medullary hemorrhage and MRI also showed additional foci of intramedullary hemorrhage involving cervical and upper thoracic spinal cord and spinal cord edema.

Discussion: Dipyridamole stress testing is routinely performed and is considered relatively safe. It is commonly known to cause headache, hypotension and bronchospasm which are relatively reversible with aminophylline. Serious adverse events associated with dipyridamole are ischemic phenomena in the brain secondary to hypotension and an arterial 'steal phenomenon' with pre-existing stenotic lesions of the carotid artery. Even though the anti-platelet effects of dipyridamole are well known, the occurrence of intracranial hemorrhage after an intravenous dose of dipyridamole has not been reported.
Post-Nephrectomy Metastatic Renal Cell Carcinoma to Duodenum Leads to Massive Gastrointestinal Bleed

Introduction:
Life-threatening upper gastrointestinal bleed due to malignancy is relatively uncommon, with the duodenum being the least frequently involved site. Renal cell carcinoma (RCC) may metastasize to almost any site; duodenal metastasis is especially rare in renal cell carcinoma. Early detection, especially in the case of a solitary mass, helps in planning further therapy. We describe a case of duodenal metastasis from renal cell carcinoma, one of only a handful that have been previously described in the literature.

Case Description:
An 80-year-old female presented with progressively worsening shortness of breath, fatigue and generalized weakness for three months. She reported black tarry stools for 3-4 days prior to admission. She denied previous use of NSAIDS. Her past medical history was significant for hypertension and right renal cell carcinoma status post nephrectomy 12 years earlier. On physical exam, she was orthostatic and appeared pale. Laboratory tests on admission were significant for a microcytic, hypochromic anemia - hemoglobin of 7 g/dl, hematocrit 16.8%. Esophagogastroduodenoscopy showed a bleeding 4cm irregular, polypoid, ulcerative mass in the duodenal bulb, and a biopsy was taken.

Discussion:
Gastrointestinal tract metastases are a very rare cause of gastrointestinal bleeding. Small bowel involvement by metastatic tumors is rare and has been reported in only 1-2% of autopsy cases. RCC metastases account for 7.1% of these lesions. Solitary duodenal metastasis from RCC is very rare and most frequently involves the periampullary region or the duodenal bulb. Appropriate awareness, recognition and aggressive work-up in post nephrectomy for RCC are of utmost importance.
Recurrent Thrombotic Microangiopathy Due to Hypertensive Emergency

Case Report:
A 26-year-old male presented to emergency room with abdominal pain, nausea, and vomiting for 4 days. Medical history was significant for hypertension and end-stage renal disease managed with hemodialysis. Patient had been noncompliant with anti-hypertensive medications. Upon arrival, blood pressure was 231/123 mmHg and laboratory investigations revealed WBC 10,300/mm3, hemoglobin 7.8 gm/dL, platelet count 46,000/mm3, reticulocyte count 7.8%, total bilirubin 1 mg/dl, LDH 1,235 IU/L, and haptoglobin <10 mg/dL. Direct coomb's test was negative. Numerous schistocytes were identified on peripheral smear. Patient was diagnosed with TMA secondary to severe hypertension and was started on intravenous nicardipine. With appropriate blood pressure control, hematologic parameters improved within 10 days. Of note, patient had 2 other similar episodes of hypertension-induced TMA within a 3 months period.

Discussion:
The initial clinical presentation of hypertension-induced TMA is indistinguishable from TTP-induced TMA except for the presence of severe hypertension. A history of hypertension, significant renal impairment, relatively modest thrombocytopenia, and lack of severe ADAMTS-13 deficiency, serve as useful clues for diagnosis of hypertension-induced TMA.
Recurrent Intra-Cardiac Mass in a Patient with a History of Intra-Cardiac Lipoma Presenting with AICD Firing

Introduction: The majority of primary cardiac tumors are asymptomatic, benign, and do not raise concern for a malignancy. However, these tumors can carry a risk of life threatening arrhythmias, embolic phenomena, cardiac tamponade, heart failure and valvular dysfunction.

Case Presentation: 55 years old female with PMH of intra-cardiac lipoma with a history of debulking and AICD placement, for secondary prevention of ventricular tachycardia in 2002, presented with a complaint of AICD firing. Upon presentation, her vitals were stable and she had an unremarkable initial work up. She was placed in the clinical decision unit for observation. Overnight the patient developed ventricular tachycardia with a heart rate of 200, and her AICD fired 5 times. She was loaded and continued on an Amiodarone drip, as well as IV beta blockers. Transesophageal echocardiogram was performed and revealed a 7cm x 6 cm mass occupying the right ventricular apex. She exhibited no further episodes of ventricular tachycardia, and remained in normal sinus rhythm. She was later switched to oral Amiodarone, and beta blockers and was discharged home with plans to follow-up at the Mayo Clinic for further evaluation of probable recurrence of intra-cardiac lipoma.

Discussion: Intra-cardiac lipomas are usually benign in nature and symptoms are varied depending on the location. Patients should be monitored for recurrence and risk of cardiovascular complications. They also should be educated regarding symptoms that warrant further evaluation.
It is Not Always CHF Exacerbation: A Case of Acute Mitral Regurgitation Secondary to Rupture Chordae Tendineae

Introduction:
Etiology of ruptured chordae tendineae include: infective endocarditis, trauma, myxomatous disease, rheumatic heart disease, or spontaneous rupture. It is a medical and surgical emergency. Survival is dependent on early recognition and prompt intervention.

Case:
A 61-year-old male with history of severe ischemic cardiomyopathy (EF of 15%), COPD, and hypertension presented with rapidly progressive shortness of breath. The patient had been hospitalized multiple times for CHF exacerbations. He denied fever, chills, cough, chest pain, or lower limbs swelling. He reported sick contacts at home. He was afebrile with normal blood pressure and heart rate. He was tachypneic and hypoxic. Physical exam showed moderate respiratory distress, bilateral coarse rhonchi in all lung fields, normal heart sound with gallop but no murmurs, and +1 pitting edema on the lower extremity bilaterally. An electrocardiogram showed sinus tachycardia with occasional premature ventricular complexes. The patient’s CBC and CMP were unremarkable, except for a WBC of 27,000. His Troponin was negative. Our patient was given multiple doses of Intravenous lasix with minimal urine output of about 20 mL/hr. Because of the poor response to treatment, urgent echocardiography was ordered and showed severe mitral regurgitation secondary to ruptured chordae tendineae. An emergent surgical referral was made for mitral valve repair.

Discussion:
Acute mitral regurgitation has variable presentation, ranging from mild dyspnea to acute pulmonary edema or even cardiopulmonary arrest. It is often misdiagnosed as heart failure exacerbation or a primary pulmonary process. Echocardiography should be ordered immediately when acute mitral regurgitation is suspected clinically.
Introduction: Listeria monocytogenes is a facultative intracellular, Gram-positive bacillus. Infectious complications tend to impact extremes of age, immunocompromised individuals and pregnant patients. Gastroenteritis, meningitis and bacteremia are the most common manifestations of infection. We present a rare case of listeria associated acute cholecystitis.

Case: A 64-year-old female with history of metastatic breast cancer (receiving chemotherapy) presented with a three day history of fever of 101.5°F, nausea, and stabbing, non-radiating right upper quadrant (RUQ) abdominal pain associated with diarrhea. Her physical exam was notable for NO fever, mild tachycardia and RUQ tenderness to palpation and positive Murphy’s sign. Laboratory studies demonstrated a normal white blood cell count with 20% band forms. Her liver enzymes and liver function studies were normal. Abdominal ultrasound showed cholelithiasis with sludge and thickening of the gallbladder wall. HIDA scan revealed cystic duct occlusion. She had laparoscopic cholecystectomy performed without complications. Purulent bile was aspirated and cultures grew Listeria monocytogenes. She was treated with intravenous ampicillin therapy. She developed a rash and was discharged on one week of oral trimethoprim/sulfamethoxazole without evidence of extrabiliary complications of her infection and with the lack of neurologic symptoms, a lumbar puncture was not performed.

Discussion: Listeria monocytogenes cholecystitis has been reported in a limited number of cases making it a rare complication of the infection. It is likely an under-reported infection due to limited microbiological examinations of the gallbladder. The exact nature and duration of therapy for patients with Listeria cholecystitis remains unknown.
An Unusual Cause of Microangiopathic Hemolytic Anemia

Introduction: Microangiopathic hemolytic anemia (MAHA) is an important but rare cause of renal impairment. The most common causes of MAHA are thrombotic thrombocytopenic purpura (TTP), hemolytic uremic syndrome and disseminated intravascular coagulation. We report an unusual case of MAHA induced by malignant hypertension.

Case: A 40-year-old woman with a history of uncontrolled hypertension presented with a two-day history of blurred vision, oliguria, nausea and vomiting. Vital signs revealed blood pressure 300/180 mmHg, heart rate 120 beats/minute, respiratory rate 27 breaths/minute and temperature 98.6°F. Her physical examination was unremarkable except for lethargy. Laboratory evaluation was significant for: hemoglobin 11.3 g/dL, platelets 91 × 10^9/L, BUN 103 mg/dL, creatinine 15.95 mg/dL, reticulocyte percentage 5%, lactate dehydrogenase (LDH) 891 U/L and haptoglobin < 10 mg/dL. Peripheral blood smear revealed numerous schistocytes and helmet cells. Given the patient’s clinical presentation, severe hypertension-induced MAHA was suspected. She was started on intravenous nicardipine and underwent emergent hemodialysis. Within one week, the patient experienced resolution of symptoms with normalization of platelets, LDH and haptoglobin levels. Kidney biopsy demonstrated morphologic changes suggestive of malignant hypertensive nephropathy. After eight months, the patient remained oliguric requiring hemodialysis.

Conclusion: Severe hypertension-induced MAHA is a rare entity that responds well to antihypertensive therapy. However, in severe cases, permanent kidney dysfunction may be an unavoidable consequence. Treatment decisions should be made after careful exclusion of other potential causes.
IgA-dominant Acute Post-staphylococcal Glomerulonephritis: An Uncommon Variant of Post-infectious Glomerulonephritis

Introduction: IgA-dominant post-infectious glomerulonephritis (PIGN) is an uncommon cause of acute renal failure. It is most commonly associated with staphylococcal infections in adult diabetics. It presents with hematuria, proteinuria and a rising serum creatinine. Here we describe a Caucasian female with baseline diabetic nephropathy who was diagnosed with IgA-dominant PIGN.

Case: Our patient is a 54-year-old woman with a history of stage IIIb chronic kidney disease due to diabetic nephropathy with baseline creatinine of 1.6 mg/dL. She presented with gangrenous toe and a serum creatinine of 3.9 mg/dL. Urinalysis showed WBC casts, RBC casts, proteinuria and eosinophiluria. Ultrasound and serological workup for glomerulonephritis (GN) were unremarkable. She underwent toe amputation and ampicillin/sulbactam treatment. Creatinine levels increased. Steroids were initiated for a presumptive diagnosis of acute interstitial nephritis secondary to antibiotics, but delivered no improvement. Ultimately she required dialysis. Renal biopsy was performed and a diagnosis of IgA-dominant PIGN on pre-existing diabetic nephropathy was made.

Discussion: IgA-dominant staphylococcus-associated GN is uncommon and primarily occurs in middle-aged or elderly patients. Risks factors include diabetes, alcoholism, cancer, or IV drug use. It is a separate entity from post-streptococcal GN which is of a predominantly IgG deposition. It is distinguished from primary IgA nephropathy by hypocomplementemia and the presence of subepithelial humps on electron microscopy. Its presence should be suspected when patients have manifestations of active glomerulonephritis with a known recent or concurrent staphylococcal infection. Treatment should focus on eradicating the infection and controlling hypertension. Immunosuppressive therapy has no role in such patients.
Scrofula an Important Consideration for Lymphadenopathy

Scrofula is defined as tuberculous lymphadenitis in the cervical region. Scrofula is a prominent cause of lymphadenopathy in the developing world however, in the United States this occurrence is much less frequently seen.

A 23 year old Vietnamese man presented to his primary care provider due to worsening bilateral cervical and supraclavicular lymphadenopathy for 6 months. The patient was found to have emigrated from Vietnam two and a half years ago with several contacts concerning for tuberculosis. A PPD performed in office was found to be positive, however chest x-ray was negative. With this he was referred to St. John. On initial presentation he denied fever, night sweats, cough, hemoptysis, and dyspnea over the last several months. Physical exam showed lymphadenopathy and lungs that were clear to auscultation bilaterally. CT of the neck was performed with findings concerning for a metastatic process or lymphoma. The patient was started on RIPE therapy empirically. Excisional biopsy of a cervical lymph node was performed and showed necrotizing, caseating granulomas highly suggestive of tuberculosis. The patient was discharged from the hospital with plans to continue RIPE therapy and follow up with the Health Department.

Recognition of tuberculosis is an important public health issue. This case illustrates the importance of recent and distant travel and sick contact history. While tuberculosis lymphadenitis is relatively rare in the United States, given the right context it becomes remarkably common and important to recognize, not only for the health of the patient, but for the community as a whole.
A Fatal Case of Idiopathic Cold Agglutinin Disease

Introduction: Idiopathic cold agglutinin disease is an acquired condition where IgM antibodies react with polysaccharide antigens on the RBC surface at low temperatures causing varying degrees of hemolytic anemia. We present a case of rapidly progressive cold agglutinin disease with fatal consequences despite aggressive management.

Case: A 60 year old male with a history of hypothyroidism, recent hunting trip in northern Michigan, presented with purplish rash on his extremities, jaundice and confusion for 1 day. Physical exam was significant for temperature of 101.9F, jaundice and livedo reticularis. Hemoglobin was 3.5g/dl, down from 8.5g/dl the day before at PCP’s office (prior baseline of 15). Lactate dehydrogenase of 1181, haptoglobin<10, indirect bilirubin of 12.6 were noted. Serologies for CMV, EBV, lyme, HIV and mycoplasma were negative. Direct Coomb’s test was positive with anti-C3d and negative with anti-IgG. Despite initiation of steroids and transfusion of 7 units of packed red cells he continued to deteriorate. As patient was receiving emergent plasmapheresis he went into cardiac arrest and could not be resuscitated.

Discussion: The incidence of cold agglutinin disease is 1 in 300,000. It presents as purplish rash and jaundice. Severe hemolysis can lead to shock and heart failure. Rarely, prolonged exposure to cold can be fatal. A positive direct Coomb’s test with anti-C3d and a negative test with anti-IgG is specific. In rapidly progressing cases early initiation of Rituximab, plasmapheresis and blood transfusions may be life-saving. Despite these efforts patients may deteriorate relentlessly.
Colonic Duplication Cysts: A Curiosity

Introduction:
Colonic duplication cysts are a rare congenital abnormality commonly presenting before the age of 2. We present a case of endoscopic ultrasound (EUS) guided diagnosis and treatment of colonic duplication cyst.

Case report:
A 42-year-old female was evaluated for complaints of constipation for one year. There was no history of anorexia, weight loss or hematochezia. Patient had colonoscopy performed which showed a large submucosal lesion at the hepatic flexure. CT scan of abdomen with contrast was done which was normal. On colonoscopy with EUS, a tattooed subepithelial mass was seen in the hepatic flexure. On EUS, a 14 mm anechoic lesion lined by mucosa was seen consistent with a duplication cyst. The mucosa was then snared exposing the cyst cavity. Pathologic evaluation was negative for any malignancy.

Discussion:
Enteric duplication cysts are a rare entity encountered in the pediatric age group and extremely rare in adults. The most common site of enteric duplication cyst is the ileum. The clinical presentation varies in patients ranging from abdominal pain, volvulus, intussusception, ischemic bowel, obstruction, and constipation. CT scan and US abdomen are helpful in making the diagnosis of duplication cyst. Cases of malignant transformation have rarely been reported in the literature. Surgical resection is done for cysts found incidentally or intraoperatively. Our patient has features consistent with duplication cyst on EUS and biopsy was negative for any dysplasia. Effective drainage was established by hot snare resulting in resolution of patient’s symptoms.
Diffuse large B cell lymphoma (DLBCL) is the most common subtype of non-Hodgkin lymphoma. Advancements in molecular diagnostics have recently identified new subsets of DLBCL. Both new entities have a worse prognosis, require more intensive treatment regimens and demand non-standard testing for detection. The first subset, Double Hit Lymphoma (DHL), is diagnosed with fluorescence-in-situ-hybridization. Double Expressor Lymphoma (DEL) is the second subset and requires immunohistochemistry (IHC) for protein expression. Presented is a case of aggressive DEL that could have been undetected had clinical suspicion not been high.

A 59-year-old woman with a history of abdominal aortic aneurysm (AAA) presented to the hospital with severe radiating abdominal pain. Imaging to evaluate her AAA demonstrated lymphadenopathy suspicious for malignancy. Pending biopsies results, severe dyspnea developed requiring immediate reimaging which displayed substantial progression of extensive adenopathy. Pathology confirmed a diagnosis of DLBCL. Initial FISH testing was negative for gene rearrangements. Further testing was requested with IHC due to her clinical deterioration, which confirmed diagnosis of DEL. The aggressiveness and poor prognosis of this subtype would have been missed without additional non-standard immunohistochemistry.

DHL and DEL are aggressive subtypes of DLBCL. They have been shown to have shorter progression free survival and inferior outcomes when treated with standard chemotherapy. Testing for these molecular entities is not routine in practice. This case highlights the importance of standard IHC to assist with treatment decisions. This is a rapidly changing field and improved survival of these patients starts with recognizing them at time of diagnosis.
Hard to Swallow: Fresh Frozen Plasma Still Has Utility in Treating Hereditary Angioedema

Hereditary angioedema type II is a life-threatening condition characterized by recurrent attacks of angioedema throughout the body due to abnormal function of the C1-esterase inhibitor protein. A handful of costly drugs aimed at inhibiting the bradykinin pathway have been developed and utilized as the primary treatment strategy for managing attacks. Fresh Frozen plasma (FFP) has traditionally been used in the past as it contains C1-esterase inhibitor, however it is currently a second-line therapy for the treatment of HAE due to potential for adverse affects. We present a case of type II HAE with Icatibant treatment failure and a rapid recovery response to FFP. A 27-year-old female with a history of HAE type II confirmed by her immunologist presented to our hospital with acute onset oro-facial and abdominal swelling after 2 home doses of Icatibant failed to relieve her exacerbation. Despite impending airway compromise, the patient refused intubation, asking specifically for 4 units of FFP. In less than 30 minutes after infusion of the first two units she was speaking clearly, and her level of distress had markedly decreased. A similar response has been documented on multiple prior admissions. Agents designed to target the bradykinin pathway are recommended as first-line treatment options for HAE. Limiting the use of FFP in favor of these targeted therapies is sensible, however our case highlights an instance where FFP offered superior utility over a bradykinin B2 antagonist, and adds evidence that FFP offers rapid symptom reversal in a specific subset of HAE patients.
Grandma’s Home Remedy: An Enema You Should Not Try at Home

Introduction: Hydrogen peroxide (H2O2) is known to cause colonic injury. H2O2 enemas were a home remedy which was found to be caustic to the colon and this practice was largely abandoned. Today, self-induced chemical rectal injuries are rare. Here we present a case of chemical proctitis secondary to H2O2 enema.

Case: 34-year-old female with history of 6 days of constipation presented to the ED with hematochezia. The patient administered an enema of 3% H2O2 on her grandmother’s advice. She immediately had a large firm bowel movement followed by abdominal pain, cramping and diarrhea with hematochezia. Vital signs stable upon admission. Physical exam was significant for rectal tenderness without visual abnormality. CT abdomen/pelvis revealed diffuse wall thickening involving the rectum. Flexible sigmoidoscopy revealed diffuse inflammation, erosions, erythema and shallow ulcerations in the rectum consistent with proctitis secondary to chemical insult. Biopsies showed small fragments of colonic mucosa with chronic inflammation, focal ulcer and mild lamina propria expansion with architectural distortion. Treatment included IV fluids, morphine and 30 days of Melasamine suppositories.

Conclusion: Proctitis is the most common complication from H2O2 enema. The damage is caused when H2O2 meets the H2O2 catalase resulting in microbubbles of oxygen which force the blood out of the intramural vasculature resulting in corrosive, lipid peroxidation and oxygen free radial injury. H2O2 enema can result in colonic gangrene, gas embolization and fulminant colitis which is potentially fatal. Precaution must be taken with home remedies to ensure they will be safe and effective to prevent injury and fatality.
An Unexpected Case of Eosinophilic Cholangitis

When evaluating biliary strictures benign and malignant causes should be considered. Eosinophilic cholangitis (EC) is a rare benign disorder caused by fibrosis and stricture from eosinophilic infiltration. Etiology of the disease remains unclear, and often presents with obstructive jaundice. A 23-year-old male presented with worsening abdominal pain, vomiting, weight loss and jaundice. Physical exam showed epigastric tenderness with negative Murphy’s sign. Lab work up revealed: WBC of 10.56 with 22.9% eosinophils, total bilirubin 18.7 mg/dl, direct bilirubin >10.0 mg/dl, AST 247 U/L, ALT 606 U/L, and alkaline phosphatase 588 U/L. Autoimmune antibodies and viral hepatitis serology were negative. Hepatic ultrasound showed mild intra-hepatic and extra-hepatic biliary dilation. MRCP depicted moderate to severe intrahepatic biliary ductal dilation at the level of the porta hepatis. ECPR revealed a 25mm stricture in the common hepatic duct. Sphincterotomy and stenting was performed. Histopathology showed benign ductal mucosa with subepithelial fibrosis with mixed eosinophils/lymphocytes inflammation. Follow up at three months after high-dose steroid therapy revealed down trending of eosinophil percentage to 4.1%. Patient underwent a Roux-en-Y hepaticojejunostomy as repeat ECRP revealed incomplete resolution of stricture, pathology was consistent with eosinophilic cholangitis.

EC is a rare cause of biliary strictures. Diagnoses of EC is based on histopathological evidence of eosinophilic infiltration with or without peripheral eosinophilia. Treatment is a course of high dose corticosteroids with resolution of stricture in 3 to 5 weeks, however there is no specific guidelines on duration and dosing of therapy. In cases of incomplete resolution surgical intervention is pursued.
Oh Rats!! An Unusual Case of Jaundice Due to Leptospirosis

Conjugated hyperbilirubinemia in the absence of hepatocellular injury requires investigation of unique pathologic entities. Leptospirosis is a rare zoonotic infection, which can result in conjugated hyperbilirubinemia and AKI. Humans are infected via rodent-urine contaminated water or soil. Complications include thrombocytopenia, renal failure, ARDS, pulmonary hemorrhage and myocarditis.

A 43-year-old man with abdominal pain for five days associated with subjective fevers, generalized weakness and jaundice. He reported living with five dogs in a rat-infested home. He denied any recent travel or sick contacts. Exam revealed jaundice, scleral icterus and marked conjunctival suffusion. Lab work revealed leukocytosis, pyuria and conjugated hyperbilirubinemia but normal transaminases. The patient deteriorated within the next 12 hours, requiring intubation and vasopressor therapy. CXR revealed bilateral patchy infiltrates suggestive of ARDS. Ultrasound and CT scan of the abdomen were unremarkable. Leptospirosis antibody(IgM) was negative. The conjugated bilirubin continued to rise to a plateau of 22.8 mg/dL. Molecular testing with RT-PCR revealed Leptospira DNA. The patient was treated with ceftriaxone leading to resolution of AKI and hyperbilirubinemia.

Leptospirosis is rare in the United States, it must be considered in cases of conjugated hyperbilirubinemia and renal dysfunction. Hyperbilirubinemia is postulated to result from endothelial damage to hepatic capillaries and hyperplasia of Kupffer cells causing intrahepatic cholestasis. Important clues include conjunctival suffusion or rodent-infested living conditions. A Jarisch-Haerxheimer reaction (fever, rigors and hypotension) may occur after initiation of. High index of suspicion and initiation of antimicrobial therapy are essential in avoiding the complications of this devastating zoonotic disease.
A Case of Infective Endocarditis Caused by Granulicatella

Introduction: Infective endocarditis caused by Granulicatella, which is a nutritionally variant streptococcus, is rare. We report a case of infective endocarditis with a large mitral valve vegetation caused by Granulicatella adiacens in an elderly male with no risk factors, who was successfully treated with combination antimicrobial therapy.

Case description: A 66 year old male presented with a five month history of fevers, arthralgia, night sweats and generalized fatigue. On physical examination, he had a 2/6 holosystolic murmur best heard at the apex. Significant laboratory findings included a normal white cell count and elevated CRP. Echocardiogram showed large complex vegetation on the mitral valve measuring 2.0 x 0.7cm. A diagnosis of subacute native mitral valve infective endocarditis was made. He was started empirically on intravenous vancomycin and ceftriaxone, which was changed to intravenous ceftriaxone and gentamicin for 6 weeks after blood cultures grew Granulicatella Adiacens. Subsequent serial transthoracic echocardiograms showed almost complete resolution of the vegetation and he did not require any surgical intervention.

Discussion: Granulicatella endocarditis is associated with high rates of morbidity and mortality due to treatment failure, need for surgery and infection relapse. Patients should be monitored closely for complications like heart failure as they may require early surgical intervention to prevent long-term sequelae.

Clinical Significance: Infective endocarditis caused by Granulicatella is rare with 11 reported cases of mitral valve endocarditis in the literature. Our case illustrates success of combination antibiotic therapy in patients with Granulicatella endocarditis, even in presence of large sized vegetations.
Candidal endocarditis (CE) accounts for only 2% of endocarditis cases, however, the mortality associated with it is around 30-47%.

51-year-old female with history of intravenous heroin use was admitted after presenting with cough, hypoxia, hypotension and new systolic murmur. Nasopharyngeal swab was positive for influenza A virus and chest CT scan showed bilateral ground glass opacities concerning for acute respiratory distress syndrome. Admission blood cultures subsequently grew Candida albicans, raising concern for CE. On hospital day 3, the patient had worsening encephalopathy and head CT scan showed multiple regions of acute ischemic changes within the cerebral hemispheres, cerebellum, and posterior occipital lobes. However, two transesophageal echocardiogram (TEEs) performed on hospital days 1 and 5 did not show any valvular vegetations or patent foramen ovale. The patient was successfully treated with a six-week course of intravenous Amphotericin B and flucytosine, with improvement of her neurologic deficits.

Although no vegetations were seen on TEE, our patient met three minor Dukes criteria for endocarditis (fever >100.4 degrees Fahrenheit, major arterial emboli, and microbiological evidence with positive blood cultures). The majority of CE involves left-sided heart valves with major risk factors being intravenous drug use, prolonged antibiotic exposure, indwelling central venous catheters, underlying valvular disease, and human immunodeficiency virus infection.

In clinically suspected infective endocarditis, current guidelines recommend screening with echocardiography. However, guidelines are unclear for diagnosis of CE. As CE is rare with high mortality, a high index of suspicion is required to avoid delays in diagnosis and appropriate treatment.
Heart Failure (HF) has steadily increased in prevalence and given diuretics are mainstay of management, we should acknowledge that diuretic doses result in side effects and can result in readmissions.

65-year-old female with medical history of HF with preserved ejection fraction, presented with weakness and reduced responsiveness of 3 days duration in the setting multiple episodes of emesis. She was discharged one month prior after a prolonged hospitalization, involving a significant increase in diuretic dose. In the Emergency Department, she was alert, non verbal and communicative with only ocular and head movements. She had proximal quadriaparesis with minimal movement of fingers and toes. Labs were significant for potassium level of 2.0 mEq/L. With potassium supplementation, her condition dramatically improved. Within hours she was moving all her extremities spontaneously and able to communicate verbally.

Hypokalemic periodic paralysis (HypoKPP) is a channelopathy characterized by transient episodic weakness of proximal muscle groups with a preserved consciousness in the setting of hypokalemia. A wide range of precipitants have been implicated involving insulin or epinephrine surge, distal renal tubular acidosis, and medications. Patients on diuretic therapy are at an increased risk for dyselectrolytemia. Interestingly, our patient had no family or personal history of HypoKPP and the incidence of diuretic induced HypoKPP is unclear. The clinical significance is that our inpatient management of decompensated HF often involves aggressive diuresis to reach euvolemic state. However, it is important to prescribe a dose that will maintain euvoolemia upon discharge and prevent readmissions.
Rare Case of Staphylococcus Haemolyticus Prosthetic Valve Endocarditis Following Transcatheter Aortic Valve Replacement

Introduction: The rate of prosthetic valve endocarditis (PVE) following transcatheter aortic valve replacement (TAVR) is similar to that after surgical AVR. PVE post-TAVR is associated with high in-hospital mortality and is often treated with intravenous antibiotics as patients are poor surgical candidates. This case illustrates the identification and treatment of PVE post-TAVR secondary to an uncommon organism, Staphylococcus Haemolyticus.

Case: A 74 year-old male with severe aortic stenosis underwent successful TAVR. Three months following TAVR, he developed Streptococcus Bovis bacteremia without evidence of PVE. He was treated with a four-week course of intravenous antibiotics. Repeat blood cultures showed clearance of bacteremia, the source of which was not identified. Three months later, he presented with Staphylococcus Haemolyticus bacteremia with evidence of PVE on ECHO without destruction of the prosthetic valve. Following a multi-disciplinary team evaluation, a six-week course of vancomycin was planned following negative repeat blood culture. Additionally, the patient was diagnosed with large esophageal varices secondary to cirrhosis and a follow-up TEE was unobtainable.

Discussion: Literature review reveals Enterococci species and Staphylococcus Aureus as the most common microorganisms associated with PVE post-TAVR. PVE post-TAVR secondary to Staphylococcus Haemolyticus is rare with only one previous case report published in 2013, in which a patient was treated with IV antibiotics and a subsequent TEE at 8 months demonstrated complete resolution of vegetation.

Clinical Significance: By illustrating this case, we aim to support the healthcare provider facing similar cases of PVE post-TAVR with Staphylococcus Haemolyticus in making evidence-based clinical decisions.
Rectal Bleeding From a Source Outside the Colon with a Negative CT Scan

Introduction:
Synchronous primary endometrial and ovarian carcinomas account for 0.7-1.8% of all gynecologic cancers. Symptoms are abnormal uterine bleeding, pelvic masses and pain. Rarely rectal bleeding is the sole presentation. Women commonly are between the ages of 41-53 and have an increased BMI. CT is used for diagnosis and staging but morbid obesity can present a challenge in diagnoses.

Case:
A morbidly obese 64yo female was admitted to the hospital with DKA, Sepsis due to UTI, AMS and lower GI bleed. Initial labs showed creatinine 5.66mg/dL, glucose 521, albumin 2.5gm/dL, WBC 27.1 thou/mcL and Hb 6.8mg/dL. Colonoscopy was performed which was limited but showed a sigmoid ulcer, biopsy of which was negative. CT abdomen/pelvis only showed a ureteric stone. Ureteric stenting was completed with resolution of sepsis; but another episode of rectal bleed occurred. Repeat colonoscopy showed, sigmoid polypoid mass with ulceration. Surgery was consulted. On laparotomy, a large necrotic mass was seen attached to the colon, uterus and right ovary. An intraoperative consult for gynecology was placed; hysterectomy with BSO, sigmoid resection and debulking was performed. Pathology revealed two synchronous primary endometroid tumors, one originating from the right ovary and one from the uterus.

Conclusion:
Our case illustrates the rare presentation of primary ovarian and uterine tumor with the absence of any cardinal symptoms. Furthermore the case emphasizes the limitations of imaging modalities and a need for considering a detailed review when utilizing radiology in morbidly obese patients.
Bilateral Lower Extremity Emboli: A Rare Complication of Atrial Fibrillation

INTRODUCTION:
Atrial Fibrillation (AF) has been reported in almost half the patients with Mitral Stenosis (MS) and the major complication is Atrial thrombus formation resulting in distal emboli. Common site for clinically evident embolism is cerebral circulation but the annual incidence of acute limb ischemia is 0.4% (lethality 16%).

CASE:
A 62-year-old gentleman with history of MS and PAF presented with acute onset, progressively worsening bilateral foot pain. Oral Anticoagulation (OAC) had been discontinued 1 month prior by his cardiologist. His feet were cold, pale with absent arterial pulses. Arterial Doppler revealed occlusion of the bilateral tibio-peroneal trunk. The patient underwent emergent bilateral embolectomy with excellent arterial flow re-establishment. Echocardiography revealed severe mitral valve stenosis; a giant left atrium (10 X 7 cm); and a large left atrial thrombus. He was started on anticoagulation therapy and discharged home.

DISCUSSION:
Unilateral limb ischemia has been commonly reported but this is the first report of presentation with bilateral acute lower limb ischemia from cardiac source emboli. Giant left atria and AF both increase the risk of thrombosis and the presence of both can cause thromboembolism even if OAC is interrupted for a short duration.

CONCLUSION:
OAC should not be interrupted without bridging in patients with giant atria and AF. Should Patient be evaluated with Echocardiogram before discontinuation of therapy in Paroxysmal Atrial Fibrillation?
Large cardiac thrombi can present as multiple site emboli.
Recurrent Liposarcoma with Invasion of the Duodenum Causing Upper GI Bleed

INTRODUCTION

CASE
A 61 year old African American gentleman with history of End Stage Renal Disease on hemodialysis presented with Hemoglobin of 5.5 (baseline Hemoglobin 10.1). Patient also has history of Liposarcoma of right kidney treated with nephrectomy, recurred one year later and treated with localized radiation. Patient presented with 4 week history of weakness and melena. Upper GI endoscopy was done and a large ulcerated duodenal mass was found at the junction between first and second segments. Biopsies of the mass revealed "Proliferative of pleomorphic, hyperchromatic spindle and multinucleated cells in a myxoid background, compatible with : Sarcoma, favor Differentiated Liposarcoma.". Abdominal CT showed "11.7 x 11.4 x 10.cm hypoenhancing, multiseptated mass arising from the right retroperitoneum, invading the inferior vena cava and abutting the right hepatic lobe with possible invasion, and abutting the duodenum and right colon" confirming the diagnosis of Liposarcoma invading the duodenum causing Upper GI bleed. Patient was started on chemotherapy.

DISCUSSION
Differentiated low-grade liposarcomas have high local recurrence rates, the potential to metastasize and a high risk of death. In our patient, not only did the liposarcoma recur, but it also invaded the duodenum causing atypical presentation of Liposarcoma with GI bleed.

CONCLUSION
In the setting of a patient with history of liposarcoma presenting with GI bleed, the possibility of metastases and invasion of the tumor to the GI tract needs to be considered.
Whipped and Weak!: Literature Review of Nitrous Oxide Abuse

Introduction
Recreational nitrous oxide use is estimated at 9.3% among adolescents. It is procured in canisters originally intended for aerosolizing whipped cream.

Methods
Literature search and review via Pubmed was performed with the terms: "Nitrous oxide", "whippet", "whippit", "addiction" and "over use". Anesthetic, occupational or accidental exposure were excluded. 58 papers met these criteria. Quantitative analysis of symptomatology, laboratory findings and treatments was performed.

Results
There was male preponderance (70%) with 55% in 17 to 25 age range. 59% reported upper and 81% lower limb paresthesia. 17% reported lower limb weakness and 16% had quadriplegia. 57% reported ataxia. Other reported symptoms included psychosis (10%), bladder dysfunction (9%), memory loss (7%), sexual dysfunction (7%), skin pigmentation (2%) and bowel dysfunction (2%). Imaging demonstrated cervical spinal cord lesions on T2 weighted MRI in 27% of the cases, 3% with thoracic spinal cord lesions and 2% with both thoracic and cervical spinal cord lesions. 10% however had normal imaging. Management was with high dose intramuscular B12 followed by oral replacement.

Discussion
N2O abuse is underreported with highest prevalence in young adult males. N2O mediated functional deficiency of vitamin B12 with inactivation of methionine synthase causes central nervous demyelination. Patients present with clinical features resembling subacute combined spinal cord degeneration with functional vitamin B12 deficiency.

Conclusion
N2O abuse presents as subacute combined degeneration of spinal cord with low to normal serum Vitamin B12 levels. Suspected cases should be managed with vitamin B12 supplementation and abuse cessation counseling.
Radiocontrast Agent Causing Iododerma in a Patient with Peripheral Arterial Disease

A 45-year-old female with a significant past medical history of left sided ischemic stroke, congenital transposition of great vessels, and polysubstance abuse was admitted for weakness, numbness, and paresthesias in her right lower extremity. Upon admission, a lower extremity arterial duplex demonstrated severe right common and external iliac artery disease. On day four of admission, the patient underwent an abdominal aortogram with runoff that showed 100% occlusion of the right external, internal, and common femoral arteries. Approximately 24 hours after imaging, the patient developed mild acneiform, papulonodular skin eruptions beginning with the lips, nose and cheeks. These lesions would become progressively severe, hemorrhagic and encompass her neck, hands, wrists, and the entire face and scalp. Two separate skin biopsies from the patient’s arm and neck revealed localized subepidermal collections of neutrophils indicating a nonspecific type of neutrophilic dermatoses. 10 days after the aortogram, a urine iodine level indicated a severely elevated value of 91,975mcg/L (705 uln).

Halogenodermas can be caused by any intake of halogens paired with a delay in clearance. This skin condition tends to occur in areas with a higher density of sebaceous glands. Though the exact mechanism for this condition is not well understood, it is believed to be a cell-mediated hypersensitivity reaction. For patients with decreased renal function it is imperative to be aware of all potential side effects that exposure may cause. Prompt recognition of iododerma is imperative as it can lead to significant deformities and potentially death.
Transforming the Structure and Process of Peripherally Inserted Central Catheter (PICC) Utilization and Ordering Practices

Introduction:
Variability in peripherally-inserted central catheter (PICC) line appropriateness can decrease quality and safety. Our aim in this project was to assess PICC line utilization and ordering practices at St. Mary Mercy to meet the appropriate utilization goal per HMS guidelines.

Methods:
A total of 228 patients were studied in this initiative. Inclusion criteria consists of patients at St. Mary Mercy aged ≥18 years who were hospitalized between 8/1/16-12/31/16 with an active order for PICC line placement. Of the 228 patients, 126 underwent proactive chart review where ordering physicians were contacted for PICC line insertion orders that were for inappropriate reasons per HMS guidelines.

Results:
There was a decrease in inappropriate PICC EMR orders from 61% to 38% (p<0.0007). Proactive chart review follow-up with ordering practitioner occurred in 41% of cases. Of orders requiring practitioner follow-up, there was 21% order modification. We observed a decrease in patients with a PICC line indwelling duration of 5 days or less from 19% to 17% (p<0.0370). Furthermore, there was a decrease in patients with PICC lines with GFRs<45 from 27% to 24% (p<0.53).

Conclusions:
Proactive chart review with follow-up with ordering practitioner significantly improved PICC line ordering practices. Specifically, there was a significant decrease in unacceptable ordering reasons for PICC line as well as in number of patients who had a PICC line indwelling duration of 5 days or less. It is therefore important to educate ordering practitioners about appropriate HMS guidelines for PICC line use and to seek alternative options for patients with poor venous access.
Case of Sudden Severe Toxicity From Methotrexate After Use for 22 Years

Methotrexate (MTX) has been used for years for management of Rheumatoid arthritis (RA). Literature suggests a favorable long-term safety but at the same time toxicity emerges as one of the main limiting factors in continuous use. The purpose of this case study is to increase awareness about MTX toxicity in long term users with a stable disease course.

74 years old lady with 22 years of methotrexate use for RA presented with mucositis, partial thickness skin wounds and diarrhea. Blood analysis showed acute renal failure, leukopenia and thrombocytopenia. Patient had failed outpatient fungal and antiviral treatment. Patient had been taking MTX 90 mg every week for 22 years without folic acid or leucovorin. We hypothesized methotrexate toxicity resulting in acute renal failure which was exacerbated by hypovolemia from poor oral intake. After 24 hours of withholding methotrexate, starting intravenous Diflucan and folic acid, skin wounds and mucositis started improving. Leukopenia resolved. Bicytopenia was likely due to drug toxicity. On discharge, MTX was discontinued and folic acid was prescribed. Platelet count was normal 2 weeks after discharge. MTX toxicity is commonly seen in the initial years of treatment and is usually not high on differential diagnosis in a patient who had been maintained on the same dose for 22 years.

This case highlights the importance of considering drug toxicity regardless of the duration of use and prior stable course. In this case, prompt recovery after discontinuation of MTX confirmed our hypothesis.
Virchow's Node - A Trojan Horse

Introduction:
Metastasis to the Virchow's node is usually associated with Gastric malignancies. Only a few cases of prostate cancer metastasized to Virchow’s node are reported. Prostate cancer frequently metastasizes to regional lymph nodes and bones. We present a rare case of prostate cancer with an initial presentation of left supra-clavicular lymphadenopathy.

Case Description:
A 59-year-old Caucasian male presented with a neck lump, hematemesis, abdominal pain, and weight loss of 20 pounds. Physical exam was remarkable for palpable left supra-clavicular lymphadenopathy. Labs revealed creatinine of 15.6. Endoscopy showed bleeding gastric ulcer. Biopsy was unremarkable. CT Abdomen/Pelvis showed multiple hepatic lesions, retroperitoneal lymphadenopathy, obstructive uropathy, enlarged lobular prostate with invasion of bladder, and sclerotic bony lesions. PSA was 128. Biopsy showed prostate adenocarcinoma, Gleason score of 4+5. PSA stain was positive in left supra-clavicular nodes. Patient underwent TURP and was started on Biclutamide and Goserelin. PSA trended down and metastatic lesions decreased in size. Patient received multiple sequential treatments and is asymptomatic and doing well after 48 months of therapy.

Discussion:
This was a unique case of Virchow's node as an initial presentation of prostate cancer which was at first spuriously suggestive of gastric malignancy. Further investigations led to a diagnosis of prostate cancer. Carcinoma of the prostate can metastasize to left supra-clavicular lymph nodes, however, this has been reported in ≤0.5% of cases. Presence of Virchow's node is traditionally associated with very poor prognosis, yet, our patient continues to survive at 48 months.
Unsuspected Malignancy Presenting as Febrile Lactic Acidosis

Introduction:
Diffuse large B-cell lymphoma usually presents with rapidly enlarging mass or nodal enlargement or B-Symptoms. Here we discuss an unusual presentation of DLBCL with febrile lactic acidosis.

Case:
82-year-old female, independent and functional on baseline, presented with acute onset of nausea, poor oral intake, generalized weakness, weight loss. Physical examination was unremarkable. SIRS 3/4, Initial labs were only significant for leukocytosis, normocytic anemia and lactic acidosis. Empiric antibiotics were started. Pan Cultures, Initial CT-ANGIO, endoscopy, colonoscopy were negative for any acute process. Lactic acidosis persisted despite all management. Later she started spiking intermittent fevers with persistent lactic acidosis and an acute drop in hemoglobin with no visible blood loss. ID, Hem-Onc, GI, Rheumatology, Critical-Care were on board. Physical examination, Blood cultures, Protein electrophoresis, hematological studies, rheumatological studies, CT chest abdomen pelvis, mammogram were normal. Lactic acidosis, anemia and fevers remained unexplained. There was a mutual consensus to go for Bone marrow biopsy, Bone marrow biopsy revealed an unexpected diffuse large B-cell lymphoma.

Discussion:
Diffuse large B-cell lymphoma usually presents with mass typically anywhere in the body, this case illustrates it can also present with Febrile lactic acidosis. In my literature search, only three cases were reported where B-cell lymphoma presented with lactic acidosis along with other symptoms, but none of those presented as isolated lactic acidosis. It is important for physicians to be aware of this condition so a rapid diagnosis can be made and therapy can be started immediately.
MBP-1 Granule Protein Deposition May Explain Esophageal Symptoms in Patients with Low Eosinophil Counts

Background: Part of the confusion surrounding the management of eosinophilic esophagitis (EoE) lies in the fact that the diagnosis and management is centered around peak eosinophil counts. However, eosinophil granule proteins, including major basic protein 1 (MBP1) likely contribute to disease pathology. We sought to determine whether recurrent or refractory esophageal symptoms in patients with low eosinophil counts (<15 eosinophils/HPF) could be explained by presence of mucosal granule proteins.

Methods: All patients with esophageal biopsy specimens stained for MBP1 at the University of Utah were collected. A total of 34 patients, who met criterion for EoE or PPI-REE were evaluated. Chart review was performed to identify clinical symptoms, age, gender, peak eosinophil counts, and their esophageal biopsy staining results for MBP1.

Results: In the EoE group, the average eosinophil counts did not vary between the symptomatic cohort and the resolved cohort (23.9 ± 25.5 versus 16.2 ± 30.0 respectively) p= 0.131. Median MBP was greater in the symptomatic group than in the asymptomatic one 3 (2-3) versus 0.5 (0-1) (p= 1.3e-06). This was also seen in the EoE with low eos group; the average eosinophil counts did not vary between the symptomatic and asymptomatic cohorts (symptomatic 4.2 ± 5.5 peak eos/HPF versus asymptomatic 2.2 ± 4.0 peak eos/HPF, p= 0.412). Median MBP was also greater in the symptomatic group than in the resolved group 2.5 (IQR 1.25-3) compared to 1 (IQR 0-1), p= 0.002.

Conclusions: MBP1 differentiated esophageal symptoms in those with EoE and those without EoE.
Non-infectious Purulent Pericardial Effusion in a Rheumatoid Arthritis Patient: A Case Report

Pericarditis is inflammation of the fibrous sac surrounding the heart (pericardium) and purulent pericarditis is a rare complication characterized by gross pus in the pericardium or microscopic purulence (>20 leukocytes per oil immersion field). A 73-year-old woman presented with left sided back pain and an extensive medical history including peripheral arterial disease and rheumatoid arthritis (RA) with lung involvement (solitary pulmonary nodules). She required chronic prednisone and methotrexate therapy for her RA although methotrexate had been recently discontinued due to a non-healing pyoderma gangrenosum wound requiring skin grafting. A CTA revealed a large, 3 cm, non-enhancing pericardial effusion compressing the right heart. A TTE showed respirophasic flow variation across the mitral valve indicating tamponade physiology. Pericardiocentesis revealed 480 cc of yellow-green purulent fluid (88 leukocytes/oil immersion field, WBC 55,000/uL, total protein>3 g/dL, and glucose<10 mg/dL). Interestingly, all microbiological studies including repeat blood cultures revealed no infectious etiology for the purulent effusion. She was placed on long term vancomycin/ceftazidime and methotrexate therapy was resumed. No further complications were subsequently reported. We describe a rare case of culture negative purulent pericarditis, complicated by cardiac tamponade requiring pericardiocentesis, in an immunocompromised patient with RA. Pericarditis is a known complication of RA although no data to our knowledge exists on purulent pericarditis. This report highlights a lesser known complication of RA and the need for additional clinical data on this potentially lethal condition.
68 year old female, smoker, presented with gradual onset weakness and falls. She also reported increased shortness of breath. Past Medical History was significant for primary biliary cirrhosis. Vital signs were unremarkable except for new onset hypoxia. Physical examination showed bibasilar crackles and diffuse neuromuscular weakness. CT scan chest showed prominent areas of parenchymal change within the region of the left upper lobe and lingula. Severe conglomerate areas of fibrotic change or infiltrates were noted in the lower lobes. Prominent mediastinal and hilar lymphadenopathy was noted. Serology worked up showed positive ANA and positive SSA/B. Paraneoplastic workup was unremarkable.

Primary Biliary Cirrhosis is frequently associated with underlying auto-immune diseases. The most common auto-immune disease associated with PBC was Sjögren’s syndrome (SS) (36.2%), followed by systemic lupus erythematosus (3.7%), polymyositis (3.1%), progressive systemic sclerosis (2.8%) and rheumatoid arthritis (2.8%). Conversely, clinically overt PBC is found in less than 2 percent of SS patients in large cohorts. PBC has been reported as associated with various ILDs, such as pulmonary fibrosis, lymphoid interstitial pneumonia (LIP), non-specific interstitial pneumonia (NSIP) and bronchiolitis obliterans with organizing pneumonia (BOOP). Concomitant SS increases the risk of ILD in patients with PBC. In a study composed of 109 consecutive PBC cases, concomitant SS was seen in 46 patients (42.2%). While the frequency of ILD was 21.7% in PBC patients with SS, it was only 1.6% in those without SS.
Implications of Donor Withdrawal from a Bone Marrow Transplant After the Recipient Has Undergone Conditioning

BACKGROUND
Allogenic stem cell transplantation (ASCT) is the treatment of choice for a variety of high risk leukemias. HLA matched full siblings or unrelated haploid donors prove to be the best match but when they are unavailable stem cells from unrelated donors or umbilical cord blood may be used as an alternative.

CASE:
A 48 year old African American woman with chronic hepatitis C and Philadelphia chromosome positive ALL underwent myeloablative conditioning with full body radiation after a potential donor was identified. Unfortunately, her donor withdrew after she started her conditioning and another donor was not located in time. Transplant team urgently searched cord blood registries and she underwent double cord blood stem cell transplant. Her post-transplant course was complicated by multiple infections, liver and respiratory failure, ultimately resulting in her death.

DISCUSSION
There are over 18million volunteer donors in registries worldwide however ethnic minorities are poorly represented. African Americans have only 76% likelihood of finding a match compared to 97% for Caucasians. The donor volunteers are very committed and we were unable to find a previous documentation in literature of donor withdrawing from stem cell collection after patient had started conditioning regimen. Donors signs a consent acknowledging that the recipient will most likely expire if they refuse to donate under above mentioned circumstances. In addition, to cord blood haploidentical donors can be considered during these emergent circumstances.
Hemophagocytic lymphohistiocytosis (HLH) is an inappropriate hyperactive immune response primarily seen in children with genetic disorders, but presents infrequently in adults as a syndrome secondary to malignancy, autoimmune disease, or infection. HLH is diagnosed when 5 of 8 criteria are met, including fever, cytopenia, hypertriglyceridemia or hypofibrogenemia, decreased NK cell activity, splenomegaly, elevated ferritin, elevated IL-2, and/or evidence of hemophagocytosis on pathology sample. Cases of HLH have been rarely associated with systemic Histoplasmosis infection, particularly in immunocompromised populations.

A 56-year-old man with rheumatoid arthritis, managed with weekly methotrexate and recent course of Humira, presented with shortness of breath, fever, chills, and hypotension. He received vancomycin and zosyn, fluid resuscitation, and briefly required norepinephrine. Chest x-ray, echocardiogram, and blood cultures were unremarkable. Significantly, the patient had recently been admitted for presumed COPD exacerbation. On admission, antibiotics were switched to levaquin and methotrexate was held. Shortly into the hospitalization, the patient developed dyspnea, intermittent fevers to 39.9°C, and worsening pancytopenia with thrombocytopenia and leukopenia. Imaging demonstrated new pulmonary congestion and splenomegaly. Further evaluation revealed elevated ferritin, low fibrinogen, and bone marrow biopsy with hemophagocytic lymphohistiocytosis and fungal forms, suggestive of histoplasmosis with secondary HLH. Confirmatory testing was performed and the patient was started on rituximab, dexamethasone, and amphotericin B, with frequent electrolyte monitoring.

Guidance regarding appropriate management of histoplasmosis-associated HLH has been limited due to the few case reports available within the literature, likely through a combination of rare occurrence and under diagnosis.
Endobronchial Amyloidosis Masquerading as Asthma

Introduction: Endobronchial amyloidosis is a rare disease which can mimic the symptoms of asthma and recurrent pneumonia. Severe airway obstruction can occur, requiring bronchoscopic intervention. Presently, no definitive treatment is available.

Case: A 59-year-old asthmatic man was admitted for an elective diagnostic and therapeutic bronchoscopy. Six months prior to presentation, the patient had been treated for multiple asthma exacerbations and pneumonia episodes, requiring multiple courses of antibiotics and prednisone without improvement. A contrast CT of the thorax showed patchy right hilar and perihilar consolidation with bronchial thickening and narrowing, mass-like along the posterior aspect of the major fissure and hilar region. A diagnostic bronchoscopy showed small mucosal nodules throughout his entire airway with near total occlusion of right upper, middle and lower lobe segmental bronchi. Pathology showed “amyloidosis with osteochondroid metaplasia”. At his subsequent outpatient pulmonology appointment, he complained of worsening cough, dyspnea and easy fatigability. An elective therapeutic bronchoscopy was therefore done which involved tumor debulking, debridement and ablation with Nd:YAG laser and argon, followed by cryotherapy, balloon dilation and deployment of three MeritAERO stents.

Discussion: Endobronchial amyloidosis is a rare localized pulmonary condition often diagnosed in the 5th-6th decade of life, presenting as bronchial asthma and recurrent post-obstructive pneumonia. Bronchoscopic intervention in cases of significant airway compromise offers symptomatic improvement and involves mechanical and laser debulking, balloon dilation and bronchial stenting. Chemotherapy is associated with significant side effects, however external beam radiation therapy has shown more promising results.
Small Bowel Obstruction Mimicking Acute ST-Elevation

Introduction: The mainstay of diagnosing Myocardial Infarction (MI) is the EKG finding of ST elevation and requires urgent intervention. However, the etiology of ST elevation can be non-cardiac in nature. Herein, a case of small bowel obstruction that presented as ST segment elevation

Case: A 55-year-old male presented with abdominal pain, nausea and vomiting for 5 days after hemiarthroplasty of left humerus. Patient denied chest pain or shortness of breath. On admission, patient was vitally stable, except for tachycardia of 110 bpm. On, examination, abdomen was tender to deep palpation and moderately distended. Blood tests showed leucocytes and normal troponin level. Initial EKG showed significant ST elevation in inferior leads II-III and aVF, leading to emergent cardiac catheterization which revealed patent coronary arteries with normal LV function. Abdominal pain and vomiting persisted. CT of the abdomen revealed small bowel obstruction. After the patient’s bowel obstruction resolved, EKG findings returned to baseline.

Discussion: Due to the nature of time dependent injury in MI, the finding of ST elevation on EKG prompts physicians to urgently intervene by emergency cardiac catheterization, even when clinical scenario and blood word direct otherwise. The postulated mechanism of these EKG changes is attributed to pain or mechanical dislocation of the heart in lieu of normal position. Our abstract highlights a case of interest to health care workers, who are first responders, where EKG findings should be correlated with other clinical findings.
Reducing Unnecessary Testing: Ultrasound in Acute Kidney Injury

Introduction: Acute Kidney Injury (AKI) is one of the most common diagnoses resulting in admission to the hospital. Ultrasound (US) abdomen is a commonly used tool to exclude obstructive uropathy in patients with AKI. An ultrasound abdomen can cost $200 to $1000. Studies have shown obstructive uropathy is only found in 2.3% of patients with AKI. Therefore US should not be done routinely in all patients with AKI, unless there is evidence on history/physical of renal tract obstruction.

Methods: We examined medical records from patients admitted to Crittenton Hospital with AKI between September 2014 and August 2015. We only included patients who were admitted with diagnosis of AKI (ICD-9 code 584.9). We reviewed the percentage of patients who had US. We then implemented resident physician education in order to attempt to decrease this percentage. We repeated analysis following physician education.

Results: Our initial analysis of 100 patients admitted to ER with AKI, showed 22% had an US. Following resident physician education repeat analysis showed 18% of patients admitted via the ER had US for AKI. Resident physician ordering of unnecessary tests decreased to zero following intervention.

Discussion: This decrease in resident physician ordering will lead to annual savings of $26,000 if this behavior is maintained (based on 1300 yearly admissions with AKI). We are currently in the process of implementing an EMR prompt for US which will act as an intervention that will affect both resident physicians and non-resident physicians. Successful implementation could increase annual savings to $52,000.
Preventing Hypoglycemia and Hyperglycemia During Hospitalization by Reducing the Use of Oral Hypoglycemic Drug

Rationale/Aim: Hypoglycemia is a potentially fatal condition that is frequently encountered during hospitalizations. Guidelines have been established for management of hyperglycemia in the hospital with the use of insulin. The aim of our study is to discontinue oral hypoglycemic use in the hospital setting.

Method: Institute Healthcare Improvement.

Plan: During a one-month period in 2016, we reviewed the charts of patients who received oral hypoglycemic agents. Only sixty percent of patients who use oral hypoglycemic agents at home were switched to insulin therapy on admission. Using a fishbone diagram as part of brainstorming, we identified areas of improvement including physician and pharmacy education.

Do: Our team created a presentation that outlined the current society recommendations regarding management of hyperglycemia in the hospital with the use of insulin. Physicians, including medicine faculty and residents, were educated during noon conference. Pharmacy managers and committee was also educated during a PNT meeting.

Study: In 2017, following education and pharmacy policy initiation, primary outcomes included overall reduction of prescribing and dispensing of oral hypoglycemic medications during hospitalizations.

Act: Although, our pilot study revealed desirable outcomes, further PDSA cycles are needed to look at the sustainability of our project. We plan to expand our study and educate private physicians in addition to nursing staff.

Results: During a one-month period in 2016, 20 patients had oral hypoglycemic agents ordered while in the hospital. In 2017, following education and policy implementation, there is a 70% overall reduction in prescribing oral hypoglycemic agents.
Rare Case of Anomalous Left Main Coronary Artery Originating From Right Coronary Cusp

Introduction: Anomalous of the coronary arteries is a rare finding. The overall reported Incidence of anomalous coronary arteries during angiography is 5.6% and for Left main coronary artery from right coronary cusp is about 0.19%. Sometimes these anomalies have malignant course which can cause arrhythmia and sudden cardiac death.

Case presentation:
We present a case of 75 years old woman with a history of hypertension, peripheral arterial disease and hyperlipidemia presented with near syncope 3 to 4 times a month prior to admission. Upon admission patient denied having any chest pain, dyspnea or palpitations. EKG revealed new T wave inversions on the inferior and antero-lateral leads. Troponins were negative and echocardiogram revealed normal ejection fraction with no wall motion abnormalities. Decision was made to do left heart catheterization. Patient was found to have anomalous of the left main coronary artery arising from the right coronary artery. This was confirmed by CT-angiography with benign course. Patient was discharged without any intervention and didn’t experience any complications.

Discussion: We present this case to illustrate the importance of anomalous coronary arteries which can be associated with malignant course. In certain patients, anomalous coronaries have malignant course running between pulmonary artery and aorta which can be affect leading to life threatening arrhythmias and sudden cardiac death. Patient must be further evaluated through CT-angiography or other imaging modalities to identify the proper course of the coronary arteries.
Pericardial Effusion in a Patient with MELAS Syndrome

Pericardial effusions are not commonly associated with mitochondrial encephalopathy, lactic acidosis, and stroke like episodes (MELAS) syndrome; however, in the literature there have been handful of cases in which patients presented with moderate to large effusions. We present a 19 year old women with a history of MELAS syndrome who initially came to our institution in status epilepticus. Per Genetics recommendations, the patient underwent an initial echo early in the admission that showed a small pericardial effusion. Patient was later noted to develop pneumonia, and R foot cellulitis. Despite treatment with broad spectrum antibiotics, patient continued to have fevers and persistent tachycardia and subsequently repeat transthoracic echocardiogram (TTE) was ordered to evaluate viral myocarditis. TTE showed moderate pericardial effusion, increased in size versus previous echocardiogram. Effusion was thought to potentially be secondary to inflammation and patient received intravenous methylprednisolone for 5 days with no significant improvement in pericardial effusion. Rheumatological work up, including ANA and anti DS DNA were negative. A decision was made to clinically monitor effusion with weekly repeat echocardiograms. During her stay, patient was further received treatment for streptococcal bacteremia. On day of discharge, repeat echocardiogram was showed trace pericardial effusion, significantly improved from the previous echocardiogram. Pericardial effusions are rarely reported in patients with MELAS syndrome. In our case, our patient's effusion resolved without any intervention, suggesting that patients with MELAS that develop pericardial effusions do not require drainage as long as they remain hemodynamically stable.
This is a 37 years old white female with a PMH of asthma, scapular dyskinesia, chronic myofascial cervical and thoracic pain was admitted for shortness of breath. She received acupuncture the day prior her admission over her cervical, upper thoracic spines and periscapular border. She started feeling short of breath and pain over the left lower shoulder blade. At the hospital she denied any fever, cough, orthopnea, PND, leg swelling or prolonged immobilization. Her vitals were B.P: 142/104, heart rate: 164, respiratory rate: 30 and she was afebrile. On physical examination, decreased breath sounds and hyperresonance on percussion was noted over left lung apex. Rest of physical examination was unremarkable. EKG showed sinus tachycardia. Chest Xray showed left apical pneumothorax. She was admitted for observation but developed radiologic worsening of the pneumothorax, therefore a pigtail catheter was placed. Patient was kept under observation for 36 hours. Patient’s symptoms improved and repeat Xray showed resolution of pneumothorax. Her pigtail catheter was subsequently removed. Pneumothorax remains a rare adverse event of acupuncture. Only two patients out of 229,230 patients experienced a pneumothorax in one prospective study. Given its increasing popularity among general population, it is very important to keep in mind associated adverse effects especially fatal adverse events like pneumothorax and cardiac tamponade.
Salmonella Enteritis Prosthetic Joint Infection in an Immunocompetent Host

Salmonella enteritidis is a bacterial pathogen known to commonly cause gastroenteritis. Prosthetic joint infection is a rare manifestation of salmonella enteritidis infection that has been described in limited case reports and occurs primarily in immunocompromised patients. This case report describes an immunocompetent patient who presented with salmonella enteritis infection of her right prosthetic joint.

The patient is a 48-year-old female with a past medical history of right total hip arthroplasty due to severe osteoarthritis four years prior who presented with acute right hip pain and low-grade fevers. She denied any history of immunodeficiency, malignancies, or recent steroid use. Upon admission, she was afebrile and hemodynamically stable. Initial c-reactive protein was 179 and erythrocyte sedimentation rate was 36. Blood cultures were drawn which were positive for salmonella enteritidis sensitive to ceftriaxone, levofloxacin, and trimethoprim-sulfamethoxazole. The right hip was aspirated and cultures confirmed salmonella enteritidis prosthetic joint infection. She underwent irrigation and debridement with exchange of the liner and head of the right hip arthroplasty. A PICC line was inserted and the patient was discharged with intravenous ceftriaxone to complete a 6-week course followed by oral antibiotic suppression.

Prosthetic joint infections with salmonella enteritidis are rare, especially in immunocompetent hosts. There are no guidelines as to whether it is necessary to remove the prosthesis and the duration of oral suppression antibiotic therapy if hardware is retained. More data about long-term clinical outcomes is required. Nonetheless, case reports have shown successful treatment of the organism with four to six weeks of antimicrobial therapy.
Evaluation of the Impact of a Pharmacist-Driven Termination Protocol to Limit Inappropriate Use of Acid Suppressive Medications

Purpose
Overutilization of proton pump inhibitors (PPIs) and histamine-2 receptor antagonists (H2 blockers) related to stress ulcer prophylaxis (SUP) commonly occurs. Adverse events from SUP create an economic burden to the healthcare system. Our study aims to evaluate the effectiveness of a pharmacist-driven termination protocol to limit inappropriate use of acid suppressive medications in the non-ICU setting.

Methods
Patients were included if they met the following criteria: 18 years of age or older, prescribed a PPI or H2 blocker, and were admitted to the hospitalist service. Patients were excluded if the PPI or H2 blocker was a home medication. If medication use was deemed inappropriate, a pharmacist contacted the health care provider to discontinue the medication. The primary outcome of this study was the proportion of patients that had acid suppressive medication discontinued before and after implementation of the pharmacist-driven termination protocol.

Results
Pre-intervention, an acid suppression medication was inappropriately prescribed in 26 of 131 patients. Post-intervention, inappropriate acid suppression was prescribed in nine of 132 patients, eight of which were discontinued based on the pharmacist-driven termination protocol (p<0.0001).

Conclusions
Acid suppressive medications are over-prescribed in the hospital setting. This may lead to an increased number of adverse events. The implementation of a pharmacist-driven termination protocol may help decrease the inappropriate use of acid suppressive medications on an inpatient hospital service.
Acute liver failure results in hemostatic imbalance, due to variable half-lives of procoagulant and anticoagulant factors, manifesting as either hypercoagulability or bleeding diathesis. A 27-year-old man, with no significant past medical illness, presented with sudden onset of right lower extremity weakness. He reported consuming six beers daily for four years, and a half gallon of whiskey daily for the past six months. On physical examination, he demonstrated signs of liver failure, such as, icterus, spider angiomata, ascites, asterixis, and mild hepatosplenomegaly. Neurological examination was significant for right lower extremity strength of 1/5, absent deep tendon reflexes and positive Babinski sign. Initial laboratory studies revealed, neutrophilic leukocytosis 20K/uL, platelet count 78K/uL, hemoglobin 12.2 g/dl, creatinine 1 mg/dl, albumin 1.9 g/dl, ALP 376 IU/L, AST 101 IU/L, ALT 18 IU/L, total bilirubin 26.4 mg/dl, prothrombin time(PT) 82.2 seconds, International normalized ratio (INR) 7.3, (aPTT) 98 seconds, and fibrinogen level 492 mg/dl. Testing for non-alcoholic etiologies of liver failure was negative. MRI brain revealed acute ischemic stroke involving the left motor strip. Trans-esophageal echocardiogram was negative for intracardiac thrombus and patent foramen ovale. Aspirin was deferred in the setting of coagulopathy and he was supportively managed for alcoholic hepatitis. Patient expired in two weeks, due to worsening liver failure. Routinely used coagulation assays such as PT/INR and aPTT do not accurately reflect the hemostatic imbalance in liver failure. This case demonstrates that thrombotic events can still occur in acute liver failure, even though bleeding diathesis is more common.
Atypical Hemolytic Uremic Syndrome: A Typical Presentation of a Rare, Devastating Disease

Introduction: Atypical HUS is a rare, life-threatening systemic complement-activating condition characterized by microangiopathic hemolytic anemia, thrombocytopenia, and marked acute renal failure. It is associated with high morbidity and mortality if not recognized and treated appropriately. Case: Patient is a 62-year-old female presenting with acute encephalopathy, her family notes she has become increasingly weak with poor oral intake over the last two weeks. Recent medical history includes the patient undergoing a craniotomy two months ago for removal of a meningioma and since has been on Dilantin. On presentation, labs were significant for a creatinine of 26, BUN 251, hemoglobin 4, platelets 38, LDH 1517, haptoglobin <10. Peripheral smear significant for schistocytes. Emergent dialysis and plasmapheresis were pursued, the patient received supportive blood transfusions and corticosteroids were started with concern for TTP versus HUS. The patient did not have any prior diarrheal or upper respiratory illness and infectious work up was negative. ADAMST13 level was normal at 57%, favoring the diagnosis of atypical HUS. Eculizumab was initiated and the patient improved, and six months later she remains on therapy and is not dialysis dependent. Discussion: Atypical HUS must be distinguished from other thrombotic microangiopathies and complement activating conditions, such as TTP, so as to not delay proper treatment. ADAMSTS13 activity is essential in making this distinction. Supportive care along with Eculizumab, a monoclonal antibody to C5, is first line treatment for atypical HUS. The optimal treatment duration is unknown, but some patients require life-long therapy.
A Rare Case of Zinc Phosphide Intoxication Leading to Severe Refractory Cardiogenic Shock

Introduction: Zinc phosphide is a potent rodenticide. In both humans and rodents, gastric acid reacts with phosphide to generate toxic phosphine gas which impairs oxidative phosphorylation in the mitochondria leading to cardiogenic shock.

Case description: A 22 year-old healthy female presents to the ED two hours after ingestion of one tablet of Zinc phosphide in a suicide attempt. In the ED, patient complained of nausea, non-bloody emesis and severe abdominal pain. Patient was afebrile, hemodynamically stable with good O2 saturation on room air. Physical exam was unremarkable. Activated charcoal was administered and potassium permanganate gastric wash was done. ABGs on admission showed pH 7.32, HCO3 18, PCO2 30 and PO2 93. Echocardiography was unremarkable with EF of 50%. Patient was transferred immediately to the ICU. Sodium bicarbonate IV infusion was started. Two hours later, patient was in severe respiratory distress. ABGs showed pH 7.1, HCO3 8, PCO2 40, PO2 60. Patient was intubated and mechanically ventilated. Repeat echocardiography showed EF of 15%. Patient was started on norepinephrine and dobutamine infusions because of severe cardiogenic shock. Fifteen minutes later, ABGs showed pH 6.9 and HCO3 3. Twenty minutes later, Patient had a cardiac arrest and expired despite appropriate resuscitative efforts.

Discussion: Circulatory failure is a common and frequent cause of death after ingestion of metal phosphides. Ejection fraction is reduced acutely by a mean of 36%. Metabolic acidosis or mixed metabolic and respiratory acidosis is very common. No intervention has shown to be effective in preventing death.
Wait and Watch: A Case of Regressing Iatrogenic Solid Tumors Following Medication Cessation

Introduction:
Although used to great impact against autoimmune disease, traditional systemic immunosuppressive therapy and emerging biologic agents targeting tumor necrosis factor-α are implicated in potentiating the risk for malignancy. We present a case of an iatrogenic immunodeficiency-associated lymphoproliferative disorder (IIA-LPD) in a patient on immunosuppressive therapy for psoriatic arthritis.

Case:
A 31-year-old man with a past history of psoriatic arthritis presented to the hospital with a cough and dyspnea for four days, with prior episodic fevers and malaise for eight months. The patient on ongoing methotrexate and etanercept treatment for the past three years. Physical examination revealed a respiratory rate of 25 with scattered end-expiratory wheeze, and splenomegaly. Initial labwork revealed platelets of 98K, but were otherwise unremarkable. Computed tomography of the chest revealed prominent cervical and thoracic lymphadenopathy, with subsequent imaging revealing diffuse intraabdominal lymphadenopathy.

A comprehensive workup for systemic viral, bacterial, and fungal etiologies was negative. The patient had a mediastinoscopy with excisional biopsy of a paratracheal lymph node. Pathology confirmed EBV-positive, polymorphic B-cell lymphoproliferative disorder associated with iatrogenic immunodeficiency. The patient’s etanercept and methotrexate were discontinued, without initiation of chemotherapy. After eight weeks, a PET scan soon after revealed significant improvement in his diffuse lymphadenopathy.

Discussion:
Our case highlights the importance of providing adequate time following cessation of immunosuppressive agents in order to promote tumor regression. As noted by Inui et al., by withdrawing these immunosuppressive agents for at least eight weeks, an appropriate immunologic response can be mounted against the tumor cells.