Illinois Chapter
Northern Region

Top 100 Posters

University of Illinois at Chicago
November 8, 2017
Abstract:
There is recent emerging trend to use over the counter Loperamide at higher doses for euphoric effects.
There is a growing demand amongst educated people and loperamide appears to fit that role.
At higher doses loperamide crosses the blood brain barrier and produces euphoric effects.
Euphoric effect starts around 300-400 mg per day as compared to recommended dose of 5-10 mg per.
As per poison control data there is recent surge in the number of cases in Illinois.
A 32-year old male patient with history of Substance use, scoliosis and back surgery presented to ED via ambulance after a witnessed seizure like activity and acute confusion. The Patient has been taking 100 to 200 capsules of loperamide Every other day. At Presentation, He appeared distressed but was oriented to person place and time. He reported no Bowel movement for three days, palpitations, fatigue and chills. Examination of Vitals, chest scan, cardiovascular examination, and abdominal palpitations were without incidence. He was noted to have prolonged QT of 634, several episodes of Vtach and Afib on EKG and Telemonitor. He received magnesium, lidocaine infusion, isoproterenol, and Bicarb. Cardioversion was done by Electrophisiology with Brief conversion to sinus rhythm. The Patient was taken to cath lab for placement of a temporary pace maker. A 2D Echo was done which showed Normal left ventricular systolic and diastolic function with dyskinetic inter ventricular septal motion due to pacemaker or LBBB. CT and MRI of the brain were unremarkable.

Patients often combine other medications in an attempt to increase loperamide absorption and penetration across the blood-brain barrier, inhibit its metabolism, and enhance its euphoric effects. If toxicity is suspected, the drug should be promptly discontinued and necessary therapy begun; if loperamide ingestion is suspected, blood levels should be measured, which may require specific testing. For cases of torsades de pointes in which drug treatment is ineffective, electrical pacing or cardioversion may be required.

Abstract:
Introduction:
Kounis syndrome (KS) has been described as an allergic insult leading to hypersensitivity reaction culminating in the occurrence of an acute coronary event. This recently identified entity has primarily been described as a dynamic vasospastic event that is mediated by mast cell degranulation. Below we present a case where aspirin allergy led to an allergic myocardial event.

Case Description:
A 63-year-old man presented with chest pain (CP) and urticaria. History was significant for, asthma, nasal polyps, sinusitis and multiple episodes of CP with a negative angiographic workup. Multiple drug allergies, which resulted in urticaria and dyspnea. Past episodes of CP and urticaria were relieved by nitroglycerin (NTG). Cardiac catheterization (CC) showed patent coronaries, but was conspicuous for coronary artery spasm (CAS), that was relieved by intracoronary NTG (IC-NTG). Patient was placed on aspirin (ASA), calcium channel blocker (CCB), statin, nitrate and ranolazine. A few weeks’ later patient presents with repeat onset of CP, dyspnea and urticaria. He is treated for a non-ST segment elevation myocardial infarction (NSTEMI) and undergoes CC that shows subtotal occlusion of a coronary. Failed trial of IC-NTG was followed by stent deployment resulting in resolution of occlusion. Patient was restarted on ASA and clopidogrel. Following day, patient again complains of severe CP, ECG shows ST segment elevation myocardial infarction (STEMI) accompanied by troponemia. Patient undergoes a repeat CC that shows patent coronaries and stent. Furthermore, there is spontaneous resolution of ECG changes. At this juncture, an immune mediated etiology to symptoms is suspected. ASA is stopped and a different platelet inhibitor is started. Patient is evaluated by an immunologist and is diagnosed with Samter-Beer syndrome characterized by asthma, nasal polyposis and ASA allergy. Patient is started on a histamine antagonist and ASA desensitization. In the following months’ patient is admitted twice with symptoms of CP and urticaria, ECG changes consistent with STEMI and troponemia. CC in both episodes shows CAS that is treated with IC-NTG. Similar to all prior instances, CC showed angiographically patent stent and preserved cardiac function. All episodes of CP were accompanied by urticaria mostly confined to the lower extremities. At this juncture the patient is diagnosed with KS. Patient was started on intense antihistamine therapy, coronary antispasmodics and discharged home with complete resolution of symptoms.

Discussion:
Based on the presence or absence of preexisting coronary disease, KS is classified into different variants. Precipitants include foods, insect stings, and various drugs. Treatment consists of revascularization, control of anaphylaxis and supportive therapy. This entails the use of antihistamines, steroids, epinephrine and mast cell stabilization.
Dysphagia in Dermatomyositis: A Forerunner of Cancer

Abstract:
Background
Dermatomyositis (DM) can be associated with dysphagia and various internal malignancies. Dysphagia has been reported due to skeletal muscle involvement in DM but is more frequent in patients with internal malignancy. DM is rarely associated with esophageal cancer as described in our patient below. Hence it is critical to differentiate the dysphagia secondary to malignancy from that of DM.

Case Description
A 59–year-old female with past medical history of diabetes and hypertension presented to our clinic for evaluation of progressively worsening rash for 2 months. The rash started underneath her eyes spreading to her face, chest, back, arms and her hands.
On exam, erythematous scaly lesions were seen in the above-mentioned areas in addition to proximal muscle weakness. Labs were significant for microcytic anemia, elevated aldolase and creatinine kinase (CK). Skin biopsy and EMG were consistent with diagnosis of DM. She was started on prednisone, mycophenolate and hydroxychloroquine with improvement in skin lesions. Four months after initial diagnosis of DM, she developed progressive dysphagia to solids and liquids with worsening skin lesions and muscle weakness. On exam she had oral thrush in addition to rash and proximal myopathy. Upper endoscopy showed ulcerating mass, biopsy of which showed poorly differentiated adenocarcinoma with presence of Helicobacter.

Discussion
DM is an inflammatory myopathy characterized by symmetrical progressive muscle weakness, characteristic skin rash, elevated CK, abnormal EMG and muscle biopsy. It is known to be associated with malignancy and the reported incidence varies between 7 to 34%. It usually precedes lung, breast, nasopharyngeal and ovarian malignancies even though less commonly associated with esophageal cancer. Malignancies are often reported within the first year of diagnosis. DM usually has a sub-acute onset and is progresses over months to years. Dysphagia has been reported to develop in 10 to 73% of patients with inflammatory myopathy during the clinical course and primarily affects the skeletal muscle-activated oropharyngeal phase of swallowing. The rapid onset and progression of disease despite use of high dose corticosteroids should ensue prompt evaluation for underlying malignancy.
Conclusion
As there is an increased risk of malignancy associated with DM, it is very important to have these patients undergo age appropriate cancer screening. Any new systemic symptoms or worsening disease despite appropriate immunosuppressive therapy should be given high index of suspicion with prompt evaluation for early diagnosis of malignancy. It is very critical to differentiate the dysphagia secondary to malignancy from that of DM for early diagnosis of an internal malignancy as described in our patient.
Abstract:

CASE:
A 62-year-old Caucasian male presented to the hospital with painless jaundice, pruritus, lethargy, dark urine and pale stool two weeks after having taken atovaquone/proguanil for malaria prophylaxis daily for 25 days. Prior to that, he has been quite healthy, had no history of drug abuse or excess alcohol drink. He had no past history or family history of liver disease. Physical examination was remarkable for sclera icterus and scratch marks in the skin. He had no hepatosplenomegaly or lymphadenopathy and no stigmata of chronic liver disease.

Laboratory testing showed elevated bilirubin to 15.9 mg/dl, ALT 973 U/L, AST 423 U/L, alkaline phosphatase 451 U/l and lipase level at 2,900 U/L. Complete blood cell count, renal function and coagulation studies were normal. Urine drug screen and acetaminophen levels were negative and serum level for Ceruloplasmin was normal. Serologic testing for hepatitis A, B, C, and E, HIV, CMV, EBV, HSV, malaria, Leptospira, and Entamoeba hystolitica were negative. Autoimmune work-up including ANA, anti-mitochondrial antibodies (AMA), liver kidney microsome type 1 (anti-LKM-1) and smooth muscle antibodies (ASMA) were also negative. Ferritin level was 3,900. Liver and portal duplex ultrasound and CT abdomen were unremarkable and MRCP did not demonstrate biliary dilation or hepatic abnormality. In the absence of a definitive diagnosis despite extensive laboratory work up, a liver biopsy was obtained and revealed mild interface hepatitis and marked centrilobular bilirubinomatoses suggestive of drug induced hepatitis. Patient medical condition was stable, accordingly was discharged. On outpatient follow up, his liver enzymes gradually returned to baseline, but cholestasis persisted. He developed persistent fatigue and progressive weight loss. A repeat liver biopsy two month later showed diffuse ductopenia diagnostic of vanishing duct syndrome (VBDS). Patient was transferred to a tertiary care center for liver transplant evaluation.

The combination of atovaquone and proguanil (Malarone™) has emerged as a promising new agent for treatment and prevention of Plasmodium Falciparum Malaria. Transient elevation of liver enzymes is a recognized side effect of the medication though the mechanism is unknown. The association of VBDS with use of Atovaquone/Proguanil was not previously reported in the literature. VBDS is a rare disorder characterized by chronic cholestasis with paucity of intralobular bile ducts in more than 50% of sampled portal tracts leading to “ductopenia” and...
cholestasis. The pathogenesis of the condition remains elusive; however, it has been reported in association with various pathologic conditions including autoimmune diseases, infections, malignancies and as a side effect of various medications. This case highlights a serious potential side effect of a usually well-tolerated medication.
Steroid Withdrawal Induced Myocarditis in Duchenne Muscular Dystrophy: A Case Presentation

Abstract:
Duchenne Muscular Dystrophy (DMD) is an x-linked recessive myopathy that affects around 20,000 males worldwide each year. Corticosteroids, especially recently FDA approved deflazacort, are a cornerstone in the treatment of DMD as they improve muscle strength and delay the deterioration of cardiac and respiratory function. This case illustrates the potential cardiac complications of the abrupt cessation of deflazacort.

An 18-year-old male with DMD presented to the emergency department with acute non-pleuritic substernal chest pain for six hours. He denied any viral prodrome or heart failure symptoms. Family history was only significant for DMD on his maternal side. Since eight years-of-age he had been on deflazacort shipped from the United Kingdom, as it was unavailable in the United States. Seven days prior to presentation he ran out of deflazacort 30 mg daily and was taking prednisone 25mg daily to substitute. His vitals, cardiopulmonary exam, and initial laboratory studies were unremarkable. An electrocardiogram revealed normal sinus rhythm at 60 bpm and ST elevations inferiorly with reciprocal changes anteriorly. Troponin I was initially 39 ng/mL and peaked above 73 ng/mL; corresponding CKMB was 359.5 ng/mL. Emergent coronary angiography revealed no coronary artery disease and normal anatomy. Subsequent echocardiogram showed a reduced left ventricular ejection fraction (LVEF) of 46% with global hypokinesis, most severe at the basal to mid inferolateral and anterolateral walls. Cardiac magnetic resonance (CMR) imaging showed diffuse myocardial edema and local fibrosis in the areas of severe hypokinesis, all consistent with acute myocarditis. Viral testing, including HIV, was negative. He received two days of colchicine with resolution of his chest pain, and was continued on prednisone. A few days later he resumed deflazacort after arrival of his shipment. Follow-up CMR three months later showed improved LVEF of 52% and resolution of the myocardial edema, with visualization of the lateral wall fibrosis seen previously.
We believe that the abrupt discontinuation of deflazacort for our patient resulted in increased inflammation and myocardial edema, manifesting clinically as myocarditis. In DMD, chronic myocardial inflammation primarily causes subepicardial fibrosis and subsequent lateral wall scarring, which can lead to a dilated cardiomyopathy. Corticosteroids have been proven to delay the onset and severity of DMD related cardiac disease. Standard of care is prednisone >0.75 mg/kg/day or deflazacort 0.9 mg/kg. Although our patient’s cardiac inflammation was controlled with deflazacort, it rapidly progressed on the equivalent dose of prednisone. Resuming deflazacort resolved his cardiac edema on CMR and his LVEF recovered. This case highlights the risk of stopping or switching between corticosteroids, especially now that...
deflazacort is FDA approved. In addition, our case reveals the utility of CMR in the detection and response to treatment of cardiac inflammation in DMD.
Abstract:
Bevacizumab or Avastin is a humanized monoclonal antibody directed against VEGF-A (vascular endothelial growth factor-A) which leads to reduced tumor vascularization and angiogenesis with increased survival. It is a newer agent added as an adjuvant therapy alone or along with platinum based chemotherapy in patients with initial or recurrent ovarian cancer. It has been used in treatment for metastatic breast cancer, non-small cell lung cancer, colorectal cancer, renal cancer, and brain cancer. Cardiovascular side effects other than hypertension are rare complications of bevacizumab therapy. Very few cardiac arrhythmias and heart failure cases have been described with bevacizumab. Cardiac syncope due to a high grade AV block along with a new reduced ejection fraction heart failure on bevacizumab therapy for ovarian cancer has not been described in the literature. We present a case of 81 year old female with past medical history of atrial fibrillation on amiodarone and xarelto, ovarian cancer on maintenance bevacizumab who presented to the hospital after a cardiac syncope. She was in her usual state of health prior to this event. Her initial electrocardiography showed a junctional bradycardia with right bundle branch block with a ventricular rate of 56. Troponin initially was negative but increased up to 2.29 (Normal limit <0.04). Brain natriuretic peptide was elevated at 430. Patient was admitted to the hospital and monitored on telemetry. During the hospital stay her heart rate dropped to 30s without symptoms, on telemetry second degree Mobitz type II AV block along with high grade AV block was noted. Echocardiography showed an ejection fraction of 20-25% with a normal previous ejection fraction two years prior. Left heart catheterization was done which showed normal coronary arteries. She had a ventricular tachycardia arrest right before the catheterization requiring intraaortic balloon pump (IABP) and temporary transvenous pacer. Subsequently, patient underwent a biventricular pacemaker with unremarkable hospital course. Patient was started on goal directed medical therapy for her heart failure with reduced ejection fraction and was discharged to an acute rehabilitation center. We conclude that it is important to have a high suspicion of a cardiac etiology of syncope in ovarian cancer patients on bevacizumab therapy. In patients presenting with syncope, careful review of medications, high suspicion for arrhythmia, and prompt intervention are vital in preventing life threatening arrhythmias and possible death in ovarian cancer patients with syncope.
Abstract:
Introduction:
Anti-MDA-5 positive amyopathic dermatomyositis is a rare type of dermatomyositis. It presents with distinct cutaneous findings, interstitial lung disease (ILD), and absence of muscle involvement.

Case:
A 56-year-old male presented with dyspnea, malaise, and pain involving his hands, wrists, and knees for five days. Review of systems, past medical history, and family history were all unremarkable. Social history was significant for a recent trip to Mexico though he had no exposure to sick contacts, wildlife or outdoor activities. Physical examination was only remarkable for violaceous macules and tenderness in bilateral DIP, PIP, and MCP joints. He had no joint swelling or warmth and had no muscle weakness. CBC was normal. CMP showed elevated AST (174 U/L) and ALT (212 U/L). Both ESR (55 mm/hr) and CRP (25.1 ng/mL) were elevated. CK (156 U/L) was normal. Rheumatologic work-up, including ANA, RF, CCP Ab, ANCA, Myeloperoxidase Ab, Proteinase 3 Ab, and Scleroderma Ab were all negative. Culture studies were unremarkable. Chest x-ray showed consolidation in bilateral lower lobes while chest CT showed diffuse ground glass opacities. Bronchoscopy with biopsy demonstrated findings consistent with organizing pneumonia. His EMG showed no myopathic changes. The constellation of findings were thought to be concerning for amyopathic dermatomyositis so myomarker panel 3 was requested and came back positive for anti-MDA-5 antibody. He was then diagnosed with anti-MDA-5 antibody positive amyopathic dermatomyositis. Prednisone taper was started with noted symptom improvement after 3 days. Within a month, he presented with worsening dyspnea. Repeat Chest CT showed an increase in bilateral ground glass opacities. He received pulse dose steroids followed by cyclophosphamide infusion with improvement of symptoms. His skin lesions were noted to have resolved. He was advised close outpatient monitoring of his pulmonary disease.

Discussion:
Dermatomyositis (DM) is an autoimmune inflammatory myopathy that classically presents with prominent weakness of skeletal muscle. Amyopathic Dermatomyositis (ADM) is a type of dermatomyositis characterized by typical dermatologic manifestations of DM, absence of muscle weakness, and no elevation in muscle enzymes. ILD is a common manifestation of DM.
but its course and severity vary according to DM classification. When ADM is suspected, a myomarker 3 panel, which includes the Anti-MDA-5(AMDA5) antibody should be requested to determine the subtype. AMDA5 antibody causes macrophage activation leading to skin, lung, and liver injury, which were all evident in this case. The presence of AMDA5 antibody is associated with lower survival rates due to rapidly progressive ILD leading to respiratory failure. Common imaging findings include ground glass opacities, as seen in this patient, followed by irregular linear opacities. Treatment consists of corticosteroid, cyclophosphamide and cyclosporine. Recognition of amyopathic dermatomyositis is challenging but a high index of suspicion can lead to timely work-up, diagnosis and treatment to prevent progression to its life-threatening pulmonary complications.
Abstract:
Introduction:
Legionella is a gram negative bacteria, found in freshwater bodies that can be transmitted via water droplets. It generally causes atypical pneumonia. Rhabdomyolysis is rapid destruction of skeletal muscles, which can lead to acute renal failure either by acute interstitial nephritis or acute tubular necrosis. We present a case of legionella pneumonia associated with rhabdomyolysis and acute renal failure.

Case Presentation:
54 year old African American gentleman with no past medical history presented for worsening cough, dyspnea, diarrhea, and severe nausea and non bloody non bilious vomiting over the previous 7 days. Review of systems was remarkable for somnolence and generalized muscle pain. On presentation, he was febrile, tachycardic, hypotensive, tachypneic and appeared in distress, with physical exam remarkable for generalized tenderness, lethargy and mild bibasilar crackles. His labs were remarkable for hyponatremia (123 mmol/L), acute renal failure (BUN 84 mg/dl, Creatinine 11.53 mg/dl), hyperphosphatemia (8.7 mg/dl), transaminitis (AST 1276 unit/L, ALT 361 unit/L), leukocytosis (WBC; 13.7 thousand/mcl) and procalcitonin elevated to 21.64 ng/ml. He was also found to have an elevated Creatinine Kinase level of 54000 unit/l. Chest Xray revealed left patchy infiltrate. Patient had emergent hemodialysis with transient improvement in the metabolic panel, as well as creatinine kinase levels. Blood cultures, HIV, hepatitis panel and viral respiratory pathogen panel were sent. Considering the hyponatremia, diarrhea and pneumonia, legionella urine antigen was also tested which came back positive. He also developed atrial fibrillation with RVR, unable to be rate controlled with diltiazem and was started on amiodarone. He was initially treated with 5 days of azithromycin with some improvement, however considering his diagnosis of complicated legionella, patient was switched to levofloxacin for 14 days. He was discharged to acute inpatient rehab with resolution of pneumonia and rhabdomyolysis,, still requiring dialysis for renal failure.

Discussion:
Legionella pneumonia complicated by rhabdomyolysis subsequently leading to acute renal failure is a rare presentation. Rhabdomyolysis is postulated to result from endotoxin mediated tissue injury as well as direct bacterial invasion of the muscles, subsequently leading to acute renal failure. It is imperative to be aware of the association between bacterial infections and rhabdomyolysis and subsequent renal failure as demonstrated by our case.
Title: Fatal outcome of Imatinib in a patient with idiopathic hypereosinophilic syndrome.

Abstract:
Fatal outcome of Imatinib in a patient with idiopathic hypereosinophilic syndrome.
Authors: Chaudhary Ahmed1, Gabriel Rodriguez1, Ashraf Abugroun1
1) Md. Advocate Illinois Masonic Medical Center

Introduction:
Imatinib is a tyrosine kinase inhibitor that is used in various hematologic malignancies including idiopathic hypereosinophilic syndrome (IHES). This case highlights the development of a cytokine storm after the initiation of Imatinib in a patient with IHES.

Case report:
A 77-year-old male with history of IHES, COPD, CKD stage III and active Mycobacterium avium complex (MAC) infection on treatment with Rifampin, Azithromycin and Levofloxacin presented to the hospital with progressive weakness, lethargy and hyperkalemia. Patient underwent extensive outpatient workup for hypereosinophilic syndrome. His disease was resistant to steroids and chemotherapy with methotrexate. On admission, patient had normal vital signs, was lethargic and cachectic. Skin examination showed widespread erythroderma, scaling and excoriations. Initial laboratory workup revealed potassium 6.9 mmol/L, creatinine 1.3 mg/dL, alkaline phosphatase 671 unit/L and WBC 18.000 cells/mcL with 58% eosinophils. Hyperkalemia resolved on the third day of admission. Subsequently, a decision was made to start Imatinib at a lower than recommended dose due to the high risk of developing tumor lysis syndrome. Shortly after initiation of Imatinib, he developed acute restlessness, agitation, shortness of breath and acute urinary retention. His condition rapidly deteriorated and progressed to acute respiratory failure requiring intubation and mechanical ventilation. He also developed distributive shock with hypothermia and non-anion gap metabolic acidosis. Absolute eosinophil count dropped dramatically from 9.500 cells/mcL to 0. Further workup revealed normal Echocardiogram and head CT scan. A CT abdomen and pelvis showed prominent bowel loops with wall thickening, suggestive of bowel ischemia. Chest x-ray showed patchy airspace infiltrates in the left lower lobe suggestive of pneumonia. Blood cultures showed no growth and respiratory culture revealed Multi-drug resistant organisms (MDRO) Klebsiella pneumoniae and moderate Pseudomonas aeruginosa. He eventually developed severe hypotension that required vasopressors, in addition to methylprednisolone and broad-spectrum antibiotics. He expired on the sixth day of admission.
Idiopathic hypereosinophilic syndrome (IHES) is a myeloproliferative disorder characterized by unexplained persistent hypereosinophilia associated with multi organ dysfunction. Available therapeutic options include corticosteroids, chemotherapeutic agents and interferon-alpha. The tyrosine kinase inhibitor Imatinib was recently proposed as a rescue medication with high response rate among patients with IHES refractory to medical therapy. Some of the adverse effects of Imatinib in patients with HES include tumor lysis syndrome, cardiogenic shock and left ventricular dysfunction. However, in this case, Imatinib caused severe acute distributive shock and multi-organ failure secondary to massive histamine release and cytokine storm evident by the temporal relation with initiation of therapy and dramatic decline of eosinophilic count to zero. This study highlights a life-threatening complication of the medication that may be under recognized.
Abstract:
Introduction: Solid Pseudopapillary Tumors (SPT) are rare pancreatic tumors [1], with low-grade malignancy and a strong female preponderance [2]. Typical presentations are usually incidental findings in asymptomatic patients or with vague abdominal pain, and thus can go undetected for extended periods of time.

Case Presentation: The patient is a 42-year-old Hispanic male who presented with nausea, vomiting and flank pain. He underwent a CT scan for nephrolithiasis and was found to have a focal calcification of the tail of pancreas. Initial physical examination and laboratory tests were within normal limits. Endoscopic ultrasound confirmed the presence of a calcified mass in the tail of the pancreas, with no associated lymphadenopathy. Fine needle aspiration (FNA) result was favoring a pseudopapillary neoplasm. Subsequently, laparoscopic distal pancreatectomy with splenectomy was performed. Pathology confirmed solid pseudopapillary neoplasm with low-grade tumor size of 3 x 2.7 x 2.2 cm focally extending into the peripancreatic adipose tissue with free surgical margins.

Discussion: SPT is an exocrine pancreatic tumor accounting for less than 1% of all primary pancreatic tumors. Since less than 10% of patients with SPT in the reported literature were males [3], this makes our case more intriguing. Almost 50% of patients are asymptomatic at time of diagnosis, and are only picked up incidentally on imaging studies for unrelated issues. Pathology reveals a well demarcated tumor with most being CD10+ and Beta Catenin+ [4, 5]. Typical treatment consists of complete tumor resection with or without splenectomy [6]. Metastasis is rare and the prognosis is favorable once the tumor is resected; unfavorable risk factors for recurrence are pancreatic parenchymal invasion and/or capsule invasion [7].
Abstract:
Introduction: Sarcoidosis is a non-caseating granulomatous disease of unclear etiology characterized by multiple organ involvement. Clinical manifestations of sarcoidosis vary widely leading to diagnostic challenges. It can mimic the presentation of lymphoma due to similar presence of constitutional symptoms with diffuse lymphadenopathy involving, but not limited to, the mediastinum. There are some reports of increased risk of lymphoma in patients with systemic chronic active sarcoidosis in the literature however the exact association or the etiology is not fully understood. We are presenting a case, in a patient with known history of chronic sarcoidosis, of newly diagnosed ALK-1 positive anaplastic large cell lymphoma which is an unusual association with sarcoidosis.

Case presentation: A 43 year old African American female with a known history of sarcoidosis for the past 13 years presented to the hospital with a complaint of fever, weight loss, pleuritic chest pain, shortness of breath, and dry cough for the past few weeks. Empiric therapy for community acquired pneumonia and sarcoidosis exacerbation by her PCP as an outpatient failed to resolve the symptoms. Physical exam was unremarkable apart from marked painless lymphadenopathy and wheezing. Imaging studies ruled out pulmonary embolism and showed diffuse lymphadenopathy of the neck, areas of nodular consolidation with multiple calcified hilar and mediastinal lymph nodes in the chest and hepatosplenomegaly. Based on the clinical presentation she was empirically started on antibiotics for tuberculosis and steroids for her presumed sarcoidosis flare. Later on, Tuberculosis and HIV were ruled out and the patient eventually underwent excisional cervical lymph node biopsy which was consistent with ALK-1 positive anaplastic large cell lymphoma. The patient was started on CHOP regimen (Cyclophosphamide, Doxorubicin, Vincristine, and Prednisone) and referred to the outpatient oncology clinic for further follow up.

Discussion: The association between sarcoidosis and lymphoma has been described in the literature but the exact etiology is not known. The onset of sarcoidosis may follow, precede or even occur concurrently with, the lymphoma complicating the clinical presentation. Our case highlights that sarcoidosis and non-hodgkin Lymphoma share many similar features and low threshold for malignancy workup should be present in cases of sarcoidosis with worsening of symptoms despite the optimal management. Our literature review suggests that patients with sarcoidosis were noted to have a 5.5 relative risk of developing lymphoma especially diffuse large B cell lymphoma. In the other hand, anaplastic large cell lymphoma has an estimated
incidence of 0.25 cases per 100,000 and although it is known to have a clinically aggressive course, it seems to be more responsive to therapy and associated with a better prognosis in patients with sarcoid disease.
Title: Starvation Ketoacidosis Rapidly Induced by High-Fat Diet in Setting of Hyperthyroidism

Abstract: Department of Medicine, University of Chicago (NorthShore), Evanston, IL

Introduction:
The causes of ketoacidosis are well known but hyperthyroidism is generally not considered part of the differential diagnosis. Given the prevalence of hyperthyroidism and the recent fad of "high-fat" or ketogenic diets, derangements in thyroid metabolism should be considered in the setting of acute ketoacidosis without another obvious cause.

Case:
A 55 year old male presented with a two-day history of nausea, vomiting, and malaise. He reported starting a “high fat” diet two days prior to presentation which included bacon, avocado, and macadamia nuts. From that evening until presentation, he experienced nausea and multiple episodes of emesis with little to no intake. On physical examination, he was tachycardic with mild abdominal tenderness. His laboratory studies revealed a B-hydroxybutyrate of >4.5mmol/L (normal 0.0-0.3mmol/L), profound ketonuria (>80mg/dL), and an elevated calculated anion gap of 31. Patient was found to have a normal lactate, osmolar gap, blood urea nitrogen/creatinine levels, salicylate levels, negative urine drug panel, and negative toxin screen. He was started on IV dextrose and normal saline with improvement of his symptoms. He was found to have abnormal thyroid studies: TSH of 0.051 u/mL (normal 0.3-5.6 u/mL) with a slightly elevated free T4 of 1.93ng/dL (normal 0.61-1.7ng/dL) and normal free T3 of 2.3 pg/mL (normal 2.0-4.4 pg/mL). Repeat thyroid studies showed: TSH of 0.086 u/mL, an elevated free T4 of 1.79ng/dL and normal T3 of 3.1 pg/dL. He was instructed to start antithyroid therapy but informed us that he would be moving to Korea and would follow up with his physician there.

Discussion:
Starvation is a known cause of ketoacidosis and is part of the traditional differential diagnosis of anion gap acidosis. However, starvation alone typically takes weeks to develop ketoacidosis to the degree seen in this case. Thus, two days of decreased intake does not explain the rapid onset of ketoacidosis. Hyperthyroidism, on the other hand, is rarely reported as a cause of ketoacidosis but the lipolytic effects of thyroid hormone on adipocytes and its promotion of hepatic beta-oxidation is well known. In this case, the high-fat or ketogenic diet likely triggered ketoacidosis in this patient with underlying hyperthyroidism. This is the first case report to our
knowledge, of a high-fat or ketogenic diet causing ketoacidosis in a patient with untreated hyperthyroidism. This case illustrates the need for including hyperthyroidism in the differential when workup for the common causes of ketoacidosis is unrevealing. Although a rare cause of ketoacidosis, hyperthyroidism is easily managed and a thyroid function test should be obtained, particularly in patients who are on a high-fat or ketogenic diet.
Myeloid sarcomas causing unilateral cranial nerve palsies in a patient with relapsed acute myeloblastic leukemia

Abstract:
Introduction
Myeloid sarcomas (MS) are extramedullary tumors composed of malignant immature myeloid cells and represent a rare complication of myeloid malignancies. In patients with acute myeloblastic leukemia (AML), the prevalence of MS ranges from 3 to 9%. Clinical presentation depends on the size and location of the mass, and mechanical compression of nearby structures. MS are commonly diagnosed with a concurrent primary myeloid malignancy; however, they can occur in the absence of bone marrow involvement, which makes diagnosis challenging.

We present the case of a patient with a diagnosis of AML who presented with numerous myeloid sarcomas at the time of first relapse.

Case description
The patient is a 43-year-old man, who presented with unintentional weight loss and abdominal subcutaneous nodules. Initial laboratory exams showed an elevated LDH level (1,165 UI/L), and myeloid blasts in peripheral blood. Bone marrow biopsy showed infiltration by 47% myeloid blasts with an abnormal karyotype (46, XY t(2;21;8) (p13;q22;q22)) and positive for AML1-ETO fusion gene. Whole-body computerized tomography (CT) revealed numerous soft-tissue masses involving anterior mediastinum, pericardium and subcutaneous tissue in the abdomen and pelvis.

The patient was diagnosed with acute myeloblastic leukemia with t(8;21) variant and extramedullary involvement and completed chemotherapy attaining a complete remission.

Four months after therapy, the patient developed oropharyngeal dysphagia and right facial asymmetry. Physical examination showed right parotid enlargement and a palpable left axillary mass. Neurological exam revealed palsies of cranial nerves VII, X, and XII. Laboratory results were unremarkable and a peripheral blood smear did not show leukemic blasts. CT and magnetic resonance imaging of the neck showed an infiltrative soft-tissue, right skull base mass extending into the right jugular and stylomastoid foramen, parotid gland and temporomandibular junction causing displacement of the internal carotid artery, encasing and occluding the internal jugular vein. CT scans showed multiple soft tissue masses in the anterior mediastinum, left renal hilum and subcutaneous tissue in the abdomen. Biopsy of bone marrow and left axillary mass confirmed a relapse of acute myeloblastic leukemia and myeloid sarcoma.
respectively. The patient received reinduction chemotherapy attaining a second complete response of medullary and extramedullary disease.

Discussion
Although rare, MS can add significant morbidity in patients with AML. As in our case, MS compressing neurological or vascular structures can lead to severe complications or permanent neurological deficits. Diagnosis can be challenging in patients with isolated MS without overt medullary disease, where disseminated infections or secondary malignancies are considered. In such scenarios histological examination by an experienced pathologist is paramount. Patients with AML and t(8;21) have a higher prevalence of MS compared to other subtypes of AML; however, the ideal chemotherapy regimen and the prognostic impact of MS remain uncertain.
Title: Paraneoplastic gastroparesis: initial presentation of undiagnosed lung cancer

Abstract: Paraneoplastic gastrointestinal (GI) syndromes rarely precede the actual detection of an overt cancer with gastroparesis being a very rare initial presentation. Likely pathogenesis is the destruction of neuronal ganglions with chronic inflammation of involved nerve fibers. To increase the clinical awareness of this rare clinical entity, we present a case of severe gastroparesis that was later proven to be associated with an overt poorly differentiated lung cancer.

A 61-year-old African American man with no significant medical history came to medical attention in December 2016 with severe post prandial bloating and vomiting for 2 months. On admission, vital signs and physical examination were unremarkable. The following laboratory tests were normal or negative: blood urea nitrogen, serum creatinine, bilirubin, alanine aminotransferase, aspartate aminotransferase, urinalysis, hemoglobin A1c, thyroid stimulating hormone, chest X-ray, electrocardiogram, computed tomography (CT) scan of the abdomen and upper Gi endoscopy. Further workup in the form of gastric emptying scan and small bowel follow through studies was consistent with generalized GI hypo-motility disorder of unclear etiology. CT scan of the chest showed a sub-centimeter spiculated nodule in right upper lobe of the lung with hypermetabolic activity seen on positron emission tomography (PET) scan consistent with malignancy. Nodule resection and biopsy showed poorly differentiated lung carcinoma. Serological testing was positive for neuronal nuclear antibodies (Anti-Hu, Anti-Ri) and cytoplasmic purkinje cell antibodies (Anti-YO). Overall clinical presentation was compatible with paraneoplastic GI hypomotility disorder and patient was started on combined modality treatment with chemotherapy and radiation therapy for his lung cancer. Further modalities in the form of dietary modifications, prokinetic agents and psychological counselling were used for relief of his severe GI symptoms and clinical improvement was observed on followup visits.

After ruling out common etiologies first, unexplained gastroparesis should always prompt workup for an overt malignancy in the right clinical setting. Serological detection of anti-Hu antibodies is of clinical significance in the early diagnosis of paraneoplastic gastroparesis with a specificity and a sensitivity of 99% and 82% respectively. Once the diagnosis is established, treatment is mainly supportive with removal of the antigen source by treating the tumour along with dietary modifications, prokinetic agents and psychological counseling for symptomatic relief.
INTRODUCTION
Methamphetamine (MA) and amphetamine-related compounds are the most commonly abused street drugs. MA toxicity is associated with 57% increase in mortality and multi-system toxicity through various mechanisms. Most widely studied effects are cardiotoxicity, nephrotoxicity, hepatotoxicity, and neurotoxicity. These effects are more pronounced when combined with other drugs like cocaine, ethanol or opioids. We present a case of methamphetamine induced necrotizing vasculitis and myositis - an uncommon toxicity challenged by the timeliness of diagnosis that poses life and limb threatening outcomes.

CLINICAL CASE
A 35-year-old man with a history of HIV on antiretroviral therapy and IV methamphetamine abuse presented with four hours of acute severe left leg pain, preceded by a few hours of left foot drop and hyperemia. On admission, he had left leg cyanosis that was spreading proximally with absent dorsalis pedis and posterior tibial pulses. He denied any skin or soft tissue infection preceding the event. Lower extremity angiogram was unremarkable; he was surgically treated for suspected necrotizing fasciitis with an above-the-knee amputation. Intra-operative findings were consistent with anterolateral and posterior myonecrosis and necrotizing fasciitis. His operative microbiologic studies and blood cultures were all negative for bacterial growth. Pathology eventually revealed focal necrotizing neutrophilic vasculitis and myositis and intravascular fibrin clots. Additional work up showed elevated IgM anti-cardiolipin antibody and elevated beta-2 glycoprotein suggestive of secondary antiphospholipid antibody syndrome (APLAS). He was started on prednisone for methamphetamine induced vasculitis and was empirically started on anticoagulation therapy. He was discharged with a follow-up confirmatory laboratory tests for APLAS.

DISCUSSION
Methamphetamine and adulterants in drug preparations cause direct irritation of blood vessel walls, inhibition of uptake of dopamine, norepinephrine, and serotonin resulting in pseudo-vasculitis from vasospasm and inflammation. Pseudo-vasculitis responds to the removal of the offending agent and usually does not require the use of immunosuppressant therapy. Unfortunately, medications and drugs are also associated with hypersensitivity leukocytoclastic vasculitis. Few case reports have discussed mononeuropathy and vasculitis similar to our patient’s presentation. Use of steroids and immunosuppressant therapy is the standard of care for the management of vasculitis that prevents complications from ongoing ischemia. The overlapping clinical picture of pseudo-vasculitis, necrotizing fasciitis, and vasculitis and the
challenge of providing emergent life-saving interventions or even amputation in necrotizing fasciitis highlight the need for earlier diagnostic tests that can differentiate these pathologies. Limb amputation carries high morbidity with chronic pain and psychiatric distress. Efforts should be placed in making clinicians aware of the different pathologic mechanisms of this particular clinical presentation of methamphetamine toxicity.
Last Name: Bavishi  First Author: Resident

First Name: Aakash  Category: Clinical Vignette

ACP Member:

Additional Authors:

Title: Young Female with Chest Pain and ST- Elevation: A Case of Spontaneous Coronary Artery Dissection

Abstract:
Spontaneous Coronary Artery Dissection (SCAD) is a rare cause of Acute Coronary Syndrome (ACS) and the risk factors and medical management of SCAD is an area of ongoing debate.

38 year old healthy woman developed substernal chest pain and shortness of breath while walking to work. Chest pain increased in intensity and patient became diaphoretic and nauseous. On exam, she was hypertensive (180/120) and tachycardic (120). Denied any cardiac history or cocaine use. Her mother had myocardial infarction at age 50. Only home medication was combined oral contraceptive (OCP).

EKG showed significant ST elevation in inferolateral leads and patient was given Aspirin 325mg and Ticagrelor 180mg. She was taken urgently for left heart catheterization (LHC). LHC was notable for distal obtuse marginal coronary artery dissection with no other obstructive lesions. Ventriculography showed inferior wall akinesis. No intervention was done. Troponin-I and CK-MB peaked at 32 and 91.4, respectively. Transthoracic Echocardiogram notable for inferolateral wall motion abnormalities with normal left ventricular ejection fraction.

Her chest pain resolved. She was started on daily aspirin 81mg, metoprolol succinate 100mg, and advised to discontinue her OCP. Renal ultrasound with doppler was within normal limits.

This case represents a unique presentation as SCAD accounts for less than 0.5% cases of ACS. SCAD is increasingly being identified as cause of ACS in women, especially in those younger than 50 years. In fact, SCAD may account for nearly up to 25% of cases in this cohort. Another risk factor for our patient was prolonged use of combined OCP. Numerous case reports have shown an association between SCAD in healthy women and prolonged use of combined OCP. There is a very strong association between SCAD and fibromuscular dysplasia (FMD). There is no consensus on how much vasculature should be evaluated to screen for FMD in SCAD patients. Only the renal vasculature was evaluated in our patient.

As was the case with our patient, most patients with SCAD- especially those who are hemodynamically stable- are managed conservatively given high rates of complications with revascularization. PCI is technically challenging given frailty of often small caliber vessels,
increasing risk for propagating dissection and stent-in-stent thrombosis. B-blockade has been shown to reduce arterial shear stress, facilitate healing, and reduce risk of recurrence. The utility of anti-platelet agents in SCAD patients not treated with stents is unclear. Given evidence of aspirin in ACS and secondary prevention of CAD, it is reasonable to commit to long-term aspirin therapy in SCAD patients. There is no benefit when clopidogrel is added for SCAD patients and our patient received aspirin alone. There is no indication for statin therapy in SCAD patients with normal lipid profile and HbA1C, as was the case in our patient.
Abstract:
Background: Foley catheter and diaper use in hospitalized women are plagued by catheter-associated urinary tract infections (CAUTIs), skin breakdown, and patient discomfort. These urinary collection devices are frequently indicated in bedbound women when incontinent or for strict intake and output (I/O’s) measurements in intensive care units, operating rooms, or on medical floors in the hospital. When not limited by urinary retention, an external female catheter presents a viable and safer option. This is an analogous premise to the use of the condom catheter in men; however, a similar device has not been widely used in women in United States (U.S.) hospitals to-date. The PureWick® external female urinary catheter is a sterile flexible rod resting against the vulva which gently vacuums urine expelled through tubing to a wall canister that has shown promise at reducing CAUTI and skin breakdown while improving comfort.

Methods: We conducted a 2 month quality improvement project implementing the PureWick catheter in Northwestern Memorial Hospital’s (NMH) Medical Intensive Care Unit (MICU) and Neurosurgical Intensive Care Unit (NSICU) with survey assessment of registered nursing (RN) reported patient outcomes. All women in these units who needed urinary catheter placement and lacked urinary retention were enrolled in this trial. Thus far, this trial has studied approximately 90 wicks in 50 bedbound women.

Results: Outcomes revealed no associated urinary tract infections or skin breakdown with use. 92% of RN’s said urine capture was excellent with accurate I/O’s. 100% recommended future use and said it was easy to use. Foley use was prevented orfoleys were removed in 67% of patients, with an absolute reduction of foley catheter usage from 31% to 18% in the MICU. Rates in NSICU remain at 34%. Diaper use was prevented altogether. These results mirror similar PureWick data gathered over 1 year at five other U.S. hospitals surveyed with decreased rates of CAUTI by 50-59%, decreased foley usage by 15-26%, and 95-100% efficacy in catching urine. Centers surveyed reported improved chronic ulcer healing during PureWick use. In all cases, the device was highly rated by staff and patients for comfort, convenience and functionality. Further, cost analyses indicated a potential net savings of approximately $1 million dollars per year due to reduced cost of CAUTIs and associated Medicare fines.
Conclusion: These data are promising and indicate that patients and hospitals could benefit from filling this void in device options for female patient care. Female external catheters should be further studied for implementation in bedbound women without urinary retention in U.S. hospitals. The PureWick device appears to provide safer and more comfortable urine collection in this population compared to standard foley catheter and diaper use.
Abstract:
Introduction:
Pott’s puffy tumor (PPT) is a subperiosteal abscess of the frontal bone. It is most commonly caused by frontal sinusitis resulting in frontal bone osteomyelitis, and subsequent extension of the infection. PPT has a risk of life threatening intracranial complications but remains a rare clinical finding, particularly in adults, due to widespread antibiotic use.

Case description:
A 55 year female with a history of systemic erythematous lupus (SLE) on chronic prednisone and hydroxychloroquine presented to the emergency department with frontal head pressure, forehead swelling, eye redness, and congestion for two weeks. She had previously been treated with eye drops and nasal sprays but without improvement in symptoms. In the ED, she was febrile (38.4C) and tachycardic. She was lethargic but arousable and without focal neurologic deficits. Laboratory findings were notable for a WBC of 17,000. CT sinuses revealed acute on chronic infection of the frontal sinuses with subperiosteal extension of infection. ENT was consulted and she was started on vancomycin and ceftriaxone and admitted to the general medicine floor. On hospital day 2, she continued to have persistent fevers and complained of headache and weakness. MRI was ordered but she became disoriented and responded to all questions with “OK” and then her name and date of birth. MRI brain without contrast showed intracranial extension of left frontal sinus infection with thin left frontal epidural abscess and cerebritis. Metronidazole was added to her antibiotic regimen and infectious disease and neurosurgery were consulted. She underwent an acute bilateral maxillary antrostomy, ethmoidectomy, and bifrontal craniotomy with washout and exenteration of frontal sinuses that was performed jointly with ENT and neurosurgery. Culture of the subdural empyema was positive for S. milleri and the sinus cultures were positive for MRSA and candida. Over the next several days, she had full resolution of headaches and aphasia and was discharged home on post-operative day 10, to complete a six-week course of IV vancomycin and PO metronidazole, and two weeks of fluconazole.

Discussion:
Pott’s puffy tumor is a rare but serious complication of frontal bone osteomyelitis, with less than 50 adult cases reported in the literature. This case is the first adult case, to the best of our knowledge, of PPT in a patient with SLE on chronic immunosuppressive therapy. PPT has a risk of intracranial complications including subdural empyema, epidural empyema, brain abscess, and cortical vein thrombosis. Although rare in adults, PPT should be on the differential for patients presenting with persistent sinusitis symptoms, especially in those with focal forehead
swelling. Early intervention and a collaborative medical and surgical approach are essential for prevention and treatment of intracranial complications.
57 year old male presented with nausea, vomiting and worsening abdominal pain for 2 days. He was recently diagnosed with advanced duodenal adenocarcinoma and got a palliative stent placed. He also had history of type 2 DM. He was on metformin and Canagliflozin. Patient was afebrile, tachycardic to 120/min, tachypneic with rate of 30/min and had a stable BP. He was in distress due to abdominal pain and breathing was labored, oral mucosa was dry, lungs were clear to auscultation, abdomen was distended and tender. Initial labs were, Glucose 180 mg/dl, creatinine 0.9mg/dl, Na 135 mmol/l, k 5.0 meq, Cl 92 mmol/l, Bicarb 9.0mmol/l, Anion gap 31, lactate 1.6 mmol/l, WBC count 11. ABG, PH 7.20, PCo2 10mmHg. Emergent abdominal imaging showed no signs of surgical abdomen. CT Angio chest was negative for PE. Serum salicylate levels, blood alcohol level and urine toxicology were negative. As the glucose level was not high and with a history of well controlled DM type 2 on oral medications, suspicion for DKA was low. ABG 2 hrs from the prior showed PH of 7.04. Urine ketones was 80mg/dl and urine glucose >500 mg/dl. Patient was diagnosed with euglycemic diabetic ketoacidosis likely secondary to SGLT2 use. He was treated with Insulin infusion and fluids according to DKA protocol. Patient responded well. Over 24 hrs anion gap closed, patients symptoms improved and was started on diet after checking KUB to rule out worsening of duodenal obstruction.

Euglycemic diabetic ketoacidosis is described as anion gap metabolic acidosis with a glucose level < 200 mg/dl. Our patient had high anion gap metabolic acidosis and respiratory alkalosis with normal salicylate and lactate levels. He had no history of alcohol use. Blood glucose was not very high, however labs showed ketonemia and ketonuria. SGLT -2 inhibitors like Canagliflozin have been reported to cause euglycemic DKA. These medications increase glucose excretion in urine and lead to drop in blood glucose levels, which decreases insulin secretion from pancreas leading to ketogenesis. Glucosuria keeps the glucose levels low to normal despite of lack of insulin. Ketoacidosis is triggered only in setting of infection, poor oral intake or acute illness. As in case of our patient who was taking canagliflozin and had poor oral intake due to malignancy. In contrast to DKA which is associated with high glucose levels, Euglycemic DKA presents with low glucose levels but is equally fatal. It is a rare entity and without awareness, the normal to slightly elevated glucose levels can be misleading and lead to diagnostic delays as in our case, whose severe acidosis worsened in a couple of hours. Treatment is prompt IV fluids and Insulin infusion.
Formation of mediastinal pancreatic pseudocysts is a rare consequence of chronic pancreatitis, with the potential to cause a variety of complications. We present a case that demonstrates pancreatic pseudocysts as a cause of dysphagia with unintentional weight loss as well as dyspnea.

A 49-year-old male with a past medical history of chronic pancreatitis secondary to alcohol abuse presented to the emergency department with left-sided pleuritic chest pain and shortness of breath for several days. Chest X-ray showed a lingular consolidation and left-sided pleural effusion. A diagnosis of pneumonia was made and the patient was discharged on oral antibiotics and analgesics. The patient returned to the same emergency department five weeks later with the persistence of left-sided chest pain and shortness of breath, but also with new-onset dysphagia causing anorexia and unintentional 20lb weight loss. Chest X-ray revealed worsening left-sided pleural effusion, and a thoracentesis was performed. Pleural fluid revealed an exudative pattern with amylase concentration of 2783 U/L. Serum amylase concentration was 300 U/L and serum lipase was 103 U/L. Liver enzymes were within normal range and the patient did not have a leukocytosis. A CT chest/pelvis/abdomen was performed to rule out pulmonary embolus and to evaluate for acute-on-chronic pancreatitis. This revealed multiple mediastinal fluid collections encasing the esophagus, consistent with pancreatic pseudocysts. The largest of these pseudocysts was estimated to be 10 x 2.3 x 4.3 cm in volume. Octreotide therapy was initiated for management of a suspected pancreatic duct leak. An ERCP performed three days later revealed a cranially-extending pancreatic tail leak, consistent with a pancreatic-pleural fistula. A 5 French 10cm pancreatic duct stent was placed in an attempt to direct fluid away from the pleural space. The patient’s pleural effusions eventually diminished in size. His dysphagia disappeared and his chest pain was reduced to a tolerable level. He was discharged on octreotide and analgesics. Follow-up chest X-ray six weeks later showed resolution with clear lung fields.

This case report demonstrates an unusual cause of dysphagia, weight loss, and dyspnea. The patient was initially suspected to have a malignancy given his tobacco history, especially in the context of weight loss, respiratory symptoms, and the suggestion of an extrinsic structural impingement of the esophagus. Other early considerations included infection, given findings on his chest X-ray; and alcohol-related aspiration event, given his history of heavy alcohol use. This report illustrates the diverse range of consequences that can arise from pancreatic pseudocyst formation, and the importance of considering pseudocysts for any patient with chronic pancreatitis who presents with new unexplained symptoms.
Non-Cirrhotic Ascites Secondary to Peritoneal Tuberculosis

A 76-year-old Hispanic female with history of type two diabetes mellitus and hypertension presented for evaluation of new onset ascites. She endorsed four weeks of abdominal distension, with bilateral upper abdominal pain and intermittent nausea. On exam, her abdomen was soft, distended with bilateral upper quadrant tenderness. No hepatosplenomegaly, rash, jaundice, bruising or skin lesions were appreciated. Initial labs were remarkable for an AST of 84, ALT 26 and albumin of 2.2. Chronic hepatitis panel was negative. UA was positive for proteinuria. CT of the abdomen and pelvis did not reveal any visceral pathology. Analysis of the ascitic fluid showed 150 PMN cells/mm³, lymphocyte predominance, SAAG of 0.8, and modestly elevated total protein. Bacterial, fungal and mycobacterial cultures had no growth. Smear for acid-fast bacilli and cytology were negative. Quantiferon gold assay was positive and CA-125 level was elevated at 523. Pelvic ultrasound was unrevealing. Decision was made to perform an exploratory laparotomy. Adhesive intra-abdominal disease, inflammatory changes, friable tissues and ascitic fluid were found, suspicious for military TB. Biopsies of the omentum and peritoneum were TB PCR positive. The patient was started on a four-drug regimen for miliary TB. Cultures were susceptible to all four drugs.

Peritoneal tuberculosis is a great mimicker. Acid fast staining is often negative and cultures are positive in less than 50% of cases. In this case, the elevated serum CA-125 was a confounder, as it can be increased in ascites due to any cause. Previous case reports of PTB coexisting with ovarian carcinoma suggest that adenosine deaminase can assist in discriminating between the two diseases. Diagnosis of PTB is confirmed by biopsy during laparoscopy. Mortality is increased with delayed treatment. Thus, a high index of suspicion is needed in patients with lymphocyte predominant ascites.
Title: Management of Nivolumab induced sudden hepatitis (NISH)

Abstract:
Background:
Nivolumab is an immune check point inhibitor that targets PD-1 receptor pathways. It is commonly used in treatment of various malignancies and is recently being used for metastatic renal cell carcinoma. It causes immune mediated hepatitis with a reported incidence of less than one percent. Corticosteroids have been used as treatment in drug induced auto immune hepatitis. Unfortunately, there are no official guidelines for management of steroid resistant hepatitis. We present a rare case of Nivolumab induced sudden hepatitis (NISH) resistant to steroids that was successfully treated with an alternative modality.

Case Presentation:
A 77 year old woman, recently diagnosed with clear cell carcinoma of kidney status post right nephrectomy with pulmonary metastasis, presented with 1 week of generalized weakness, fever, abdominal pain, nausea and non-bloody vomitus. She received her first dose of Nivolumab one week prior. Physical examination revealed scleral icterus and right upper quadrant tenderness. Initial lab workup revealed transaminitis with an AST of 995 and ALT of 1486, hyperbilirubinemia (total bilirubin 4.4), mild leukocytosis. Her liver enzymes 2 days prior to the start of chemotherapy were normal. Ultrasound of the abdomen showed no abnormalities or any gall bladder pathology. Clinical status and liver function declined within the next 24 hours, therefore she was started IV Hydrocortisone 100 mg q 8 hours. Autoimmune workup, serum alpha1 antitrypsin and iron studies were negative. Despite therapy, her levels continued to drastically elevate and thus she was started on IV N-acetylcysteine (NAC) and Mycophenolate Mofetil (MMF) and planned for liver biopsy. Biopsy revealed acute pericholangitis with rare neutrophils in the lumen of the bile ducts, reactive bile ductal epithelial changes, focal lobulitis and abundant micro vesicular steatosis. The acute pericholangitis without any biliary obstruction was suggestive of drug reaction. After three days of the new treatment regimen, her liver function started to improve. She was discharged home with a prolonged three month steroid taper.

Discussion:
This is likely the first case of early onset steroid resistant hepatitis secondary to Nivolumab, which was successfully managed with the use of MMF. There seems to be an added benefit with NAC in severe hepatitis. Only a few cases of Ipilimumab induced hepatitis have been treated successfully with MMF therapy. There are no reported cases in the literature of Nivolumab induced steroid refractory hepatitis and hence no guidelines exist on how to
appropriately manage this pathology. Furthermore, autoimmune hepatitis has been shown to occur after long periods of therapy, between 7 to 12 weeks, but our patient presented acutely within the first 3 days. Our case puts forth the importance of early detection of NISH that is steroid refractory and provides additional information in understanding the etiology, pathogenesis and formulation of guideline treatment.
Sorry to burst your bubble, but this common chief complaint isn't so simple

Abstract:
Case:
A 66 year old man with a history of coronary artery disease, atrial fibrillation on warfarin, and chronic neck pain presented to the emergency department with three days of acute onset chest pain, low back pain (LBP) and testicular pain. The patient was discharged with a course of antibiotics for presumed epididymitis after workup revealed stable vitals, negative cardiac enzymes, EKG without ischemic changes, baseline renal function on labs and urinalysis negative for blood and leukocyte esterase. Twelve days later, at outpatient follow up, he reported improved chest and testicular pain, but continued to endorse the LBP, described as sharp and constant, radiating to the front. His vitals and exam were again reassuring, and he was referred to physical therapy and to pain clinic, where he is already seen for his neck pain. Five days later, he was seen by urology for follow up of his epididymitis, at which time he continued to endorse the LBP which had evolved to radiate into the left lower quadrant. On exam, the patient had bilateral costovertebral angle tenderness. A noncontrast CT to evaluate for nephrolithiasis revealed a possible distal leaking aortic aneurysm. The diagnosis of distal aortic aneurysm with contained rupture was confirmed on CT angiography, with active contrast extravasation seen at the time of the study. He underwent an emergent endovascular aneurysm repair that same day, 19 days after initial presentation. He did well postoperatively without any complications, with significant improvement in LBP.

Discussion:
LBP is one of the most common chief complaints in the outpatient setting. Given that the majority of cases are nonspecific and resolve without intervention, imaging is not routinely recommended, particularly as evidence suggests imaging often does not improve outcomes and may lead to unnecessary procedures. When assessing patients with LBP, clinicians are often taught to look for “red flag” symptoms of medical emergencies including spinal cord compromise, infection or malignancy. Ruptured abdominal aortic aneurysm (rAAA) are not often thought to fall into the category of LBP emergency. However, studies have suggested that the classic triad of abdominal pain, pulsatile mass and shock is only present in about half of patients with rAAA, with LBP being the presenting symptom in about 42% of patients. In this patient, definitive diagnosis and treatment of a medical emergency was delayed due to presentation of a common complaint and reassurance with stable vitals and labs. It is important to be aware of atypical presentation of rAAA as well as maintain a wide differential diagnosis
for unusual causes of LBP, particularly in patients with significant vascular risk factors including diabetes, hypertension and cardiovascular disease.
Intestinal tuberculosis (TB) is an uncommon cause of abdominal pain and fever that can clinically and histologically mimic Crohn’s Disease. The diagnosis should be entertained in patients with presumed Crohn’s and risk factors for TB whose symptoms do not respond to immunosuppressants.

A 30-year-old woman from Poland was diagnosed with Crohn’s Disease based on colonoscopy and biopsy results after presenting with several months of abdominal pain, nausea and vomiting. She was started on prednisone 40mg daily with initial improvement in her symptoms. After several months her symptoms recurred so infliximab, a tumor necrosis factor (TNF) alpha inhibitor, was added to her medication regimen. Prior to initiation of infliximab she had a negative tuberculin skin test. Her symptoms were initially stable, but four months later she again presented with abdominal pain, vomiting, fevers, and shortness of breath. Her labs were notable for pancytopenia, hypoalbuminemia and coagulopathy. A computed tomography scan of her chest, abdomen and pelvis showed apical scarring and scattered ground glass opacities in bilateral lung fields as well as ileocecal wall thickening and extensive mesenteric adenopathy. Induced sputum samples were obtained and positive for many acid fast bacilli (AFB). Her course was complicated by hypovolemic shock from an upper gastrointestinal bleed and acute respiratory distress syndrome. She also developed acute encephalopathy and underwent a lumbar puncture with +AFB staining. She was initiated on therapy for disseminated tuberculosis with isoniazid, rifampin, pyrazinamide and levofloxacin, as well as a prolonged steroid taper. After four weeks she was discharged from the hospital with a plan for direct observed therapy as an outpatient. The pathology slides from her original biopsy were eventually obtained and reviewed. They showed non-caseating and caseating granulomas, indicating that she was likely initially misdiagnosed as Crohn’s Disease when in fact had intestinal TB. Infliximab was presumed to be the precipitant for rapid dissemination.

This case represents an usual presentation of disseminated TB and highlights the similarities between Crohn’s Disease and intestinal TB. In this case, the initial misdiagnosis and subsequent treatment with infliximab likely precipitated disseminated TB and led to serious complications for the patient. Reactivated TB should be a diagnostic consideration in patients who clinically deteriorate on a TNF-alpha inhibitor, even in the setting of a prior negative screening test.
Silent Abdominal Pain

Nicholas Daane, DO
PGY2 Internal Medicine, Advocate Lutheran General Hospital, Park Ridge, IL

45 yo male with spastic quadriplegic cerebral palsy, nonverbal secondary to tongue weakness, presents to Internal Medicine outpatient clinic for complete physical, after not being seen by a physician for over 20 years. No presenting symptoms, but labs obtained for screening revealed mild elevations in both cholestatic and hepatocellular liver function tests. A thorough outpatient workup was performed, and imaging revealed cirrhosis, ascites, and multiple liver nodules with arterial phase enhancement with no definite washout. Before patient could see gastroenterology as an outpatient, was admitted through the ER for worsening bilateral lower extremity edema and abdominal distension. On physical exam, no jaundice, no abdominal pain to palpation, but did have shifting abdominal dullness, as well as lower extremity pitting edema bilaterally in the lower extremities, extending proximal to the knees. Labs remarkable for a new hyperbilirubinemia, but other liver enzymes relatively stable from outpatient baseline a few months prior. GI workup revealed a positive AMA, rest of autoimmune workup and tumor markers negative. Paracentesis performed, and fluid was transudative, showed no signs of infection, and cytology negative for malignancy. Transjugular liver biopsy performed, and hepatic venous pressure gradient was slightly elevated. Biopsy was inconclusive, sent to Cleveland Clinic for further review. Patient was discharged home with diagnosis of Primary Biliary Cholangitis with cirrhosis and portal hypertension, started on ursodiol pending pathology review. Was only home for short time before being readmitted for intractable nausea and vomiting. Biopsy results revealed hepatic sinusoids lined by atypical endothelial cells with enlarged hyperchromatic nuclei, CD34 positive, but p53 negative. Concerning for angiosarcoma, but not diagnostic without demonstration of solid growth pattern. A repeat ultrasound guided liver biopsy was performed, but showed similar pathology. Oncology consulted, Palliative care recommended.

While this case has an unfortunate ending, it has very interesting pathology. Moreover, it illustrates the significance of clinical Internal Medicine, and is a great example of how to properly work up asymptomatic elevated liver function tests as an outpatient, with proper escalation of laboratory investigation and imaging.
Abstract:
Tramadol is a common pain medication available under various brand names. Tramadol and its active metabolite (M1) binds to μ-opiate receptors in the CNS causing inhibition of ascending pain pathways, altering the perception of and response to pain; also inhibits the reuptake of norepinephrine and serotonin, which are neurotransmitters involved in the descending inhibitory pain pathway responsible for pain relief. Serious anaphylactoid reactions (including rare fatalities) often following initial dosing have been reported in <1 % of cases.

A 18-year-old female with past medical history of allergy to ibuprofen presented to the emergency department with acute onset of shortness of breath after taking 2 tramadol pills. She stated that she had a dental procedure 1 day prior to admission for which she was prescribed tramadol. The patient had some dental pain for which she took 2 pills of tramadol. Shortly thereafter she developed shortness of breath and generalized pruritis. She called an ambulance and was brought to the ED. She received a dose of IM epinephrine on her way to the ED. In the ED, the patient's SBP was initially in the 70s but then improved to 90s. She initially had mild shortness of breath which later developed into severe respiratory distress. On physical exam in the ED, the patient had diffuse crackles in all lung fields. She had increased work of breathing, tachypnea and was using accessory muscles for breathing. She was put on bipap and given, Rocephin, solumedrol, famotidine, Benadryl and 1 L of normal saline bolus. CT Angio of the chest showed diffuse patchy airspace disease noted in both lungs and more prominent at lung bases consistent with moderate ARDS. Troponins were mildly elevated, which was most likely from demand ischemia due to underlying acute lung injury/acute respiratory distress syndrome, hypotension and acidosis. An EKG showed normal sinus rhythm. She was transferred to the ICU for further care. Upon arrival to the ICU, the patient was seen in severe respiratory distress on bipap. Her respiratory rate was in the mid-60s. She was intubated and was kept on ventilator for 48 hours and then extubated when the chest x-ray showed clear lung fields. After 3 days of ICU stay, she was transferred to the general medical floor where she remained stable and was discharged on tapering dose of methylprednisolone.

This case illustrates potential for severe anaphylactic reaction secondary to tramadol, a commonly used pain medication. Understanding the severity of the reaction, which occurs very rarely with Tramadol, helps us in better treating the patient.

References:
**Title:** Abdominal Pain as a Rare Presentation of HTLV-1-driven adult T-cell Lymphoma/Leukemia

**Abstract:**
Adult T-cell Lymphoma/Leukemia (ATLL) is a rare lymphoproliferative malignancy, diagnosed in up to 5% of Human T-cell Lymphotropic Virus-1 (HTLV-1) infected patients. Endemic in Japan, Africa, Caribbean and South America, the blood-borne (horizontal, vertical and blood transfusion transfusion) HTLV-1 can induce transformation of T-cells over decades, (although infecting B and mast cells as well). Typical presentation of ATLL can vary from local to systemic manifestations. A patient is described who initially presented with a rare constellation of advanced symptoms of papular rash, hypercalcemia, lymphocytosis, abdominal pain and liver function tests consistent with cholestasis. Diffuse type ATLL was subsequently diagnosed.

A 60-year-old Jamaican female with past medical history of hypertension presented to an outside hospital in Suburban Chicago with diffuse papular pruritic rash present for approximately one month and a five-day history of epigastric pain with nausea without constitutional symptoms. Initial workup was notable for hypercalcemia, mild lymphocytosis with liver function tests consistent with cholestasis and diffuse biliary duct dilation on ultrasound, which was treated medically as acute cholangitis. Due to worsening transaminitis with negative autoimmune and infectious workup, the patient eventually underwent cholecystectomy with liver biopsy of a suspicious lesion found intraoperatively. As her fever curve and liver function tests worsened postoperatively, further workup revealed new diffuse lymphadenopathy with splenomegaly. Peripheral blood review with flow cytometry, CSF sampling and biopsies of rash, bone marrow, liver and axillary lymph node were obtained. Results revealed ALK negative, CD30 positive HTLV-1-driven Adult T cell leukemia/lymphoma in acute leukemic phase. Further clinical course was complicated by Cytomegalovirus (CMV) and urine BK viremia. The patient was started on multiple therapeutic regimens: AZT and IFN (HTLV-1) with intrathecal methotrexate followed by CHOP (ATLL) as well as Ganciclovir (CMV) with plan for allogeneic stem cell transplant.

Among all HTLV strains, only HTLV-1 has oncogenic potential. While estimated 20-30 million people live with HTLV-1 infection, only 1-5% is known to seroconvert into ATLL. Abdominal pain and acute cholangitis is a never described presentation of this disease and its rarity makes the recognition difficult. First line therapy for ATLL involves chemotherapy with CHOP; additional agents may include NF-kB or proteasome inhibitors, Arsenic trioxide, monoclonal antibodies, ultimately leading to stem cell transplant in those who respond to treatment. The median
survival is 4 to 60 months, with treated acute leukemia carrying the worst and smoldering type carrying the most favorable prognosis.

This case illustrates the importance of considering ATLL in patients who present with abdominal pain with associated lymphadenopathy, papular rash, lymphocytosis and hypercalcemia, which do not respond to standard treatment. Due to decreased cellular immunity, these patients are more susceptible to complications with opportunistic infections such as CMV and BK, as illustrated in this case.
Abstract:
Recurrent meningitis is defined as having two or more separate episodes of meningitis with full recovery. Typical causes include congenital conditions, anatomical abnormalities, asplenism, immunodeficiencies and chronic infections. While identifying a pathogen is crucial for management and tailored therapy, a recurrence in meningitis requires a detailed history and further workup.

A 35-year-old female with no PMH presented to the emergency department with sudden onset of headache, photophobia, fever and chills. She had a similar episode a few years prior where she was diagnosed with bacterial meningitis. Physical exam demonstrated a temperature of 102 Fahrenheit, the patient was restless, in acute distress, and nuchal rigidity was present. The remainder of her physical exam was unremarkable. A computed tomography (CT) scan without infusion of the head was done, and a lumbar puncture was performed. Cerebrospinal fluid studies demonstrated a glucose of two, protein of 197, white count of 5750, negative HSV PCR, and gram stain demonstrated gram positive cocci in pairs and chains consistent with bacterial meningitis. The patient was started on intravenous antibiotics, steroids and a marked improvement in her symptoms was observed. Further laboratory work ruled out immunologic deficiencies, and complement levels were normal. The patient denies any history of head trauma, or ear trauma, or any congenital anomalies. However, the patient mentions she has been experiencing clear rhinorrhea for four years that did not resolve with over the counter therapy or courses of antibiotics. A more detailed review of head imaging demonstrated a small bony defect seen within the superior wall of right ethmoid and frontal sinus, consistent with an ethmoid fracture. She mentions she had a history of complicated cesarean section four years ago, where a nasogastric tube was inserted (NGT). Two weeks after the NGT insertion the patient developed her first episode of bacterial meningitis.

NGT insertion is a relatively common practice in medicine, many physicians and nurses are unaware of potential complications. Inadvertent introduction of nasogastric tubes in the cranial cavity have been described, particularly in patients who have marked septal deviation, underdeveloped turbinates, and a high-grade pneumatization of the paranasal sinuses. After complete resolution of symptoms, the patient was referred for neurosurgical evaluation. This case also highlights means of preventative medicine; in which patients with CSF leaks should receive pneumococcal vaccinations.
Title: Diabetic Ketoacidosis from Pembrolizumab in a Patient with Malignant Melanoma

Abstract:
INTRODUCTION
Since its approval by the Food and Drug Administration, programmed cell death protein 1 (PD-1)/programmed cell death ligand 1 (PD-L1) inhibitors such as Pembrolizumab have been used in different malignancies. Given the novelty of the drug, many of its side effects are yet to be discovered. A rare toxicity of this drug class is autoimmune diabetes which oftentimes present as Diabetic Ketoacidosis (DKA).

CASE
We present a case of an 80-year-old female with past medical history of malignant melanoma (s/p excision and radiotherapy, currently on Pembrolizumab), hyperlipidemia, osteoarthritis and GERD who came in with altered mental status, nausea and vomiting. For her malignant melanoma, she was given her Cycle 2 of Pembrolizumab two weeks prior to presentation. Patient was doing well until she developed symptoms three days before presenting to the ED. Physical examination was significant for somnolence, tachycardia and diffuse abdominal tenderness to palpation. Pertinent laboratory findings include Glucose 1,399 mg/dL, pH 7.062, Bicarbonate 7 mmol/L, Anion Gap 41, Lactate 2.7 mmol/L, Ketones 7.1 mmol/L and Hemoglobin A1c 7.7%. Infectious work-up was negative. She was diagnosed with DKA and was started on IV fluids and insulin drip. Patient has no family history of diabetes mellitus. Further investigation identified high serum titers of anti-glutamic acid antibody and anti-islet cell antibody supporting an autoimmune etiology. C-peptide was low. Pembrolizumab was assumed to be the most likely cause of the DKA given the temporal relation and the absence of other risk factors. Ketones normalized and anion gap closed with above interventions. She was then transitioned to subcutaneous insulin. Patient eventually discharged with close follow-up with Oncology and Endocrinology.

DISCUSSION
To date, there are only a few cases reported in literature of autoimmune diabetes in patients getting Pembrolizumab for malignant melanoma. The onset of Type 1 DM varied greatly from 1 week to 12 months, with the majority of patients presenting with diabetic ketoacidosis. The PD-1/PD-L1 pathway is crucial for lymphocyte maintenance of self-tolerance. There is evidence showing a strong pathogenic link between Type 1 DM and inhibition of the PD-1/PD-L1 axis. Studies have also shown that blockade of PD-1 and PD-L1 accelerates the onset of autoimmune diabetes in non-obese diabetic mouse through the expansion of self-reactive T cells in the
pancreatic lymph nodes. The use of PD-1/PD-L1 inhibitors is expanding. This case is important as it reminds clinicians that although rare, DKA can be an adverse effect of these drugs. Awareness of this can help us monitor patients on these drugs more closely. It can also lead to earlier detection of DKA leading to prompt intervention and better outcomes for our patients.
Abstract:
A 32-year-old male presented with progressive shortness of breath and leg swelling. He has a past medical history significant for HIV disease, Renal Cell Carcinoma (RCC) s/p left nephrectomy and was recently found to have metastasis to his mediastinal lymph nodes and lung, which were detected 2 months prior to admission. The patient has been on salvage treatment with atezolizumab since the time of the metastatic diagnosis.

On admission, the patient’s physical exam was significant for tachycardia, respiratory distress, decreased breath sounds in the right hemithorax and lower extremity swelling. A Chest X-ray showed a right sided pleural effusion and an enlarged nodular density in the left upper lobe. Laboratory data revealed anemia and an elevated Troponin I. His viral load was undetectable and his CD4 count was 444 cells/mm³. Pleural fluid analysis showed atypical cells weakly positive for RCC marker. Transthoracic echocardiography revealed normal left ventricular function and a large mobile cystic mass in the left atrium. Transesophageal echocardiography revealed a large mass composed of solid and cystic components and a left atrial appendage thrombus. The solid component, a 5 x 2.3 cm mass, invaded the basal half of the interatrial septum and the cystic component, a 4 x 1.6 cm mass was found to be protruding to the left atrium. Multiple enhancing neoplastic masses in the muscular compartment in both calves were detected on MRI of the lower extremities.

Renal cell carcinoma (RCC) is the seventh most common cancer in the United States and causes approximately 13,000 deaths annually. RCC occurs in younger patients who are HIV positive and has unfavorable prognosis in HIV patients with advanced stage. Distant metastasis usually develops in the lung, bone and lymph nodes. Although tumor thrombus involving the renal vein and inferior vena cava (IVC) happen in up to 10 % of patients with RCC, there are rare reported cases of RCC metastasized to the heart without IVC involvement. The majority of these cases involved the lymph nodes before reaching the heart. Metastasis to the muscles is a rare phenomenon but has been reported in RCC. Metastatic RCC responds poorly to chemotherapy and most patients do not survive beyond 6 months of diagnosis. Here, we reported a solid cystic cardiac mass in a patient with an undetectable level of HIV viremia and RCC. This patient had been treated with atezolizumab prior to the presentation with disseminated metastasis. The metastatic mass was mobile in the left atrium with cystic and solid components. Unfortunately, our patient’s course ended with death, secondary to hemoptysis leading to respiratory failure a few days after admission to our hospital.
Abstract:
Biotin (Vitamin B7) is commonly used in hair, skin and nail supplements. For hair fall, Biotin use in high doses is becoming increasing popular. However, a lot of primary care physicians are unaware of its effects in high doses. Hereby we present an interesting case related to Biotin use.

A 67-year-old male was referred by his PCP to Endocrinologist for hypothyroidism management. Patient was diagnosed with Hypothyroidism by his PCP and was being treated with 25 mcg of levothyroxine for the last 2 years. When he was first diagnosed his TSH was elevated and free T4 was in the normal range. However, despite being on Levothyroxine therapy, his TSH kept increasing but interestingly his T4 was high too. Repeat labs showed normal T4, very high T3 and mildly high TSH. After digging deeper into his medication history, it was revealed that he was taking very high doses of Biotin. He was asked to stop Biotin and repeat labs in 1 week. Repeat labs showed normal T4, T3 and mildly elevated TSH at 7. Levothyroxine was stopped. The problem had nothing to do with the patient’s thyroid: it was biotin that was interfering with the tests.

Institute of Medicine recommends a daily intake of 30 mcg of Biotin. But some patients are taking milligram amounts. Almost all immunoassays contain biotin because they rely on the biotin–streptavidin attraction to either anchor the assay’s antibodies to a capture surface or capture them once they have reacted with a patient sample. Large amounts of biotin in a patient sample can interfere with this process. However, the effects can be confusing because, depending on the particular assay, biotin can skew the results to be either falsely high or falsely low. In the case of competitive immunoassays, usually used for low molecular weight targets (such as T4, T3, and cortisol), biotin interference causes a falsely high result. In immunometric (sandwich) assays, it gives a falsely low result. Biotin can also cause thyrotropin receptor antibodies to be falsely positive, which could lead to a misdiagnosis of Graves’ disease. For the interference to occur, the patient’s biotin level needs to be at least three times the upper limit of the healthy adult reference range.

Close attention should be paid to OTC supplements as patients usually don’t mention them when medications are reviewed. The doses of these medications should be noted as well. If Biotin is suspected to be interfering with the results, it should be stopped and labs repeated in 1 week as Biotin is water soluble, so it washes out of the body quickly and even a single day can make a big difference in the test results.
Last Name: Flores  First Author: Resident

First Name: John  Category: Clinical Vignette

ACP Member: 1548654

Additional Authors: Suman Setty, MD, Esteban Cedillo-Couvert, MD, Yasmine Lame’, and Shiva Arami, MD

Title: A Case of Proliferative and Membranous Lupus Nephritis Complicated by Cryoglobulin-like Hyaline Thrombi

Abstract:
Introduction: Renal disease is a devastating complication of systemic lupus erythematosus (SLE). In addition to glomerular involvement, vascular disease is not uncommon. We present a case of lupus nephritis (LN) in the setting of thrombotic microangiopathy (TMA) with a rare instance of cryoglobulin-like thrombi.

Case: a 21-year-old woman with past medical history of SLE complicated by Class VI nephritis was admitted with worsening kidney function which showed a serum creatinine of 2.9 mg/dL from a baseline of <1.5. She reported shortness of breath, cough, and a headache. She endorsed compliance with all of her medications and did not notice any urine output changes. Blood pressure was 200/123. On exam malar rash, Cushingoid appearance, and hair loss was noted. Initially, serum creatinine trended to > 6, but over the next few days, downtrended. Renal biopsy was significant for evidence of class IV and V, diffuse proliferative and membranous lupus nephritis with glomerular capillary loop thrombi. Ultrastructurally, the thrombi appeared similar to those seen in cryoglobulinemia. However, serum cryoglobulins and hepatitis B and C viral panels were negative. Patient received one dose of cyclophosphamide and discharged on high dose steroids and hydroxychloroquine. Unfortunately, the patient’s renal function declined and is she is currently on hemodialysis.

Discussion: Lupus nephritis is primarily caused by anti-dsDNA immune complex deposition in the glomerular basement membrane and mesangium, leading to activation of the complement cascade, but is often multifactorial. Vascular involvement in LN is most often associated with immune complex deposition, TMA, vasculitis, atheroembolic disease or antiphospholipid antibodies. The majority of acute TMA cases are the result of fibrin thrombi, and there is scant literature documenting cryoglobulinemic thrombi. Cryoglobulin clot formation has primarily been documented in case reports with cryoglobulinemia associated with HCV or HBV infection. Mixed cryoglobulinemia may be complicated by glomerulonephritis and TMA, but only 6.4% of the time is it associated with rheumatologic disease. While rare, cryoglobulin clots may be present even with a negative serum cryoglobulin. Cryoglobulin deposition beneath renal vascular endothelium causes complement cascade activation, and a hypercoaguable state with fibrin clots, but rarely cryoglobulin clots. All patients with class IV and V LN are treated with aggressive antihypertensive, antiproteinuric, and statin-based, and induction immunosuppressive therapies with cyclophosphamide, followed up by 1-2 years of
maintenance therapy with mycophenolate-mofetil or azathioprine. Patients with confirmed essential mixed cryoglobulinemia may consider rituximab instead of cyclophosphamide, but our patient was treated according to standard Class IV recommendations.

Conclusion: This patient documents a rare case of acute lupus nephritis complicated by renal vascular involvement with cryoglobulin-like thrombi. It is important to keep a broad differential when an SLE patient presents in renal failure - atypical thrombotic microangiopathy may be present suggesting another concomitant infectious or autoimmune process.
Abstract:
Vascular occlusion serves as the underlying mechanism for the multitude of clinical manifestations of sickle cell disease, one of the most prevalent hemoglobinopathies worldwide. Recurrent painful episodes are the hallmark of this condition. Abdominal pain attributed to an acute painful episode can be indistinguishable from a separate and/or more serious intra-abdominal disease process, representing a significant diagnostic dilemma for clinicians.

A 28-year-old gentleman with a history of sickle cell disease (HbSS) complicated by multiple strokes necessitating prophylactic chronic exchange transfusion therapy presented to the emergency department with abdominal pain, back pain, and diarrhea. Of note, the patient was previously admitted to the hospital twice within the month for similar symptoms with an unrevealing work-up. On presentation, the patient was afebrile and hemodynamically stable, in mild discomfort but non-toxic, with normoactive bowel sounds and a soft, non-distended, diffusely tender abdomen most prominently in the left lower quadrant without guarding or rebound tenderness. Initial laboratory studies were significant for a white blood cell count of 22,500 with 90% neutrophils; otherwise, additional blood count and chemistry parameters were unchanged from the patient’s baseline studies. Intravenous fluid hydration and patient-controlled analgesia (PCA) was initiated. On hospital day 2, the patient developed two episodes of bright red blood per rectum, a first reported occurrence per the patient; rectal exam was unrevealing. Flexible sigmoidoscopy revealed friability, decreased vascular pattern, and ulcerated areas from the sigmoid to splenic flexure suggestive of ischemic colitis. Biopsies of the descending colon demonstrated focal acute inflammation, mucosal hemorrhage, fibrin and distorted crypts, also consistent with ischemia. The patient subsequently received an exchange transfusion with successful reduction of HgbS levels below 30% per hemoglobin fractionation studies. The patient’s hematochezia resolved and abdominal pain was well controlled on his home analgesics. He was discharged on day 6 with plans to increase exchange transfusion frequency from bimonthly to monthly.

Abdominal pain, comprising about 25% of acute pain episodes, is often ascribed to microvessel occlusion and infarcts of the mesentery and abdominal viscera. Progression to frank colonic ischemia despite adequate hydration and analgesia is exceedingly rare in sickle cell disease, owing to the significant collateral blood supply of the colon as well as the low degree of oxygen extraction by the bowel. Diagnosis of ischemic colitis in sickle cell disease is often obscured as typical signs of intestinal ischemia may be masked by opiate analgesia; patients
who do not respond to conservative measures or whose clinical course worsens should prompt further investigation. Once established via colonoscopy, treatment is aimed at optimizing colonic perfusion through hemodynamic monitoring, bowel rest, and in the case of sickle cell disease, exchange transfusion, which acutely lowers HgbS levels and thereby reduces blood viscosity, improving blood flow and tissue oxygenation.
Abstract:
Learning Objective 1: Neuropsychiatric manifestations in systemic lupus erythematosus (SLE) are common; however, psychosis per se is bit uncommon. They include cognitive deficit, lupus headache, psychoses, seizures, peripheral neuropathy, and cerebrovascular events.

Learning Objective 2: Psychiatric symptoms in SLE can be functionally independent psychiatric disorders. It can be due to drugs (steroids) used for SLE or secondary to SLE because of its brain involvement, which is termed as neuropsychiatric systemic lupus erythematosus (NPSLE)

Case: We had a 23-year young lady with history of SLE presented with change in behavior. She’s stopped talking since two days with continuous withdrawn behavior. No personal or family history of psychiatric disorders.
She was diagnosed with lupus 1 year ago treated with low dose prednisone and the dose was recently increased to 40mg.
On physical exam, she was pale and dehydrated. On mental status exam, she was noted to have anxiety, poverty of speech, delusions and auditory hallucinations. Labs were significant for pancytopenia, low complements and high ESR and CRP. MRI was unremarkable. Initially it was thought to be steroid-induced psychosis but on further questioning of the family, she was found to be non-adherent with her medications for the past 5 months. Based on this clinical presentation and high levels of antibodies to P ribosomal proteins a diagnosis of lupus psychosis was made.

Our patient was started on 2mg/kg IV steroids as well as Zyprexa 10mg qd. Significant improvement observed after 1 week of treatment. She became more conversant and interactive. Started eating and taking her medications as well. eventually she was discharged home on oral steroids and outpatient follow up.

Discussion: Case reports show that paranoia and psychosis may be found as co morbidity in patients of SLE, especially those who are partial responders to antipsychotics. These patients showed marked improvements on addition of steroids.

Lupus psychosis has to be differentiated from steroid-induced psychosis though it can be extremely challenging and the dilemma resides in whether to discontinue the steroids or actually give high-dose steroids.
Steroid dosage, time intervals, and the duration of mental changes may help in the differentiation. Brain MRI findings in neuropsychiatric lupus are frequently unremarkable, and abnormalities are nonspecific. No single laboratory test is sufficient to establish a definitive diagnosis of lupus psychosis is available at the present time. However, elevated levels of antibodies to P ribosomal proteins has been associated with neuropsychiatric lupus.
Title: Mistaken Identity: intraductal papillary mucinous neoplasia (IPMN) initially thought to be a pancreatic pseudocyst.

Abstract:
Introduction: We describe a case of pancreatic intraductal papillary mucinous neoplasia (IPMN) initially thought to be a pancreatic pseudocyst and we discuss biomarkers that may aid in risk stratification in the future.

Case: A 64-year-old man with a past medical history of alcohol dependence presented with epigastric tenderness and negative Murphy’s sign. Abdominal CT revealed lesions at the proximal pancreatic neck (3.1x3.2 cm) with upstream dilatation of the main pancreatic duct and fluid density in a side branch. EUS with FNA showed 3 cystic lesions located at the head and neck of pancreas in the main and branch ducts, with cystic CEA of 156, amylase of 38000, more consistent with cystic etiology. Cytology revealed rare glandular epithelial cells. MRCP demonstrated cystic communication with the main pancreatic duct. The overall clinical picture was thought secondary to pancreatic pseudocyst.

Five months later the patient presented with recurrence of symptoms and weight loss. Repeat abdominal CT showed a 5.9x5.8x5.7 cm pancreatic cyst with ductal dilatation. Heterogeneously attenuating lesions were noted in the spleen, and liver with omental caking. Repeat EUS with FNA showed a cystic thick-walled lesion in pancreatic head, with an unequivocal CEA of 15423. Cytology revealed hypocellular fluid without malignant cells. Repeat EUS was scheduled, however, the patient was lost to follow up.

Discussion: IPMN is the most common pre-malignant mucinous cyst. Usually asymptomatic and incidentally discovered, IPMN is stratified for malignant potential using imaging with surgical resection required for lesions involving the main pancreatic duct and having dysplastic histologic changes. Cystic CEA is used to differentiate serous and mucinous lesions (>200), but cannot accurately predict dysplasia. As such, the current models of assessing the malignant potential of IPMN are lacking.

In our patient, initial workup was consistent with pancreatic pseudocyst and annual surveillance was planned. Within one year, the patient decompensated clinically. Repeat imaging showed new distant metastases as well as a consolidated pancreatic structure. Patients with IPMN are at risk for developing pancreatic malignancy, one study reports a rate of 1%
yearly. We hypothesize the patient underwent rapid malignant transformation with abdominal metastases.

Recently validated biomarkers for improved risk stratification of IPMN include cystic VEGF and PGE-2. Elevated VEGF and CEA has been shown to differentiate between serous and mucinous pancreatic cysts with sensitivity and specificity >95%. Moreover, elevated PGE-2 and CEA levels have been shown to predict IPMN dysplasia with sensitivity and specificity of 78 and 100%, respectively.

The clinical utility of these assays may provide an avenue for IPMN risk stratification.

Conclusion: Together with imaging, biomarkers show potential in early IPMN detection and may improve clinical outcomes in patients with pancreatic lesions, such as the patient presented here.
INTRODUCTION: Blastomycosis, a close mimic of a multitude of diseases, is a pyogranulomatous infection primarily involving the lungs with pneumonia as its usual initial clinical presentation. Skin, bones and genitourinary systems are the most common sites of dissemination, while the thyroid is an unusual site of spread owing to its rich lymphatic supply and ability to generate hydrogen peroxide. As such, fungal thyroiditis is extremely rare especially in the absence of an immunocompromised state. We present a rare case of thyroid blastomycosis in an immunocompetent young male with disseminated blastomycosis.

CASE DESCRIPTION: A 29-year-old male with no significant past medical history presented with an enlarging thyroid nodule. Several weeks prior, he had also been experiencing dysphagia, fevers, chills, arthralgias, and had developed multiple exophytic, hyperpigmented, verrucous skin lesions. He had no history of recent travel and had lived in Chicago for the past 20 years. Thyroid ultrasound was done which showed a heterogeneous hypoechoic nodule within the midpole of the right thyroid lobe and an overlying heterogenous mass. Thyroid function tests were normal. Biopsy of the thyroid and overlying mass demonstrated broad based budding yeast consistent with blastomycosis. Skin biopsy from his right nasal ala showed similar yeast microscopically. Patient was then admitted for initiation of amphotericin B for disseminated blastomycosis. Further work-up with magnetic resonance imaging revealed extensive osteomyelitis of left knee and right hand and a large thoracic spine destructive mass. Hospital course was complicated by atrioventricular reentrant tachycardia and presumed blastomycotic pericardial effusion, improved on serial echocardiography after 4 days of amphotericin B. He was discharged on itraconazole to complete a 1-year treatment course. On his 2-month follow-up, there was continued improvement of rash and joint pain.

DISCUSSION: Fungal thyroiditis is a rare diagnosis, with Blastomyces only second to Aspergillus as an etiologic organism. Thyroid blastomycosis usually occurs in immunocompromised individuals, making this case in the immunocompetent one of the very few. Diagnosis is confirmed with tissue cultures growing broad based budding yeast. Duration of treatment depends on the extent of systemic involvement, which typically ranges at least 12 months. This case highlights the importance of an increased suspicion for thyroid blastomycosis in patients with risk factors for blastomycosis who present with thyroid mass or dysfunction. Moreover, it emphasizes how immunocompetence does not negate patients’ risk for this disease. Since timely diagnosis and initiation of treatment with itraconazole provide a 90-95% cure rate of symptoms, the physicians’ role in facilitating diagnosis is central in championing
against thyroid blastomycos
Title: Phytophotodermatitis: When Plants, Skin and Sunlight do not Mix.

Abstract:
Introduction: Photosensitivity disorders occur when the skin reacts to UV radiation exposure. Classes of photosensitivity disorders are divided based on etiology such as idiopathic, exogenous agents or genetics. Photosensitivity due to exogenous agents can elicit a toxic or an allergic cutaneous response after the agent is exposed to the skin. Phytophotodermatitis is a phytotoxic condition that occurs when cutaneous exposure to photosensitizing chemicals in plants is followed by exposure to UVA radiation.

Case Summary: A 30 year old man was admitted for left facial cellulitis. On the day of admission, patient presented with painful swelling of his left face with an overlying blister that had been present for the past two days. Patient also had single non-tender blisters formed on his hands and feet. The pain was constant, rated 5/10 on the pain scale, radiating down his left neck, and worse with chewing. Patient denied pruritus, sore throat, hoarseness or drooling. Prior to the swelling, the patient had been cooking outdoors and did not appreciate any trauma or insect bites. On exam, the patient was afebrile with poorly demarcated warmth, erythema and edema of left face with a honey-colored crust overlying the swelling and non-tender bullae on both hands and feet. CT was remarkable for left jaw face and upper neck cellulitis. Patient was admitted to the MICU for possible airway compromise and treated with IV steroids and subsequently left against medical advice. The next morning, patient presented again with progression of his symptoms to include blurry vision of his left eye. Repeat imaging revealed no involvement of the orbit. Dermatology evaluated the patient and believed the swelling and bullae were secondary to phytophotodermatitis. Infectious disease evaluated the patient and believed there to be a secondary streptococcus cellulitis infection. Patient was treated with IV vancomycin and piperacillin-tazobactam and transitioned to cephalexin as symptoms improved.

Discussion: We present an interesting presentation of phytophotodermatitis with an overlying streptococcus cellulitis. Phytophotodermatitis typically occurs after the skin is exposed to plants such as celery, wild parsnips and limes. These plants contain furocoumarins which are photosensitizing chemicals. Within 24 hours after exposure, patients typically present with painful, non-pruritic, erythema, edema and bullae in linear or bizarre patterns on sun-exposed skin that reflect the contact that they had with the plant. Treatment is symptomatic. Regarding this patient, his exposure to cutting limes prior to being outdoors was the trigger. The patient then developed an overlying streptococcus infection secondary to scratching and touching the swelling on his face with his hands. Awareness of phytophotodermatitis is important as it can
be mistaken for fungal infections or angioedema. Recognizing this benign condition can help to provide effective and efficient treatment and prevention from occurring again.
Abstract:
Introduction: Immunotherapies, like ipilimumab, are increasingly used as first line treatment for advanced melanoma. However, these treatments can cause highly morbid immune-mediated adverse reactions. Further investigation into the treatment of these adverse reactions is needed.

Case Description: An otherwise healthy 62 year old man with stage III melanoma presented with diffuse abdominal pain two days following an ipilimumab infusion. His pain was managed with opiates. On day five, patient was newly somnolent and hypoxemic thought secondary to opiate overdose and seemed improved after naloxone. He became ataxic and increasingly delusional over the following days despite weaning opiates and starting risperidone. MRIs of the brain and spine were negative for metastases. Lumbar puncture revealed elevated protein and lymphocytic pleiocytosis; empiric coverage for meningitis was started but stopped after bacterial cultures and viral PCR were negative. He was diagnosed with ipilimumab-induced encephalitis, and methylprednisolone was started at 2 mg/kg BID. The next day, he developed acute hypoxic respiratory failure due to aspiration pneumonia and required intubation. Course was complicated by persistent encephalopathy resulting in recurrent aspiration pneumonias and multiple intubations. His encephalopathy resolved after one month of steroids. He was discharged to rehab on prolonged prednisone taper. Unfortunately, he returned five weeks later with colonic perforation due to ipilimumab-induced colitis and underwent right hemicolecystectomy and diverting ileostomy. He has not received any further doses of ipilimumab or other immunotherapies.

Discussion: Melanoma is the most fatal form of skin cancer and the sixth most common cancer in the United States with 270% increase in incidence from 1973 to 2002. It is estimated 1 in 63 Americans will be diagnosed with melanoma during their lifetime. Most cases are detected at an early stage and can be cured by surgical removal of the lesion(s). However, with increasing lesion thickness and nodal involvement, long term survival decreases and systemic therapies are needed.

Ipilimumab is a recombinant IgG1 immunoglobulin monoclonal antibody, which blocks cytotoxic T-lymphocyte antigen 4 (CTLA-4). Blocking CTLA-4 results in increased proliferation and activation of T-cells, subsequently enhancing the immune system response to tumors like melanoma. Ipilimumab results in disease control at 6 months in 38% of patients and a median increase in overall survival of 9.6 months. However, adverse events include severe immune-mediated reactions of any organ system, most commonly the skin and GI tract. Encephalitis is a diagnosis of exclusion and reported in <1% of cases. Studies show one-third of patients...
receiving ipilimumab have severe (grade 3 to 5) adverse reactions requiring corticosteroid treatment. Steroids fail to improve symptoms in 1/3 of cases. In these instances, infliximab should be administered. It is unclear why infliximab was not given in this case and potentially contributed to increased morbidity.
 ***Abstract***

Introduction: Intussusception is a rare clinical condition in adults, and cases caused from lymphangiomas are rarer still. Most cases of lymphangioma present as cramping abdominal pain, abdominal fullness, or bleeding. However with larger lesions, patients can present with obstruction, volvulus, and intussusception.

Case: A 38-year-old woman presented to the Emergency Department (ED) with recurrent nausea, emesis, and lower abdominal pain. Her past medical history was notable for four prior episodes of intussusception at multiple regions of the gastrointestinal tract over the prior two months. Upon arrival, she was afebrile with a soft, non-peritonitic abdomen. Labs revealed a low albumin level of 2.8 g/dl. Radiographs of the abdomen demonstrated normal-appearing bowel loops, while contrast-enhanced computed tomography (CT) revealed a small bowel intussusception in the left mid abdomen. The patient underwent small bowel enteroscopy that revealed a submucosal 1.1 centimeter jejunal mass that was tattooed and biopsied. She was discharged with plans for video capsule endoscopy, however, returned to the ED two days later with recurrent nausea and emesis. Repeat CT imaging demonstrated new right upper quadrant intussusception, and she underwent surgical resection with primary anastomosis. She was discharged with improvement in her symptoms. Surgical biopsies returned positive for submucosal lymphangioma.

Discussion: Lymphangiomas are rare, benign tumors that arise from abnormalities of the lymphatic system. More often discovered in children, just 5% of lymphangiomas arise outside of the head and neck region, and less than 1% occur in the jejunum or ileum. The most common manifestations of intestinal lymphangiomas are non-specific, and thus these tumors are often overlooked in the workup for gastrointestinal complaints. Manifestations include intermittent abdominal pain, bleeding, abdominal fullness, or a protein losing enteropathy. However, in rare cases and with larger lymphangiomas, patients can develop more serious conditions such as obstruction, volvulus, and intussusception. Diagnosis can be assisted with the use barium enema, colonoscopy, and endoscopic ultrasonography. Lymphangiomas are characterized endoscopically as smooth, translucent lesions with a bluish or pinkish hue and with a pedunculated or broad base. The cushion-sign will often be positive. Treatment has historically been surgical, but the role of endoscopic resection has been rising in particular with small, pedunculated tumors. However, in cases of intussusception, the preferred treatment is still surgical as the rate of neoplasm is high.
Conclusion: Intussusception is a rare condition in adults, with 5 – 10% of all cases occurring in this population and the remaining 90 – 95% occurring in children. When intussusception does occur in adults, the likelihood of neoplasia is high. While lymphangiomas of the intestinal tract are uncommon, particularly in Western countries, the incidence has been increasing and these tumors remain an important part of a thorough differential.
Unexpected Aortic Dissection: Identifying Additional Clues to Diagnosis

Abstract:
Introduction: The classic presentation of tearing chest pain suggesting aortic dissection (AD) may be missed in an unresponsive patient. We present a case of AD incidentally discovered in a sedated and intubated patient, and discuss additional findings that may raise clinical suspicion.

Case: A 60-year-old male with history of hypertension and alcohol abuse presented to the hospital after heavily drinking. Initial labs were remarkable for elevated lactate and BNP. CXR demonstrated signs of vascular congestion, and transthoracic echocardiogram demonstrated a newly reduced LVEF of 30-35% with severely dilated LA and moderate aortic regurgitation. He experienced severe alcohol withdrawal and required intubation with sedation for airway protection. Vitals alternated between tachycardic with hypertension and bradycardic with hypotension, attributed to autonomic dysfunction secondary to delirium tremens. A DVT was identified by lower extremity ultrasound. CT chest was performed to rule out PE, which revealed an aortic aneurysm-dissection extending from the ascending to distal abdominal aorta, involving the aortic root and valve. He underwent emergent open repair of the aneurysm/dissection with CABG to the right coronary artery. Postoperatively, the patient was transitioned from IV to PO antihypertensives and was successfully extubated. Mental status improved and he was discharged to a rehabilitation facility.

Discussion: AD involves blood entering a tear between the aortic intima and media, which creates hemodynamic compromise by obstructing the true aortic lumen. Ascending AD, in particular, is associated with poor outcomes, as aortic root involvement can lead to valve insufficiency or compromise coronary flow, as in the patient described. Classically, AD presents with severe, tearing chest pain radiating to the back. In a recent retrospective study, however, 20% of patients diagnosed with AD did not endorse chest pain, and 6% of cases were painless. Furthermore, between 1996 and 2013, the frequency of the “tearing” descriptor has decreased from 64% to 24%. In an unresponsive patient, it is especially important to consider additional signs and symptoms, such as hypertension, diminished peripheral pulses, or variable systolic blood pressure (>20 mmHg) between upper extremities. A finding of pulse/BP differential is highly specific, with a positive likelihood ratio (LR+) of 46.7. Dissection involving the aortic valve may present with a diastolic murmur consistent with aortic regurgitation (LR+ = 1.67). Widened mediastinum on CXR can be helpful (LR+ = 3.4), however the frequency of normal CXR in documented cases of AD has increased. In the patient above, earlier suspicion of AD based on
alternating hypertension/hypotension coupled with aortic regurgitation as identified on echocardiogram may have prompted earlier imaging and diagnosis.

Conclusion: AD may not always be associated with chest pain. Heightened awareness of clinical signs including BP lability, diminished pulses, and aortic insufficiency may improve time to diagnosis and ultimately reduce mortality.
Abstract:
Sinistral (or left sided) portal hypertension is a rare, life-threatening cause of upper gastrointestinal variceal bleeding. It is often caused by splenic vein thrombosis. A 60-year-old male with a significant past medical history of an undifferentiated myeloproliferative disorder presented to the hospital with 3 days of melena and lightheadedness. Upon presentation, the patient was hemodynamically stable. Although the patient was previously diagnosed years ago with portal hypertension attributed to cirrhosis, physical exam lacked stigmata of chronic liver disease (i.e. jaundice, spider telangiectasias, caput medusa, etc.). His hemoglobin had dropped to 12 g/dL from 14 g/dL the year prior. His liver function tests were normal. A computed tomography exam of his abdomen with contrast showed a markedly dilated and tortuous splenic artery, an enlarged spleen measuring 27.8 centimeters, and marked dilation of the splenic vein. This imaging also showed multiple mesenteric collaterals arising from the splenic hilum and a few prominent varices near the gastroesophageal junction. The portal veins appeared patent without thrombus. While there was some hepatomegaly, no mention was made of typical cirrhotic changes of the liver. The patient underwent initial esophagogastroduodenoscopy (EGD) which showed large type 2 gastroesophageal varices (GOVs) along the fundus with evidence of recent bleeding. The patient was initially managed with intravenous proton pump inhibitor therapy, octreotide drip, ceftriaxone prophylaxis, and nil per os (NPO) status. He underwent pelvic angiography which revealed splenic vein opacification likely due to chronic splenic vein thrombosis. The angiography was also notable for hepatopedal flow via known varices with opacification of the portal vein further ruling out portal vein thrombosis. Despite therapy with proton pump inhibitor and octreotide, the patient continued to have melena and his hemoglobin dropped to 9 g/dL. He underwent repeat EGD which again revealed type 2 GOVs extending along the fundus and gastric body. Cyanoacrylate was successfully injected to one bleeding varix. After this EGD, the patient was monitored for 3 days on octreotide therapy and he no longer had melena. A trans venous liver biopsy was performed, and his hepato-portal venous gradient was 1 mmHg speaking against portal hypertension from cirrhosis. Final results of the liver biopsy are pending. The patient has decided to pursue splenic artery embolization with interventional radiology for further treatment of gastric varices. If splenic artery embolization does not prevent further episodes of upper gastrointestinal bleeding, then splenectomy may be pursued. This case illustrates the importance of creating a differential not limited to one diagnosis. Although portal hypertension is most often caused by cirrhosis, if physical exam and investigative studies do not fit the picture of cirrhosis, then sinistral portal hypertension must
be considered. Management of sinistral portal hypertension is drastically different & appropriate treatment can prevent death.
The Imitation Conundrum: Kaposi Sarcoma versus Bacillary Angiomatosis

Abstract:
Kaposi sarcoma (KS) is often clinically indistinguishable from bacillary angiomatosis (BA). Both KS and BA primarily affect the skin and mucous membranes with bone involvement occurring more commonly in BA. Bone involvement is rare in KS and usually occurs by invasion from adjacent skin lesions. Below is a challenging case of AIDS-related osseous KS presenting with skin lesions, lymphadenopathy and osteolytic bony lesions.

A 34-year-old African American male with AIDS (CD4 count 92cells/mm3, viral load 128,000 copies/ml) presented with a 2-day history of fever, chills and left leg pain. He has not been adherent to highly active antiretroviral therapy (HAART) for the past 4 weeks. Vitals were stable except for a temperature 39.3°C. Physical examination revealed multiple blackish scaly skin lesions on bilateral lower extremities with new onset bilateral nontender inguinal lymphadenopathy. CT abdomen and pelvis with contrast to evaluate lymphadenopathy revealed multiple new lytic lesions in the right pubic body, left ischium, bilateral iliac bones, left hemisacrum and L4 vertebral body with no overlying skin involvement. Azithromycin was started empirically for a presumptive diagnosis of BA. Bartonella henselae IgM/IgG, Bartonella quintana IgM/IgG and PCR were negative. Skin biopsy revealed epidermal hyperplasia with increased vascular proliferation positive for CD34 and HHV-8 stains. Left deep inguinal lymph node biopsy revealed spindle-shaped cells positive for HHV-8 and negative for bartonella, spirochetes and lymphoma. Although bone biopsy couldn’t be performed due to patient refusal, given skin and lymph node biopsies indicative of KS, HAART was resumed.

Kaposi’s sarcoma is an angioproliferative neoplasm associated with HHV-8 infection. AIDS-related KS with osseous involvement is rare with an unknown prevalence. Patients are mostly asymptomatic. It commonly involves the axial and/or maxillofacial skeleton. Radiologically, these lesions are mainly osteolytic with cortical osteolysis and infrequent periosteal reaction. Although osteolytic, they are not visualized by conventional plain radiography. MRI is preferred over CT to identify these lesions and has a high sensitivity for detection of marrow involvement and adjacent soft tissue masses. Bacillary angiomatosis is an important differential for osteolytic lesions in patients with HIV/ARDS. BA typically presents with systemic symptoms like fever, night sweats and weight loss. In BA, bone pain is more common with a predilection for distal long bones of lower extremities. BA lesions demonstrate an uptake on technetium-99m and gallium bone scans unlike KS. Biopsy is the gold standard for definitive diagnosis. On biopsy, spindle-shaped cells positive for CD34, CD31 and negative for Warthin-Starry stain are seen in
KS. As KS is a radiosensitive tumor, radiation therapy has been shown to provide palliation. The role of surgery, chemotherapy and radiation has yet to be determined and warrants further research.
Abstract:
Diverticulosis is a very common bowel disease and the prevalence is increasing. Complications like diverticulitis and diverticular bleeding appear in 10-15% of patients with diverticular disease. Genetics predispositions and medications such as nonsteroidal anti-inflammatory drugs (NSAIDs) remain a potential risk factor of developing diverticular disease. Foreign body (FB) digestion might also be one of the uncommon causes of diverticulitis. Most people who accidentally ingest FBs are unaware of the incident. It is also associated with alcohol and drug abuse, mental illness and retardation and denture-wearing elderly.

A 54-year-old male with a history of chronic complicated diverticular disease with bouts of acute diverticulitis presented abdominal pain and fever started a couple of years ago. He was initially treated conservatively with bowel rest and antibiotics. He presented again, a few days later, with lower abdominal pain, fevers, and nausea. At that time, imaging showed thickening of the descending and sigmoid colon consistent with acute diverticulitis. A colonoscopy was attempted but was aborted after an inflammatory mass in the sigmoid colon was impassable. The mass was biopsied and revealed an inflammatory process secondary to Actinomyces. A few weeks later, the patient underwent elective sigmoid resection for chronic diverticulitis and possible micro-perforation and abscess formation. The sigmoid colon along with the inflammatory mass was resected, with the creation of a primary colorectal anastomosis. The pathology report showed severe acute diverticulitis and a foreign body within the inflamed segment of sigmoid colon. The foreign body was a dental prosthesis that had lodged itself inside a diverticulum.

We are reporting this case to shed the light on FB ingestion as a rare, yet important, cause of diverticulitis. Even though complications such as hemorrhage, obstructions, perforations occasionally occur, most foreign bodies pass spontaneously without interventions, with only 10-20% requires endoscopic removal and around 1-14% (4.8%) need surgery for removal. However, having a predisposing factor such as intestinal stricture due to inflammation or cancer, or diverticular disease may increase the risk of obstructions and perforations. Most impactions and/or perforations occur in the terminal ilium or ileocecal and recto-sigmoid regions where changing in intestinal caliber happens. Often, foreign bodies are only discovered intraoperatively or in pathology, as in our case. Standard treatment was conducted in our case to treat the diverticulitis episode by bowel rest, antibiotics, then elective surgery.
Foreign body ingestion should be considered in patients with recurrent episodes of diverticulitis or presenting with a diverticular abscess. It is most likely that the FB ingestion and impaction was the trigger of the diverticulitis. To our knowledge, we are among the first to describe such case.
Risk of mechanical valve dysfunction is 0.4-1% per year. Mechanical aortic valves are most commonly complicated by pannus or thrombus formation accounting 10% and 12%, respectively. Here we report a rare case of mechanical aortic valve being complicated by a foreign body granuloma.

A 55 year old female presented to the ED with history of one episode of syncope. Her past medical history was significant for anti phospholipid lipid antibody syndrome (APLAS) with multiple miscarriages, Lupus valvulitis s/p AVR with St. Jude Mechanical valve 9 years ago, and hypothyroidism. She denied any associated symptoms of chest pain, palpitations, blurring of vision, or other neurologic deficits. On examination her vitals were significant for normal orthostatics and auscultatory findings significant for holosystolic murmur best heard in the aortic area. TTE showed aortic stenosis and TEE showed normal EF 55-65% with 6 x 6 mm fixed echogenic mass on posterior aspect of aortic valvular annulus with features of severe aortic stenosis of peak velocity of > 4m/s and valve area of < 1cm2. Valve cineflouroscopy showed one valve leaflet was immobile/frozen. Her h/o APLAS and prosthetic valve raised the suspicion of thrombus causing stenotic symptoms and was subsequently started on Alteplase with f/u TTE x 4 cycles, but the stenotic findings on TTE and video cineflouroscopy didn’t change, which raised the suspicion of pannus formation. Her h/o of adequate anticoagulation (average INR >2.5), unresponsiveness to Alteplase, immobile leaflet on video cineflouroscopy, and onset of symptoms years post valve replacement raised the suspicion of pannus formation. Hence cardiothoracic surgery was consulted for AVR and during surgery no thrombus or pannus was found, rather a shelf of subaortic hypertrophic tissue was found. The membrane along with aortic valve were replaced with biosynthetic pericardial valve. The pathology report of the specimen suggests fibrosis, neovascularization, and foreign body granuloma with sutures.

There are multiple ways by which pannus can be differentiated from thrombus and interestingly foreign body granuloma has a similar presentation as of pannus. Barbeteas et al showed time for valve replacement to reoperation was 62± 57 months for thrombus and 178± 52 months for pannus (p=0.006). Patients with thrombus formation had a lower rate of adequate anticoagulation (21% vs 89%, p=0.028). Abnormal valve motion was detected by TEE in all cases with thrombus formation but in 60% with pannus (p=0.0198) and pannus formation is more common in aortic position (70% vs 21%, p=0.035). Our patient presenting after 9 years
of AVR, adequate anticoagulation, and immobile aortic valve leaflet points towards pannus but in reality it was foreign body granuloma, though the management for both is equivalent.
Can thyrotoxicosis cause acute reversible cardiomyopathy?

INTRODUCTION: Thyrotoxicosis mimics a state of sympathoadrenal excess and augments all the metabolic processes in the body. These effects are seen in many organ systems including the cardiovascular. Takotsubo cardiomyopathy is an acute, reversible, nonischemic cardiomyopathy triggered by stressful events. It is characterized by normal coronary arteries and a very distinctive left ventricular contraction pattern of apical or midventricular transient systolic dysfunction. Herein, we report a rare case of possible thyrotoxicosis induced Takotsubo cardiomyopathy.

CASE PRESENTATION: A 43-year-old female with type 2 diabetes mellitus and Graves disease with poor adherence to methimazole presented to the emergency room with two-day history of progressively worsening left-sided chest pain with radiation to the back and associated dyspnea, nausea and diaphoresis. On exam, the patient was afebrile, tachycardic (with rate of 122/min), hypertensive and tachypneic. Skin was warm to touch with diaphoresis. She had minimal proptosis, exothalmus and lid lag. Thyroid gland was significantly diffusely enlarged, with no bruit. Lungs were clear to auscultation. Cardiovascular exam was remarkable for tachycardia with grade 1 holosystolic murmur. Abdominal exam was benign. No tremor was present.

Chest x-ray showed minimal sub-segmental atelectasis of left lung base. Electrocardiogram showed sinus tachycardia with non-specific ST-T wave abnormalities inferolaterally and troponin was elevated to 0.7 ng/mL. TSH was found to be less than 0.03 IU, freeT4 of 4.02ng/dL, freeT3 of 6.33ng/dL and thyroid peroxidase antibody 38 IU/ml. Patient was admitted with diagnosis of acute coronary syndrome and symptoms of uncontrolled hyperthyroidism due to non-compliance. Treatment for NSTEMI was initiated with aspirin, clopidogrel, low molecular weight heparin, and propranolol resulting in resolution of chest pain and decrease in troponin level. Patient was also treated with propylthiouracil (PTU) and hydrocortisone. Considering the severity of cardiovascular presentation, PTU was chosen over methimazole because of its additional effect on suppressing peripheral conversion of thyroid hormone. Echocardiography showed dyskinesia of left ventricular apical segments encompassing more than one coronary artery territory. Coronary angiography showed only mild nonconstructive coronary artery disease. Five days after initiation of therapy patient felt well with resolution of tachycardia and repeat echocardiography showed normalization of systolic function. She was discharged home with close outpatient follow-up.
DISCUSSION: Based on clinical presentation, transient troponin elevation, EKG and typical echo findings as well as absence of significant coronary disease we made the diagnosis of thyrotoxicosis associated Takotsubo cardiomyopathy. Takotsubo cardiomyopathy is a rare presentation of thyrotoxicosis with few cases described in the literature. Patients can present with signs and symptoms that mimic myocardial infarction and distinctive wall motion abnormalities on echocardiogram. Left ventricular dysfunction usually improves to normal levels within 1-4 weeks of initiating treatment. In our case of thyrotoxicosis induced cardiomyopathy, we observed the recovery of systolic dysfunction within 1 week.
A unique case of Acute Decompensated Heart Failure

Abstract:
Introduction: Acute decompensated heart failure (ADHF) is a clinical syndrome consisting of volume overload in the setting of ventricular dysfunction and may be complicated by end organ dysfunction. Determining the etiology of new-onset heart failure is key to determining the best treatment approach to prevent future decompensations.

Case: A 30-year old male presented to the University of Illinois Hospital with a 1-day history of chest pain, shortness of breath, headaches, and two episodes of syncope over the past 6 months. Four months before admission, he was diagnosed with anxiety disorder and prescribed propranolol after reporting multiple episodes of palpitations. On presentation his blood pressure was 196/89, pulse 113, respiratory rate 24, and he was saturating 82% on room air. He was noted to have jugular venous distension, lower extremity edema and an S3 gallop. Electrocardiogram showed sinus tachycardia without ST segment elevation Initial troponin was 20.69 (normal < 0.06). Echocardiography showed biventricular systolic dysfunction with a left ventricular ejection fraction of 20%. Left heart catheterization showed normal coronary arteries. Invasive hemodynamics with a right heart catheterization showed biventricular pressure overload and low cardiac index. Following diuresis and initiation of an ACE inhibitor, the patient’s blood pressure and clinical exam improved, and he was started on a beta blocker. Cardiac MRI 5 days after his initial presentation showed a recovered ejection fraction of 55% without evidence of myocardial scarring. A few days after discharge, urinary metanephrines returned highly elevated, and an abdominal MRI showed a 5.7 x 4.7cm mass consistent with a pheochromocytoma. He subsequently underwent a successful right adrenalectomy and has been doing well for the past year.

Discussion: Pheochromocytoma can cause catecholamine-induced cardiomyopathy and should be considered in previously-healthy patients who present with hypertension and new-onset ADHF. Most patients present with the classic triad of headaches, palpitations, and hypertension, however some patients can present with signs and symptoms of acute coronary syndrome. It is estimated that up to 10% of patients present with cardiomyopathy and cardiogenic shock similar to Takotsubo’s cardiomyopathy (TTC), which accounts for 0.7 - 2.5% of suspected acute coronary syndrome presentations and is thought to be the result of excessive sympathetic stimulation that induces coronary vasospasm. Because it is a rare clinical entity, the etiology of pheochromocytoma-induced cardiomyopathy is unknown, however several theories exist. First, cAMP mediated calcium overload during catecholamine surge has been implicated with direct myocyte toxicity. Also, catecholamines may result in the production of oxygen-derived free radicals. Finally, catecholamine-induced apical outpouching can result in
midventricular obstruction leading to reduced cardiac output. Similar to TTC, patients with pheochromocytoma-induced cardiomyopathy can experience rapid improvement of cardiac function with medical therapy, however patients in cardiogenic shock may require mechanical support.
Primary Pulmonary Kaposi Sarcoma (KS) in a Newly Diagnosed Cisgender Heterosexual HIV Patient Without Cutaneous Manifestations

Introduction:
Although AIDS-related KS is a well described and recognized disease, visceral involvement is rare.

Case description:
A 24-year-old man presented to the emergency department with one month history of pleuritic chest pain, shortness of breath, and dry cough. Three weeks prior to admission he was diagnosed with HIV-1 infection. He had missed his scheduled follow up visit and was not initiated on antiretroviral therapy. Review of systems was significant for a 15 pound weight loss over 3 months. He denied fever and night sweats. Physical examination revealed non-tender cervical lymphadenopathy and bilateral rhonchi at lung bases. There were no skin findings on initial presentation. Laboratory results were significant for WBC 3200/mm3 with 13% lymphocytes, hemoglobin 11.6 g/dL, LDH 272 U/L, creatinine 1.9 mg/dL, CD4 count 54/mm3, and HIV viral load 334991 copies/mL. CXR showed coarse bilateral lower lung interstitial infiltrates and mild perihilar infiltrates. He was admitted with a suspected HIV related respiratory disease and started on Pneumocystis jirovecii (PJP) and Mycobacterium avium complex (MAC) prophylaxis. An extensive workup was negative for tuberculosis, legionella, histoplasma, cryptococcus, and viral panel. Bronchial lavage was performed and silver stain was negative for PJP. Chest CT revealed diffuse irregular opacities with linear and nodular features and interlobular septal thickening. Bronchoscopy with bronchial lavage was done a second time, but the pathology and cultures were negative. On further examination he had developed a maculopapular rash on his left shoulder. Shoulder skin biopsy showed a dermal vascular tumor consistent with KS and positive for HHV-8 immunostain. On day 3 of hospitalization, his condition deteriorated. He developed fever, diarrhea, tachypnea, tachycardia and hypoxemia. His lung auscultatory findings worsened with diffuse bilateral crackles and rhonchi. CXR showed worsening pleural effusions. Patient underwent a video assisted thorascoscopic surgery, which showed vascular proliferations in the right middle lobe. Lung biopsy was positive for HHV-8. The diagnosis of respiratory AIDS related KS was made and treatment with antiretroviral therapy (ART) and chemotherapy with doxorubicin were initiated. His condition improved over 33 days of hospitalization and he was transferred to a chemotherapy facility. He received 6 rounds of chemotherapy over 4 months. He was seen at the Infectious Disease outpatient clinic three
months after diagnosis and was doing well with a CD4 count of 211/mm3 and a viral load of 431 copies/mL.

Discussion:
Pulmonary Kaposi sarcoma is a very rare presentation of AIDS related KS and can represent a diagnostic challenge as in our case. If there is lack of dermatological findings with nonspecific respiratory symptoms, then Pulmonary KS becomes a diagnosis of exclusion. Patients with mucocutaneous presentation of KS, low CD4 count and high viral load are usually at higher risk for pulmonary involvement.
Title: PROGRESSIVE MOTOR NEUROPATHY IN ABSENCE OF SENSORY DEFICIT: A RARE NEUROPATHY ASSOCIATED WITH ZOSTER

Abstract:
Introduction:
Motor weakness is relatively less common manifestation of neuropathy associated with zoster. The latency of onset of motor paralysis has been found to be up to 4 months from appearance of the rash, but more commonly 2-3 weeks. Motor weakness is usually associated with sensory deficit in respective dermatome. The pathogenesis of motor involvement has been linked to extension of inflammation from dorsal ganglia to the ventral horn. Complete recovery from weakness in most cases indicates radiculopathy rather than myelitis; the nerve conduction velocity studies have indicated radicular, plexus or peripheral nerve involvement.

Case:
78 year old woman presented with itchy and painful rashes over left arm and left upper back for 2 days. Medical history was significant for chronic lymphocytic leukemia (CLL), hypertension and history of stroke, without residual weakness. Medications included aspirin, hydrochlorothiazide and atenolol. Exam showed scattered, scaling and vesicular rashes with salmon colored base over left upper back extending to left deltoid area. Clinical diagnosis of Herpes zoster in the cervical (C6) distribution was made. Patient was discharged with a 7-day course of valacyclovir. 2 weeks later, she returned with constant pain in the area of distribution of rash. She had developed new ‘stiffness’ of left shoulder, with exam revealing inability to abduct beyond 15 degrees. There were few pink bumps in skin, but much of the lesions had resolved. She was prescribed valacyclovir for 7 more days, given persistent rash. After 6 weeks of initial presentation, she returned with progressively worsening weakness of left upper extremity, inability to abduct left shoulder, and new weakness in both flexor and extensor muscle groups across the elbow. Bicipital and supinator reflexes were absent on left upper extremity. Power and deep tendon reflexes were normal in other extremities. Sensation was intact in all extremities during all visits. Labs were only remarkable for a high white cell count (131,000 per microliter) associated with untreated CLL. Nerve conduction study revealed severe left cervical root lesion in the C5-C6 distribution.

Discussion:
Literature describing gradual progression of motor neuropathy, and in absence of sensory deficit, such as in this patient is limited. Also, there is lack of data regarding timeline in which stable weakness sets in. This case is an example that motor neuropathy secondary to zoster can present as progressive disease beyond one motor nerve root and over several weeks’ duration despite patient receiving appropriate treatment at onset. Due to high prevalence of zoster
among immunocompromised patients, presentation such as this raises insight into consideration of zoster as a cause of progressive motor neuropathy, especially in such population. It also opens door to further classification of zoster associated neuropathy and identify them in a timely manner.
A case of severe acute pancreatitis complicated by abdominal compartment syndrome due to tamoxifen-induced hypertriglyceridemia

Abstract:
Introduction: About 20% of patients with acute pancreatitis have a complicated course with development of persistent organ failure. While hypertriglyceridemia (HTG) is a known cause of acute pancreatitis, acute pancreatitis due to tamoxifen-induced HTG is rare.

Case report: 57-year-old woman presented to the emergency room with a sudden onset of severe abdominal pain and nausea with bilious vomiting. Patient had a history of hypertension, and breast intraductal hyperplasia treated with lumpectomy almost 2 years ago. Her medication list included atenolol, losartan, atorvastatin, and tamoxifen. No history of alcohol drinking. On physical exam, patient was in moderate distress due to pain, normotensive but tachycardic (105/min), tachypneic (20/min) with normal oxygen saturation. Abdomen was distended with right upper quadrant tenderness and voluntary guarding, and no rebound tenderness. Laboratory findings revealed neutrophilic leukocytosis (13,500/mm3), lipase 2,600 U/l, sodium 128 mEq/L, triglycerides (TG) of 3,905 mg/dl, glucose 144 mg/dl, calcium 8.8 mg/dl. Records from 4 years prior to admission revealed TG of 120 mg/dL. Abdominal CT showed edematous pancreas without apparent necrosis, gallstones, or free fluid. Patient was admitted to ICU and supportive care started, despite initial low Ranson and BISAP scores. Patient’s condition deteriorated rapidly, calculated Ranson at 48 hours was 7, consistent with a poor prognosis. ICU course was complicated by ARDS requiring mechanical ventilation, shock requiring multiple vasopressors, acute kidney injury (AKI) requiring hemodialysis; and abdominal compartment syndrome which was diagnosed based on high pre-test probability and high bladder pressure measurements and was treated conservatively. Patient required tracheostomy and gastrostomy. HTG was successfully treated with insulin drip: in 72 hours TG was below 500 mg/dl. Over 2 weeks, patient’s condition somewhat improved: abdominal compartment syndrome resolved, she followed commands and participated in physical therapy. Unfortunately, kidney function did not recover and she suffered from ICU myopathy. She was enrolled into acute rehabilitation.

Discussion: In this report, we identified multiple learning objectives: 1) abdominal compartment syndrome indicates poor prognosis with 70-100% mortality rates; 2) the risk of pancreatitis is up to 20% with TG > 2000 mg/dL; 3) multiple initial scoring systems (clinical and radiographic) are used to predict the severity of the pancreatitis but could be misleading if low; 4) tamoxifen is known to raise the serum TG level. Few cases of tamoxifen induced HTG leading to acute pancreatitis have been reported. Similar to our case, four women with no history of previous hypertriglyceridemia experienced pancreatitis associated with tamoxifen-induced
HTG. The time of onset of pancreatitis after tamoxifen exposure was quite long (at least a few months) in all cases. Clinicians should be aware of the risks of developing severe and even fatal acute pancreatitis with tamoxifen therapy and should consider periodic testing of lipid levels.
Title: A shadow from the past: Going back to the basics

Abstract:
An incidentally discovered lung mass can be catastrophic news to patients. Malignancy usually tops the list of differentials and tissue diagnosis remains the gold-standard. A thorough history, in rare circumstances, may spare the patient a biopsy.

An 86-year-old gentleman with a past medical history of COPD, pulmonary tuberculosis (TB) - diagnosed and treated in the 1950s - and localized prostate cancer presented to the hospital with a 3-day history of progressive dyspnea on exertion. He had no associated fever, productive cough, chills, or chest pain. On examination, he was tachypneic with bilateral wheezes. The rest of his exam was unremarkable. As part of the workup of his likely COPD exacerbation, a chest X-ray was obtained, revealing a huge, partially-calcified lung mass, measuring approximately 16.5 x 10 cm, that was projecting over the left lung apex and likely external to the lung parenchyma (figure 1). A chest CT-scan was then performed for further evaluation of the suspicious mass. This revealed a very large, peripherally-calcified soft tissue mass in the left upper chest wall measuring 9.4 x 11.8 x 14.7 cm, with destruction of adjacent left 1st-3rd ribs (figure 2). Differentials considered at this point included osteosarcoma, metastatic prostate cancer, and mesothelioma, among many other possibilities. Tissue diagnosis was deemed necessary. However, before rushing to any invasive measures, we went back to the basics. Based on the patient's history, we asked ourselves a simple question: What were the treatment options for TB in the 1950s? Our patient had the answer.

The patient did recall he had underwent a “surgery that healed TB”. On further investigation, we learned that a historical procedure known as "Plombage” was used to treat cavitory TB of the upper lung lobe. It involved the insertion of an inert material (such as paraffin) into the pleural cavity, thereby actively forcing the diseased lung lobe to collapse and allowing rapid healing. It was practiced between the 1930s to the late 1950s. It became obsolete with the advent of potent antituberculous medications. Given the historical time-frame, it is not unlikely that we would encounter similar cases among elderly patients in today's practice. Although not free from complications, surgical management may be revisited with the rise of multi-drug resistance. This case demonstrates the value of a proper history and how it may significantly alter management. The huge mass discovered on our patient's imaging was merely a sequel of the Plombage procedure, and thus no longer required tissue biopsy or invasive measures. He
was reassured and grateful for the good news - it is not cancer, it is just battle scars from when you defeated TB!
Abstract:
Spontaneous atraumatic hematomas typically occur in the jejunum and rarely in the colon. A 71-year-old man with history of renal cell carcinoma, prostate adenocarcinoma, and chronic extensive aortic dissection presented with a 2-day history of right lower quadrant abdominal pain and hematochezia. The patient reported daily use of low-dose aspirin. He denied family history of colon cancer and bleeding disorders. On physical examination, the patient was afibrile and normotensive with mild tenderness to palpation in the right lower quadrant. Three large skin hematomas were noted. Digital rectal exam noted trace maroon-colored blood. Laboratory findings were notable for hemoglobin 8.9 g/dL decreased from baseline 12.0 g/dL, platelets 73 K/μL, International Normalized Ratio 2.8, partial thromboplastin time 32 seconds, hypofibrinogenemia (34 mg/dL), and elevated D-dimer (>20 μg/mL). Factor V, VII, and VIII levels were normal. Computed tomography angiography revealed a 12 cm segment of the ascending colon with marked edema and wall thickening with areas of mild hypervascularity. Colonoscopy revealed a smooth, blue-purple non-obstructing mass in the cecum and two smaller, similar masses on the ileocecal valve. Biopsies of the mucosa near the largest mass showed hemorrhage in the lamina propria with no thrombi or ischemic changes, consistent with colonic wall hematoma. A subsequent bone marrow biopsy demonstrated non-malignant hypercellular marrow consistent with peripheral destruction of red blood cells and platelets. Thus, the patient was diagnosed with chronic DIC secondary to shearing from the dissection, which subsequently caused the soft tissue and colonic hematomas.

Intramural hematoma is a rare condition. Most commonly found in the duodenum in children following blunt abdominal trauma due to the relatively fixed position of the duodenum anterior to the spine. Spontaneous atraumatic hematomas in the colon are thought to occur less frequently, which may be due to the taenia coli protecting against the expansion of the hemorrhage. Etiologies of atraumatic hematomas include vasculitis, hematologic malignancies, anticoagulation, and iatrogenesis. The consumptive coagulopathy was the culprit in this patient. Radiographic findings are variable including bowel thickening. Endoscopy often reveals a dark-colored submucosal mass. This case demonstrates the importance of considering intestinal intramural hematomas when a patient presents with abdominal pain, obstructive symptoms, and/or hematochezia, particularly in the setting of anticoagulation or bleeding diatheses. Timely diagnosis leads to the appropriate management, which is typically treatment of the underlying cause. Surgical intervention is required for uncontrolled hemorrhage or bowel obstruction. Recurrence is rare and anticoagulation can be safely resumed. The consumptive
coagulopathy in this patient was reversed with cryoprecipitate, and repeat imaging six weeks later revealed resolution of the colonic wall thickening and edema.
Abstract:
Acute urinary retention is a urologic emergency and is uncommon except in older males. Our case is unique as acute urinary retention provided an important clue to diagnose Meningitis Retention Syndrome (MRS) in a young healthy adult with subtle symptoms.

A previously healthy 20-year-old male presented with sudden onset urinary retention. He reported mild headaches, fevers, fatigue later followed by acute urinary retention associated with lower abdominal and back pain. Last void was over 24 hours ago. Vitals were stable except for a temperature of 39.9°C. No cervical neck stiffness, meningeal irritation or focal neurologic deficits on physical examination. Rectal tone and prostate examinations were normal with no sensory loss in perineum. Foley catheter was inserted with >1L of clear urine output with resolution of abdominal and back pain. CT head, abdomen and pelvis were unremarkable. Due to acute urinary retention with unexplained fevers and headaches, a lumbar puncture was performed, revealing nucleated cell count of 27/mm3 with monocytic pleocytosis, mildly decreased glucose level of 67mg/dl (serum glucose 198mg/dl), and mildly elevated protein of 71mg/dl. CSF HSV, VZV, EBV, CMV, enterococcus and HTLV were negative with no bacteria identified. Given abnormal CSF analysis suggestive of aseptic meningitis, MRI brain and lumbar spine were performed to rule out Acute Disseminated Encephalomyelitis (ADEM) with unremarkable results. He was started empirically on dexamethasone, vancoymcin, acyclovir, ampicillin and ceftriaxone. He remained afebrile without identifiable source of infection hence antibiotics and antivirals were discontinued. Despite multiple voiding trials, he continued to have high post void residuals and was discharged on clean intermittent catheterization and tamsulosin.

Meningitis Retention Syndrome is a combination of aseptic meningitis and acute urinary retention. MRS is a rare disorder with an unknown prevalence, mainly caused by viruses with the reported exception of Neisseria meningitidis and Listeria monocytogenes. Patients typically present with headaches, fever, stiff neck and urinary retention. The diagnosis of MRS is challenging and is often confused with ADEM, which typically presents after vaccination or exanthematous infections along with signs of encephalitis, myelitis, including brain and spinal cord lesions. MRS is considered to be a mild variant of ADEM with selective lower urinary tract involvement usually of 2-10 weeks’ duration. Urodynamic studies demonstrate an acute shock phase of detrusor areflexia with an unrelaxing sphincter, followed by a phase of detrusor overactivity suggesting an upper motor neuron etiology. MRS is a benign, self-limiting disease,
diagnosed by exclusion and has a very good prognosis. Early management of acute urinary retention by clean intermittent catheterization prevents bladder injury from over distention. The role of steroids is still unclear and warrants further research.
Title: Subacute combined degeneration with normal vitamin B12 level – finding an under-recognized cause in gastric bypass surgery patient

Abstract:
Introduction
General neurological symptoms including ataxia, paresthesia, and weakness carry a wide differential. Various nutrient deficiencies should be strongly considered in the setting of gastric bypass.

Case description
A 51 year-old female with history of morbid obesity status post sleeve gastrectomy one year prior presented to the emergency department due to frequent vomiting, pre-syncope on standing, neuropathic lower extremity pain, impaired balance, and lower extremity weakness, all of which progressed over months. Examination demonstrated orthostatic hypotension, diffuse weakness limiting ambulation, brisk reflexes in the upper extremities and absent reflexes in the lower extremities, ataxia, positive Romberg sign, and vibratory loss to the bilateral knees sparing pin and temperature sensation.

Glycosylated hemoglobin, thyroid function tests, vitamin B12, methylmalonic acid, fluorescent treponemal antibody absorption, HIV, hepatitis B and C, zinc, and thiamine were normal or negative. Copper was low at 43 mcg/dL (normal 70-175 mcg/dL). Electromyogram and nerve conduction studies demonstrated a length-dependent polyneuropathy without myopathy. MRI of the cervical spine was normal. EGD demonstrated an ulcer in the gastric antrum with severe edema causing gastric outlet obstruction. Biopsy of the ulcer showed no evidence of H.pylori or malignancy.

She was treated with proton pump inhibitor therapy with resolution of vomiting and normalization of oral intake over days. Orthostatic hypotension and weakness did not respond to boluses of IV fluids, supporting a neurological etiology. She was then treated with IV copper, fludrocortisone, and compression stockings. After 2 mg per day of IV copper for 10 days serum copper increased to 62 mcg/dL. Orthostatic hypotension resolved and weakness improved to allow ambulation with assistance. Peripheral neuropathic pain improved following copper repletion and the addition of gabapentin. She was discharged on 2 mg per day of oral copper.

Discussion
The patient’s final diagnosis was copper deficiency myeloneuropathy caused by poor oral intake from gastric outlet obstruction and malabsorption following gastric bypass surgery. In patients presenting with signs of the subacute combined degeneration, consider testing for copper deficiency with serum copper and ceruloplasmin levels, regardless of vitamin B12 sufficiency. The exact site of copper absorption is unclear, although likely occurs in the entire
upper gastrointestinal tract (1). Copper is a component of key electron transport enzymes responsible for the structure and function of the nervous system (1). Patients with prior gastric surgery, enteropathies, excessive zinc ingestion, and those receiving parenteral nutrition are at risk for acquired copper deficiency (1). Consideration and testing for copper deficiency can be critical as copper supplementation prevents further deterioration though improvement is variable (1).

References
A case of severe hyperemesis gravidarum complicated by cerebral venous thrombosis and a new diagnosis of myasthenia gravis

Introduction
Although nausea and vomiting during pregnancy is extremely common, hyperemesis gravidarum has been reported in the literature to occur in around 0.5% of all live births. Since the implementation of intravenous fluids the morbidity and mortality of hyperemesis gravidarum has improved; however, severe vitamin and electrolyte disturbances can still lead to significant complications for the fetus and the mother.

Case
A 23 year old female presented at 18 weeks and 6 days of pregnancy with persistent nausea and vomiting. She had a history of hyperemesis gravidarum in previous pregnancies and again throughout the first trimester of the current gestation. Despite her symptoms, she had delayed seeking medical care. She was found unresponsive by family members at home and was brought to the emergency department via emergency medical services. She had severe hypokalemia and hypomagnesemia with electrocardiographic changes, a urinary tract infection, and acute kidney injury with oliguria. After initial stabilization, she developed acute altered mental status, hypercapnic respiratory failure, and became obtunded. She was emergently intubated and initiated on dialysis. Magnetic resonance imaging showed a dural transverse venous sinus thrombosis and she was initiated on heparin therapy with improvement in mental status, and eventually extubated. On review of systems and records, the patient had evidence of a likely underlying neuromuscular disease. She ultimately had a positive serum titer for acetylcholine receptor antibodies and was diagnosed with myasthenia gravis. She improved with management and successfully delivered a healthy, full term baby girl.

Discussion
The complications of hyperemesis gravidarum have been well described. Of these, neurological complications are most frequently focused on the prevention of Wernicke’s encephalopathy with intravenous thiamine infusion. Although part of the differential diagnosis in any pregnant female with altered mental status, central venous thrombosis is three times more likely to occur in the post-partum period than during the pregnancy itself. The presentation is variable from headaches or vision changes to coma. The unique complicating factor for this patient was hypercapnic respiratory failure. This likely occurred due to both the acute development of cerebral venous sinus thrombosis along with her underlying myasthenia gravis that was yet to
be diagnosed. Myasthenia gravis is well known to worsen in early pregnancy, as was likely the case of this patient.

Conclusions
This patient presented with very severe complications of hyperemesis gravidarum and myasthenia gravis. Due to a delay in seeking care, she had severe dehydration, acute renal failure, and hypercoagulability. Ultimately, the etiologies of the patient’s presentation were managed and she successfully completed pregnancy without further complication.
Title: Seeing “Whispers” of People: A Case of Primary Central Nervous System Vasculitis

Abstract:
Case Description:
A 57 year old female with past medical history of right superior cerebellar artery aneurysm status post coiling and remote history of blastomycosis lung infection was transferred from an outside hospital for a 3 day history of confusion and hallucinations. Initially she noted worsening lumbar back pain and loss of balance, and then developed brief episodes of headaches and word-finding difficulties. On admission, she was afebrile and hemodynamically stable. Physical examination was notable for inappropriate affect and episodes of hallucinations. Labs revealed a mildly elevated erythrocyte sedimentation rate. Initial head computed tomography (CT) was negative for acute intracranial pathology. On CT angiogram (CTA) of head and neck, there was generalized thin caliber of intracranial vasculature suggestive of vasospasm. Magnetic Resonance Imaging (MRI) showed minimal T2/fluid-attenuated inversion recovery (FLAIR) hyperintensities within bilateral frontal periventricular white matter along with diminutive caliber of bilateral M1 and A1 segments. Diagnostic cerebral angiogram was consistent with multifocal areas of vasculopathic changes of large, medium and small vessels of anterior and posterior intracranial circulation. Lumbar puncture revealed results concerning for inflammatory etiology with normal cell count, protein 256, glucose 92. Serum and cerebrospinal fluid studies for infectious, malignant, and autoimmune pathologies were unrevealing. Given strong evidence for primary central nervous system vasculitis (PCNSV), a brain biopsy was indicated, however the family deferred biopsy. High dose pulse intravenous methylprednisolone was started, which resulted in resolution of her hallucinations and neurological findings.

Discussion:
PCNSV is a very rare form of vasculitis that is limited to the brain and spinal cord. Pathogenesis is poorly understood. The presentation can vary as there are no pathognomonic findings. Common symptoms are subacute headache, cognitive impairment, and/or stroke. As a result, it makes diagnosis challenging, as it is a diagnosis of exclusion. Cerebrospinal fluid analysis needs to exclude infectious or subarachnoid hemorrhage causes. Typical image findings on MRI or MRI angiography (MRA) or CTA can include diffuse, bilateral and alternating areas of stenosis and dilatation, often called “beading.” Brain biopsy is the gold standard, particularly if angiographic studies are unrevealing. Given the rarity of diagnosis there has not been a consensus for treatment. However early recognition and treatment of PCNSV prevents serious complications and poor outcomes. Proposed treatments include 1mg/kg/day prednisone.
monotherapy initially for small cortical and leptomeningeal vessels. High-dose intravenous methylprednisolone 1000mg daily for 3-5 days and cyclophosphamide are proposed for severe large/proximal vessel disease.

Conclusion:
The patient developed strokes prior to initiation of treatment thought to be a complication of her vasculitis, but fortunately, she did not have any permanent neurological damage that affected her functional performance. In conclusion, given the uncommon and non-specific presentation, it is important to diagnose and treat early to prevent complications of PCNSV.
A Case of Angioimmunoblastic T-Cell Lymphoma Presenting as Volume Overload

Introduction:
Angioimmunoblastic T-cell lymphoma (AITL) is an aggressive subtype of peripheral T-cell lymphoma. Its initial presentation is highly variable and rarely can feature signs of hypervolemia comparable to heart failure exacerbations rather than the classic B symptoms.

Case Description:
An 84-year-old man, with a history of hypertension, presented with 6 weeks of cough and dyspnea on exertion. He endorsed significant weight gain and denied constitutional symptoms. Of note, he reported being treated with steroids for mononucleosis a month ago. On exam, he had bivascular crackles and bilateral upper and lower extremity edema. Initial laboratory analysis was remarkable for Hb 9.6, Cr 2.3 (baseline ~1.0), albumin 3.2, total bilirubin 1.8, and direct bilirubin 0.3. Chest x-ray showed bilateral pleural effusions. He underwent a cardiac workup including BNP and echocardiogram, which were unremarkable. Subsequent CT abdomen found extensive axillary, hilar, mesenteric, and retroperitoneal lymphadenopathy, as well as splenomegaly to 18.4 cm. Axillary lymph node biopsy confirmed the diagnosis of EBV+ angioimmunoblastic T-cell lymphoma. Further laboratory workup was remarkable for LDH 497, undetectable haptoglobin, and positive Coombs. Uric acid was also elevated at 14.2.

This patient was treated with high dose steroids for autoimmune hemolytic anemia and with rasburicase for suspected tumor lysis syndrome. Given his poor functional status, he was unlikely to tolerate CHOP. He was thus tapered off steroids and enrolled in a phase II clinical trial of romidepsin and lenalidomide in peripheral T-cell lymphomas. His volume status improved with treatment of his lymphoma.

Discussion:
Angioimmunoblastic T-cell lymphoma is a rare and aggressive neoplasm of follicular T helper cells, accounting for just 1-2% of non-Hodgkin’s lymphomas. The median age at diagnosis is 65 years. AITL has a poor prognosis with a five-year survival rate of 30-35%. There is little consensus on the optimal initial treatment, but therapy often involves a CHOP-based regimen with auto-HCT for those who can tolerate it.

Most cases of AITL present in the advanced stages with generalized lymphadenopathy (76-95%), hepatosplenomegaly (50-70%), and B symptoms (70-85%). Less common manifestations
include skin rashes, ascites, pleural effusions, neurologic symptoms, and autoimmune phenomena such as hemolytic anemia. Acute kidney injury from spontaneous tumor lysis syndrome is rare and may be mistaken for cardiorenal syndrome when noted in the setting of fluid retention. Fluid retention in AITL can result from several mechanisms including lymphatic obstruction, malignant infiltration, and cytokine-mediated capillary leak. The low incidence of AITL combined with its variable clinical presentation can make early diagnosis challenging. This case highlights the need to keep an index of suspicion for lymphoma in presentations of rapidly progressive hypervolemia, particularly when the cardiac evaluation is normal, and even in the absence of B symptoms.
Abstract:
Introduction: In the United States, one-third of patients are exposed to heparin products while hospitalized. Heparin induced thrombocytopenia (HIT) is a drug reaction that can increase the risk of thrombotic events by 50% with a mortality rate of 20%. The incidence is low at 0.2%. When HIT is suspected, it is recommended to calculate a pre-test probability score, called the 4Ts. 4Ts is a well validated test with a high negative predictive value, therefore a score of 3 or less excludes HIT and no further testing is needed. In those with an intermediate or high score, HIT antibody testing can be considered. We believe at our institution, HIT antibody testing is done in many whose 4Ts score is low. An iFORM that allows for the calculation of the 4Ts was implemented in the electronic medical record system (EMR) to remind physicians when trying to order the HIT antibody test. We also reviewed the patient charges associated with the improper ordering of HIT antibody before and after the iFORM was implemented.

Methods: Retrospective chart review identifying patient that were admitted to our hospital from January 1, 2015 to December 31, 2016(pre-iFORM) and February 1, 2017 to June 30, 2017(post-iFORM) who had HIT antibody testing. The iFORM was implemented in January 2017. 4Ts were calculated by evaluating the timing and degree of thrombocytopenia, timing of the decrease in platelet count, presence of thrombosis, and other potential causes of thrombocytopenia. Patient costs associated with testing for HIT antibody were calculated.

Results: 57 patients were identified in the pre-intervention cohort of which 45(79% of patients) had a low 4Ts score. Total patient costs were laboratory testing was $43,542.45. 12 patient had intermediate and high score. Patient costs for those patient were $11,611.32. In the post-intervention group, 12(57% of patients) had a low score and patient charges totaled $11,611.32. 9 patients had a score in the intermediate and high score and patient charges totaled $8,708.49.

Conclusion: Prior to the iFORM, $31,931.13 were incurred due to improper testing. Post iFORM revealed that there is significant HIT antibody testing being done, despite a iFORM that guides the ordering physician to complete the 4Ts testing prior to proceeding with HIT antibody testing to avoid testing in those with low scores. These was a waste of $11,611.32 within 5 months. The average 4Ts score was 0.8 pre-intervention group and 3.09 in the post-intervention group. ABIM's Choosing Wisely Campaign recommends not to test or treat for suspected HIT in patient with a low pre-test probability of HIT. In our continued focus on high value care, we need to
continue to educate physicians on unnecessary testing and the downstream costs associated with it that burdens our already fragile healthcare system.
Title: Solitary cutaneous plasmacytoma an unusual diagnosis for a lesion on the lip

Abstract:
Background:
Primary cutaneous plasmacytomas are rare, and represent only 4% of all extramedullary plasmacytomas. Lesions involving the skin on the chest, back, and abdomen are more common, while the lips are unusual site.

Discussion:
To the best of our knowledge there are only four published case reports of solitary cutaneous plasmacytomas occurring on the lip. Our patient, a 65-year-old, gentle man presented to the clinic with complaints of a burning, non-healing ulcer on his lower lip. Based on his history of tobacco abuse, alcohol abuse and sun exposure he was at risk for squamous cell cancer but his biopsy results revealed findings that were consistent with an extramedullary plasmacytoma. He underwent an extensive work up, confirming that he had a solitary cutaneous plasmacytoma, and was treated with radiation therapy. Unlike previously published case reports our patient had lambda light chain type restriction on in situ hybridization, rather than kappa, and was treated with radiation therapy, rather than with surgical resection.

Conclusion:
Nine months later the patient did not have a reoccurrence of the lesion signs of transformation to multiple myeloma.
Title: Asymmetric Paralysis: Just another stroke? No.

Abstract:
West Nile Virus (WNV) disease is indigenous to Asia, Europe, Australia and Africa. It migrated to the Americas in 1999; by 2003 it had spread to 46 states. The primary reservoir of WNV is birds, and cycles via arthropod vectors; usually via mosquitoes. Risk factors include outdoor activity, contact with birds, and exposure to mosquitoes. Most people infected with WNV have a self-limited flu-like illness, characterized by fever, myalgias, headaches, gastrointestinal issues, and a maculopapular rash. Less than 1% of infected people develop West Nile Encephalitis (WNE); of those only 5-10% get poliomyelitis. Here we present a case of a patient who presented with symptoms mimicking a stroke who was found to have West Nile Encephalitis with poliomyelitis.

A 60-year- old Latino Male with no past medical history presented with altered mental status (AMS) and dizziness for one day, while driving. His only complaint prior was a few days of a bad headache. In the emergency department (ED), a CT of the head and abdomen was performed and was negative. He was sent home with close follow up. Later that same day he was found unresponsive on the floor and presented again to the ED. Only additional history noted was tobacco use, and smoking cocaine 1-2x a week. His physical exam was thought to be consistent with an ischemic stroke, with left sided weakness, hyporeflexia, dysphagia, and dysarthria; complicated by aspiration pneumonia. MRI however showed no acute ischemia, with abnormal signal intensity in the right periventricular region, with possible focal demyelination. EEG showed severe slowing and disorganization. Several days later he spiked a fever of 100.9, and had a WBC count of 20.6 with 5% bands. A lumbar puncture was performed. CSF results revealed protein of 208.6, LDH of 38, lymphocytes of 95%, and the West Nile IGG and IGM were positive. Patient was diagnosed with West Nile Encephalitis with poliomyelitis and antibiotics stopped and supportive care continued. Patient was extubated and was sent to a nursing home with a PEG tube with no improvement of weakness or AMS. One year later the patient is able to speak well and eat normally, but he still has the left sided flaccid paralysis which is slowly improving.

WNE, unlike other viral encephalitis, can present uniquely with muscular weakness, or poliomyelitis, which can mimic a stroke. WNV affects the lower motor neuron which causes asymmetric flaccid paralysis, and hyporeflexia with no sensory abnormalities. Age of 50 or greater has a higher risk of getting neurologic involvement in WNV disease. Patients with poliomyelitis usually require significant ICU care as with our patient. In patients such as ours
who have a significant risk for getting WNV, WNE with poliomyelitis should be in your differential diagnosis.
Title: Extramammary Paget disease with diffuse spine metastasis complicated by macroangiopathic hemolytic anemia, a case report.

Abstract:
Extramammary Paget disease (EMPD) is a rare oncologic entity, best described as adenocarcinoma of the apocrine glands. EMPD presents as a skin lesion, with some patients complaining of burning, pruritus, edema, irritation, pain or even bleeding. EMPD progresses slowly and due to nonspecific presentation, can present as advanced disease. This case of Right inguinal/Right scrotum EMPD with diffuse spine metastasis reveals the rare complication of macroangiopathic hemolytic anemia (MAHA) that may often be overlooked.

A 47-year-old Asian man presented with a groin lesion in 2011. Antifungals and vinegar were used without substantial improvement. Biopsy was performed in 2016 and revealed EMPD. Prior to scheduled excision, patient developed constant lower back pain with radiation to his legs. MRI showed compression vertebral fracture at L4 causing severe spinal stenosis with extension of tumor into ventral epidural space at T12. L4 decompression and L2-S1 fusion were performed. Unfortunately, treatment with conventional RT to T12 lesion (T11-L1) and SBRT to L4 lesion were not successful. Back pain progressed and repeat imaging showed epidural spread of tumor in the thoracic spine at the levels of T5-6 and T7-T10.

He was later admitted to the hospital due to dyspnea, fatigue, severe anemia with hemoglobin of 3.8 and thrombocytopenia of 12. Further testing was significant for ADAMTS13 activity 57/inhibitor 16, elevated absolute reticulocyte count, indirect hyperbilirubinemia and an elevated LDH. Peripheral blood smear showed a normochromic, normocytic anemia with schistocytes, polychromasia, and no spherocytes. Immature platelet fraction was elevated. Direct Coombs test was negative. Fibrinogen was 312. Other coagulation parameters were normal.

Patient was started on Prednisone, carboplatin and paclitaxel. After 2 cycles, repeat imaging demonstrated stable to improving disease. After an additional two cycles, new imaging studies demonstrated worsening metastatic burden within the liver. Considering this finding, chemotherapy was terminated. He was supportively managed with as needed blood products, but unfortunately passed away one month later.

MAHA in oncologic patients can be induced by cancer itself or by chemotherapy. In our patient MAHA most likely was induced by systemic microvascular metastases. In these cases, treating
underlying cancer is the only option for controlling MAHA. Multiple chemotherapy regimens are used in different centers and there is no gold standard treatment for metastatic EMPD. Thus, early diagnosis is crucial to prevent unfavorable outcomes.
Abstract:
Introduction: Tuberculous peritonitis (TbP) is an uncommon extrapulmonary site of infection with Mycobacterium tuberculosis. Ascitic fluid acid-fast bacillus (AFB) smears and cultures have an exceedingly low yield (<10% and <20%), yet studies show that adenosine deaminase (ADA) levels have 100% sensitivity when using cut-off values between 36-40 IU/L. We describe a patient with culture-proven TbP and repeatedly normal ADA levels and hypothesize as to why this occurred.

Case: An 87 y/o Chinese male patient presented with abdominal swelling for one week prior to admission. Patient denied alcohol abuse, fevers, chills, abdominal pain, nausea/vomiting, chest pain, weight loss, cough or sputum production. On exam, vitals were unremarkable. His abdomen appeared distended with shifting dullness, normal bowel sounds and no tenderness to palpation. Paracentesis and ascitic fluid analysis revealed WBC 1875 (59% lymphocytes, 4% neutrophils), LDH 260, total protein 4, and serum-ascites albumin gradient (SAAG) 0.7, suggesting an exudative process such as infection or malignancy. Cytology for malignancy and AFB smear and culture testing of ascitic fluid were negative, and adenosine deaminase (ADA) level was normal (6.6 IU/L). Blood work-up of patient showed Hgb: 7.8, WBC: 3.6 (absolute lymphocytes 0.8, absolute neutrophils 2.0, absolute monocytes 0.7), Platelets 55. Bone marrow biopsy showed a hypercellular marrow and myelodysplastic syndrome with excess blasts (17%). Patient was discharged without a clear diagnostic cause for ascites. A week later, patient was seen at clinic with recurrent ascites. A second paracentesis showed similar exudative characteristics, ADA was again normal (4.4 IU/L), but AFB culture on this specimen was positive. Patient was started on RIPE treatment and followed up with hematology/oncology for management of myelodysplastic syndrome.

Discussion: TbP is an extrapulmonary presentation seen in about 5% of pulmonary tuberculosis and usually results from reactivation of latent tuberculosis present in the peritonium, which was established through hematogenous spread coming from a primary lung focus. It has a ~50% mortality which can be lowered by early diagnosis. ADA is an enzyme that is disproportionately present in proliferating T lymphocytes, which are commonly present in serosal tuberculosis infections. It has a reported sensitivity of 100% in TbP when using a cut-off between 36-40 IU/L, but our patient’s ascitic fluid ADA was normal on two occasions in spite of AFB culture positivity on the second paracentesis. We hypothesize that the lymphocytopenia associated with his
myelodysplastic syndrome may have contributed to the unexpectedly normal ADA level in this TbP case.

Conclusion: This case demonstrates the uncommon presentation of TbP with a normal ascitic fluid ADA level. Although it is a highly sensitive test in most patients, caution should be used when interpreting it in patients with lymphocytopenia and should consider other diagnostic tests available to rule out TbP if clinical suspicion is high.
The Use of Beta Blockers in COPD patients on Long Term Oxygen Therapy

Abstract:
Chronic Obstructive Pulmonary Disease (COPD) is a major cause of morbidity and mortality in the United States. Patients with this disease suffer complications from tissue hypoxia and are at increased risk for a variety of hypoxia related comorbid medical conditions including heart disease in the form of coronary artery disease, atrial fibrillation, and congestive heart failure, and patients with these comorbid medical problems face higher mortality rates. However, there is controversy in the literature about the treatment of ischemic heart disease in patients with COPD. Prior studies for the use of beta blockers in patients with COPD including a meta-analysis by Etminan et al demonstrated an improved mortality rate in patients with the use of these medications, however confounding medical problems and their treatments in patients with COPD may make this data inaccurate, indeed subsequent studies as with those performed by Ekstrom et al demonstrated worsening mortality rates on patients using cardiovascular drugs in the setting of patients COPD on long term oxygen therapy, however to date no study has investigated the use of beta blockers in patients with COPD on long-term oxygen therapy.

This study is a randomized retrospective analysis of patients age 18-95 with a diagnosis of COPD on long term oxygen therapy defined as the need for oxygen supplementation due to a PaO2 of <55mmHg, (O2 sats <88%) on rest or ambulation, or patients with a PaO2 of 55-59 mmHg (O2 sats <89%) and who exhibit signs of tissue hypoxia between April 2009 to April 2015. This cohort of patients was divided into those who utilized any class of beta blocker (of 6-month duration or more) to those who did not and compares all-cause mortality in both groups as the primary outcome. Patient charts were reviewed to determine the use of and criteria for use of long term oxygen therapy, the use of beta blockers, pulmonary function test results, and the presence or absence of coronary artery disease, atrial fibrillation, and congestive heart failure. A sample size of 383 patients was calculated to produce a 95% confidence interval. This hypothesis was tested using the McNemar test with convexity correction at the 0.05 significance level.

The results of this study demonstrated no increase in all-cause mortality in patients who used beta blockers during the study interval time relative to those who did not when correcting for confounding variables from cardiac comorbidities. However, subgroup analysis determined that atrial fibrillation was an independent risk factor for mortality in patients with COPD (p < 0.05). As beta blockers are a treatment for atrial fibrillation, this suggests that atrial fibrillation is a confounding factor for mortality in patients with COPD on long term oxygen therapy.
A 74-year-old male with a history of vitiligo presented to an outside hospital with dyspnea on exertion, a 20 pound weight loss, and generalized fatigue for two months. His physical exam was notable for temporal wasting, scleral icterus and splenomegaly. On admission, abnormal labs included an ALT of 455 U/L and AST of 593 U/L as well as a leukocyte count of 1.3 thous/uL, Hgb 12.3 mg/dL, and platelets 83 thous/uL. Hepatitis viral serologies were unremarkable; however, serum autoimmune markers were elevated: ANA titer was 1:1280, Anti-Smooth muscle was 42 units, and serum IgG was 2639 mg/dL. A liver biopsy demonstrated autoimmune hepatitis (AIH) with Batts-Ludwig stage three fibrosis while a bone marrow biopsy was without malignancy. The bone marrow biopsy was complicated by persistent non-reabsorbing hematomas. He was started on 60 mg of prednisone for presumed AIH. After two weeks of treatment the patient began spiking fevers and was clinically worsening so he was transferred to our facility. Despite recurrent fevers, all cultures and tests for atypical organisms were negative. A repeat liver biopsy showed decreased inflammation while a repeat bone marrow biopsy showed evidence of hemophagocytosis. The patient was diagnosed with macrophage activation syndrome and he was started on high dose IV methylprednisolone and then transitioned to cyclosporine and prednisone. Despite immunosuppressant medications and supportive care with total parenteral nutrition, the patient developed multi-organ failure and died on day 30 of his hospitalization.

Macrophage activation syndrome (MAS) is the uncontrolled regulation of T and natural killer cells resulting in cytokine-mediated uncontrolled macrophage activation. While primary MAS is an X-linked recessive disease, type 2 MAS occurs in the setting of an underlying autoimmune disease or infection. Commonly associated autoimmune conditions include systemic Juvenile Idiopathic Arthritis, Adult Onset Still’s Disease, and Systemic Lupus Erythematosus. Given the rarity of the disease process, no accepted diagnostic guidelines exist for MAS. The only proposed diagnostic algorithm is based on a publication of 74 cases of MAS in the setting of various autoimmune diseases. Diagnosis relies on at least 2 of the following: leukopenia, thrombocytopenia, hypofibrinogenemia, elevated AST, CNS dysfunction, hemorrhages and hepatomegaly. Our patient fit six of the seven criteria. Patients with hepatic involvement tend to have poor outcomes as mortality rate has approached 75%. Patients with MAS are initially started on high dose steroids and in refractory cases, azathioprine and cyclosporine have been considered as second line therapy. We report a case of AIH leading to MAS, a rare etiology of an
even rarer disease. MAS should be considered in patients with severe refractory AIH as early treatment with high dose steroids and immunosuppression may lead to improved outcomes.
Title: Rare Organism Causing Gastric Outlet Obstruction

Abstract:
Sarcina is a gram positive anaerobic coccus that has been implicated in cases of gastric perforation, gastritis, and peritonitis and noted to occur in the background of gastric adenocarcinomas. Although numerous fatal cases have been attributed to it in veterinary literature, only a few human cases have been documented. We report a Sarcina-like organism isolated from upper gastrointestinal endoscopic biopsies in a patient with gastric outlet obstruction secondary to a pyloric channel ulcer.

A 23 year-old female presented with worsening burning epigastric pain, as well as nausea, and emesis for 1 week. Patient had a history of similar episodes in the past that were relieved with antacids, but no history of peptic ulcer disease. Liver function tests, gastrin, amylase and lipase were within normal limits. Right upper quadrant ultrasound showed cholelithiasis with no cholecystitis. Patient was admitted to general medicine floor and treated with IV pantoprazole with some relief of symptoms. Esophagastroduodenoscopy (EGD) showed a large pyloric channel ulcer causing deformity of the pylorus with outlet obstruction. Biopsy of gastric antrum showed chronic inflammation and Sarcina-like organisms in tetrads. Helicobacter pylori could not be ruled out. Patient was treated for Helicobacter pylori with metronidazole, clarithromycin, and PPI for two weeks. Repeat EGD was done 6 weeks later which showed pyloric channel stricture and resolution of ulcer. The stricture was dilated and biopsy of gastric antrum was taken which showed no Sarcina-like organisms or H. pylori.

The bacterium is known to cause deadly abomasal bloat in livestock. Of note Sarcina has been found in patients with gastric outlet obstruction or delayed gastric emptying with reports in the literature of increased risk of complications such as perforation with concurrent gastric ulcer. Any patient found to have Sarcina with gastric ulcer should be followed for potential life-threatening complications. Due to its association with delayed gastric emptying and gastric outlet obstruction such patients should also be evaluated for underlying etiology such as malignancy.
The efficacy of D-Dimer values in identifying venous thromboembolism in oncology patients

Abstract:
The risk of venous thromboembolism (VTE) is elevated in cancer patients. VTE is associated with significant morbidity and mortality. However, a universal thromboprophylaxis strategy in oncology patients is non-existent due to the questionable efficacy in the outpatient setting, unequal risk of VTE across different cancer types and the associated risks with anticoagulation therapy; such as bleeding. D-Dimer is used as an initial screening test in the emergency department to diagnose VTE in patients who have the associated signs or symptoms. However, there is little known regarding the diagnostic value of a D-Dimer test in cancer patients with clinically suspected DVT. Our group set out to demonstrate an additive or synergistic effect on D-Dimer level in those patients with VTE plus malignancy.

Clinical, laboratory and radiological data were retrospectively collected from the Jackson Park Hospital medical records. Records of ninety-seven patients were gathered from 2014 to 2016. These patients were assigned to one of three groups: 1) VTE alone (n=15), 2) malignancy alone (n=63) or 3) VTE plus malignancy (n=19). The diagnosis of VTE was confirmed by reported imaging studies (venous Doppler, CT angiogram or V/Q scan) and the diagnosis of cancer was established by the appropriate diagnostic criterion for the malignancy in question. Cancer patients were excluded if their disease was considered cured or in remission. The peak D-Dimer value was documented in patients with active malignancy within two days of radiological evidence of VTE. The mean D-dimer levels between all groups were compared using a single factor ANOVA to establish any significant difference between the means (P < 0.05). A Bonferroni corrected post-hoc two-sample T-test was employed to identify significant variations of the means between: 1) malignancy alone versus VTE alone, 2) malignancy alone versus VTE plus malignancy and 3) VTE alone versus VTE plus malignancy. An intergroup significant difference between the means was established if the T-test generated P-value exceeded the Bonferroni corrected P-value (P = 0.0167).

Our group found the mean D-dimer values for each group to be 7.69, 4.55 and 19.85 for the VTE alone, malignancy alone and VTE plus malignancy groups; respectively. The mean D-dimer value in the mixed VTE plus malignancy group was significantly elevated compared to the VTE alone and malignancy alone groups (p < 0.05). Based on these data, it appears oncology patients with VTE tend to have supraelevated D-Dimer values compared to a patient with each disease process in isolation. Future effort will be focused on establishing a meaningful D-dimer
cutoff value for oncology patients with suspected VTE. With this, it is our hope to reduce the time to diagnosis of VTE in cancer patients to subsequently reduce overall morbidity and mortality of delayed intervention in this population.
Abstract:
Purpose: Hypertension affects more than a quarter the world’s adults and is considered the leading cause of death globally. It is the most important risk factor for cardiovascular disease, and among the most common, affecting more than fifty million Americans. A wide body of research has demonstrated benefits to controlling BP to systolic levels of 139 or lower, including fewer strokes, long-term care placements, and deaths. However, only 50% of hypertensive patients have controlled BP.

Many interventions aimed at hypertension control have been implemented in recent years, spanning a broad spectrum of patient- and provider-centered methods. Despite a proliferation of interventions, less research has been done to elucidate patients’ opinions on factors impeding control of their hypertension or perceptions of proposed interventions. Patient viewpoints are likely to be beneficial in designing targeted interventions.

Methods: Fifty patients were recruited from a primary care practice within an urban Midwestern hospital. Inclusion criteria were age over 18; ability to read and speak; and a prior diagnosis of hypertension. Each patient completed a 2-page survey during an office visit. The survey utilized a 4-point Likert scale to determine what issues patients identify as major obstacles to controlling BP, as well as their assessment of how helpful each of 18 proposed interventions would be in achieving BP control. Obstacles and interventions were selected from three categories: medical, lifestyle, and social factors.

Results: The majority identified medical management issues as primary obstacles to BP control; 40% did not want to take daily medication, 28% did not want to take medication at all, and 10% reported forgetting to take medication or feeling too healthy for it. The other most frequently selected obstacles were stress (36%), not sure what foods to avoid (18%) or consume (16%), cost of healthy foods (16%) and medication (16%), tobacco (12%), and lack of support from family/friends (12%). Fewer than 10% identified inconvenience of making or keeping appointments with physicians, obtaining medication, or consumption of caffeine and alcohol.

Interventions most identified as likely to be helpful were nutritional counseling (42%), medication vouchers (28%), cooking or group exercise classes (22%), home monitoring (20%), mental health counseling (18%), support groups (18%), and time management workshops.
The least popular interventions were text-message reminders (12%), cell phone apps (6-10%) and counselor phone calls (2%).

Conclusions: Our data suggests that patients want to avoid taking medications, and instead hope to control BP through lifestyle modifications. Consideration of the discrepancy between identified obstacles and recommended interventions may provide a useful way forward in planning an intervention for this population. A support group approach combining education about medication with support in lifestyle changes could address reluctance to take medication and capitalize on eagerness to try lifestyle modification counseling.
Abstract:
We report a case of ischemic proctitis in a patient who presented with hematochezia and was found to have secondary amyloidosis. A 49 year-old male with a history of hypertension, chronic kidney disease, and cholecystitis s/p cholecystectomy presented with acute on chronic diffuse abdominal pain, persistent anorexia and rectal bleeding for two days. He also admitted to worsening malaise, nausea, vomiting, and an unintentional 50 pound weight loss. He was afebrile and normotensive. Laboratory studies showed a microcytic anemia, an isolated elevation of serum alkaline phosphatase, hypoalbuminemia, hyperglobulinemia, and an elevated gamma-glutamyltransferase. A CT scan revealed rectal wall thickening and perirectal fat infiltration. A colonoscopy showed distal proctitis with superficial erosions and ulcerations with pathology compatible with ischemic colitis. Due to the suspicion for infiltrative disease, Congo Red staining was requested. Biopsies were positive for amyloid deposition in the mucosal vessel walls and lamina propria. Subsequent staining of peri-gallbladder vessels from prior cholecystectomy confirmed amyloid deposition. A bone marrow biopsy revealed small amount of amyloid in the blood vessels. A renal biopsy confirmed secondary amyloidosis.

This unique case emphasizes the clinical importance of systemic diseases and their vague presentations. Amyloid deposition in the gallbladder is quite uncommon and has been reported in very few cases. Interestingly, this patient’s cholecystitis may have been triggered by unusual localization of amyloidosis. Amyloidosis can often present with misleading elements, such as abdominal pain, anemia, lower GI bleed, and weight loss. This wide spectrum of clinical manifestations can make amyloidosis a mimic for colorectal cancer. Given such a varied and ambiguous presentation of amyloidosis, further discussion is warranted for diagnostic methods for such an infiltrative disease. Needless to say, this case is also a testament to the timeless principle of forming an extensive differential diagnosis to avoid a missed or delayed treatment.
**Title:** Recurrent sigmoid colon diverticulitis complicated by inferior mesenteric vein thrombosis

**Abstract:**
We report a 48 year old male with a past history of diverticulitis who presented with fever for one week and mild abdominal pain. CT imaging of his abdomen confirmed recurrence of diverticulitis; it also showed filling defects in the inferior mesenteric vein and induration of the adjoining mesentery consistent with septic thrombophlebitis. Blood cultures were positive for E coli. Antibiotic therapy was started with IV cefoxitin and the patient was anticoagulated with enoxaparin and then transitioned to rivaroxaban. Surgery was consulted and recommended conservative management. The patient improved with these measures and was discharged on oral antibiotics and anticoagulation. He was seen by Surgery as an outpatient and is planned for elective colectomy.

Septic thrombophlebitis also known as pylephlebitis is an unusual but recognized complication of intra-abdominal infections such as diverticulitis and appendicitis. It is typically seen with severe sepsis and associated with a mortality approaching 30%. While antibiotics comprise the central pillar of medical management, the role of anticoagulation in these patients remains a matter of debate in part due to the absence of evidence that anticoagulation positively impacts mortality. In this instance, the extent of thrombosis, the presence of bacteremia and the absence of risk factors for bleeding tipped our judgement in favor of anticoagulation. The prevalence of diverticulitis in the US is already among the highest in the world and is expected to rise further as its population continues to age. Physicians caring for such patients should be aware of this uncommon complication and the potentially grave prognosis that it carries.
Title: Routine Screening Colonoscopy Yielding Anything but Routine Pathologic Findings: A Case Series

Abstract:
According to the American Cancer Society, only half of all at-risk populations receive appropriate colon cancer screening, leading to a delay in diagnosis and a worse prognosis in those who do not. We present a case series to emphasize the importance of screening and diagnostic colonoscopy in patients presenting with common gastrointestinal signs and symptoms but found to have uncommon underlying primary pathologic processes.

The first case is a 44 year-old African American female with family history of colorectal carcinoma who presented with 5 year intermittent rectal bleeding and bowel habit changes. On colonoscopy, she was found to have a large 5 cm pedunculated polyp. Upon excision, it was diagnosed on pathology as a submucosal cavernous hemangioma.

The second case is of an 80 year-old Caucasian male with non invasive colonic adenocarcinoma status post resection, who upon surveillance colonoscopy, was found to have a flat 1.1 cm polyp histologically diagnosed as mantle cell lymphoma, which represents less than 0.2% of colonic neoplasms.

Our third patient is a 53 year-old female with unremarkable colonoscopy except for hemorrhoids 1-year prior, presenting with abdominal pain and hematochezia for a 6-month period. She was found to have a 1.5 cm colonic polyp on repeat colonoscopy. Histologic diagnosis of malignant melanoma was made. She did not have any cutaneous or ocular lesions.

The fourth and most unexpected case was a 75 year-old Caucasian male with a history of internal hemorrhoids and chronic myelogenous leukemia who presented with abdominal pain, nausea, vomiting, diarrhea, and reduced appetite. His hemoglobin level continued to decrease, and an esophagogastroduodenoscopy was unrevealing. On colonoscopy, a 0.3 cm semi-pedunculated polyp in the distal sigmoid was found and later diagnosed as metastatic papillary renal cell carcinoma (RCC). This case represents a rare metastasis of a RCC to the GI tract manifesting as a colonic polyp.

Polyps are not necessarily colonic in origin, rather, they may be the first indication of metastatic disease or an uncommon location of a primary carcinoma. Under half of at risk
populations do not receive appropriate screening colonoscopies. In this era of preventative medicine, the importance of screening colonoscopies cannot be overstated. It is imperative that all patients are screened to ensure the early detection and prevention of cancer progression, even those pathologies which we may never expect to encounter. In this manner, we can optimize high-value and effective care for our patients.
Title: “Now You See It and Now You Don’t: A Case of Disappearing Toxic Epidermal Necrolysis after Cessation of Cefepime”

Abstract:
Toxic Epidermal Necrolysis (TEN) is a severe mucocutaneous reaction, characterized by extensive epidermal necrosis and desquamation involving greater than 30% of the body’s surface, often secondary to medication administration. TEN is a rare diagnosis with an estimated incidence of 1-2 cases per million people per year and a mortality rate of 30%. While several antibiotics, particularly sulfur and penicillin containing compounds, are recognized causes of TEN, cephalosporin induced TEN is rare. The first reported case of cefepime induced TEN was in 2015.

A 37 year old Hispanic man brought from nursing home (NH) with a past medical history of seizure disorder, TBI secondary to gunshot wound with ventilator dependence presented to the emergency department for hypotension and altered mental status. According to NH records, the patient was diagnosed with ventilator associated pneumonia five days prior to admission with tracheal aspirate cultures positive for pseudomonas aeruginosa. He was being treated with amikacin, vancomycin and cefepime. Patient was febrile, diaphoretic, less active than baseline, only responsive to pain stimuli and had a breakthrough seizure controlled on a stable dose of Keppra for 10 years. On admission, patient met 3/4 SIRS criteria with temperature 38.3°C, tachycardic to >100, and tachypneic to > 30. Examination of the patient’s face, particularly the bridge of the nose, back and upper lower extremities revealed areas of erythema, blistering and skin desquamation. Patient had an elevated IgE of 698. Punch biopsy was done with histopathological findings consistent of TEN. Histology revealed detached fragments of the epidermis with full thickness necrosis and overlying basket weave stratum corneum. Within the dermis, there was perivascular lymphocytic infiltrate. Cefepime was stopped and the patient was given a two-day course of solumedrol 125mg q8, which resulted in the resolution of the rash on the face, back and extremities.

Drug reactions have been reported in 85-90% of cases of TEN most often caused by antibiotics, NSAIDS, antimitobalites, antiretrovirals and anticonvulsants. We believe that this patient’s TEN was secondary to cefepime since he was started on cefepime in the nursing home, had never been exposed to it previously and symptoms started to resolve after discontinuation of the medication. Most published cases on cephalosporin induced TEN have been on first, second and third generations. There have only been three reports of cefepime induced TEN, a fourth-generation cephalosporin, in the literature thus highlighting its rarity. While our patient was on Keppra for his seizure disorder, he had been on a stable dose for the past 10 years without any
seizure episodes. Our case emphasizes the need to recognize early TEN in patients with active skin desquamation and blisters being treated on cefepime to ensure prompt discontinuation of the antibiotic.
Abstract:
Introduction:
Stress ulcer prophylaxis (SUP) is a common indication for Proton Pump Inhibitors (PPIs) and H2 receptor antagonists (H2RA) use in the hospital setting. It has been postulated that SUP is effective for prevention of upper gastrointestinal bleeding (UGIB) in sick hospitalized patients. Estimates of UGIB from gastric or esophageal ulceration ranges from 1.5 to 8.5% in patients on SUP as compared to about 15% in patients not on SUP. Multiple risk factors for stress ulceration have been identified, but no consensus exists regarding the criteria for SUP. Physician and patient perception of “no harmful effects” of SUP medications has led to major overuse of medications, especially PPI in both inpatient and outpatient practices. A previous study showed that only 45% of inpatient on SUP actually met the appropriateness criteria. The objective of this study was to assess the appropriateness of SUP administration in a community-based hospital setting, including ICU.

Methods:
This retrospective study was conducted at a teaching community hospital in Chicago, IL. 292 inpatients receiving either PPI or H2RA were identified during their hospitalization from October 1st, 2015 to November 30th, 2015. We performed explicit/implicit chart review of 109 randomly selected patients. In addition to demographic data, we collected data on indications for SUP. We deemed SUP as appropriate if there was a presence of major criteria per hospital pharmacy policy i.e. coagulopathy and mechanical ventilation and/or the list of other minor criteria i.e. burns, traumatic brain injury, sepsis, high dose steroid usage or history of GI bleed.

Results:
Ninety-five patients (out of 109) (87%) were admitted under Internal Medicine service. Only Internal medicine patients were included in the results. 33 patients (30%) were admitted to the ICU and 68 patients (59%) were already on antacids at the time of admission. Indications for acid suppression were listed as follows: 27 (28%) were given the medication for SUP, 31 (33%) received it as part of treatment for various medical conditions and 37 (39%) were given the acid suppression medications without any specific indication documented in the charts. Out of 27 patients in SUP category, only 10 (37%) patients were deemed as meeting appropriate indications for SUP criteria. Moreover, 18 patients (67%) were inappropriately discharged from the hospital with the prescription for SUP.
Discussion:
These data show that acid suppression therapy is over utilized in the hospital settings. This finding was compounded by a lack of proper documentation. This preliminary study will further prompt us to deliver a real time SUP indication support system to promote the appropriate use of SUP hopefully leading to the reduced prescription of unnecessary medication.
Introduction
Hemoglobin A1c (HbA1c) is one of the most commonly used criteria for diagnosis and treatment of diabetes mellitus type II (DMII). However, it is less likely known that there exists hemoglobin variants that can falsely elevate HbA1c. Below is a rare case of a hemoglobin variant called Hemoglobin Wayne which is clinically silent but runs the risk of misdiagnosing a patient with DMII, committing them to a lifetime of antiglycemic agents and its complications.

Case presentation
A 40-year-old asymptomatic obese female with significant family history of DMII was evaluated for elevated HbA1c. Over the past four years, she consistently had elevated HbA1c of 10.3%, 10.5% and 10.9%. However, every glucose level on her basic metabolic panel had been normal, with point of care fasting glucose < 126mg/dL, random glucose < 200mg/dL. Oral glucose tolerance test (OGTT) showed fasting glucose of 84mg/dl (reference: 65-99mg/dL), 2 hour glucose of 90mg/dl (reference 65-152mg/dL). Hgb 14.0g/dL. Given the discrepancy between her HbA1c and OGTT, a fructosamine test was ordered which revealed a normal value of 228 µmol/L (reference: 205-285µmol/L). Fasting insulin and C peptide were normal. Hemoglobin electrophoresis showed reduced hemoglobin A1 of 91.4% (reference 96.4-98.2%) with normal hemoglobin A2, undetected Hgb F, S or C. Hemoglobin electrophoresis cascade revealed the patient’s final diagnosis: Hemoglobin Wayne. Because this diagnosis was clinically silent, our patient was never started on antiglycemics.

Discussion
Hemoglobin Wayne is a rare hemoglobin variant caused by a frameshift mutation which alters the alpha globin molecule in heme and confers a specific charge to the hemoglobin similar to the charge of HbA1c. It exists in two different isoforms, Hemoglobin Wayne I and Wayne II. The prevalence of Hemoglobin Wayne is unknown as it is a very rare variant. Studies have shown that Hemoglobin Wayne modifies charge of hemoglobin and confounds HbA1c by interfering with the commonly used cation exchange high performance liquid chromatography assay. Methods such as the boronate affinity assay, molecular analysis and DNA sequencing which are not affected by hemoglobinopathies can be used. In cases of discrepancies between HbA1c and fingerstick testing, further investigations are warranted such as: serum hemoglobin, B12, folate, iron panel to look for decreased RBC turnover; serum fructosamine to measure glycated protein unlike glycated hemoglobin in HbA1c; fasting insulin and C peptide to assess endogenous
insulin; hemoglobin electrophoresis to look for hemoglobin variants. Physicians should be aware of hemoglobinopathies like Hemoglobin Wayne that can falsely elevate HbA1c to prevent the misdiagnosis of DMII that can negatively impact a patient’s quality of life.
Title: The Patient Portal: Improving Patient Engagement and Patient-Provider Communication in Community Health Centers

Abstract:
Introduction: In this digital age, electronic tools for patient-provider communication are becoming the standard of care. Patient portals tethered to the electronic health record (EHR) are expected to improve healthcare quality by streamlining communication, increasing workflow efficiency, and improving patient satisfaction. However, federal incentives for electronic-based health communication do not translate to increased and equitable adoption of patient portals. Portal use in community health centers (CHCs) has the potential to decrease the digital divide and close the gap on healthcare disparities. Despite this, utilization of patient portals has lagged in CHCs nationwide. This study describes current utilization of the MyNearNorth patient portal, explores barriers to portal registration and use, and provides strategies for engaging portal users in community health centers.

Methods: Data collected reflected patients empaneled to Near North Health Service Corporation (NNHSC), a federally qualified health center, across its eight Chicago sites. In Phase I, we used the AllianceChicago customized GE Centricity platform to generate monthly reports for the number of portal PINs generated, active portal accounts, and secure messages sent from July 2016 - July 2017. In Phase II, we provided a small cohort (n=20) of patient participants with a semi-structured MyNearNorth portal tutorial. We recorded participant responses to open-ended questions about perceived portal usage barriers and benefits. In Phase III, we collected observational data on the current NNHSC portal workflow and interviewed key stakeholders.

Results: A total of 16,285 portal PINs were generated, and the portal PIN activation rate was minimal (9.5%). There was a spike in the number of secure messages initiated by the care team following the introduction of a new integrated EHR-to-portal function (124 in April vs. 330 in May). Most portal tutorial participants reported avoiding unnecessary visits as a benefit of portal use (58.3%), while those who declined participation cited discomfort with technology as a barrier (39.1%). Following our workflow observations, we produced revised PIN instructions for clinic use. We found that the patient portal was not consistently discussed with Near North patients and key stakeholders were most concerned about lack of staff engagement.

Conclusion: Low computer literacy and lack of staff engagement pose significant barriers to portal adoption, which could further exacerbate disparities in health systems serving vulnerable
populations. Incorporation of portal features like secure messaging into the EHR has the potential to greatly improve patient-provider communication. Strategies for increasing portal utilization in CHCs must address older patients with limited technological skills. Policies to reduce disparities in patient portal usage should provide support to patients at all three stages: portal access, account activation, and portal use. These findings may be useful in designing workflow protocols and targeted interventions to improve patient portal engagement and patient-provider communication in community health center settings.
Title: Sudden episode of priapism in 58 year old patient who presented with chest pain

Abstract:
Introduction: Priapism is rare and is defined as the persistent erection of the penis not associated with sexual stimulation or desire lasting for at least four hours. Priapism can either be ischemic or non-ischemic.

Case Description: A 58 year old male with uncontrolled type 2 diabetes mellitus, coronary artery disease status post coronary artery bypass grafting and tobacco use who was admitted to the hospital due to complaints of chest pain for the past 3 weeks. He had been seen at a different hospital and discharged without any definitive diagnosis. His blood pressure was 120/62, heart rate 89, respiration 18, and oxygen saturation on room air 98%. He had a tender protruding epigastric mass. His platelet was 203, alkaline phosphate 559, lipase 170, and negative troponin. CT chest revealed left lower lobe pulmonary embolism and an ill-defined mass involving the tail the pancreas extending to the spleen. Anticoagulation with a heparin drip was started. A CT guided biopsy was confirmed by cytopathology as pancreatic adenocarcinoma with metastases to the liver. An echocardiogram showed an 18x18 mm apical ventricular blood clot. On day 4 of bridging to warfarin his INR became supra-therapeutic at 4.4 and his warfarin was held. The next day he developed painful priapism. Physical examination revealed a very rigid shaft of the penis and softer glans penis. Ultrasound of penis didn’t show thrombosis and revealed normal blood flow. His platelet count was 164 and his INR was 8.6. His priapism was relieved after penile injection of epinephrine. The patient was discharged with apixaban and will be following up with his oncologist for further treatment. Further work up has revealed he has both protein C and protein S deficiencies.

Discussion: The exact mechanism of warfarin induced priapism remains unclear and further studies are warranted regarding the action of warfarin and its effect on penile physiology. This case study shows that in hypercoagulable states, susceptible patients with thrombocytopenia can develop priapism and if not recognized and treated early enough, penile necrosis may occur. Previous case studies have suggested that unopposed warfarin be avoided in patients with protein C deficiency. There are suggestions that high warfarin loading doses and premature heparin discontinuation predispose development of warfarin-induced skin necrosis. Also in previous case studies with warfarin induced priapism and necrosis, patients had priapism for approximately 24 hours or longer. Our patient had it for less than 7 hours when it was recognized and treated. Patients taking anticoagulants especially warfarin should be
alerted to the peril signs of priapism and INR levels should be monitored daily till they are therapeutic at least twice 24 hours apart.
Abstract:
Introduction: Infective endocarditis is an infection of the endocardium of the heart with an incidence of 3 to 7 per 100,000 people per year. It typically affects the heart valves and can cause a wide range of systemic complications. If untreated, neurological complications, including embolic stroke, brain abscesses, and meningitis, can occur in up to 30% of patients. 80% of bacterial meningitis cases are caused by Streptococcus pneumoniae and Neisseria meningitidis. There have been far fewer cases reported in the literature of meningitis due to Streptococcus bovis, which is more commonly linked with endocarditis and colonic disease.

Case: An 81-year-old male with a history of multiple myeloma, severe mitral regurgitation, and history of infective endocarditis presented with 1 week of increased fatigue and 1 day of altered mental status. He reported neck pain, headache, and fevers with chills. On presentation he had a maximum temperature of 103 and rigors. An MRI of his brain showed an acute ischemic stroke without evidence of abscess. Lumbar puncture was performed. Cerebrospinal fluid analysis showed 1730 white blood cells with a 92% neutrophilic predominance, elevated protein of 181, and a low glucose of 38. Empiric antibiotic coverage for meningitis was started with Vancomycin, Ceftriaxone, Acyclovir, and Ampicillin. Within 12 hours, the patient improved from minimally responsive to an alert and oriented state. A transthoracic echocardiogram revealed known severe mitral regurgitation with flail leaflets. Blood cultures from admission grew Streptococcus bovis, confirming a diagnosis of infective endocarditis with the fulfillment of 1 major and 3 minor Duke’s criteria. He was discharged on a prolonged course of Ceftriaxone, to treat meningitis and infective endocarditis, with appropriate Cardiothoracic Surgery, Infectious Disease, and Gastroenterology follow-up.

Discussion: Here we present a patient with Streptococcus bovis endocarditis and meningitis, complicated by ischemic stroke, and without evidence of adenocarcinoma on recent colonoscopy. Endocarditis affects up to 2% of patients with meningitis. Streptococcus species and Staphylococcus aureus are often the causative agents of bacterial meningitis in the setting of endocarditis. Streptococcus bovis, recently renamed Streptococcus gallolyticus, is a member of the Lancefield group D Streptococci that is commonly linked with infective endocarditis and colonic neoplasia. Bacterial meningitis due to S.bovis is far more rare. Though our patient’s CSF culture was ultimately negative, we believe his meningitis to be related to his Streptococcus bovis endocarditis. Ischemic stroke affects up to 22% of bacterial meningitis patients and 15.2% of endocarditis patients. The incidence of ischemic stroke rises to 38% in patients with both bacterial meningitis and endocarditis. Risk factors for S.bovis meningitis include
immunocompromised state, prior endocarditis, and colonic disease. Notably, this patient had a colonoscopy in 2015, which revealed only a sessile serrated polyp and diverticulosis.
Mixed connective tissue disease (MCTD) presents in diverse ways. A high index of suspicion is warranted in patients with unexplained Raynaud’s phenomenon. Renal involvement is recognized in MCTD, occurring in up to 25% of cases; with membranous nephropathy and nephrotic range proteinuria being the most common renal manifestation. The case below highlights an unusual complication of MCTD requiring complex medical decision making.

A 35-year-old African American female presented with blunt head trauma after losing consciousness while in the bathroom. She subsequently regained full consciousness. There was a history of headaches and nausea one week prior to this event. One year prior to presentation the patient had been having recurrent Raynaud’s phenomenon. She was evaluated at the time for SLE but ANA was negative making SLE unlikely. Three months prior to her presentation, she was having recurrent pedal swelling and facial edema. Examination on admission revealed no neurological deficit but significant bilateral pedal edema was noted. CT venogram done showed transverse, oblique and sagittal sinus thrombi. This was confirmed with MRI and MRV. She was diagnosed with dural sinus thrombosis and started on anticoagulation with enoxaparin. Urinalysis revealed proteinuria with an albumin/creatinine ratio of 10g/g. Serum albumin was 1.5g/dl, creatinine was 0.6mg/dl. Work up for nephrotic syndrome showed positive ANA (1:160 titers) with a speckled pattern, normal anti-dsDNA, anti-SSA, and anti SSB but Anti U1-RNP was positive and very high (>8). This immunologic profile supporting the diagnosis of MCTD. Hepatitis B and C screening was negative and C3 and C4 levels were normal. Due to the risk of ongoing cerebral venous thrombosis, anticoagulation could not be stopped to perform a renal biopsy. The patient was thus commenced on prednisone and tacrolimus after extensive multidisciplinary discussion. The goal being the empiric treatment of the most likely renal lesion of membranous nephropathy; to reduce proteinuria, improve the serum albumin and decrease the risk of thromboembolism thereby allowing a renal biopsy down the line.

Hypercoagulable states secondary to severe nephrotic syndrome due to MCTD may present with thromboembolic complications. This may necessitate the use of anticoagulants in these patients and preclude an immediate renal biopsy. This case illustrates the importance of suspecting MCTD in patients with unexplained Raynaud’s phenomenon and the use of immunosuppressants as a bridge to renal biopsy when an immediate renal biopsy is contraindicated.
Background: Reverse Takotsubo cardiomyopathy (RTCM) is a rare variant of the classic Takotsubo stress CM (TCM), characterized by akinesis of the basal and mid segments of the left ventricle (LV) with preservation and/or hyperdynamic apical function of the LV. The pathophysiology behind TCM is believed to be secondary to catecholamine release. RTCM tenders to be more prevalent in younger patients tend to have higher concentration of adrenergic receptors in the basal, and mid LV segments. Recovery is anticipated with treatment of underlying stress and HF medical therapy.

Case Description: A 61-year-old woman with history of stage IV endometrial carcinoma, initially presented at outside hospital (OSH) with Acinetobacter ursingii sepsis. OSH workup included, an electrocardiogram, notable for sinus tachycardia with non-specific T wave abnormalities and prolonged QT interval. Troponin was checked and trended due to acute onset of dyspnea: 0.03, 3.82 and 0.50 ng/ml. A transthoracic echocardiogram (TTE) was significant for akinesis of the basal and mid left ventricular (LV) walls, with preservation of apical function. Due to an elevated troponin and new wall motion abnormalities, a nuclear regadenoson stress test was done which was demonstrated normal perfusion and negative for myocardial ischemia and infarction. She was treated for her line associated bacteremia and discharged in stable condition. She then presented a month later to our institution for her cancer care. Cardiology was consulted for the history of LV dysfunction from the OSH. She had denied any shortness of breath on exertion, orthopnea, or paroxysmal nocturnal dyspnea. She had trace pedal edema she attributed to blood clot in the past. Repeat TTE with Optison showed complete normalization of her LV function, which confirmed the diagnosis of RTCM. She was discharged on an ace inhibitor and a beta blocker with continued outpatient Cardiology care at our institution.

Conclusion: This case highlights the importance of TTE follow up to reassess LV function regardless of the patient’s profile in establishing the correct diagnosis, especially, when subsequent therapy(s) may be altered, as in our case of a woman with advanced cancer and the need for additional chemotherapy. The repeat TTE confirmed the diagnosis of RTCM, a rare variant of stress cardiomyopathy, with complete restoration of LV function in a post menopausal woman. This patient will continue HF medical therapy and chemotherapy. Her LV
systolic function coupled with strain will be monitored yet there was no interruption in her cancer treatment plans.
Parasitic infections are often overlooked in developed areas of the world. Because they are infrequently encountered in clinical practice, diagnosis and management can be challenging. In this case, a 23 year old male previously living in Hawaii presented with pain in his feet for 3 weeks. He described a burning sensation with radiation to the calves bilaterally. He was initially prescribed oral methylprednisolone and indomethacin for presumed gout, although later a BMP and uric acid were found to be normal. The patient’s foot pain progressed and the day prior to admission, he was seen at the infectious disease clinic and found to have eosinophilia. He was started on ivermectin for suspected strongyloides infection. However, he had no improvement of symptoms and was sent to the hospital. Upon admission, erythrocyte sedimentation rate proved normal, though c-reactive protein was elevated at 20.2. Complete blood count showed eosinophilia of 15.8%. CT abdomen/pelvis to evaluate for intra-abdominal parasite infection was unremarkable. An MRI w/ and w/o contrast the following day revealed findings concerning for myelitis. Lumbar puncture was performed the next day, notable for 35% eosinophils. Angiostrongylus cantonensis infection was suspected after these findings. A review of the patient’s history revealed that the patient worked at an organic food store in Hawaii, and ate salads daily given his vegan diet. PCR of the CSF obtained from the lumbar puncture confirmed presence of Angiostrongylus cantonensis. The patient was treated with supportive care, including IV methylprednisolone. His pain improved and he was discharged with prednisone and appropriate follow up. Later, he was seen in ophthalmology clinic for retinal detachment and found to have a worm in the back of the retina, which was removed.

Angiostrongylus cantonensis, a parasitic roundworm, is the most common cause of eosinophilic meningitis. These parasites reside in the pulmonary arteries of rats, with snails as a common intermediate host. Humans become infected through ingestion of the larvae. The larvae eventually migrate to the meninges and develop into their adult form. Once matured, the parasite cannot survive and dies, leading to the inflammatory response causing the symptoms of infection. It was suspected that the patient consumed raw vegetables contaminated by snails while in Hawaii. A retrospective analysis of cases of eosinophilic meningitis from 2003 to 2005 showed that a majority of cases were in Hawaii, with similar descriptions of paresthesias that this patient experienced.

This case demonstrates the importance of a broad differential diagnosis and thorough diagnostic testing when approaching potential infectious causes of illness, especially given
history including travel and specific dietary considerations. Although rare, eosinophilic meningitis can cause devastating sequelae including myelitis and retinopathy. Complete information gathering and testing is therefore crucial to avoid missing such a diagnosis.
Abstract:
Hyperthyroidism secondary to malignant tumors secreting high levels of human chorionic gonadotropin (HCG) is a rare condition. Germ cell tumors can be associated with elevated HCG and subsequent activation of thyroid stimulating hormone (TSH) receptors in predisposed patients. This paraneoplastic phenomenon in testicular germ-cell tumors and elevated HCG has an estimated prevalence of 3.5% varying according to the level of HCG. Although this diagnosis can be challenging in patients with advanced disease, early intervention is of paramount importance to prevent potentially life-threatening complications.
A 27-year-old man with no past medical history presented with a 6-week history of right hemiscrotal swelling. On further questioning, he described a 2-month history of weight loss, night sweats, palpitations, hemoptysis and clear nipple discharge. Physical examination showed a regular tachycardia to 100 bpm with normal thyroid exam, bilateral gynecomastia and a midline abdominal mass. Genital exam demonstrated an indurated and edematous right hemiscrotum. Ultrasonography revealed a right testicular mass measuring 8 cm in its largest dimension. Serum HCG and alpha-fetoprotein (AFP) were elevated to 199,000 IU/L and 135 ng/mL respectively. The patient underwent right radical orchiectomy, with testicular pathology revealing a mixed germ cell tumor composed of 90% seminoma and 10% choriocarcinoma. CT imaging demarcated a large retroperitoneal mass and innumerable lung nodules with multiple enlarged mediastinal lymph nodes consistent with metastatic disease. The findings of weight loss, palpitations and persistent tachycardia prompted testing of thyroid function, revealing low levels of TSH (0.088 IU/mL) and increased free thyroxine (FT4) (2.88 ng/dL). Thyroid-binding inhibitory immunoglobulins (TBII) were undetectable. He was started on atenolol for hyperthyroidism and chemotherapy with bleomycin, etoposide and cisplatin. After 6 weeks of treatment his tachycardia had resolved, serum tumor markers declined (serum HCG 8000 IU/L and AFP 6 ng/dL), and thyroid function tests normalized.
Paraneoplastic hyperthyroidism in patients with testicular cancer is a rare condition. In patients with high serum HCG levels (>50000 IU/L), the estimated prevalence of thyrotoxicosis is approximately 50%. This supports the theory that high levels of HCG are capable of inducing hyperthyroidism through stimulation of the TSH receptor. However, this phenotype is not constant possibly due to the existence of HCG isoforms with diminished affinity for the TSH receptor. Chemotherapy can reduce HCG levels but carries a risk of inducing thyroid storm due to the prolonged half-life of HCG and an initial surge upon commencement. Preemptive treatment with beta-blockers may ameliorate this complication. The role of early treatment with thionamides is uncertain. Recognition of paraneoplastic hyperthyroidism may be difficult
in patients with malignancy as symptoms overlap with those of metastatic disease. Hence, a high degree of clinical suspicion is required to prevent this potentially catastrophic presentation.
Strumal Carcinoid Tumor: A Rare Incidental Finding

Abstract:
Background:
Strumal Carcinoid is a rare ovarian germ-cell tumor with features of both mature thyroid and carcinoid neuroendocrine tissue. Most patients are post-menopausal, but may occur in the third to eighth decade of life. Primary ovarian carcinoid tumors only account for 0.1% of all ovarian malignancies and 0.5%-1.7% of all carcinoid tumors[1,2]. Most patients with strumal carcinoid do not exhibit carcinoid-like symptoms of flushing or diarrhea [3], rather have been reported to have cons pa on. Patients present with abdominal mass, pain, and hirsutism. These tumors are usually found incidentally.

Case Study:
40-year-old G3P3 with a history of uterine fibroids and anemia who presented for a scheduled left ovarian cystectomy after experiencing fevers greater than 101°F, vaginal bleeding, progressively worsening abdominal and pelvic pain. She had been diagnosed with ovarian cysts 3 years prior. A recent pelvic ultrasound prior to surgery showed a 12x8x7cm and 6x8x6cm cysts. At that time, the decision to remove the cysts was made. Laboratory workup prior to surgery showed no abnormalities. Surgery was scheduled with the intent to relieve the patient of her worsening symptoms since her initial diagnosis.

Results:
During surgery, a large left ovarian cystic structure with both solid and cystic properties were found. Samples sent for pathology revealed a 129g collapsed multi-cystic mass measuring 9.6 x 7.8 x 4.2 cm filled with serous fluid. There was a focal area consisting of hemorrhagic fibro-fatty tissue while the remaining serosal surface is purple-pink, focally roughened and congested. Within the cystic wall, a firm mass measuring 4.5 x 3.3 x 3.0 cm was found. The tumor shows thyroid tissue intermixed with carcinoid. The thyroid component was positive for TTF-1, while carcinoid component was positive for synaptophysin, pancytokeratin and chromogranin. No significant mitotic activity was identified in the carcinoid component. Octreotide scan showed no abnormal focal lesions. Staging CT scan of the chest, abdomen & pelvis after resection of the cysts revealed post-surgical changes.

Conclusion:
We report the case of a 40-year-old female with a strumal carcinoid tumor found during a scheduled ovarian cystectomy. Surgical removal of the tumor in this patient proved to be
curative based on post-surgical lab work and imaging. Prognosis for strumal carcinoid after surgical removal is considered excellent [4].

References on request.
Abstract:
Sternocostoclavicular Hyperostosis (SCCH) is a rare clinical entity that may overlap with clinical symptoms consistent with the diagnosis of Paget’s disease. SCCH typically presents with erythema, swelling, and pain at the sternoclavicular joint with an often associated pustular rash known as palmoplantar pustulosis. The course is frequently episodic with exacerbations and remissions. A variety of treatments have shown benefits including NSAIDs, IV bisphosphonates, Cyclosporin A, DMARDS, and TNF-alpha inhibitors.

A 59 year-old Chinese male with no significant past medical history presented to the emergency department with bilateral clavicular pain that was chronic in nature and new onset diffuse body rash. Recent CT scan and bone scan had previously demonstrated sclerosis and thickening of the clavicles, first ribs, and manubrium suggestive of Paget’s Disease and the patient was treated with IV pamidronate. At the time of the current presentation, the patient’s vital signs were significant for tachycardia. Physical examination revealed a man of Asian descent in significant distress. Marked tenderness to palpation at the clavicles bilaterally with overlying erythema and warmth was appreciated. A diffuse petechial, non-blanching rash on the upper arms and gluteal region was noted with areas of confluence. Labs were significant for elevated inflammatory markers and negative alkaline phosphatase, ANA, ANCA, RF, Hepatitis serologies. MRI was obtained revealed enhancing inflammatory signals seen along the periosteal margins of both clavicles extending into the pectoralis anteriorly concerning for Paget’s disease. Skin biopsy revealed leukocytoclastic vasculitis thought to be either idiopathic or secondary to bisphosphonate use. He was treated with high dose prednisone with resolution of his rash along with extensive analgesia prior to resolution of the patient’s pain. He was followed in rheumatology clinic with improvement of his symptoms and had subsequently initiated sulfasalazine therapy before being lost to follow up.

This case illustrates that SCCH may be a common mimic of Paget’s disease given the radiographic similarities. The diagnosis of SCCH is typically confirmed by hyperostosis and sclerosis of the sternum and first ribs on computed tomography although inflammation may extend beyond those areas. Although Paget’s has similar radiographic findings, Paget’s will often present with axial skeleton findings and an elevated alkaline phosphatase level, which were both absent in our patient. Retrosternal proliferation of soft tissue is an important diagnostic criterion that can also help separate SCCH from other benign processes. Since recent data shows SCCH may respond well to early initiation of treatment, this is an important entity
to differentiate from Paget’s since delayed treatment can lead to severe restriction in mobility and degenerative changes.
Abstract:
Infective endocarditis is a known cause of cerebrovascular accidents (CVA) in 35% of cases (1). Of these cases, about 35% are found to be due to culture-negative endocarditis (2-3). Cryptococcus represents an extremely rare pathogen in endocarditis with only seven reported cases (4). Little is known regarding the natural history and optimal management given the limited available data. We report the first known case of a patient with native aortic valve endocarditis secondary to Cryptococcus neoformans in the context of active malignancy treated with targeted immunotherapy.

The patient is a 60 year-old woman with metastatic non-small cell lung adenocarcinoma with widespread osseous and lung metastases (T1bN2M1b, EGFR+) treated with erlotinib and multiple deep vein thromboses (DVTs) managed with rivaroxaban who presented to the emergency room with right-sided weakness and facial droop. MRI revealed embolic strokes to the left hemisphere (temporal and thalamic). Transthoracic echocardiogram demonstrated new aortic insufficiency. A subsequent transesophageal echocardiogram demonstrated a round echogenic mass on the left ventricular side of the non-coronary cusp of the aortic valve measuring 5.1 x 6.7 mm and a smaller mass on the right coronary cusp measuring 3.3 x 3.6 mm. During this admission, the patient was hemodynamically stable, afebrile, and had no leukocytosis. Blood cultures were negative for bacterial growth. Culture-negative endocarditis work up was subsequently initiated, including Histoplasma antigen, Blastomyces antigen, Legionella antigen, Brucella antibodies, Coxiella antibody, Bartonella antibody, and Whipple's DNA PCR. Due to suspicious morphology of lung nodules on CT and no prior biopsy to confirm metastatic disease, serum Cryptococcal antigen was also obtained. This later came back positive with a titer of 1:320. Lumbar puncture was performed, revealing an elevated opening pressure of 33 cm, lymphocytic predominance (88%), glucose of 37 mg/dL, and protein of 32 mg/dL. CSF Cryptococcal antigen titer was elevated at >=1:2560. Treatment was initiated with amphotericin B lipid complex and flucytosine. Fungal blood and CSF cultures later grew Cryptococcus neoformans at 5 and 1 days, confirming diagnosis. Repeat LP demonstrated decreased Cryptococcal antigen titer of 1:320. Flucytosine was discontinued due to pancytopenia. The patient was to complete 6 weeks of the amphotericin B and to be transitioned to fluconazole for consolidation therapy. However, her clinical status declined. The risks were deemed to outweigh benefits of further lumbar puncture or valve replacement, and the patient and her family decided to transition to hospice care.
This represents the first known case of Cryptococcal endocarditis reported in a patient with active malignancy receiving targeted immunotherapy. With the advent of several new targeted immunotherapeutic agents and limited long-term data, further investigation is needed regarding possible increased risk of infectious diseases. The astute clinician will avoid anchoring bias and consider a broad differential diagnosis in immunocompromised patients.
**Title:** Thrombolytic Therapy in Stroke Patients within tPA window: “Are we FAST or merely getting by standards?”

**Abstract:**

Introduction

American Heart Association’s target of Tissue Plasminogen Activator (tPA) administration within 60 minutes to eligible stroke patients is achieved only in 30% of US patients and there is currently a national momentum to improve this rate to at least 50%. Studies have shown that for every 15 minute reduction in door-to-needle times, there was a 5% lower odds of risk-adjusted in hospital mortality and 4% increase in odds of independent function at discharge. Therefore, it is imperative to assess these barriers and implement best practices to reduce door to tPA times.

The objective of the study was to assess the impact of structured changes to our existing hospital stroke care system (intervention) on door-to-needle times for IV tPA administration (outcome).

The changes (intervention) were implemented on ten key barriers to target tPA administration time that were identified using root cause analysis and focus group discussions on a separate retrospective study, a prelude to our current study.

Methods

The study design was a quasi interventional outcomes study with a pre (June 2014-November 2016) and post (December 2016 to May 2017) system intervention comparison.

Subjects were patients who arrived to hospital with clinical suspicion of acute ischemic stroke to the Emergency Department within 3.5 hours of last known well and are eligible for tPA. Exclusion criteria included hemorrhagic stroke/TIA and uncontrolled blood pressure.

Primary outcome of the study was door-to-needle time for IV tPA administration. System intervention in the 10 key barriers were: Telestroke robot implementation prior to patient’s arrival, replace full NIHSS assessment by a shorter and more efficient "Estimated" NIHSS assessment, patient taken directly to CT, new order set "Nursing Stroke Order Set" for ED RNs to place w/CT brain, Code Stroke toolbox, assigned tasks for team members, designate one IV pump for exclusive tPA use, redesign Code Stroke process to include ISTATs on all patients, ED MD education on assessing LVO/posterior occlusion and add Code Stroke CTA Brain order reflex to Initial Stroke Order set.

**Results**
Our current analysis includes 70 pre-intervention (baseline) and 14 post-intervention (intervention) subjects.
The percentage of tPA eligible stroke patients who met goal of tPA administration within 60 minutes of arrival improved from a pre-intervention rate of 36% to a post-intervention level of 81%.. The improvement phase depicted mean door to tPA time of 52.2 minutes compared to baseline 88.2 minutes.

Conclusions
The analysis concludes that by correctly identifying limiting barriers and implementing steps to decrease delays, we can improve door to tPA times. Additional analysis would be useful to examine secondary outcomes such as: increasing the odds of independent function at discharge and reduce risk of in hospital mortality. Limitations include small sample size and geographic bias in regard to patient population.
Abstract:
INTRODUCTION:
POEMS stands for Polyneuropathy, Organomegaly, Endocrinopathy, Monoclonal protein and Skin disease. Although the etiology of this rare syndrome remains anecdotal, vascular endothelial growth factor (VEGF) and interleukin-6 seem to play central roles, leading to microangiopathy and increased vascular permeability, resulting in the typical clinical features. We present a 39 year old woman who presented with neuropathy, initially diagnosed with chronic inflammatory demyelinating polyneuropathy (CIDP), and later diagnosed with POEMS syndrome.

CASE:
39 year old woman presented with progressively worsening sensory loss and motor weakness in extremities. Deficits were confirmed on exam. Labs showed a hemoglobin of 16.2 g/dL, a platelet count of 732,000 per microliter. Electromyography (EMG) showed demyelinating motor predominant polyneuropathy. Cerebrospinal fluid analysis showed white blood cell count of 2 per microliter, 214 mg/dL protein, with oligoclonal bands. She was diagnosed with CIDP and treated with prednisone (1mg/kg). Symptoms improved initially, but started worsening 3 months prior to her second admission when she presented with worsening weakness and distal (both anterior and posterior column) sensory deficits in extremities. This time she also had Raynaud’s phenomena, clubbing and anasarca. The protein gap had always been < 4 g/dL, however, serum immunoelectrophoresis (IFE) showed monoclonal IgA lambda chains, and a serum free light chain ratio of 0.5. Bone marrow biopsy showed 28% plasma cells, monoclonal for lambda light chains. Fat pad biopsy showed only calciphylaxis, and no amyloid. Repeat EMG showed worsened demyelinating sensorimotor polyneuropathy with secondary axonal loss. No lytic lesions were seen on bone survey. As a part of workup, VEGF level was sent for and it was elevated: 523 pg/mL (normal: 31-68). A diagnosis of POEMS syndrome was made and chemotherapy with cyclophosphamide, bortezomib, and dexamethasone was started.

DISCUSSION:
POEMS syndrome usually presents in the fifth to sixth decade of life as an inflammatory demyelinating sensorimotor polyneuropathy, and a monoclonal lamda light chain disorder. Sclerotic bone lesions are very frequent (97% of cases). Multitude of skin findings can be present (like hyperpigmentation and hemangioma, among others). This syndrome falls under the umbrella of plasma cell dyscrasias (PCD). Multiple myeloma, the most common PCD, was
ruled out in this patient in the absence of CRAB criteria (hyperCalcemia, Renal insufficiency, Anemia, Bone lesions). Diagnosis of POEMS syndrome is based on International Myeloma Working Group criteria which mandates 2 mandatory major criteria (polyneuropathy and monoclonal plasma cell disorder), 1 major criteria (sclerotic bone lesions, Castleman's disease and elevated VEGF), and 1 minor criteria (organomegaly, extravascular volume overload, endocrinopathy, skin changes, papilledema, thrombocytosis/polycythemia). This presentation highlights the importance of recognizing this rare diagnosis attached with CIDP. In the presence of a protein gap and/or positive IFE studies, a clinician must always entertain a diagnosis of rare PCDs.
A Case of Submandibular Masses?

Abstract:
IgG4-related disease (IgG4-RD) is an immune-mediated fibro-inflammatory condition of unknown etiology and poorly understood pathophysiology. It was not recognized as a systemic condition until 2003. Epidemiology requires further studying as its confusing nomenclature as well as non-specific laboratory, imaging and even histopathological findings led to under recognition of the disease by physicians, and it might be more common than previously thought.

A 61 year old man was referred to the emergency room by his physician for the evaluation of progressively worsening chronic dry cough, sinus pain, nasal congestion, fatigue and submandibular masses. Patient recollected that about 2 years ago he had nodules on his left submandibular area and he was told that it might be related to his acne. No further work up was done at that time because the nodules had decreased in size. Physical examination was significant for multiple, nontender submandibular masses. Laboratory findings revealed leukocytosis of 14500/mm3, elevated sedimentation rate (55 mm/h), and C-reactive protein of 181.6 mg/L. Chest radiography raised the concern of granulomatous disease. CT head and neck showed extensive paranasal sinusitis and diffuse enlargement of the left parotid and submandibular glands. The patient underwent an extensive diagnostic work up as the differential diagnosis was broad. CT chest showed several spiculated bibasilar pulmonary masses. Infectious work up was negative. Fine needle aspiration biopsy of the submandibular mass was performed, but pathology was non-diagnostic, showing abundant neutrophils. A core biopsy was done, showing fibrotic tissue with focal mixed inflammation including eosinophils, neutrophils, and mild lymphocytic infiltrate. The possibility of IgG4 related disease was entertained. Serum IgG4 level was elevated, and ANA was negative. Oral prednisone was started with significant response within a few weeks. On the most recent visit was noted that the submandibular masses have completely resolved.

This case illustrates the diagnostic challenge of the IgG4-RD which can mimic various inflammatory and neoplastic diseases. It typically affects middle aged and older men. Several manifestations can occur in the same patient including, but not limited to: autoimmune pancreatitis, Mikulicz disease, retroperitoneal fibrosis, pulmonary nodules, thyroiditis, IgG4-renal disease. Major salivary gland involvement is a common feature of IgG4-RD: lacrimal and parotid gland enlargement (previously called Mikulicz disease), and/or submandibular gland enlargement (previously called Küttner’s tumor). Several entities of pulmonary involvement have been described: it can mimic sarcoidosis but can also manifest as solitary nodule, bronchovascular or alveolar interstitial pattern. Biopsy is still considered a gold standard.
Recent meta-analysis reported that elevated serum IgG4 level has a sensitivity of 87% and a specificity of 83%. Recognition of IgG4-RD is important to implement appropriate management and prevent further organ damage and disease recurrence.
Abstract:
Protective mechanical ventilation is a widely accepted initial strategy in the treatment algorithm for acute respiratory distress syndrome (ARDS). In cases of refractory hypoxemia, extracorporeal membrane oxygenation (VV-ECMO) is commonly used in experienced centers. It has been postulated that early initiation of VV-ECMO for severe ARDS in otherwise healthy individuals may provide sufficient support with minimal complications, avoiding some of the inherent risk of lung injury by mechanical ventilation.

A 49-year old male presented to the emergency department with acute-onset dyspnea and hypoxemia one day following a septoplasty and rhinoplasty for obstructive sleep apnea. A chest x-ray showed diffuse bilateral pulmonary infiltrates. Supplemental 100% FiO2 via face mask was required to maintain his oxygen saturation above 90%, and an arterial blood gas demonstrated PaO2:FiO2 of 67. He started and ultimately completed antibiotic treatment for presumed aspiration pneumonia and received intravenous diuretics. Transthoracic echocardiogram demonstrated normal heart function. A clinical diagnosis of severe ARDS was promptly made. Based on patient’s preference on avoidance of intubation and following a multidisciplinary approach, VV-ECMO was initiated as an alternative strategy to mechanical ventilation. He required a brief period of intubation to support oxygenation while ECMO cannulation via femoral-jugular vein approach occurred. Bronchoscopic examination was performed and revealed a neutrophilic bronchoalveolar lavage, which was negative for diffuse alveolar hemorrhage and positive for Parainfluenza 3 virus. He received one day of ribavirin and intravenous immunoglobulin, both of which were discontinued shortly thereafter. He was successfully extubated after 24 hours and remained on awake, ambulatory VV-ECMO, receiving only 3 liters of oxygen via nasal cannula. The patient denied dyspnea during this period of time and had access to oral intake. After decannulation he was rapidly down-titrated to room air with maintenance of normal oxygenation. Overall, the patient spent a total of 5 days on VV-ECMO and a total of 8 days in the intensive care unit. Six days after discharge, the patient’s pulmonary function testing demonstrated mild symmetric reduction in FEV1 and FVC with a normal ratio as well as mildly reduced diffusion capacity, however the patient had no respiratory complaints and was oxygenating appropriately on room air at rest and on ambulation. One month after discharge, the patient reported no symptoms and had no limitations in physical activity with radiographic normalization of his chest-x-ray.

This case illustrates the successful use of early VV-ECMO in the treatment of severe ARDS. This single case report aligns with similar cases reported in some other centers around the world.
Given the complexity of this therapy which is not risk-free, center-experience driven and applied to a subset of a very sick patient population, a comprehensive well-designed clinical trial is encouraged before establishing conclusions on its application.
Abstract:
Introduction
Hypereosinophilic syndrome (HES) is a sustained overproduction of eosinophils that results in eosinophil-mediated organ damage. Can be primary (neoplastic), secondary and idiopathic. The estimated prevalence is between 0.36-6.3 per 100,000, but the actual prevalence is unknown. We present a case of a patient with peripheral neuropathy secondary to HES with myelodysplastic features.

Case Report
A 69 year old male came to our institution with progressive tingling and burning sensation in both feet for the past two months. In addition to that, he has been noticing pedal muscle stiffness that progressed to the thighs and worsens with walking. Gabapentin provided minimal relief. Past medical history includes idiopathic angioedema resolving with an antihistamine and eosinophilia with extensive work up done in 2013, significant for elevated IgE level and eosinophilic count of 14.876 UI/ml. The patient is being followed by a hemato-oncologist due to myelodysplastic features including low C4 count, elevated CRP and ESR level; elevated Vitamin B12, tryptase level, and M protein on SPEP (IgG Kappa). Bone marrow biopsy reported: hypercellular bone marrow with 55-60% cellularity and intact trilineage hematopoiesis, with a significant increase of mature and immature eosinophils. As neurologic symptoms persisted, electrical muscle stimulation (EMS) was done and was positive for asymmetric, predominately axonal, sensorimotor polyneuropathy of significant severity. The patient was diagnosed with hypereosinophilic syndrome with myeloproliferative features and neurologic symptoms as evident by hypercellular marrow, dysplastic eosinophils, increased B12 and tryptase with no direct evidence of clonality as JAK2, FISH for 4q12, 5q33.1, 8p11, 16q22 and EMS findings. The patient was prescribed with low dose prednisone with subsequent tapering and continued follow-ups.

Discussion
HES is defined as an absolute eosinophilic count (AEC) > 1500 cells/microL for longer than six months. Eosinophils can damage the tissues and common target organs include the skin, lungs, gastrointestinal tract and only in 4% of the cases can damage the nerves. Eosinophilia can be seen in a variety of conditions like collagen vascular disease, sarcoidosis, ulcerative colitis and human immunodeficiency virus infection. HES has been subdivided into several clinically relevant variants as myeloproliferative, T-lymphocytic, familial, idiopathic and organ-restricted conditions. Among patient with multisystem organ involvement, some demonstrate features of a myeloproliferative disease, including increased vitamin B12 levels, chromosomal
abnormalities, anemia and circulating leukocytes precursors. Neurological manifestation of HES can include cerebral thromboemboli, encephalopathy. However, peripheral neuropathy accounts for approximately half of cases with neurological involvement. Neuropathy may present as symmetric or asymmetric and may involve sensory or motor nerves. As our case illustrates, early recognition of hypereosinophilia as a significant laboratory abnormality can aid in the prevention of end-organ damage.
Title: Role of Corticosteroids in Subclinical Atherosclerosis in SLE: A Systematic Review and Meta-Analysis

Abstract:
Background/Purpose: Atherosclerosis in SLE results from a complex interplay between traditional risk factors, SLE-specific factors, chronic inflammation and multifaceted effects of SLE therapeutics. In particular, corticosteroids may exert a double-edged effect by increasing traditional risk factors on one hand and inhibiting the inflammatory process on the other. In this meta-analysis, we aim to determine the association between corticosteroids and subclinical atherosclerosis in SLE patients and investigate the influence of strength, duration and cumulative dose of steroids.

Methods:
A comprehensive literature search was conducted in Cochrane Library, Scopus, MEDLINE, PubMed and EMBASE for articles published in English from January 1985 through June 2016 in adult age group. Studies were eligible if they presented the dose of steroid administered to the SLE group and used mean CIMT as evaluated by high-resolution ultrasound (surrogate marker of subclinical atherosclerosis). Two independent reviewers performed study selection and data extraction. All articles with multiple publications were considered for data extraction but only one was used for final analysis. The required data to estimate the effect-sizes associated with each study was extracted. Using Hedges’ random-effect model, the estimated effect-sizes were pooled together. Heterogeneity was explored using subgroup analysis and meta-regression technique. Publication bias was tested using funnel plot and Eggers test.

Results:
Out of 254 citations, 24 studies were eligible. Disease characteristics and quality score of the studies are shown in Table 1. The pooled effect size showed statistically significant increase in subclinical atherosclerosis in SLE patients (SMD=0.821, P=0.000; 95% CI, 0.512 to 1.22). In a univariate meta-regression model, corticosteroid consumption significantly increased the risk of subclinical atherosclerosis in SLE patients (B=0.018, P= 0.027; 95% CI, 0.002 to 0.035). Result also showed, there was insignificant relationship between the duration as well as cumulative dose and the risk of subclinical atherosclerosis in SLE patient. Subgroup analysis showed that the above association of corticosteroid and subclinical atherosclerosis in SLE patients is not affected by the dose of the steroid.
Conclusion:

Our findings concluded that corticosteroids increase the risk of early atherosclerosis in SLE patients. We also found that subclinical atherosclerosis is not influenced by the strength, duration or cumulative dose of steroids. The biggest challenge to this analysis is the heterogeneity of the studies included. Further research is needed to better understand the adjusted effect of SLE disease activity in the role of steroids in subclinical atherosclerosis.
Excitable cause of relapsing-remitting paralysis: Unusual case of Thyroid Periodic Paralysis.

Abstract:
Introduction: Patients presenting with constellation of hypokalemia and acute paralysis have a heterogeneous group of underlying etiologies from familiar thyrotoxic/familial periodic paralysis to rare cases of licorice ingestion. In US, most cases were due to familial periodic paralysis but in recent times there is a rise in the cases of thyrotoxic periodic paralysis (TPP). This rise has been secondary to increase in immigrants from Asian/Latin American countries, which are the predisposed populations.

Case: 28-year-old Hispanic male with history of hyperthyroidism presented with diffuse muscle weakness after stopping his anti-hyperthyroid medications since last 1 month, as he was no longer able to afford the medications. He then noticed painless weakness in his legs and arms. Vitals were otherwise normal except for a heart rate of 108bpm. Exam was remarkable for symmetric non-focal muscular weakness with depressed reflexes. No respiratory distress noted. His BMP was remarkable for K of 2.8meq/dL. Given the history of hyperthyroidism, thyroid function tests were sent: TSH 0.002 mU/L and free T4 14.6 mcg/dL. The patient was admitted to the intensive care unit and was started on potassium supplementation, propranolol and methimazole. Within 24 hours of initiation of therapy he had complete resolution of his paralysis. Of note, patient lacked the classical symptoms of thyrotoxicosis like diaphoresis, palpitations, diarrhea, anxiety or tremor. Serum thyroglobulin was elevated consistent with probable Graves’ disease. In the setting of having elevated thyrotoxicosis and hypokalemia he was diagnosed with Thyrotoxic Periodic Paralysis. Patient was discharged on methimazole daily and a betablocker and is currently doing well.

Discussion: In this case, the patient presented with thyrotoxicosis solely manifesting as muscular paralysis without other characteristic signs or symptoms of hyperthyroidism. A good initial history and physical exam will help ascertain any focality of neurological signs, which point toward a stroke. Presence of diffuse muscle paralysis with intact consciousness was pointer towards an extracranial pathology. Presence of a sole severe hypokalemia with muscle paralysis should raise the suspicion of a problem at a muscular level, especially if other causes have ruled out. TPP one of the compelling cause of such presentation and thus all such cases warrant thyroid function testing. Excessive thyroxine stimulates the Na-K ATPase pump causing cellular influx of potassium leading to decreased muscle excitability. Thus, hypokalemia is
secondary to transcellular shift and not true total body potassium deficit. Correcting the underlying hyperthyroid state and judicious potassium supplementation (to avoid rebound hyperkalemia) are appropriate management strategy. There is evidence that such ionic channels have a predilection in hyperthyroid states with completely normal function in euthyroid states. TPP should be differentiated from familial periodic paralysis with features of presence of tachycardia (HR >105), later age of onset and history of hyperthyroidism in the former.
INTRODUCTION
Non-compaction cardiomyopathy (NCC) is a condition characterized by ventricular trabeculae and deep intertrabecular recesses. [1, 2] Its prevalence among patients with heart failure (HF) is approximately 3 to 4 percent [3, 4]. Anti-thrombin (AT) and protein C (PC) deficiency in the setting of cardiomyopathy is not well understood, but well explained in the setting of liver congestion. We present a rare case of AT and PC deficiency resulting in acute arterial and venous thrombosis in the setting of rapidly progressive HF secondary to NCC.

CASE PRESENTATION
27 year-old, Hispanic male patient, with no pertinent PMH presented with acute onset, R lower extremity edema (LEE), ipsilateral calf pain and foot coldness and numbness. CTA of RLE showed a thrombus to the common iliac artery and an occlusion of the popliteal artery. ECHO demonstrated a borderline EF 50-55% with moderately reduced right ventricular systolic function (RVSF). The PC and AT levels were decreased. He underwent an uncomplicated RLE embolectomy and was discharged on LMWH to bridge to warfarin.

Two years later, he presented to our institution with scrotal and bilateral LEE after discontinuing anticoagulants. VS were unremarkable. Positive findings included a palpable, pulsatile, non-nodular hepatic left lobe with LEE 2+ and palpable pulses 2+ throughout.

Relevant workup included persistent decreased AT and PC levels. ECHO showed an EF 29% with a fixed thrombus on the apical wall of the LV and severely reduced RVSF. MRI of the heart showed multiple intracardiac thrombi in both ventricles. Multiple areas of contrast enhancement were seen on the lateral wall of the LV, apex, and basal aspect of the RV free wall consistent with embolic infarcts/trabeculae. The US abdomen showed a partial portal vein thrombosis (PVT).

He required a single chamber ICD placement and was given therapeutic doses of LMWH (1mg/kg) for the intracardiac thrombi and PVT secondary to NCC and PC and AT deficiency improving his clinical condition.

A 3-month follow-up ECHO showed an EF 48%, with a persistent fixed apical thrombus.
DISCUSSION
The mechanism of a significant reduction in AT and PC levels resulting in acute limb ischemia and venous thromboembolism in the setting of HF is unclear. Some authors have reported that the hepatic synthesis of these natural anticoagulant proteins is affected in critically ill patients with septic or cardiogenic shock. [5, 6, 7] Thrombin generation increases as a result of increased synthesis of pro-inflammatory cytokines and tissue factor. The downregulation of thrombin synthesis is obtained via activated PC that inactivates factors Va and VIIIa. The reduced hepatic synthesis of AT and PC predisposes to a hypercoagulable state that in our case is thought to be caused by a severe, rapidly progressive HF secondary to an uncommon condition such as NCC.
Abstract:
A 67-year-old man with type II diabetes, on metformin, was brought to the emergency department with nausea, vomiting, diarrhea, generalized weakness, and recent inability to stand. He had lost 30 pounds and become forgetful over the preceding 3 months. Neurological examination revealed mild generalized weakness with significant weakness in the lower extremities, compromised vibration and proprioception, and decreased sensation in a stocking-glove distribution. His deep tendon reflexes were hypoactive.

The complete blood count showed pancytopenia, with hypersegmented neutrophils and MCV of 107 (80-96 fl/red cell). Further laboratory results were: vitamin B12: 61 (normal 180-912 ng/L), methylmalonic acid: 24,890 (87-318 nmol/L) and homocysteine 244.1 (< 11.4 mmol/L). MRI of the cervical spine without contrast showed scattered areas of hyperintense T2 signal in the posterior columns of the spinal cord (Figure). Based on these findings and the patient’s clinical picture, including severe vitamin B12 deficiency, he was diagnosed with subacute combined degeneration (SCD) and given daily vitamin B12, 1000 mcg, IM x5. After 5 days, ataxia was decreased and he was able to get up from the bed. His mental status improved and 6 days later he was transferred to rehab. Two weeks later his B12 was corrected; except for mild anemia, CBC including MCV was normal, and neurological exam including mental status had returned to baseline.

Our patient demonstrated many signs and symptoms of B12 deficiency, including weakness, ataxia, forgetfulness, pancytopenia, and most notably, SCD: central and peripheral nervous system manifestations of decreased myelin sheath synthesis.

Vitamin B12, when ingested in its unbound form, binds with R-protein (also called haptocorrin or transcobalamin. R-protein, found in saliva, gastric and small intestinal secretions, protects vitamin B12 from acidic pH, allowing the B12/R-Protein complex to pass to the duodenum where it is digested by pancreatic enzymes, isolating B12 from R-protein and allowing it to combine with intrinsic factor. The Vitamin B12/Intrinsic Factor complex is taken up by the ileal surface receptor; this uptake is calcium-dependent.

Pathology of gastric or small intestinal mucosa or the pancreas can lead to vitamin B12 deficiency. Our patient did not have pernicious anemia, chronic pancreatitis or ileal mucosal abnormalities such as Crohn's or celiac disease. However, he had been taking metformin since his diagnosis with type II diabetes several years earlier.

Metformin, a primary therapy for type II diabetes, competes antagonistically for binding at the ileal surface receptor site, impeding B12/Intrinsic Factor transport into the enterocyte. About 10-30% of patients on metformin are reported to have vitamin B12 deficiency. It is likely that
our patient’s B12 deficiency and ensuing subacute combined degeneration was caused, at least in part, by metformin. In patients with Type II diabetes and peripheral neuropathy, consider metformin-induced B12 deficiency as a possible contributing factor.
Title: A case of Idiopathic C3 Glomerulopathy

Abstract: Glomerulonephritis is the 4th leading cause of ESRD in the US with almost 56,000 renal transplants done annually for the same reason. It is a heterogeneous syndrome with various histological and immunological changes in the glomerulus. Membranoproliferative glomerulonephritis (MPGN) is one of the subtypes denoting the characteristic proliferative injury pattern and accounting for approximately 7 to 10% of all biopsy-confirmed cases of glomerulonephritis. Rapid diagnosis of acute glomerulonephritis is critical for initiation of therapy and preserving renal function.

A 42 years old male presented with dyspnea, orthopnea, bilateral lower extremity edema and decreased urine output for 1 month. Vital signs were normal. Physical exam showed decreased bibasilar breath sounds and differential right lower extremity (RLE) edema. RLE ultrasound revealed deep vein thrombosis. Labs showed; BUN 51 mg/dl, Creatinine 3 mg/dl, Bicarbonate 13 meq/l, Albumin 1.5 g/dl and urinalysis with 2.6 g proteinuria, hematuria with dysmorphic RBCs.

Workup was negative for lupus, hepatitis B, C or HIV. Complement C3 levels were low with normal C4 levels. SPEP showed polyclonal gammopathy without M Proteins on serum immunofixation. Anti-Phospholipase-A2-Receptor (PLA2R) autoantibodies were positive. Renal ultrasound was normal.

The patient was started on empiric treatment for membranous glomerulonephritis with pulse steroids yet his renal function declined rapidly, he became anuric with volume overload requiring hemodialysis. Renal biopsy showed membranoproliferative glomerulonephritis with dominant C3 staining consistent with C3 glomerulopathy. One glomerulus had a cellular crescent and thus patient was started on plasmapheresis and cyclophosphamide therapy. Further workup came back positive for cryoglobulinemia. Bone marrow biopsy and CT imaging were unremarkable for underlying malignancy. Immunological work up including C3 nephritic factor, complement factor H (serum CFH) activity, serum factor I, serum F B levels, mutations for complement factor H related protein (CFHRP), membrane cofactor protein (MCP) level and soluble membrane attack complex levels (C5b-9) was unremarkable. The patient was managed as idiopathic C3 MPGN.

Acute glomerulonephritis (GN) with low complement levels is a characteristic and limited phenotype. It can be due to systemic diseases like lupus, subacute bacterial endocarditis, cryoglobulinemia or primary renal disease like acute post streptococcal GN and
membranoproliferative GN. Ruling out systemic causes, the histology helps differentiate between PSGN and MPGN, with the former being rich in immunoglobulin subendothelial deposits. Cause of MPGN can be elusive and patient should be thoroughly evaluated before labelling them “idiopathic” MPGN for a chance to offer targeted therapy. C3 glomerulonephritis (C3GN) is extremely rare cause of GN with an incidence of 2-3 cases/million. The characteristic picture on immunohistochemistry is the lack of staining for immunoglobulin with mesangial and capillary deposits for C3. No randomized control trials occur for optimum treatment of this uncommon disease. Treatment should be based on the underlying etiology.
Title: Ebstein’s anomaly presenting as a stroke in a young female.

Abstract:
Ebstein’s anomaly is a rare congenital heart disease with a wide spectrum of clinical presentation. We report a case of a 35-year-old female presenting with left middle cerebral artery paradoxical cardio-embolic stroke from Ebstein’s anomaly associated atrial septal defect (ASD) and a right to left shunt.
A 35-year-old female with no medical history presented with difficulty speaking and inappropriate behavior for five days. Her vitals were within normal limits and neurological exam showed expressive aphasia. Cardiovascular exam showed a fixed split S2. Electrocardiogram (EKG) showed normal sinus rhythm. Computerized tomography and magnetic resonance imaging of brain showed acute left temporoparietal infarct in the middle cerebral artery territory. Hypercoagulability and other secondary workup for stroke was negative. Transthoracic echocardiography showed apical displacement of septal leaflet of tricuspid valve with mild tricuspid regurgitation, atrialised right ventricle (RV) with a small true functional RV, atrial septal defect and a right to left shunt. The findings of Ebstein’s anomaly and ASD were confirmed on a subsequent transesophageal echocardiography (TEE) and cardiac magnetic resonance imaging (MRI). Lower extremity doppler was negative for any deep vein thrombosis. Case was discussed in multidisciplinary cardiology conference and patient was scheduled for surgical repair/replacement of tricuspid valve with concurrent ASD closure and put on warfarin in the meantime for cryptogenic stroke in the setting of ASD.
Ebstein’s anomaly is a rare congenital anomaly first described by Wilhelm Ebstein in 1866. It has a wide spectrum of presentation with oldest age of diagnosis reported in the ninth decade. The cardinal symptoms are cyanosis, right-sided heart failure, arrhythmias and sudden cardiac death. Clinical presentation depends on severity of tricuspid valve distortion and presence of other defects like ASD, right to left shunt, pulmonary stenosis or arrhythmias. Early ages of presentation reflects worse hemodynamics and have poorer prognosis with mortality of up to 40% by one month of age when diagnosed as neonates. EKG may show pre-excitation pathway (up to 25% have accessory conduction pathways) or the so-called “Himalayan P waves”. An inter-atrial communication is present in 80% to 94% of patients with Ebstein’s anomaly most commonly being ASD. There is often an associated right to left shunt across the ASD which represents higher pressure in the atrialised RV rather than pulmonary hypertension and Eisenmenger’s physiology. Patients with Ebstein’s anomaly almost never have pulmonary hypertension. Tricuspid valve repair/replacement with ASD closure, if present is indicated in patients with compromised exercise capacity, cyanosis/hypoxia with oxygen saturation < 90%,
paradoxical embolism, progressive cardiomegaly or RV systolic dysfunction. EP studies should be offered to patients with symptoms suggestive of arrhythmias or EKG findings suggestive of pre-excitation. Anticoagulation with warfarin is strongly recommended in patients with paradoxical embolism associated with Ebstein’s anomaly.
New onset facial droop in a woman with a rare granulomatous myopathy

Case:
A 70 year-old Caucasian female with past history of hypothyroidism and recently diagnosed granulomatous myopathy presents with 1 week of right sided facial droop. She was diagnosed with granulomatous myopathy due to isolated sarcoidosis approximately one year prior to current presentation. The diagnosis was made due to bilateral painless lower extremity weakness, elevated creatine phosphokinase (236 units/L), elevated corrected calcium (13.6 mg/dL), and muscle biopsy revealing granulomatous inflammation which stained negative for infectious or amyloid etiology. She denied any history of cardiac, pulmonary or integumentary involvement of sarcoidosis. She was treated with a prolonged prednisone taper with very minimal improvement in her lower extremity weakness. She had completed the steroid taper about one week prior to onset of her current symptoms of right sided facial droop. On this presentation, she had obvious drooping and muscle weakness in the distribution of the right cranial nerve VII, as well as occipital headache and right ear pain. She had slightly elevated serum angiotensin converting enzyme (50 units/L), elevated corrected calcium (11.9 mg/dL), and intracranial magnetic resonance imaging revealed abnormal enhancement of the intracranial segments of the facial nerves bilaterally. While neuroimaging showed characteristics of Bell’s Palsy, this may also be characteristics of neurosarcoidosis. Due to the patient’s known biopsy proven granulomatous disease, neurosarcoidosis of bilateral cranial nerves VII is a likely diagnosis. She was treated with 60 mg prednisone daily with rapid improvement in her 7th nerve palsy, however she continued to have weakness in her bilateral lower extremities. She was discharged with a slow steroid taper.

Discussion:
Sarcoidosis is characterized by noncaseating granulomatous infiltration in multiple organ systems. This disorder typically involves bilateral hilar lymph nodes, lungs, skin or eyes, however can involve any organ system in the body. While asymptomatic skeletal muscle involvement is common in sarcoidosis, symptomatic sarcoid myopathy causing severe weakness, as described in this case, is exceedingly rare and found in only 0.5-2.5% of sarcoidosis cases. Isolated sarcoid myopathy without other organ system involvement is even more rare and has only been described in a handful of cases. The diagnosis of isolated sarcoid myopathy requires muscle biopsy showing granuloma formation, without possibility of other granuloma forming diseases such as fungal infection or vasculitis. Our patient had this rare isolated sarcoioid myopathy and did not respond to steroid therapy. One year later she returned
with 7th cranial nerve palsy which may be explained by sarcoidosis involvement of the peripheral nervous system.

Conclusion: Sarcoidosis can rarely present with isolated skeletal muscle involvement. In addition, as seen in this case, with involvement of other organ systems such as the nervous system, a possibility of neurosarcoidosis remains to be explored as the disease progresses.
**Title:** An under-recognized cause of low back pain - It would be RASH to Ignore it.

**Abstract:**
Case Report
59 year old lady with poorly controlled diabetes mellitus was admitted for one week of intractable low back pain radiating to the right leg accompanied by numbness of the dorsal foot and great toe, she denied lower extremity weakness or loss of bowel and bladder continence, denied recent trauma, fevers, chills or intravenous drug use. Vital signs were within normal limits. Neurologic exam was significant for weakness of right foot dorsiflexion, Knee and ankle jerks were normal and there were no sensory abnormalities. Labs were significant for hyperglycemia to 514 mg/dl, otherwise complete blood count, basic metabolic panel and urinalysis were unrevealing. CT of the lumbar spine was negative for fracture. MRI of the lumbar spine showed no herniated disc. Cerebrospinal fluid (CSF) analysis revealed lymphocytic pleocytosis with normal protein and glucose levels. Polymerase chain reaction (PCR) of cerebrospinal fluid eventually disclosed VZV DNA. Interestingly two days after admission to the hospital a typical zoster exanthem involving the left L5 dermatome appeared. Patient was started on oral valacyclovir that lead to rapid decrease in pain and exanthem and patient was discharged to complete a seven day course of oral valacyclovir.

**Discussion**
Varicella Zoster Virus is an exclusively neurotropic virus that causes varicella (chickenpox) during primary infection after which the virus becomes latent in cranial nerve, dorsal root and autonomic ganglia along the entire neuroaxis. Decades later virus may reactivate to cause (shingles) An extremely painful vesicular skin rash confined to one or more sensory dermatomes, the incidence of zoster is increased in elderly and immunocompromised individuals. VZV reactivation can also lead to multiple other neurologic and ocular disorders including vasculopathy, myelopathy, meningoencephalitis, Cerebellitis and necrotizing retinitis. However, VZV reactivation can produce radicular pain without rash “Zoster sine herpete” (ZSH) making this diagnosis more challenging. The appropriate investigations to establish a diagnosis of ZSH are PCR for VZV DNA and anti-VZV IgG in CSF. The work up of radiculopathy in elderly immunocompromised patients with normal MRI of the lumbosacral spine should include CSF analysis with PCR for VZV-DNA even in the absence of classic rash as early diagnosis and treatment can prevent significant morbidity.

**Conclusion**
Herpes zoster infection can mimic lumbosacral radiculopathy caused by disc herniation, especially in elderly immunocompromised patients thus zoster radiculitis should be included in the differential diagnosis of Low back pain. The work up of radiculopathy in elderly immunocompromised patients with normal MRI of the lumbosacral spine should include CSF analysis with PCR for VZV-DNA even in the absence of typical rash.
**Abstract:**

Spontaneous Psoas muscle hemorrhage (SPH) is a rare, deadly complication of Alcoholic Liver Cirrhosis (ALC) and requires a high index of suspicion in patients presenting with hemodynamic instability and anemia without overt bleeding. We present a patient with ALC, found to have SPH and survived.

A 59-year-old man with Child C Alcoholic cirrhosis (MELD 27) was admitted with 2 months of abdominal distension. At presentation, vital signs were normal; he had distended abdomen and bilateral pitting pedal edema. Workup revealed hemoglobin (Hb) 12.9 g/dL, platelet count of 55 k/ul, serum creatinine 2.5 mg/dL, total bilirubin 5.1 mg/dl, Albumin of 3 g/dl and INR 1.6. CT Abdomen showed a right liver lobe arterially enhancing mass consistent with Hepatocellular Carcinoma (HCC). Bilateral lower extremity Sequential Compression Device was placed as venous thromboembolism prophylaxis. On day 3, the patient complained of right groin pain, he was hypotensive, tachycardic, right hip flexion elicited inguinal pain. His Hb dropped to 7g/dl. He was transferred to the Intensive Care Unit (ICU), and started on vasopressors and given blood products. Repeat CT abdomen revealed a right psoas hematoma. Interventional Radiology (IR) was unable to localize the bleeding vessel for embolization. The General Surgery service assessment was that peri-operative mortality risk was too high for intervention. To prevent abdominal compartment syndrome, he had paracentesis with removal of 2.2L of hemorrhagic fluid. He received 5 units packed RBC, 1 unit of platelets and 9 units of FFP in total. After 5 days in the ICU, his hemoglobin stabilized above 8 g/dl, he was transferred to General Medicine and discharged home.

In Cirrhosis, there is reduced synthesis of pro-coagulant factors and fibrinogen, which contributes to increased bleeding risk. In a case series of cirrhotic patients with muscle or retroperitoneal bleeding, 71.4% of the cirrhosis was caused by Alcohol. Alcohol intake directly leads to impaired platelet function, decreases fibrinogen, factor VII, and vWF levels and leads to increased fibrinolysis. Also, in a case series of patients, not on anticoagulation, who developed spontaneous muscle hemorrhage, mean INR was 1.2 ± 0.3 which suggests INR may not correlate with bleeding risk. Management of SPH can be supportive or aggressive with either surgical or intra-arterial embolization. Panetta et al. found that hemodynamic instability despite four or more units of blood transfusion within 24 h is an indication for urgent endovascular treatment. Open surgeries are indicated if the patient remains unstable or if IR is unsuccessful or unavailable. However, there is a linear increase in the relative risk of death for MELD scores...
greater than 8. Our patient was not a good surgical candidate because of his high MELD and survived after conservative treatment.
Last Name: Weiner  
First Author: Resident

First Name: Rebecca  
Category: Clinical Vignette

ACP Member: 2889583

Additional Authors:

Title: Zika Virus, Persistent Arthralgias, and a Positive Anti-CCP Antibody: A Conundrum in Diagnosis and Management

Abstract:
Zika virus is a flavivirus transmitted by the Aedes mosquito with protean manifestations. It is associated with neurologic and other autoimmune phenomenon, however, it is uncertain if it can potentiate an already underlying autoimmune predisposition.

A 51 year old Caucasian female presented three days after returning from Nicaragua, complaining of low grade fevers, rash, and neck pain. On exam, anterior/posterior cervical lymphadenopathy and a diffuse maculopapular rash were present. Reference labs for Zika, Dengue fever, and Chikungunya were obtained, and Zika virus serum RNA PCR was positive. Progressively worsening symptoms of low grade fever, conjunctival injection, and diffuse arthralgias prompted referral to rheumatology. Years prior, rheumatology evaluated her for hand swelling and positive anti-ccp antibody at 10.3 (normal <3) with negative rheumatoid factor and antinuclear antibody. At that time, she was not diagnosed with rheumatoid arthritis (RA) and her positive serology was attributed to smoking. Current rheumatology consultation revealed a positive anti-ccp at 74.1; assessment was negative for synovitis. Her symptoms were attributed to Zika virus infection; treatment consisted of NSAIDS. Clinical course was complicated by parasthesias and EMG demonstrated polynuropathy and demyelination attributed to Zika induced Guillain-Barre syndrome (GBS). She received three rounds of intravenous immunoglobulin with improvement in neuropathic symptoms and her arthralgias persisted. On follow up exam, trace swelling of the small joints of hands and diffuse muscular tender points were noted. She was diagnosed with seropositive RA and fibromyalgia, and given prednisone, which decreased joint swelling, but did not minimize her pain; methotrexate was added. Despite these interventions polyarthralgias persisted and small joint imaging showed no erosive changes. Ultimately, she was labeled “post-Zika syndrome” as she did not meet strict criteria for RA, and immunosuppression was discontinued. Her diagnosis and management remain a question one year later as her arthralgias have persisted longer than expected for typical transient rheumatic complications of viral arthropathies.

When Zika virus infection occurs in a patient with pre-existing positive autoimmune serology or diseases, differential diagnosis and management becomes quite challenging given the number of autoimmune phenomenon that Zika virus has been associated with. While unknown, one can postulate that this patient had a propensity to develop GBS based on her history of positive autoimmune serology. This case raises questions of how viral induced arthropathies interplay or
potentiate other autoimmune phenomenon, such as anti-ccp antibody, to prolong the course of polyarthralgias and chronic pain. Zika virus has been reported as a transient cause of arthralgias, most cases are mild and self-limited, unlike this case presentation. It may be beneficial to advise patients with autoimmune disease predilections to avoid travel to endemic areas of flaviviruses, or to take extra precautions against mosquito exposure to avoid potential sequela.
Title: Ring-Enhancing Lesions in Brain due to Mycobacterium avium-intracellulare Complex in Acquired Immunodeficiency Syndrome

Abstract:
Introduction: Mycobacterium avium-intracellulare complex (MAI) infections commonly present in a disseminated manner. Symptoms can be nonspecific and may include fever, night sweats, weight loss, vomiting, diarrhea, and abdominal pain. MAI can commonly affect the reticuloendothelial system, but can also affect the lungs and the brain in rare cases. Traditionally, ring-enhancing lesions in acquired immunodeficiency syndrome (AIDS) have been most commonly attributed to Toxoplasma infections or central nervous system (CNS) lymphoma. However, the possibility of unusual manifestations of MAI infections underscore the importance of formulating a broad differential diagnosis for ring-enhancing lesions in brain.

Case Presentation: The patient was a 44-year old male with poorly controlled human immunodeficiency virus (HIV) infection, AIDS, and adrenal insufficiency who initially presented to an outside hospital for altered mental status. Initial evaluation included computed tomography of the head, which was notable for bilateral frontal lobe lesions with extensive edema and a 7 mm midline shift. The patient was transferred to Loyola University Medical Center (LUMC) for further evaluation and neurosurgical consultation. Initial testing at LUMC revealed CD4 lymphocyte count of 77 cells/μL and HIV viral load of 70 copies/mL. Magnetic resonance imaging of brain showed multiple periventricular ring-enhancing lesions. Serologic testing for Toxoplasma was negative. Quantitative polymerase chain reaction tests for Epstein-Barr virus and cytomegalovirus were also negative. Lumbar puncture was obtained, and the cerebrospinal fluid (CSF) analysis showed low glucose (41 mg/dL) and elevated protein (108 mg/dL) levels. However, bacterial, fungal, and acid-fast bacteria (AFB) cultures of the CSF were all negative. Subsequently, neurosurgery was consulted and the patient underwent stereotactic biopsy of the brain lesion. Histology indicated presence of Mycobacterium, and AFB culture of the brain biopsy specimen confirmed Mycobacterium avium-intracellulare complex.

Management and Outcome: The infectious disease service was consulted. After the AFB culture of brain biopsy with sensitivity testing was finalized, the patient was started on azithromycin, ethambutol, and moxifloxacin for anti-mycobacterial treatment. He was also continued initially on his home antiretroviral therapy including darunavir, ritonavir, stavudine, and zidovudine, but stavudine was later switched to dolutegravir. His initial presenting complaint of altered mental status showed gradual improvement with treatment. He was transferred to acute inpatient rehabilitation facility for further recovery on hospital day 32.
Discussion: With the widespread use of combination antiretroviral therapy, MAI incidence has significantly decreased since the 1980-1990s. Even so, there have been few cases of MAI brain abscesses reported, mostly in HIV-infected or AIDS patients. While toxoplasmosis and CNS lymphoma are common causes of ring-enhancing lesions in immunocompromised patients, MAI infection should be also considered on the differential diagnosis. This case illustrates the importance of recognizing unusual disease manifestations in order to provide timely and appropriate therapies.
Abstract:
Rationale: Acute respiratory distress syndrome (ARDS) is a severe form of non-cardiogenic pulmonary edema that is under-recognized in clinical settings, with only 60.2% of cases recognized by clinicians. Previously, an automated electronic screening tool for ARDS was developed and retrospectively validated in critically ill patients with cirrhosis. We aim to evaluate the test characteristics of this screening tool prospectively in all critically ill patients admitted to the medical intensive care unit (MICU) at a tertiary academic medical center.

Methods: The authors have previously designed an automated electronic screening tool in a structured query language (SQL)-based integrative database from the institution’s clinical research database. Electronic health records (EHR) served as the data source for the rule-based approach with linked mechanical ventilation data, arterial blood gas data, and keywords from chest imaging reports that fulfill the Berlin definition. The automated tool prospectively analyzed the EHR of critically ill patients within 24 hours of admission to the MICU, on a daily basis between March 1, 2017 and July 31, 2017. Providers were alerted electronically once the patients screened positive for ARDS. The pulmonary and critical care physician responsible for the patient’s care served as the reference standard. The automated tool’s performance was evaluated by calculating its sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV) against the reference standard.

Results: A total of 217 patients admitted to MICU were analyzed by the automated screening tool. Thirty-nine patients (18.0%) screened positive by the automated tool for ARDS. Of these, the provider agreed with the ARDS diagnosis in 16 patients (41.0%), and disagreed in 23 patients (59.0%). Common reasons for false positives included inability to distinguish hydrostatic pulmonary edema from ARDS and inappropriate pairing of laboratory and mechanical ventilation data for the fraction of arterial-to-inspired oxygen ratio. Five patients (2.3%) were identified by the provider as having ARDS, but were not screened positive by the screening tool. Common reasons for false negatives were variations in semantics and syntax in the chest radiology report that did not qualify as a keyword in the automated rule-based approach. The automated screening tool had a sensitivity of 76.2% (95% confidence interval [CI] 52.8-91.8%), specificity of 88.3% (95% CI 82.9-92.4%), PPV of 41.0% (95% CI 30.7-52.2%), and NPV of 97.2% (95% CI 94.1-98.7%).
Conclusions: The prospective validation of an automated screening tool demonstrated good sensitivity for early identification of ARDS in critically ill patients. Disagreements in distinguishing hydrostatic pulmonary edema contributed to the majority of false positive cases. Improving methods in linguistic processing could improve the test characteristics of the automated system, and its implementation as a tool for clinical decision support can facilitate early identification and treatment of ARDS.
Spontaneous bladder rupture: when ascites is not ascites and renal failure is not renal failure

Abstract:
A 46-year-old man presented with a two-day history of abdominal pain and decreased micturition after a weekend of heavy alcohol consumption. The patient lost consciousness after the binge and awoke the next day with the above symptoms. His medications include daily celecoxib 200mg and losartan-hydrochlorothiazide 100-25mg for his shoulder osteoarthritis and hypertension, respectively. Vital signs were remarkable for blood pressure 190/110 and heart rate 113 beats/min. Blood work was notable for the following: sodium 124, chloride 90, bicarbonate 16, BUN 63, creatinine 6.39, albumin 4.4, total bilirubin 1.5, alkaline phosphatase 131, AST 97 and CPK 2923. Arterial blood gas showed pH 7.298, pCO2 38 and pO2 96. Urinalysis showed small blood and 5 red blood cells. CT of the abdomen/pelvis demonstrated large volume ascites but no urinary obstruction. The patient underwent paracentesis with clear fluid output and studies consistent with portal hypertension. Despite multiple fluid boluses, there was no evidence of renal recovery. A Foley catheter was placed with an immediate 5 liters of diuresis. Due to suspicions for bladder injury, the ascitic fluid was analyzed for creatinine and returned elevated to 27.42. A CT cystogram demonstrated contrast extravasation into the peritoneal cavity. Laparoscopic repair confirmed the diagnosis of bladder rupture and revealed a 1 cm defect in the wall. Majority of bladder rupture cases is reported in association with a history of bladder disease, such as neurogenic bladder or history of pelvic radiotherapy. In rare instances, bladder rupture occurs spontaneously. Nonetheless, there are a few reports describing spontaneous bladder rupture in the context of alcohol intoxication. It is speculated that alcohol intoxication may lead to a decreased sensorium and response to bladder filling. The volume of alcohol ingested and its diuretic effect further increase bladder filling. The combination of the two places an already stretched bladder at risk, such that even minor trauma can cause it to rupture. Once recognized, it is treated surgically with good prognosis. Historically, bladder rupture has been associated with significant mortality since most cases experience a diagnostic delay due to its nonspecific presenting symptoms. Furthermore, serum electrolytes can appear suggestive of acute kidney injury (AKI), when in reality, it is a reflection of the reabsorption of urine across the peritoneum that causes an increase in serum urea and creatinine. In our case, it was not until the placement of the Foley catheter and the large amount of output that followed that led to suspicions of bladder rupture. This prompted analysis of the ascitic fluid for creatinine, revealing that the patient’s ascites was actually composed of urine. This case highlights the importance of recognizing key features of
spontaneous bladder rupture, which classically masquerades as abdominal pain in the context of ascites and AKI.
Title: Pylephlebitis – A rare and dangerous complication of diverticulitis

Abstract:
Pylephlebitis is the infective thrombosis of the portal vein or one of its tributaries. It can complicate any type of abdominal sepsis and is a rare but dangerous cause of morbidity and mortality. The most common cause of pylephlebitis is diverticulitis but pylephlebitis remains one of the rarest complications of diverticulitis with no exact numbers for epidemiology. 48 year old male with a past medical history of HIV and diverticulitis presented to the ED with a high grade fever of 104 F for the past 2 days. He denied any abdominal pain, nausea, diarrhea or blood in his stool. He was also complaining of a headache and photophobia with no neurological deficits. He did endorse partial neck stiffness. He denied any history of recent travel or sick contacts. He had had 2 episodes of acute diverticulitis in the past 2 years and each time had presented with severe abdominal pain and LLQ tenderness associated with nausea and vomiting. His physical exam was unremarkable except for his fever. His initial work up showed a mildly elevated white count of 11.4. With this information in mind, he was initially worked up for meningitis with an LP and CT head which were normal. He was then sent home from the ED with cefoxitin as all his work up was negative with instructions to return if his symptoms did not resolve. He returned to the ED the next day as his condition had not improved. Blood cultures were sent and he underwent a CT abdomen to evaluate for an atypical presentation of diverticulitis. His CT abdomen showed acute sigmoid diverticulitis and inferior mesenteric thrombophlebitis. GI, ID and General Surgery were consulted and flagyl was added to his medications. At this point, his blood cultures came back positive for E coli resistant to ciprofloxacin and his antibiotics were adjusted. He was diagnosed to have pylephlebitis complicating acute diverticulitis and started on anticoagulation alongwith the antibiotics. His condition improved with treatment, his white count normalized and his fever resolved. General surgery discussed the possibility of a colectomy due to his multiple episodes of acute diverticulitis and he was instructed to follow with them in 2 weeks after discharge to plan the procedure. He remained stable and so was discharged with ID, GI and GS follow up. This case represents the importance of a careful history and shows the spectrum of diverse presentations of diverticulitis. It also casts light on a rare but dangerous complication of diverticulitis – pylephlebitis – which requires immediate attention and treatment. Once a universally fatal complication, it now has improved outcomes due to the advent of antibiotics. Hence, swift recognition and treatment can be lifesaving.
Illinois Chapter
Northern Region

Top E-Posters

University of Illinois at Chicago
November 8, 2017
Last Name: Abou Mrad   First Author: Rachel

Category: Clinical Vignette

ACP Member: 3362172

Additional Authors: Mahmud Samra, Rasheed Hammadeh, Firas Aubeid, Armand Krikorian

Title: Colonic large cell neuroendocrine carcinoma: A rare and aggressive tumor

Abstract:
Neuroendocrine tumors (NETs) are rare epithelial neoplasms with predominant neuroendocrine differentiation. They can arise in different organs, have clinical and pathologic features unique to the site of origin and also share common characteristics. The annual incidence in the USA of NETs of the digestive system is about 3.65 per 100,000 population. Colonic NETs in particular are rare with only 0.6% of patients with colorectal cancer having NET and 0.2% of those were large cell neuroendocrine neoplasms.

A 62 yo male presented to the emergency department with abdominal pain and change in bowel habits. No nausea, vomiting, melena or hematochezia. Physical examination was positive for mild right upper quadrant tenderness. Laboratory work up revealed microcytic anemia with a normal complete metabolic profile. A CT scan of the abdomen and pelvis showed a colonic mass at and above the ileocecal valve with full thickness and adjacent fat involvement. A colonoscopy confirmed the presence of a malignant looking cecal mass. The patient subsequently underwent a right hemicolecotomy and primary anastomosis. On pathology, the tumor cells exhibited ample cytoplasm and numerous mitotic figures. Immunohistochemistry showed diffuse cytoplasmic staining for synaptophysin and focal staining for chromogranin A, consistent with the diagnosis of large cell neuroendocrine carcinoma of the colon (LCNEC). A PET scan was positive for hypermetabolic abdominal para-aortic lymph nodes. Patient was started on a chemotherapy regimen based on cisplatin/etoposide. A follow up PET scan showed a significant decrease in hypermetabolic activity of abdominal lymphadenopathy. LCNEC is a rare and aggressive subtype of NETs characterized by high mitotic index and neuroendocrine appearance under light microscopy. Most are located in the cecum and rectum and most are metastatic at presentation. Prognosis is poor for all stages of disease with median survival of 38 months for localized disease and 5 months for metastatic disease. While surgery is the primary treatment modality, the benefit of chemo- or radiation therapy, as used for conventional colorectal adenocarcinoma, has not been established for these tumors. Our case consists of a rarely described localized LCNEC with a favorable response to adjuvant chemotherapy. In the literature the only factor associated with a favorable outcome was the absence of metastatic disease. Age, gender and site of the tumor did not affect overall survival.
Dry mouth and iron is the culprit: A case of sicca syndrome associated with haemochromatosis.

Case report:
A 35-year-old male came to the outpatient clinic with complaints of fatigue, dry eyes, dry mouth and occasional sore throat together with non-specific hand tremors and generalized joint pains for the past several years. His past medical history was significant for Raynaud phenomenon treated with Nifedipine. Physical examination revealed dry mucus membranes, no abnormal pigmentation of the skin, soft abdomen without organomegaly, and no adenopathy or salivary gland enlargement. Laboratory workup revealed negative ANA, borderline low level of C3 and C4 [C3 81 mg/dl and C4 14.6 mg/dl], negative serology for Sjogren’s A and B antibodies, normal ESR, CRP. Initially, the patient was diagnosed with sicca syndrome and was treated with pilocarpine and hydroxychloroquine which helped with the dryness, however the fatigue persisted.

Few months later, his hand tremors worsened and he developed transient unsteadiness, but had no focal weakness, sensory symptoms, vertigo, or vision changes. Lab work including CBC, CMP, B12/Folate, TSH, HbA1c, homocysteine, and methyl malonic acid were all normal. His hand tremors remained undiagnosed and he received no treatment. A year later he developed right upper quadrant pain with tenderness. Abdominal ultrasound showed no gall stones and abdominal CT was normal. Further laboratory work revealed slightly elevated liver enzymes with total bilirubin 0.3 mg/dL. Patient had negative serology for HCV and HBV. Iron studies showed elevated serum ferritin level at 535 ng/mL, TIBC was 232 mcg/dL, and iron saturation was 61%. Genetic analysis showed that he was a carrier of the haemochromatosis-associated alleles C282Y/H63D. He was diagnosed with hemochromatosis. He was treated with maintenance phlebotomies and with this treatment he remained well without evidence of disease progression.

Hereditary hemochromatosis is the most common genetic disorder in Caucasians. Despite the increased awareness of hemochromatosis, the disease is under diagnosed. In
hemochromatosis, iron deposits within various organs lead to organ dysfunction. When the disease is fully established clinical findings include liver cirrhosis, cardiomyopathy, hypogonadism, diabetes mellitus, and skin pigmentation. In early stages, the disease is either asymptomatic or can present with nonspecific symptoms such as weakness, fatigue, and joint pain. Other rare manifestations include night sweats, movement disorders such as chorea Parkinsonism, and hand tremor, sicca-like symptoms including dry eyes and mouth, as well as nonspecific GI symptoms. Our patient had a long-standing constellation of nonspecific symptoms including hand tremors, arthralgia, and sicca-like symptoms, all of which can be explained by hemochromatosis. Our report emphasizes not only the importance of early recognition of hemochromatosis, but also highlights the association between serology negative Sjogren’s syndrome and hereditary haemochromatosis.
A 54 years old man presented with gait unsteadiness, palpitations, and tremors for 10 months. He denied diplopia, dysphagia and sensory deficits. Physical examination was remarkable for; heart rate 104/min, hand tremors, diffuse non-tender goiter, dysarthria, bilateral horizontal nystagmus, ataxic wide based gait, dysdiadochokinesia and 3+ bilateral knee reflexes with intact sensations. His initial work up, at an outside hospital, with MRI Brain and lumbar puncture was unremarkable.

Clinical presentation was consistent with a cerebellar syndrome with presumed etiologies as; paraneoplastic, autoimmune, post viral or degenerative ataxia. Labs were remarkable for normal CBC, CMP, RPR, vitamin E, B12, lactate, pyruvate, anti-gliadin antibodies. Thyroid profile showed; TSH <0.015, T4 3.61, TPO antibodies 104 (Normal<9), TSI 293 (Normal<140). Thyroid ultrasound showed increased vascularity. Neurological imaging with CT head, MRI/MRA brain was unrevealing of any pathology. CSF analysis was remarkable for normal protein and cell counts, negative paraneoplastic antibody panel and oligoclonal bands. CSF fungal and mycobacterial cultures showed no growth.

Diagnosis of Graves' disease was made, based on clinical and biochemical evidence of thyrotoxicosis in the setting of TPO and TSI antibodies. The patient responded to metoprolol and methimazole, with improvement in tremors. Given the negative work up for structural, metabolic, infectious, and vascular or paraneoplastic etiologies of cerebellar dysfunction, symptoms were attributed to autoimmune brain disease associated with Graves’ disease. Definite treatment with radioactive iodine ablation therapy resulted in clinical and biochemical resolution of hyperthyroidism. Patient has demonstrated complete recovery of cerebellar signs and symptoms on subsequent outpatient follow up.
The neurological disorder associated with thyroid autoimmunity can present as Hashimoto encephalopathy, cerebellar ataxia, exacerbation of myasthenia gravis and peripheral neuropathy. Cerebellar ataxia is a known feature of Hashimoto thyroiditis in the setting of hypothyroidism, but it’s a rare presentation of hyperthyroidism related to Graves' disease. To the best of our knowledge, only a few cases have been reported. Recurrent and reversible episodes of cerebellar ataxia with concomitant episodes of hyperthyroidism have been reported, highlighting the importance of achieving and maintaining a euthyroid state. Structural, degenerative, paraneoplastic and infectious etiologies need to be ruled out by neurological imaging and CSF analysis. Thyroid function tests and the presence of thyroid antibodies make the diagnosis of Graves’ associated cerebellar ataxia once all other possibilities have been excluded. Accurate diagnosis in these patients is critical because they may be misdiagnosed as spinocerebellar ataxia. Prognosis is good since patients respond to anti-thyroid medications and RAI ablation.
Title: From Hypo to Hyper: Post-partum Hashimoto Thyroiditis Complicated with New Onset Graves’ Disease.

Abstract:

Introduction:
This case demonstrates the unusual occurrence of two distinct autoimmune diseases within the post-partum period of the same pregnancy. A previously healthy young female with post-partum depression was originally diagnosed with hypothyroidism due to Hashimoto’s thyroiditis. One year later, she underwent transition to the hyperthyroid state and the diagnosis of Graves’ disease was confirmed.

Case:
A 27-year-old Hispanic female was referred to the endocrine clinic for evaluation of abnormal thyroid function test. She had delivery complicated with postpartum depression treated with sertraline. She had no past medical history or family history of thyroid or autoimmune diseases. Physical examination was unremarkable. Six months following delivery she presented with hyperthyroidism with a thyroid stimulating hormone (TSH): 0.025 (0.35-5.0 mcU/ml), free thyroxine (FT4): 1.7 (0.8-1.5 ng/dl), free triiodothyronine (FT3): 4.1 (2.2-4.0 pg/ml). Findings were suggestive of post-partum thyroiditis. Six weeks later, she developed severe hypothyroidism with TSH: 35 mcU/ml, FT4: 0.5 ng/dl. She also had markedly elevated serum anti-thyroglobulin antibody: 35.7 (<0.0-4.0 IU/ml) and anti-microsomal antibody: 1787 (<60 IU/ml). Accordingly, she was diagnosed with Hashimoto thyroiditis and started on levothyroxine replacement. One year later she developed increased episodes of palpitations, anxiety, heat intolerance and fatigue. She was clinically hyperthyroid and repeat testing revealed severely suppressed TSH: < 0.008 mcU/ml, FT4: 3.8 ng/dl, FT3: 12.2 pg/ml. Her levothyroxine was discontinued. Upon follow up one month later, repeat testing showed persistently suppressed TSH: < 0.01 mcU/ml. Surprisingly, she also had markedly elevated thyroid stimulating immunoglobulin :1756 (<150%). A diagnosis of Graves’ disease was confirmed. She was treated with β-blockers and Methimazole which helped controlling her symptoms.

Pregnancy is associated with variable adaptive metabolic, hemodynamic and immunological changes that affects the function of the thyroid gland. Different types of autoimmune thyroid disease may occur during pregnancy and post-partum period, including Graves’ disease, Hashimoto’s thyroiditis and postpartum thyroiditis. The relationship between postpartum depression and autoimmune thyroid disease was investigated in multiple studies with no firm conclusion to be reached. The variation in immune tolerance during pregnancy might play a
role in triggering thyroid autoimmunity. No other risk factor for autoimmune thyroid dysfunction was identified in our patient. Although the incidence of postpartum autoimmune disorders of endocrine glands is frequently reported, the development of two distinct autoimmune diseases following same pregnancy is extremely rare. Our patient developed Hashimoto thyroiditis treated with thyroxine and converted a year later to Graves’ disease. Further studies are needed to investigate this abnormal clinical course.
Abstract:
Introduction:
Sclerotic bone lesions often represent osteoblastic metastasis of select tumors known to spread to bone. However, not all sclerotic appearing lesions are osteoblastic metastases. This case reports a patient whose diffuse sclerotic lesions were a direct manifestation of his progression to acute leukemia.

Case Description:
An 84-year-old man with past medical history of myelodysplastic syndrome (with thrombocytopenia), hypertension, and chronic lower back pain presented to the emergency department with 1-week of acute on chronic lower back pain which he attributes to having overexerted himself with yard work. On history he endorsed 14-pound weight loss over the previous two months. On exam he had mild tenderness to palpation of his lumbar spine, range of motion at this hip was limited due to pain. He had no signs or symptoms of spinal cord compression. Laboratory findings were significant for serum calcium within normal limits (9 mg/dL corrected for albumin), serum alkaline phosphatase of 794 IU/L, and protein specific antigen < 3 ng/mL. CT imaging showed sclerotic lesions within the vertebra of T12 and L1 concerning for metastatic disease. Follow up MRI showed widely disseminated vertebral lesions, primarily in the thoracic and lumbar segments, consistent with osseous metastasis. Bone biopsy of the left anterior iliac crest yielded hyper cellular marrow with immature myeloid cells, consistent with a high grade myeloid neoplasm. Possible etiologies included high-grade myelodysplastic syndrome and acute myeloid leukemia. Ultimately, patient underwent prophylactic femoral neck pinning per concern for pathologic fracture and was referred for outpatient chemotherapy.

Discussion:
Given the patient’s history of bone pain, lab findings consistent with an osteoblastic process (high alkaline phosphatase, low calcium), and images concerning for metastatic disease we did not suspect leukemia until the bone pathology was read. We originally narrowed the differential to include the classic osteoblastic tumors: prostate, small cell lung cancer, hodgkin lymphoma, and carcinoid. However, prostate specific antigen was low, imaging did not suggest small cell lung cancer, and there was no history to suspect carcinoid (no diarrhea, no flushing). Upon literature review, there have been case reports of sclerotic bone lesions caused by
leukemic bone remodeling, with at least one instance accompanied by hypocalcemia driven by sclerotic bone lesions.
A 24-year-old man with PMH of recurrent bronchitis and pneumonia presented to the ED with shortness of breath for 4 days. One month prior to presentation he was hospitalized elsewhere for aspiration pneumonia. An extensive workup for infectious, immunologic, and hematologic causes was negative. At presentation the patient was afebrile with heart rate 133 beats/minute and respiratory rate 22 breaths/minute. WBC was 25.1 cells/microliter; respiratory PCR panel was positive for rhinovirus. Chest CT showed bronchiectasis with patchy infiltrates and mediastinal necrotic lymphadenopathy.

The patient was admitted to ICU for acute respiratory failure and atypical pneumonia, and placed on bipap. Repeat infectious workup was negative. Abdominal CT showed retroperitoneal adenopathy. CT-guided biopsy of retroperitoneal lymph nodes revealed polygonal cells with eosinophilic cytoplasm, cells with cytoplasmic mucin vacuoles, and cells with prominent nucleoli and mitoses consistent with metastatic mucoepidermoid carcinoma. The plan was for palliative chemotherapy. However the patient had a cardiac arrest and could not be resuscitated.

Mucoepidermoid carcinoma (MEC) tumors of the lung are rare, comprising fewer than 0.1% of primary lung tumors. They derive from salivary gland-like epithelium located in the tracheobronchial tree. Men and women are equally affected. Most affect young people in their 20s and 30s, and non-smokers. MEC presents with nonspecific respiratory symptoms including dyspnea, cough, fevers, and hemoptysis. Because of this, diagnosis is often delayed. Chest xrays show solitary nodules in the proximal tracheobronchial tree which eventually enlarge and cause obstruction. Definitive diagnosis is by biopsy.

Mucoepidermoid tumors can be either low-grade or high-grade. Classification is determined by morphologic, histologic, and prognostic features. Grossly, low-grade MEC tumors tend to be well-circumscribed and noninvasive. Microscopically, tissue shows a mix of solid, cystic and sheet-like intermediate cells separated by mucus-filled cysts. These patients have a much better prognosis than those with high-grade tumors. Surgery is usually curative. High-grade tumors are larger and more likely to infiltrate the parenchyma. Histologically, they have mucus-producing cells, mitoses, cell atypia, and necrotic tissue. Patients with these tumors do not benefit from surgery. Median survival after diagnosis is less than 12 months.
Our patient had a history of chronic respiratory symptoms. Chest CT findings included necrotic tissue. On admission to our hospital, he was in acute respiratory failure; bronchoscopy was contraindicated. Tissue obtained from retroperitoneal lymph nodes showed mitoses, and cells with cytoplasmic mucin vacuoles and prominent nucleoli. His presentation was consistent with high-grade tumor and, unfortunately, his prognosis was grim.
Sclerosing Encapsulating Peritonitis is a rare chronic inflammatory condition often seen as a consequence in less than 5% of patients being treated with peritoneal dialysis. A prompt diagnosis is essential due to its often-fatal outcome with a mortality rate as high as 55%. A 32-year-old female with a history of lupus complicated by end-stage renal disease presents to the emergency room with persistent right lower abdominal and pelvic pain accompanied by a 16-pound weight loss in one month. The pain is located in her right lower quadrant and radiates to her back. She also complains of weakness, nausea, and decreased appetite. Physical exam findings are notable for extreme RLQ tenderness and pain. Lab findings are significant for leukocytosis. Further history exposed that she was recently treated for sepsis secondary to pseudomonas peritonitis from an infected peritoneal dialysis catheter. On day one of hospitalization, Computed Tomography with contrast of the abdomen and pelvis showed scattered multiloculated fluid collections, one of which was drained via ultrasound guided paracentesis. The 100-cc specimen yielded no growth on culture with normal cell counts and provided minimal relief to the patient. Two subsequent paracentesis attempts yielded minimal fluid (less than 20-cc) with normal cell counts and no growth on culture. Laparoscopic surgery was discussed with the patient, however she elected to continue with percutaneous drainage attempts only. A multidisciplinary discussion between internal medicine, interventional radiology, nephrology, and infectious disease determined that two catheters were to be placed in order to facilitate continuous therapeutic and diagnostic drainage until further diagnosis could be made. On day 4, she was started on low dose steroids for extensive peritoneal inflammation, but this did not change the clinical course. On day 7 after repeatedly negative fluid samples and highly suspicious multiloculated peritoneal fluid collections, high dose steroids and tamoxifen were started due to suspected sclerosing encapsulating peritonitis. This suspicion arose from the patient’s previous bout of peritonitis, thickened peritoneum, multiple negative fluid cultures, recurrent and persistent abdominal pain accompanied by elevated inflammatory markers. Furthermore, the patient’s recent history of peritoneal dialysis and peritonitis just one month prior suggests a correlation with sclerosing encapsulating peritonitis. On day 11, the patient reports improvement in pain for the first time with concurrent improvement in physical exam and lab findings. Upon discharge on day 14, all drains have now been removed and the patient is transitioned to oral medications with recommendations for close outpatient follow-up.
This case portrays a rare outcome for a patient suffering with this often-fatal condition. Due to its rarity and nonspecific clinical features, diligent history taking, strong clinical suspicion, and a multi-disciplinary approach is imperative for a favorable prognosis.
The use of weight-loss herbal therapy has increased tremendously in the past few years, and it represents a multi-million-dollar industry. Although many Americans use herbal supplements many are unaware of the potential hidden risks [2]. Alternative herbal therapies are oftentimes poorly understood in the medical world as substantial research in the health effects of these products is scarce. Consequently, providers may miss the hazardous effects they can have on a patient’s health. Nephrotoxic effect of herbal medicines may be due to the presence of undisclosed compounds, heavy metals, interaction with the pharmacokinetic profile of concomitantly administered drugs, or misidentification of the herbal species. Renal involvement has been reported after the use of weight-loss herbal therapy, including but not restricted to acute tubular necrosis, acute or chronic interstitial nephritis, Fanconi's syndrome, hypertension, papillary necrosis, nephrolithiasis, and urinary tract neoplasms [8]. Thus, it is crucial that physicians and healthcare providers consider the potential risks of such often poorly reported herbal therapies and carefully investigate their use in patients under their care.

Herein we report a case of acute kidney injury requiring hemodialysis and steroids treatment due to Garcinia cambogia, an herbal supplement widely promoted for weight loss [2].
A rare etiology of aortic valve stenosis.

Unicuspic aortic valve (UAV) is a rare valve anomaly which usually presents with significant aortic stenosis and can be associated with other cardiac anomalies. This case describes a rare etiology of aortic valve stenosis.

A 29 year old man with history of aortic stenosis was sent to our institution for evaluation of chest pain. His exam was notable for a loud systolic murmur at the second right intercostal space with radiation to the carotids. Electrocardiogram (EKG) revealed sinus rhythm with non-specific T wave abnormalities, troponin-I was negative. His chest pain was thought to be non-cardiac and resolved spontaneously. Furthermore, Transthoracic echocardiogram (TTE) showed severe aortic stenosis with unclear valve morphology. Subsequently, Transesophageal echocardiogram showed eccentric aortic valve orifice with a tear drop appearance and a single commissure consistent with a UAV. CT aortogram and coronary arteries to evaluate for aortopathies and anomalous coronary arteries revealed normal coronaries and ascending aorta diameter of 3.8 centimeters. Due to his unclear functional capacity he underwent EKG stress test and was able to complete 9 minutes of Bruce protocol achieving heart rate of 176 beat per minute and 10.5 METs without symptoms. Given his lack of symptoms it was decided to follow him up closely and repeat TTE every 6 months to decide surgery timing.

Unicuspid aortic valve is a rare congenital valvular disease with reported incidence of about 0.02% among adults. A normal aortic valve consists of three cusps and three commissures. Failure of aortic cusps separation results in development of UAV. Two morphological forms of UAV have been described; acommissural and unicommissural UAV. The former results in a pinhole-like orifice and usually require intervention during infancy. On the other hand, a unicommissural UAV has a slit-shape appearance resulting in a larger orifice and usually presents during adulthood (30-50 years of age). When compared with bicuspid aortic valve, UAV results in valve dysfunction at an earlier age.

Aortic stenosis and aortic regurgitation are the most common presentations. In addition, UAV can be accompanied by aortic dilation, increased risk of aortic dissection, and other congenital heart diseases. Echocardiography, cardiac CT and cardiac MRI can be used to make the diagnosis pre-operatively, however, most UAV are diagnosed intraoperatively or during autopsy.
The American College of Cardiology recommends surgical valve replacement for severe aortic stenosis in the setting of symptoms (syncope, angina and dyspnea), left ventricular ejection fraction less than 50%, or in patients undergoing other cardiac surgeries. Moreover, an aortic root or ascending aorta diameter of more than 5.5mm is another indication for surgery. This case illustrates a rare congenital valve condition which should be a consideration in patients presenting with severe aortic stenosis at a young age.
Abstract:
Introduction:
Acute Eosinophilic Pneumonia (AEP) is a rare, life threatening condition commonly mistaken for infectious pneumonia. AEP presents with severe respiratory distress and diffuse infiltrates on chest X-Ray in previously healthy individuals. We present a case of AEP in the setting of hypogammaglobulinemia.

Case:
18-year-old female with PMH of uncomplicated premature birth presented with fever, rash, sore throat and shortness of breath for 1 week. Desquamating rash began on hands, feet and spread to legs and trunk. She had two younger sisters who presented with similar rash and sore throat 1-week prior as well. Patient and her siblings had been started on Keflex by her PCP due to an amoxicillin allergy, however, patient noticed swelling of lips, tongue, hands and feet after starting therapy.

At time of presentation, patient was febrile, tachycardic, tachypneic, hypoxic and became hypotensive within a few hours. CBC was significant for an elevated absolute eosinophil count without leukocytosis. Chest X-Ray showed diffuse interstitial infiltrates and small bilateral effusions. Patient tested positive for Group A strep. Of note, both siblings also tested positive for group A strep and were concurrently being treated in the PICU.

Patient was started on BiPAP and transferred to the MICU. Ceftriaxone and Azithromycin were started for strep pneumonia. Hypoxia continued to worsen and patient was intubated. Patient was started on prednisone and was eventually extubated. Patient was sent home without steroids and recommended to follow up with Pulmonology.

Two days after discharge, patient was visiting her sister in the PICU when she became dyspneic. Patient was intubated. Chest CT showed diffuse parenchymal alveolar areas of consolidation in subapices. Patient was prone positioned and treated with steroids. Bronchoalveolar lavage showed elevated eosinophils. Immunology was consulted for both the patient and her siblings in PICU. They were both found to have hypogammaglobulinemia.

Discussion
Etiology of Acute Eosinophilic Pneumonia is largely unknown. Many theories allude to environmental factors, cigarette smoking or unusual outdoor activity. Usually these patients will be HIV positive. There have not been many cases of AEP in immunosuppressed states such as hypogammaglobulinemia. AEP typically presents in previously healthy individuals with pulmonary symptoms less than a week and is accompanied by fevers, night sweats and pleuritic chest pain. X-ray or CT chest shows diffuse nonperipheral infiltrates. Although, blood
eosinophils are normal or mildly elevated, bronchoalveolar lavage shows eosinophils >25%. Most patients are admitted to the ICU and intubated due to the acuity and severity of symptoms. Patients that do not have an obvious cause of AEP, an immune work up may be warranted. Recovery is typically very fast with steroid treatment and relapse is rare. However, without treatment, AEP can be life threatening making early diagnosis imperative to successful treatment.
Title: Clinicians’ Learning Sources and Adherence to Guidelines Regarding Individualizing Glycemic Goals

Abstract:
Introduction: ADA guidelines (2017) recommend individualizing glycemic (A1C) goals for patients with diabetes based on patient characteristics and preferences. Effects of glycemic control vary with diabetes duration and history of complications. Intensive control soon after diagnosis is associated with reduced complications for two to three decades. In contrast, in patients with diabetes for ten years, predisposed to cardiovascular disease, intensive control may cause harm and mortality. We looked at how clinicians’ self-reported practices relate to current guidelines.

Methods: In 2015, a survey was mailed to endocrinologists and primary care physicians at an academic medical center (AMC; University of Chicago) and the AMC-affiliated community health system (North Shore University CHS).

Main outcomes were physicians’ 1) Source of learning about individualizing goals 2) Degree of difficulty individualizing goals 3) Reasons for difficulty 4) Factors considered when managing goals.
We also considered these variables: clinical site, gender, specialty, years in practice, practice size, and percent of patients 65 or older.

Statistics were calculated using means and proportions as appropriate. Bivariate relationships between physician/practice characteristics and outcomes were tested using chi-square tests.

Results: Response rate was 73% (156/213). Nearly 40% reported learning about individualizing goals from journal articles; 30% each learned from educational seminars and guidelines. About 20% each learned online or from colleagues. Individualizing goals was described as very challenging by 45% because of patient preferences (26%), vague recommendations (22%), or insufficient patient information (21%). There were significant differences depending on practice site. AMC physicians were more likely to learn from colleagues (41% vs.16%, p=0.001) and to report more challenge due to vague recommendations (41% vs.16%, p=0.001). They were also more likely to report learning from published guidelines: American Diabetes Association (45% vs.20%, p=0.001), American Geriatrics Society (14% vs.4%, p=0.016), and Choosing Wisely (10% vs. 2%, p=0.026). In addition, AMC physicians more often reported not knowing patients’ duration of diabetes (24% vs.5%, p=0.001) and hypoglycemia risk (33% vs.8%, p<0.001), or how
to estimate life expectancy (43% vs.7%, p<0.001). AMC physicians were more likely to consider life expectancy (79% vs.50%, p=0.002), while CHS physicians were more likely to consider diabetes duration (23% vs.5%, p=0.009) and history of diabetic complications (57% vs.38%, p=0.04). Only the difference in clinical site (but no other physician/patient characteristics) was consistently associated with the outcome measures.

Conclusion: In our survey, adherence to guidelines regarding individualizing A1C goals varied significantly by clinical site. This may be because of closer interactions between specialists and primary care physicians in AMC settings. Interestingly, the AMC physicians reported greater difficulty individualizing glycemic goals, possibly due to higher complexity of patients or to inherent differences in physician characteristics. Developing system-level interventions may help decrease physician-level variation in adherence to guidelines for individualizing glycemic goals.
A rare case of Pneumocystis Jiroveci pneumonia in a COPD patient, after a short steroid course

Abstract:
Pneumocystis pneumonia is a potentially life threatening infection well recognized in HIV population. With the development of the new immunosuppressive therapies, the epidemiological picture has changed, making its diagnostic challenging for physicians, especially in immunocompetent patients. We report a rare case of PCP pneumonia in COPD patient. A 79-year-old male, with a history of oxygen dependent COPD, presented to the ER for sudden onset dyspnea after visiting a horse farm. One month ago, the patient was hospitalized for COPD exacerbation and received a tapering course of 40 mg oral prednisone for 7 days. On admission, Patient was Afebrile, Tachycardiac and in respiratory distress, his O2 saturation 88% on 4 liters O2, requiring intubation shortly after presentation due to increased work of breathing. His ABG revealed hypoxemic respiratory insufficiency with PO2 57 mmHg, PCO2 36 mmHg. His CBC, lactic acid, pro-BNP, D-dimer and chemistry were normal. LDH was elevated to 385. Chest XR revealed bilateral reticular interstitial infiltrates. The patient was empirically started on Zosyn & Azithromycin. Rifampicin was added considering Rhodococcus infection, due to exposure history to horses. Fiberoptic bronchoscopy and bronchoalveolar lavage were performed. Bacterial, fungal, viral, AFB stains and cultures, were inconclusive. PCP DNA was detected in bronchial washing and the patient was started on trimethoprim/sulfamethoxazole and prednisone for PCP pneumonia. Patient's clinical condition significantly improved, he was extubated after 11 days of treatment with complete clinical recovery.
Pneumocystis Jiroveci is an opportunistic fungal infection transmitted human to human through inhalation, a known cause of pneumonia in immunocompromised individuals. Its pathogenesis, based on the unbalanced relation between T-CD4 & T-CD8 cells, which leads to a hyper inflammatory state what compromise the air interchange and damage the lung.
In a study from Helwed-Larsen of non-HIV patients, PCP-DNA was found in 16/367 (4.4%) cases, 12 of which were on corticosteroids. A prospective study in France, found an increased rate in non-HIV patients 461/604, of which 357/461 (77.4%) were receiving corticosteroids, 305/461 (66.1%) were hematological malignancies and 34/461 (7.4%) were solid transplant organ.
But what about COPD patients? There is still much to be defined: The course of steroid that put patients at risk, and the role of the prophylaxis. Among solid transplant organ, prophylaxis decreases the incidence of PJP from 10% to 1%.
A prospective study in Taiwan found that the delay in the diagnostic and therapy in days was 10 in HIV v/s 1 non-HIV group. Although multiple predictors of mortality have been proposed. The most robust indicator of mortality in those vulnerable patients is the early suspicion and treatment, PJP infection should be considered in COPD patients. Steroids use may be defined as possible risk factor for the development of PJP pneumonia.
Title: Celecoxib Augmentation of Escitalopram in Treatment-Resistant Bipolar Depression: Effects on Kynurenine Pathway Metabolites

Abstract:
In bipolar depressed patients, failure to respond to antidepressant therapy occurs very frequently. The chronic pro-inflammatory state that has been associated with stress and depression is believed to lead to a pathological shunt of tryptophan metabolism toward the kynurenine pathway and thereby interfere with drug efficacy. This study was undertaken to determine if modulation of the inflammatory response by inhibiting cyclooxygenase-2 with the anti-inflammatory agent celecoxib, co-administered with the SSRI antidepressant escitalopram, would convert treatment-resistant bipolar depression to an improved overall outcome. Additionally, we hypothesized that the neurotoxic metabolite Quinolinic Acid (QA) blood level would decrease over time. This randomized, double-blind, two-arm, placebo-controlled study consisted of a screening visit, a 2-week washout, and a 1-week placebo run-in phase. Subjects underwent a physical exam, medical history, laboratory tests, and completed several rating instruments. At their baseline visit, they were rated in a blinded manner. If they continued to score ≥18 on the 17-item Hamilton Depression scale (HAM-D), they were randomized to receive escitalopram + celecoxib, or escitalopram + placebo. The overall study was powered for 70 patients to complete 8 weeks of active medication. Blood levels of inflammation biomarkers and metabolites were determined at baseline and weeks 4 and 8. A Wilcoxon Rank Sum test assessed differences in QA levels between treatment groups at baseline. Univariable exact logistic regression analyses compared rates of treatment response or remission by drug therapy. A linear mixed effects model estimated QA values over time by drug therapy after adjusting for sex, age, and body mass index. The combination of escitalopram with celecoxib over 8 weeks led to decreased levels of patient-reported depressive symptoms vs. escitalopram with placebo. Patients receiving celecoxib were 4.13 (95 CI: 1.03-18.48) times more likely to respond to treatment compared to those receiving placebo (p =0.04), and 14.34 (95 CI: 2.59-153.17) times more likely to experience remission compared to those receiving placebo (p <0.001). Overall, patients receiving celecoxib (mean = 55.69, standard error [SE] = 6.27) had comparable QA values to patients receiving placebo (mean = 64.90, SE = 7.20, p = 0.34). QA values did not change significantly over time independent of which therapy the patients received (p = 0.28). In a prior study of major depressive disorder, we had demonstrated reduction of neurotoxic metabolites, indicating that escitalopram may exert its antidepressant effect in part through inhibition of synthesis of certain neurotoxic metabolites and through reduction of the inflammatory response. Although normalization of metabolites did not occur in this study, there were greater symptom response and remission in the
escitalopram+celecoxib group at 8 weeks, showing the effects of modulating the inflammatory response on the efficacy of an anti-depressant.
Abstract:
Lyme Carditis is seen in 4-10% of all patients with Lyme disease. Lyme Carditis occurs in the early disseminated phase within weeks to a few months after exposure. Electrocardiogram (EKG) in affected patients may have ST depression and T wave inversions particularly in the inferior leads. Patients typically also have varying degrees of atioventricular (AV) conduction block ranging from first degree heart block to complete heart block.

A 61 year old female with history of hypertension and isolated amyloidosis in her left eye presented with syncope. While sitting on her couch, she suddenly felt lightheaded. She was found unconscious and woke up a few minutes later. Then almost an hour later while tying her shoelaces, she became lightheaded but did not lose consciousness. There was no associated chest pain, dyspnea or prodrome prior to each of the episodes nor did she have a prior history of syncope. EMS was called and they noted irregular heart-rate on the monitor which was found on EKG to be complete 3rd AV block with a junctional escape rhythm.

Four weeks prior to this event, the patient was treated with Bactrim for a UTI. Three days later, she developed a pruritic, erythematous rash on her calves, abdomen and back that expanded circumferentially with blanchable purpura in the center. She was given steroids for a presumed drug induced rash by the ER. She stated that she hiked daily in the woods near her home in Northern Illinois. A screening test for Lyme disease was negative at that time.

The patient underwent a cardiac MRI with gadolinium which did not reveal any evidence of cardiac infiltration. A Nuclear Myocardial (NM) perfusion scan showed basal to mid inferolateral defect which was new compared to a prior NM scan. Transthoracic Echocardiogram revealed an ejection fraction of 60% with no structural heart disease. A repeat ELISA screening test for Lyme disease was positive. She was subsequently started on intravenous Ceftriaxone. Her heart block slowly improved to a first degree block over a week. The Western blot for Lyme IgG/IgM was positive (11 bands) confirming the diagnosis of Lyme Carditis. She was discharged home symptom free to complete a 4 week course of Ceftriaxone.

Third degree heart block usually requires permanent pacemaker implantation. However, in the case of Lyme disease, it is usually reversible with appropriate antibiotics and can save the patient from a life altering procedure. When the diagnosis is not readily apparent, one must diligently pursue further studies based on good clinical suspicion. Diagnosing Lyme Carditis
requires a strong clinical assessment and appropriate ordering of diagnostic studies, so that when life gives you Lyme’s, you can treat appropriately and continue to make lemonade.
Title: Acute Phlegmonous Gastritis with distal small bowel obstruction: A rare case of successful medical management

Abstract:
Introduction: We present a case of emphysematous gastritis with concurrent small bowel obstruction which was treated with broad spectrum antibiotics and supportive therapy.
Case Description: An 87 year old female with history of atrial fibrillation and colon cancer successfully treated with colon resection presented with a two day history of severe abdominal pain and vomiting. The patient was initially vomiting clear yellow liquid however it later developed into hematemesis. Patient had also developed severe sepsis on presentation with lactic acid of 7, leukocytosis of 25 and was severely hypotensive requiring urgent fluid resuscitation in the intensive care unit. The patient had a CT abdomen done which revealed a distal small bowel obstruction and a small volume of portal venous gas within the left hepatic lobe with small volume of air in gastric wall and adjacent draining veins suggesting emphysematous gastritis. Surgery was emergently consulted who recommended non-surgical management due to high intra-operative risk. An EGD was performed which revealed erythema, edema and purplish discolouration in the antrum, prepyloric area and body of the stomach. Biopsies were taken which revealed acute necrotizing erosive gastritis with full thickness ulceration and marked regenerative atypia. The patient was kept on empiric antibiotic coverage of Vancomycin, Aztreonam and metronidazole and supportive intravenous fluids. The patient also had a nasogastric tube placed to suction for small bowel obstruction treatment. The patient made good clinical recovery on this regime and her severe sepsis resolved within the next two days. Aztreonam was replaced with Levofloxacin after consultation with the infectious disease team for broader gram negative coverage. The patient underwent a repeat CT abdomen 3 days later which revealed no air in the portal venous system or in the gastric wall and resolution of small bowel obstruction. The patient was resumed on oral nutrition and was transferred to medical floor. Antibiotics were continued for a total of 15 days with the triple antibiotic regimen of Vancomycin, Levofloxacin and metronidazole escalated to Vancomycin and meropenem on day 8 of antibiotics due to persistent asymptomatic leukocytosis. The patient had successful resolution of leukocytosis and all clinical symptoms and was discharged to subacute rehab.
Discussion: Acute phlegmonous gastritis is associated with a high rate of mortality however treatment has been successful when detected early and treated adequately with broad spectrum antibiotics and supportive therapy. Our case reflects a more complicated management owing to the presence of a small bowel obstruction. This appears to be the first documented case of these two conditions occurring concurrently.
Cardiopathia Fantastica: The Munchausen Syndrome of Cardiology

Abstract:
Introduction
Cardiopathia fantastica is a factitious disease wherein cardiac symptoms are intentionally produced or feigned in the absence of overt external incentives. Affected patients can be obsessed with the prompt attention and intensive interventions associated with cardiology, even at the expense of possible harm and death.1 Appropriate recognition of the diagnosis is important because treatment to reduce procedural risks, incidental diagnoses, excessive health care costs and potential legal implications are available.

Case Presentation
A 51-year-old female presented to the ER with acute left-sided sharp chest pain only partially relieved by nitroglycerin. It was associated with dyspnea and vomiting. Her self-reported medical history consisted of cerebral aneurysm, coronary artery disease, diabetes, hypertension, pulmonary embolism, deep venous thrombosis, and asthma. Physical exam and routine laboratory tests were normal. Chest x-ray showed mild pulmonary vascular congestion in the setting of low lung volumes. EKG was only remarkable for poor R-wave progression. Serial troponins were within normal range. Echocardiogram showed left ventricular hypertrophy and no ischemic changes. Nuclear stress test was performed and showed a moderate severity reversible defect in the RCA territory. Upon review of records, it was found that the patient had hospital visits in at least 15 different hospitals, more than 20 CT scans of the chest, 23 nuclear stress tests, 6 stress echocardiograms and 5 coronary angiograms in the past 5 years. It was also found that the patient carries the diagnosis of bipolar disorder, multi-substance abuse, poor follow-up and non-compliance, and possible malingering behavior. Because the patient’s symptoms and stress test findings are chronic and given concern for a factitious disorder, further ischemic workup was not pursued. Patient was discharged and instructed to follow-up with her primary care physician.

Discussion
Patients with cardiopathia fantastica may present with chest pain, syncope, arrhythmia, hypertensive crises, and abnormal cardiac enzymes and electrocardiographic findings. It is important to rule out organic cause as the differentials may include life-threatening or easily reversible conditions. Medical records should be obtained and reviewed. Suspicion for cardiopathia fantastica is raised by clues like dramatic presentations, fragmented care, extensive knowledge on medical terminologies, psychiatric co-morbidities, apparent
senselessness of actions, and even specific timing of presentation (at night, in July or during shift changes) are present. When suspected, it is best to avoid confrontational treatment and blacklisting. Patients should be referred to psychiatry early, preferably while the patient is still in the hospital. Anxiolytics and behavioral therapies were shown to have some benefits. Routine and brief supportive visits to prevent unexpected ER visits should be scheduled. In these patients, the goal should be to break the cycle of recurrent admissions and evaluations for organic cause of symptoms.
Abstract:
Introduction
Sarcoidosis is a systemic granulomatous disease with variable presentation capable of affecting a wide variety of organ systems. It frequently presents with fever, fatigue, dyspnea on exertion, retrosternal chest pain, cough, and weight loss. In acute onset of sarcoidosis patients can present with Löfgren’s syndrome, a triad of symptoms: erythema nodosum, polyarthralgias or polyarthritis, and bilateral hilar lymphadenopathy. This is similar to the symptomatology of tuberculosis which can also present with fever, weight loss and cough. Here we describe the case of a 35 year old Hispanic male with Löfgren’s syndrome.

Case report
A previously healthy 35 year old Hispanic male presented to the emergency department with one month of fatigue that had worsened over the previous four days. He also reported weight loss, dry cough, fevers, night sweats, muscle pain, and lower extremity rash on both legs with swelling. History of immigration from Mexico seven years ago and currently working in construction. Physical exam demonstrated anterior shins and left forearm with indurated, tender subcutaneous nodules with overlying bright red erythema. A portable chest x-ray showed bilateral hilar convexities. CT of the chest, abdomen and pelvis demonstrated mediastinal and hilar lymphadenopathy correlating with a granulomatous entity. Laboratory results reported: elevated ESR and CRP, negative ANA, histoplasmosis urine antigen, blastomycosis antigen and AFB samples. TB quantiferon was indeterminate. Following an endobronchial ultrasound with biopsy and culture RIPE therapy was initiated. Biopsy results showed non-caseating granulomatous lymphadenitis that was consistent with sarcoidosis. Prednisone therapy was added to the RIPE therapy. Patient was discharged on prednisone and RIPE therapy. On follow up patient felt better with improved cough and resolved erythema nodosum. AFB cultures and repeat quantiferon were negative and RIPE therapy was discontinued.

Discussion
Distinguishing sarcoidosis from tuberculosis is difficult due to the similarities in presentation; both are capable of producing weight loss, dry cough, and fevers. Even the symptoms of Löfgren’s syndrome are less commonly produced by tuberculosis. This case was complicated by its epidemiology. The incidence of sarcoidosis and tuberculosis are 4.1 and 30.8 per 100,000 respectively. The greater incidence resulted in elevated clinical suspicion of tuberculosis. Even
after sarcoidosis was confirmed, indeterminate quantiferon required addition time before the AFB cultures could be ruled definitely negative. Furthermore, treatment for sarcoidosis is steroids, increasing the risk of reactivation tuberculosis and was the reasoning for discharging the patient on RIPE therapy and steroids. The difficulty differentiating these two diseases and the possibility of reactivation when treating sarcoidosis complicate clinical decision making. In conclusion, it would be prudent to treat tuberculosis in any high risk population even if sarcoidosis is confirmed until the culture results are definitely negative.
Abstract:
Toxic and metabolic disorders produce widespread, symmetric patterns of injury in brain that often involves the deep gray nuclei and cerebral cortex. Topographic distribution of lesions on MRI may help to deduce the list of differential diagnosis. Here we present a case of a middle aged man who presented with acute onset altered mental status with peculiar MRI finding. A 48-year-old male with past medical history of uncontrolled diabetes mellitus, presented to the ED with altered mental status, which was sudden in onset, worsening over a period of 24 hrs and was associated with loss of memory. He denied fever, altered body movements, blurring of vision or weakness. He denied alcohol, smoking or drug intake. On physical examination he appeared distressed and confused, was not oriented to time, place and person. He had an altered mini mental status examination with inability to recall objects and decreased attention but without neurological deficits. Laboratory work up was normal except for glucose of 400 mg/dl and sodium of 130 meq/ml. Brain computed tomography scan showed an area of diffuse bilateral thalamic enlargement with possible edema. Further brain magnetic resonance imaging (MRI) confirmed T2/FLAIR hyperintense signal within bilateral thalamus, dorsal midbrain sparing the red nucleus, and cerebellum peduncle extending into dorsal pons and medulla. He was admitted to ICU for observation. He was treated with insulin, thiamine, aspirin and atorvastatin. His mental status improved significantly over the course of his hospital stay and was discharged with almost normal functioning and no neurological deficit 1 week after discharge.

Thalamus plays an important role in memory, emotions, sleep-wake cycle, executive functions, and processing of sensory information. Bilateral thalamic lesions are un-common and have a limited differential diagnosis that includes metabolic, toxic, infectious, vascular and neoplastic causes. In our patient, the DWI imaging on MRI, was negative for acute ischemia. Magnetic resonance venography (MRV) was negative for any venous thrombosis. Viral encephalitis panel including West Nile serology was negative. Heavy metal screening, copper studies, Sedimentation rate, C-reactive protein, ANA, ANCA, Carbon monoxide levels, Lyme Antibody, Cryptococcus Antigen, Thiamine levels, syphilis, TSH, ammonia levels were normal. The pattern of involvement of brain including exact symmetrical involvement of thalamus, dorsal midbrain, pons and medulla may exclude neoplasm as one of the cause. Sudden osmotic changes associated with glucose, as in our case, may cause similar lesions especially at the common sites of extrapontine myelinolysis including thalamus. Usually in these cases clinical improvement precedes changes in MRI. Extrapontine myelinolysis is a rare manifestation of non ketotic hyperglycemia and thus describes the peculiarity of the case.
INTRODUCTION:
Kaposi’s Sarcoma (KS) is the second most common HIV-related malignancy causing increased mortality in this population. Patients with HIV often present with respiratory illnesses most commonly from an infectious etiology, but KS should also be considered on the differential. Here we present an HIV patient, non-adherent to antiretroviral therapy (ART), admitted with respiratory failure secondary to pulmonary KS causing diffuse alveolar hemorrhage and subsequent death.

CASE DESCRIPTION:
A 44 year old male with advanced HIV/AIDS (non-adherent to his antiretroviral therapy (ART) presented with productive cough, fevers, respiratory distress, hypotension and tachycardia, requiring vasopressor support and intubation for mechanical ventilation.

Patient previously had presented with diffuse lymphadenopathy and nodular pulmonary and liver lesions. Left inguinal lymph node biopsy was obtained. Histo-cytologic markers were diagnostic of KS (+CD34, +vimentin, +HHV8). He completed 11 chemotherapy cycles with doxorubicin but was lost to follow-up thereafter.

During current admission in the Intensive Care Unit, patient was found to have low CD4 count (12) and high viral load (38,000 copies/ml). Empiric therapy was initiated (piperacillin/tazobactam, azithromycin, oseltamivir, sulfamethoxazole/trimethoprim and steroids). Physical exam showed new lower extremity cutaneous lesions, consistent with KS. CT chest demonstrated progression of the previously known metastatic bilateral KS lesions. The following cultures, stains and antigens were sent with negative results- bronchial, blood, legionella, endotracheal aspirate, aspergillus, histoplasmosis, cryptococcus and pneumocystis jiroveci pneumonia. Acid fast bacilli sputum cultures were negative. Bronchoscopy showed minimal hyperemic airways without secretions. Bronchoalveolar lavage demonstrated macrophages present with hemolyzed blood; CMV was detected, so patient received ganciclovir. However, the patient’s condition continued to deteriorate with increased ventilatory requirements. He eventually had multiple massive episodes of hemoptysis from diffuse pulmonary hemorrhage, and ultimately died from cardiac arrest.

DISCUSSION:
Kaposi’s Sarcoma is an angiosarcomatous neoplasm which is associated with human herpesvirus 8 and has been associated with pulmonary manifestations, including hemorrhagic pleural effusion, peribronchovascular opacities and airway lesions. Rare presentations include Immune Reconstitution Inflammatory Syndrome (IRIS), chylous effusions and diffuse alveolar hemorrhage (DAH), as seen in our patient. KS therapy includes bleomycin and vincristine, but pegylated liposomal doxorubicin has demonstrated greater efficacy. Our patient underwent 11 cycles of doxorubicin, however, his disease progressed, and he succumbed to pulmonary hemorrhage. This case report demonstrates the importance of recognizing that pulmonary infiltrates may be a manifestation of KS in an HIV positive patient, especially since it can mimic infectious etiologies. Currently, no effective therapy exists for DAH caused by pulmonary KS other than supportive care, especially given the difficulty in cauterization or embolization of the highly vascular pulmonary lesions. Patients with KS should be counseled extensively on ART adherence, since adherence provides them with the greatest chance to prevent pulmonary KS and its devastating sequelae.
Abstract:
Introduction:
In the post-antibiotic era, purulent pericarditis is a rare complication of S. pneumoniae infection. We present a case of a man who developed cardiac tamponade and sepsis secondary to purulent pericarditis.

Case:
A 68-year-old male with past medical history of ESRD on HD, Afib, HTN, and HFrEF who presented with 1 week of fatigue, malaise, and dyspnea. He denied having fevers, cough, or chest pain. In the ED, he was afebrile, tachycardic, and normotensive. His physical exam showed conversational dyspnea, bibasilar crackles, and a pericardial friction rub. Labs revealed a leukocytosis to 30.4, BUN of 159, BNP of 781, and a troponin of 0.02. CXR showed cardiomegaly with a left lower lobe consolidation. EKG showed inferior-lateral ST elevations. He went for emergent left heart catheterization for suspected STEMI, which showed non-obstructive CAD. Repeat EKG post-catheterization showed now diffuse ST elevations and PR depressions consistent with pericarditis. He was admitted to the ICU for urgent dialysis for suspected uremia. Two hours later, he became hypotensive. Broad-spectrum antibiotics and fluid resuscitation were started for suspected sepsis. Bedside TTE showed early evidence of cardiac tamponade with negative pulsus paradoxus. Soon after, he went into PEA arrest. During resuscitation, he underwent emergent pericardiocentesis, with drainage of 650 cc of purulent brown fluid with improvement in his blood pressure. Cultures from the blood and pericardial fluid grew S. pneumoniae. Patient passed away within two days and his autopsy confirmed suppurative fibrinous pericarditis.

Discussion:
Purulent pericarditis is an uncommon entity characterized by infected fluid in the pericardial space. Historically, it was commonly caused by dissemination of streptococcal pneumonia to the pericardial space. With development of antibiotics and streptococcal vaccines, it has become rare. Most present-day cases present in patients with immunosuppression or recent thoracic surgery; however, high clinical suspicion remains important to prevent delay in diagnosis. Case series show fever being present in most patients, but was absent in our case. Chest pain is present in less than half of cases. Profound leukocytosis and abnormal CXR findings such as enlarged cardiac silhouette, consolidations, or pleural effusions can help guide towards an infectious etiology. EKG is normal in 35% of cases, but may reveal a pericarditis pattern. Cardiac tamponade develops in 79% of patients. Early antibiotic therapy and pericardiocentesis for diagnosis, source control, and prevention of tamponade remains the
standard of treatment for this disorder. In this case, the presence of ST elevations in the setting of normal cardiac biomarkers, non-obstructive CAD, and leukocytosis were consistent with the diagnosis.

Conclusion:
This case illustrates a presentation of purulent pericarditis likely resulted from dissemination of streptococcal pneumonia. The patient presented with characteristic laboratory and imaging findings, but with atypical clinical presentation. His autopsy results confirmed the diagnosis.
Abstract:
Pseudoseptic arthritis, although not limited to rheumatoid arthritis (RA), is a clinical syndrome that simulates septic arthritis-presenting with acute monoarticular pain, often with fever/chills, and synovial fluid analysis (SFA) revealing leukocytosis in the septic range, with negative culture. In a patient with pre-existing RA, it is much more common for a flare to present with polyarthritis, but monoarticular inflammation can sometimes occur in early stages of disease. Additionally, patients with RA are predisposed to contracting septic arthritis, due to a combination of chronic synovial membrane damage, systemic and intraarticular corticosteroid use, and use of other immunosuppressive agents.

The patient is a 46-year-old female with a 20-year history of RA, currently on Prednisone, Azathioprine and Abatacept injections, who presented with 1-day history of severe, constant right knee pain and swelling. She was noted to be poorly adherent with her medications, as she had experienced several RA flares in the past year mostly affecting her hand and ankle joints bilaterally. She denied other symptoms including fever and chills, history of trauma, recent illness, or recent travel.

Pertinent on examination was swollen, erythematous, warm right knee with significantly decreased range of motion. Laboratory studies showed WBC 18,000, ESR was 49 mm/hr and CRP was 2.17 mg/dl. SFA demonstrated yellow, cloudy fluid with 33,500 WBCs/uL with neutrophilic predominance, absence of crystals, and a negative gram stain. Empiric Ceftriaxone and Vancomycin were started. On the following day, she showed marked clinical improvement as she was able to ambulate with minimal pain. Synovial fluid, blood and urine culture were negative at 48 hours. She was given intraarticular corticosteroids, for which she responded well. On the third day, right knee pain had completely resolved. Synovial fluid culture remained sterile. She was eventually discharged improved with continuation of RA therapy.

Major causes of acute monoarthritis in RA patients include trauma, infection, crystal-induced arthritis, and RA flare, presenting as pseudoseptic arthritis. A case series had explored this clinical presentation in 10 patients with preexisting chronic rheumatologic conditions, including 5 with RA, presenting with pseudosepsis. All patients were treated empirically with antibiotics, and were discontinued after negative synovial fluid cultures. The average length of hospitalization was 6 days, and all 10 patients experienced resolution of symptoms within two days of intraarticular corticosteroid injection.
In summary, we present a patient with RA, sudden onset monoarticular pain, PMN-predominant synovial leukocytosis, and negative synovial fluid culture - a presentation that has previously been described as pseudoseptic arthritis. While it is crucial to treat a patient with suspected septic arthritis empirically, recognition of this entity as part of spectrum of RA flare is essential in preventing prolonged hospital stays and unnecessary prolonged antibiotic therapy.
Glomerulonephritis can often be a preceeding diagnosis of malignancy. Classically, the paraneoplastic presentation of glomerulonephritis has been associated with membranous nephropathy. Goodpasture syndrome is a rare autoimmune disease that is mediated by anti-glomerular basement membrane antibodies. It is estimated to affect 1 case per million per year and is a known cause of acute renal failure in approximately 20% of cases of rapidly progressive or crescentic glomerulonephritis. Of note, an appreciable percentage of patients with anti-glomerular basement membrane disease also have antineutrophil cytoplasmic antibodies (MPO antibodies) which is also known as double-positive Goodpasture syndrome. Double-positive Goodpasture syndrome is an important clinical entity as early diagnosis and early aggressive therapy can prevent progression to end-stage renal disease and improve survival.

Unfortunately, presentation with severe disease often portends a poor prognosis. There is minimal literature currently available paraneoplastic double-positive Goodpasture syndrome in the setting of rectal adenocarcinoma.

An 80-year-old female with history of Stage III chronic kidney disease was admitted for evaluation of abnormal creatinine detected on routine outpatient labs. A few days prior to admission, she had three episodes of nonbloody diarrhea. She also reported progressively worsening fatigue, poor appetite and weight loss over the preceding month. Patient denied hematuria, epistaxis, rash or joint pain and no recent use of NSAIDs. On physical examination, she was confused, had mild bibasilar crackles and +1 bipedal edema. Her admission laboratory results were remarkable for a creatinine of 11 mg/dL. Further workup revealed nephrotic range proteinuria and gross hematuria. During her hospital course, she developed anuria, and hemodialysis was initiated. Due to this progression, additional tests were ordered and revealed surprisingly high levels of anti-GBM and MPO-ANCA antibodies. A kidney biopsy showed diffuse crescentic glomerulonephritis with immunofluorescence findings compatible with anti-glomerular basement membrane glomerulonephritis. She was started on daily plasma exchange, intravenous solumedrol, and cyclophosphamide. Her hospital stay was further complicated by discovery of a rectal mass which biopsy proved to be infiltrating moderately differentiated adenocarcinoma. She further elected hospice care and declined any further interventions. She unfortunately did not recover renal function prior to discharge.

Diagnostic of Goodpasture syndrome is based on clinical, histological and immunological findings. Detection of both anti-MPO antibodies and anti-GBM antibodies in this patient gives clues that both antibodies might participate in the pathogenesis of crescentic
glomerulonephritis. Double-positive Goodpasture syndrome has been linked to poor outcomes. The association between Goodpasture syndrome with double-positive antibodies and rectal cancer has not been reported in the past. Although this association might be purely coincidental, it is possible that release of tumor antigen triggers autoimmune antibodies as in paraneoplastic membranous nephropathy. Further studies are needed to investigate this association.
Abstract:
Sir William Osler said “He who knows syphilis knows medicine” referring to the disease’s myriad of possible clinical presentations which earned Treponema pallidum the name of the “Great Imaginator”. The Centers for Disease Control and Prevention’s recent data are very concerning reporting the highest rates of syphilis in more than 20 years.

CASE REPORT
A 43-year-old African American transgender female with HIV presented with a four week history of painless, watery diarrhea and intermittent, diffuse abdominal pain. Review of systems revealed generalized weakness, lack of appetite due to nausea but no vomiting, and weight loss of 15 pounds over 1 month. Patient reported no rash, no joint pain, no fever, no headache, and no cough. Patient was on antiretroviral therapy since the diagnosis of HIV in 1990 with current CD4 count of 280/uL and undetectable viral load. She had a history of primary syphilis which was treated with IM penicillin 10 years ago. She has receptive anal intercourse with multiple MSM partners. On physical examination patient was with mild volume depletion which resolved with intravenous fluids; abdominal and rectal exams were benign. Complete blood count and comprehensive metabolic panel were normal. Testing of stool samples did not identify any parasites including Microsporidia and Cryptosporidium spp.. PCR tests for Clostridium difficile and Norovirus were negative. Stool cultures did not isolate any organisms. Patient underwent colonoscopy which showed focal areas of erosive colitis changes in the splenic and hepatic flexures as well as the mid transverse colon. Biopsies were obtained from the colonic lesions and histologic analysis of the colonic mucosa revealed: active colitis with lymphocytic infiltrates, full thickness ulceration, pseudomembrane formation, and acute cryptitis with crypt abscesses without dysplasia. Further immunohistochemical staining was positive for T. pallidum antibodies, CD3+ T-cells and CD20+ B-cells. Rapid plasma reagin (RPR) was negative. Patient was treated with penicillin with clinical improvement.

DISCUSSION
Colonic syphilis is very rare and is difficult to diagnose due to its non-specific gastrointestinal symptoms such as (sub)acute or chronic diarrhea, abdominal pain, weight loss. It could be a visceral manifestation of secondary or tertiary syphilis. Most cases have been reported in patients who practice receptive anal intercourse as well as in patients with HIV infection. Colonoscopy may visualize non-specific inflammatory colitis with ulcers, erosions, or even masses. Typically, histology reveals severe inflammation predominantly with plasma cells. To make a definite diagnosis, it is necessary to identify T. pallidum histologically.
Immunohistochemistry staining with anti-T. pallidum antibodies can be used to detect it in
tissue, as in the present case. In our patient, RPR was negative, prompting us to consider the possibility of either tertiary syphilis (though no gummae were identified) or atypical presentation of secondary syphilis.
A 32-year-old male presented with progressive shortness of breath and leg swelling. He has a past medical history significant for HIV disease, Renal Cell Carcinoma (RCC) s/p left nephrectomy and was recently found to have metastasis to his mediastinal lymph nodes and lung, which were detected 2 months prior to admission. The patient has been on salvage treatment with atezolizumab since the time of the metastatic diagnosis.

On admission, the patient’s physical exam was significant for tachycardia, respiratory distress, decreased breath sounds in the right hemithorax and lower extremity swelling. A Chest X-ray showed a right sided pleural effusion and an enlarged nodular density in the left upper lobe. Laboratory data revealed anemia and an elevated Troponin I. His viral load was undetectable and his CD4 count was 444 cells/mm3. Pleural fluid analysis showed atypical cells weakly positive for RCC marker. Transthoracic echocardiography revealed normal left ventricular function and a large mobile cystic mass in the left atrium. Transesophageal echocardiography revealed a large mass composed of solid and cystic components and a left atrial appendage thrombus. The solid component, a 5 x 2.3 cm mass, invaded the basal half of the interatrial septum and the cystic component, a 4 x 1.6 cm mass was found to be protruding to the left atrium. Multiple enhancing neoplastic masses in the muscular compartment in both calves were detected on MRI of the lower extremities.

Renal cell carcinoma (RCC) is the seventh most common cancer in the United States and causes approximately 13,000 deaths annually. RCC occurs in younger patients who are HIV positive and has unfavorable prognosis in HIV patients with advanced stage. Distant metastasis usually develops in the lung, bone and lymph nodes. Although tumor thrombus involving the renal vein and inferior vena cava (IVC) happen in up to 10 % of patients with RCC, there are rare reported cases of RCC metastasized to the heart without IVC involvement. The majority of these cases involved the lymph nodes before reaching the heart. Metastasis to the muscles is a rare phenomenon but has been reported in RCC. Metastatic RCC responds poorly to chemotherapy and most patients do not survive beyond 6 months of diagnosis. Here, we reported a solid cystic cardiac mass in a patient with an undetectable level of HIV viremia and RCC. This patient had been treated with atezolizumab prior to the presentation with disseminated metastasis. The metastatic mass was mobile in the left atrium with cystic and solid components.
Unfortunately, our patient’s course ended with death, secondary to hemoptysis leading to respiratory failure a few days after admission to our hospital.
Blastomycosis is a systemic pyogranulomatous disease with varied clinical presentation and can mimic other diseases, 50% cases are asymptomatic. In endemic regions, yearly incidence rates are approximately 1-2 cases/100,000 population annually with Wisconsin having the highest incidence averaging 10-40 cases/100,000 population. T-cells play a major role in host defense against Blastomycosis with immunocompromised individuals at higher risk for severe forms of disease. Here we present a case of acute pulmonary blastomycosis.

A 54-year-old male with past medical history of poorly controlled insulin dependent diabetes with retinopathy and neuropathy status post below-the-knee amputation, hypertension, hyperlipidemia, and chronic respiratory failure on home oxygen hospitalized with complaints of 1-week of sudden onset of nonproductive cough, daily low grade intermittent fever, and loose nonbloody stools (up to 6 episodes daily). Patient denied recent travel, sick contacts or weight loss. In the ED patient was afebrile, hemodynamically stable with oxygen saturation at 93% on room air laying comfortably in bed, not in acute distress. Physical exam showed diminished breath sounds in left lower lobe. Labs were remarkable for leukocytosis, normocytic normochromic anemia, thrombocytosis, and poorly controlled blood glucose. Imaging studies showed near complete consolidation of left lower lobe and loculated left pleural effusion. Patient was started on IV fluids and antibiotic therapy, later underwent thoracentesis for left pleural effusion with suspicion for empyema. Due to worsening left and new right parapneumonic effusions, bronchoscopy was performed. Patient had to be intubated during procedure but was unable to be successfully extubated and continued to require high FiO2 and PEEP. Respiratory cultures obtained resulted positive for Blastomycosis and patient started on IV amphotericin. Despite ongoing mechanical ventilation, patient had a protracted and complicated course as he continued to desaturate, became profoundly acidic (pH as low as 6.97), hypotensive, developed renal failure, multiple cardiac arrests and anoxic encephalopathy despite management. He passed away after decision was made to withdraw life sustaining measures, 15 days from admission.

This case demonstrates the rapid progression to respiratory failure and high mortality associated with severe blastomycosis. Endemic regions should have lower threshold for suspicion of Blastomycosis infections. Antigen detection from urine or serum can take up to one week once specimen is received to accepting laboratory, which does not take into account time necessary for specimen collection, processing and delivery to laboratory. Despite
immunocompetent status within endemic regions, if no etiology for pneumonia can be ascertained within 72-96hrs, we may consider obtaining bronchial aspirate sooner or consider empirical antifungal therapy when appropriate as early diagnosis and treatment portends a survival benefit.
Abstract:
Background
Spurred by federal incentives through the HITECH Act, a majority of all US physicians now own certified electronic health record (EHR) technology. Despite the growing adoption of EHRs, physicians increasingly report dissatisfaction with their EHRs. Common reasons for dissatisfaction include increased clerical burden, decreased time with patients, and alert fatigue. Due to the availability of a variety of EHR products, it is possible that providers dissatisfied with their EHR could switch to a competing product in search of a better experience. We sought to determine the usage trends and transitions between specific EMR products utilized by outpatient primary care providers.

Methods
Meaningful Use attestations from 2012-2017 were obtained from the Medicare EHR Incentive Program Data. Outpatient attestations made by Internal Medicine, Family Medicine, General Practice physicians were included in analysis. Non-EHR product attestations (ie. modular patient portal) were excluded and EHR products were grouped by the 24 most common EHR vendors (as measured by attestations), with the remaining EHR products grouped as “Other.” The transition between EHR vendors was recorded when a physician attested for different products during the period of analysis.

Results
141,345 physicians were included in analysis. Using the most recent attestations available, Epic, Allscripts, and eClinicalWorks were the most common EHRs utilized by physicians (31%, 12%, and 10%, respectively). More physicians switched to Epic and AthenaHealth than any other product (net gain of 8,376 and 3,347 providers, respectively). Conversely, Allscripts and NextGen saw the greatest number of providers switch away from their product (net loss of 3,665 and 2,040 providers, respectively). Allscripts and NextGen users who switched products were more likely to switch to Epic than any other vendor. The share of physicians who attested for the 10 most common EHRs increased from 89% to 91% over the study period.

Discussion
There is evidence of increasing user consolidation among top EHR vendors in the ambulatory primary care setting. A growing trend of adoption of Epic and AthenaHealth products was observed, perhaps indicating that these vendors are more consistently able to provide physicians with a satisfactory user experience. Further work is needed to elucidate causes of switching EHR systems.
Abstract:
Anti-neutrophil cytoplasmic antibody (ANCA)-associated vasculitis could be life-threatening even in patients known to have the disease. Patients can present with severe multi-system failure requiring prompt identification and management of the disease and its complications.

A 32-year old man with known history of ANCA-associated vasculitis presented with a 2-week history of dyspnea, fevers, dark urine, and a 1-day history of hemoptysis. Upon examination, he was febrile, tachycardic, tachypneic, and hypoxic. A systolic ejection murmur (grade 3/6) was noted, consistent with a previous history of hypertrophic obstructive cardiomyopathy. Soon, the patient went into respiratory failure and shock, requiring mechanical ventilation and vasopressor support in the medical intensive care unit (ICU). He was also found to have acute metabolic acidosis and acute renal failure. Differential diagnoses at this time included systemic infection, acute cardiac ischemia, acute venous thromboembolism, and underlying vasculitis exacerbation. Empiric treatment was started for possible infectious etiologies of shock based on available clinical data. Since no definitive diagnosis could be reached, invasive testing was performed. A bronchoscopy was done and bronchoalveolar lavage showed findings consistent of diffuse alveolar hemorrhage with no signs of infection or malignancy. Furthermore, immunologic testing showed a negative anti-glomerular basement membrane (anti-GBM) antibody, low levels of proteinase 3 (PR-3), and an elevated myeloperoxidase (MPO) level. These findings were consistent with the diagnosis of microscopic polyangiitis. The patient was promptly started on methylprednisolone and cyclophosphamide. He also required plasmapheresis because of suspected rapidly progressive glomerulonephritis. He responded well with reversal of organ dysfunction and was transferred out of the medical ICU on day 15.

ANCA-associated vasculitides affect 10 to 20 persons per million per year. With initial immunosuppressive therapy, remission is easily achievable with rates as high as 70 to 90%. Yet in these patients, relapses are still common—affecting 10 to 60% of cases. Therefore, it is imperative to recognize and treat these conditions promptly. Delay in treatment can result in permanent sequelae. Initial signs of relapse should be easy to catch and worsening presentations easy to prevent. Careful monitoring of symptoms is thus of utmost importance, both by clinicians and the patients themselves.
Abstract:
Introduction:
The increasing global burden of gout disease and its impact on the patient’s quality of life calls for new strategies in management. Gout is among the worst-managed diseases due in part to barriers in patient knowledge and perception about the disease. Our aim was to assess the disease-related knowledge and attitude among gout patients in inpatient setting and to identify the barriers to optimal management.

Methods:
A cross section survey of patients admitted to a community hospital for various reasons including acute gout flare from March 2016 through March 2017 was conducted using a 10-item gout related survey. Diagnosis of gout was based on American college of rheumatology classification criteria. Patients were considered to have “good” knowledge if they answered 70% of the questions correctly. Gout survey performance was compared to demographic and medication data. In bivariate analysis, we examined differences in mean survey performance using 1-way analysis of variance (ANOVA) and multivariable Poisson regression. Analyses were conducted using Stata, version 14.2.

Results:
Fifty-eight patients’ surveys were conducted. Most were aged 65 and older (60%) with equal number of males and females. Only 29% of respondents completed college or above and 24% were currently employed. Mean duration of gout in years was 11.8 years. Thirty-nine (81%) patients had at least one visit to the treating physician and thirty-five patients had at least 1 flare (60%) in last year. Most of the patients were taking allopurinol (69%). Good knowledge was demonstrated by 60% of the respondents. Knowledge strengths in terms of percentage of patients having correct answers included the majority of patients identifying that gout was related to elevated uric acid levels (86%); that pain with swelling in the joint as being a sign of acute flare (82%); and that hypertension was associated with gout (89%). Knowledge gaps included: uric acid goals (19%), treatment of acute flare (29%), prophylaxis of flare (37%), duration of ULT (42%), and lifestyle modification (14%). In multivariable regression analysis, gout related knowledge was found to be higher among females (RR-1.5; P=0.03; 95% CI, 1.04 to 2.28); patients with higher level of education (RR-3.4; P=0.04; 95% CI, 1.08 to 10.9) and in those patients using allopurinol (RR-1.9; P=0.05; 95% CI, 0.97 to 4.00).

Conclusion:
We conclude that a large disease related knowledge gap is identified in our survey of gout patients in inpatient setting. Our data informed the areas of major knowledge gaps. We also identified certain demographic and medication factors influencing gout knowledge. To our knowledge this is the first patient education study in gout focusing on a hospitalized patient population, and we believe the inpatient setting may be a focus of opportunity for gout education.
Abstract:
Mrs. S is an 80 year old female with a history of paroxysmal atrial fibrillation (CHAD-Vasc 6 not on anticoagulation), HTN, DM, renal insufficiency, hypothyroidism who was transferred to the cardiac intensive care unit (CCU) for management of NSTEMI and CVA. Three days prior to admission Mrs. S was found down with left facial droop and abnormal gait. CT head at outside hospital demonstrated findings consistent with large subacute/chronic ischemic infarction localized to Right posterior portion of the frontal lobe and head of the Right temporal lobe extending into Right caudate nucleus of basal ganglia. Labs were significant for troponin 0.86 with a peak of 1.99, BNP 4111, lactate 2.6, BUN 59, Creatinine 1.69, WBC 13.8. Patient was transferred to our CCU for further management. Electrocardiogram was significant for QT prolongation and diffuse T wave inversions. Echocardiogram showed normal LV size with akinesis of the distal half of the LV with an overall EF of 35%. Most recent echocardiogram 3 weeks prior to this admission showed an LVEF of 55% with normal LV size and normal global contractility. Patient was loaded with amiodarone and continued on the heparin drip. In our opinion these findings were consistent with apical ballooning syndrome in the setting of an acute ischemic stroke. An optimal heart failure regimen was started and patient was eventually discharged to rehab for her residual left sided weakness.
This case exemplifies the neurohormonal interdependence of the cerebral and cardiovascular system. The condition in which myocardial stunning is of neurogenic origin is termed neurogenic stress cardiomyopathy. (NSC). It is theorized that a sudden increase in intracranial pressure secondary to structural brain damage induces an autonomic storm with elevation in tissue and plasma catecholamine levels. Subsequent release of endogenous catecholamines from myocardial sympathetic nerve terminals has been demonstrated in a baboon model. This causes increased myocardial concentrations of norepinephrine result in myocyte calcium overload, ATP depletion, and cell death causing dysfunction in myocardial contractility. The resulting apical akinesis of the heart is not surprising as the apex is structurally more vulnerable to catecholamine-mediated toxicity than the basal regions due a greater concentration of adrenergic receptors. The area of infarct in this patient is consistent with the case reports resulting in NSC. The insular cortex, lying in the region of the middle cerebral arteries, contains autonomic somatotropin representation and plays a pivotal role in autonomic function integration with dysfunction causing increased norepinephrine and epinephrine tissue and plasma concentrations. ECG changes in NSC: QT prolongation, ST depression, T-wave inversions, and ventricular arrhythmias.
Catch Me If You Can !!! Crohn’s disease that attacks the brain (Case of Crohn's-associated CNS vasculitis)

Abstract:
Central nervous system (CNS) vasculitis is a known rare complication of Chronic Bowel Inflammatory disease. Our case is a patient with Crohn’s disease presenting with neurologic deficits and MRA findings consistent with CNS vasculitis.

A 62-year-old male with a medical history of type 2 diabetes mellitus, Hyperlipidemia, Alcoholism and known Crohn’s disease for 35 years that is currently well controlled on Azathioprine and Mesalamine. The patient presented to the ED few minutes after experiencing disorientation, left-sided facial numbness, mild slurring of the speech, numbness and tingling in the right upper and lower extremity. The symptoms lasted for fifteen minutes. Patient on presentation to the ED had a normal neurologic exam. Non-contrast head CT scan showed right frontal lobe lacunar infarcts of unclear chronicity. Complete blood and metabolic panels, B12, TSH, RPR, hypercoagulability and vasculitis serology were only significant for macrocytic anemia. Patient at that time was considered to have TIA and he continued on Aspirin and high dose statins. TTE with Bubble study, routine 30 minutes EEG, bilateral carotid duplex were unremarkable. On the 2nd day of admission, the patient had a recurrence of his symptoms. Neurologic exam was significant for a mild left facial droop, right upper and lower motor weakness 4 /5, intact sensations, right hyperreflexia, positive right Babinski and positive Romberg's. The patient was transferred to the ICU for close monitoring. MRI brain showed punctate right superior cerebral cortical diffusion hyperintensity with a right old cerebral white matter infarction. MRA of the brain showed extensive areas of stenosis and beading deformities throughout anterior and posterior circulations involving the anterior, middle and posterior cerebral vessels as well as vertebral and basilar arteries consistent with vasculitis. Patient symptoms improved and he was eventually discharged to home and to continue on his immunosuppressive therapy.

Crohn’s disease rarely associates with CNS vasculitis. It's not uncommon in those cases to have negative serologic markers for vasculitis. Crohn’s disease presenting with neurological symptoms with the findings of multifocal cerebral events in the blood supply territories of various cerebral arteries and multiple segmental narrowing of cerebral arteries should raise the suspicion for Crohn's-associated CNS vasculitis. The pathophysiology for Crohn's-associated CNS vasculitis is not well-known. Immunosuppressive therapy to control Crohn's disease is the recommended treatment.
Abstract:
Introduction
For decades, radiotherapy (RT) has been used effectively in the treatment of cancer involving the chest and mediastinum. The late effects of radiation generally become evident most frequently appearing in the second to third decade post-therapy.

Case Report
A 57-year-old man with a past medical history significant for Hodgkin’s lymphoma, dyslipidemia and hypertension, who underwent extensive radiation therapy to the chest and neck 35 years earlier, presented to the Emergency Department complaining of progressively worsening chest tightness and shortness of breath for the last 5 weeks. The patient was experiencing chest tightness radiating to the arms during exertion that immediately resolved with rest. An initial electrocardiogram showed sinus rhythm, a heart rate of 104 bpm, a normal axis and non-specific ST changes. Initial troponin level was 0.497 ng/ml. He was admitted and managed as a non-STEMI. Transthoracic echocardiogram showed decreased left ventricular function with an ejection fraction (EF) of 40% (Image 1), and mid anteroseptal severe hypokinesis, basal anteroseptal and mid inferoseptal mild hypokinesis. Twenty four hours after admission troponins trended up to 0.556 ng/ml.

A cardiac catheterization showed left main disease with 90% stenosis (Image 2), with diffuse disease in the left circumflex artery (Image 2), normal ramus intermedius, dominant right coronary artery (RCA) with 65% stenosis (Image 3) and normal left anterior descending artery (LAD). The EF was 55%. He underwent a quadruple coronary artery bypass surgery without complications. During surgery moderate pericardial adhesions were found.

Discussion
Hodgkin’s lymphoma (HL) is one of the most common cancers in young adults. Coronary artery disease (CAD) has been reported in 5.5–12% of patients undergoing therapeutic chest radiotherapy, usually manifesting 3 to 30 years after radiation exposure. The likelihood of developing CAD secondary to radiation therapy is predicted by the volume of heart radiated, younger age at the time of radiation, pre-existing CAD, inadequate or absent shielding, concomitant chemotherapy, anterior or left chest irradiation and conventional risk factor (smoking, hypertension, diabetes, and hyperlipidemia).

Radiation injury leads to intimal proliferation with collagen deposition and fibrosis. There is a higher incidence of severe left main disease, followed by ostial RCA and LAD artery stenosis correlating with the anatomic location of radiation therapy. Percutaneous revascularization
should generally be favored over surgical revascularization. Aggressive modification of cardiovascular risk factors is essential in patients at risk. Long-term follow-up with regular screening plays an important role in the management of cancer survivors who have undergone radiation therapy.

The importance of this case is to remind us that RT can cause severe structural damage to the heart and that it can develop years to decades after exposure. Surveillance monitoring is essential, because the timing of medical or surgical intervention can be crucial for optimal outcomes.
**Title:** Paraneoplastic Limbic Encephalitis (PLE): Amnesia as a neurologic presentation of thymic malignancy.

**Abstract:**
Paraneoplastic Limbic Encephalitis (PLE): Amnesia as a neurologic presentation of thymic malignancy.
Thomas Joseph MD, Taimur Abbasi MD, Gaurav Sharma MD

**Introduction**
Paraneoplastic syndromes (PNS) and amnesia are common and most often spatially distinct entities that are encountered in clinical practice. The following is a case of Paraneoplastic limbic encephalitis (PLE) related to thymoma with amnesia and confusion as a neurologic presentation.

**Case Description**
A 54-year-old homeless man presented to the emergency department (ED) with complaints of intermittent chest discomfort for the past 1 year. On exam patient seemed confused and anxious, unable to recount his reason for presenting to the ED. Vitals, basic blood chemistries, EKG were unremarkable with a normal troponin level. CT head was negative for any acute intracranial event. Chest x-ray was suggestive of a mediastinal mass. A CT angiography of the chest was performed which showed a large superior mediastinal mass exerting mass effect on the pulmonary trunk along with multifocal pleural and pulmonary nodules. The patient was admitted, needle biopsy was performed on the mediastinal mass and subsequent pathology reported a diagnosis of AB type thymoma. Patient admission was complicated by his persistent state of confusion, progressive amnesia and personality changes. Patient continued to be afebrile and repeat neurologic exams were negative for sensory/motor deficits. Labs showed no vitamin deficiencies. Neurology was consulted and a MRI was subsequently ordered which showed bilateral hyperintense signals within the hippocampal region, suggestive of limbic encephalitis. Paraneoplastic antibodies, associated with thymoma were suspected to be the presumed etiology. Blood screening for neuronal antibodies was positive for anti-Voltage Gated Potassium Channels (VGKC) antibodies. Patient was started on neoadjuvant chemotherapy to reduce tumor burden prior to planned resection. During the course of chemotherapy there was observed a drastic improvement in the patients overall neurologic symptoms, memory and mentation as monitored by regular mini mental exams.

**Discussion**
This patient presented with PLE, a PNS of the nervous system that is characterized by anti neuronal antibodies and malignancy. It often manifests with rapidly progressive memory deficits, psychiatric symptoms and seizures. VGKC are exchange complexes that are associated predominantly with the limbic neuronal cell surfaces. Only about 30% of patients with such antibodies are found to have tumors and an even smaller number are associated with thymoma’s. Usual treatment comprises the use of steroids, plasma exchange, IV immunoglobins or tumor resection. This case illustrates an important educational point for clinicians in regards to formulating a differential when presented with patients having malignancies that are compounded with atypical neurologic symptoms.
Abstract:
Lupus nephritis is a common presenting feature of idiopathic systemic lupus erythematosus (SLE) but uncommonly described in drug-induced lupus. Here we present a case of a 73 year old gentleman with past medical history of hypertension who presented with fatigue for around 6 weeks. He denied any other complaints. Physical exam including vitals were unremarkable except for pronounced conjunctival pallor. Complete blood count revealed pancytopenia and Comprehensive metabolic panel was significant for BUN of 101 and creatinine of 5.2 mg/dL. His baseline creatinine from 6 months was 0.8 mg/dl. A renal ultrasound was done which showed no evidence of obstruction and urinalysis revealed coarsely granular casts, proteinuria and microscopic hematuria. He was admitted to general medical floor for blood transfusion and further evaluation of acute kidney injury. Work up revealed positive antinuclear antibody (ANA ) with titre of double stranded DNA antibodies of 1:1280, low complements level (C3 and C4), positive antineutrophil cytoplasmic antibodies (c-ANCA 1:320) and negative p-ANCA. Anti histone antibody titre was checked as the patient was on hydralazine for 3 years for blood pressure management. It was found to be significantly elevated (15 units) and hence hydralazine was discontinued. A renal biopsy was performed which showed focally necrotising and crescentic glomerulonephritis pauci-immune type, with patchy mild interstitial fibrosis and tubular atrophy. The diagnosis of drug induced lupus nephritis (class III A/C) was suspected in this case. In addition to discontinuing the offending agent, we started pulse dose steroids and one dose intravenous cyclophosphamide was given and was then switched to oral prednisone and cyclophosphamide. The patient’s renal function improved during the hospital stay without needing renal replacement therapy. Over the subsequent period of three months his creatinine improved to 2.8 mg/dL and his pancytopenia improved. Our case highlights the importance of considering the possibility of lupus as a cause of rapidly deteriorating renal function regardless of age and gender. Renal involvement in Hydralazine induced Lupus is uncommon but can result in significant morbidity. Early diagnosis and appropriate treatment are crucial to prevent irreversible loss of renal function.
Case presentation:
A Case of Bowel Perforation From Bevacizumab

Soumyasri Kambhatla MD, Shilpa Arora MD, Saad Alvi MD
John H Stroger Hospital of Cook County, Chicago, Illinois

Discussion:
Bevacizumab is a monoclonal antibody against VEGF. It was approved by FDA in 2004 for use in metastatic colon cancer. Bowel perforation from bevacizumab is a rare but fatal complication. The incidence ranges from 1-4% with mortality rate as high as 50%. Several clinical factors have been postulated to increase the risk of spontaneous bowel perforation from bevacizumab which include history of peptic ulcer disease, diverticulitis, colitis, intestinal obstruction, tumor necrosis, recent sigmoidoscopy or colonoscopy, intact primary tumor, radiotherapy, higher cumulative dose of bevacizumab, or emergent surgery while receiving bevacizumab. Various
mechanisms are thought to mediate bevacizumab induced bowel perforation including disruption of vascular regeneration and resultant bowel ischemia, enhanced chemotherapy delivery to the tumor leading to necrosis, or impaired wound healing. The management depends on the timing of presentation and the overall condition of the patient. Decision regarding surgery should be based on severity, clinical signs, expectation of outcome, patient’s wishes, risks of bleeding and non-healing. Contained perforations are often managed medically with percutaneous drainage only. Bevacizumab should be discontinued permanently if bowel perforation occurs.

References:
Bevacizumab-Induced Bowel Perforation by Sarunas Sleisoraitis DO, Bernard Tawfik MD Incidence and Management of gastrointestinal perforation from Bevacizumab in advanced cancers by Abu-Hejleh T et al
Abstract:
Purpose:
Encephalitis is the most clearly established clinical manifestation of human herpes virus 6 (HHV6) reactivation in allogeneic hematopoietic cell transplants (HCT) and other immunocompromised patients (Gewurz, 2008). Current literature on the subject supports non-specific antiviral therapy normally used for cytomegalovirus—primarily ganciclovir, foscarinet, and cidofovir. Multiple cases of resolved symptomatic HHV6 activation have been described with these therapies. However, they are not without risk. 20-27% of individuals treated with these drugs develop acute renal failure (Izzedine et al, 2005). Immunosuppression with ganciclovir has also been well documented (Bedford Laboratories, 2014). There is limited research activity towards the development of HHV6 specific therapies—perhaps secondary to a lack of clear association between infection and symptomatic disease (Prichard & Whitley, 2014). Treatment of patients with reactivation of HHV6 thus poses a clinical conundrum involving two questions. First, is the reactivation truly symptomatic? Second, in light of the toxic effects of therapy, do the severity of symptoms warrant treatment? Our purpose in this presentation is to contribute to the evidence counseling clinicians against the usage of potentially harmful agents for patients in whom HHV6 treatment is not absolutely indicated.

Case Report:
We present a two-case series of conservative therapy in the setting of equivocal central nervous system (CNS) symptoms.
Patient 1: 38-year-old female presented with 6 hours of radiating occipital headaches down the spine, fever, nausea, and photophobia. Patient had a history of acute lymphoblastic leukemia and was on maintenance chemotherapy. CSF was positive for HHV6 PCR by viral amplification panel. Blood cultures were positive for Acinetobacter jejunii, and tunneled catheter was removed soon after admission as suspected source for the bacteremia. Fevers, radiating headache, and chills resolved entirely with empiric antibiotic coverage within two days. In regards to clinical conundrum above, we saw resolution of concerning symptoms with antibiotic therapy alone, deterring us from proceeding with toxic foscarinet, cidofovir, or ganciclovir therapies. HHV6 levels were undetectable after 14 days of intravenous antibiotic treatment and oral valacyclovir.
Patient 2: 22-year-old female with T-lymphoblastic lymphoma presented with ascending paresthesia, burning pain, and weakness following vincristine seven days prior to admission. Initial concern was for vincristine-induced neuropathy. Two days after admission, lumbar
puncture was performed due to worsening paresthesia. CSF was found to be HHV6+, but due to concern for toxicity she was started on valacylovir instead of standard anti-HHV6 antiviral therapy. The following day, electromyography confirmed diagnosis of Guillain-Barre Syndrome, acute motor axonal neuropathic (AMAN) type. Patient underwent plasma exchange with resolution of neurologic symptoms.

Discussion/Conclusion:
No formal guidelines currently exist for the treatment of HHV6 reactivation. We propose here a brief guide for withholding toxic antiviral agents in patients with concurrent CNS symptoms potentially attributed to etiologies other than HHV6 reactivation.
Title: Fish handler's disease

Abstract:
Introduction: Sepsis is a leading cause of mortality in the United States. Thorough history and physical examination often provides clues to the identity of the responsible pathogen. Moreover, some comorbidities can influence susceptibility to certain organisms that rarely cause bacteremia in healthy individuals.

Description: A 68-year-old male presented to the E.D. with complaints of left shoulder pain that began one month prior. Hypertension was his only known medical problem and he did not regularly follow up with a physician. Review of systems revealed subjective fevers, generalized malaise, and weakness. He was a non-smoker, drank 2 beers daily, and did not use recreational drugs. None of his close contacts were sick. He had recently traveled to Colombia, and on further investigation he recalled puncturing his finger with a fishing hook three months prior. On physical examination he was in minimal distress and initial vital signs were within normal limits. Cardiovascular examination revealed a left sternal border systolic murmur. Chest examination showed bibasilar rales. Abdominal examination was normal. There was left sternoclavicular joint tenderness without overlying warmth or erythema. No focal neurologic deficits; however, during the hospital course he developed altered mental status. Labs showed WBC 9600, bands 8%, hemoglobin 9.8, platelet 52000, BUN 23, creatinine 1.39, liver function tests normal. Chest X-Ray was normal and MRI of the left shoulder was unremarkable. Blood cultures were positive for Erysipelothrix rhusiopathiae. Transthoracic echocardiography showed ejection fraction 60%, moderate to severe aortic regurgitation. Transesophageal echocardiography showed aortic valve vegetations. Brain MRI showed deep white matter and subcortical infarcts, suggestive of an embolic source. Hepatitis C antibody was positive. HCV RNA 1.9 log IU/mL.

The patient was started on meropenem for treatment of sepsis, endocarditis and suspected septic arthritis involving the left sternoclavicular joint due to E. rhusiopathiae. He was scheduled for a follow up with a cardiovascular surgeon and hepatologist at discharge.

Discussion and conclusion:
Erysipelothrix rhusiopathiae is a gram-positive bacillus that is infrequently responsible for infections in humans. It is transmitted through direct contact with animals or their products. Infection with this pathogen is classified into three forms: a localized cutaneous form, a generalized cutaneous form and a bacteremic form. Case reports describe an association between developing sepsis with E. rhusiopathiae and chronic liver disease.
In our patient, the identification of *E. rhuzipathiae* sepsis lead to further investigation for an underlying cause that could have predisposed him to the severe form of the infection. Eventually, we were able to diagnose cirrhosis and HCV infection which were asymptomatic.
Abstract:
Introduction:
Diagnosing peripheral polyneuropathy with overlapping features and negative etiological workup can be a diagnostic and therapeutic challenge. We present a case of a progressive polyneuropathy with mixed features with significant clinical response to Intra Venous Immunoglobulins (IVIG).
Case:
A 60-year-old female presented with progressive pain & paresthesia involving upper and lower extremities for 8 weeks. Paresthesia was ‘glove and stocking’ in distribution and progressed to involve proximal extremities and face with no motor involvement with poor response to Gabapentin and Amitriptyline. Neurological exam on admission showed symmetrical sensory deficits to light touch, severe loss of vibration and moderately reduced proprioception with reduced deep tendon reflexes. Motor strength and was normal. Comprehensive lab works only showed elevated ESR and CRP with negative autoimmune panel and paraproteins. There was no evidence of any paraneoplastic syndrome but the Electromyography (EMG) and Nerve Conduction Velocities (NCV) were consistent with ‘axonal large fiber sensory neuropathy’ affecting upper and lower extremities without evidence of motor involvement or demyelination. A lumbar puncture showed increased cerebrospinal fluid protein without pleocytosis but the neuraxial imaging failed to highlight any other cause. Negative inspiratory force was normal. The patient was suspected to have ‘Chronic idiopathic axonal polyneuropathy’ (CIAP) or variant of ‘Chronic inflammatory demyelinating polyneuropathy’ (CIDP). Patient was started on IVIG for five days resulting in significant improvement of symptoms.
Summary:
There is a temporal continuum between acute inflammatory demyelinating polyneuropathy (AIDP), the demyelinating form of Guillain-Barre syndrome, and CIDP and observation of the patient over time can clarify whether the clinical course is that of AIDP or CIDP. The classic form of CIDP is fairly symmetric and motor involvement is greater than sensory. Weakness is present in both proximal and distal muscles. Sensory impairment in CIDP is usually greater for vibration and position sense than for pain and temperature sense. Cranial nerve and bulbar involvement occur in a minority. However, in the absence of electrodiagnostic evidence of demyelination the diagnosis becomes challenging. At the crossroads of such diagnostic dilemma this case proves the therapeutic significance of IVIGs, which is well established in the treatment of CIDP and less so in cases of CIAP.
Pancreaticopleural fistula is an uncommon complication of chronic pancreatitis and a rare cause of pleural effusion (less than 1% of cases). Diagnosis is often obscured given the extrathoracic source of pulmonary symptoms. Depending on its severity, pancreaticopleural fistulas may be treated conservatively, endoscopically, or surgically; however, at present, there are no established guidelines regarding these treatment strategies.

Here, we present a 49-year-old gentleman with a past medical history of alcohol-induced pancreatitis who presented to the hospital with pleuritic left-sided chest pain. He previously sought medical care twice in the same month for similar symptoms for which he was prescribed antibiotics and opioids for presumed pneumonia on both occasions. On presentation, he was afebrile with a normal heart rate, in mild discomfort but no respiratory distress, with a non-tender abdomen and decreased bibasilar breath sounds on physical examination. Initial labs were unremarkable including a troponin level. Chest x-ray demonstrated a moderate left pleural effusion. Ultrasound-guided thoracentesis produced 1000 mL of brown fluid, exudative per Light’s criteria, with a pleural fluid amylase level of 2783 U/mL. Computerized tomography of the abdomen and pelvis revealed numerous fluid collections extending superiorly from the pancreatic tail, concerning for mediastinal pseudocysts and pancreaticopleural fistula, for which octreotide therapy was initiated. Endoscopic retrograde cholangiopancreatogram (ERCP) demonstrated mild diffuse dilation of the main pancreatic duct and evidence of a pancreatic tail leak for which pancreatic duct stenting was performed. Post-procedure course was complicated by post-ERCP pancreatitis (PEP), which resolved with supportive care and IV hydration.

Following treatment for PEP, the patient’s presenting symptoms had resolved, and interval improvement of the pleural effusion and pseudocysts was noted on imaging. The patient was discharged with continued octreotide therapy and follow-up in six weeks to assess closure of the fistula.

Pancreaticopleural fistula is a rare entity, only seen in 0.4% of patients presenting with pancreatitis. Posterior disruption of the pancreatic duct and ascension of the fistulous track into the pleural cavity gives rise to large-volume pleural effusions. In conjunction with markedly elevated amylase levels within the pleural fluid, computerized tomography, magnetic resonance cholangiopancreatography, and ERCP may aid in delineating the fistulous tract, with sensitivities approaching 47%, 78%, and 80%, respectively. Treatment largely depends on
pancreatic ductal anatomy and is predicated on thoracentesis, administration of somatostatin analogues (octreotide) and, if amenable, endoscopic repair of obstructions/strictures and pancreatic stent placement. If conservative treatment fails or endoscopic therapy is not feasible, surgical intervention is the definitive line of management, usually in the form of distal pancreatectomy.
Extraocular Carcinoid Tumor

Carcinoid is a slow growing malignant neuroendocrine tumor that can lead to carcinoid syndrome when its vasoactive substances bypass hepatic degradation. Majority is found in the GI tract or lungs, but metastases can be found in other sites.

Patient is a 63 year old Caucasian male with past medical history of htn, dm, ckd stage 2 who presents to the oculoplastic clinic at Loyola university medical center by referral from his primary care physician for asymptomatic left sided proptosis for 6 months. Patient denies pain, diplopia, blurry vision, flashes, or floaters but has noticed gradual increasing proptosis of his left orbit throughout the past 6 months. His primary care physician ordered a MRI orbit that revealed enlargement and enhancement of the left superior rectus musculature. His last known eye exam 2 months ago by his optometrist was noted to be within normal limits other than minor refractive error. His exam at the initial visit revealed pupils equally reactive to light without APD, full visual fields bilaterally, intraocular pressures within normal limits, and full color vision. His manual refraction revealed correctable hyperopia in his right eye. Patient, however, had minor restriction in upgaze in his left extraocular movement. Hertel exophthalmometer revealed a 4 mm greater protrusion of his left orbit. Slit lamp exam revealed superior nasal and temporal injection of his conjunctiva on the left. Fundus exam was unremarkable bilaterally. Patient received a biopsy of the mass which revealed a well differentiated neuroendocrine tumor with CDX-2 positivity suggesting
gastrointestinal origin. CT abdomen/pelvis revealed 4 small lesions in the liver and moderate right sided hydronephrosis. Patient noted symptoms of diarrhea and abdominal pain for the past 8 months, and was referred to Heme/Onc. Chromogranin and 5HIAA was shown to be elevated, and patient was started on octreotide treatment for carcinoid. An octreotide scan was ordered, demonstrating abnormal uptake in the left orbit, mediastinum, myocardium, pericardium, abdomen, and pelvis. Patient is now scheduled to receive a MRI cardiac to identify heart metastases, a urology referral for his increasing right hydronephrosis, and may receive a frontalis sling for his new onset ptosis of the left eye after biopsy.

This case illustrates how investigation in an asymptomatic finding can expose an underlying extensive pathology that is treatable. This patient’s proptosis revealed his carcinoid syndrome symptoms that he had always neglected. Patients may not reveal their problems unless specifically asked, so a complete history and physical is important for every patient. Presentation of carcinoid to the extraocular muscles is a rare finding; only a handful of cases are documented in the literature with no current guidelines on treatment.
Abstract:
Introduction
Individual risk assessment for Venous Thromboembolism (VTE) coupled with Caprini Risk Score (CRS) targeted prophylaxis based on the score is effective in reducing the probability of post-operative VTE. Critics contend that the tool is time-consuming for healthcare providers and limited to the English Language.

Aim
To compare scores calculated by a patient to scores in the same patient calculated by a blinded physician.

Methods
We translated, a validated patient-completed CRS from English to Polish following a standardized process. First, two native polish speaking authors independently translated each form into Polish (WK, AI,). Then, a third (LP) author unified each individual translation to create a single version for each language. Finally, all the translators reviewed the last form for accuracy. We conducted a pilot study to identified additional challenges. In this phase, we conducted a standardized interview on hospitalized patients. During the first part of the interview, patients calculated their CRS using the Polish form. Subsequently, a native-speaker physician blinded to the patient’s answer, scored the CRS for the same patient. At the end of the interview we tabulated both forms for analysis. Based on the interim analysis the forms did not require additional modifications and therefore was used in the final validation process. In a 15 week process, we prospectively enrolled patients for the validation phase. We calculated the sample size to be 37 assuming power of 80% and an alpha of 0.05. We calculated the individuals’ questions and categorized scores using SPSS version 24 to estimate Kappa, linear correlation and Bland Altman test. A Kappa value over 0.8 was defined as “almost perfect agreement”.

RESULTS
We recruited 39 patients, 23 (59%) were women, with a mean age of 62 years (range 29-91), with less than college education (74%). The mean CRS calculated by the physician was 4 (range 0 - 13). Patients spent on average 6 minutes (range 3 - 8) filling the form. The agreement level was almost perfect when the CRS was categorized following ACCP guidelines recommendations
Spearman's correlation coefficients between patient and physician-completed forms were 0.981 (p < 0.01). The Bland Altman plot did not show any trend for extreme values.

CONCLUSION
We translated and validated a self-reported CRS form in Polish to assess peri-operative thrombotic risk. The new version has an almost perfect agreement between patient and physician completed. Completing the form was not time-consuming or complex.
Abstract:
A 70-year-old male smoker with history of COPD and Crohn’s (not on immunosuppressants) presented with a three-day history of increased shortness of breath and labored breathing. He denied fever, cough, sputum production, or night sweats. A week prior, he was treated for two days at an outside hospital for community-acquired pneumonia (CAP). The patient was discharged with levofloxacin and home oxygen. On admission to our facility, the patient was found to be hypoxemic with an oxygen saturation of 55% and required intubation. The patient had leukocytosis (15,000/mcL) and an elevated procalcitonin (0.85 ng/mL). Vancomycin and meropenem were started for empiric treatment.

Chest x-ray and CT chest demonstrated large confluent airspace consolidation throughout the right upper lobe with central hypo-attenuation, multiple internal cystic/cavitary spaces, and surrounding irregular septal thickening consistent with extensive necrotizing cavitary pneumonia. Blood cultures, legionella urine antigen, fungal smear and culture, fungal serologies, AFB smear and culture (x3), beta-D-glucan assay, and HIV antigen were all negative. Bronchoscopy was performed, and bronchoalveolar lavage (BAL) smear was unremarkable. However, BAL culture grew Rothia mucilaginosa identified by Matrix Assisted Laser Desorption/Ionization-time of flight mass spectrometry along with catalase-positive identification and gram-positive staining. The patient was stabilized and extubated within 36 hours. Cefdinir was prescribed for 14 days, and the patient was instructed to follow up for a repeat chest x-ray four weeks after discharge.

R. mucilaginosa is a gram-positive, catalase-positive coccus found in the upper respiratory tract and oral cavity that can cause opportunistic infection in immunocompromised hosts and is mainly associated with device infection and endocarditis. This bacterium was previously known as Stomatococcus mucilaginosus and is often misidentified as stapholococcus, streptococcus, or micococcus. A recent literature review found 20 cases of infection with R. mucilaginosa between the years 1970 and 2014. Four of the 20 cases involved an immunocompetent host. Necrotizing pneumonia accounts for only 4% of all CAP, and infection with R. mucilaginosa in an immunocompetent patient amplifies the rare nature of this case.

R. mucilaginosa is often resistant to aminoglycosides and fluoroquinolones but has shown susceptibility to vancomycin and beta-lactams. R. mucilaginosa is usually covered by a standard beta-lactam-based CAP regimen but not fluoroquinolones alone. This could be why our patient
did not improve after discharge from an outside hospital when treated with only levofloxacin. We suspect impaired pulmonary defenses due to COPD led this immunocompetent patient to develop necrotizing pneumonia secondary to R. mucilaginosa infection. It is important for providers to consider R. mucilaginosa as a potential cause of pneumonia in both immunocompromised and immunocompetent patients, since early detection and early treatment can help prevent medically unnecessary invasive interventions.
Abstract:
Introduction: IgG4 related disease (IgG4-RD) is a relatively new fibroinflammatory condition characterized by lymphoplasmacytic infiltrate rich in IgG4 positive plasma cells. It is linked to autoimmune pancreatitis (AIP) and recognized in 2003. Elevated serum IgG4 is often present but not required for diagnosis. IgG4-RD has been described virtually in all organ system.

Case presentation: 89 years old white male with past medical history of atrial fibrillation, coronary artery disease, mechanical heart valve, on warfarin, presented to the Emergency department(ED) with an episode of epistaxis following a few weeks of upper respiratory infection symptoms such as a cough, pleuritic chest pain, runny nose, sneezing etc. Chest X-ray(CXR) was showing Right lower lobe(RLL) consolidation. The patient was treated for pneumonia and follow-up CXR was recommended in 3 months to confirm resolution of CXR finding. The patient did not follow-up, but 6 months later he presented in ED with low back pain, fatigue and weakness. Patient was a retired Janitor, living with family, and has never smoked tobacco. His vital signs, CBC and BMP were unremarkable. CXR showed a similar finding, now raising a question of mass. A contrast enhanced CT scan of the chest was done which showed 4 x 4.5 x 3cm mass in RLL, with no lymphadenopathy. A core biopsy of the mass was done by an interventional radiologist. Meanwhile, we worked up for his back pain and weakness. MRI of back showed degenerative disc disease. CPK, aldolase, ESR, CRP are within normal limit (WNL). A neurologist was consulted. CT and MRI of the head were WNL. Lumbar puncture was performed for the concern of leptomeningeal disease in this context of the big lung mass. The CSF analysis was WNL. The patient was then transferred to inpatient rehabilitation facility. Biopsy result showed marked lymphoplasmacytic infiltration with increased IgG4 positive plasma cells. There were mean of 133 IgG4 positive plasma cells per hotspot, counted over three hotspots, confirming the diagnosis of IgG4-RD. Oncology was also consulted for concern of plasma cell neoplasm. Serum protein electrophoresis with immunofixation was WNL. Lipase was normal. Serum IgG levels including IgG4 were WNL. Rheumatologist started the patient on oral prednisone.

Discussion:
This case illustrates that IgG4-RD with isolated lung involvement can occur, and it may not be associated with AIP. IgG4 related respiratory disease can be asymptomatic or can present with hemoptysis, cough, pleurisy, pseudo-tumor, pleural thickening, and interstitial pneumonia. This rare and relatively new disease has a limited study on epidemiology. The recommended first
line treatment is an induction of remission with glucocorticoid unless a contraindication exists, followed by maintenance therapy for 3-6 months. Research is underway for other treatment options like rituximab and plasmablast directed therapy with CD19 monoclonal antibody.
Postpartum Induced Thrombotic Thrombocytopenic Purpura.

Abstract:
Introduction

Post partum induced Thrombotic Thrombocytopenic Purpura (TTP) is a rare but feared complication of pregnancy. We present a 28 year old gravida 1 para 1 female who recently had a full term normal spontaneous vaginal delivery who presented with severe acute thrombocytopenia and anemia in whom ultimately etiology of thrombocytopenia was found to be due to TTP.

Case Report:

A 28 year old Gravida 1 Para 1 female with no significant past medical history, who recently had a full term pregnancy without any complications via normal spontaneous vaginal delivery presented with lightheadedness, nausea and vomiting for 1 week. Upon presentation she was hemodynamically stable. Her physical examination was unremarkable. Her blood work showed severe acute anemia with Hgb of 6.1 and severe acute thrombocytopenia with platelet count of 27. Previous lab works showed mild thrombocytopenia. Creatinine was elevated at 2.15 and bilirubin elevated at 1.9. Her LFTS and PT/INR were within normal limits. Her peripheral blood smear showed 6-7 schistocytes. Clinical picture was concerning for TTP and a ADAMS13 level was checked and was found to be less than 5 %, this confirmed the diagnosis and she was immediately started on plasmapheresis. Patient underwent a total of 10 session of plasmapheresis. She received pre treatments with solumedrol and Benadryl before each plasmapheresis session.Her symptoms improved and her acute anemia and thrombocytopenia resolved. Her creatinine and bilirubin were also normalized and patient was discharged home with a short course of prednisone.

Discussion

Post partum induced TTP is a very rare yet, feared complication of pregnancy. Since the differences between causes of thrombocytopenia associated with pregnancy are subtle, its very important to have correct diagnosis since the management are different. It is important to note that the classical pentad of symptoms associated with TTP may not be present in all patients. Our patient, had no neurological symptoms or fever upon presentation. Therefore a high index of suspicion for TTP is warranted in the setting of thrombocytopenia, microangiopathic
hemolytic anemia and kidney dysfunction alone. Another important consideration is to check for ADAMS 13 level before starting Plasmapheresis since post plasmapheresis ADAMS13 values are not accurate. A value less than 10% suggest TTP.

Conclusion
There are numerous causes of thrombocytopenia associated with pregnancy, yet the differences between them are very subtle. This case report further illustrate the importance of diagnosing the cause of thrombocytopenia since treatment depend upon the diagnosis.
**Last Name:** Kwan, MD PhD  
**First Name:** Jennifer  
**Category:** Patient Safety/QI  
**ACP Member:** 2135063  
**Additional Authors:** Bharath Raju MD, Michelle Yoo, Renee Cheng, Deepa Yohannan MD

**Title:** Reminder Increases Statin Adherence in High Risk ASCVD Score Patients

**Abstract:**

Introduction: Statin therapy contributes significantly to decreasing the risk of cardiovascular events including coronary artery disease (CAD) and stroke. Evidence based guidelines for the use of statins leverage the Atherosclerotic Cardiovascular Disease Score (ASCVD). Patients 40-75 years of age with an ASCVD >7.5% are at significantly increased risk for CAD over the next 10 years and are categorized as high risk for CAD. Significant risk reduction for CAD can be achieved by starting a statin in this group. However, many patients in this high risk group may not have been started on a statin and do not have contraindications for starting one. Our aim was to identify these patients and to see if a letter/phone call reminder to such patients could help increase starting statin therapy compared to a group that did not get this reminder.

Methods: 100 high risk ASCVD score patients at the VA hospital who were not on a statin and fit the criteria for starting a statin (no statin allergies, no significant LFT abnormalities, LDL <70, not already on a statin from outside hospital) were identified and extracted for each the intervention and control groups. For the intervention group, a letter was mailed to each patient educating them about their risk score and recommended that they discuss the use of statin therapy with their primary care physician. A phone call was made to the patient to remind them to schedule a primary care visit to discuss statin therapy. For the control group, we identified one hundred comparable patients who were not on a statin but did not receive the intervention. Over a three-month period, we tracked how many patients were started on statin therapy for both groups.

Results: 34 of the 100 patients originally identified in the intervention group versus 8 of the 100 patients originally identified in the control group were started on statin therapy. Compared to the control group, a 4 fold greater number of patients were started on statin therapy in the intervention group over the same three month period.

Conclusion: A letter/reminder phone call sent to high cardiovascular risk patients educating them about their risk score and recommending that they discuss the use of statins with their primary care physician, helped increase adherence to recommended statin therapy. The low cost and effort of the intervention suggests that this could easily be generalized to a much larger population. However, further study with a larger population size over a longer period of time will need to be done to fully validate these results. Additionally, automation of such a
reminder for high risk ASCVD patients who are eligible for statins can help increase adherence while further reducing time needed on the clinician’s part.
SUMMARY OF BACKGROUND DATA:
Amyloidosis is a broad disease, including a number of disorders all of which involve intracellular and/or extracellular deposition of protein through a variety of pathological mechanisms which we will discuss. Primary solitary amyloidosis, referred to as an amyloidoma, are more rare than their systemic counterparts; and are commonly found in bone, heart, liver, kidneys, skin, and gastrointestinal system. In these focal cases, the deposition of the abnormal protein is localized and not secondary to any systemic process or plasma cell dyscrasia.

CASE DESCRIPTION:
A 59-year-old Haitian woman was admitted to our internal medicine department with severe shortness of breath, weakness, and epigastric discomfort. Chest x-ray showed a shifting of the mediastinum due to a mass previously found to be a solitary amyloidoma of the left lung. Surgery was planned for removal of the mass. She underwent a left extended posterolateral thoracotomy with excision of 6th rib, left lower lobe resection, partial left parietal pleurectomy, excision of left chest wall tumor, excision of left paraspinal tumor, excision of mediastinal tumor and repair of left diaphragm. There was no cancer on pathology. Her follow up appointment showed the patient to be afebrile, hemodynamically stable, with no evidence of fat necrosis. She showed appropriate recovery from the surgery with the exception of some drainage from the thoracotomy wound. She will be continued to be monitored via outpatient clinic to ensure proper recovery and complete resolution of her disease.

METHODS:
In this report, we present a rare case of primary solitary amyloidoma of the lung in a 59 year old Haitian female and provide a literature review of solitary amyloidoma.

CLINICAL SIGNIFICANCE:
It is important to differentiate primary solitary pulmonary amyloidoma from granulomas, pneumonia, pneumothorax, primary lung tumors, or metastasis. A diagnosis of amyloidoma is made only after special staining of the tissue via biopsy. Patients are expected to have excellent prognosis after surgical intervention, but there are limited follow up studies at this time.

CONCLUSIONS:
Although primary solitary amyloidosis is a rare form of amyloidosis, it often has an excellent prognosis. Depending on the severity of the case and the type of amyloid involvement, treatment may range from observation to a surgical resection. In these cases of surgical intervention, management must include aggressive local resection of mass and appropriate
supportive and symptomatic care. Complete evaluation for systemic amyloidosis is crucial to determine follow up care.
A positive blood culture of coagulase-negative staphylococcus is not always a contaminant. Staphylococcus lugdunensis is a coagulase negative staphylococcal (CoNS) skin commensal. Blood stream infections caused by S. lugdunensis are uncommon but may have an increased rate of serious complications reminiscent of Staphylococcus aureus. We present a case of native valve, permanent pacemaker (PPM) lead associated infective endocarditis that was successfully treated with antibiotics and surgery.

A 56 year old woman with a history of complete heart block requiring PPM placement 20 years ago presented with intermittent fever and chills for 2 weeks. She was afebrile with unremarkable PPM site and chest examination. Blood cultures drawn in ED grew CoNS in one set of culture bottles, which was interpreted as a contaminant. She presented 2 weeks later with increasing frequency and severity of fever with chills and PPM site soreness. Physical examination was remarkable for BP 101/76, HR 92/min, temperature 101.3F and RR 18. PPM site warmth and fluctuance was noticed without discharge. Labs were remarkable for hypochromic normocytic anemia. Empiric Vancomycin was given in the ED after two sets of blood cultures were drawn. She was admitted for work up of infective endocarditis/PPM site infection. Patient was hemodynamically stable so antibiotics were held while awaiting blood culture results.

Transthoracic echocardiography (TTE) showed aortic and tricuspid valve mobile masses with thickened biventricular pacemaker leads. Blood cultures started growing Staphylococcus lugdunensis by day 4. Transesophageal echocardiogram confirmed the findings of TTE. Intravenous vancomycin therapy was started and later deescalated to intravenous oxacillin based on susceptibilities. Repeat cultures were drawn to document clearance. She underwent PPM removal and temporary pacemaker placement. Repeat ECHO showed unchanged aortic and tricuspid valve mass, and a superior vena cava (SVC) mass extending to right atrium concerning for infected thrombus. Patient was continued on culture directed therapy with serial TTE showing regression of valve masses but persistent right atrial and SVC mass. She underwent successful operative removal of the suspected thrombus/mass with new PPM placement followed by 6 weeks of IV oxacillin. Follow up is remarkable for clinical and imaging evidence of resolution of infection.

This case highlights the need to maintain an index of suspicion when the blood culture grows CoNS in the right clinical setting. Identifying S. lugdunensis bacteremia with proper speciation of CoNS can prevent life threatening complications. Pathogenicity is related to biofilm.
formation and binding to Von Willebrand factor which enables it to cause more aggressive infections. Hardware associated, native valve endocarditis, as in this case, responds to culture directed antibiotic therapy and surgical removal of the device.
Abstract:
Introduction: Flail chest is commonly managed by surgical teams, however, medical physicians increasingly have to manage this condition in patients with multiple medical comorbidities. We present a patient with flail chest and highlight key aspects of medical management.

Case: A 46-year-old female with history of HTN, DM, OSA, and CHFpEF presented after a motor vehicle accident. Physical exam was significant for paradoxical right anterior chest wall motion on inspiration and tenderness over her left distal femur. CT imaging revealed posterolateral and peri- sternal fractures of multiple ribs, as well as a left femoral fracture. She underwent repair of the femoral fracture and received an epidural catheter for lower extremity pain. Postoperatively, she continued to have respiratory difficulties with frequent desaturations. Imaging demonstrated bilateral pleural effusions more significantly on the right side. Presumed causes of hypoxemia included respiratory splinting from rib pain, atelectasis from poor inspiratory effort, obstructive sleep apnea, and vascular congestion. The patient was managed with oxygen by nasal cannula and nighttime CPAP, diuresis for pleural effusions and vascular congestion in the setting of HF, incentive spirometer, and scheduled oxycodone and acetaminophen, with morphine for breakthrough pain. The patient was ultimately discharged without oxygen requirement.

Discussion: The intercostal muscles create negative intrapleural pressure that is necessary for air entry into the lungs. Flail chest is a phenomenon where multiple ribs are fractured in two or more places, creating a flail segment that experiences, on inspiration, the same phenomenon as outside air—being pulled in; the opposite applies upon expiration. Such paradoxical motion is the pathognomonic finding of flail chest. Traditionally, flail chest is managed conservatively with adequate pain control and respiratory support. When using opioids, however, a balance should be reached between effective pain control and suppression of respiratory drive. Epidural catheters represent an infrequently used but promising method of aggressive pain control, as one retrospective analysis found that only 8% of patients received this treatment modality. This patient received epidural pain control for lower extremity pain, but may have additionally benefited from epidural therapy targeting thoracic pain. Additionally, although not performed in this patient, surgical treatment for flail chest is becoming more prevalent. In a recent systematic review, Cataneo et al. found no difference between surgical or conservative management in terms of in mortality or overall length of hospital stay. Surgical rib fixation,
however, may be effective in reducing pneumonia, length of ICU stay, duration of mechanical ventilation, and likelihood of requiring tracheostomy.

Conclusion: For cases of flail chest with minimal complications and without necessary ICU stay, a conservative approach is indicated. The case presented above highlights key aspects of medical management for flail chest including adequate pain control and respiratory support.
Abstract:
Corticobulbar tracts originate in the primary motor cortex of the frontal lobe, descend through corona radiata and genu of the internal capsule and terminate in the brain stem. Here they synapse on to the lower motor neuron nucleus (LMN). In classic neuroanatomy, the corticobulbar projection to the hypoglossal nucleus has been described as bilateral, predominantly from contralateral side and hence cortical lesions of the internal capsule affect the contralateral half of the tongue. A study involving 300 patients with acute supranuclear unilateral ischemic motor strokes looked into the presence of tongue deviation in these patients. Tongue deviation was found in 29% of stroke patients and 5% of controls. Tongue deviation was most common in patients with clinical features of the non-lacunar stroke subtype (56%) or in those with cortical or large subcortical infarctions on brain CT scan (55 and 45%, respectively). Weakness of the arm was significantly associated with presence of tongue deviation and all tongue deviations were associated with supranuclear 7th nerve palsy. We are presenting a case of 64-year-old man presenting with isolated upper motor neuron (UMN) palsy of hypoglossal nerve from an ischemic stroke.

64 years old male with past medical history of cocaine abuse and newly diagnosed hypertension presented with slurring of speech and abnormal sensation in his right cheek for past 2 days. He denied weakness or abnormal sensation anywhere else and the review of system was otherwise negative. Neurological exam was only remarkable for dysarthria and deviation of the tongue to the right side upon protrusion, with preserved power of all the other tongue movements (UMN palsy). Remaining part of the neurological exam and physical exam was within normal limits. Routine labs including complete blood count, basic metabolic panel, lipid profile and hemoglobin A1C were also within normal limits. CT brain showed chronic microvascular disease, with age in-determinate bilateral corona radiata lacunar type infarcts. CT angiogram of the neck vessels and intracranial vessels did not show any flow limiting occlusion or dissection of internal carotid artery. Echocardiography done as part of stroke workup did not show any intracardiac shunt or thrombus. Management of stroke was initiated with aspirin, atorvastatin and blood pressure control while awaiting the MRI. MRI Brain showed a small acute infarct in the left posterior frontal and periventricular areas. The location of the stroke (corticobulbar fibers of the hypoglossal nerve) corroborated with the clinical presentation. Thereafter, patient’s symptoms dramatically improved and he was asymptomatic by the 3rd day. The tongue deviation also resolved.
Hence this case presents a rare and subtle presentation of ischemic stroke, the supranuclear XII nerve lesion, where a thorough and timely neurological exam and its transient nature are usually the most important clues.
Title: An amoeba at a local restaurant

Abstract:
65 year old heterosexual male presents to ER complaining of bloating sensation for 5 days with mild abdominal pain and diarrhea for 2 days. According to patient he started to feel bloated immediately after eating a beef burger which was one week old; two days ago he had three episodes of nonbloody and watery diarrhea 3 episodes. His abdominal pain is described as achy, 2/10 pain throughout all quadrants, generalized, nonradiating. He denies nausea, vomiting, fever, chills, recent travel stating he has not left Chicago for last 18 years, denies sick contacts, no one sick in the household, pets in house or contact with pets. His usual diet consists of home made food consisting of fish, burgers, hotdogs and pork which his whole family also consumes. Patient is from Arkansas, moved to California at age 18 and then moved to Chicago in 1988; has never traveled outside the country. Objective findings were significant for a fever and abdominal exam showed tenderness to mild palpation diffusely, no masses, no organomegaly. CT revealed a is small amount of fluid along the posterior right lobe of the liver with areas a septated cystic mass that measures 9.4 x 6.6 x 6.5 cm. There is surrounding decreased attenuation in the liver suggesting edema and Amoebas antibody was positive. Final diagnosis was Invasive Entamoeba histolytica infection with liver abscess due to local beef consumption. Entomoeba histolytica infections are normally uncommon in urban areas except for travellers, immigrants or men who have sex with men but uncommon to those without these risk factors. Patient was treated with ceftriaxone and metronidazole.
Abstract:
Introduction: Acute pancreatitis is an inflammatory disorder of the pancreas, with alcohol and gallstones being the most common etiologies. Serum lipase is a sensitive marker for diagnosis with a negative predictive value approaching 100%. Here we describe a case of acute pancreatitis without elevation of lipase levels, in the setting of hypertriglyceridemia.

Case: A 49-year-old Hispanic man presented with 1-day history of severe epigastric pain radiating to the back that started after drinking several cans of beer over the previous 2-3 days, associated with two episodes of non-bloody, non-bilious vomiting. On abdominal exam he had epigastric tenderness with normal bowel sounds. Laboratory studies were ALT 39 U/L, AST 47 U/L, GGT 114 U/L, lipase 64 mg/dl, triglycerides 1803 mg/dl. BMP, CBC, protein, INR and bilirubin were normal. He had presented with similar complaints after binge drinking 1 year ago, with lipase of 61 U/L and triglycerides of 728 mg/dl. He was diagnosed with acute pancreatitis after CT imaging had shown peripancreatic edema involving the head and neck of pancreas. Repeat CT on current admission showed resolution of edema, and no pancreatic necrosis, calcifications, pancreatic/biliary duct dilation or gallstones.

Our patient was treated for acute alcoholic pancreatitis with bowel rest, hydration and pain control. Hypertriglyceridemia was managed with insulin, fenofibrate and atorvastatin. He improved rapidly with resolution of pain by day three, and could tolerate oral diet. Triglycerides improved to 515 mg/dl. He was discharged home with fenofibrate and atorvastatin. He remained asymptomatic on follow-up with improvement of triglycerides levels to 81 mg/dl.

Discussion: Diagnosis of acute pancreatitis is generally established by two of the following criteria: 1) abdominal pain consistent with the disease, 2) serum amylase/lipase levels three times the upper limit of normal, 3) characteristic findings on imaging. Serum amylase and lipase levels are commonly used biochemical markers for diagnostic workup, with lipase being more sensitive than amylase. Plasma triglycerides of more than 500 mg/dL have been known to interfere with amylase measurement by causing a falsely low level, believed to be related to a circulating inhibitor and interference of light transmission from the serum. It can be corrected for by serially diluting the sample. Although this effect of hypertriglyceridemia on measurement of lipase has not been described in literature, further studies may be indicated to investigate this
relationship. It is rare to find acute pancreatitis without elevation of serum lipase1,2,3 and few cases of pancreatitis with normal lipase have been reported.

In conclusion, acute pancreatitis can have a variable presentation and severity. It is important to consider this diagnosis in appropriate clinical setting without elevation of serum biomarkers and to be aware of the effect of hypertriglyceridemia on interpretation of these results.
Streptococcus bovis: A case of bacteremia associated with non-malignant colon neoplasia.

Abstract:
Streptococcus bovis is part of the normal flora of the human gut. Colorectal cancer is one of the most significant associations with S. bovis bacteremia; however, other benign conditions like large polyps are also infrequently associated with it. We report the case of a patient who developed infective endocarditis (IE) secondary to S. bovis bacteremia and was found to have a large tubular adenoma.

A 76 year old man with past medical history of hypertension presented with generalized fatigue and increased urinary frequency for two months. Physical examination was significant for pallor and hepatosplenomegaly. Laboratory work-up was remarkable for pancytopenia (hemoglobin-10 g/dL, platelets-86 k/uL, leukocyte count-3.6 k/uL). A bone marrow biopsy was done for evaluation of pancytopenia, which showed infiltration with metastatic adenocarcinoma that was positive for prostate specific antigen (PSA). Following this, a digital rectal examination showed an enlarged, nodular prostate and serum PSA levels were found to be elevated (38 ng/mL). A transrectal prostate biopsy was done which confirmed prostate adenocarcinoma. The patient was started on androgen deprivation therapy (ADT) and discharged home.

Two weeks after the biopsy, patient presented to the clinic with a fever of 102°F. Physical examination was unchanged from recent admission. Laboratory work-up redemonstrated pancytopenia. Blood cultures grew Streptococcus galloluticus subsp. pasteurianus. As part of further Group D Streptococcus workup, a 2-dimensional trans-thoracic echocardiogram was done that showed aortic vegetation with cusp perforation. CT scan of the abdomen showed a 3x5cm hepatic flexure mass which was biopsied via colonoscopy. The pathology from the biopsy showed tubular adenoma. Since starting ADT, PSA levels have normalized (<0.01 ng/mL) and plans for concomitant resection of colonic mass and repair of aortic valve are currently under way.

S. bovis bacteremia is usually associated with colorectal cancer. However, it is also related with various non-malignant and extra-colonic pathologies, such as neoplastic polyps, inflammatory bowel disease, IE and discitis/osteomyelitis. Of these, large colon polyps and adenomas bear considerable significance because of their malignant potential. S. bovis may further enhance oncogenic transformation in these lesions by promoting a protracted inflammatory state. Moreover, as S. bovis accounts for normal microbiota in up to 16% of the population, it is likely that invasive procedures such as transrectal prostate biopsy lead to S. bovis bacteremia by
hematogenous seeding of gastrointestinal flora, thereby presenting an avenue for further exploration. This report illustrates the association of S. bovis bacteremia with benign diagnoses such as large polyps and transrectal biopsies, in addition to more vicious etiologies like colon cancer, that necessitate vigilant observation.
Invasive Pneumococcal Endocarditis with Psoas Abscess Masquerading as Sciatica

Abstract:
Pneumococcal Endocarditis is a rare disease process accounting for less than 3% of all native valve endocarditis cases. This infection is highly invasive with a very high morbidity and mortality rate. Most often the infection starts with lung invasion, later disseminating to various organs including the heart.

We present the case of a 66-year-old woman who presented with a four-day history of low back pain radiating to her buttocks bilaterally. She was afebrile, with sinus tachycardia (103 bpm) and a BP of 145/82 mm Hg at admission. Physical examination was unremarkable except for poor dentition, lower para-spinal tenderness, positive straight leg raise bilaterally. CT abdomen revealed left lower lobe lung consolidation concerning for pneumonia and no concerning spinal findings.

During the admission, the patient spiked a fever to 102F with blood culture growing Streptococcal pneumoniae. Patient also had a new holosystolic mitral murmur two days after admission. TTE confirmed Infective Endocarditis. TEE revealed tricuspid and mitral valve vegetations and mitral valve perforation. CT Surgery was consulted for surgical intervention and patient was transferred to the Cardiac Care Unit for close monitoring. Further lumbar imaging also showed iliopectineus and epidural abscess. Patient received antibiotic therapy in the interim to surgery. Patient underwent surgical intervention with unfortunate complications of massive bleeding and hypotension. She was transferred to the Surgical ICU and passed away one day later.

The risk for invasive pneumococcal disease varies among different ethnic groups. African-American ethnicity has been identified as an independent risk factor for invasive pneumococcal disease. The likely disease trajectory in our patient was a primary lung infection which seeded into the bloodstream resulting in Pneumococcal Endocarditis. This was further complicated by septic emboli to the lumbar spine and musculature resulting in epidural and psoas abscesses. The patient did not receive Pneumococcal vaccination which could potentially have limited the seriousness of her course. Her initial back pain could potentially have been mis-diagnosed as Sciatica and the more perilous diagnosis could have been missed.

Pneumococcal Infective Endocarditis simultaneously affecting the mitral and tricuspid valve is a very rare phenomenon. The risk is higher for African American Ethnicity and in patients above
age 65. We would like to highlight the need for Pneumococcal vaccination when indicated as Pneumococcal Endocarditis is a rare but dangerous entity. In addition, we would like to emphasize the consideration for Psoas abscess and other complications when working up the problem of back pain.
A Curious Case of Small Bowel Obstruction

Abstract:
Patients with small bowel obstruction (SBO) typically present with abdominal pain, distension, nausea, vomiting and obstipation. However, some will have an atypical presentation and represent a diagnostic challenge.

A 20 year old female was referred to the medicine service for management of hypochloremic, hypokalemic, metabolic alkalosis. Review of the chart revealed that she was admitted 26 days ago with multiple gunshot wounds, and underwent exploratory laparotomy, left nephrectomy, small bowel resection and repair, transverse colon repair, and ventral hernia repair, as well as three orthopedic surgeries. Her early post-operative course was unremarkable; however, two weeks post-operatively, she was reported to have episodes of self-induced vomiting. Physical examination consistently revealed a soft, non-distended abdomen with normoactive bowel sounds. Serum chemistry initially showed mild metabolic alkalosis, and abdominal x-ray showed non-specific bowel gas patterns in three instances. With passage of flatus and bowel movement, the patient was started on clear liquids then regular diet as tolerated. She was diagnosed with acute adjustment disorder with features of depression, and mirtazapine, prazosin and as needed lorazepam was initiated by psychiatrist.

Review of the patient’s current serum chemistry revealed sodium 134 mEq/L; chloride 83 mEq/L, potassium 2.6 mEq/L and bicarbonate 41 mEq/L. The cause of this hypochloremic, hypokalemic, metabolic alkalosis was not apparent from the history and physical examination. Additional work-up showed random urine pH 9.0, chloride 22 mEq/L and sodium 110 mEq/L. Severe hypokalemia and contraction alkalosis were thought to have occurred partly secondary to the diuretic phase of acute tubular necrosis, and partly due to vomiting. Aggressive hydration and electrolyte replenishment were initiated but did not result in resolution or improvement of her metabolic alkalosis. Since vomiting was considered the likely cause of her electrolyte/acid base disturbances, abdominal x-ray was repeated which again showed non-specific bowel gas patterns. An abdominal CT scan with contrast was then ordered which showed a massively distended stomach and small bowel loops proximal to the anastomosis. The patient was placed on NPO and gastric decompression was started. Vomiting and the patient’s electrolyte abnormalities resolved in the course of two days.

In the case presented, despite being clinically unapparent, SBO was the cause of the electrolyte abnormalities, and “mild self-induced vomiting” was not psychogenic. Properly identifying risk
factors of SBO even in the atypical clinical picture, would prompt one to perform imaging studies which are usually confirmatory; however, their sensitivity and specificity vary (plain radiograph 50% and 75%; CT 93% and 100%, respectively). When the signs, symptoms and diagnostics become equivocal such as in this case, diagnosis may be delayed; therefore we should have a high index of suspicion of bowel obstruction especially in patients with a history of abdominal surgery.
Title: Wellen's sign seen in patient with Takotsubo Cardiomyopathy

Abstract:
Wellen’s syndrome is an electrocardiographic finding of T wave changes that is associated with critical stenosis of the left anterior descending artery, which has a high likelihood of progressing to an extensive anterior myocardial infarction. Due to these facts, detection and early intervention are extremely important in suspected Wellen’s syndrome. However, less commonly Wellen’s sign is detected on electrocardiogram but does not correlate with the critical stenosis of the LAD. We report a patient with Wellen’s sign not correlating with critical stenosis of LAD but instead Takotsubo Cardiomyopathy: 87 year old lady presented to the ED with chest pain and shortness of breath, negative cardiac biomarkers, biphasic T waves in V1-V6 and 50% stenosis of LAD. Transthoracic echo and cardiac catheterization demonstrated severe apical hypokinesis consistent with diagnosis of Takotsubo Cardiomyopathy. Although, the knowledge and high clinical suspicion for Wellen’s syndrome is key in avoiding catastrophic situations, it is also important to keep in mind other causes for Wellen’s sign on electrocardiogram.
Abstract:
Introduction: Tarlov cysts (TC) represent a rare form of meningeal cysts. These cysts are abnormal fluid collections in the space between the endoneurium and perineurium of spinal nerve roots, they are usually incidentally found on MRI images of the spine; they are generally asymptomatic. However, when symptomatic they can cause further debilitating symptoms such as weakness, numbness, and urinary and fecal incontinence. Management of these cysts has no consensus. Case: A 35-year-old female with no significant past medical history presented complaining of lower back pain radiating down the right leg. Her initial pain episode occurred 1 year before that self resolved, she then had a recurrence of the pain 9 months later which gradually worsened and persisted thereafter. She described it as a burning sensation in the right leg and foot, exacerbated by standing, bending, or sitting for prolonged periods of time and alleviated by lying down. She had been prescribed oral methylprednisolone, tramadol, and NSAIDs with partial relief of her pain. On examination, her body habitus and joint flexibility was normal. There was focal tenderness overlying the right L5-S1 area. Lumbar spine range of motion was painful with limited flexion. Supine straight leg raise testing was positive on the right side. Physical therapy was instituted with no improvement. At that time, the patient was referred to neurosurgery and Contrast-induced MRI of the lumbosacral spine was performed which revealed multiple TC causing nerve root compression. Workup for connective tissue disorders was performed; this included ESR, CRP, ANA, and RF, which were all within normal limits. She underwent conservative epidural spinal injection of the right L5-S1 space, the injection provided the patient with temporary pain relief. Hence, she is currently seeking a second neurosurgical opinion and continues to suffer from pain. Discussion: Perineural cysts are abnormal collections of fluid that arise from the spinal dorsal nerve root membrane. These cysts were first described by neurosurgeon Isadore M. Tarlov in 1938. TC are most commonly found incidentally on imaging studies in asymptomatic patients; this has led to a false belief that they are invariably asymptomatic. When symptomatic, these cysts cause lower-back pain, pseudo-claudication, and may potentially cause spinal-root compression symptoms. There is no consensus regarding the treatment of symptomatic TC; most approaches are directed at symptom control, whereas surgical resection is preserved for pain refractory to conservative management. Surgical treatment has had a variable, and sometimes disappointing, outcome. Our patient presents a case of symptomatic TC in a young female with no associated medical conditions; her pain had rapid progression with significant impact on her quality of life. Hence, it is safe to conclude that a wider recognition of this medical condition with further investigations to generate treatment consensus is warranted.
Abstract: Nephrotic syndrome is a common subset of glomerular disorders. Clinically it manifests as heavy proteinuria >3 grams daily, edema, and hypoalbuminemia. Of the primary nephrotic diseases, minimal change disease is predominantly described in the pediatric population and rarely in adults. Although the major feature of this disorder is edema, there are significant morbidities associated with its heavy proteinuria.

We present a case of a 60 year-old female with a past medical history of multiple sclerosis and hypertension, who presented with generalized weakness, anuria and shortness of breath over one week. Exam revealed anasarca with diffuse edema of all four extremities, and a distended abdomen. Venous dopplers revealed an acute deep vein thrombosis in the left femoral and common femoral veins and a V/Q scan showed high suspicion for pulmonary embolism. Urinalysis demonstrated 3+ protein and an unusually high urine protein creatinine ratio (UPCR) of 31.63. Her blood work was remarkable for an albumin level of 0.4 g/dl and a creatinine of 1.5 mg/dl. Review of past medical history revealed that she had initially been treated at an outside hospital 6 months prior for acute renal failure, hemolytic anemia and proteinuria. A renal biopsy revealed thrombotic microangiopathy (TMA) likely secondary to chronic interferon therapy for multiple sclerosis. Of note, light microscopy and immunofluorescence were unremarkable. Electron microscopy demonstrated diffuse podocyte effacement of glomerular membranes, without subendothelial and subepithelial deposits. Patient’s proteinuria persisted after interferon was discontinued. The hemolysis, anemia, and acute renal disease resolved. Patient was started on high dose steroids to treat a possible second primary process. She continued to have elevated UPCR and she was switched to tacrolimus which led to decrease of UPCR below 10. Patient discontinued tacrolimus due to fatigue 4-5 weeks prior to her second admission to our hospital. On this admission, the nephrology team evaluated the patient and the pathologists reviewed the prior biopsy. She was treated with anticoagulation, diuretics, albumin replacement, and angiotensin inhibitors. On discharge the patient was started on cyclosporine. As an outpatient, her UPCR ratio has decreased to 4, but this is in the context of increased creatinine to 1.9 mg/dl. She remains edematous with low albumin of 1.1 g/dl.

This case is an unusual presentation of severe nephrotic syndrome with podocytopathy by renal biopsy. The severity of the proteinuria and the discontinuation of intervention leading to venous thromboembolism highlight the multiple potential morbidities of heavy proteinuria, including hypercoagulability, hyperlipidemia, and anasarca. The patient’s general prognosis and
likelihood of recovery is low given the degree of proteinuria and suboptimal responsiveness. Our case emphasizes the need of further research in therapies for aggressive minimal change disease in the adult population.
The missing triad: Diffuse alveolar haemorrhage in granulomatosis with polyangiitis with coexistent rheumatoid arthritis

Authors: Shoeb Mohammed1, Ashraf Abugroun1, Beenish Zulfiqar1
1) MD. Advocate Illinois Masonic Medical Center

Case report:
A 64-year-old Asian female with past medical history of rheumatoid arthritis and granulomatosis with polyangiitis (GPA) was brought to the Emergency Room after she developed massive hemoptysis. She was hospitalized for same reason five years earlier, during which time she had purpuric skin rash, and a progressive decline in renal function. Skin biopsy revealed leukocytoclastic vasculitis and renal biopsy confirmed focal segmental glomerulosclerosis. Her blood test was positive for C-ANCA and PR3 antibodies. She was treated with cyclophosphamide, steroids, and had been on a maintenance dose of Azathioprine for several years before it was stopped two years ago due to stable disease course.

On current admission, physical examination revealed diffuse crackles in both lungs and pulse oximetry showed oxygen saturation of 94% on room air with absence of A-a gradient in arterial blood gas. She was intubated for airway protection. Laboratory work up showed hemoglobin 11.2 gm/dl, WBC 11.1x10^9/l (neutrophils 8.9x10^9/l), serum creatinine 5.06 mg/dl, BUN 46 mg/dl, INR 1, PTT 25, lactic acid 2.5, C-reactive protein 2.1, ESR 56, and procalcitonin 0.67 ng/mL. Urinalysis revealed protein level > 300 mg/dl, but had no RBCs, casts or dysmorphic cells. She had negative serology for myeloperoxidase and serine protease. CT scan of the chest showed patchy ground glass opacities throughout both lungs. Bronchoscopy revealed clots and bloody returns on lavage consistent with alveolar hemorrhage. Microbiological analysis of the bronchoalveolar lavage specimens revealed Haemophilus influenza and Pseudomonas aeruginosa. Patient recurrence of hemoptysis was attributed to a flare of granulomatosis with polyangiitis along with concomitant pneumonia. Patient condition drastically improved following a pulse dose of steroids, cyclophosphamide, and antibiotic treatment.

Granulomatosis with polyangiitis is characterized by a granulomatous inflammation of the respiratory tract and necrotizing vasculitis of small to medium sized blood vessels. The disease commonly involves the kidneys in the form of necrotizing glomerulonephritis. PR3-ANCA is found in around 95% of the cases. Although the lung is commonly involved in GPA, diffuse
alveolar hemorrhage is a rare presentation reported in only 5% to 10% of patients. While it is known that GPA has a characteristic triad of hemoptysis, anemia and progressive hypoxemia. Another triad of three distinct pathologies seems to be recognized. Our patient had two episodes of massive hemoptysis caused by diffuse alveolar hemorrhage, each required intubation for airway protection. This is the second case report (Vaishnav, Bhatt, and Desai 2012) where a patient with known GPA with coexisting rheumatoid arthritis presents with DAH. Further studies are needed to study the immune mechanism behind such clinical presentation.
Abstract:
Objective/Background: Diseases do not occur randomly. This study applies a time tested descriptive epidemiology method called “Person - Place –Time” analysis as an initial critical review of underlying patterns of firearm violence admissions at Mount Sinai Hospital, Chicago.

Method: After obtaining IRB approval, data on trauma patients from Mount Sinai Hospital entered to Illinois Department of Health Trauma Registry from December 2003 to July 2016 was used for analysis. A total of 4539 gun-violence related injuries were identified.

Results:
Person: Firearm victim were predominantly (92.73 %) male, with an average age of 25.3 years (SD +/- 9.4 yrs.) with majority of victims being younger (median 23.0 yrs.). Majority of victims identified themselves as Black (69.8 %) and Hispanic race (26.33 %).

Time: Most (33.58 %) firearm injuries occurred in early morning hours between midnight and 6:00 am, followed by late evening (9:00 pm and midnight) (22.85 %) and early evening (6:00 pm to 9:00 PM) (16.72 %). Saturday and Sunday saw the highest incidence (19.67 %) and (18.15 %), followed by Friday (13.33 %) and Thursday (13.26 %). Firearm violence was highest in summer months – July, June August and September- (10.99 %), (10.24 %), (9.14 %) and (9.87 %), respectively. February (5.35 %) and January (6.32 %) saw the lowest level of firearm violence, where February figure was almost half of summer statistics.

Place: Most firearm violence took over at zip codes 60623, 60624 and 60608, (14.5 %), (11.79 %) and (9.01 %), respectively. Further analysis shows that most (49.7 %) violence took place in the same residential zip code of victims. As to the exact location of violence, an overwhelming majority (70.9 %) of shootings were reported to have occurred on a street or highway. Shooting at home (6.43 %) and recreational and sport parks (0.46 %) only accounted for the minority of cases.

Discussion:

Compared to national and Chicago data, the average age of victims at Mount Sinai hospital is slightly older, literature review suggests a younger age might be indicative a thrill-seeking
motivation as opposed to intentional and utilitarian intent seen in older ages. A difference between a local hospital data and citywide statistics warrants further examination. The PPT analysis highlights potential areas of priority for future intervention or research.
An Unusual Cause of SIADH

Abstract:
Endemic mycoses are commonly found in Northern Illinois. Especially in the immunosuppressed, it is imperative to keep these fungi on the differential when a patient presents with non-specific symptoms and unexplained fevers. Moreover, it is important to understand that the differential for SIADH is broad, including pulmonary infections/diseases.

A 68-year-old female with history of kidney transplant on immunosuppression was sent to the emergency room after a routine lab draw showed a sodium level of 122 and a potassium of 6.0. Patient was recently discharged after being treated for hyponatremia and pyelonephritis. She completed 14 days of treatment with imipenem for her pyelonephritis and was sent home on a one liter per day fluid restriction as well as salt tablets. She reported compliance with her home regimen on presentation. The patient was completely asymptomatic at the time of presentation. On further evaluation, she was found to have a euolemic hypo-osmolar hyponatremia, felt to be due to SIADH. Work up for other causes of hyponatremia (adrenal insufficiency, hypothyroidism, adverse drug effect) was negative. During her hospital course, the patient was noted to have nightly febrile episodes up to 39.3. The patient was asymptomatic during each of these fever spikes, and infectious workup was initially negative. Based on transplant nephrology recommendations, a CT of the chest was ordered to search for the etiology of SIADH. CT chest revealed diffuse centrilobular lung nodules not previously seen on any imaging. Tuberculosis was ruled out with serial AFBs, and pulmonology was consulted. Bronchoscopy with BAL was performed, but no appropriate tissue could be found to biopsy. Subsequent BAL analysis showed many WBCs but was negative for any specific etiology. Urine histoplasmosis and blastomycosis antigens were sent, and both came back positive. Patient was discharged on a 12 week course of itraconazole with close ID outpatient follow up.

This case illustrates the underlying risks of immunosuppression, including the myriad of potential complications and how they can present. In this patient, a fungal pulmonary infection was most likely driving the SIADH, even though she was otherwise asymptomatic. Had it not been for the daily monitoring of temperature that occurs in the hospital, her infection may have gone undiagnosed. As this case shows, endemic mycoses can often be indolent in their course and presentation.
Iatrogenic retained intracoronary foreign body necessitating emergent bypass surgery

Abstract:
We report a 80 year old Caucasian male with an extensive cardiac history including prior myocardial infarction requiring coronary bypass grafting and multiple sessions of percutaneous coronary angioplasty, who was admitted after cardiac catheterization. Cardiac stress testing performed prior to admission for crescendo pattern of angina was strongly positive. Coronary angiography showed restenosis of the saphenous vein graft to the left anterior descending artery as well as restenosis of the stent in the right main artery with dense calcification around the lesion. After failing to dilate the stenotic lesion in the right coronary, rotational atherectomy was attempted with a Rotablator device. Although the device was successfully negotiated through the stenosis, it could not then be withdrawn, the burr having lodged within the lesion. After several unsuccessful attempts to retrieve the device, it was decided to convert the procedure to redo open bypass grafting. Despite direct intra-operative traction on the retained rotablator it could not be dislodged, however the visible portion of wire fractured and was removed along with the adjacent section of the device. Bypass grafts were then placed to the left anterior descending and right posterior descending arteries. The post-operative period was complicated by multiple episodes of ventricular tachycardia and fibrillation ultimately necessitating placement of an implanted cardioverter/defibrillator and initiation of antiarrhythmic therapy. The patient was later discharged to acute rehabilitation where he made a satisfactory recovery.

Coronary artery disease remains the leading cause of death in America and percutaneous intervention is an important tool in the armamentarium of cardiologists. Densely calcific disease however poses special challenges as were demonstrated in this instance. The rotablator device was designed specifically for calcified stenotic disease and was its use was entirely justified in our patient; nevertheless the patient suffered significant morbidity in the aftermath of its use. Due caution should be exercised when attempting percutaneous interventions in highly calcified coronary artery disease and the patient should be made aware of potential need for bypass grafting should such interventions fail. Rotational atherectomy should be reserved for management of refractory angina in patients who are otherwise not candidates for surgery.
Title: Hemiplegic migraine mimicking stroke triggered by acute pre-eclampsia

Abstract:
Introduction:
Hemiplegic migraine (HM) is a rare type of migraine with aura associated with weakness. HM commonly presents with variable neurological deficits, including visual, sensory, or motor disturbances. It is linked to various genetic defects that are either inherited as AD (familial HM) or acquired (sporadic type). Here, we describe a sporadic type of hemiplegic migraine in a 44-year-old female, triggered following acute pre-eclampsia.

Case:
A 44-year-old female with past medical history of migraine headaches, hyperactive parathyroid adenoma treated with resection presented to the emergency room with severe generalized headache for one week, associated with left-sided weakness for the last day. Symptoms were preceded by a head injury following a fall from a height. Her headache was worsened by noise or light, and relieved partially with analgesics. Neurological exam was remarkable for flattening of the nasolabial fold on the left side, grade 4/5 weakness of the left limbs. Patient has had repeated admissions for headache associated with left-sided weakness in 2012, 2014, and 2015. Extensive workup included normal ANA, CRP, HIV, TSH, protein C, PT/INR, fibrinogen level, lupus anticoagulant, and mixing studies. She also had repeated brain Computed Tomography (CT), Computed Tomography Angiography (CTA), Magnetic Resonance Imaging (MRI), Magnetic Resonance Angiography (MRA), and Magnetic Resonance Venography (MRV) as part of stroke workup with each admission. Tests were significant for only stable 1.6 mm aneurysm at the at the junction of the left anterior cerebral artery (ACA) and the anterior communicating artery (ACOM) seen on CTA, as well as fixed 4 mm T2 high signal in the right parietal subcortical white matter, suggestive of focal demyelination seen in different MRIs. Patients repeated weakness was finally diagnosed as hemiplegic migraine.

Hemiplegic migraine being a diagnosis of exclusion, this patient fulfilled diagnostic criteria of the illness as she had fully recurrent reversible unilateral weakness with headache. Each episode lasted for several hours and resolved in less than 72 hours. Extensive workup ruled out all other potential causes of weakness. The patient has a characteristic focal area of increased intensity on MRI. Various imaging findings were reported in prior studies in association with HM. Little is known on the pathogenesis of hemiplegic migraine. Having this patient’s unique presentation with onset of HM triggered by pre-eclampsia and presence of focal brain area suggestive of demyelination on imaging might contribute to greater understanding of the pathogenesis of the condition. This case highlights the complexity and diagnostic challenge of
the disease. It also sheds light on the concept of clinical waste as expensive sets of investigations were requested with limited clinical outcome.
Abstract:
Burning mouth syndrome (BMS) is characterized by oral mucosal burning sensations, with normal clinical and laboratory results. Menstrual synchrony of migraines and epilepsy have been discussed; however, catamenial BMS has not heretofore been described.
A 29 year old right-handed female exhibited intermittent BMS symptoms, one month after suffering from a right frontal infarction. She describes the BMS pain as a burning sensation, localized on the bilateral and anterior aspects of her tongue. It lasts for 4 days, starting 3 days prior to her menses, and occurs twice a month. She is unable to correlate any patterns or triggers that may cause to exacerbate her BMS. She denies any taste disturbances, hot-flashes, night sweats, and perspiration. Abnormalities during neurological examination were noted. Cranial nerves (CN) III, IV, and VI showed bilateral lateral first-degree end-gaze unsustained nystagmus. CN IX and X showed decreased bilateral gag reflex. A right pronator drift with a right abductor digiti minimi sign was seen in the motor examination. The cerebellar examination was positive for bilateral dysmetria during the Finger-To-Nose examination, and exhibited Holmes rebound phenomena, right more than left. Sensory examination showed decreased light touch in the lower extremities, right more than left. Hoffman reflex was bilaterally positive. Mental status examinations demonstrated poor similarity interpretation and calculation ability. Her neuropsychiatric testing was normal, and included the Go-No-Go and Animal Fluency Testing. MRI of brain with and without gadolinium indicated Arnold-Chiari malformation with the cerebellar tonsils descending 8mm below the foramen magnum.
This case illustrates BMS to be catamenial. Estrogen and progesterone both have nociceptive properties. Premenstrual drop or reduction of estrogen and progesterone may act to disinhibit pain [Vincent 2008], with pain modulation being more effective during the ovulatory phase (high estrogen and low progesterone) [Rezaii 2012]. Depression in the presence of Late Luteal Phase Dysphoric Disorder may function to exacerbate the perception of underlying pain throughout the body, including the mouth and tongue. Decrease in estrogen and progesterone levels may also alter salivary output and composition. This may allow baseline reduction of proprioceptive input on the tongue, thus acting through Melzack and Wall’s Gate Control Theory of Pain to disinhibit small C fibers, which is perceived as burning pain [Melzack 1978]. Along with menses, olfactory ability drops, and food preferences are often reported to change [Keller 2013]. A decrease in estrogen and progesterone can also enhance trigeminal nerve sensitivity [Martin 2007], which exacerbates pain. This may indirectly influence or be associated with her BMS. Such observations justifies a trial of hormonal agents for therapy of BMS.
Title: Improving Pneumococcal Vaccination Rates in adults ≥ 65 years of age: A Matter of Simple Education

Abstract:

Introduction
Pneumococcal infections are important source of morbidity and mortality, with the highest rates of mortality occurring among individuals who are ≥65 years of age. Despite the clear updated guidelines for pneumococcal vaccination, the nationwide adherence rate is still below the goal set by the Office of Disease Prevention and Health Promotion (ODPHP). No data on current pneumococcal vaccination rates was available at the Arvey Community Health Center located at Weiss Memorial Hospital in Chicago, which is a resident-run outpatient clinic.

Methods

A retrospective chart review using the electronic medical records was performed from October to November of 2015 to evaluate the current pneumococcal vaccination rate. All enrolled patients were ≥65 years of age with at least 1 visit documented. Then we implemented 3 improvement strategies to increase vaccination rates based on education and reminders. The first intervention was directed to the internal medicine residents by sending e-mails about updated vaccination guidelines, and by providing a single lecture included in the core curriculum about pneumococcal vaccination. The second intervention was directed to the patients, using handouts and personalized education. The final intervention was directed to the medical assistants by educating about the vaccination schedule, proper documentation and screening. Data was gathered weekly as successful vaccination or as documented vaccination refusal from September to November 2016. Updated run charts were visually available in our clinic.

Results

785 patients attended the clinic during the period from October to December 2015. 208 (26%) patients were ≥65 years of age and only 40% of this group had a documented vaccine administration or refusal. 54% of these patients were actually vaccinated (22% of the patients ≥ 65 years of age). Our primary outcome was the improvement in the percentage of patients receiving the vaccination and the documentation of “offered/declined” after the 3 improvement interventions. There was an average of 4 % increase in compliance per week during the first 10 weeks after the interventions. The highest increase of 16.1% per week was
seen after implementing a patient directed handouts in the waiting area. The final compliance rate was calculated 10 weeks after the initial intervention and was 80%.

Conclusions

Educating the healthcare providers had a good impact on the pneumococcal vaccine compliance rates, likely explained by eliminating the confusion due to frequently changing practice guidelines. However, patient education was the paramount of our interventions. With the patient being educated there was a higher chance of discussing his vaccination status during the regular office visit. The work of discussion and documentation even if a patient chose to decline should be considered a success. These initial results are promising and we encourage clinics to educate their patients about the pneumococcal vaccination.
Title: Clinical significance of inflammatory biomarker to determine the severity of hypertriglyceridemia-induced acute pancreatitis

Abstract:
Aim:
To investigated the performance of inflammatory biomarkers like neutrophil-lymphocyte ratio (NLR), Platelet-Lymphocyte ratio (PLR), and red cell distribution width (RDW) for detecting severity of disease in patient with hypertriglyceridemia-induced acute pancreatitis (HTG-AP).

Methods: We retrospectively reviewed 110 patients with HTG-AP and compared the NLR, PLR, and RDW in different severity groups. We performed receiver-operating characteristic (ROC) analysis to identify the optimal cut-off value for NLR to predict severe AP.

Results: The average value for NLR was significantly higher in patients with severe AP than in those with mild/moderately severe AP (14.6 vs. 6.9, p < 0.001) or patients with persistent organ failure (13.2 vs. 6.9, p < 0.001). After dichotomization by the optimal cut-off value of 10 as determined by the ROC curve, the high-NLR group had a significantly longer length of stay (9.1 vs. 6.6 days, p = 0.001), duration of nil per os (4.9 vs. 3.7 days, p = 0.007), and higher rates of complications, including systemic inflammatory response syndrome (81.5% vs. 44.6%, p = 0.001) and persistent acute kidney injury (25.9% vs. 3.6%, p < 0.001).

Conclusion: NLR represents an inexpensive, readily available test with a promising prognostic value in patients with HTG-AP. Among the three inflammatory biomarkers, the NLR has the highest discriminatory capacity for severe HTG-AP, with an optimal cut-off value of 10

KEYWORDS:
Hypertriglyceridemia
Acute pancreatitis
Neutrophil lymphocyte ratio
Organ failure
Title: Evaluation of the Prognostic Value of Platelet to Lymphocyte ratio in patients with Hepatocellular Carcinoma

Abstract:
Introduction:
Hepatocellular carcinoma (HCC) is increasingly common, potentially fatal cancer type globally. Platelet-lymphocyte ratio (PLR) as a biomarker for systemic inflammation has recently been recognized as a valuable prognostic marker in multiple cancer. The aim of the present study was to assess the prognostic value of PLR in HCC patients and determine the optimal cut-off value for risk stratification.

Methods:
We retrospectively analyzed patients with diagnosis of HCC (screened by ICD-9 code, confirmed with radiographic examination and/or biopsy) at a large public hospital during 15 years (Jan 2000 through July 2015). PLR, among other serology laboratory values were collected at diagnosis of HCC. Its association with overall survival were evaluated with Cox proportional hazard model.

Results:
Among 270 patients with HCC, 57 (21.1%) patients died within an average follow-up of 11.9 months. Direct comparison of laboratory values collected at diagnosis of HCC between groups revealed that survivors had significantly lower INR, AST, bilirubin, PLR, and higher albumin. PLR at diagnosis was significantly different between survivors and deceased (128.9 vs 186.7, p=0.003). In multivariate analysis, aspartate transferase (HR 2.257, p<0.01) and PLR (HR 1.768, p=0.004) independently predicted mortality. The optimal cut-off value for PLR was determined to be 220 by receiver-operating characteristics curve, and high PLR group had significantly higher mortality (HR 3.42, p<0.001).

Conclusion:
Recently, the utility of PLR has been extensively explored in various non-cancerous populations, including patients with acute coronary syndrome, immunoglobulin-resistant Kawasaki disease, premature ovarian insufficiency and acute pancreatitis. These associations were attributed to the role of PLR as a systemic inflammation marker. The exact underlying mechanism responsible for the role of PLR in HCC has yet to be elucidated. However, it is postulated to be closely related to the cancer-induced systemic
inflammatory response, which suppress recruitment of immunosuppressive cells such as regulatory T cells, leading to tumor progression and metastasis. Platelet secretes various growth factors which promotes angiogenesis, cell proliferation and metastasis. PLR is a low-cost and convenient tool, which may serve as a useful prognostic marker for HCC. Our results indicated that elevated PLR at diagnosis above 220 predicted poor prognosis in HCC patients.
Title: Autonomic dysfunction secondary to copper deficiency-associated myeloneuropathy caused by sleeve gastrectomy

Abstract:
Copper deficiency-associated myeloneuropathy typically presents as a subacute gait disorder with associated peripheral neuropathy. Although the pathophysiology is not completely understood, acquired copper deficiency as a delayed complication of gastric bypass surgery is thought to be the most common cause. Further investigation into this syndrome is warranted given the limited reports in the literature, potential for permanent neurologic dysfunction and the rise in bariatric procedures in the US. Although typically reported in cases of gastric bypass, here we report a unique manifestation of copper deficiency-associated myeloneuropathy caused by sleeve gastrectomy.

A 51-year-old woman presented to the emergency department for persistent lightheadedness, non-traumatic falls, near-syncopal episodes and burning pain in her lower extremities. Her symptoms began two months after undergoing a sleeve gastrectomy and remained despite physical therapy at a skilled nursing facility. On evaluation, the patient was found to have orthostatic hypotension that was refractory to intravenous fluid boluses. Initial workup was unremarkable including normal TSH, AM cortisol, vitamin B12, methylmalonic acid, homocysteine, ECG and echocardiogram. Given this negative work up and likely presence of malabsorption in the setting of recent gastric sleeve, nutritional deficiencies were pursued as possible etiologies for patient’s neuropathic pain and autonomic dysfunction. Serum copper levels were found to be abnormally low and an electromyogram study confirmed mild, axonal, predominantly sensory length dependent polyneuropathy. Therefore, the orthostatic hypotension and neuropathic pain were thought to be secondary to autonomic and peripheral neuropathy. This would be consistent with acquired copper deficiency in the setting of recent gastric surgery and poor nutritional status. Patient was started on intravenous cupric chloride, fludrocortisone for postural hypotension, gabapentin for peripheral neuropathy and physical therapy for muscular deconditioning. Serum copper levels increased to normal and patient’s symptoms improved allowing for the patient to be discharged with PO copper supplements and outpatient physical therapy. The patient was subsequently hospitalized for similar symptoms of autonomic dysfunction despite adequate copper repletion. Her constellation of symptoms was determined to be due to a combination of poor PO intake and medication non-compliance exacerbating her underlying neuropathy. Patient was restarted on intravenous cupric chloride and fludrocortisone. The fludrocortisone was tapered down and replaced with midodrine with
subsequent improvement in symptoms. She was discharged in stable condition with a walker for ambulation.

Here we present a case of neuropathy and orthostatic hypotension secondary to autonomic dysfunction from copper deficiency myeloneuropathy. Although rare, this case demonstrates the range of neurologic manifestations that can result from acquired copper-deficiency caused by gastric sleeve surgery. Prompt copper supplementation has the potential to treat neuropathic symptoms and avoid permanent deficits. For these reasons, evaluation of copper status is essential in patients who develop symptoms of peripheral neuropathy or autonomic dysfunction following bariatric surgery.
Abstract:
Background: Stroke is a major cause of death and disability in the United States with a disproportionate impact on underserved communities. Establishment of formal certification of primary stroke centers and financial incentives have shown to improve overall care for patients with stroke nationwide. However, it is uncertain if these measures are generalizable to an underserved community, where decreased access to care, lower health literacy, and lack of stroke preparedness could represent an additional challenge.

AIM: To determine baseline characteristics, revascularization rate and overall survival in an underserved population with acute stroke.

Methods: Retrospective chart review of patients with diagnosis of acute stroke at John H. Stroger Hospital in Chicago, between January 1st and December 31st, 2014. Stroke events were clinically diagnosed by certified stroke physicians in the Neurology Division and confirmed by magnetic resonance imaging or computed tomography. All events were independently adjudicated by four investigators. Demographic variables were independently extracted. We present continuous variables as mean±standard deviation and categorical variables as percentages. SPSS version 24 was used for calculations. We used Kaplan Meier method to estimate overall survival (OS).

Results: A total of 451 patients were included, with mean±SD age of 59.1±12.2 years and predominantly male (57.4%). The majority with ischemic stroke (94.5%), out of which only 10 (2.4%) met criteria for tissue plasminogen activator (tPA) therapy. By race, predominantly African American (58.8%) followed by Hispanic (21.7%), Caucasian (10.9%), and Asian (5.5%). Patients had a history of tobacco use (46.1%), hypertension (73.4%), diabetes (36.8%), recurrent stroke (22%), and atrial fibrillation (AFIB) (3.8%). Also, new AFIB was diagnosed in 2.2%.

The mean±SD follow up was 21.9±16.5 months, and a total of 30 patients died (6.7%). Patients with hemorrhagic stroke had a worse OS when compared to those with ischemic stroke (mean 32.8 months vs 39.7 months, p<0.01) (Fig.1). We did not find differences in OS among the different ethnic groups (p>0.05).
Conclusion: Despite similar baseline characteristics to the Stroke Statistic of 2016 reported by the American Heart Association, in our cohort, only 2.4% of patients met criteria for revascularization therapy, compared to the 3.4% to 5.2% reported nationwide. Our findings prompt the question of whether targeted interventions are needed to ameliorate health disparities, such as health insurance coverage, access to and use of care, in an underserved population.
Primary pulmonary salivary gland tumors are exceedingly rare and comprise less than 1% of all lung tumors. Mucoepidermoid tumor is one of the subgroups that is under recognized since most cases are grouped under more common histological varieties. Presentation, treatment and prognosis of these tumors vastly differ and hence it is important to recognize this histopathological entity.

An 80 y/o gentleman with a 30 pack year smoking history, quit 30 years ago, presented to pulmonary clinic with complaints of cough and R sided chest pain for a month that failed to resolve with antibiotics. CT chest showed partial obstruction of the segmental RLL bronchus and atelectasis. Bronchoscopy revealed a whitish hard glistening endobronchial mass occluding the airway and it was biopsied. Initial cytology showed NSCLC. Further imaging showed no evidence of metastases and patient underwent right lower lobectomy with lymph node dissection. Histopathology revealed low grade primary pulmonary MEC measuring 2.2 cm and negative margins. None of the lymph nodes were involved.

Primary mucoepidermoid carcinoma (MEC) is a very rare subgroup of NSCLC that has distinct morphological and histological and pathophysiological characteristics, similar to that of salivary gland tumors. They have weak association with smoking and are commonly seen in patients who were remote or never smokers as our patient. These tumors generally present in younger age group as one case series reported a mean age at presentation of around 35 years. Incidence is equal among men and women. Since these cancers arise from the bronchial epithelium, post obstruction pneumonia, atelectasis or collapse are frequent presenting features and therefore often warrant further work up is necessary in such situations. Histologically, mucoid, epidermoid and intermediate cells are seen with intercellular bridges, but keratinization is absent in contrast to squamous cell carcinoma. Immunohistochemistry can be positive for p63, p40, CK7, CK5/6 but negative for CK20. A case series of primary pulmonary MEC revealed MAML2 overexpression as a common finding.

Another unique feature of this sub class is the histological grade of the tumor which determines prognosis. Low grade MEC that are resected have excellent 5 year survival rates approaching 95%. On the other hand higher grade MECs have poor prognosis. In advanced or metastatic tumors, role of tyrosine-kinase inhibitors targeting the MAML2 over expression are being studied.

Our case highlights the importance of recognizing this rare entity as a unique subclass to avoid mislabeling the diagnosis. It also demonstrates the need for prompt evaluation of solitary
endobronchial lesions even in non/remote smokers. Our case adds to the published literature as our patient is much older than the usual age group in which these tumors have been reported thus far.
Abstract:
Diffuse alveolar hemorrhage (DAH) is characterized by bleeding into the alveolar spaces of the lungs, mechanism being disruption of alveolar-capillary basement membrane. Hemoptysis is the common presenting symptom. DAH associated with cocaine use is reported commonly whereas DAH with cannabis use is uncommon.

Here we present a case of a 31 year old gentleman with no past medical history who presented with hemoptysis for 1 week duration. He denied any other symptoms. Social history was significant for cannabis smoking but denied cigarette smoking or cocaine use. Physical examination including vitals were normal. Complete blood count, comprehensive metabolic panel and coagulation profile were normal. Urine toxicology was positive for cannabinoids. Chest X-Ray was unremarkable. Chest CT scan showed wedge shaped pneumonic type infiltrate within anterior segment of left upper lobe extending into left hilum. Infectious workup including human immunodeficiency virus, influenza A and B, sputum gram stain and culture, quantiferon and acid fast bacilli (AFB) smear were negative. Vasculitis workup consisting of antinuclear antibody, rheumatoid factor, anti glomerular basement membrane antibody, anti neutrophil cytoplasmic antibody was negative. Bronchoscopy revealed a pattern of DAH. Bronchoalveolar lavage showed reactive bronchial cells, foamy and pigmented macrophages, mixed acute and chronic inflammatory cells. AFB smear and culture, viral cultures and fungal stain and cultures from bronchoalveolar lavage were negative. He was started on intravenous methylprednisolone for cannabis induced DAH with good clinical response. He was discharged on tapered dose of corticosteroids. He was readmitted 2 weeks later with hemoptysis after he smoked cannabis again. Corticosteroids was restarted with resolution of symptoms.

Our case report aims to draw clinicians’ attention and add to the increasing literature about cannabis associated DAH. With legalisation of and increasing use of cannabinoids for medical and recreational purposes, physicians should be aware of and be able to educate our patients regarding the possibility of DAH- potentially life threatening condition with cannabinoids use along with other effects in our body. Our patient responded well to corticosteroids but we do not have enough studies to guide us in the management of cannabis associated DAH. We certainly need more studies to evaluate the role of corticosteroids in the management of cannabis associated DAH.
Abstract:
Paraneoplastic syndromes occur at sites distant from primary tumor and are seen in 20% of cancer patients. Their timely recognition substantially affects prognosis. Our goal with this case is to highlight the importance of follow-up after cancer diagnosis, along with emphasizing nonspecific presentation, and rapid progression of Paraneoplastic Cerebellar Degeneration (PCD).

A 62-year-old Caucasian female with history of uncontrolled hypertension and uterine carcinoma presented with slurred speech. She was diagnosed one year prior with uterine papillary serous carcinoma (UPSC) with negative nodal disease but positive pelvic floor washing cytology, stage IA Grade 3 status post total abdominal hysterectomy and bilateral salpingo-oophorectomy. She was offered adjuvant chemotherapy but had declined. She presented to the hospital with sinus congestion and dysarthria. She denied difficulty formulating thoughts, ophthalmoplegia or focal neurological deficits. Stroke workup was unremarkable and she was discharged home. She returned four days later for non-resolving dysarthria. She was evaluated for neuromuscular junction disease and tested for ACh-R Ab, antimuscarinic Ab, V-gated calcium channel Ab and paraneoplastic panel. She was discharged before autoimmune workup resulted and was recommended to follow-up in the movement disorder clinic. Three weeks later, she presented with ataxia. Repeat MRI brain was unremarkable. Prior workup revealed anti-Yo and anti-Purkinje antibodies and the diagnosis of PCD was confirmed. Staging revealed a large left pulmonary artery embolus along with a 3.5 cm right paratracheal mass. She underwent bronchoscopy with endobronchial ultrasound with biopsy of mediastinal lymph node. She was started on IVIG and prednisone. Pathological evaluation revealed metastatic high-grade serous carcinoma. The tumor was positive for CK CAM 5.2, CK7, PAX–8, CA–125 and p53 compatible with known primary endometrial carcinoma.

UPSC makes up 10% of all uterine cancers. It is highly malignant even without myometrial invasion and accounts for 50% of relapses or death from endometrial cancers. Therefore long-term follow-up and adjuvant chemotherapy is recommended to avoid late metastases. Paraneoplastic syndromes occur in <1% of cancer patients and are made up of several different entities. PCD is a tumor associated autoimmunity against cerebellar antigens. Anti-Yo antibody accounts for 50% of associated antibodies in PCD and have a very low prevalence. 90-98% of patients with this antibody have underlying breast or gynecological malignancy. PCD is associated with poor neurological recovery with <10% regaining function, the majority of which
become bed-bound. Standard therapy involves treatment of malignancy and suppression of immunologic response. Chemotherapy is the treatment of choice. High-dose steroids, intravenous immunoglobulin or plasma exchange should be initiated within the first month of symptom onset to improve survival outcome, though studies demonstrate only short-term relief.
Abstract:

Introduction:
Most Internal Medicine Residency Programs provide exposure to Hospitalist Medicine through a conventional “General Medicine” rotation in their curricula. General medicine rotations usually consist of interns, senior residents, and an attending physician with or without medical students. Management and medical decision making on these rotations usually made after discussions amongst the team members. We identified a need for a model that closely reflects the rigors of Hospitalist Medicine, including direct patient care, scheduling, involvement in quality improvement and patient flow. There is a paucity of data on this topic. We designed our study to have objective data on resident perspectives on conventional versus the hospitalist style rotation.

Methods:

We implemented an innovative Hospitalist Medicine rotation for Internal Medicine residents at a tertiary care hospital in the academic year 2016-2017. During this rotation, each team consists of two senior residents (either PGY2 or PGY3) and an attending physician. Each senior resident works a day shift in the hospital for 7 days in a row followed by 7 days working on Quality improvement projects, two half day clinics and time-off. This was compared to the traditional General Medicine rotation (GMF) where each team consist of two interns, a managing senior resident, an administrative senior resident, medical students and an attending physician. Compliance with ACGME duty-hours restrictions was ensured on both rotations. Surveys were sent to all residents, through Survey Monkey, who completed a GMF rotation as seniors as well as all residents who completed both GMF and Hospitalist Medicine. Surveys were administered anonymously and participation was voluntary. Questions in both surveys queried perception of autonomy, involvement with patient’s families, time management, clinical teaching during rounds, discharge planning and the level of satisfaction with the rotation in general. Responses were on a scale from 1 (not comfortable or satisfied at all) to 10 (extremely comfortable or satisfied). A T test with alpha 5% was used to evaluate for statistical significance of the results comparing hospitalist medicine Rotation and GMF rotation.

Results:

31 residents out of 33 completed the GMF survey (a response rate of 93.9%) and 23 residents out of 27 completed the Hospitalist Medicine survey (a response rate of 85.1%). Based on the survey’s results, the residents felt a statistically significant difference in their confidence with time management during the Hospitalist Medicine rotation (p <0.05). They also reported being
more satisfied with teaching during rounds and interaction with patient’s families and more confident with discharge planning while on the Hospitalist Medicine rotation as compared to GMF rotation (p<0.05). The level of autonomy was not statistically significant between the two rotations (p 0.145) Residents’ overall satisfaction for the Hospitalist Medicine rotation was higher than GMF(p <0.05).
Introduction
Hemiplegic Migraine is a rare condition (prev .01%) characterized by migraine attacks with motor weakness during the aura phase and can be sporadic or familial. Due to its rarity and mimicking an acute CVA/TIA can be initially a challenge in obtaining proper diagnosis.

Case Description
46 year old Caucasian male presented with left upper extremity weakness with subsequent left facial droop while working as a housekeeper. Additionally, the patient stated of having bifrontal headache with blurry vision. Patient with past medical history of hypertension and intermittent headaches, but denied history of migraine headaches or cerebrovascular accident. Patient with family history of hypertension, but denied cerebrovascular accident. Neurological exam revealed cranial nerve II-XII intact except left sided facial droop (CN 7), LUE 0/5, remaining extremities 5/5, sensation intact, cerebellar function intact. Electrolytes were unremarkable. CT head without contrast (stroke protocol) to rule out hemorrhage was negative for acute findings. Patient was initiated on tPA as treatment of acute CVA and transferred to tertiary care stroke center. Subsequently, the patient had a negative MRI of the brain. Patient’s symptoms resolved in about 8 hours including his headache. Upon resolution of symptoms and the presentation with negative imaging the patient was diagnosed with Complex Migraine (Hemiplegic Migraine). Patient was discharged home with outpatient follow up with neurology.

Discussion
This case illustrates the importance of recognizing uncommon presentation (motor auras) of a common condition (migraine) which can be easily confused with another condition (CVA). Recognition of this rare presentation is essential in providing appropriate treatment, potential prevention of serious harmful treatment associated with misdiagnosis, and future prevention.
Abstract:
Adult intussusception accounts for 5% of all intussusception cases and 1-5% of bowel obstructions. About 8-20% of adult intussusception cases are idiopathic occurring in the small intestine. The remaining are attributed to a lead point lesion such as post-surgical adhesions, lipomas, inflammatory bowel disease or metastatic neoplasm.

A 46-year old male presented to the emergency room with one-day history of severe stabbing left lower abdominal pain and rectal bleeding. He woke up with an urge to defecate and found blood "trickling" down his legs. He had abdominal pain, tenesmus, ten episodes of bloody diarrhea, and one episode of syncope. He had a history of AIDS on HAART, anal condyloma with high grade dysplasia status post excision, hypothyroidism, morbid obesity status post gastric bypass, and a polyp removed two years ago due to a palpated mass found on DRE. On admission, physical exam revealed bradycardia, a soft, non-distended abdomen, normoactive bowel sounds, and suprapubic and left lower quadrant abdominal tenderness to palpation. Rectal exam demonstrated dark dried blood at perineum. CT scan of the abdomen on admission demonstrated proctitis, sigmoid colitis, and distention in the sigmoid colon. The distention was attributed to intraluminal blood products from rectal bleeding and was being managed conservatively with intravenous antibiotics. Stool studies were unrevealing. On the fifth hospitalization day, due to persistence of symptoms and pain out of proportion to physical exam, a repeat CT scan of abdomen was performed. A persistent 7.6 x 4.7 cm intraluminal mass was once again visualized in the distal sigmoid colon and rectum and seemed less likely to be intraluminal blood product. Tumor markers were negative. Further diagnostic work up with flexible sigmoidoscopy with colonic and rectal biopsy revealed mucosa with fresh stromal hemorrhage and congestion consistent with sigmoid intussusception into the rectum. He underwent a diagnostic laparoscopy which found redundant sigmoid colon with obvious ischemic changes at the rectosigmoid junction, edematous sigmoid colon, evidence of intermittent volvulus of the sigmoid colon, biopsied obtained were positive for ischemic changes but negative for malignancy.

Idiopathic colo-rectal intussusception is rare hence making the diagnosis challenging in the beginning. Patients can present with nonspecific symptoms such as nausea, vomiting, abdominal pain and bloody stools and duration of symptoms can vary. The triad of abdominal pain, palpable mass and bloody stools is only seen in 10% of all cases. CT abdomen is the most sensitive diagnostic tool and prognosis largely depends on the underlying causative lesion.
Gingival Hyperplasia is an unwanted side effect from certain medications that can create concern and discomfort for patients. Calcium channel blockers; a commonly used antihypertensive with great advantages can also cause side effects. Gingival hyperplasia is one of the least known and expected side effect. Among calcium channel blockers, incidence of gingival hyperplasia for Amlodipine, a third generation calcium channel blocker, is very limited.

We present the case of a 55 year old male who came to the outpatient clinic with several months of slowly increasing gingival hyperplasia and rapid decline in dental health that developed over the two years of using Amlodipine as antihypertensive medication. Patients past medical history include Cerebrovascular accident, hyperlipidemia and hypertension. On examination the patient had poor dentition, and gingival enlargement of both upper and lower gums, without bleeding, discomfort or pain. Amlodipine was discontinued.

Studies have reported that chronic gingival hyperplasia can start after approximately 260 days of amlodipine use. Chronic gingival hyperplasia may lead to infections, bleeding and functional difficulty. The management is still uncertain, but includes cessation of the medication and switching to alternate drug, oral hygiene, and surgical excision of enlarged gingival tissue.
Abstract:
Introduction
Cardiomyopathy in the setting of pheochromocytoma is rare but recognized clinical entity. It should be considered in patients with unexplained cardiac failure along with features of catecholamine excess.
Case
A 49 year old male with a history of coronary artery disease, peripheral artery disease and hypertension was evaluated for worsening heart failure. He was diagnosed with heart failure one year ago after presenting with progressive dyspnea, orthopnea and bilateral leg swelling. Transthoracic echocardiography at that time revealed ejection fraction of 40-50%. He had multiple hospital admissions for worsening dyspnea and orthopnea since last year. He also had episodic diaphoresis, palpitations, fluctuating blood pressure and 40-pound weight loss in addition to sudden onset of cold fingers with color changes and skin pallor. On examination, he appeared cachectic with temporal wasting and BMI of 16.7kg/m2. Pertinent findings on physical exam included: Livedo reticularis and Raynaud’s phenomenon. Cardiopulmonary exam was unremarkable. Workup included: EKG and troponins: Negative for acute ischemia, Chest X-ray: mild interstitial edema and cardiomegaly, serum BNP: 3421pg/ml, TTE: Severe left ventricular dilation and global left ventricular dysfunction with regional variation and apical dyskinesia, LVEF<20%; Cardiac MRI: Severely dilated left ventricle and markedly reduced LVEF with no evidence of infiltrative or inflammatory process. Coronary catheterization was performed that did not reveal any significant coronary stenosis to explain the severity of his heart failure. Thyroid stimulating hormone, dexamethasone suppression test, plasma aldosterone-to-renin ratio, autoimmune screening and HIV testing were unremarkable. 24-hour urine metanephrines were markedly elevated i.e. 10,154mcg/24hr (Normal: <1,300mcg/24hr). Abdominal MRI revealed a well circumscribed 4.5x5.4x4.6cm mass in the right adrenal gland with mixed solid and cystic features and increased T2 signal intensity. A diagnosis of pheochromocytoma-induced cardiomyopathy was made. Treatment was started with phenoxybenzamine and carvedilol. Endocrine surgery was consulted for surgical resection of tumor.
Discussion
Cardiomyopathy in pheochromocytoma occurs in 10% cases, and results from elevated catecholamines leading to beta receptor down-regulation, myocardial stunning and oxidative damage of myocardial cells. Such deleterious effects of high catecholamines are also seen in
tako-tsubo cardiomyopathy and systolic dysfunction due to acute brain injury. Reversible cardiomyopathy with global or focal dysfunction is common in all these conditions. Initial medical therapy of pheochromocytoma consisting of phenoxybenzamine, an irreversible alpha-1 blocker and a non-selective beta blocking agent is critical prior to definitive surgical therapy. This reduces perioperative risk of hypotension and hypertensive crises. Laparoscopic adrenalectomy is preferred over open laparotomy especially with tumor diameter <8 cm and no malignant radiologic features.

Pheochromocytoma induced cardiomyopathy is reversible, and in the presence of an unexplained systolic dysfunction and features of catecholamine excess, a thorough evaluation should be und

### 翻译

细长卡氏肌病和由于急性脑损伤引起的收缩功能障碍。可逆性心肌病与全球或局部功能障碍在所有这些条件中都很常见。

初始的药物治疗包括酚苄明，一种不可逆的α-1阻滞剂和非选择性β阻滞剂，对于决定性手术治疗来说是至关重要的。这可以减少术期低血压和高血压危机的风险。腹腔镜切除术优于开放性腹腔镜，尤其是在肿瘤直径<8 cm且没有恶性放射学特征的情况下。

嗜铬细胞瘤引起的心肌病是可逆的，如果出现未解释的收缩功能障碍和 catecholamine 超量，应该进行彻底的评估。
Abstract:
Introduction:
Ewing’s sarcoma (ES) is the second most common primary bone malignancy in children and adolescents. Fewer than 30% of cases occur in adults over the age of 20. ES can also occur as a primary soft tissue neoplasm in approximately 5% of cases. This is a case of ES in an adult who presented with a large sigmoid mass.

Case Description:
A 36-year-old previously healthy male presented with a three-month history of intermittent abdominal cramping. Computed tomography imaging revealed a large 13 x 10 cm mass-like thickening of the sigmoid colon infiltrating into the adjacent mesentery, as well as multiple hepatic and peritoneal masses. Biopsy of a mesenteric mass revealed sheets of malignant cells with enlarged nuclei and scant cytoplasm. Immunostaining was positive for CD99 and FLI-1. Findings were consistent with small round blue cell tumor. Molecular genetic testing was negative for the fusion transcript EWSR1-FLI1. Patient’s course was complicated by malignant small bowel obstruction (SBO), Eikenella bacteremia, and acute pulmonary embolism. He was initiated on standard chemotherapy with VDC (Vincristine, Doxorubicin, Cyclophosphamide).

Discussion:
ES and Primitive Neuroectodermal Tumor (PNET) are small round blue cell tumors. Both show neuroectodermal differentiation, though ES tends to be poorly differentiated. ES and PNET are viewed as members of the Ewing family of tumors and grouped together for treatment and prognostic factor analysis.

Biopsy is essential for diagnosis, and is made by the classic light microscopy features of primitive-appearing round cells with high nucleus-to-cytoplasmic ratios. Immunostaining profile shows positive CD99, positive FLI-1, and negative WT-1. The cytogenetic translocation t(11;22)(q24;q12) is positive in 88-92% of ES/PNET cases. The most commonly expressed aberrant fusion protein, EWS-FLI1, is present in 90-95% of ES/PNET cases. However, its absence does not exclude the diagnosis. Those with the EWS-FLI1 translocation have an improved prognosis independent of tumor size and location.

There is a relative paucity of literature relating to outcome for adults with this disease. Adults of age 26 and older have inferior survival rates. This may be due to reduced bone marrow...
reserve in older patients limiting the intensity of the chemotherapy regimen. Unsurprisingly, metastatic disease at diagnosis and primary extraosseous tumor are adverse predictors of survival. Our patient has many negative prognostic factors: age, extraosseous tumor, metastatic disease at diagnosis, and cytogenetics. His SBO persisted through cycle one of chemotherapy requiring jejunal tube placement and total parenteral nutrition.

Extraosseous ES arising from the abdominal cavity is extremely rare. Cases of intra-abdominal ES occurring in the omentum, colon, liver, and small intestine have been occasionally described. Our patient shows us that ES/PNET should be included in the differential diagnosis of an adult patient presenting with intra-abdominal masses.
A 40 year-old African-American male diagnosed with cutaneous sarcoidosis following a biopsy for generalized papulosquamous eruption was referred to renal service due to worsening renal function. Over a period of one year, creatinine progressively increased from a baseline of 1.9 mg/dL to as high as 5.8 mg/dL. Urine studies were significant for trace proteinuria on dipstick, pyuria without growth on culture and 0.8-gram proteinuria without hematuria. Serum calcium levels were normal. Systemic evaluation revealed normal spirometry, HRCT of the chest showed axillary and retroperitoneal adenopathy without evidence of pulmonary sarcoid. Ultrasound-guided biopsy of the kidney revealed granulomatous interstitial nephritis (GIN) consistent with sarcoidosis. Oral prednisone 60 mg/day was initiated with improvement in creatinine plateauing at 2.6 mg/dl after three months.

Sarcoidosis is a systemic inflammatory condition that affects multiple organs, commonly involving the lungs, hilar lymph nodes, eyes, and skin. The estimated incidence in the US is 15-20 per 100,000, with African-Americans affected threefold more than White Americans. Extrathoracic involvement is observed in half of the cases, and are associated with intrathoracic involvement in 80-90% case. Kidney disease is seen in about one-third of cases, as evidenced by nephrolithiasis, nephrocalcinosis, and acute interstitial nephritis with or without granuloma formation. Definite diagnosis is made with biopsy demonstrating non-caseating granulomatous interstitial nephritis.

The incidence of renal sarcoidosis in the absence of pulmonary involvement in unknown, but presumed to be very rare. Although seen in 20% of renal sarcoidosis, GIN rarely causes symptomatic renal insufficiency in of itself without hypercalciuria or hypercalcemia. This abnormal calcium homeostasis is caused primarily by high levels of calcitriol produced by macrophages, found mainly in the lung and lymph nodes (hilar), which is absent in this patient. Granuloma formation in renal interstitium can potentially generate calcitriol subsequently increasing serum calcium levels. Among patients with this histopathologic finding, important differentials that must be kept in mind include adverse drug reactions, mycobacterium infections, GPA and histoplasmosis.

Interstitial nephritis due to sarcoidosis is very rare in the absence of pulmonary involvement. This case study shows importance of considering sarcoidosis in the differential diagnosis of patients with AKI and sterile pyuria in the absence of systemic and pulmonary sarcoidosis.
Title: Shwachman-Diamond Syndrome presenting as chronic recurrent abdominal pain in a 36 year old male

Abstract:
Introduction: Schwachman - Diamond syndrome is a rare autosomal recessive disorder presenting with multisystemic abnormalities including chronic neutropenia, neutrophil chemotaxis defects, metaphyseal dysostosis, and multiple organ involvements. We present a case of a 36 year old male with frequent healthcare visits for recurrent abdominal pain which was eventually diagnosed as Shwachman-Diamond syndrome.

Case Description: A 36 year old male presented to the ER for colicky abdominal pain and vomiting which was present for the past 2 days. The patient had a history of multiple emergency room visits in the past with similar complaints with the last admission just a few days prior to this admission. Pain was worsened with oral intake and was of a severe intensity prompting the emergency room visit. He was admitted to general medical floor for further workup, which revealed CT abdomen/pelvis findings consistent with multiple nodules within liver parenchyma, diffuse fat infiltration in pancreas and elevated lipase on lab evaluation. Patient recently had a normal endoscopy and colonoscopy and previous imaging with CT of the abdomen showing atrophic pancreas, leading to diagnosis of recurrent chronic pancreatitis of unknown etiology. Upon further investigation, it was revealed that patient has history of skeletal and dental abnormalities since childhood, along with chronic neutropenia, Type I diabetes mellitus and schizophrenia. Physical examination was consistent with short stature, dental carries, bowing like appearance of limbs (musculoskeletal abnormality). Patient was treated for chronic pancreatitis with resolution of symptoms and eventually discharged with follow up with hematology. Patient had outpatient genetic testing which revealed that the patient was homozygous for the C.258+2T>C pathogenic mutation in the SBDS gene consistent with Schwachman-Diamond syndrome. Patient also had an MRI abdomen done which showed fatty infiltration of the liver producing nodularity and complete fatty replacement of the pancreas. Patient was continued on pancreatic enzymatic replacement along with disease education and genetic counselling with instructions for regular primary care, orthopedic and hematology follow up for closer monitoring.

Discussion: Schwachman-Diamond syndrome is a rare disease with estimated incidence of 1 in 75000. It presents as pancreatitis and is often misdiagnosed until adulthood leading to increased morbidity and healthcare burden. Our case illustrates the need for increased awareness of the disease manifestations in order to diagnose and manage this condition earlier
Title: Hypertriglyceridemia-induced Acute Pancreatitis in a young male presenting with Diabetic Ketoacidosis

Abstract: Hypertriglyceridemia is the third most common cause of acute pancreatitis (AP). Serum triglyceride levels above 1000mg/dl are most frequently implicated. Acute elevations of circulating triglycerides may be observed in diabetic ketoacidosis (DKA). This is attributed to increased lipolysis, and decreased activity of the lipoprotein lipase enzyme found in the capillary endothelial cells of adipose tissue, as a result of insulin deficiency. Occasionally, DKA may present with severe hypertriglyceridemia that leads to AP.

We present the case of a young, obese, diabetic male who presented to the emergency department with mid-abdominal pain, nausea and emesis. He reported no previous history of cholelithiasis, dyslipidemia or recent alcohol use. He admitted to non-compliance with his anti-diabetic medications. On examination, his abdomen was diffusely tender. Laboratory testing revealed blood glucose of 383 mg/dl, anion gap 23, urine ketones 80mg/dl and an acidic blood pH, consistent with DKA. Serum lipase was elevated to 2595 IU/L. Serum samples were lactescent in appearance. Initial serum triglyceride level was reported as > 3000 mg/dl. An abdominal CT scan showed marked induration surrounding the head of pancreas. Treatment for DKA with intravenous (IV) fluids, IV regular insulin infusion and potassium supplementation was begun per protocol. Oral feeding was held. The anion-gap normalized 36 hours later. Serum triglyceride levels were followed every 12 hours, and a decreasing trend became apparent after 24 hours of continuous insulin infusion. We continued the IV insulin beyond resolution of DKA to treat the acute severe hypertriglyceridemia. 5% dextrose-0.45% saline was infused simultaneously to prevent hypoglycemia. Our goal of serum triglycerides lower than 500 mg/dl was reached after 80 hours of continuous IV insulin, following which transition to subcutaneous long-acting insulin was made. Serum lipase had trended down to 200 IU/L by this time. As the patient’s symptoms resolved, oral feeding was resumed.

In this case, medication non-compliance led to poor glycemic control and DKA, which in turn led to severe hypertriglyceridemia causing acute pancreatitis. In the management of hypertriglyceridemia-induced acute pancreatitis (HTGAP), lowering the serum triglyceride level is a priority. Therapeutic plasma exchange (TPE) is often the preferred option when available. However, in our case, triglyceride levels dramatically declined only with IV insulin, so TPE was deferred. From our experience, DKA-associated HTGAP can be rapidly, efficiently and cost-effectively treated with IV regular insulin and close biochemical monitoring. Additionally, our
patient’s abdominal pain prompted a CT scan which revealed findings of AP early and unexpectedly. Signs and symptoms of DKA may mask those of co-existing AP. Since hypertriglyceridemia, non-specific elevations in serum amylase and lipase are common in DKA, a diagnosis of AP may easily be overlooked. A high index of suspicion for AP is necessary in patients with DKA, especially with co-existing hypertriglyceridemia.
Acute Pericarditis; an uncommon complication of Graves’ disease.

Introduction:
Graves’s disease is an immune mediated pathology considered to be the most common cause of hyperthyroidism. Most common clinical symptoms comprise of palpable goiter, tachycardia, tremors, heat intolerance, and exophthalmos. The most common cardiac manifestations include atrial fibrillation, angina, pulmonary hypertension, and heart failure. Here we describe a case of pericarditis as an initial presentation of Graves’ disease.

Case Description:
A 49 year old man presented to the Emergency department with complaint of chest pain (CP) for the past 24 hours. Medical history was significant only for psoriasis. Patient described the pain as sudden in onset, substernal, “band like”, and non-radiating. Pain started at rest and was exacerbated by deep inspiration with positional variations in intensity. Patient did not report any other associated symptoms except for unintentional weight loss, about 18kgs, over the past 10 months. Patient was given sublingual nitroglycerin en route to hospital with minimal relief. General examination was notable for thyroid bruit, systolic murmur and psoriatic plaques. Vital signs were blood pressure of 142/81, pulse rate 82/min, temperature 36.8 and saturation of 99% on room air. EKG showed diffuse ST segment elevation in the precordial leads. Patient was given aspirin, and clopidogrel. Initial laboratory results were only significant for normocytic anemia and normal cardiac troponins. Based on presentation, pericarditis was high on the differential, however in light of persistent CP and EKG findings, decision was made to perform cardiac catheterization (CC). CC was negative for stenosis, only showed 60-70% LAD lesion with appropriate flow. At this juncture treatment for pericarditis was initiated and patient was started on NSAID and colchicines with improvement in symptoms. Additional labs were significant for normal repeat troponins, low TSH and elevated T4. The thyroid functions were indicative of hyperthyroidism. Thyroid ultrasound showed hyper vascular thyroid, consistent with hyperthyroidism secondary to Graves’ disease. This provided a plausible explanation for both the patient’s weight loss and pericarditis, which is very uncommon complication of graves’ disease. Patient was started on anti-thyroid medications. Further testing to rule out infectious, inflammatory and connective tissue causes for the pericarditis were negative.

Discussion:
Graves’ disease is an autoimmune disease, mediated by TSH receptor antibodies, with multi system clinical symptoms. These include common cardiovascular complications such as tachycardia, atrial fibrillation, pulmonary hypertension and heart failure. A documented but rare complication includes pericarditis. The exact patho-physiological pathways for pericarditis
are unknown but most commonly attributed mechanisms include; infectious, inflammatory and autoimmune. The exact mechanistic association between Graves’ disease and pericarditis still requires study; however clinicians should still be educated on this widely unrecognized complication of Graves’ disease.
Late onset Chemo/radiation induced tracheoesophageal fistula in squamous cells cancer of the lung.

Introduction:
Chemo and radiotherapy are associated with a relatively high incidence of tracheoesophageal fistulae (TEF) formation in lung cancer. In the setting of previous radiation therapy, various anti-angiogenic agents were found to increase risk for fistula formation. We here report a case of a 72-year-old male who developed a TEF upon re-irradiation of squamous cell cancer one year after treatment with gemcitabine/carboplatin.

Case report:
A 72-year-old male with past medical history of stage IV squamous lung cancer was brought to the hospital on 07/16 with anorexia, shortness of breath, and increased hemoptysis for preceding four weeks. His lung cancer was diagnosed in 2008, presented with T4N2M0 non-small cell lung cancer treated with combined modality therapy consisting of chemotherapy and radiation to a dose of 6480 cGy in 180 cGy fractions which led to a 7-year progression-free interval. On 08/15, he had recurrence of the cancer. Patient was not candidate for further radiation therapy. He received palliative chemotherapy with gemcitabine and carboplatin and after four cycles was placed on a chemotherapy holiday. Patient condition had been stable till one month ago when he developed progressive hemoptysis, right sided chest pain and fatigue. On admission, chest X-ray (CXR) showed new worsened reticular markings along left lung base suggestive of infectious process. CT chest showed findings consistent with interval progression of malignancy with an area of consolidation along the medial right lower lung. Sputum culture showed mixed bacterial growth. Patient received short course of antibiotics followed by 10 fraction course of palliative radiation therapy. Immediately after completing his 8th cycle of radiotherapy, he developed progressive coughing and difficulty swallowing, together with shortness of breath, fever and confusion. He was readmitted, CXR showed increased interstitial prominence at the bilateral lung bases. Laboratory work-up revealed signs of infection with a WBC of 21,000, Procalcitonin 0.12 ng/ml. He was diagnosed with hospital acquired pneumonia.
and received broad antibiotics coverage. His sputum culture revealed poly-microbial growth without organism identified. Due to concern of progressive coughing and difficulty swallowing a video swallow evaluation was done. Surprisingly, it showed a tracheoesophageal fistula in the middle third of the esophagus. Patient underwent upper endoscopy and esophageal stent placement.

Tracheoesophageal fistulae are rare complications of thoracic cancers and their treatments. It commonly present with aspiration pneumonia and difficulty swallowing and heralds rapid decline in overall condition. Re-irradiation carries significant risk for esophageal fistula formation. Although delayed complications associated with various chemotherapeutic agent were described among different cancers. Such delayed effect was not widely reported in patients with lung cancer.
Last Name: Smart                 First Author:

First Name: Keith                Category: Clinical Vignette

ACP Member: 3128453

Additional Authors: John Wille

Title: Sudden Cardiac Death Prevented in a Patient with Brugada Syndrome Exacerbated by Hashimoto's Thyroiditis

Abstract:
Introduction:
Brugada syndrome is an autosomal-dominant inherited arrhythmic disorder associated with one of several EKG patterns characterized by incomplete right bundle-branch block and ST-segment elevations in the anterior precordial leads. Brugada syndrome is a channelopathy caused by an alteration in the transmembrane ion currents that constitute the cardiac action potential. Patients with Brugada syndrome are prone to develop ventricular tachyarrhythmias that may lead to syncope, cardiac arrest or sudden cardiac death (SCD) in the absence of structural heart disease.

Case Presentation:
A 59-year-old male with a past medical history of benign prostatic hypertrophy presented to the hospital with syncope. Neurological workup for seizures and stroke were negative. Upon further questioning the patient had a positive family history of SCD in both his father who passed away at age 40 and his sister who was found dead at age 50. EKG showed ST elevations and T wave inversions in leads V1 and V2 consistent with type I Brugada pattern. Echocardiogram demonstrated a preserved ejection fraction and no evidence of structural heart disease. Evaluation by an electrophysiologist solidified the diagnosis with a positive Brugada provocation test and subsequently an implantable cardioverter defibrillator (ICD) was placed. Thyroid function was tested given the patient's increasing fatigue leading up the syncopal event. He was found to have an elevated TSH and a low T3 and T4 consistent with hypothyroidism. The patient was discharged in good condition on Levothyroxine.

Discussion:
This case shows the impact of a careful review of family history. Our patient’s account of SCD in both his father and sister at an early age directed us to the testing needed to diagnose Brugada syndrome. This case also shows the importance of recognizing Brugada pattern on EKG because it went unnoticed on our patient’s prior EKG’s. Our patient had type 1 “coved” Brugada pattern given his EKG with ST segment elevation that descended with an upward convexity into an inverted T wave in leads V1 and V2. Many events are known to unmask or exacerbate the Brugada syndrome such as fever, electrolyte disturbances, alcohol and specific medicines. No such triggers were identified in our patient but he did present with increasing fatigue and was subsequently diagnosed Hashimoto’s thyroiditis. It is most likely that his hypothyroidism
exacerbated the underlying Brugada syndrome resulting in syncope. Many case reports document Brugada EKG pattern caused by hypothyroidism and correction of the Brugada EKG morphology after normalization of thyroid function. However, these patients were asymptomatic and did not meet criteria for Brugada syndrome as our patient did given his inducible electrophysiology provocation testing, syncope and family history of SCD. Therefore, our patient received ICD placement because it is indicated to prevent SCD in patients with Brugada syndrome.
Rhinovirus associated respiratory failure: A rare course of a common cold virus.

Abstract:
Upper and lower viral respiratory infections in adult patients are predominantly mild and self-limited. Though in the past few years, human rhinoviruses/enteroviruses have been associated with severe respiratory infections leading to respiratory failure and Acute Respiratory Distress Syndrome (ARDS), but most cases have been reported in the pediatric population. Here we present a case of a middle aged woman who developed ARDS secondary to a rhinovirus infection.

A 53-year-old female with past medical history of seronegative rheumatoid arthritis, on methotrexate and leflunomide, presented to the ED with one-week history of dyspnea that was gradual in onset, progressively worsening, associated with fever of up to 102 F, dry cough and pre-syncopal episodes. On physical examination she appeared distressed and had bilateral crackles on her chest examination and was saturating at 71%. Laboratory work up revealed leukocytosis and a rhinovirus infection identified on respiratory viral panel. Blood cultures, sputum cultures for bacteria, acid fast bacterial smear, quantiferon gold test and fungal antibody tests were negative. An extensive rheumatological workup was also negative. A CT chest showed bilateral interstitial infiltrates with patchy areas of dense consolidation. An echocardiogram revealed normal cardiac function. She was admitted to ICU for severe hypoxemic respiratory failure, initially requiring high flow rates of oxygen with Optiflow, followed by Bipap support. She was treated with steroids, broad spectrum antibiotics to cover for bacterial pneumonia and likely PCP infection. Her respiratory function worsened despite being on above treatments, eventually requiring intubation and mechanical ventilation. Cultures obtained from bronchosopic aspirates did not show growth. Three days post-intubation, she was weaned off the ventilator and transitioned back to Optiflow. She was discharged after a 16-day hospital course to a subacute rehab facility on supplemental oxygen.

Rhinoviruses/Enteroviruses cause severe respiratory infections mainly in the pediatric population, but they can also affect adults especially those who are immunocompromised or elderly. Mechanisms involved may include direct cytopathic effect to diffuse inflammatory cascades affecting the lungs. Medical institutions from the states of Illinois and Missouri reported to the CDC in 2014 about an increase in the number of cases of severe respiratory illness from enterovirus D68, serotype belonging to the family of enteroviruses/rhinoviruses. In the following weeks, EV-D68 was identified in other Midwestern and Northeastern states and as of October 29, 2014, the CDC had reported EV-D68 in 47 states. Such cases have also been observed in areas near Chicago in past couple of years. Rhinovirus infections are usually ignored and thus the diagnosis is often delayed. Rhinoviruses need to be regarded as pathogens.
responsible for much more than just the common cold and should be suspected as a cause of ARDS, especially in the absence of a more likely cause.
Abstract:
Introduction: Systemic lupus erythematosus (SLE) is a chronic system inflammatory disease of autoimmune origin that may affect the central nervous system (CNS) which has been linked to increased morbidity and mortality. The risk of cerebrovascular events (CVEs) is markedly increased in SLE, and stroke represents one of the most fatal complications, with an incidence rate of 3% and 20%. Stroke can be caused by ischemic or hemorrhagic events, and its occurrence can be justified by several mechanisms, including the presence of antiphospholipid antibodies (aPL) causing disease specific hypercoagulable state. It is estimated that for every aged year, the cardiovascular risk increases by 3% and that CEVs may be responsible for 20% to 30% of the mortality in these patients.
Case: A 23-year-old female with past medical history of lupus since age 7 complicated by nephritis, hypertension, and heart failure presented with confusion and aphasia associated with unsteady gait. In the Emergency Department, she was hemodynamically stable. Laboratory results on admission were remarkable for creatinine 1.59, BUN 53, INR 1, PTT 33, WBC 8.5, hemoglobin 10.8, platelets 111, ESR 26, CRP 0.4, HCG negative. Physical exam was noteworthy of receptive and expressive aphasia, normal strength bilateral upper and lower limbs with a National Institutes of Health Stroke Scale (NIHSS) of 6. MRA head showed early hyperacute nonhemorrhagic infarction of the left parietal lobe and demonstrated slightly irregular vessel lumens at left anterior cerebral artery in the 11th segment in M2, M3 and P3 segmental branches bilaterally suspected to be due to underlying vasculitis from known lupus. Patient received tissue plasminogen activator (tPA). Further workup revealed circulating Lupus Anticoagulant. She was later discharged on enoxaparin 30mg and warfarin 5mg with some improvement in speech.
Discussion: The best clinical monitoring strategy to prevent CVEs in SLE patients is to identify the risk factors related to cerebrovascular disease. Performing a clinical and serological risk profile (aPL and inflammatory markers) is extremely important for the appropriate surveillance of these patients. Several studies have revealed that lupus anticoagulant is a good predictor of thrombosis in SLE. Anticoagulation therapy is the first-line treatment in the prevention of stroke and stroke recurrence in patients with SLE that are at high risk of thrombosis (comorbidities including hypertension, diabetes mellitus, and hyperlipidemia). Recent studies revealed that only those patients maintained at high dose warfarin (target INR 3.1–4.0) was there a significant 10-year benefit. Based on which some experts recommend that SLE patients with ischemic stroke-secondary to APS should receive long-term anticoagulation therapy with a
target INR of 3.0–4.0, rationale being that the danger of thrombosis and stroke in these patients far outweigh the risk of bleeding induced by anticoagulants.
Title: C. Difficile infection, just give antibiotics? THINK AGAIN!

Abstract:
One of the initial thoughts when a patient present to the hospital with diarrhea would be to work up nosocomial causes like C. difficile infection. However, it should be noted that there are other contributing factors. This case is a presentation of diarrhea that was initially thought to be secondary to C. difficile only, but showed improvement after treatment with pancrelipase, suggestive of an atypical presentation of chronic pancreatitis with pancreatic exocrine insufficiency in the setting of severe C. difficile colitis.

A 41-year-old female with history of alcohol abuse and chronic abdominal pain presented to the ED with worsening abdominal pain and diarrhea for four days. Patient stated that her abdominal pain was present for years occasionally radiating to her back. Her pain was mostly in the right lower quadrant and was associated with watery, nonbloody diarrhea. She reported a 40lb unintentional weight loss in less than a year. Denied recent hospital admissions or antibiotic use. Her last admission to a hospital was five months prior to presentation for the same abdominal pain where she underwent an extensive workup which were unrevealing. On admission, she was found to be extremely cachectic, tachycardic and afebrile, with hypoactive bowel sounds. Laboratory tests revealed leukocytosis, metabolic derangements and severe malnutrition. An HIV test was negative, lipase <50, and C. difficile PCR positive. Abdominal imaging revealed pseudomembranous colitis.

She received appropriate treatment for severe C. difficile colitis. She did not improve and thus rifaximin was added after two days. Due to her history of chronic alcoholism and chronic abdominal pain, suspicion for a possible underlying exocrine pancreatic insufficiency were confirmed with surprising low level of fecal elastase-1. She was started on pancrelipase after a week of admission and showed marked improvement within 24 hours. She was discharged to continue a course of vancomycin and pancrelipase.

There is no internationally accepted diagnostic gold standard for chronic pancreatitis. Studies have shown that fecal elastase-1 has the highest sensitivity for diagnosing pancreatic exocrine insufficiency. The mainstay of management of chronic pancreatitis is meal-time pancreatic enzymes. Two double-blind, randomized, placebo-controlled studies by Safdi et al and Whitcomb et al showed that pancrelipase substantially improved clinical symptoms, including stool consistency and abdominal pain, in patients with chronic pancreatitis. There is no study that correlates patients with C. difficile in the setting of chronic pancreatitis, though some studies have shown that changes in the gut environment in acute pancreatic inflammation may place hospitalized patients at higher risk for developing an active infection with C. difficile. This case demonstrates the importance in recognizing the presence of two underlying diagnoses.
that share the same presentation in order to effectively treat patients simultaneously suffering from both illnesses.
Abstract:
The diagnosis of abdominal pathologies are difficult in patients with sensory deficits secondary to spinal cord injuries (SCI). The following is a case of acute cholecystitis in the setting of SCI rendering physical examination limited.

An 80 year-old man with history of ischemic cardiomyopathy and partial SCI at C6-C7 level presented with palpitations. The patient was afebrile and hemodynamically stable. Serum chemistry, complete blood count, and liver profile were unremarkable. Cardiac and abdominal exam were normal. Hours later, the patient became hypoxic, tachycardic, febrile, hypotensive, and somnolent. Cultures were obtained and the patient was started on broad spectrum antibiotics. He was transferred to the intensive care unit and subsequently intubated and required vasopressors. Computed tomography of the chest/abdomen/pelvis revealed a distended, thickened, and ruptured gallbladder with surrounding edema. Cholecystostomy tube was inserted for drainage. Blood and cholecystostomy fluid cultures resulted Escherichia coli and antibiotics were narrowed. Given history of SCI, the patient had reduced abdominal sensation and no symptoms suggestive of intra-abdominal pathology. The patient clinically improved, completed treatment, and was eventually discharged. He later underwent successful laparoscopic cholecystectomy.

Acute cholecystitis presents with right upper quadrant abdominal pain, positive Murphy’s sign, nausea, and fevers. This constellation of symptoms is most commonly due to cystic duct obstruction from cholelithiasis. Laboratory evaluation may reveal a leukocytosis; generally, a liver panel is normal. In addition to not displaying the signs and symptoms of acute cholecystitis upon presentation, this patient also did not have laboratory abnormalities suggesting infection, further obscuring the underlying pathology. Ultrasonography is sensitive (88%) and specific (80%) for cholecystitis; and, will demonstrate gallbladder wall thickening to >4mm with or without cholelithiasis. If diagnosis is uncertain, cholecintigraphy is a highly sensitive (90-97%) and specific (71-90%) method to diagnose cholecystitis. The most common complication of untreated cholecystitis is rupture resulting in sepsis as this patient experienced. Treatment involves drainage and antibiotics targeted against Gram negative organisms and anaerobes. Low risk patients undergo cholecystectomy; whereas, high risk patients undergo cholecystostomy tube insertion with possible cholecystectomy later. Studies have detailed the difficulty of diagnosing acute abdominal pathology in SCI patients who usually present with vague or no symptoms. Diagnoses are often made after clinical deterioration and development.
of complications such as perforation which this case highlights. Advanced age, cervical spinal injuries, male gender are poor prognostic factors. A single-center retrospective study established acute cholecystitis as the most common abdominal emergency seen in SCI patients. Furthermore, this population has an increased incidence of biliary sludge and cholelithiasis compared to non-SCI patients.

SCI patients present with acute abdominal pathology such as acute cholecystitis without classic signs and symptoms. A high degree of suspicion and early advanced imaging are required to promptly diagnose and improve outcomes.
**Abstract:**
Introduction. Legionella species account for almost 10% of community acquired pneumonia. Previous observational studies have shown Legionnaire’s disease to be largely unrecognized, estimating that only 3% of patients carry the discharge diagnosis. Severe cases involving multi-organ dysfunction and intensive care treatment are infrequent but can result in mortality rates that can reach up to 35% if early diagnosis is not made and proper treatment is not initiated. We describe a severe case of Legionnaire’s disease presenting as multi-organ dysfunction in an immunocompetent host with complete recovery after completion of antibiotic treatment.

Case Description. A 42-year-old, previously healthy man presented to the emergency department with a 4-day history of generalized weakness, decreased exercise tolerance and orthopnea. He did not take any medications and his medical history was unremarkable. He denied smoking, drug use and claimed to be a social drinker. On admission, he exhibited increased work of breathing and physical examination was pertinent for tachycardia (116 bpm), tachypnea (33 rpm) hypertension (164/77 mmHg) and late inspiratory crackles in the right upper lobe. Laboratory data showed evidence of coagulopathy, troponin elevation, hepatic dysfunction, and acute kidney injury. Chest radiography and chest computed tomography revealed irregular opacities in the right upper lobe and the diagnosis of sepsis secondary to community-acquired pneumonia was made. Broad-spectrum antimicrobial therapy was initiated (vancomycin, azithromycin, ceftriaxone, and clindamycin) and he was transferred to the MICU. The patient underwent rapid clinical deterioration, including worsening mental status, acute respiratory distress requiring intubation and mechanical ventilation, heart failure with reduced ejection fraction (EF: 10 – 15%), cerebral infarction, rhabdomyolysis, and kidney failure requiring hemodialysis. Microbiological tests, including blood cultures, sputum and endotracheal aspirate analysis, and fungal and viral studies, were all negative. On day 3, Legionella by urine antigen came back as positive. Antibiotic therapy was appropriately changed to levofloxacin, vancomycin and piperacillin-tazobactam. Microbiologic directed treatment resulted in progressive clinical improvement with subsequent successful extubation, normalization of left ventricular ejection fraction, and recovery of urine output with discontinuation of renal support.

Discussion: This case report illustrates the importance of recognizing Legionella species as a potential cause of sepsis with multi-organ dysfunction. It should be considered in any patient who presents with clinical symptoms of community acquired pneumonia regardless of risk factors or identifiable epidemiological exposure since early treatment can avoid progression of the disease and decrease mortality risk.
Abstract:
Lactic acidosis occurring in association with β-2 agonist treatments such as salbutamol, ritodrine, metaproterenol, and albuterol have been reported, although, in most such cases, the lactate elevation was transient. We report a case of severe lactic acidosis in a patient that remained persistently elevated in spite of stopping β-2 agonist therapy.

Our patient was a 48-year-old Caucasian female who presented with complaints of worsening cough, chest discomfort and shortness of breath for 5 days. She also c/o 2 e/o fever. Her medical history was significant for bronchial asthma, essential hypertension, anxiety, depression, and hyperlipidemia. Her asthma was fairly well controlled and she was on salmeterol/fluticasone by inhalation twice a day and albuterol rescue inhaler as needed for wheezing. Since the onset of her symptoms, she had used her albuterol inhaler several times, in addition to this, she also visited an urgent care center and received multiple rounds of albuterol nebulizations which brought about some improvement in her symptoms.

Upon arrival to the ED, her vitals were checked which showed a heart rate of 152/min, blood pressure of 90/60 mm of Hg, respiratory rate of 40/min, SpO2 of 88% on room air with difficulty in speaking and wheezing associated with chest retractions.

Initial labs showed an elevated white count of 14,100 cells/mm3, mild hypokalemia of 3.2 mEq/L with a blood lactate level of 12.9 mmol/L. ABGs showed a pH of 7.23, PCO2 28, PO2 84, HCO3 12 mmol/L. Her chest radiograph was normal.

She was treated with three doses of albuterol combined with ipratropium bromide, hydrocortisone along with injectable magnesium sulfate. She received 3 one liter fluid boluses and was also given a single dose of Zosyn and Vancomycin.

Blood cultures were sent and she was admitted to the intensive care unit with an impression of acute hypoxic respiratory failure and possible septic shock.

During the next 8 hours, her condition gradually improved with a decrease in dyspnoea, normalization of blood gases. Interestingly, a rapid flu test done returned positive. Antibiotics and aggressive fluid resuscitation were subsequently discontinued.

As all common causes of metabolic acidosis were excluded, this lactic acidosis was suspected to be secondary to increased nebulized albuterol which was consequently reduced to intermittent as needed inhalation. She was transferred to the floors the next day. Her lactate levels remained elevated throughout her hospital stay.

Whilst the exact mechanism of lactate elevation with β-2 agonist therapy remains unknown, Increased glycogenolysis leading to overproduction of pyruvate and subsequent lipolysis in addition to the presence of a previous hyper adrenergic state with increased work of
respiratory muscles are postulated as possible explanations for this phenomenon. Prompt recognition of this phenomenon can avoid unnecessary interventions and prevent untoward iatrogenic complications.
Abstract:
Introduction:
Apical hypertrophic cardiomyopathy (ApHCM) is a rare variant of hypertrophic cardiomyopathy, first described in Japan in 1976, characterized by a spade-like left ventricular cavity. ApHCM is more commonly seen in the Asian population, however it has been well documented among many other population groups worldwide. Few cases were reported among African-Americans. This case highlights the rare incidence of the disease among African-Americans as well as the challenging diagnostic and presentation features of the disease.

Case:
A 58-year-old African American female with past medical history of hypertension presented for evaluation of exertional chest tightness, palpitations and headache. Over preceding three years, patient had multiple hospital admissions with similar complaints, and she underwent extensive cardiac workup, including multiple stress tests as well as an angiogram, but a correct diagnosis was not reached. She had no family history of premature coronary artery disease or sudden cardiac death, and no history of tobacco use. Physical examination was within normal limits. Electrocardiogram (EKG) showed normal sinus rhythm, left ventricle hypertrophy and marked T waves inversions in leads II, III and aVF. Troponin was minimally elevated but stable at 0.08 ng/mL, and other labs were unremarkable including TSH and hemoglobin A1C. In light of persistent symptoms, decision was made to take patient for cardiac catherization. coronary angiography showed preserved ejection fraction as well as angiographically normal coronary arteries. Left ventriculopgraphy revealed apical hypertrophy of the left ventricle with a spade like left ventricular cavity that was typical of the Japanese variant of asymmetrical apical hypertrophy known as “Japanese heart disease”, or Yamaguchi syndrome.

ApHCM is often clinically silent, when symptomatic it usually presents with chest pain mimicking acute coronary syndrome. Due to low familiarity, a high degree of suspicion is required to diagnose this condition. Typical EKG findings for ApHCM include repolarization changes and giant (>10 mm), inverted T waves in the anterolateral leads. Diagnostic imaging modalities include two-dimensional Echocardiogram and left ventriculopgraphy however cardiac magnetic resonance remains as the best diagnostic tool. Long term outcomes in term of mortality and sudden cardiac death are rare, however complications such as atrial fibrillation, ventricular arrhythmias and apical thrombosis occurs in up to a third part of the patients, making this diagnosis of critical importance, since these patients need close follow up. With the
increasing cases of ApHCM reported among non-Asian populations, physicians need to consider ApHCM in the evaluation of typical chest pain. Awareness of the unique EKG features, including giant T-wave inversions, provides the initial clues to making the diagnosis.
Title: Horses on the Mind, Zebras in the Brain: a Case of Balo’s Concentric Sclerosis

Abstract:
Balo’s concentric sclerosis is a rare, rapidly progressive, and often fatal variant of multiple sclerosis. The most common symptoms include headache, aphasia, cognitive or behavioral symptoms, and seizures. It is usually diagnosed on MRI (magnetic resonance imaging) by the pathognomonic concentric hyper- and hypo-intensities (“onion ring” appearance).

The patient is a 62 year-old female with a history of mitral valve prolapse, who presented to an outside hospital with one day of word finding difficulties and right upper extremity weakness. On initial physical exam, she was found to have an expressive and comprehensive aphasia, but without any upper extremity weakness. There was initial concern for embolic stroke because the patient had a history of valvular disease and underwent a dental procedure 2 days prior to presentation. Initial CT of the head demonstrated a small hypodense area within the left parietal lobe with mild vasogenic cerebral edema. The patient received dexamethasone. A follow up MRI of the brain with and without contrast showed a 2.8 cm left parietal non-enhancing hypointense lesion with surrounding diffusion restriction. MRA, TTE, TEE, and carotid ultrasound were all negative. Lab work results included normal TSH, elevated ESR (36), elevated CRP (18.5), negative blood cultures, and mildly positive ANA. Hypercoagulable work-up and CSF studies (including oligoclonal bands) were negative. A repeat MRI done a few weeks later showed enlargement of the lesions. The patient continued to receive corticosteroids without significant clinical improvement. One month after presentation, the patient was transferred to UIC Neurosurgery for higher level of care.

At our institution, repeated MRI demonstrated a large nonenhancing, nonhemorrhagic lesion without increased vascularity in the left frontoparietal and temporal junction. These findings were most consistent with demyelinating disease vs. progressive multifocal leukoencephalopathy. Brain biopsy confirmed a demyelinating lesion. Patient was started on plasma exchange and high dose steroids. Repeat MRI to evaluate response to treatment showed the lesion was stable in size and without any new lesion. The patient had only mild improvement of her symptoms over her one month inpatient stay, and was eventually discharged to follow up with rehab.

In summary, MRI played a key role in diagnosis of BCS, and the final diagnosis was confirmed by biopsy. A pitfall to starting steroids before contrast enhanced imaging is that steroids may
affect the enhancing characteristics of the lesion, delaying the diagnosis. In our case, the lesion showed no post-contrast enhancement because the patient was already treated with steroids. Physicians should be mindful about the treatment options they are giving to patients, not only for what side effects they may have, but also for how they may impact the procedures, including imaging, to establish an accurate diagnosis.
Title: Heparin resistance and the challenge in MDS associated thromboembolism: Complications of a complication

Abstract:

Background: The myelodysplastic syndrome (MDS) is a bone marrow disorder of peripheral blood cytopenias, dysplastic cells and occasionally thrombocythemia that can result in venous thrombosis. Qualitative changes in platelet function have also been proposed. Heparin resistance requiring >35,000u/24hrs to achieve therapeutic activated thromboplastin time (aPTT) is best described in inherited or acquired anti-thrombin (AT) deficiency but is an uncommon finding in MDS. We present a case of venous thromboembolism (VTE) from MDS complicated by heparin resistance.

Methods: 51 year old man presented with syncope, pleuritic chest pain and dyspnea for 2 weeks. Physical exam revealed pallor and a flow murmur. Initial studies revealed hemoglobin of 8.2g/dl and thrombocythemia of 575k/ul. CT chest revealed pulmonary embolism and lower extremity ultrasound revealed bilateral acute DVT. Patient did not attain therapeutic aPTT (~39,000u/24hr) on heparin therapy and was started on Argatroban treatment. Evaluation for a malignancy was unremarkable. Hypercoagulability work up was negative for factor V leiden mutation, homocysteine level, and anti-cardiolipin antibodies. Patient had elevated ferritin, normal vitamin B12 and folate levels. Peripheral blood smear revealed severe thrombocythemia. Patient was discharged on warfarin. Further work-up at a tertiary care center revealed MDS.

Results: Limited literature describes an increase in platelet factor 4 that neutralizes heparin in MDS, and an elevation in acute phase reactants which decrease heparin efficacy. Given that AT deficiency usually presents with VTE at an earlier age and our work-up was negative for an identifiable cause for an acquired deficiency; our patient’s resistance is a possible complication of his MDS.

Conclusions: This case highlights the risk of suboptimal treatment response to heparin in patients with MDS, a population that is also at an increased risk for thrombotic complications. A better understanding of the complex mechanisms of heparin resistance, particularly intensified by MDS, would help guide therapeutic management.