New York Chapter ACP
Resident and Medical Student Forum

Poster Presentations
Saturday, October 28, 2017

Staten Island Garden Hilton
1100 South Avenue
Staten Island, NY 10314
New York Chapter ACP

Resident and Medical Student Forum

Medical Student Clinical Vignette
66 year old male with past medical history significant for autoimmune hemolytic anemia, fatty liver, pulmonary embolism, hypertriglyceridemia, seasonal allergies and varicose veins presented with a fever and rash for 4-5 days. Rash was diffuse, vesicular and associated with low grade fever, nausea, vomiting and malaise. Five weeks prior, the patient was prescribed methylprednisolone acetate (Medrol) for new onset back pain and right thigh pain which was attributed to nerve root inflammation. Few days after starting steroids, new rash appeared on his back and right posterior calf for which valacyclovir was started for presumed shingles.

Initial work up on admission to hospital revealed low grade fever (100.5°F). Complete blood count and basic metabolic panel were unremarkable except for a platelet count of 100,000 per microliter and serum sodium of 134 mmol/L. Microbiology and immunology from vesicular fluid revealed varicella zoster virus in viral culture and antibodies against varicella zoster virus antibody on direct fluorescence. He was started on intravenous (IV) acyclovir and steroids were stopped.

On day four of admission, patient complained of right leg numbness which gradually progressed over next 24 hours to involve perineal and sacral areas and the left foot. Also right leg motor weakness (power 4/5) was noted on neurologic exam. Magnetic resonance imaging (MRI) at that point revealed scattered intramedullary thoracic cord signal abnormality with post-contrast enhancement suggestive of transverse myelitis. Spinal tap showed that cerebrospinal fluid was positive for Varicella zoster virus DNA. A diagnosis of post-infection transverse myelitis (PITM) was made and IV acyclovir 10mg/kg every eight hours was started. Methylprednisolone and IV immunoglobulin therapy was added for seven days. Acyclovir was continued for 21 days. Patient responded very well to treatment. After three weeks, all neurological deficits were resolved except that of slight residual numbness in his right foot.

PITM is a rare complication of herpes zoster often observed in immunocompromised patients. Our patient had no history of being immunocompromised, however, his recent steroids for back pain made him vulnerable. Interestingly, steroids are cornerstone of treatment for PITM, yet the same steroids can make an immunocompetent person vulnerable for disseminated shingles and PITM. Steroids are indeed a double edged sword.
Chelidonium majus intoxication: a rare cause of hepatotoxicity

Complementary and alternative medicine (CAM) has become increasingly popular amongst Americans for the past decade. Although many “natural” products seem harmless, there is little awareness about the rare, but serious adverse effects. Chelidonium majus, (Greater Celandine) from the poppy (Papaveraceae) family is used externally for skin conditions (warts, eczema) or internally for gastric and biliary disorders. It has a rare yet known cause of herb induced liver injury (HILI) causing typical symptoms and signs of cholestatic hepatitis. A 54-year-old female of Eastern European descent presented with sudden onset jaundice and abdominal discomfort 6 days prior. She had no medical conditions or drug allergies other than chronic rash on her back for 7 years. She denied any medication use, alcohol or substance abuse, or exposure to hepatitits viruses. Physical examination revealed marked jaundice, scleral icterus, and tenderness of the right upper quadrant without rebound or guarding and an enlarged liver on palpation. Her laboratory work-up showed significantly elevated liver enzymes with alanine aminotransferase (ALT) of 1247 IU/L, aspartate aminotransferase (AST) of 894 IU/L, total bilirubin of 8.6 mg/dL, alkaline phosphatase (ALP) of 190 IU/L. Labs confirmed an absence of acetaminophen and alcohol in the blood and a negative hepatitis panel. Common causes of liver diseases including autoimmune hepatitis were ruled out with negative antinuclear antibody, anti-smooth muscle antibody and urine porphobilinogen tests. Ultrasound examination showed mild hepatomegaly with no evidence of focal hepatic mass. Gallbladder findings were suggestive of adenomyomatosis of the gallbladder, a benign condition that would not lead to the findings in our patient. Upon detailed questioning, patient admitted to drinking Chelidonium tea daily for two months prior to presentation to treat her chronic rash. Intake of Chelidonium was discontinued immediately and she was treated with N acetyl-cysteine (NAC) for 4 days. Symptoms of abdominal discomfort and jaundice improved with treatment. Upon discharge, ALT and AST levels had decreased to 984 IU/L and 607 IU/L, respectively.

Using the Naranjo Drug Reaction Probability Scale in Drug Induced Liver Injury, a score of 7 was calculated which was consistent with a PROBABLE interaction as there was a temporal relationship to a substance with known toxicity in the literature that improved upon withdrawal and which could not be explained by other causes.

Our case provides another significant source of causality between the oral use of Chelidonium with HILI. It raises concern as Chelidonium is legally sold in the United States with little oversight on the potential risks involved with its consumption. Prescribers and customers should be made aware of risks involved in herbal remedies. As there is no diagnostic means for herbal-related toxic hepatitis, clinicians should always inquire about alternative remedies especially when there is liver damage of unknown etiology.

Anomalous Coronary from the Opposite Sinus: A Case of Sudden Cardiac Arrest

Anomalous coronary artery from the opposite sinus (ACAOS) is an extremely rare type of congenital coronary artery anomaly. Typically these patients are asymptomatic and the diagnosis is often made as an incidental finding. Case Presentation We report a case of a 29-year-old male with undetected ACAOS and no other significant medical history who presented with syncope and cardiac arrest while running. A bystander was unable to palpate a pulse and provided chest compressions for several minutes with return of a spontaneous pulse. In the emergency room, the patient was tachycardic to 130 bpm and intermittently in atrial flutter with rapid ventricular response but spontaneously reconverted to normal sinus rhythm. He had elevated troponins but no ST-segment changes on preliminary EKG. Bedside echocardiogram did not reveal any abnormalities. Head CT as well as brain and cervical spine MRI revealed no acute process. Video EEG was negative for any seizure activity. A coronary CTA was performed and demonstrated an anomalous RCA. Subsequent cardiac catheterization demonstrated anomalous take-off of a small non-dominant RCA from the left coronary sinus, with an angulated orifice and an intramural course anteriorly between the pulmonary artery and aorta. Patient elected for surgical intervention and received an unroofing procedure. He was discharged home and has since made a full recovery.

Discussion ACAOS is a challenging case to diagnose and treat with very high mortality if undetected. The presence of ACAOS is difficult to uncover with physical exam and echocardiogram. The detection of the diagnosis requires coronary CTA. Surgery is recommended for all patients with symptomatic ACAOS, and most often for asymptomatic patients with ACAOS of the right coronary artery from the left sinus, as seen in our patient.

Conclusion The consequences of ACAOS can be detrimental; therefore particular attention should be given to cardiac etiologies, specifically for coronary artery abnormalities when investigating a case of exertional syncope. Clinicians should have a high index of suspicion for the possibility of ACAOS of the right coronary artery from the left sinus as a cause of cardiac arrest.
POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME MANIFESTING DURING HEROIN WITHDRAWAL

A 40 year old Caucasian female presented from an inmate facility with new onset seizure activity and blurry vision during a two day period of heroin withdrawal. The patient endorsed crack cocaine use in her teens and regular alcohol use from ages 14 to 21 but denied any recent use of substances besides IV heroin. In the facility, the patient had been undergoing withdrawal symptoms, including diarrhea, nausea, abdominal cramps, and headache. She experienced tonic-clonic convulsions and an elevated blood pressure on the way to the hospital with repeated convulsions in the waiting room and in her hospital room.

An MRI was performed which showed multiple patches of abnormal signal, involving both white and gray matter throughout both cerebral hemispheres, predominantly posteriorly, and in both cerebellar hemispheres. These findings were consistent with Posterior Reversible Encephalopathy Syndrome (PRES). Symptoms improved after normalization of blood pressure and initiation of antiepileptic drugs. Reimaging confirmed the diagnosis of PRES.

Our case is a very typical clinical presentation of PRES with generalized tonic-clonic seizures, visual disturbance, elevated blood pressures, and MRI findings consistent with PRES. However, our literature review suggests this is a unique case of PRES manifesting during a period of heroin withdrawal, which has not been previously described. We see in our case that the patient had significantly elevated blood pressures upon her arrival in the emergency room in the setting of active opioid withdrawal. Previously, heroin has been shown to cause anoxic brain injury and leukoencephalopathy from microvascular damage of cerebral vessels due to apoptosis of oligodendrocytes. However, the MRI findings in our patient were not suggestive of anoxic brain injury. MRI findings in our patient can sometimes be seen in vasculitis as well, but this was inconsistent with the patient’s clinical picture. Moreover, resolution of MRI findings on follow-up imaging with simultaneous resolution of hypertension was not suggestive of a vasculitic process.

The underlying etiology of PRES in the patient described in this report was most likely secondary to uncontrolled hypertension in the setting of opioid withdrawal. This unusual case of heroin withdrawal-induced PRES highlights the importance of early recognition and prompt treatment for patients withdrawing from substance use.
INTRODUCTION

Drug-induced aseptic meningitis (DIAM) is a rare disorder that has been associated with non-steroidal anti-inflammatory drugs (NSAIDs), most commonly ibuprofen. DIAM is correlated with autoimmune connective tissue disorders such as systemic lupus erythematosus, although it has been described in a number of healthy people as well. The following case describes a patient with no history of autoimmune disease, who developed aseptic meningitis following ibuprofen use.

CASE SUMMARY

A 41-year-old female with a history for Ehlers-Danlos syndrome and fibromyalgia presented to the emergency department with intractable back pain, associated with photophobia, headache, and neck stiffness. A lumbar puncture was performed. Cerebrospinal fluid (CSF) analysis showed clear, colorless fluid, proteins 56 mg/dL, glucose 81 mg/dL, and 8 WBC/µL, with a differential of 79% lymphocytes, 21% monocytes/macrophages. PCR analysis of CSF was negative for E. coli, H. influenzae, L. monocytogenes, N. meningitidis, S. agalactiae, S. pneumoniae, cytomegalovirus, enterovirus, HSV1, HSV2, HHV 6, human parechovirus, VZV, and C. neoformans/gatti. Upon reviewing the patient’s history, it was found that she regularly takes multiple 400-800 mg doses of ibuprofen daily for her pain. Her signs and symptoms of meningitis subsided over the next few days.

DISCUSSION

Aseptic meningitis is a rare complication of NSAID use; a recent literature review reported 72 cases of DIAM associated with NSAIDs, 46 of those caused by ibuprofen. Symptoms of NSAID-induced DIAM include fever, headache, meningismus, nausea/vomiting, and altered mental status. Signs and symptoms of meningitis often resolve soon after removal of the offending agent. While the pathophysiology of this disorder remains unclear, its association with autoimmune disorders may point to an autoimmune mechanism.

CSF findings in DIAM usually include pleocytosis with lymphocytic predominance, as in this case. CSF protein is often significantly elevated, though this patient only had moderately high levels. The clinical and laboratory similarities to infectious causes of meningitis may lead to a low clinical suspicion of this rare disorder, but the lack of culture or PCR findings should lead physicians to consider DIAM in their differential diagnosis for cryptogenic meningitis. Furthermore, the increased use of over-the-counter NSAIDs should give clinicians a lower threshold to consider DIAM in the differential. DIAM is a diagnosis of exclusion, and the patient should first be worked up and empirically treated for possible infectious cause of meningitis.
### Medical Student Clinical Vignette

<table>
<thead>
<tr>
<th>Nicole Lifson</th>
<th>Benjamin Parnes</th>
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<tr>
<td><strong>First Author:</strong> Nicole Lifson <strong>Second Authors:</strong> Alexander Fein, John Lofrese <strong>Third Authors:</strong> David Lehmann, MD Lauren Krowl, MD</td>
<td><strong>Renee McDonald-Fleming MS4, Marie Abdallah M.D.</strong></td>
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<td><strong>SUNY Upstate Medical University</strong></td>
<td><strong>SUNY Downstate Medical Center</strong></td>
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<td><strong>Thiazide-induced unmasking of Type 2 (proximal) renal tubular acidosis</strong></td>
<td><strong>Laryngeal Coccidioidomycosis in a Healthy 34-year-old Male</strong></td>
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**Type 2 renal tubular acidosis, or proximal renal tubular acidosis, is a genetic defect in the ability of the proximal renal tubular cells to reabsorb bicarbonate and electrolytes that may go undiagnosed. This leads to excretion of bicarbonate and subsequent acidaemia, proteinuria, phosphaturia, glycosuria, and aminoaciduria.**

**Background:** A 58-year-old male with hypertension presented with syncope. The patient stood up to use a copier machine when he collapsed and struck his head. The patient stated that his primary care physician instructed him to administer an additional dose of hydrochlorothiazide if his blood pressure were to rise. One day prior to admission the patient took an extra dose of hydrochlorothiazide. On admission the neurological examination was unremarkable and remained so throughout hospitalization. On admission his blood pressure was 107/66. Serum values were: sodium 116 mmol/L, chloride 77 mmol/L, bicarbonate 22 mmol/L, glucose 138 mg/dL, creatinine 1.6 mg/dL, BUN 28 mg/dL. The patient’s urine chemistry indicated a chloride of 20 meq/L, potassium of 50 meq/L, and urine pH of 5. The patient’s hydrochlorothiazide was discontinued. Intravenous saline bicarbonate was administered and other electrolytes were repleted.

**Discussion:** Thiazide diuretics inhibit the thiazide-sensitive Na+-Cl- cotransporter in the distal convoluted tubule. The hypovolemic, hyponatremic, and hypochloremic presentation may initially indicate a thiazide overdose, but the patient’s metabolic acidosis with a bicarbonate of 22 mmol/liter suggests otherwise. Hypovolemia and hyponatremia trigger the renin-angiotensin-aldosterone system, stimulating the nephron to correct for volume and sodium loss. Angiotensin II triggers the Na+-H+ exchanger in the proximal convoluted tubule, promoting reabsorption of bicarbonate and secretion of hydrogen ions into the tubular lumen. In other words, hypovolemic hyponatremia should lead to an alkalotic response by increasing serum bicarbonate. The metabolic acidemia and glucosuria (despite normal blood glucose) indicate an underlying proximal tubular defect that produces bicarbonate loss.

**Conclusion:** This case shows that thiazide overdoses can precipitate a serious electrolyte abnormality while simultaneously aiding in the diagnosis of an underlying renal tubular acidosis. It is important to identify renal tubular defects in patients before prescribing any medications that affect electrolyte transport in the renal tubules. Routine electrolyte screening and urinalysis should be considered for those on high dose diuretics.

| **Introduction:** Coccidioides is a dimorphic fungus that is known to live in the soil in the Southwestern United States, parts of Mexico, and in Central and South America. Coccidioides is commonly acquired through inhalation of airborne arthroconidia, which are found in soil in these various different geographic regions. The presentation can range from asymptomatic to a mild self-limited acute disease to disseminated disease with involvement of the tissues lining the brain, soft tissues, joints, and bones. Immunocompromised patients are at an increased risk for disseminated Coccidioidomycosis.
| **Case:** In this case, we present a 34-year-old male who presented with a 2-month history of hoarseness without any associated respiratory symptoms or other associated symptoms of a Coccidioidomycosis infection. He was originally from Puebla, Mexico, and his last visit to Mexico was in 2013. He had no significant history of tobacco use, illicit drug use, or alcohol consumption. On presentation to the ED, he appeared physically well and had stable vital signs. Routine blood work, including a CBC and CMP, were unremarkable. The patient also had a chest radiograph, which was also unremarkable. Due to the patient’s continued complaints of hoarseness, a fiberoptic endoscopy was performed, which revealed a 1 cm supraglottic mass, and he subsequently underwent an excisional biopsy of the mass. Pathological samples of the specimen stained with Grocott’s methenamine silver stain and periodic acid-schiff stain were positive for fungal elements consistent with spherules containing endospores. Serologic testing for Histoplasma and Blastomyces were both negative. Serologic testing for Coccidioides antibodies were positive for IgG antibodies (1:4) and negative for IgM antibodies. HIV antigen/antibody testing was negative. Real-time PCR testing of a tissue specimen from the patient’s excised mass was positive for Coccidioides psadasii, negative for Coccidioides immitis, negative for Blastomyces dermatitidis, and negative for Cryptococcus species.
| **Discussion:** Coccidioidomycosis is an infection that is found in the Southwestern United States, parts of Mexico, and in Central and South America. Symptomatic Coccidioidomycosis commonly presents in immunocompromised patients. Our patient’s presentation was unique because he was a healthy young patient with no other medical issues who presented with involvement of his larynx. Our patient not only presented with no history of an immunocompromised state, but also had no symptoms of a systemic infectious process. The etiology of our patient’s laryngeal Coccidioidomycosis infection was not clear. On review of the literature, two reported cases of isolated laryngeal Coccidioidomycosis without any other evidence of disease were reported. While our patient’s presentation of laryngeal Coccidioidomycosis is rare, it demonstrates that endemic fungal infections always need to be considered as part of the differential diagnosis in a patient with a laryngeal mass.
Metastatic Sarcomatoid Carcinoma in a Young, Female, HIV-Positive Patient

Sarcomatoid carcinoma is a rare malignancy of the lung, accounting for less than one percent of all lung cancers (1,2). It has a male-to-female predominance of almost 4-to-1, has a mean age of onset of 60 years, and is associated with a poor prognosis (1). This is, to the best of our knowledge, the second case of metastatic sarcomatoid carcinoma in HIV patients (3) and the first in a young, female patient. We present a case of sarcomatoid carcinoma in a 37-year-old patient with a history of Hodgkin’s Lymphoma treated with chemotherapy and uncontrolled HIV initially presenting with unresponsiveness, tachycardia, and hypoxia after several days of vaginal bleeding. Chest CT on admission revealed a cavitary lesion in the right upper lobe, which was suspected to be tuberculosis. A lung biopsy performed revealed highly atypical spindle epithelial cells highly suspicious for sarcomatoid carcinoma. A biopsy of a left iliac spine lesion found on CT revealed cells similar in morphology and immunohistochemistry to the lung specimen, consistent with metastatic sarcomatoid carcinoma. After lengthy discussions, the patient opted to hospice care secondary to her poor functional status. Overall, this case highlighted the importance of a broad differential in the approach to patients with unconfirmed diagnoses, and expands the metastatic profile of sarcomatoid carcinoma.

References:

AL Amyloidosis & Multiple Myeloma: A Case of Coexistence

Background: AL amyloidosis and multiple myeloma (MM) represent a spectrum of similar diseases that are characterized by monoclonal plasma cell proliferation. The clonal plasma cells produce misfolded light chains, leading to organ deposition and subsequent organ dysfunction. In approximately 10% of patients with AL amyloidosis, multiple myeloma may also be present at the time of initial diagnosis. We describe a case of a 73-year-old asymptomatic female with acute onset renal insufficiency and absence of overt MM findings. The patient was subsequently found to have AL amyloid deposition on renal biopsy and coexisting bone marrow findings diagnostic for multiple myeloma. This case therefore highlights the importance of considering both AL amyloidosis and multiple myeloma as differential diagnoses for acute, unexplained renal dysfunction in elderly patients, regardless of whether clinical MM findings exist.

Case Presentation: A 73-year-old female was admitted after a routine primary care appointment revealed an elevated creatinine of 7.2 from her baseline of 0.7 three months prior. Initial physical examination was unremarkable aside from mild lower extremity edema, which the patient endorsed had been improving. The patient otherwise denied any fever, chills, significant fatigue, shortness of breath, or bone pain. Laboratory examination revealed nephrotic range proteinuria of 12 grams in the setting of a hypoalbuminemia of 2.6. Thus, given the acuity of decompensation and to further understand the etiology of this patient’s acute onset proteinuria, a renal biopsy was performed, revealing a positive Congo red stain for amyloid by light microscopy. Follow-up immunofluorescence staining confirmed the presence of lambda light chain deposition. These results were considered diagnostic for AL amyloidosis, so a bone marrow biopsy was subsequently performed to rule out an underlying plasma cell tumor. The BM biopsy revealed a hypercellular marrow with diffuse infiltration of clonal plasma cells at >25%, and immunophenotyping confirmed CD38+ plasma cells, both consistent with an additional diagnosis of multiple myeloma.

Discussion: This patient presented with an acute onset of nephrotic syndrome and lack of “classic” MM myeloma findings such as anemia (observed in 73% of patients), bone pain (58%), and hypercalcemia (28%). This case demonstrates that a targeted work up for plasma cell dyscrasias should be considered in elderly patients who present with unexplained renal insufficiency in the setting of acute nephrosis. In addition, this patient’s renal function declined significantly within a matter of three months, which is less characteristic of the indolent progression of renal insufficiency in amyloidosis and MM, normally progressing from one to two years. Therefore, a high index of suspicion should be maintained for plasma cell proliferative disorders in the diagnostic workup of elderly patients who are otherwise asymptomatic but present with unexplained renal insufficiency.
New York Chapter ACP
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Medical Student
Research
SECONDARY MALIGNANCIES IN BREAST CANCER PATIENTS TREATED WITH RADIOTHERAPY

Purpose: Breast cancer (BC) is the most commonly diagnosed cancer in women in the western world, and the second leading cause of death among women. Radiotherapy (RT) for breast cancer (BC) appears to be effective for the reduction of both cancer recurrence and mortality. However, it remains unclear if RT in combination with smoking increases the risk of second primary cancers (SPCs) and mortality. The purpose of this study is to evaluate the relationship between RT and smoking status on the risk of SPCs and mortality among BC patients with a follow-up period of up to 15 years.

Methods: This study utilized data from the Northwell Cancer Registry. We conducted a retrospective cohort study of BC patients treated with post-operative RT from 2000 to 2014 to evaluate long-term SPC risks of RT in combination with smoking habits. The total number of BC patients (stage 0-III) from 2000-2014 was 14,106. Cox proportional hazard model was used to assess the hazard ratios (HRs) of developing a SPCs or dying based on RT and smoking status.

Results: The risk of developing SPCs in smokers treated with RT was 79% higher (HR=1.79, 95%CI=1.43-2.23) when compared to the reference (never-smokers/never-treated with RT), while the risk for never-smokers treated with RT was 31% higher (HR=1.31, 95%CI =1.06-1.63). Smokers treated with RT were at greater risk of developing hematological (HR=5.87, 95%CI=1.97-17.53), gastrointestinal (HR= 2.46, 95%CI =1.26-4.81), urological (HR= 4.02, 95%CI=1.13-14.26), gynecological (HR= 2.84, 95%CI =1.53-5.29) SPCs, and lung cancer (HR= 4.98, 95%CI=2.06-12.05) when compared to the reference. No significant risk was observed for thyroid, skin and breast cancers. Moreover, smokers who received RT were at higher risk of dying after the occurrence of SPCs (HR=2.53 95%CI=1.61-3.99). In contrast, the risk of dying was lower among never-smokers that received RT but did not develop SPCs (HR= 0.78, 95%CI=0.66-0.92).

Conclusion: Our results suggest that smoking before or at time of BC diagnosis can elevate the risk of developing SPCs as well as the risk of dying among those who received RT. This study suggests that careful evaluation of smoking history should be considered in the decision process of therapeutic treatment and subsequent surveillance among BC patients. This study provides further understanding of the relationship that exists between RT and smoking history in the development of new malignancies. The investigation of this topic may lead to the improvement of clinical care for patients by providing a better understanding of the effects of RT.

Role of esophageal dilation in patients with ineffective esophageal motility

Background:
Ineffective Esophageal Motility (IEM) is diagnosed by manometry based on the strength of contractions in the esophagus. Patients with IEM may report dysphagia, angina, globus, regurgitation and acid reflux. The mechanism behind this diagnosis involves an imbalance of inhibitory and excitatory forces on the esophagus. Furthermore, there has not been sufficient evidence supporting any treatment. Esophageal dilation is frequently used in IEM patients with dysphagia.

Objective:
To evaluate effectiveness of esophageal dilation in relieving dysphagia in IEM patients.

Methods:
Retrospective cohort analysis of 130 adult patients with IEM between 2006 and 2015. Of the 130 patients with IEM, 37 underwent esophagogastroduodenoscopy (EGD) with dilation. Of the 37 who were dilated, 31 returned for a follow-up appointment. Resolution or return of dysphagia was recorded. Data was then analyzed to determine if dilation improved dysphagia.

Results:
28% underwent EGD with dilation
73% were female
Mean age was 53
43% had symptom improvement, 33% has no improvement after 1 dilation and 24% did not follow up
Out of the 37 patients with IEM who underwent 1 EGD with dilation, 16 had symptom improvement, 8 had recurrence of symptoms with 4-6 months, 12 had no improvement after 1 dilation and 9 did not follow up. 4 patients underwent multiple EGD with dilation and none had improvement in symptoms.

Discussion:
Even though EGD with dilation has been a common practice to achieve for symptom improvement in patients with IEM, this study shows that this method is not as effective as once thought. Less than half of the patients who underwent this procedure for IEM symptoms saw any improvement. Even though this study does not offer alternative treatments, it does show that this widely used procedure may not be the best first line treatment.

Conclusion:
This data suggests that esophageal dilation may, at least temporarily, help relieve dysphagia in about 40% of patients with IEM. Unfortunately, half of those who had recurrence of dysphagia within a few months. Further studies are needed to better define the role of esophageal dilation in this population.
Comparing the National Inpatient Sample and the MarketScan Databases: Can We Rely on Them?

Study Design: Inter-database reliability and validity study of two observational national databases.

Objectives: Ascertain similarities and discrepancies in extracted population demographics and outcomes between two commonly used national databases.

Methods: International Classification of Diseases, 9th edition (ICD-9) codes were used to identify elderly spine surgery patients in Truven Health Analytics MarketScan claims database (2000-2012) and National (Nationwide) Inpatient Sample (NIS) discharge database (1998-2011). Patient baseline characteristics, comorbid status, insurance enrollment, and outcomes were queried.

Results: We analyzed 15,303 and 206,957 patients between ages 80 to 100 years (mean 83.0) from MarketScan and NIS databases, respectively. MarketScan participants tended to have better comorbidity status, receive fusion relatively more often, and were insured by Medicare insurance more frequently than those in the NIS database. The risk of sustaining one or more complications was lower in the MarketScan database compared with NIS (11.7% vs 16.9%). No significant difference was observed in mortality rate (0.30% vs 0.34%). Consistently, older age, female gender, fusion surgery and worse comorbidity status represented increased odds of complications.

Conclusions: This study highlights the discrepancies in demographics and outcomes of spine surgery in two commonly used databases. Overall, it appears that NIS patients have more complications and comorbidities, and are likely a sicker population than those in the MarketScan database. As databases gain popularity in Medicine, clinicians and reviewers should be cautious to ascribe generalizability of results from just the statistical significance.

Ketogenic diets have become an alternative treatment for childhood epilepsy, cancer, Alzheimer's and other neurogenic diseases. Little or no carbohydrate intake with adequate protein and high fat consumption is thought to starve the body of glucose and force it to use fatty acids and their metabolites, ketone bodies, as the main energy source. Ketogenic diets have demonstrated efficacy in specific epileptic syndromes and in some pediatric patients with drug resistant epilepsy. Major ketone bodies studied include ß-hydroxybutyrate (BHB), acetoacetate, and acetone with BHB. Research on ketone bodies has become more prevalent due to the application of ketogenic diets to neurologic conditions. This investigation looked at the effect of a common ketogenic diet metabolite, BHB, on human microglia in vitro, specifically whether BHB could induce polarization of unstimulated microglia to an M1 inflammatory or M2 resolving state. HMC3 at or below passage six were grown in lower glucose medium (10mM glucose EMEM) and normal glucose medium (25mM glucose DMEM) before being treated with BHB at concentrations of 0.0mM, 0.5mM, 2.5mM and 5.0mM to simulate a ketogenic milieu. Trypan Blue viability tests were done in both glucose conditions on all concentrations of BHB resulting in no significant difference between BHB groups in viability with treatment. To test for polarization, real-time PCR was run and analyzed for the M1 marker gene Arginase1 and the M2 marker gene iNOS with GAPDH as the housekeeping gene. There was a significant increase in expression of iNOS in the 5.0mM BHB treatment group compared to the 0.5mM BHB treatment group (p=0.0240). There was no significant difference in expression of Arginase1 between groups. Our results suggest that unstimulated HMC3 cells respond to higher concentrations of BHB and polarize to the M1 state. Polarization to the M1 state is typically associated with adverse inflammatory events so these results may suggest that an M1 phenotype could be reparative in the presence of BHB. Future directions include stimulating HMC3 cells with lipopolysaccharide (LPS) before treating with BHB to see if BHB blocks the effect of LPS in a dose dependent manner. Additionally, other polarization markers including COX2, CX3CR1, CCR7, mannose receptor, and the mitochondrial function marker PGC1-a will be analyzed. This foundational work will help lead to an understanding of the relationship between a ketogenic diet and the role of inflammatory microglia in neurologic diseases.
Defining the Role of Hypoxia Inducible Factor (HIF) in Non-pulmonary Vascular Smooth Muscle Cells

Peripheral vascular disease (PVD) affects tens of millions of people in the United States, and is a significant cause of morbidity. PVD is a pathological condition that is caused by insufficient tissue perfusion, which could lead to the loss of a limb or even death. Insufficient tissue perfusion leads to ischemia, which is characterized by low oxygen tension (hypoxia) and reduced nutrients. Hypoxia leads to the activation of Hypoxia Inducible Factor (HIF), which is a transcription factor regulating the expression of angiogenic genes. Vascular smooth muscle cells (VSMC), present in the tunica media layer of vessels, modulate their phenotype in response to physiological and pathological cues. We hypothesize that HIF-1 is an essential regulator of smooth muscle cell activation and phenotype in the peripheral vasculature required for effective angiogenic and arteriogenic responses to ischemia. Our research is concerned with testing the requirement for HIF in non-pulmonary VSMC responses to stresses including hypoxia and starvation by examining the expression of contractile genes in peripheral VSMC and subsequent phenotypic changes. Additionally, we utilized an in vivo carotid artery ligation model to examine the vascular remodeling in VSMC-HIF deficient mice. Our preliminary data depicts trends of differing levels of Alpha-actin-2 and Calponin-1 gene expression between starved and unstarved conditions, in arterial and venous VSMCs. However, we observed no difference in the remodeling of ligated carotids between the control and VSMC-HIF knocked out mice. These preliminary results will help further our understanding of the role of HIF in regulating peripheral VSMC phenotype.
Patient Awareness of Atrial Fibrillation: Major Risk Factors and Treatment Options

Background: Atrial fibrillation (AF) is a common arrhythmia that carries a significant risk of stroke, however, patients are often unaware of their own diagnosis. Furthermore, patients often have little understanding of the complex treatment options available including rate control, rhythm control, anticoagulation, and catheter ablation. We aimed to assess patients’ understanding of AF, and their insight into the consequences of having such arrhythmia.

Methods and Results:
A survey was conducted amongst 50 patients who were already diagnosed with AF in both inpatient (n=35) and outpatient (n=15) settings between September 2016 and May 2017. The median age was 69.5 and the average CHA2DS2-VASc score was 4.1. Forty-seven out of 50 patients (94%) identified to have known about their diagnosis of AF. Only 34% were able to identify if they had persistent or paroxysmal AF. Of those with knowledge of their diagnosis, 85% knew that AF could increase their risk of a stroke, and 92% understood that anticoagulation could decrease that risk. Subsequently, 87% were currently treated with anticoagulants and 98% indicated that they were seeing a cardiologist and/or electrophysiologist for their AF. However, only 26% were aware of whether they were currently on rate or rhythm control and only 60% identified catheter ablation as a treatment option.

Conclusions:
The vast majority of patients are aware of their diagnosis of AF, understand that they are at an increased risk for stroke, and that anticoagulation can decrease that risk. However, only a minority of patients have an understanding of rate or rhythm control, and catheter ablation as treatment options. Education should be targeted at patient awareness of the various different types of medical treatments including catheter ablation.

Medical Student Research

DOES ORDERING A BLOOD TEST STAT VS TIMED VS ROUTINE REALLY MAKE A DIFFERENCE?

Purpose of Study: To determine the difference in collection and laboratory processing time between blood specimens ordered “STAT” versus “timed” versus “routine” when done by phlebotomy versus intensive care unit (ICU) staff.

Methods: The current state in the surgical ICU at Emory University Midtown Hospital requires all blood work to be drawn by a centralized phlebotomy team. A randomized, monocentric, prospective study was undertaken from June 1, 2017 through July 1, 2017 to compare the difference in blood collection and laboratory processing times, whether ordered STAT, timed, or routine, between our current system (phlebotomy team) versus a model we created in which the ICU healthcare staff drew blood work as well.

Results: When drawn by a centralized phlebotomist, the average time needed to collect a STAT, timed, or routine specimen was 74.9, 65.5, and 79.3 minutes, respectively. When drawn by ICU staff, the average time needed to collect a STAT, timed, or routine specimen was 22.2, 29.9, and 34.6 minutes, respectively. The time required for the laboratory to process a STAT, timed or routine sample was 68.3 minutes, 62.3 minutes, and 68.5 minutes.

Conclusion: Ordering a specimen as STAT or “timed” utilizes alternative phlebotomy staff and can increase healthcare costs. To date, this is the first study to examine the difference in collection or processing time of a STAT, timed, or routine specimen. Our study has shown that the ordering label makes little impact in collecting and processing time, whether done by a phlebotomist or ICU staff. Providers should therefore avoid unnecessary STAT or timed orders and utilize ICU staff for tests needed urgently. In time, this may ultimately improve specimen prioritization.
**Shannon Lance BA**  
Shaikha, Zarya; Yang, Joanna; Xu, Lily D.O.; DeLauro, Salvatore M.D.; Becker, Maureen D.H.Sc.; Smith, Marianne M.D.  
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**A BRIDGE TO HELP: DEPRESSION SCREENINGS ON COLLEGE CAMPUSES- A PILOT STUDY**  

**Purpose:** The purpose of this study is to determine to the effectiveness of depression screenings among study participants in a college campus wellness pilot study.  
**Methods:** Eighteen College of Staten Island (CSI) Dolphin Cove dormitory residents were recruited in October 2016. A depression screening questionnaire (PHQ-2) was administered to all participants. If participants scored a one or higher they were subsequently administered a PHQ-9. Participants scoring above a four on the PHQ-9 were counseled regarding results and referred to the CSI student health center for mental health services.  
**Results:** 72% (13/18) of students tested positive on the PHQ-2 depression screen and 83% (10/12) of students who were subsequently administered PHQ-9 questionnaires were referred to the student health center. 100% (10/10) of students were compliant with student health center referrals.  
**Conclusions:** This pilot study supports the value of proactive depression screenings for college students. Our process also emphasized the importance of private discussions of positive screening results to empower help-seeking behavior in students. Our findings support the need for future studies of global depression screenings on campuses.

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**Effects of NSAID Use and Race on Heart Failure Readmissions and Hospital Resource Utilization**  
**Background:** NSAIDs are widely used and have been shown to cause renal dysfunction, elevated blood pressure, and volume retention. In the setting of heart failure, these adverse effects are especially deleterious. The effects of NSAIDs on readmissions and hospital resource utilization in patients with a heart failure diagnosis have not been well investigated.  
**Material and Methods:** Medical charts of 1551 consecutive patients admitted with heart failure diagnosis to a single tertiary center hospital during 2007-2016 were reviewed in this IRB-approved study. There were 132 patients with chronic NSAID use. Control group consisted of 173 age, gender, race, diabetes, coronary artery disease, hypertension, peripheral vascular disease, atrial fibrillation, and severe renal insufficiency or hemodialysis matched control subjects from the same cohort. Analysis of variance, chi-square, and logistic regression analyses were used.  
**Results:** NSAID patients experienced more readmissions (17% vs. 4%, 2 or more readmissions, p<0.001 when compared to controls) and longer cumulative length of stay (15.9±21.8 vs. 9.7±12.9 days, p=0.002, respectively). In multi-variable logistic regression analysis, when adjusted for all recorded demographic variables and co-morbidities, only NSAID use (HR 4.366, 95%CI 1.772-10.757, p=0.001) and non-white race (HR 2.815, 95%CI 1.248-6.349, p=0.013) were associated with more than 1 heart failure readmission. Compared to Caucasians, non-white patients were younger (58.4±177;21.1 vs. 68.7±177;13.9 years old, p<0.001), with lower LV ejection fraction (39.7±177;14.6 vs. 43.9±177;13.2, p=0.028, respectively), and with significantly increased prevalence of end stage renal disease or hemodialysis (19.1% vs. 8.4%, p=0.013, respectively). Non-white patients experienced more readmissions (20.6% vs. 6.8%, 2 or more readmissions, p=0.003 when compared to Caucasians) and numerically longer cumulative length of stay (15.9±177;26.2 vs. 11.4±177;14.1 days, p=0.062, respectively).  
**Conclusions:** NSAID usage is associated with more frequent heart failure readmissions and increased hospital resource utilization. When adjusted for demographic variables, co-morbidities, and NSAID use, race remained an important predictor of heart failure readmission. Future studies of this important subject are warranted.
Background: One in three American adults has prediabetes, which increases their risk of developing diabetes mellitus type 2, as well as comorbidities including cardiovascular and renal diseases. In rodents, epoxyeicosatrienoic acids (EETs), cytochrome P450 metabolites of arachidonic acid, are known to decrease blood pressure, inflammation and pain, and increase insulin sensitivity. Unfortunately, EETs are rapidly hydrolyzed by the enzyme soluble epoxide hydrolase (sEH) into functionally less active dihydroxyeicosatrienoic acids (DHETs). sEH inhibition is known to increase endogenous EET concentrations, and our group recently found that circulating EETs increase insulin sensitivity in both mice and humans. Despite recent characterization of EET concentrations and sEH activity in rodent tissues and human plasma, these molecules have never before been measured in human insulin-sensitive tissues. Methods: EETs, DHETs, EpOMEs* and DiHOMEs* (*metabolites of linoleic acid, also hydrolyzed by sEH) were extracted from plasma, adipose tissue and muscle of overweight or obese prediabetic individuals and quantified by negative ESI–LC/MS/MS using stable isotope labeled internal standards. sEH activity in these samples was measured as the rate of conversion of pharmacological EETs to DHETs, and EpOMEs to DiHOMEs. Results: Our assays successfully measured three isomers of EETs and DHETs (14,15, 11,12 and 8,9) and two isomers of EpOMEs and DiHOMEs (10,11 and 12,13) in human adipose tissue and muscle for the first time. Consistent with studies in human plasma, EpOMEs and DiHOMEs were measured in adipose and muscle in concentrations 100-1000x greater than those of EETs and DHETs. EET and DHET concentrations were comparable in mice and human adipose tissue, with significant differences seen only in 8,9-EET levels and 14,15-DHET levels (p<0.05). We found a positive correlation between DHET/EET ratios and sEH enzymatic activity for all isomers, as hypothesized, except 8,9-DHET/EET, and we observed strong positive correlations for metabolite concentrations and sEH enzymatic activity between human plasma and adipose tissue. Finally, we found a correlation between patient BMI and sEH activity in adipose tissue, consistent with our knowledge that obesity is a risk factor for insulin resistance. Conclusions: Our data show for the first time that EETs and sEH activity are measurable in human adipose tissue and muscle. Along with past data, this suggests that pharmacological inhibition of sEH activity is a feasible and promising approach for increasing insulin sensitivity in insulin-resistant individuals. Further, we can conclude that sEH activity in plasma can be used as an accurate biomarker of the enzyme’s activity in insulin-sensitive tissues, requiring only a blood test and not an adipose biopsy for monitoring.
Taking Off the Training Wheels in Medical School

Background: According to American Board of Internal Medicine (ABIM) guidelines, residents should have been involved in at least five instances of each of the following procedures: cardiopulmonary resuscitation (CPR), arterial blood gases (ABGs), phlebotomy, and placing intravenous (IV) lines. Clinical rotations are a time for medical students to gain experience with hands-on procedures that are commonly encountered during residency. However, many students enter residency feeling unready to do common procedures, so in this study, the aim was to determine what procedures clinical students were commonly gaining practice with to investigate their preparedness for residency.

Methods: Medical students at 11 different schools were emailed a link to a digital survey. The survey queried how many times student had been involved with common procedures. Procedures included were placing IV lines, phlebotomy, ABGs, suturing, CPR, bag mask ventilation, Foley catheter insertion, and incision/drainage (I&D) of abscesses.

Results: 121 responses were initially collected; 8 were excluded because they were completed by medical school graduates, and a further 8 responses were excluded because the students were not in clinical rotations. Of the included 105 responses, 52 were completed by third-year medical students and 53 by fourth-year medical students. 91.4% had completed core rotations in at least internal medicine, and 65.7% had completed core rotations in at least surgery. For each procedure, students were dichotomized into having never done the procedure, or done it at least once. Phlebotomy and suturing were the procedures that the largest proportion of eligible students had done at least once, with 71.4% and 66.6% respectively. The remaining procedures with proportion of students having done them at least once are as follows: IVâ€™s (59.0%), CPR (54.3%), Foley catheter insertion (52.4%), and bag mask ventilation (49.5%). The least commonly done were I&Ds (45.7%) and ABGs (49.5%). The average total number of the listed procedures done by each student was 13.3.

Discussion: The survey results indicate that clinical students have good exposure to certain procedures such as phlebotomy in working towards the ABIM guidelines. Students also showed a moderate degree of exposure to IV placement, CPR, and Foley catheterization. However, in general, the rates of procedure exposure is less than desired overall (especially for ABGs and I&Ds), and begs the question of whether students are getting the practice they need prior to residency. This data will be utilized in conjunction with medical schools and hospitals to further improve the quality of clinical rotations.

Daily Coffee Promotes Sustained Viral Response in HCV Infected Patients

Background
Chronic hepatitis C virus (HCV) infection can progress to severe liver disease including cirrhosis and hepatocellular carcinoma. There are 150,000 new cases of HCV infection diagnosed annually in the United States. Elimination of the virus is determined by attainment of a Sustained Viral Response (SVR). SVR is defined as maintenance of undetectable HCV RNA by PCR for six months after completing anti-viral therapy and now classified as three months with newer forms of therapy. Coffee consumption has been associated with reduced incidence of chronic liver disease, liver cancer, a lower rate of disease progression and viral loads in patients with HCV. Such effects are also seen similarly with tea. We hypothesize that daily caffeine intake will lead to lower initial viral loads and higher sustainment of SVR. We also believe that these effects can also be seen with other forms of intake that is composed of caffeine.

Method
A prospective observational study was designed to determine if daily caffeine intake is associated with higher achievements of SVR in HCV infected patients on antiviral therapy including Peg interferon-Ribavirin and Direct acting antivirals (DAA). Study participants were patients of Coney Island Hospital Hepatology clinic. Patients over the age of 21 years and with HCV infection were enrolled. Patients with all other forms of hepatitis and liver disease unrelated to HCV were excluded. Data was collected via a XX-tem questionnaire. High viral was defined as >800,000 IU/ml and the degree of liver disease determined by either liver biopsy or Fibrosure lab testing.

Results
Data acquisition is still being actively processed with a target sample size of 316 patients that would provide a 97% power. Currently 17 patients are enrolled for which preliminary results reveal 10 patients with SVR and 7 who did not attain SVR. There were 13 pts who had frequent coffee intake for which 8 revealed low viral loads prior to therapy.

Conclusion
Current data demonstrates a trend towards a lower initial viral load and higher SVR in pts who had caffeine intake on a consistent basis. Further data analysis is to be determined based on SVR among different Genotypes and their degree of liver fibrosis.
RATE OF PANCREATIC PSEUDOCYST IN PANCREATITIS ACCORDING TO ETIOLOGY

Purpose for Study:
To evaluate the rate of pancreatic pseudocyst in patients with pancreatitis in our population with high rates of alcohol abuse and to compare patients with pancreatic pseudocyst to the overall patients with pancreatitis and gender distribution.

Methods:
Chart review of patients from an urban community hospital, as a quality improvement project from January 2010 to May 2017. Using ICD 9 code for pancreatitis patients with the diagnosis of pancreatitis were selected and their charts reviewed for clinical data, demographics and imaging results. Main outcome was rate of pancreatic pseudocyst formation and subsequent complications.

Summary of Results:
Four hundred forty patients with pancreatitis were identified, of which 21 patients (4.8%) had pancreatic pseudocysts. Overall, 263 patients (59.77%) with pancreatitis were male and 177 patients (40.23%) with pancreatitis were female. There were 17 male patients with pancreatic pseudocyst (80.95%) and 4 female patients with pancreatic pseudocyst (19.05%).

Overall, the age range of pancreatitis patients was 3 to 92 years and the median age was 46 years. The age range of pancreatic pseudocyst patients was 26 to 72 years and the median age was 49 years. The etiologies of the pancreatitis of the pseudocyst patients were: 11 Alcohol (52.4%), 4 Unspecified/Undetermined (19%), 2 Gallstone (9.5%), 2 Post-ECRP (9.5%), 1 Post-surgical procedure (4.8%), and 1 Hypertriglyceridemia (4.8%). The location of the pancreatic pseudocysts was as follows: 7 Solely Pancreatic Head (33.3%), 9 Solely Pancreatic Tail (42.9%), 2 Solely Pancreatic Body (9.5%), 1 Extending from Pancreatic Body to Tail (4.8%), and 2 Pancreatic Body and Tail (9.5%).

Overall, 277 patients with pancreatitis abused alcohol. Of these 277 patients, 207 patients (74.7%) were male and 70 patients (25.3%) were female. Of the 21 pseudocyst patients, 2 patients (9.5%) abused alcohol and had gallstones, 16 patients (76.2%) abused alcohol but did not have gallstones and 3 patients (14.3%) did not abuse alcohol or have gallstones.

All 17 male patients with pseudocyst (80.95%) were alcohol abusers and 1 female patient with pseudocyst (4.8%) was an alcohol abuser. One patient (4.8%) with pancreatic pseudocyst had a pseudocyst rupture as a complication.

Conclusions:
Alcohol abuse appears to result in the formation of pancreatic pseudocyst more than the presence of gallstones in patients with pancreatitis. Male patients were the majority of pancreatitis patients (59.77%) and the majority of pancreatic pseudocyst patients (80.95%). Increased emphasis on alcohol abuse counseling as well as monitoring for the formation of pancreatic pseudocysts in patients with pancreatitis due to alcohol abuse might be useful intervention to avoid complications. This study is limited due to small sample size and results are from one center with large number of patients with high alcohol abuse as well as the lack of follow up and images for all patients.
New York Chapter ACP
Resident and Medical Student Forum

Resident/Fellow
Clinical Vignette
Cavitation and fibrosis are the end results of caseous necrosis in tuberculosis and nontuberculous mycobacterial pulmonary infections. The lung destruction can be so extensive that fibrosis and cavities replace the entire lung, the process called autoneumonectomy (APE). This case report describes APE in Mycobacterium Avium Complex (MAC) infection with a large air-filled cavity replacing the whole lung. A 76-year-old man presented to the emergency department complaining of progressive shortness of breath for one-month duration, cough with small amount of yellowish sputum, generalized weakness, weight loss and poor appetite. Past medical history of pulmonary MAC infection, first diagnosed at age of 67. He was treated with appropriate therapy at that time and was lost to follow-up. Never smoked but was a construction worker. No surgeries. On physical examination, he was afebrile, tachycardic (118 beats/min) and tachypneic (20 breaths/min). Oxygen saturation was 92% on ambient air. He was cachectic and in mild respiratory distress. On percussion of the right posterior chest, tympanic sound was elicited. Amorphous sounds were heard in the same area on auscultation. Rales were noted in the left chest. Laboratory work showed mild leukocytosis. Chest radiograph and CT scan revealed a large air-filled cavity in place of the right lung. Right pleura was diffusely abnormal and thickened. Left lung was hyper-expanded with multifocal bronchiectasis and opacities. Left lower lobe consolidation noted with small left pleural effusion. The radiograph was compared to the one from nine years ago, which only had multiple opacities in the right lung with some fibrosis. Patient was placed in airborne isolation and sputa for AFB and regular culture were sent. Presumptive diagnosis of reactivated MAC infection was made. The patient was placed on anti-MAC treatment and therapy for possible concurrent community-acquired pneumonia (CAP). Three sputum samples came back smear positive for AFB. Polymerase chain reaction for Mycobacterium tuberculosis was negative and airborne isolation was discontinued. The culture grew MAC. With therapy, the patient’s constitutional and pulmonary symptoms improved. Within nine years, the insidious MAC infection caused the affected lung to be completely replaced with an air-filled cavity. There have been case reports of patients with APE being asymptomatic until a pathologic process developed in the remaining lung. The reactivation of MAC and possible superimposed CAP made the patient seek medical attention. Given public health concerns, the patient was placed in airborne isolation until pulmonary tuberculosis could be excluded. Cases of coexistence of MAC and Mycobacterium tuberculosis infections had been reported.
Resident/ Fellow Clinical Vignette

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**Pembrolizumab associated Colitis**

**Introduction**
Pembrolizumab is an anti-PD-1 Monoclonal Antibody recently approved for various malignancies, including melanoma and non-small cell lung cancer, with positive PD-1 markers and has been associated with improved survival. It has several potential side effects involving the gastrointestinal tract ranging from decreased appetite and diarrhea to immune mediated colitis.

**Case report**
A 70 year old male with a history of Coronary artery disease and Stage IV adenocarcinoma of the right lung with liver and cerebellar metastases presented with bloody diarrhea. He reported 4-5 bloody bowel movement a day associated with abdominal cramping but no fever or chills. He was recently treated with antibiotics for toe osteomyelitis and reported a 20 pound weight loss over the last few months. Medications include pembrolizumab for his lung cancer. On examination, he was afebrile and normotensive. Abdominal exam was unremarkable. Labs revealed hemoglobin 11.2 mg/dl, hematocrit 34.1 and white blood cell 6000. Extensive stool testing including ova/parasite and Clostridium Difficile toxin were negative. Serum and liver chemistries were also unremarkable. Colonoscopy revealed pan-colitis with diffuse ulcers and erythema. Biopsy revealed active chronic colitis without dysplasia. Given his presentation and colonoscopy findings pembrolizumab induced colitis was suspected and he was started on high dose steroids with marked improvement in his diarrhea.

**Conclusion:**
Pembrolizumab associated immune colitis can occur as early as 10 days after starting therapy and usually presents as bloody diarrhea. Most patients respond well to high dose steroids, 1 mg/kg/day, for seven days. Severe cases, refractory to steroids, may require termination of drug therapy. In a growing era of new anti-neoplastic agents attention should be paid to potential unique side effects, as timely and appropriate intervention may allow continuation of anti-neoplastic therapy with a survival benefit. In this case the patient was treated with high doses of steroids with marked improvement in his symptoms.

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**MANDIBULAR AMELOBLASTOMA AND THROMBOEMBOLISM**

**Introduction**
Acquired venous thromboembolism (VTE) is known to be associated with malignancy. Although the incidence is low, VTE has been reported in head and neck cancer but it is unclear in benign tumors of the head and neck. We present a case of spontaneous extensive VTE in a patient with histologically confirmed ameloblastoma and no other identifiable risks.

**Case Detail**
A 76-year-old Haitian man with no significant medical history and a previous smoking history presented to local ENT clinic for evaluation of a right mandibular mass. The mass has been growing slowly for the past 2 years, with sudden increasing size over the last 15 days. The mass started to erode his gums and teeth, causing ulceration with intermittent bleeding. He reported a 3-week history of dyspnea on exertion and chest pressure. He was admitted electively for a planned surgical biopsy. He underwent cardiac work-up as part of pre-operative risk stratification. Echocardiography revealed elevated pulmonary artery pressure of 73 mmHg with normal left ventricular ejection fraction and right ventricular function. CT scan of chest revealed extensive bilateral pulmonary emboli (PE) and lower extremity sonogram demonstrated occlusive deep vein thrombosis (DVT) of bilateral popliteal veins. Patient was placed on a heparin drip, but administration was frequently interrupted due to development of severe bleeding from the oral ulcers requiring transfusions. Due to difficulties in anticoagulating the patient, an inferior vena cava filter was placed and patient was placed on lovenox 48 hours later when bleeding was slowed. Pathology revealed areas of keratinization replacing stellate reticulum and cystic follicles filled with keratinized epithelium, consistent with the acanthomatous variant of ameloblastoma.

**Discussion**
Ameloblastoma is a rare benign tumor of the jaw that is odontogenic in origin and often locally aggressive. Acanthomatous type is one of the rarest variants. This tumor can occur at any age, has a higher prevalence in men, and is treated with surgical resection. One case series reported the development of DVT in one patient, however it occurred after the mandibular reconstruction surgery. To our knowledge, there has never been a case report regarding the spontaneous development of preoperative VTE in a benign ameloblastoma patient as described here. VTE is often described as a complication of malignancy due to the production of procoagulant factors by cancer cells. There are limited reports of VTE in benign tumors, especially in patients with older age, larger tumor size and obesity.

**Conclusion**
In a patient presenting with a mass or tumor of any origin, a physician should consider a lower threshold to evaluate for VTE with appropriate clinical signs and symptoms.
Resident/ Fellow Clinical Vignette

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Impact of Standard Mean Arterial Pressure on Acute Kidney Injury in Patients with Shock according to Age Groups

Background: Management of mean arterial pressure (MAP) to a target value is crucial in shock. Recent studies advocate the use of higher MAP target in patients in shock who are at risk of developing acute kidney injury (AKI). However, there is limited clinical data to support this approach. Our objective was to compare the AKI outcome in patients with shock according to age and the achieved MAP.

Methods: We performed a retrospective chart review of patients admitted to the Intensive Care Unit (ICU) of one tertiary care center from Jan 2012 to May 2015. We obtained three MAP readings per day for the first three ICU days (D 0,1,2), with one mean value per day. Patients were stratified into three MAP groups (65-70,70-75 and 75-80mmHg). Patients were also grouped according to age (less than 60 year of age and equal or more than 60 year of age). The study’s primary outcome was the incidence of AKI according to both age and MAP.

Results: Our sample size included 255 patients (104 were less than 60 year of age, 151 were equal or more than 60 year of age). The incidence of AKI was similar regardless of the achieved MAP. Within each age group, MAP did not have an impact on the incidence of AKI (incidence of AKI in patients aged less than 60 years on D0: 86.6% in MAP 65-70 mmHg, 73.9% in MAP 70-75 mmHg, 78.9% in MAP 75-80 mmHg, p=0.65; Incidence of AKI in patients aged 60 years or above on D0: 67.8% in MAP 65-70 mmHg, 76% in MAP 70-75 mmHg, 75% in MAP 75-80 mmHg, p=0.81). Furthermore, there were no statistically significant differences in the incidence of AKI for all age and MAP groups on all studied days (D0, D1, D2).

Conclusion: Older adults with shock have a higher incidence of AKI compared to younger patients, with no associated reduction in AKI incidence with higher MAP. Larger studies are needed to confirm whether a more conservative MAP target achieves the similar AKI outcomes compared to a higher, more aggressive one.

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A Case of Hirschsprung Disease in an Adult Male

Hirschsprung disease (HD) is usually diagnosed early in childhood, but can rarely manifest in later childhood (age > 10 years old) or adulthood. Complications of HD include bowel obstruction, bowel necrosis, bacterial overgrowth and infection from stool stasis. Our case highlights the need for a high index of suspicion for HD in patients with chronic constipation without a clear medical etiology, in order to avoid long term complications.

A 42-year-old man with a past medical history of chronic constipation presented with abdominal pain, nausea, vomiting, and diarrhea for 4 days. He had similar episodes in the past requiring intravenous fluids during Emergency Department visits. On physical examination, the patient had hypoactive bowel sounds, abdominal distention, and stool in rectum. Computed tomography of the abdomen/pelvis revealed severe constipation and dilated rectosigmoid colon measuring 19 cm. The patient was placed on an extensive bowel regimen with moderate stool clearance. He refused elective colon resection or biopsy. Follow-up imaging showed decreased stool content. He was discharged home on Miralax twice a day and Linzess daily, with adequate bowel movements. Outpatient colonoscopy demonstrated erythematous rectal mucosa with a hyperplastic polyp. Outpatient anorectal manometry showed normal resting and squeeze pressures and an absent rectal sensation and recto-anal inhibitory reflex suggestive of HD. The patient eventually became amenable to a rectal biopsy that revealed aganglionic neural bundles on S100 immunostain consistent with a diagnosis of HD.

In 75% of cases, the rectosigmoid is the affected colonic segment and in 10% there is total aganglionosis of the colon. Usually, symptoms present within the first year of life and account for 80% of cases; late diagnosis is often consistent with ultra-short segment hirschsprung disease (USHD), which lacks increased acetylcholinesterase in the mucosal layers when compared to HD. Anorectal manometry is a reliable tool to aid in the diagnosis of HD with specificity and positive predictive value of 97% and 95 % respectively when combined with rectal biopsy. There is controversy surrounding the diagnostic criteria for USHD. Some believe the criteria should be modified to include =4 cm of aganglionosis; this would help define proper biopsy technique and decrease recurrence from insufficient resection. This area of contention warrants further study.
Hypereosinophilic syndromes (HES) are a rare group of myeloproliferative disorders characterized by a persistently elevated absolute eosinophil count (>1.5 x 10^9/L) with evidence of organ involvement, usually the skin, heart, lung and nervous system. Historically, HES was classified as idiopathic or secondary, due to parasitic infections, allergic reactions, autoimmune disorders and malignancies. However, the recent identification of the clonal molecular genetic rearrangement, FIP1L1-PDGRa fusion gene, has led to the reclassification of HES as chronic eosinophilic leukemia (CEL). As such, patients previously thought to have Idiopathic HES may actually have CEL. The product of FIP1L1-PDGRa is an imatinib-sensitive protein tyrosine kinase, which allows the use of targeted therapy.

A 28-year-old man of Caribbean descent with no known comorbidities presented to the infectious diseases clinic with a 3-month history of recurrent painless oral and genital ulcers. Acyclovir was given by his PCP with no improvement. Besides having Herpes labialis years ago, he denied penile discharge, lymphadenopathy, fever, night sweats or weight loss, current drug use or allergies, alcohol or tobacco use. Complete blood count was normal except for eosinophilia (absolute eosinophil count of 2.96 x10^9/L), confirmed with repeat study. Serologic studies for HIV, Hepatitis A & B, HTLV I/II and Syphilis were negative. HSV Ig G was positive. Cultures from the oral and penile ulcers were negative for HSV.

Stool studies for ova, cysts and parasites, and serology for Strongyloides, ANA, anti-neutrophil cytoplasmic antibody, and rheumatoid factor were negative. The finding of eosinophilia without an obvious infectious cause prompted referral to the Hematology service. At the time, he had healed tongue ulcers, no other abnormalities on examination. Blood film revealed normal red cell morphology and predominance of eosinophils. Molecular testing of peripheral blood revealed FIP1L1-PDGRa rearrangement, which confirmed the diagnosis of HES/CEL. Chest CT was unremarkable. Transthoracic echocardiography finding moderate concentric left ventricular hypertrophy with a normal ejection fraction and so, Prednisone 1mg/kg/d (80mg) was given, along with Imatinib 400mg po daily. Prednisone was discontinued after 2 weeks and Imatinib lowered to 200mg given the resolution of eosinophilia (0.11 x10^9/L).

This case highlights several pertinent points. In a young patient of Caribbean descent, as in this case, an infectious etiology is usually the first to be considered as a cause of eosinophilia but once ruled out other etiologies must be explored. HES should be considered in a patient with mucosal ulcers and hypereosinophilia. Patients with FIP1L1-PDGRa positive HES/CEL have been found to have more extensive organ involvement. End-organ damage is the most feared complication of HES/CEL, most notably cardiac dysfunction which is a major cause of morbidity and mortality. Rapid and complete hematologic, cytogenetic and molecular responses have been seen with Imatinib, which targets FIP1L1-PDGRa and has led to improved patient outcomes.

Hypereosinophilic syndromes with suspected cardiac involvement

Hypereosinophilic syndromes with suspected cardiac involvement

The prevalence of non-neoplastic PVT in patients with liver cirrhosis ranges from 8.4% to 15% and is higher in patients with more severe liver disease. Anticoagulation therapy for PVT in cirrhotic individuals is associated with complete recanalization rates between 33% and 45% after 6 months. Anticoagulation is a challenging therapy in individuals with liver cirrhosis because of the well-recognized coagulation abnormalities observed in that setting and because of the increased risk of bleeding, especially from gastrointestinal tract caused by portal hypertension.

Case description:

76-year-old woman with past medical history of NASH induced liver Cirrhosis with portal hypertension, esophageal varices, Spontaneous bacterial peritonitis, Hypertension presented after worsening bilateral leg swelling and abdominal distension. Physical examination was remarkable for a non-tender, distended abdomen with bilateral shifting dullness with pedal edema. Labs showed sodium 141, potassium 4.5, BUN 12, creatinine 0.59, alkaline phosphatase 98, AST 43, ALT 27, albumin 3.1, total bilirubin 1.3, Hemoglobin 12, hematocrit 35, INR 1.4, PTT 12. CT abdomen and pelvis with IV contrast revealed acute non-occlusive thrombus in the main portal vein and right portal vein with occlusive thrombus in the posterior right portal vein along with moderate ascites and bilateral pleural effusion. An US Doppler of hepatic vessels was done and it was consistent with the CT findings and demonstrated hepatofugal flow within the portal venous system. Patient was initiated on anticoagulation with unfractionated heparin with a PTT of 81. The following day patient had an episode of hematemesis and melena with a drop in hemoglobin and hematocrit. Heparin drip was discontinued and patient was scheduled for an emergent upper esophagogastroduodenoscopy (EGD) which showed three columns of grade II esophageal varices with red wale sign status post placement of 6 bands with complete control of bleeding. Patient remained clinically stable with no more episodes of gastrointestinal bleeding and had an uneventful stay in the hospital. A decision was made to hold the anticoagulation until a repeat surveillance EGD in 3-4 weeks.

Discussion:

Anticoagulation is considered the therapy of choice in patients with non-cirrhotic portal vein occlusion but concerns of AT for PVT in patients with liver cirrhosis are founded on the high risk of bleeding related to clotting impairment and portal hypertension. In the setting of decompensated cirrhosis are founded on the high risk of bleeding related to clotting impairment and portal hypertension. In the setting of decompensated cirrhosis is it necessary to show convincing and definitive safety of AT before starting therapy of PVT. It is preferable to screen for varices before starting anticoagulation and endoscopic variceal ligation (EVL) should be performed if patient has grade II or grade III esophageal varices prior to starting AT. More data are needed to make evidence-based recommendations on the use of these agents in patients with acute PVT, particularly for those with cirrhosis and portal hypertension.

Resident/ Fellow Clinical Vignette

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<th>Odeth Barrett-Campbell MD</th>
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<td>Tracian James-Goulbourne MD, Rochelle Hardie MD, Jason P. Gonsky MD, PhD</td>
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<td>SUNY Health Sciences Center in Brooklyn (Downstate)</td>
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<td>HYPEREOSINOPHILIC SYNDROME/ CHRONIC EOSINOPHILIC LEUKEMIA WITH SUSPECTED CARDIAC INVOLVEMENT</td>
<td>ANTICOAGULATION THERAPY (AT) FOR PORTAL VEIN THROMBOSIS (PVT) IN LIVER CIRRHOSIS: A DILEMMA TO TREAT</td>
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Rheumatoid arthritis and sickle cell disease (RA-SCD) have been rarely described together. In our experience, these two entities are found at a prevalence (0.94%) similar to that observed for rheumatoid arthritis in the general population (0.5% - 1%)[1]. The mainstay of therapy for sickle cell disease is glucocorticoids have been reported to cause rebound vaso-occlusive crisis (VOC). We describe two cases where this specific dilemma was encountered.

Case 1: 26 year-old woman with established RA-SCD presented with subacute bilateral knee pain and swelling worse on the right. Patient was diagnosed with RA years ago in her native country. Four months prior to presentation, patient was put on a 40mg to 10mg prednisone taper and methotrexate. She endorsed adherence to her RA medications and was not on hydroxyurea. Physical exam demonstrated swelling of the knees. Initial laboratory studies were not suggestive of acute hemolysis. Given this presentation, RA flare was suspect as compared with VOC. Patient received 500mg IV methylprednisolone followed by 40mg of prednisone daily with significant improvement in her pain. Three days after steroids were started, she developed bilateral hip and back pain consistent with her sickle cell crisis. Hemolysis labs also echoed a sickle cell crisis. Patient's crisis was treated with hydration and opiates with discharge after 11 days.

Case 2: 22 year old woman with recently established RA-SCD on the inpatient rehab service begins to complain of bilateral wrist pain and swelling. Patient endorsed compliance with recent 20mg prednisone taper as well as hydroxyurea. Physical exam demonstrated bilateral swollen wrists. Pain was not consistent with her typical crises and labs were not suggestive of hemolysis. Patient was given methylprednisolone 500mg IV followed by 40mg prednisone daily for a suspected RA flare. Three days after receiving steroids, she developed diffuse generalized body aches and pruritis on her arms, face, and chest. Hemolysis labs echoed a sickle cell crisis. Patient’s crisis was treated with hydration and opiates with discharge after 10 days.

Although a rare entity, treating RA flares in patients with sickle cell disease poses a unique challenge. From assessment of our patients’ disease course, we were intrigued to observe improvement of RA flare followed by acute sickle crisis. Literature search identified that a rebound pain crisis, although rare, has been documented in pediatric populations [2,3]. Given conflicting published results, there is still no consensus on the net effect of these medications in sickle cell crises [4,5]. Our series will remind clinicians to be mindful of high dose systemic steroid use in RA flares in this niche population.

Introduction: We present a case of diabetes mellitus (DM) diagnosed as type 2 in a young obese patient on routine examination who later developed liver cirrhosis. Further workup revealed hemochromatosis and its common presentation as diabetes mellitus was missed, with delay in diagnosis leading to liver cirrhosis and subsequent hepatoma formation.

A 54-year-old male was referred to Endocrinology for uncontrolled diabetes mellitus type 2 (diagnosed 15 years ago, sub-optimal control with metformin, A1C 9.8%). Past medical history was noted to be significant for chronic stable mild thrombocytopenia, alcohol abuse and hepatic steatosis. Family history was significant for DM in several family members. Physical exam revealed no complications of DM and no hepatosplenomegaly. Labs showed elevated AST-75 (Normal (N): 17-59U/L), ALT-93 (N: 21-72U/L), alkaline phosphatase-354 (N: 38-125U/L) and thrombocytopenia of 88K (N: 140-425K). Elevated liver enzymes were presumed to be from alcohol abuse. Labs 5 months later showed persistently elevated AST-81, ALT-93, alkaline phosphatase-349, and GGT-1756 (N: 8-78U/L). CT abdomen showed hepatic steatosis, mild enlargement of spleen and nodularity concerning for evolving cirrhosis. A follow-up abdominal ultrasound 3 months later showed 2 small hypoechogenic liver lesions, suspicious given background of cirrhosis. Workup for hepatitis A, B and C, HIV -1, HIV-2, ANA, AMA, alpha-1 antitrypsin deficiency, Wilson’s disease, and celiac disease were negative. Iron panel showed TIBC-244 (N: 250-450ng/dl), ferritin-1735 (N: 20-400ng/dl), iron-227 (N: 50-150ug/dl) raising suspicion for hemochromatosis and AFP was elevated to 15, suspicious for hepatocellular carcinoma (HCC). MRI abdomen showed a 1.4 cm nodule with arterial enhancement and washout, cirrhosis with portal hypertension and a siderotic nodule adjacent to the gallbladder fossa. EGD showed esophageal varices. Repeat MRI after 2 months showed progression of the lesions consistent with stage 2 HCC. Genetic testing for HFE gene mutations revealed C282Y negative, but H63D positive. With an official diagnosis of hemochromatosis, he was started on phlebotomy, and ferritin improved to 27. He underwent transarterial chemoembolization for his liver lesions. Pt HbA1c improved to 8% with diet, exercise, weight loss and addition of glipizide.

Conclusion:
Secondary causes such as hemochromatosis should be strongly suspected in patients with early onset diabetes mellitus not well controlled with oral hypoglycemics, especially when associated with elevated liver enzymes and strong family history of early-onset diabetes. In retrospect, our patient most likely developed diabetes secondary to undiagnosed hemochromatosis, which also led to liver cirrhosis and subsequently hepatoma. The association between hemochromatosis and diabetes was first recognized in the late 1800s, when doctors coined the term “sorbonne diabetes.” It is due to selective beta-cell damage due to uptake of iron, leading to impaired insulin synthesis and release. Treatment with phlebotomy in this patient population has the benefit of reducing the degree of hepatic fibrosis if cirrhosis is absent.

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| Madiha Alvi, MD                    |
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| Bassett Medical Center             |
| **A missed case of common presentation of hemochromatosis with diabetes mellitus, complicated by cirrhosis leading to hepatoma** |

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COMPLAINTS OF MONO- OR POLYARTICULAR JOINT PAIN ARE UBQUITOUS IN THE CLINICAL SETTING. SINCE ADULT ONSET STILL’S DISEASE (AOSD) IS A DIAGNOSIS OF EXCLUSION, IT SHOULD BE CONSIDERED ANYTIME JOINT PAIN WORKUP IS NEGATIVE FOR THE MORE COMMON CAUSES OF ARTHRALGIA.

A 49 YEAR-OLD WOMAN, WITH NO SIGNIFICANT PAST MEDICAL HISTORY, PRESENTED TO OUR EMERGENCY DEPARTMENT WITH A CHIEF COMPLAINT OF SUDDEN ONSET OF DIFFUSE JOINT PAIN FOR TWO DAYS. A REVIEW OF SYSTEMS REVEALED A MILD, NON-PRODUCTIVE COUGH WITH SUBJECTIVE NIGHTLY FEVERS FOR THE PAST TWO WEEKS, AND AN 11.3 KG WEIGHT LOSS IN THE SETTING OF WATERY DIARRHEA SHE ATTRIBUTED TO AN HERBAL DIET SUPPLEMENT OVER THE LAST THREE MONTHS.

UPON EXAMINATION, THE PATIENT WAS LYING VERY STILL IN BED, HER JOINTS TENDER TO PALPATION. ON HOSPITAL DAY TWO, THE PATIENT SPIKED A NIGHT-TIME TEMPERATURE OF 39.3°C AND CONTINUED TO SPIKE NIGHTLY FEVERS FOR THREE DAYS. AT THAT TIME, THE PATIENT ALSO DEVELOPED A WARM, PATCHY, MACULAR RASH ON HER CHEST THAT SUBSEQUENTLY RESOLVED. THERE WAS AN INITIAL LEUKOCYTOSIS OF 17.3 K/μL AND BLOOD, URINE, AND SPUTUM CULTURE SCREENS WERE NEGATIVE. X-RAYS OF HER JOINTS DENTROITED NO ACUTE OR CHRONIC PATHOLOGY. FERRITIN LEVELS WERE AT 10300 μG/L. RHEUMATOLOGIC SEROLOGIES WERE NEGATIVE. ANGIOTENSIN CONVERTING ENZYME, CREATINE PHOSPHOKINASE, HEPATITIS VIRAL PANEL, LYME SEROLOGY, CYTOMEGALOVIRUS, EBSTEIN-BARR VIRUS, HIV 1/2 ANTIBODY AND ANTIGEN, RESPIRATORY VIRAL PANEL, SYPHILIS IgG, PARVOVIRUS B19 PCR, CHLAMYDIA, AND GONORRHOEA WERE NEGATIVE. ASPIRIN 1,000 MG WAS STARTED ON DAY TWO AFTER WHICH SHE WAS SWITCHED TO PREDNISONE 20 MG DAILY ON DAY FOUR. PATIENT’S SYMPTOMS RESOLVED, AND SHE WAS SUBSEQUENTLY DISCHARGED FROM THE HOSPITAL.

JOINT PAIN AND ARTHRITIS IS FOUND IN 64-100% OF AOSD PATIENTS, AND FEVER IS PRESENT IN 82-100% OF PATIENTS. RELATIVELY UNIQUE TO AOSD IS THAT THESE PATIENTS TYPICALLY WILL SPIKE HIGH FEVERS (>39°C) ONCE TO TWICE DAILY, ON A BACKGROUND OF NORMAL TEMPERATURES OR MILD PERSISTENT FEVERS.


FURTHERMORE, THERE ARE OTHER LESS SPECIFIC FINDINGS THAT SUPPORT THE DIAGNOSIS OF AOSD INCLUDING: SORE THROAT/PHARYNGITIS, HEPATOMEGALY OR SPLENOMEGALY, ABNORMAL LIVER FUNCTION TESTS, AND ELEVATED FERRITIN LEVELS4. ANYTIME THE TRIAD OF FEVER, RASH, AND JOINT PAIN APPEARS IN THE CONTEXT OF CONSISTENTLY NEGATIVE OR NONSPECIFIC WORKUP, IT IS IMPORTANT TO LOOK BACK OVER THE PATIENT’S COURSE TO SEE IF THERE IS A TEMPORAL RELATIONSHIP BETWEEN THE PRESENTING SYMPTOMS. THESE KEY POINTS WILL HELP TO KEEP AOSD ON THE DIFFERENTIAL.
Resident/ Fellow Clinical Vignette

Raja Chandra Chakinala MBBS
George P. Jolly, MBBS; Shashvat Gupta, MBBS; Lavneet Chawla, MBBS; Leanne Forman, MD; Ronald Cho, MD
New York Medical College at Westchester Medical Center
DIABETIC KETOACIDOSIS ASSOCIATED WITH DAPAGLIFLOZIN, A SODIUM-GLUCOSE CO-Transporter 2 INHIBITOR

Introduction:
Sodium-glucose co-transporter 2 (SGLT2) inhibitors such as canagliflozin, dapagliflozin and empagliflozin belong to a newer class of antihyperglycemic agents which inhibit glucose reuptake in the proximal tubule of the kidney, causing glycosuria, thereby improving glycemic control. Hypoglycemia, dehydration, and urinary tract infection are some of the adverse effects of this class of drugs with recent reports raising concern for the development of euglycemic diabetic ketoacidosis (DKA). Although majority of cases of DKA were reported with off label use of SGLT2 inhibitors in type 1 diabetes mellitus (DM) patients, rare cases have also been reported in patients with type 2 DM. We present a case of SGLT2 inhibitor associated DKA developed in a type 2 DM patient after initiating therapy with dapagliflozin.

Case report:
61 year old male with history of type 2 DM presented to the emergency department with complaints of dizziness. His medication list includes metformin 1000mg twice daily, glimepiride 8mg twice daily and dapagliflozin 5mg once daily. He was initially started on canagliflozin 6 months prior to admission, which was switched to dapagliflozin 3 months prior to admission for insurance related issues. On admission, he was found to have a hemoglobin A1C of 8.1, blood glucose level of 409 mg/dL, serum bicarbonate of 14 mEq/L, anion gap of 22 mEq/L, pH of 7.34 and urine positive for ketones. He was appropriately managed for DKA, and dapagliflozin was discontinued. He was discharged home on an insulin regimen. He had no further re-admissions for DKA.

Discussion:
Diabetic Ketoacidosis, a fatal complication of DM, is triad of hyperglycemia (>250 mg/dL), increased serum ketones and high anion-gap acidosis > 10. Recent concerns pertaining to the risk of developing DKA in type 2 DM patients with the use of SGLT2 inhibitors led to FDA issuing a drug safety warning in 2015. The etiology of SGLT2 inhibitor associated DKA is multifactorial. Low levels of insulin leading to insufficient suppression of ketogenesis, SGLT2 inhibitor promoted glucagon secretion and SGLT inhibitor mediated decreased urinary excretion of ketone bodies are some of the commonly proposed mechanisms. Our patient had a blood glucose level of 409 mg/dL at the time of admission, but many cases of DKA related to SGLT2 inhibitors have been reported in patients with blood glucose levels less than 250 mg/dL. Physicians must be cautious as euglycemia could be misleading resulting in delayed diagnosis of DKA. The treatment for SGLT2 inhibitor associated DKA is discontinuing the drug along with the standard DKA management protocol. Three cases have been reported with prolonged hyperglycosuria even after discontinuing SGLT2 inhibitors. Hence, clinicians must be aware of the persistent effect of SGLT2 inhibitors beyond their expected half-life, which can complicate the management of DKA.

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NY-Presbyterian Brooklyn Methodist Hospital, Internal Medicine Department
Removal of Partially Covered Esophageal Metal Stents Using Argon Plasma Coagulation

Introduction
Esophageal stents have utility in temporizing esophageal leaks and benign or malignant strictures, but their removal can be complicated by tissue embedment which requires special techniques. Two commonly used methods are argon plasma coagulation induction of tissue necrosis (APC) and stent-in-stent technique (SIS). APC is a non-contact thermal technology that uses argon gas to deliver plasma of thermal energy to an area adjacent to the probe, and it is widely used as a method to control bleeding. APC can be set at a precise setting to limit necrosis to the superficial layer of the esophageal mucosa, freeing an embedded esophageal stent. On the other hand, SIS utilizes a second esophageal stent that is placed within the first esophageal stent to induce necrosis of embedded tissue, enabling removal of both stents.

Case Presentations
We present two cases with successful removal of embedded, partially-covered self-expandable esophageal metal stents (SEMS) using APC at a precise setting. One case showed the flexibility of APC in removal of a migrated partially-covered SEMS in a patient with metastatic esophageal cancer. The stent had migrated into the stomach with the distal tip eroding the gastric mucosa, but the proximal end of the stent had embedded itself into the esophageal mucosa. Given the difficult positioning, SIS was not possible. APC was used on the area of tissue embedment to dislodge the stent with subsequent removal on repeat endoscopy. The second case demonstrated the effectiveness of APC in removal of a partially-covered SEMS placed for an esophageal leak after sleeve gastrectomy. A partially-covered SEMS was chosen to minimize the risk of migration with expected embedment of the esophageal stent into the esophageal mucosa. After the esophageal leak had resolved, planned removal of the esophageal stent was performed with APC. On repeat endoscopy, the esophageal stent was easily dislodged from the esophageal mucosa. Minimal bleeding was stopped with hemostasis clips.

Discussion
We propose that APC is a more effective esophageal stent removal technique compared to the SIS. APC is more readily available with decreased cost and complications compared to the SIS, which requires placement of a second esophageal stent. Placing an esophageal stent within another can be complicated by incomplete induction of necrosis in embedded tissue and severe pain requiring early reintervention. APC and SIS both require at least two separate days of endoscopy, but APC stent removal in our study has only needed an interval time of 2-4 days versus median time of 9 days for SIS described in one case series. Given the small sample sizes, further investigation is needed to determine the optimal technique for esophageal stent removal.
Lithium for the Treatment of Amiodarone Induced Thyrotoxicosis

Lithium for the Treatment of Amiodarone Induced thyrotoxicosis: A case study

M.Chhetry, M.D.[1], J.Harewood, M.D.[2], Siu, Ma,M.D.[3]
1. Resident, Internal Medicine, NYPQ 2.Department of Endocrinology, NYPQ

Background: Amiodarone is a potent class III anti-arrhythmic drug which possesses both beta-blocking and myocardial potassium channels blocking properties. Amiodarone is used for the management of various tachy-arrhythmias and to a lesser extent, in the management of severe Congestive Heart failure. It is very rich in iodine, with a 100mg tablet containing an amount of iodine that is 250 times the recommended daily iodine requirement [1]. Hyperthyroidism is more common with Amiodarone; while Amiodarone induced thyrotoxicosis (AIT) is less common. AIT I is caused by increased synthesis and release of thyroid hormones while, AIT 2 is a due induced destructive thyroiditis. We present a case of Lithium for the treatment of AIT.

Case presentation: A 30 year old male with Traumatic Brain Injury, aphasia, and hemiplegia, HTN, Ventricular Tachycardia and recently diagnosed Hyperthyroidism presented with hypoxia and tachycardia and was admitted for Pneumonia. He was on home dose of Methimazole 15mg/day but TFTs were overactive on admission. His initial TSH was less than .005, T3 of >2.200, and T4 of >16.40. Amiodarone was stopped, Methimazole was increased to 20mg q6 with no improvement in TFTs; Hydrocortisone was started as 100mg q8. TFTs remain unchanged. Being a poor surgical candidate he was started on Lithium for AIT. He was started on Lithium 100q8. Patient was lost to follow up initially however he eventually had repeat TFT’s 4 months after starting Lithium, which showed hypothyroid with TSH of 35.

Discussion: AIT type I is usually treated with large doses of antithyroid drugs while AIT type II is treated with prednisone. Lithium is not routinely used for the treatment of AIT, only in cases where other measures fail. Lithium carbonate has been used since 1948 to treat manic-depressive states [2], but it was not until the late 1960s and early 1970s that hyperthyroidism and goiter were noted as side effects of long-term use of this medication [3, 4]. However there are no randomized trials or guidelines for its use [5]. Lithium has been shown to inhibit iodine uptake, interfere with tyrosine iodination, change the thyroglobulin structure, and interfere with iodotyrosine synthesis [6].

There are a very few case and one small controlled trials reviewing Lithium for AIT, involving 21 patients, the group receiving Lithium normalized TFTs earlier then only using antithyroid drugs [7]. In one study of Graves’ thyrotoxicosis, lithium (800 to 1200mg daily), serum T4 and T3 levels fell by 85% and most patients became clinically euthyroid within 2 weeks of treatment [8].

Introduction:
Gastroperforations related to PUD has declined significantly due to wide use of H2 blockers, PPIs and eradication of H. Pylori. Perforations complicates 2 to 10 percent of the patients with peptic ulcer disease. The absolute frequency of penetration into adjacent organs by PUD is unknown because it can only be reliably diagnosed by surgery or endoscopic biopsy which reveals the tissue of an adjacent organ.

Case Report:
76 yo woman who presented with complaints several month history of loss of appetite, nausea, vomiting/regurgitation of food, significant weight loss of about 80 pounds in 2-4 months. She also reports that her stools have become intermittently black, but denied BRBPR. She has never had EGD or colonoscopy in the past. She also reports early satiety, reflux symptoms, but denied odynophagia or dysphagia. She did endorse vague epigastric abdominal pain in band-like fashion. Initial abdominal film showed multiple nonspecific air fluid levels without evidence of bowel dilatation, CT abdomen/pelvis without contrast showed questionable gastric wall thickening cannot suspicious for mass vs infection, nonspecific mesenteric edema was also noted. Gastroenterology was consulted and decision was made to perform upper endoscopy which showed a deformed antrum with nodular tissue at the base of a giant ulcer, malignant- appearing tumor in the antrum which was biopsied. Histopathology revealed normal liver tissue. Possible etiology for this mass was thought to be perforated peptic ulcer that self-contained and walled-off with the liver vs possible penetration of the liver by PUD.

Patient evaluated by surgery who recommended conservative approach initially and subtotal gastrectomy eventually when patient continued to have poor oral intake tolerance, but patient decided against surgical intervention.

Discussion
Our case is a rare case of gastric ulcer that appeared malignant endoscopically along with positive red flags of early satiety loss of appetite, significant weight loss but histologically found to be normal liver tissue. In general, gastric ulcer perforation being walled-off by liver or penetration into the liver by a peptic ulcer is not common. There are few reports of liver penetration by gastric ulcer diagnosed by endoscopy however we did not find any cases of gastric perforation being walled-off by liver. Most of the publish cases of PUD perforation were found to have usual risk factors of either NSAID use, H. Pylori or anastomoses from previous surgeries which we should always look for to establish the etiology but our patient had no such risk factors.
## Resident/ Fellow Clinical Vignette

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<th>Melissa Cohen MD</th>
<th>Anusha Devarajan MBBS</th>
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<td>Rakhi Rubinova, D.O. Sohi Ashraf, M.D. Sarun Thomas, D.O. Peter Spiegler, M.D. NYU Winthrop University Hospital <strong>INSULIN PLUS PLASMAPHERESIS FOR TRIGLYCERIDE INDUCED PANCREATITIS</strong></td>
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**Case Presentation**

43-year-old male with a history of hypertension, hypothyroidism, recently diagnosed Type 2 diabetes mellitus (T2DM), and obstructive sleep apnea presented to the Winthrop University Hospital Emergency Department with left upper quadrant abdominal pain, nausea, vomiting and a glucose level over 500mg/dL. He complained of blurry vision, thirst, and increased urinary frequency. Laboratory results were remarkable for: serum Sodium of 128 mg/dl, calcium of 6 mg/dl, creatinine of 2.0 mg/dl, venous lactate of 4.7 mmol/L, HbA1c of 11.6%, severe hypertriglyceridemia (SHTG) with triglyceride (TG) level > 5000 mg/dL, and ketoacidosis. Abdominal computed tomography was positive for "peripancreatic inflammation", suggestive of acute pancreatitis. The patient required acute critical care and was started on an insulin drip. Ranson’s criteria calculated an estimated mortality of 100% after 48 hours. Plasma apheresis was initiated on day 2, for a worsening clinical course. After two rounds of apheresis TG levels decreased from 5115 to 541mg/dL. His condition improved with aggressive fluid resuscitation, electrolyte repletion, insulin drip, and apheresis. He was discharged home on insulin and oral hypertyrighceridemia treatment.

**Literature Review**

No definitive guidelines for treatment of triglyceride induced pancreatitis exist. Plasmapheresis alone, or in conjunction with traditional insulin therapy, has been proposed. Plasmapheresis compared to insulin demonstrated a triglyceride level reduction of 65-70%, in a retrospective study by Lennertz. Studies have documented successful reduction of TG levels (less than 500mg/dL) with continuous insulin infusion initiated at 0.1-0.5 units/hr/kg, with adjustments to maintain normoglycemia.

**Clinical Significance**

Acute triglyceride induced pancreatitis results in a prolonged hospital stay and a high mortality rate. Overall mortality in hospitalized patients is about 10% and up to 30% in a subset of patients. Our case highlights the prompt treatment of SHTG induced acute pancreatitis in newly diagnosed T2DM. Insulin drip plus apheresis drastically improved the patient’s triglyceride levels and clinical course.

**Research Question**

Controversy exists for the treatment of TG induced pancreatitis. Insulin drip is mainstay of therapy. Apheresis is a potential treatment option. Is one treatment more effective? Should they be used in conjunction? Does combined therapy reduce hospital length of stay?

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<th>Dr. Marc El Khoury</th>
<th><strong>EPSTEIN BARR VIRUS RELATED HEMOPHAGOCYTIC LYMPHOHISTIOCYTOSIS : THE CYTOKINE STORM</strong></th>
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**Introduction**

Hemophagocytic Lymphohistiocytosis (HLH) is a rare, but life-threatening disease caused by excessive immune activation. HLH can be either familial or secondary to infections, malignancies, drugs and rheumatic disorders. Clinical presentation is variable and non-specific. We present here a case of HLH triggered by Epstein Barr virus (EBV) infection.

**Case Presentation**

A 19 year old man with no recent travel history was admitted to our hospital with onset of high grade- fever, chills, fatigue, headaches and abdominal pain since 3 days. He reported a recent EBV infection 4 weeks ago. Physical examination showed a temperature over 39?C, tachycardia, hypotension and mild confusion. His WBC was 2000 cells /dL, with an absolute neutrophil count of 900. Hemoglobin 8.6 g/dL, platelets 65000/dL, ALT 64 U/L and LDH 348 U / L. A computed tomography scan of the abdomen showed splenomegaly. Blood parasite, buffy coat smears, HIV antibodies, CMV serology and blood cultures were negative. Given the lab work up and clinical presentation, few weeks after his recent EBV infection, there was a suspicion for HLH. On day 2, a bone marrow biopsy was done and did not show hemophagocytosis. Ferritin level was 5875 ug/L, Fibrinogen 144mg/dL. EBV viral load was 426000 copies/ mL. Interleukin 2 receptor (CD25) was 14690 pg/ mL. Dexamethasone with weekly Rituximab were started. Patient symptoms resolved by Day3 and all cell counts improved significantly by Day 9.

**Discussion**

HLH results in excessive release of cytokines, tissue infiltration of histiocytes and lymphocytes and multiorgan failure. One in 800,000 persons per year develops HLH; 90% acquired and a third related to EBV infection. The diagnosis is established if 5 out of the following 8 diagnostic criteria are present; Fever; Splenomegaly; Cytopenia ( 2 lineages); Hypertriglyceridemia or hypofibrinogenemia; Hemophagocytosis in bone marrow, spleen or lymph nodes, Elevated Ferritin >500mg/L; IL-2 receptor >2,400 U/ mL, Low NK- cell activity. Our patient had met 6 criteria and was EBV related, given the recent infection and current high viral load. Presence of hemophagocytosis on bone marrow has a sensitivity of 80% and a specificity of 60%, thus its absence does not exclude the diagnosis of HLH which can be negative in the initial stages of the disease. Other infectious and non-infectious etiologies should be considered and excluded. In EBV related HLH, rituximab in conjunction with standard treatment has been recently shown to significantly improve clinical outcome by depleting the B-cells and halting inappropriate EBV activation.

**Conclusion**

Acquired EBV related HLH is a frequently fatal disease. If left untreated the median survival is estimated to be less than 2 months, with an over-all mortality up to 75%. Therefore early diagnosis with recognition of the triggering factor and prompt therapy can prevent progression to fulminant disease.
Resident/ Fellow Clinical Vignette

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Lenox Hill Hospital

Pseudohypobcarbocarbonatemia in Monoclonal Gammopathy

Accurate estimations of serum bicarbonate levels, as determined by direct measurement or calculation, are essential in the assessment of acid-base disorders. We present a case where direct measured bicarbonate was inaccurate due to an artifact caused by a paraprotein. The rarity of its occurrence can lead to diagnostic challenges in the evaluation of acid-base disturbances.

An 82-year-old female with a past medical history of congestive heart failure, atrial fibrillation, diabetes mellitus, and coronary artery disease presented to the emergency department with the chief complaint of dyspnea on exertion and worsening lower extremity edema. On examination, she was in no acute distress with slight tachypnea, bilateral basilar rales, and pitting edema in both lower extremities. Chest x-ray was significant for cardiomegaly and small pleural effusions. These findings were consistent with the diagnosis of acute exacerbation of congestive heart failure. However, the emergency room physician received a call from the laboratory to report a critical result: initial basic metabolic panel (BMP) returned with a calculated bicarbonate value of 24 mmol/L. Arterial blood gas, however, revealed a pH of 7.49, pCO2 33 mmHg, with a calculated bicarbonate value of 2 mmol/L. Additional laboratory results were unremarkable. Repeat BMP drawn in the emergency department confirmed a serum bicarbonate level of 2 mmol/L. Arterial blood gas, however, revealed a pH of 7.49, pCO2 33 mmHg, with a calculated bicarbonate level of 2 mmol/L

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Das, D, Sayeed, I, Chaudhry, S, Tsaur, Jy, Conetta, R, Beekman, K
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Persistent Hypotension Following Dantrolene Use and a Potential Reversal Agent

ABSTRACT
Dantrolene sodium is essential in treatment of malignant hyperthermia. Dantrolene acts on skeletal muscle cells to reduce the release of calcium from the sarcoplasmic reticulum via interaction with ryanodine receptor channels. It is believed to have minimal effect on smooth and cardiac muscle. However, recent studies have suggested that dantrolene causes vasoconstriction with resultant hypotension. We present a case in which a patient treated with dantrolene developed refractory hypotension which transiently improved with calcium gluconate.

CASE PRESENTATION
A 49-year-old male presented with altered mental status, fever of 105°F, and sinus tachycardia. Blood pressure was 110/65 mmHg, respiratory rate 44 bpm, and oxygen saturation 92% on 100% non-rebreather mask. On physical examination he was comatose and diaphoretic with generalized muscle spasticity. The patient was intubated, with succinylcholine used as the paralytic agent. Ice packs, cooling blankets, and targeted temperature management were applied, however core body temperature rose to 109.4°F intravenously. Dantrolene was administered, initially decreasing core body temperature to 101.8°F. Continuous monitoring of rectal temperature showed core body temperature increase to 106°F five hours later. Subsequently a higher dose of dantrolene was given after which the patient became hypotensive with systolic blood pressure ranging from 60-80 mmHg. Despite fluid resuscitation along with initiation of norepinephrine infusion, patient remained hypotensive prompting initiation of phenylephrine and vasopressin infusions without improvement in blood pressure. Serum chemistry revealed hypocalcemia and calcium gluconate was administered, after which systolic blood pressure immediately improved and pressors were stopped. Core body temperature remained within normal range. However, the patient developed multi-organ failure and disseminated intravascular coagulation and expired five days after presentation.

DISCUSSION:
This case suggests the possibility that dantrolene might have a vasodilatory effect and cause severe persistent hypotension. As presented in this case, calcium supplementation may potentially reverse this fatal adverse effect. Calcium supplementation did not, however, counter the desired effects of dantrolene: normalization of body temperature and reduction of muscle rigidity. Calcium gluconate may therefore potentially be used as an antidote for dantrolene-induced hypotension in the treatment of malignant hyperthermia.
Severe Pancytopenia and a Markedly Elevated LDH: More to Consider than Microangiopathy

Abstract:
This is a case of a 36 year old African American female who presents with generalized weakness and paraesthesia. Laboratory work revealed profound anemia, severe pancytopenia and a markedly elevated lactate dehydrogenase (LDH). Initial clinical presentation indicated possible microangiopathic hemolytic anemia (MAHA). Correlation between the pancytopenia, LDH, and Vitamin B12 levels were key components in ascertaining the diagnosis and avoiding the use of plasmapheresis.

Clinical course:
36 year-old African American female with past medical history of hypertension, iron deficiency anemia, and menorrhagia presented with complaints of generalized weakness, worsening fatigue and peripheral paraesthesias in her extremities. Vital signs showed blood pressure of 130/63 mm Hg, heart rate of 108 bpm, respiratory rate of 19 with 100% oxygen saturation. Physical exam revealed mild scleral icterus, pallor and loss of tongue papillae. No signs of hepatosplenomegaly or petechia. Laboratory work was remarkable for initial pancytopenia (WBC 2.5 cells/mm3, RBC 1.13 cells/mm3 and Platelet 94 cells/mm3), severe anemia (Hemoglobin of 3.7 g/dL, Hematocrit of 10.8 g/dL, Mean Corpuscular Volume of 96 and Red cell Distribution Width of 34.9) and acute kidney injury (Serum Creatinine of 1.2 mg/dL). Further workup revealed elevated liver function test (Indirect bilirubin 1.1 mg/dL, ALT 50 U/L, AST 113 U/L, LDH 4,320 U/L, normal PT/INR and aPTT, haptoglobin <1mg/dL and sedimentation rate 113 mm/hr). Concern for possible MAHA urgent peripheral blood smear was performed revealing the presence of tear drop cells, reticulocytosis, anisopoikilocytosis, and hypersegmented neutrophils. Hematology consult revealed that Vitamin B12 levels were significantly decreased (91 pg/mL) presenting with a pseudo MAHA.

Conclusion:
MAHA is a medical emergency requiring immediate attention as initiating plasmapheresis is crucial to decrease mortality. Here we present a case of Vitamin B12 deficiency presenting with features indicative of pseudo MAHA. Clinically the patient presented with mainly neurological symptoms and labs that revealed intravascular hemolysis with kidney injury. Fever and purpura were absent in this patient. The peripheral smear had no evidence of schistocytes but showed high cellular turnover intravascularly and in the bone marrow hence the elevated LDH. It also showed both microcytosis and macrocytosis for which the RDW was elevated. The normal MCV can be explained by the combination of two types of concurring anemias; iron deficiency anemia from chronic menorrhagia superimposed by Vitamin B12 hypovitaminosis. Thus in this case hemolysis was due to ineffective erythropoiesis secondary to defective DNA synthesis and cell maturation from low Vitamin B12 levels. The patient was started on Vitamin B12 supplementation with rapid correction of her anemia and pancytopenia in less than month. Initial workup was negative for antibodies against intrinsic actor; the cause of her hypovitaminosis remains to be determined.

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IMMUNOGLOBULIN A MEDIATED VASCULITIS IN PATIENTS WITH ALCOHOLIC LIVER CIRRHOSIS: A CASE SERIES

ABSTRACT:
Immunoglobulin A (IgA) mediated vasculitis, also termed as Henoch-Schönlein purpura (HSP), is a small vessel systemic vasculitis, predominantly affecting children, characterized by a tetrad of manifestations, specifically palpable purpura, arthralgia, abdominal pain, and renal disease. Very few cases have been reported of HSP with liver cirrhosis in adults. We present two cases with alcoholic liver cirrhosis patients with altered IgA processing leading to the development of IgA immune complexes and ultimately HSP.

CASE 1:
A 43 year old male with history of hepatic cirrhosis consequent to alcohol abuse presented with colicky abdominal pain, associated with watery, bloody diarrhea since past few days. On examination, abdomen was diffusely tender with diffuse petechial rashes. Initial blood tests showed thrombocytopenia and acute kidney injury. CT scan showed pancolitis, hepatic cirrhosis with sequel of portal hypertension. Complete stool work up was negative and antibiotics were discontinued. Later, Immunofixation showed IgA of 1385 mg/dl. Although skin biopsy showed sparse perivascular dermatitis with hemorrhage, patient refused renal biopsy. Steroids were administered with improvement of symptoms, along with improvement of renal function.

CASE 2:
A 46 year old female with alcoholic cirrhosis, chronic venous stasis bilateral one-third lower legs came with complain of dull abdominal pain and leg pain, worsening over the past few weeks. A tender 4-5 cm hyper pigmented rash was present on left leg. Laboratory findings were significant for anemia, thrombocytopenia and acute kidney injury. Urinalysis showed proteinuria with gross hematuria. Given worsening renal function, renal biopsy was done. Immunofluorescence microscopy revealed granular global mesangial and glomerular capillary wall immune deposits which stain IgA. Serum IgA levels was 1794 mg/dl. Patient was started on oral steroids with improvement of symptoms and renal function.

DISCUSSION:
We came across many case reports HSP associated with Hepatitis C but liver cirrhosis in itself possess a significant risk in development of IgA vasculitis. The role of liver cirrhosis in the development of HSP is intriguing since this patient’s chronic liver disease may have precipitated the development of HSP with defective liver metabolism of IgA circulating immune complexes, leading to deposition in the skin and kidneys. Our first patient had received short course of steroids with almost complete resolution of symptoms and significant increase of platelets counts. In our second patient, due to worsening renal function, initial suspicion for hepatorenal syndrome was made but patient did not respond to albumin and diuretics. Kidney biopsy was the key lead here.

CONCLUSION:
The diagnosis of HSP/ IgA vasculitis can be easily missed. A high degree of suspicion and requesting immunofluorescence studies in suspected cases are mandatory to establishing the diagnosis. Skin biopsy and immunofluorescence confirms the presence of IgA deposition which is the pathognomonic finding in HSP.
Case presentation:
A 69 year old man with a history of ST segment elevation myocardial infarction status-post left circumflex artery stenting and lung cancer presented with seven days’ history of severe intermittent substernal chest pain. Five months ago He failed platinum based chemotherapy for lung cancer therefore was started on nivolumab and ipilimumab. The last infusion was six days prior to his symptoms. His electrocardiograms revealed new prominent ST segment elevation in II, III, avF, V4~V6 leads, with serum troponin of 10.4 ng/mL. Unexpectedly, the emergent cardiac catheterization demonstrated a patent stent and minimal occlusion to distal right coronary artery, with unchanged ejection fraction of 40 percent. Initially there was a spontaneous improvement in chest pain however it recurred three days later after receiving next dose of nivolumab, with similar electrocardiographic changes and Troponin of 2.1 ng/mL. At this point checkpoint inhibitor induced myocarditis was suspected. A regimen of colchicine and methylprednisolone with a single dose of infliximab infusion. Eventually, the pain subsided after high dose steroid was started but unfortunately his pain persisted. Eventually, the pain subsided after high dose methylprednisolone with a single dose of infliximab infusion. Patient remained chest pain free five weeks after discharge and his electrocardiograms as well as serum cardiac biomarker abnormalities all resolved by then.

Discussion:
Incidence of immunotherapy related cardiotoxicity remains low affecting less than 1 percent of patients, the exact mechanism is still unknown. Current main treatment approaches are based on experience of existing autoimmune diseases. Myocarditis usually responds well to high dose glucocorticoid, however death was also reported regardless of aggressive treatment. In our case, there was a rapid and almost complete resolution after infliximab with high dose steroid. To the best of our knowledge, this is the first case of combined immune blockade induced myocarditis that presented with dramatic ST segment changes. And the patient was successfully treated with above regimen.
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ENDOBRONCHIAL LEIOMYOMA, A RARE AND BENIGN TUMOR OF BRONCHIAL TREE, MAY CAUSE WHEEZE IN ASTHMATIC PATIENT

Introduction: We are all taught that "all that wheezes is not asthma." This useful mantra is typically applied to patients without a history of wheezing and expands the important differential diagnosis (Table 1). Asthma patients may present with a change in wheezing pattern and should be considered for alternative diagnosable and treatable mimics of asthma. We present an asthmatic case who had a change in pattern before being diagnosed with endobronchial leiomyoma. Primary endobronchial leiomyoma is a rare tumor of the bronchial tree (Table 2) and represents less than 2% of all benign tumors of the lung.

Case Description: An obese 47 years old longstanding asthmatic caucasian male presented with complain of dyspnea on exertion and change in wheeze that began intermittently about one year ago. Medical history was significant for Hyperlipidemia and Asthma; family history significant for emphysema in his grandfather who had shipyard exposure. Patient never smoked but has history of secondary exposure.

Physical examination was benign, except for wheezing; further workup revealed normal CXR, echocardiogram, and exercise nuclear stress test.

Patient was initially diagnosed with seasonal allergy and asthma exacerbation and treated with Singular, Advair, and desensitization therapy with improvement in dyspnea but exercise capacity did not improve.

CT of the chest reveals 1.9 x 1.4 cm soft tissue mass in the left main stem bronchus (figure 1). Fiber optic microscopy with biopsy showed a mass seen immediately at the takeoff of the left main stem bronchus along the membranous portion of the distal trachea (figure 2). Grossly the lesion was large, smooth, with visible blood vessels, reminiscent of a carcinoid tumor. Brushings were obtained and negative for malignant cells.

Biopsy of the lesion demonstrated a leiomyoma (figure 3). The patient underwent flexible bronchoscopic right thoracotomy sleeve resection of the left mainstem as a definite therapy for leiomyoma. The final pathology report confirmed leiomyoma.

Discussion: Leiomyoma of the lung is a rare tumor that can present anywhere along the tracheobronchial tree or within the lung parenchyma (Table 2). Clinical presentation depends on the site, size, and the lung changes distal to the lesion. Symptoms are related to partial or complete obstruction of the affected bronchus. Symptoms may include wheeze, hemoptysis, fever, pleural effusion, lung collapse, recurrent pneumonia, and subsequent bronchietasis. Definitive diagnosis is made via bronchoscopy to visualize and biopsy for histopathologic analysis. Management should be conservative, surgical resection is the mainstay of treatment in symptomatic patients. The adage "all that wheezes is not asthma" should also apply to patients with a previous history of asthma. The recent Clinical Problem Solving article in the NEJM of August 3, 2017 highlights this message, where a patient with asthma evolved to have eosinophilic polyangiitis. (Table 3).

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LATE ONSET COMMON VARIABLE IMMUNODEFICIENCY: A CASE REPORT

Introduction:

Common Variable Immunodeficiency (CVID) is a form of primary immunodeficiency encompassing a group of heterogeneous disorders resulting in a failure of antibody production. Patients present with recurrent sino-pulmonary infections. Diagnostic evaluation reveals marked reduction in serum immunoglobulin G (IgG) levels along with low levels of Immunoglobulin A (IgA) and/or immunoglobulin M (IgM) levels. A majority of patients are diagnosed between 20-45 years of age. We present a case of late onset CVID who presented at the age of 75.

Case Report:

A 75-year-old gentleman with past medical history of CAD, hypertension, and hyperthyroidism presented to the pulmonology clinic with seven months of productive cough and 25lb unintended weight loss. Cough had been treated with multiple courses of antibiotics and a course of systemic steroids with partial relief. The patient had a remote smoking history, but quit 47 years ago. He recalled being diagnosed with outpatient pneumonia twice in his life, and each episode was treated with antibiotics. He denied a history of recurrent sinusitis or chronic diarrhea.

Pulmonary function testing revealed a modest obstructive pattern with significant bronchodilator response. A CT chest showed bilateral lower lobe mucus impaction/peribronchial thickening and enlarged mediastinal and upper abdominal lymphadenopathy. The patient was treated for asthma with fluticasone but experienced minimal improvement. A bronchoscopy was performed, and cultures from the bronchoalveolar lavage grew Moraxella catarhais. A 10 day course of moxifloxacin resulted in some symptomatic improvement.

A repeat CT chest showed worsening multifocal pneumonia. Sputum culture grew pan-sensitive Pseudomonas. The patient was treated with a 10 day course of levoflaxacin but experienced a recurrence of symptoms and was started on inhaled gentamycin.

Given the persistent multi-lobar pneumonia, he was evaluated for immunodeficiency and found to have IgG < 70 mg/dL, IgM < 8 mg/dL, IgA < 18 mg/dL, and IgE of 2 IU/mL CD19 = 6% (8 ±1%), or 75/mm3 (111 :480 /mm3). CD4 and CD8 were normal. A bone marrow biopsy showed no evidence of malignancy and revealed decreased B cell numbers.

The patient was started on subcutaneous immune globulin replacement therapy and had an IgG level of 580 mg/dL by the 3rd month of therapy. He reported symptom improvement and repeat sputum cultures were negative. A repeat CT chest showed near-complete resolution of pneumonia.

Conclusion:

CVID affects up to 1 in 10,000 individuals and typically presents with infectious complications in teenagers and young adults. CVID is underrecognized and underdiagnosed, with a significant delay in diagnosis. This case serves as a reminder that CVID may present at any age, even late adulthood. Additionally, it highlights the importance of screening for immunodeficiency in patients with an appropriate clinical history of recurrent or unusual infections.
Introduction: Temozolomide (TMZ) is an oral alkylating agent indicated for treatment of adult patients with glioblastoma multiforme (GBM) as concurrent treatment with radiotherapy and adjuvant treatment. In the landmark trial that proved efficacy of TMZ, a total of 6 cycles were given, although often recommended more than 6 cycles in practice. While generally well-tolerated, myelosuppression is a relatively common side effect of TMZ, with a nadir typically 21-28 days after administration and recovery within 14 days. Here we report a rare case of late-onset aplastic anemia following the use of TMZ.

Case: The patient is a 63-year-old female originally diagnosed with GBM, IDH-wildtype of the left temporal lobe in June 2015. She underwent a left sided craniotomy and tumor excision, followed by concurrent chemoradiation with TMZ and completed 42 days of treatment. Her tumor was found to have MGMT promoter methylation, which carries a more favorable prognosis with higher response to TMZ. She participated in a clinical trial randomizing patients to veliparib or placebo in combination with adjuvant TMZ for newly diagnosed GBM with MGMT promoter hypermethylation. She was noted to have recurrence in the surgical cavity on MRI in January 2016 and underwent tumor excision. Of note, the pathology showed residual glioma but substantially reduced cellularity when compared to the original resection, with significant reduction of Ki-67 from >30% to 2-4%. Given the substantial treatment response on the surgical pathology, adjuvant TMZ was continued and given for a total of 11 cycles (off the study regimen as per the study protocol).

Her last TMZ cycle was administered approximately 6 months prior to this admission, when she presented to us with right-sided weakness and was found to have another recurrence of GBM. Her stay was complicated by persistent pancytopenia requiring multiple transfusions. All potentially myelotoxic agents were discontinued and infectious causes of aplastic anemia were excluded. The patient underwent a bone-marrow biopsy which revealed a hypocellular marrow with minimal hematopoiesis, consistent with aplastic anemia.

Discussion: TMZ is an oral alkylating agent and mild thrombocytopenia is common. However, severe myelosuppression involving all cell lines seems much less common. Myelosuppressive effects occur during each treatment cycle and normally recover prior to next cycle. The current literature shows rare reported cases of aplastic anemia attributed to TMZ use, however, these have typically occurred early in a patients’ treatment. Here we show our patient developed aplastic anemia well after the conclusion of 11 cycles of TMZ. Clinical awareness should be heightened to patients who are fortunate to have a prolonged survival.

Conclusion: Patients with a history of prolonged use of TMZ may be at increased risk for the development of aplastic anemia and this should be considered in the correct clinical setting.
A RARE CASE OF COMPLETE HEART BLOCK IN AORTIC DISSECTION

Introduction:
Aortic dissection is a relatively uncommon disease that can have a catastrophic presentation with hemodynamic instability. It typically presents with chest pain without any specific EKG changes. We present a case of type B aortic dissection presenting with complete heart block.

Case:
A 53-year-old man with hypertension (non-compliant with medications), presented with sudden-onset chest and thoracic back pain, associated with nausea, vomiting, diaphoresis, and dyspnea. His blood pressure was 197/140 mmHg with heart rate of 62/minute. Initial EKG revealed normal sinus rhythm with non-specific ST wave changes. Due to underlying kidney disease, emergent non-contrast magnetic resonance angiogram was performed, which revealed type B aortic dissection. Labs revealed normal hematocrit and worsening creatinine. He became hypotensive after one hour, with repeat EKG revealing complete heart block. Despite transcutaneous pacing, he became asystolic. ACLS protocol was initiated, but he could not be revived despite maximal efforts.

Discussion:
Aortic dissection is relatively uncommon, with an estimated incidence of 2.6 to 3.5 per 100,000 person years, most common in men aged 60 to 80. Stanford classification classifies dissections involving the ascending aorta as type A, regardless of the site of the primary intimal tear, and all other dissections as type B. The most important predisposing factor of acute aortic dissection is systemic hypertension, which is present in 25-35% of patients with type A and 70% of patients with type B dissections.

Patients with type A dissections typically present with anterior chest pain, while patients with type B dissection typically present with back pain. Pain can be an isolated symptom, or may be associated with syncope, symptoms of stroke, myocardial infarction, heart failure, or other clinical signs of end-organ ischemia. EKG is often non-specific. However, it is useful to rule out myocardial ischemia/infarction, which can occur when the coronary arteries are affected. Few small studies have shown that hematoma spreading into the atriocentricus junctional tissues can lead to various degrees of AV block. However, some believe that long-standing, uncontrolled hypertension causes fibrosis of the conduction system, which leads to AV block especially in type B aortic dissection. Syncpe following acute dissection may be due to the rapid progression to complete heart block without allowing the ventricular escape rhythm to take over. Thus, continuous EKG monitoring is important in patients with suspected aortic dissection, as they may rapidly decompensate into complete heart block and asystole.

Learning objectives:
- Aortic dissection can present rarely with heart block
- Continuous cardiac monitoring is important in patients with aortic dissection

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**AORTIC DISSECTION**

**Gangrenous Cholecystitis Secondary to Extension of Celiac Artery Dissection into Hepatic Artery**

A 58-year-old man with a history of hypertension and hyperlipidemia presented to the hospital complaining of four days of band-like lower abdominal pain. The pain began suddenly and felt like a seatbelt tightening around his waist. Upon arrival to the emergency department, the patient was afebrile and mildly hypertensive. Physical exam revealed moderate tenderness to palpation across his lower abdomen, without guarding or abdominal distension. Laboratory tests showed a normal WBC count, serum and liver chemistries, lipase, urinalysis, lactic acid, ESR, and CRP. Patient underwent urgent CT aorta, which revealed wall thickening of the celiac trunk, common hepatic, and proper hepatic arteries with intraluminal stenosis along the common hepatic artery. A 1.3 cm pseudoaneurysm was noted at the trifurcation of the celiac trunk. Patient’s abdominal pain continued to worsen, localizing in the right upper quadrant. Liver tests showed rising values with AST/ALT 368/339, Alk Phos 153, GGT 290, and total/direct bilirubin 2.0/1.0, while other labwork remained normal. Abdominal ultrasound revealed new gallbladder wall thickening concerning for subacute ischemia and 70% stenosis in the celiac trunk with extension of celiac artery dissection into the hepatic artery, without evidence of infarction, stones, or sludge. The spleen, liver, and pancreas were unremarkable. HIDA scan was negative for cholecystitis but MRI/MRCP revealed a significantly distended gallbladder with wall thickening and pericholecystic edema. Anticoagulation with heparin drip was initiated for management of arterial dissection and the patient was started on a morphine PCA to control his unrelenting pain. The next day the patient was sent for an exploratory laparoscopy, which demonstrated gangrenous cholecystitis and he underwent urgent cholecystectomy. He reported almost complete resolution of pain in the immediate post-op period and the morphine PCA was discontinued. After overnight observation, he was discharged home with therapeutic enoxaparin and with outpatient follow-up to complete evaluation for possible large vessel vasculitis or underlying connective tissue disorder.

This case is the first known report of gangrenous cholecystitis caused by a spontaneous celiac artery dissection. Although isolated spontaneous celiac artery dissection is an extremely rare phenomenon in the absence of trauma, the case shows it should be considered in the differential diagnosis of acute abdominal pain.
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PREGNANT OR PARANEOPlastic: BETA-HUMAN CHORIONIC GONADOTROPIN AS THE FIRST CLUE TO COLON CANCER.

Introduction:
Serum beta-human chorionic gonadotropin (hCG) is an indicator of pregnancy. Ectopic hCG secretion has been found to be elevated in different gynecologic and non-gynecologic tumors ranging from lung cancer to osteosarcoma. The free beta subunit is produced not just by gestational trophoblastic cancers but also by approximately 16% of colorectal cancers. Here we present a young female with a positive ß-hCG marker who was subsequently diagnosed with colon cancer.

Case Presentation:
A 34 year old multiparous female with a history of hyperthyroidism presented with worsening right upper quadrant abdominal pain for 3 weeks. She also reported night sweats, bloating and painless bright red blood in the stool. She was sexually active and had an intrauterine contraceptive device. Physical examination was significant for right upper quadrant tenderness and hepatomegaly. There was no leukocytosis but her labs revealed a mild transaminitis and an elevated hCG of 1954IU/L. Ectopic pregnancy was suspected due to the elevated hCG in the presence of an intrauterine contraceptive device and a negative transvaginal ultrasound. Oral methotrexate was administered but hCG levels remained persistently high. Further work-up was done including a CT abdomen which revealed hepatomegaly with extensive metastatic disease, ascites and thickening of the proximal sigmoid colon. A liver biopsy was taken and colonoscopy showed a sigmoid mass which was biopsied. The patient was to follow up with oncology for the results but returned to the gynecologist with worsening abdominal pain and ascites. The hCG level had increased to 3023IU/L and a dilation and curettage was being considered

Discussion:
This case illustrates the example of a rare paraneoplastic syndrome masking the symptoms of a serious disease behind the innocuous guise of a positive pregnancy marker. Although the positive hCG pointed towards an ectopic pregnancy, the history of blood in the stool, upper abdominal pain, hepatomegaly and a lack of response to methotrexate led us to investigate further. This case highlights the importance of considering gastrointestinal malignancy as a differential for elevated hCG in cases where the clinical scenario is not consistent with pregnancy or gynecologic diseases.

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Paroxysmal Nocturnal Hemoglobinuria in Aplastic Anemia

Paroxysmal Nocturnal Hemoglobinuria in Aplastic Anemia
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Case Presentation:
A 70 year old male presents with two weeks of fatigue, dyspnea, and jaundice. He has a history of aplastic anemia (AA) with intermittent treatment adherence.

In the months prior to presentation, he experienced episodes of fatigue and dyspnea in the Dominican Republic treated with red blood cell (RBC) transfusion with symptomatic improvement. His symptoms gradually worsened, prompting his current presentation. Physical exam was notable for jaundice. Labs were significant for hemoglobin of 6.3 g/dL, elevated LDH, low haptoglobin, and low reticulocyte index. Urinalysis showed large blood, but only 2-5 RBCs/hpf. Coombs Test was negative. He was restarted on cyclosporine for AA, however, he then had hematuria with one E-GebackâŒŒ urine on morning void and labs consistent with worsening hemolysis. Flow cytometry showed a large paroxysmal nocturnal hemoglobinuria (PNH) clone; his hematuria was thought to be secondary to PNH exacerbation after AA treatment. Cyclosporine was discontinued and the patient was started on eculizumab for his PNH.

Discussion:
PNH is an intravascular complement-mediated hemolytic anemia and the only hemolytic anemia caused by an acquired RBC cell membrane defect. Its namesake feature, morning hematuria, is thought to be due to increased hemolysis from transient acidemia during sleep. The acquired defect, a PIGA gene mutation, creates RBCs lacking the glycosylphosphatidylinositol (GPI) membrane anchor, causing loss of Complement Delay (CD) proteins CD55 and CD59. These antigen markers normally protect RBCs from complement-mediated hemolysis. Suppression of GPI-positive hematopoietic cells as in AA causes GPI-negative clone expansion and worsening PNH. Free intravascular hemoglobin causes renal toxicity and scavenges nitric oxide, resulting in vasospasm, hypertension, and hypercoagulability with venous or arterial thromboses. Definitive diagnosis is made with flow cytometry for CD55/CD59 and FluorescentAERolysin (FLAER) for direct GPI anchor binding.

Eculizumab is a direct inhibitor of the terminal complement cascade, inhibiting hemolysis in PNH. Eculizumab reduces blood transfusions and increases quality of life without changing mortality or progression of AA to myelodysplastic syndrome and/or acute myeloid leukemia. Eculizumab’s complement suppression increases the risk of N. meningitidis infection, requiring vaccination and/or lifelong penicillin prophylaxis.

Conclusions:
PNH occurs commonly as a sequelae of AA, particularly after immunosuppressive treatment. This rare disorder has multiple clinical manifestations including symptomatic hemorrhagic anemia, hemoglobinuria, free hemoglobin-mediated acute kidney injury, hypertension, and thrombosis. Eculizumab, a terminal complement inhibitor, can be used to treat PNH.
ACIDOSIS IN A STATUS ASTHMATICUS

This is a case of a 29 year old patient that was admitted for status asthmaticus and treated aggressively with albuterol nebulizers in the emergency department. At baseline, prior to nebulizer treatment, she had a normal lactate of 1.7 and a bicarbonate of 22. After receiving multiple rounds of albuterol aerosols prior to admission, a repeat lactic acid and an arterial blood gas were drawn. Results were significant for metabolic acidosis with respiratory alkalosis and a lactic acidosis of 6.6. She became tachycardic to 143bpm with persistent tachypnea while remaining normotensive. The amount of B2 agonist nebulizer treatments, lactic acid levels, and arterial blood gases were trended throughout the hospital course. It was concluded that there was a direct correlation between the quantity of aerosolized albuterol and lactic acid levels. Additionally, albuterol nebulizer frequency reduction paradoxically caused an overall improvement in the patient clinically. Physicians that recognize this phenomenon early can help prevent prolonged hospital stays and improve patient outcomes. There have been many case reports showing parenteral B2 agonists inducing lactic acidosis, but this is one of the few known cases of albuterol nebulizers inducing lactic acidosis.

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A CASE OF ALBUTEROL AEROSOLS INDUCING LACTIC ACIDOSIS IN A STATUS ASTHMATICUS

Introduction:
Estimated number of newborns with sickle cell disease globally will increase from about 300,000 to >400,000 by 2050 according to current projections. Despite hematopoietic stem cell transplant being curative in SCD, its impact on DFS remains unknown, partly due to limited donor options and perceived mortality in adults with myeloablative conditioning. Non-myeloablative and reduced intensity conditioned protocols have made HCT available to adult SCD patients but number of HCT attempts globally is relatively small, primarily due to non-availability of adequate donors, referral bias, procedure risks, costs and limited logistic resources. Study focused on the recent data for HCT for adult patients with SCD with emphasis on comparative outcomes with pediatric patients transplanted for SCD.

Methods:
Scientific literature review did from January-June 2017, contains only adult (>16 years) SCD studies. Outcome measures include: median OS, FTF, mortality (overall and at 100-days, and 1-year post-HCT), TRM, GVHD,PFS, late organ complications, graft rejection with recurrence or persistence of SCD. Found 391 articles out of which 177 were shortlisted and among these only 28 articles met our inclusion criteria.

Results:
GVHD: Out of 28 studies,16 showed minimal incidence of GVHD, while only five studies showed a high incidence of GVHD. Graft Rejection: Found low incidence of graft rejection compared to historical controls in pediatric populations, however, multiple exposures to the blood transfusion prior to HCT and chelation therapy usage for iron overload were found to be associated with increased incidence of graft rejection. Adult HCT studies showed a very low incidence of graft rejection once above-mentioned risk factors had been controlled.
OS: Observed a similar transplant outcome, seen in pediatric HCT for SCD, were the unavailability of a healthy HLA-identical sibling lead to alternate donor transplantation, with associated increase in overall mortality, though no association was found on overall survival in relation to age of patient. Nine studies reported median OS of 100%, and two studies reported 70-80% overall survival. PFS: Sixteen studies revealed an acceptable PFS of more than 90%, whereas four studies revealed PFS of below 90% (87%, 85%, 82%, and 88% respectively) TRM: Observed that TRM has significantly reduced and most optimum outcomes were observed when recipients had good functional status at baseline. The main causes of death in transplanted patients were found to be sepsis, hemorrhage, severe lung injury and CNS hemorrhage with organ failure.

Conclusions:
Clinical data from current studies in our review demonstrates improved outcomes using HCT a curative therapy for adults with SCD. However, lack of suitable HLA-identical sibling and matched unrelated donors severely limits accessibility of HCT therapy for eligible adults with SCD. Encouraging preliminary results with use of umbilical cord blood and haploidentical transplantation for SCD may solve the problem of this rarity.
ARTIFICIAL HYPOGLYCEMIA: WHEN NOT TO PANIC ABOUT HYPOGLYCEMIA.

Introduction
Falsely low point of care glucose levels are rarely seen in patients with poor peripheral circulation. Early clinical recognition will help to avoid complicated testing, thus reducing treatment cost and length of hospital stay.

Case description
87 year old woman with a medical history of coronary artery disease, congestive heart failure, raynaud’s syndrome, sicca syndrome and hyperlipidemia was brought in after choking on food. Her hospital course was complicated by a brief episode of respiratory failure requiring intubation, central line placement, aspiration pneumonia, CHF exacerbation, lower GI bleed, lumbar compression fracture and candidemia. She received IV antibiotics & stress dose steroid course, which was slowly tapered to PO steroid (prednisone 10mg daily). On the third week of hospitalization, she was noted to have multiple fingersticks in 20s without any associated hypoglycemia symptoms such as tachycardia, tremors, irritability or change in mental status. She received treatment of hypoglycemia according to hypoglycemia protocol (dextrose 50gm IV push) during these episodes, but continued to have these episodes. Hypoglycemia was initially contributed to poor oral intake, but other causes including adrenal insufficiency and insulin secreting tumor were considered. On physical examination, she was noted to have cold extremities with cyanosis. Normal blood glucose was noted on the basic metabolic panel done from the same day. Morning cortisol was also checked, which was within normal limit. At this point, possibility for artificial hypoglycemia was considered. On the subsequent episode, venous blood was drawn from the central line before treatment of hypoglycemia and tested with same glucometer. She was found to have POC glucose of 81, thus confirming the diagnosis of pseudo hypoglycemia.

Conclusion
Raynaud’s syndrome can cause falsely low finger stick measurements. This case emphasizes the importance of meeting whipple’s triad to define true hypoglycemia & considering possible causes of artificial hypoglycemia in a patient with asymptomatic low finger stick measurements. Glucose measurement from venous sample would help to confirm diagnosis of artificial hypoglycemia, thus preventing unnecessary work up.

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SPINAL EPIDURAL ABSCESS AS A RARE CLINICAL COMPLICATION OF GROUP A BETA HEMOLYTIC TOXIC SHOCK SYNDROME.

Introduction
Streptococcal Toxic Shock syndrome (sTSS) is an uncommon complication with Group A &ß223; hemolytic streptococci. When present, sTSS has a very high mortality of 30 to 70 percent. Furthermore, group A &ß223; hemolytic streptococcal bact eremia leading to spinal epidural abscess is another rare complication. Only 9 percent of Spinal epidural abscesses (SEA) are caused by streptococcus species. Over the years, SEA are on the rise due to more invasive spinal procedures being performed, as well as, improved diagnosis via the use of magnetic resonance imaging (MRI). This case discusses a sTSS complicated by spinal epidural abscess successfully managed by current treatment guidelines.

Case
A 40 year old African American female with Previous Medical history of Diabetes, Hypertension visited the clinic with acute pharyngitis and was started on azithromycin. Three days later, she returned to the Emergency Department after a fall with a wrist injury and a persistent temperature. Subsequently, she developed Diabetic Ketoacidosis, Shock and Acute Renal failure. Streptococcal bacteremia was isolated and she was diagnosed with sTSS. Initially, a clinical improvement was seen after treatment with appropriate antibiotics. However, she developed paraparesis and was diagnosed with a thoracic spinal epidural abscess after magnetic resonance imaging. She was immediately started on intravenous steroids and antibiotics. Moreover, a surgical decompression with drainage of SEA was performed. After surgery, rehabilitation and a completed course of antibiotics, patient was discharged home. She continues to follow-up in clinic and mobilizes with the help of a walker.

Discussion
When an individual with a new onset bacteremia develops any neurological symptoms of back pain or paraparesis, it is important to obtain a MRI of the entire spine, as well as the cranium. On the other hand, it is also important to start empiric antibiotic therapy in bacteremic patients, which can help to decrease the bacterial load and risk of abscess formation. In patients with severe sepsis, a high index of clinical suspicion is required to diagnose SEA early. Once diagnosed, immediate drainage of the abscess reduces the long term neurologic sequelae and mortality.
Resident/ Fellow Clinical Vignette

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Assessing the link between two inflammatory markers: Helicobacter pylori and microalbuminuria

Helicobacter pylori (H.pylori) infection and microalbuminuria are known inflammatory markers. H.pylori has been shown to be involved in extra-gastric processes, including atherosclerosis and insulin resistance. Microalbuminuria is a predictor of diabetic nephropathy. As a known early marker of atherosclerosis and has also been associated with metabolic syndrome and insulin resistance. It has recently been reported that H.pylori seropositivity may be independently associated with microalbuminuria. As diabetics are at a higher risk for developing atherosclerosis, and if H.pylori infection plays a role in the pathology of atherosclerosis, there may be a possible association between active H.pylori infection and microalbuminuria in diabetics. Retrospective data from 2014 to 2016 was obtained for 500 patients. The study population included previously diagnosed diabetic adults who underwent screening for H.pylori via stool antigen or esophagogastroduodenoscopy with biopsy. Of these subjects, those who had been screened for microalbuminuria within 1 year of being screened for H.pylori were included. The presence of diabetes was defined as HbA1C of ≥6.5% and/or those taking diabetic medications. The presence of microalbuminuria was defined as a urine albumin/creatinine ratio (UACR) of 30 to 300mg/g. Demographic variables such as age, gender, BMI, and ethnicity were also noted. Lipid levels, smoking status, GFR, serum creatinine and the presence of existing coronary artery disease were also measured and adjusted for. Statistical analysis was performed using SAS 9.3 software. Out of 500 patients, 80 were excluded (subjects with ESRD on dialysis and/or with macroalbuminuria). Of the 420 subjects, 21.4% had microalbuminuria and 47.6% had active H.pylori infection. HbA1C, BMI, triglyceride and LDL levels were significantly higher in patients with microalbuminuria and prevalence of H.pylori was significantly higher in subjects with microalbuminuria. After controlling for confounding factors, diabetic patients with microalbuminuria were 5.74 times more likely to have an active H.pylori infection (HR, 5.74, 95% CI, 2.97-11.07, p<0.01). In addition, as UACR increased, the likelihood of H.pylori infection also increased. This study indicates that active H.pylori infection is independently associated with the presence of microalbuminuria; and, that the prevalence of H.pylori infection showed a positive association with the severity of the UACR in diabetics.

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Mesalamine Induced Myocarditis in a Young Athlete. Can he run again?

Background:
Mesalamine containing products are often used in the treatment of Inflammatory Bowel disease (IBD). Cardiotoxicity is rare possible side effect of Mesalamine. We report a case of mesalamine induced myocarditis that was confirmed with Cardiac MRI and resolved after immediate cessation of the medication.

Clinical vignette:
We present a case of a 21 year- old professional football player whose medical history is only significant for a recent diagnosis of Crohn’s disease, for which he was started on Mesalamine daily, four weeks prior to his Emergency Department (ED) presentation. Patient presented to the ED with recurrent intermittent episodes of chest pain over a 24-hour period. He described the chest pain as sharp, sub-ternal pain, 8/10 in severity that started while he was at rest. He encountered 2 self-resolving episodes, each lasted for an hour before he encountered a third prolonged episode prompting him to present to the ED. Patient denied having any shortness of breath, cough, fever, runny nose, watery eyes, or other systemic symptoms prior to his chest pain. He denied smoking or having any family history of heart disease
Work up included an EKG showing non-specific ST-T abnormalities changes with 1st-degree heart block. Cardiac biomarkers were elevated with Troponin I 2.215 ng/ml and CK 220 IU/L. Echocardiogram showed no evidence of wall motion abnormality and an Ejection fraction of 55-60%. Patient’s presentation and lab work up raised the suspicion for Mesalamine induced myocarditis. A decision was then made to perform a confirmatory Cardiac MRI which showed subepicardial to mid-myocardial delayed hyper-enhancement and edema involving the basal inferior to infero-lateral wall, a non-ischemic pattern that is consistent with myocarditis.
Mesalamine was then discontinued, which resulted in subsequent resolution of patient’s chest pain and down-trending of troponins over the following 48 hours.

Discussion:
Mesalamine induced cardiotoxicity has been reported in the literature and is a rare entity. Mechanism is not fully understood. It was proposed that it is due to drug hypersensitivity, which explains why development of symptoms is dose independent and could start early in the course of the treatment or subsequently. Our patient had no symptoms suggestive of viral illness and his symptoms resolved upon discontinuing the medication which makes Mesalamine the most likely cause of his myocarditis. Most Mesalamine induced cardiovascular toxicity cases occurred 2-4 weeks after treatment was started and in most cases symptoms resolved within 1 week of medication discontinuation.
In conclusion, Mesalamine induced cardio toxicity is a rare, yet serious side effect that physicians should be aware of. Patients on Mesalamine who present with chest pain or SOB or concerning cardiovascular complaints should have the medication stopped immediately and receive the appropriate work up to rule out this potentially lethal drug side effect.
Resident/ Fellow Clinical Vignette

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LETHAL BUT REVERSIBLE ENCEPHALOPATHY SECONDARY TO LITHIUM TOXICITY

Lithium is a first line drug for bipolar disorder and is also effective in treating acute mania and major depression. Due to its narrow therapeutic window, the risk of toxicity remains a concern. Clinical manifestations include gastrointestinal, cardiac, renal symptoms, and, in chronic intoxication, development of neurological symptoms. There are several case reports of lithium-associated reversible and irreversible encephalopathy with therapeutic lithium level. Here we present a rare, life-threatening case of lithium-induced reversible encephalopathy at toxic lithium levels of 5.0 mEq/L.

A 43-year-old Caucasian male with psychiatric history of bipolar disorder I was brought in by ambulance in an unresponsive state. He was noted to be febrile (103°F), hypotensive (BP of 90/60 mmHg), tachycardic (124 bpm) with rapid shallow breathing (RR of 32/min), covered in vomitus, and only responsive to painful stimuli. The patient had no other significant past medical problems except for hospitalizations for bipolar and schizoaffective disorders treated with lithium 450 mg twice daily and chlorpromazine.

On physical examination, the patient was found to be somnolent and sedated; he was intubated. His pupils were equal, round, and reactive to light. His heart, lung, abdomen and extremities were unremarkable. He scored 4 out of 15 on the Glasgow Coma Scale and had brisk reflexes. No tremor was noted.

Initial labs showed elevated serum lithium level of 5.0 mEq/L (0.8-1.2 mEq/L) leukocytosis (16.3X10^9/L) elevated BUN (95 mg/dL), creatinine (5.6 mg/dL), CPK (1426 mcg/L) and lactic acid (2.5 mmol/L). Serum and urine toxicology were negative. ECG showed sinus tachycardia with prolonged QTc interval of 516 ms. CT of the brain was normal.

The patient was treated with isotonic saline, colonic decontamination with polyethylene glycol and two sessions of hemodialysis. Lithium levels trended down to 0.6 mEq/L on day 4. Despite normal lithium levels, he persisted to have an altered state of consciousness and generalized weakness for 4 days. Hence the syndrome of irreversible lithium effectuated neurotoxicity (SILENT) was considered. After 20 days of inpatient stay (treated with isotonic saline, colonic decontamination and hemodialysis X 2) the patient’s condition improved and he was able to ambulate by himself. A wide range of differential diagnoses were considered for our patient’s unresponsive condition. Diagnosis was concluded to be lithium induced encephalopathy after ruling out other causes of altered mental status. EEG revealed mild, diffuse slow activity, indicating cerebral dysfunction consistent with encephalopathy. Although the literature states EEG can show triphasic waves with lithium toxicity, it is not absolutely needed for diagnosis.

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HELICOBACTER PYLORI NEGATIVE GASTRIC MALTOMA AS A RECURRENTE OF AN EYELID TUMOR

The most common site of extra nodal marginal zone cell lymphoma also known as, mucosa-associated lymphoid tissue (MALT) lymphoma is the stomach. Dissemination to other sites is about 30%. Extra nodal marginal zone B-cell lymphoma is the most common lymphoma of the ocular adnexa (conjunctiva, orbit, lacrimal gland, and eyelid.) 90% of gastric MALT lymphomas are associated with Helicobacter pylori (H. pylori) infection. We report a case of H. pylori-negative gastric MALToma in a patient who had previously diagnosed MALT lymphoma of the right upper eyelid.

An 80-year-old Caucasian male presented after a near syncopal episode, without loss of consciousness. He complained of intermittent, epigastric discomfort for the last few weeks. He described the pain as dull in nature, 5/10 in intensity, and occurring after eating a meal. In the ambulance, he had an episode of a black vomitus. He denied any difficulty swallowing, weight loss, or reduced appetite. Review of other systems was unrevealing. His significant past medical history included left eyelid MALT lymphoma, diagnosed a year ago, which was in complete remission after radiation therapy. Patient had 4-5 colonoscopies in the past, which were unremarkable but never had an Esophagogastroduodenoscopy (EGD). He denied tobacco, alcohol, or recreational drug use and denied having allergies or significant family history of cancer. He was taking aspirin 81 mg daily but denied use of nonsteroidal anti-inflammatory drugs. His orthostatic vital signs were positive. Physical examination was unremarkable except for guaiac positive melena. Labs showed significant drop in hemoglobin from 14 g/dl two months prior to this 8.8 g/dl at admission. Labs were also significant for hypoalumininemia (3.0 g/dl) with total protein of 5.9 (6.3-8.2 g/dl), elevated BUN of 41 (9-20 mg/dl), creatinine of 1.0 (0.7-1.3 mg/dl), and mild hyponatremia (136 mEq/L); liver function tests were normal.

He was treated with intravenous fluid, intravenous proton pump inhibitors, packed red blood cells and platelet transfusion. EGD showed multiple gastric masses with hypertrophic gastric folds and ulceration of one of the masses. Pathology of the gastric mucosa was consistent with a brisk lymphoid infiltrate composed of predominantly small to medium sized CD20+ B-cells that focally encroach glandular structures suggesting a the diagnosis of extra nodal marginal zone lymphoma of the MALT lymphoma. H. pylori were negative. His condition improved and had no further episodes of melena or hematemeses. Patient was treated as an outpatient with local radiation to stomach with successful remission.

Stomach is the most frequent site of MALToma with local radiation to stomach with successful remission. Pathology of the gastric mucosa was consistent with a brisk lymphoid infiltrate composed of predominantly small to medium sized CD20+ B-cells that focally encroach glandular structures suggesting a the diagnosis of extra nodal marginal zone lymphoma of the MALT lymphoma. MALToma in a patient who had previously diagnosed MALT lymphoma of the right upper eyelid.
Sex Life Bland? Maybe it’s your Adrenal Gland

Non-classical congenital adrenal hyperplasia is a common genetic condition, yet it is extremely difficult to diagnose in adult males and often goes unnoticed. When detected, the clinical signs of this disease are primarily from androgen excess.

A 59-year old man was referred to an endocrinologist with low testosterone levels after complaints of sexual dysfunction. He was started on intramuscular testosterone replacement therapy by his urologist at this time. This patient has a past history significant for obesity, diabetes mellitus type 2, COPD, precocious puberty, and inability to impregnate his wife due to azoospermia. Preliminary laboratory data revealed almost undetectable gonadotropin levels. LH and FSH values were measured at 0.2 and 0.7 mIU/mL respectively. Physical exam was remarkable for small soft testes. MRI revealed no pituitary abnormalities. Cosyntropin stimulation test was ordered to assess cortisol levels and elicited a suboptimal response. An abdominal CT showed a left-sided adrenal mass measuring 21x13x20 cm and a smaller right-sided mass measuring 7.2x2.7x6.2 cm. Scrotal ultrasound ruled out a testicular adrenal rest tumor. A 17-hydroxyprogesterone level was measured and came back markedly elevated at 14,953 ng/dl. At this time, the diagnosis of non-classical congenital adrenal hyperplasia was made and treatment with hydrocortisone was initiated. Since the patient was experiencing pain related to the left-sided adrenal mass, he underwent an uncomplicated left-sided adrenalectomy. Post-operative pathology revealed a myelolipoma. Upon recovery, the patient was discharged. He continued on testosterone and hydrocortisone therapy and reported full restoration of sexual function on follow-up. This case demonstrates how early diagnosis and treatment are crucial in cases involving non-classical congenital adrenal hyperplasia. Early identification and intervention can help restore reproductive and sexual function. Non-classical congenital adrenal hyperplasia is rarely diagnosed in adult patients. This disorder should be considered in the differential diagnosis for sexual dysfunction or infertility in males. However, non-classical congenital hyperplasia deserves even more careful consideration if the patient has a past medical history significant for early-onset puberty. Multiple case reports exist of male patients being able to father children when early diagnosis and treatment are initiated. Additionally, it is also rare to encounter bilateral myelolipomas in the setting of non-classical congenital adrenal hyperplasia. Our case demonstrates a common endocrinological pathology presenting at an uncommon age with rare radiological findings.

Atypical Presentation of an Organ of Zuckerkandl Paraganglioma

Paragangliomas are catecholamine secreting neuroendocrine tumors that arise from chromaffin cells. About 10% of pheochromocytomas are extra-adrenal and are considered paragangliomas. The classic triad of symptoms, present in 24% of cases includes headache, diaphoresis, and palpitations. Other common symptoms include fever, weight loss, hypertension, hyperglycemia, and abdominal pain.

A 54-year-old man presented after a brief episode of loss of consciousness that occurred one day prior to admission to the hospital. The patient’s son reported a fall followed by an unresponsive episode lasting 3-5 seconds, the symptoms resolved spontaneously within seconds and the patient regained consciousness without deficits. The patient’s blood pressure immediately after the episode was 100/90 mmHg. Information obtained in the emergency department revealed a history of multiple similar episodes that began 3 years ago. Reportedly, these episodes had increased in frequency over the past 6 months, with a current frequency of 4-5 times per week. Upon arrival in the emergency department, the patients’ blood pressure was 218/107. IV hydralazine was given and 30 minutes later the blood pressure was 153/82. Twenty minutes later, blood pressure was 232/118. These fluctuations continued for six hours. Physical exam, including orthostatic vitals were within normal limits. Family history was non-contributory. Electrocardiogram revealed sinus bradycardia. The patient had an episode of loss of consciousness witnessed by ER staff. Although the patient initially presented with syncope, the rapid and dramatic fluctuations in blood pressure prompted a cardiac work-up. Echocardiogram revealed EF of 65%, LVH, and impaired ventricular relaxation. Due to marked hypertension, catecholamine levels were evaluated and revealed markedly elevated normetanephrine, total metanephrine, and 24-hour urinary vanillylmandelic acid levels. Coronary Angiography was unremarkable. Non-contrast CT of the abdomen and pelvis showed a 4.1 x 4.3 cm pre-aortic mass between the aorta and inferior vena cava. Tumor imaging Octreotide Indium-111 scan revealed a somatostatin positive tumor in the mid-paraaortic region of the abdomen. A diagnosis of an extra-adrenal pheochromocytoma (paraganglioma of the organ of Zuckerkandl) was made. MRI of the brain with and without contrast revealed no abnormality. The patient was treated with alpha-blockade prior to surgery and underwent an uncomplicated resection of the mass. Patient reported resolution of symptoms on post-operative follow-up.

Paragangliomas can represent a number of diagnostic, management, and surgical challenges. Patients may present with subtle clinical signs and non-contributory family histories. Additionally, paragangliomas associated with multiple syncopal episodes have been documented when the mass is located on the carotid body or when the mass causes a ventricular outflow tract obstruction. Paragangliomas of the organ of Zuckerkandl do not typically present with syncope. This case demonstrates a very atypical presentation of a paraganglioma and outlines the appropriate work-up and management.
Histoplasmosis is treated with amphotericin B or itraconazole.

Bowel perforation in HIV patients may be secondary to opportunistic infections especially with low CD4 counts.

Histoplasmosis is a progressive extra pulmonary infection caused by Histoplasma capsulatum. Almost 5-27% cases of histoplasmosis are seen in HIV infected individuals. The risk of disease in HIV infected patients has declined with effective antiretroviral therapy.

Case Description:
34 year old man with Human Immunodeficiency virus (HIV) diagnosed a year ago on elvitegravir, cobicistat, emtricitabine and tenofovir presented with severe diffuse abdominal pain with sudden onset, beginning in right lower quadrant and right lumbar region, associated with nausea and vomiting. Examination revealed- heart rate of 121/min, generalized abdominal tenderness with guarding and hypoactive bowel sounds. Leucocyte count was 15,000mm3, lactate 3.8 mmol/ L, HIV RNA viral load undetectable (was 580559 a year ago), cluster of differentiation (CD4) count 77 cells/microliter( a year ago127 cells/microliter). CT abdomen and pelvis showed distended stomach and proximal small bowel with possible transition in mid pelvis, foci of extraluminal gas in pelvic mesentery and mesenteric venous gas with extensive mesenteric edema and small pelvic ascites. Exploratory laparotomy revealed perforated small bowel, affected mesentery was resected with end to end anastomosis. Surgical pathology showed fungal-associated necrotizing granulomatous inflammation, perforation and acute peritonitis. Granulomas were positive for budding spores suggestive of histoplasmosis. Patient improved with broad spectrum antibiotics (Ertapenem) and fluconazole for peritonitis, which was changed to itraconazole based on histopathology results. Patient continued to do well at 2 month follow up.

Discussion:
Histoplasmosis should be considered in a HIV/Immunocompromised patient with CD4 counts below 150 cells/microl. Our patient was treated with HAART a year ago, possibly developed immune reconstitution inflammatory syndrome with subsequent bowel perforation secondary to histoplasmosis. Diagnosis is confirmed by microscopic demonstration or isolation using stains for fungi, cultures, antigen detection, and serologic tests of Histoplasma from extra pulmonary sites. GI histoplasmosis is most commonly seen in males in fifth decade of life. Treatment involves antifungal therapy with Amphotericin B or itraconazole therapy based on immune function and severity of illness. Treatment is highly effective but relapse can occur in severely immunocompromised individuals.

Conclusion:
? Bowel perforation in HIV patients may be secondary to opportunistic infections especially with low CD4 counts.

Histoplasmosis is treated with amphotericin B or itraconazole.
Resident/ Fellow Clinical Vignette

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Use of Factor VII as a bypass agent in a Hemophilia B patient with inhibitors to Factor IX

INTRODUCTION:
Hemophilia is an X-chromosome linked congenital bleeding disorder. There are currently an estimated 20,000 patients in the US. Hemophilia A (Factor VIII deficiency) is four times more common than Hemophilia B (Factor IX deficiency). The treatment of severe Hemophilia with intravenous clotting factor infusions is complicated by the development of inhibitory antibodies to these factors. Patients with inhibitors can be treated with a "bypassing agents" activating different parts of the coagulation cascade to bypass the deficient factor. We present a patient with Hemophilia B with inhibitory antibodies who was treated with repeated Factor VII infusions.

CASE:
Our patient is a 28 year old male with a history of Hemophilia B, chronic subdural hematomas (SDH), intra-cerebral hemorrhages and soft tissue hematomas. He had a history of anaphylactic reaction to Factor IX, with reported swelling and wheezing. He presented with a thirty-day history of occipital headaches and four-day history of severe lower back pain. The pain was associated with tingling sensation and numbness over the left posterior thigh, radiating to the front of his left thigh. On examination, he was noted to have motor and sensation intact bilaterally, normal anal sphincter tone and tenderness to palpation over T1 and L1 spine, including the left paraspinal area.

CT Head on admission showed interval evolution of the left subdural hemorrhage, with no evidence of delayed bleeding. MRI Thoracic spine showed abnormal signals in the ventral spinal canal at T1 and T2, at the T11 level and at the mid T12 level extending into the superior aspect of the lumbar spinal canal. These findings were concerning for possible subdural hematoma (SDH). He had a low level of 26% of Factor IX. He was started on prophylactic Factor VII infusions every 3 hours, to prevent further extension of possible SDH.

A repeat MRI of the spine, after 5 days of Factor VII infusions, showed stable linear foci of abnormal signals at the ventral T1 T2 and dorsal T11 T12 levels. His coagulation parameters and hematocrit remained stable during hospitalization and he was discharged to home with plans for outpatient Hematology and Neurosurgery follow up.

DISCUSSION:
Inhibitory antibodies are seen in 25-30% of patients with severe Hemophilia A and 1-5% of those with Hemophilia B. Patients with an underlying molecular defect affecting F8/F9 gene, younger age at first treatment, Afro-Caribbean ethnicity and treatment during co-existent inflammation are at higher risk of developing these antibodies. Studies to risk stratify Hemophilia A patients on the risk of antibody development have been performed and externally validated, helping to potentially individualize treatment.

This case was particularly unique because it employed the use of Factor VII to activate the extrinsic coagulation pathway and bypass the inhibitory antibodies to Factor IX.

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POLYARTERITIS NODOSA WITH NEGATIVE INFLAMMATORY MARKERS AND LOCALIZED VASCULITIS OF THE GASTROINTESTINAL TRACT: A CASE REPORT

Introduction:
Polyarteritis nodosa (PAN) is a rare systemic necrotizing vasculitis affecting 2-33 persons per million. Most cases of PAN are idiopathic, however Hepatitis B and C infections and hairy cell leukemia can be the trigger in some cases. PAN typically affects medium-sized arteries and can affect different organs in various combinations, including skin, muscle, joints, kidneys, heart, nervous system and gastrointestinal tract. Laboratory testing usually reveals a prominent nonspecific acute phase response and anti-neutrophil cytoplasmic antibody (ANCA) is typically negative. Diagnosis is based on biopsy and pathologic confirmation of vasculitis in medium-sized arteries of symptomatic organs. When biopsy is not feasible or negative, visceral angiography revealing multiple microaneurysms can support the diagnosis.

Here we describe a rare case of PAN with negative inflammatory markers and localized gastrointestinal involvement.

Case presentation:
A 56 year-old caucasian man with no significant past medical history presented to our center with acute left upper and lower quadrant abdominal pain. CT angiography of the thorax was performed to rule-out pulmonary embolism which showed thickening and enhancement of the splenic and hepatic arteries without aneurysmal dilatation. To evaluate this further, a dedicated CT angiogram of the abdomen was performed, suggestive of splenic infarct. Serologic evaluation was negative for antinuclear antibody (ANA), cytoplasmic and perinuclear ANCA (cANCA, pANCA), anti-phospholipid antibodies and revealed normal ESR, CRP, C3, C4, complement levels and IgG4. Hepatitis B and C serologies were negative. On the fifth day of hospitalization, our patient developed severe left flank pain and an erythematous papular rash in the left groin. MRI and MRA of the kidneys revealed left renal infarction and biopsy of the rash revealed spongiotic interface dermatosis, without evidence of vasculitis. Treatment with high dose steroid was initiated with improvement in symptoms. Repeat CT angiography of abdomen revealed significant mural thickening and luminal narrowing of the celiac axis and its branches with small pseudoaneurysms along the celiac trunk, confirming the diagnosis of PAN.

Discussion:
We report a rare and challenging case of PAN. Despite negative inflammatory markers and skin biopsy, our patient developed vasculitis associated infarction of abdominal organs. In PAN vasculitis, arterial narrowing in the setting of vessel wall inflammation and intimal proliferation can reduce blood flow and predispose to thrombus formation in the involved organs, resulting in ischemia or infarction. Diagnosis was confirmed on repeat CT angiography of the abdomen showing small pseudoaneurysms along the celiac trunk. Our patient responded appropriately to high dose steroids. This case demonstrates the challenges in making the diagnosis of PAN in a patient presenting with normal inflammatory markers and atypical vasculitis with limited organ involvement.
**Resident/ Fellow Clinical Vignette**

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**Immunosuppressive and dual antiplatelet therapy related spontaneous intramuscular hematoma**

**Introduction:**
Spontaneous intramuscular hematoma (SIH) is usually seen in patients with clotting disorders and those who are on anticoagulation. Rarely, retroperitoneal hematomas are seen in transplant patients on immunosuppressive medications, especially in patients who are on concurrent dual antiplatelet therapy (DAPT) or anticoagulation. However, there are no prior cases reported of SIH in such patients. This case shows that patients on DAPT (or with platelet dysfunction) and/or immunosuppressive therapy can also have SIH.

**Case:**
A 62-year-old woman with history of systemic lupus erythematosus (SLE), Lupus nephritis post renal transplant (20 years ago), chronic transplant rejection (on tacrolimus and prednisone), hypertension, overt lower gastrointestinal bleeding secondary to diverticulosis post hemicolectomy and atrial fibrillation on aspirin and clopidogrel. She presented with sudden onset of right hip pain radiating to the right thigh and gluteal region. She denied any recent trauma.

**Physical examination** showed decreased range of motion of the right hip secondary to pain. No erythema or swelling was observed. Laboratory tests showed normocytic anemia (hemoglobin 11g/dl), elevated creatinine, elevated blood urea nitrogen and normal INR. Right hip and pelvic x-rays showed no acute fracture or abnormality. She underwent pelvic computerized tomography (CT) scan which showed a large intramuscular hematoma extending through the right gluteus medius and minimus muscles. Her hemoglobin remained stable during the hospitalization and pain improved with conservative care. Initially, clopidogrel was withheld, which was later restarted as outpatient.

**Discussion:**
We report a rare case of SIH in a renal transplant patient with chronic transplant rejection on immunosuppressive and DAPT. To the best of our knowledge, no cases of SIH have been reported in association with renal transplant. Although, DAPT and platelet dysfunction is rarely associated with retroperitoneal hematomas and concurrent immunosuppressive therapy use can increase the risk significantly, there is limited data on SIH in these patients. CT imaging without contrast is the most commonly used diagnostic modality. In most cases only conservative care is warranted as tamponade effect limits the bleeding. In some cases with uncontrolled bleeding, hemodynamic instability and/or high suspicion for compartment syndrome, surgical intervention and/or interventional radiological embolization is required. It can result in significant morbidity and requires a high clinical suspicion, as early initiation of supportive care and/or intervention can decrease the long-term complications and result in good prognosis.

**Conclusions:**
SIH is commonly seen in patients with clotting disorders. DAPT and immunosuppressive therapy are common risk factors for spontaneous retroperitoneal hematomas, and can increase the risk of SIH. Patients who are on DAPT and/or immunosuppressive therapy presenting with sudden onset of localized symptoms or a drop in hemoglobin warrant a CT scan without contrast to rule out SIH and retroperitoneal hematoma, since prompt diagnosis and early supportive care has shown superior clinical outcomes.

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**SEIZURES UNFOLDING A COMPLICATED CASE OF OVERLAPPING SLE AND GPA**

**Introduction:**
Multiple autoimmune phenomena can occur in a patient that are consistent with more than one rheumatologic disease; in such cases, the patient is said to have an overlap syndrome. In this case, we discuss a patient who was diagnosed with Systemic Lupus Erythematosus (SLE) and Granulomatosis with Polyangiitis (GPA). An overlap between SLE and GPA is extremely rare, with very few case reports having cited this occurrence. Furthermore, no prior case has initially presented with neuropsychiatric lupus, an additional unique feature of this patient.

**Case Report:**
Our patient is a 19 year old Afro-Caribbean male presenting to the emergency room with new onset generalized tonic-clonic seizures. The patient was treated with anti-epileptics and was briefly intubated for airway protection. Initial workup was significant for acute kidney injury, proteinuria, and pancytopenia. CSF analysis was consistent with aseptic meningitis. EEG and brain imaging showed nonspecific findings of generalized cerebral slowing and mild parenchymal loss, respectively. Rheumatologic workup was done to investigate the etiology of aseptic meningitis. Findings included elevated levels of ANA, Anti-dsDNA, Anti-SSA/Ro, and Anti-RNP antibodies. Additionally, the patient was found to have low levels of complement C3, C4. A kidney biopsy confirmed Class III(A) and Class V lupus nephritis as well as focal segmental glomerulosclerosis. The patient was started on Mycophenolate Mofetil, Hydroxychloroquine, Methylprednisolone, and Lacosamide for SLE nephritis and presumed Neuropsychiatric lupus. The patient clinically improved over the course of a month and was discharged to an acute rehab facility. One week later, the patient began developing orthopnea, lower extremity edema, and abdominal swelling. An echocardiogram showed elevated pulmonary artery systolic pressure and mild tricuspid regurgitation. Further imaging studies showed multiple pulmonary nodules as well as opacifications and inflammatory changes of the paranasal sinuses. Of note, the patient on previous labs was found to have elevated Myeloperoxidase antibodies. The clinical picture was therefore consistent with GPA. The patient was initiated on IV Rituximab therapy in conjunction with SLE treatment and found to have significant improvement.

**Discussion:**
Our patient’s presentation had multiple unique features. First, our patient’s profile did not fit the epidemiologic pattern of GPA. The mean age of diagnosis for GPA is between 41 and 52, with a predominance in the caucasian population. In addition, the overlap of SLE and GPA with a presenting diagnosis of CNS lupus is an exceedingly uncommon occurrence. Our patient’s conditions were only unmasked following his seizures, exemplifying the importance of maintaining a wide differential diagnosis when approaching patients with acute neuropsychiatric conditions.
## Resident/ Fellow Clinical Vignette

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<th>Aireen Kuan MD</th>
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<td>Marie Louies Lamsen MD, Stephen Jesmajian MD</td>
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<td>Montefiore New Rochelle Hospital</td>
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### “START FROM THE HEART” Resolution of recurrent gastrointestinal bleeding post aortic valve replacement

**Introduction:**
Heyde’s Syndrome (HS) is the association of aortic valve stenosis (AS) and gastrointestinal angiodysplasia causing gastrointestinal bleeding (GIB). We present a case of a patient with severe AS that had successful hematologic recovery from recurrent GIB following aortic valve replacement (AVR).

**Case Presentation:**
This is a case of a 68-year-old female with Diabetes Mellitus Type 2, Hypertension, and severe AS with an initial peak aortic gradient of 71 mmHg and an aortic valve area of 0.9 cm² presenting with 1-week history of exertional chest discomfort. Physical examination showed conjunctival pallor, slow capillary refill time, and a grade 3/6, high-pitched aortic systolic murmur, radiating to the carotid area. She was diagnosed with severe acute symptomatic anemia from possible GIB. Stool was positive for occult blood. Hemoglobin (Hgb) level was low at 5.1 gm/dl, with an iron panel consistent with iron deficiency. Reticulocyte production index: 1.8%. Von Willebrand factor (vWF) and ristocetin cofactor were normal. Direct coomb’s test was negative. Initial endoscopies were unrevealing except for Helicobacter pylori gastritis.

Patient was discharged with parenteral and oral iron supplements in addition to triple therapy for H. pylori. In the following months she required multiple hospitalizations from symptomatic anemia due to recurrent GIB which required repeated blood transfusions. A repeat capsule endoscopy showed active bleeding with the presence of multiple arteriovenous malformations in the proximal small bowel. Repeat echocardiogram showed progressive AS with an increase in peak gradient of 98 mmHg and an aortic valve area of less than or equal to 0.7 cm². Cardiac catheterization and bioprosthetic aortic valve replacement (AVR) were successfully performed. On follow-up, 7 months post AVR; she had normal iron studies, and Hgb was stable at 11.5 gm/dl. There were no more recurrences of GIB requiring blood transfusions and hospitalization.

**Discussion:**
The most convincing mechanism in patients with HS is the deficiency or defect in high molecular weight multimers of vWF. Moreover, AS would also give rise to GIB by reducing gastrointestinal perfusion leading to hypoxemia-induced dilatation of blood vessels. Correlating gastrointestinal angiodysplasia and AS is challenging since both occur in the elderly such as in our case. There are multiple emerging literatures which support AVR as a curative option in more than 80% of patients with HS that decreases the need for recurrent blood transfusions, and endoscopic intervention; furthermore decrease the risk of recurrent GI bleeding and overall mortality. HS is not yet included in the guidelines for AVR but should possibly be considered especially in patients with recurrent GIB.

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<th>Aireen Kuan MD</th>
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<tr>
<td>Olvas Dallaku MD, Marie Louies Lamsen MD, Stephen Jesmajian MD</td>
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<td>Montefiore New Rochelle Hospital</td>
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### “HE TURNING POINT: HOW SAFE ARE WE FROM A COMMON DRUG?”

**Introduction:**
Acquired QT prolongation is a rare adverse effect of antihistamines which are commonly used over-the-counter medications. We report a case of a patient that developed progressive QT prolongation leading to Torsade de pointes (TdP) from Nyquil (Acetaminophen, Doxylamine Succinate, Phenylephrine Hydrochloride, Dextromethorphan Hydrobromide) overdose.

**Case Presentation:**
A 26-year-old Caucasian male with a 3-day history of Nyquil use was initially found unresponsive from a suicide attempt with a 12 ounce bottle of Nyquil and alcohol ingestion. He was immediately brought to the emergency department but had already recovered on presentation. He was not in distress. Electrocardiogram (EKG) showed sinus bradycardia with a ventricular rate of 40 bpm with a corrected QT (QTc) interval of 544 ms. Urine toxicology was positive for cannabinoids. Blood alcohol and acetaminophen levels were negative. Thyroid stimulating hormone and serum electrolytes were normal except for a magnesium (Mg) level of 1.2 meq/L. He was immediately given 4 grams of intravenous Mg. After six hours, the patient became lethargic and hypoxic, we administered a magnesium Mg infusion at a rate of 0.5-1 gram/hour for 24 hours. He had a normal echocardiogram Doppler study. Subsequent EKG’s showed normalization of acquired QTc prolongation.

**Discussion:**
Antihistamines are postulated to cause dose-dependent QTc prolongation by blocking the rapidly activating delayed rectifier potassium channel, a key repolarizing current in the ventricular myocardium. Doxylamine Succinate is a first generation antihistamine commonly found in Nyquil: a combination of Acetaminophen, Doxylamine Succinate, Phenylephrine Hydrochloride, and Dextromethorphan Hydrobromide. It has a lethal dose of at least 25mg/kg body weight, although our patient only took 150 mg single dose, Doxylamine and Dextromethorphan are both metabolized by CYP2D6 enzymes and they may compete for the same enzyme, altering their metabolism. At the same time 7-10% of Caucasians are poor metabolizers. Retrospective cases have reported diphenhydramine induced QT prolongation but there have only been four reported cases of TdP caused by diphenhydramine overdose that we are aware of. As an ethanamine derivative similar to diphenhydramine, this may be the first reported case of Doxylamine induced TdP. In such case, patients and clinicians should be aware of the possible dangerous side effects of this frequently used over-the-counter medication.
Consider the patient as a whole. The clinician’s need to have a broad differential diagnosis and to consider the patient as a whole.

**Resident/ Fellow Clinical Vignette**

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<th>Persio LÃ­pez Loyo MD</th>
<th>Sandrine Lebrun MD</th>
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<td>New York Medical College - Metropolitan Hospital Center</td>
<td>Craig Basman MD, Atul Kukar DO. Lenox Hill Hospital</td>
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<td><strong>Cardiac Tamponade: An Unusual Presentation of Systemic Lupus Erythematosus</strong></td>
<td><strong>SUDDEN CARDIAC ARREST IN A 53 y/o HEALTHY MALE: A RARE CASE OF SPONTANEOUS CORONARY ARTERY DISSECTION (SCAD)</strong></td>
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**Introduction**

Pericarditis may be the initial presentation of Systemic Lupus Erythematosus (SLE), however cardiac tamponade is rarely the presenting symptom.

**Case Presentation**

A 55-year-old Ghanaian man was admitted to our service with dyspnea, which had progressed over the course of 4 days. He had an acute decrease in his exercise tolerance and developed orthopnea the night prior to his admission. Review of systems revealed progressive asthenia, anorexia and low grade fevers for the last few months, with an unintentional weight loss of approximately 40 lbs. No other symptoms were elicited. The patient’s physical examination was within normal limits, except for mild periorbital edema. Cardiac sounds were unremarkable, and there were no signs of volume overload or circulatory abnormalities. Initial workup revealed leukopenia and a normocytic, nonmegaloblastic anemia. He had decreased renal function and subnephrotic proteinuria. His troponin level was 0.078 ng/mL, his N-terminal proBNP level was 5,011 pg/mL, and his erythrocyte sedimentation rate was 60 mm/h. His EKG was unremarkable; his chest x-ray revealed cardiomegaly. A bedside transthoracic echocardiogram (TTE) on admission revealed a moderate pericardial effusion, without any diastolic collapse. The differential included autoimmune diseases, malignancy and tuberculosis, in light of the above findings. An official TTE was obtained 24 hours after admission, showing progression of the effusion and partial diastolic collapse of the right atrium and ventricle. The patient was transferred to the intensive care unit and a pericardial biopsy with pericardial window were scheduled for the following day. Approximately 12 hours later, the patient developed dyspnea at rest and pulsus paradoxus, and was urgently operated due to cardiac tamponade.

The diagnosis of SLE was established once serology became ruled out malignancy and tuberculosis. The differential included autoimmune diseases, malignancy and tuberculosis, in light of the above findings. An official TTE was obtained 24 hours after admission, showing progression of the effusion and partial diastolic collapse of the right atrium and ventricle. The patient was transferred to the intensive care unit and a pericardial biopsy with pericardial window were scheduled for the following day. Approximately 12 hours later, the patient developed dyspnea at rest and pulsus paradoxus, and was urgently operated due to cardiac tamponade. The diagnosis of SLE was established once serology became available. The pericardial biopsy confirmed SLE pericarditis, and ruled out malignancy and tuberculosis.

**Discussion**

Acute pericarditis is characterized by sharp, pleuritic, postural chest pain and diffuse ST-segment elevations. With diverse etiology, up to 24% of the cases of pericarditis can be attributed to an autoimmune disease. Pericarditis is the most common cardiac manifestation of SLE, the prototypical autoimmune disease, and it has been associated with poor survival. Non-Caucasian patients are more likely to present with SLE pericarditis. The incidence of pericarditis is greatest in African patients (43%) compared to Chinese (26%) and Thai (16%) populations. Interestingly, although most SLE pericarditis presents with typical symptoms of pericardial disease, 26% of African patients present solely with dyspnea. Although cardiac tamponade has been described in children with SLE, it is seldom part of the initial presentation in adults. This case is a rare presentation of a new case of SLE. It illustrates the clinician’s need to have a broad differential diagnosis and to consider the patient as a whole.
**Resident/ Fellow Clinical Vignette**

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<td>Renee Dougherty DO, Rebecca Mazurkiewicz MD, Rachel Bond MD</td>
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## CLOZAPINE INDUCED MYOCARDITIS: A POTENTIALLY SILENT AND FATAL THREAT

**Case:** A 23-year-old man presented to the hospital from a psychiatric facility, with fever for four days. His medical history was significant for paranoid schizophrenia with multiple hospitalizations for auditory hallucinations, refractory to olanzapine and risperidone. In the setting of recent deteriorating psychiatric condition, clozapine was initiated and steadily titrated up over three weeks to 200mg daily, with clinical improvement. Upon presentation, his physical exam was unremarkable except for T_max of 103.1 with a heart rate of 123. Labs showed mild leukocytosis with eosinophilia to 9.8%, Troponin T of 1.28 peaking at 1.88 ng/ml, total CK of 993 u/L, CK-MB of 34 ng/ml and a CRP of 157 mg/L. An infectious workup including a respiratory viral panel, chest xray, blood and urine cultures, was negative. ECG showed sinus tachycardia with new T wave inversion in lead III. Echocardiogram revealed mild global LV dysfunction, with an EF of 35%. Cardiac catheterization showed no coronary disease. A diagnosis of clozapine induced myocarditis was strongly considered and clozapine was substituted with quetiapine and haloperidol. Further workup was limited as patient deferred cardiac MRI, the diagnostic imaging method of choice for myocarditis. For that reason, along with the inherent risk of perforation and tamponade, endomyocardial biopsy was not pursued. He was managed medically with an ACE Inhibitor and beta-blocker and discharged to a psychiatric facility with instructions to permanently discontinue clozapine. A month later, he remained asymptomatic with repeat Echo showing an improved EF of 50%.

**Impact:** A highly effective atypical antipsychotic, clozapine is used in treatment-refractory schizophrenia. The cardiac complications of this drug are rare, potentially fatal and include myocarditis, cardiomyopathy and heart failure. Alas, myocarditis has a wide range of nonspecific symptoms, making it difficult to identify clinically. Persistent sinus tachycardia and fevers may be the only warning signs. Prompt discontinuation of the agent is imperative soon as suspicion arises, to improve outcome.

**Discussion:** Though myocarditis can occur at any given time during clozapine treatment, the risk is especially heightened within 6-8 weeks of therapy. Because its heterogeneous nonspecific clinical presentation can be misleading, patients on clozapine should be closely monitored and undergo weekly lab testing (Cardiac enzymes, CBC with differential, CRP etc). A high degree of suspicion must be maintained for adverse cardiac effects, with a low threshold to stop the drug. Studies suggest that partial or complete recovery follows early diagnosis and drug cessation. Higher doses and longer duration of therapy are associated with poor overall outcome including death.

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## NO ONE IS LISTENING TO ME! NEAR DEATH AWARENESS, DELIRIUM AND CAPACITY AMONG CULTURALLY DIVERSE PERSPECTIVES AT THE END OF LIFE

**Introduction:** At the end of life, people may experience delirium or near death awareness (NDA). These are not the same but many clinicians may not recognize the difference. Delirium may influence capacity to make decisions, but NDA usually does not.

**Case Description:** Mr. J is a 66-year-old Jamaican male with recently diagnosed advanced cholangiocarcinoma who presented with generalized weakness and RUQ pain. He was treated for sepsis secondary to cholangitis and improved. Palliative chemotherapy was offered but he declined. Mr. J wished to go home and treat himself with a traditional Jamaican â€œcleansing regimen.â€ Mr. J confided that he had been â€œseeingâ€ his deceased mother, the visions were telling him heâ€™ll be â€œjoining her in her world soon,â€ and that he was comforted by these occurrences. These comments became a red flag for the primary team, prompting a psychiatric consultation, which felt that the patient lacked capacity to make medical decisions. Primary team called a palliative care consult, which believed Mr. J did have capacity, was able to recount his â€œlife review,â€ including regrets about breaking up with his wife and not having close relationship with his daughter. Patient stated he wanted to go home, he did not want artificial machines or resuscitation. Primary team identified his daughter, who lived in another state, to serve as surrogate decision maker. Daughter did not know his wishes, but requested chemotherapy. Subsequently, Mr. J had a cardiac arrest and was resuscitated. He was transferred to the ICU and died.

**Discussion:** Mr. J’s case illustrates the crossroads among capacity for making medical decisions, cultural values, and near death awareness. All three factors influenced the way he died. Patient’s cultural preference for herbal treatments and his NDA made clinicians question his capacity. NDA can be confused with delirium/psychosis and a proper assessment is critical, especially when deciding about patient’s capacity. There are five domains to assess when determining a patient’s decision-making capacity: Communication, Understanding, Appreciation, Reasoning, and Consistency. It is crucial to keep in mind that patients make decisions based on their beliefs, values and cultural background, which may not align with the Western medical model; however, this does not mean the patient lacks capacity. If the primary team, psychiatry, and palliative care teams had all agreed that Mr. J had a terminal illness, allowed him to go home to pursue his traditional Jamaican treatments, and recognized that NDA was a common phenomenon at the end of life, Mr. J could have gone home and died a â€œgood deathâ€ as he wished.
NDI is a condition in which polyuria occurs due to lack of ADH at the level of the kidney. NDI can be primary or secondary. Secondary NDI is induced by many causes, ranging from restricted secretory function due to diseases of the posterior pituitary to acquired defects due to a wide variety of toxic agents. NDI can be complete or partial (as in this case) and can be caused by a variety of factors, including diabetes mellitus, hypothyroidism, and renal tubular acidosis. NDI can also be caused by drugs such as lithium, which can inhibit ADH secretion, or by conditions such as congestive heart failure, which can reduce water reabsorption in the distal tubule. NDI is also present in patients with diabetes insipidus (DI), a disease characterized by a lack of ADH secretion, which can be induced by a variety of factors, including tumors, inflammation, or trauma. NDI can also be caused by other acquired causes, including hypercalcemia, which can be caused by a variety of conditions, such as primary hyperparathyroidism or malignancy, and which can be treated with ADH replacement therapy.

We present a case of a 50-year-old male with a history of developmental disability and hypothyroidism who presented to the hospital with fatigue and cyanosis. The patient was found to have a left upper lobe consolidation on chest x-ray consistent with pneumonia. Labs revealed neutrophilic leukocytosis. He was started on vancomycin and tazobactam for healthcare-associated pneumonia (HCAP). His course was complicated by acute hypernatremia to 166 mmol/L and a creatinine of 1.97 mg/dL, reaching a peak of 2.41, from a baseline of 0.6. Further work-up revealed a serum osmolality of 335 mosm/kg and urine osmolality (Uosm) of 174 mosm/kg, raising suspicion for diabetes insipidus (DI). He also had a fractional excretion of sodium (FENa) of 2.6%, eosinophilia, peripheral eosinophilia, and polyuria (> 3 liters/day). Piperacillin-tazobactam was discontinued due to concern for acute interstitial nephritis (AIN). His serum Na remained elevated despite aggressive parenteral hydration, and urine osmolality, consistent with partial nephrogenic diabetes insipidus (NDI). Serum creatinine improved to 1.29 after discontinuation of piperacillin-tazobactam. Piperacillin-tazobactam has been well-established as a cause of drug-induced AIN. The diagnosis can be made definitively with kidney biopsy, although most cases are diagnosed clinically. Timing of onset as related to initiation of the suspected offending agent is important in this patient. Peripheral eosinophilia, eosinophiluria, acute renal failure, and initiation of piperacillin-tazobactam with subsequent improvement after discontinuation point to a diagnosis of AIN. NDI is a condition in which polyuria occurs due to lack of response to ADH at the level of the kidney. NDI can be primary (genetic or congenital) or acquired, most commonly due to lithium use. Other acquired causes include hypercalcemia, hypercalciuria, and obstructive uropathy, none of which were present in our patient. NDI can be completor partial (as in this case). Given the time course of symptoms and laboratory derangements, we propose that piperacillin-tazobactam was the offending agent causing AIN, which subsequently led to NDI. AIN associated with NDI has not been previously reported. Broad-spectrum antibiotic use for suspected HCAP is being called into question. Antibiotic stewardship programs are being deployed to reduce overutilization. This unique, rare case of piperacillin-tazobactam causing AIN and also NDI introduces another reason to de-escalate antibiotics and limit use as feasible. Hypernatremia without another apparent cause should raise suspicion of NDI in a patient on piperacillin-tazobactam.
Background: Carbon monoxide (CO) poisoning is one of the most common types of poisoning. It can be underdiagnosed as it can present with non-specific symptoms: nausea, vomiting, headache, chest pain and fatigue. We present a case study of a couple who presented with CO poisoning to emphasize the importance of early recognition of CO poisoning and its potentially fatal pro-thrombotic complications.

Case 1
53 year-old woman with hypertension and anxiety was transferred to our facility for CO poisoning. A few days prior to presentation, the patient was experiencing headache, dizziness and fatigue. She was evaluated at an outside ED and was diagnosed with viral syndrome. The following day she was found down in her apartment. The patient recalled turning on the gas furnace earlier that week. She was anxious, confused and complaining of chest pain. Initial CoHgb was 14.3% (normal 0-5%). Troponin was elevated and the EKG showed ST depression in II, III, aVF, and V4-V6, and ST elevation in aVR. She was treated for acute coronary syndrome and transferred to our facility for hyperbaric oxygen treatment. The patient received hyperbaric treatment with a significant improvement in her mental status but continued to have chest pain. She underwent cardiac catheterization that revealed significant one vessel disease of mid LAD and moderate distal CFX disease. A single drug eluding stent was placed in the LAD. The patient eventually improved and was discharged home.

Case 2
53 year-old male with hypertension who presented with his partner (described in case 1) with CO poisoning. The patient was initially confused but later gave history of progressively feeling weak and dizzy a few days prior to presentation. His mental status improved after receiving hyperbaric treatment, but later he reported bilateral thigh pain, left leg pain and pleuritic chest pain. Physical exam was unremarkable. Labs and imaging were consistent with rhabdomyolysis, bilateral DVT and sub-massive PE. Otherwise, troponin, BNP, EKG and coagulopathy studies were all unremarkable. He was treated with anticoagulation and discharged on Apixaban.

Discussion: These cases demonstrate acute CO poisoning with a thrombotic complications. Carbon monoxide is directly toxic to the mitochondria and endothelial cells, consequently triggering ischemia, as well as arterial and venous thrombosis. The risk of DVT is 3.85 times higher in patients with CO poisoning than in the general population. Common presentations of CO poisoning include dizziness, confusion, headaches and flu-like symptoms. Larger exposure can lead to significant neurological, myocardial, renal and pro-thrombotic complications. Clinicians should be trained to recognize the early signs of CO poisoning and to detect life threatening complications (hypoxic and pro-thrombotic). Thorough history taking can prevent discharging patients back to harmful environments which can further worsen their prognosis.

Neha Mehta DO
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Lenox Hill Hospital
Improving rate of appropriate vancomycin trough levels and dosing
Introduction: Vancomycin is one of the most commonly prescribed medications in hospitalized patients. Appropriate monitoring is necessary to minimize adverse effects including nephrotoxicity and overall patient outcomes. In our hospital, approximately 49 initial serum vancomycin troughs are ordered weekly. However, laboratory samples are often ordered and collected incorrectly due to lack of standardized protocol. We investigated these trough orders and collection times to ultimately improve dosing and adjustment. Methods: A retrospective chart analysis of initial vancomycin troughs for patients admitted to the Internal Medicine service on the general medical floor, telemetry, or ICU at Lenox Hill Hospital in October 2016 was performed. Additional variables collected included timing of provider order for vancomycin trough collection, specimen received by the lab, result posted, and the vancomycin dose adjustment made. The trough results were divided into 4 categories which included appropriate order, appropriate draw, inappropriate order, or inappropriate draw. An appropriate draw/order was defined as a trough ordered and/or performed an hour before the 4th dose or after dialysis prior to next dosing. An inappropriate order/draw was defined as a trough ordered and/or performed before or after the 4th dose in non dialysis patient or before dialysis prior to next dosing. Although we do not know exact time of specimen collection at bedside, based on internal lab department data, we used 60 minutes prior to the time the specimen was received in the lab as our surrogate for actual time collection at beside.

Results: Of the 49 vancomycin trough lab orders, 33% were ordered appropriately while 67% were ordered inappropriately. Of the inappropriate orders, 76% were ordered early and 23% were ordered late. Of the 49 vancomycin trough draw times, 55% were drawn appropriately, 24% were drawn inappropriately, and 21% were unable to determine. Of the inappropriate draws, 33% were drawn too early and 67% were drawn too late. The 21% were unable to determine due to unknown collection time.

Discussion: This study examines closely the errors associated with vancomycin use. The primary issue identified is timing of provider orders for trough collection (ordered too early) and actual phlebotomy collection time (drawn too late). Understanding our rate of appropriately timed troughs and management thereafter will help decrease potential medication toxicity and improve bactericidal rates by achieving therapeutic concentrations. Future studies involve a multidisciplinary approach involving residents, phlebotomists, pharmacists, and nurses to formulate a protocol for appropriate vancomycin trough collection orders and time. This will also guide dosing and adjustments based on vancomycin trough results.
Multiple myeloma (MM) is characterized by the monoclonal proliferation of plasma cells in the bone marrow, usually presenting with painful lytic bone lesions along with one or a combination of other systemic symptoms including hypercalcemia, anemia and renal failure. The traditional diagnostic hallmark of MM is the presence of an M-spike on either the serum (SPEP) or urine (UPEP) protein electrophoresis, representative of the monoclonal M protein produced by malignant plasma cells. Less than five percent of MM cases however are non-secretory which result in either the absence or diminishment of the M-spike. An even smaller percentage of cases lack the other usual characteristics of the disease mentioned earlier. The lack of these features poses a challenge when it comes to the diagnosis of the rare forms of the disease. Here we present a case of non-secretory multiple myeloma.

53-year-old male with no PMH with a chief complaint of nonspecific back and left thoracic pain. During workup, CT scan imaging revealed multiple osteolytic bone lesions in bilateral ribs, sternum and multiple thoracic vertebral bodies with pathological compression fractures. Lab work showed an elevated alkaline phosphatase level despite lytic process of myeloma, a normal calcium level, and normal renal function. Immunologic studies showed an inconclusive free kappa/lambda ratio, and an equivocal gamma globulin spike on protein electrophoresis, as well as inconclusive initial and repeat bone marrow aspirates. Diagnosis was finally confirmed by bone marrow biopsy, showing marked cellularity with 90% of which identified as plasma cells. Chemotherapy was immediately started. Given the paucity of typical multiple myeloma findings in the context of advanced disease and overtly positive bone marrow biopsy findings, this case represents an exceptional presentation of non-secretory multiple myeloma.

Toxoplasmosis encephalitis manifests similarly to metastatic disease in immunocompromised patients, and can delay diagnosis (2-4). Our case reinforces principles that are challenging to implement in a timely fashion.

Case
A 79-year-old Caucasian female with a 3-year history of CLL presented with a 6-week history of LLE weakness, recurrent falls and 2 days of focal seizures manifesting as 1 minute episodic, involuntary, rhythmic jerking motions of her left leg. CLL had been in remission for 4 months after completing 6 rounds of bendamustine and rituximab. Physical exam revealed LLE with 4/5 muscle strength, clonus in left foot with abnormal plantar reflex, normal sensation and deep tendon reflexes. Neurological exam was otherwise non-focal.

Results/Course
Initial CT brain showed areas of vasogenic edema and MRI brain with contrast revealed three ring enhancing lesions surrounded by vasogenic edema (Figure 1 and 2). CT chest, abdomen and pelvis revealed a spiculated right upper lobe nodule. Lung biopsy showed necrotizing granulomas. Lumbar puncture showed elevated protein levels with a normal glucose, initially non-diagnostic, but later returned positive for toxoplasmosis antibody. A transcranial brain biopsy of the left frontal lobe lesion was initially read as negative, but second opinion found the biopsy positive for bradyzoites and tachyzoites with multiple necrotizing granulomas consistent with the diagnosis of toxoplasmosis encephalitis (Figure 3 and 4). Serologic studies were positive for toxoplasmosis IgG 31.7 IU/mL and negative toxoplasmosis IgM. Patient was treated with prednisone, sulfadiazine and atovaquone. At one month follow up patient is alive with some residual weakness and no recurrent falls. MRI shows smaller lesions, but not completely resolved (Figure 5). She remains on lifelong immunosuppression therapy with pyrimethamine and sulfadiazine.

Discussion
Toxoplasmosis in immunocompromised patients is the result of a reactivated latent infection, and often has a self-limiting course without treatment in immunocompetent patients (7,11). In CLL, PubMed found 3 case reports of CNS toxoplasmosis (5-9). One lived a month rounds of bendamustine and rituximab. Physical exam revealed LLE with 4/5 muscle strength, clonus in left foot with normal plantar reflex, normal sensation and deep tendon reflexes. Neurological exam was otherwise non-focal.

Conclusion
Our CLL case with non-specific neurological complaints and radiographic findings mimicking neoplastic disease required a high clinical suspicion to aggressively pursue the diagnosable and treatable CNS toxoplasmosis. CLL patients with similar neurologic presentation require aggressive pursuit if hoping to avoid fatality.
Resident/ Fellow Clinical Vignette

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**New Onset Heart Failure and Pericardial Effusion Due to Severe Hypothyroidism**

Abstract
Systolic heart failure and pericardial effusions are two uncommon consequences of hypothyroidism in the community. We report a case of new onset heart failure and large pericardial effusion in a patient with longstanding hypothyroidism.

Introduction
Thyroid disease has profound effects on the cardiovascular system. The lack of thyroid hormone causes decreased cardiac output, cardiac contractility, and heart rate as well as impaired vascular smooth muscle relaxation. (3,6) It is well recognized that exudative pericardial and pleural effusions can accumulate in chronic hypothyroid states. (1) Pericardial effusions are prevalent in myxedema or late stages of hypothyroidism. (9) Pleural effusions are found in approximately 25% of patients with primary hypothyroidism. (10)

Case
A 93 year old woman with a past medical history of hypertension, hyperlipidemia, type 2 diabetes, and hypothyroidism presented to our facility with 2 months of progressive bilateral lower extremity edema and erythema. Dyspnea on exertion was also present. The patient had suffered from hypothyroidism for over 50 years secondary to hashimoto’s disease. Her home medications included levothyroxine, diltiazem, rosuvastatin, lisinopril, and sitagliptin. Upon arrival temperature was 97.3°F, blood pressure 176/112 mm Hg, pulse 90 beats per minute, respiratory rate 20 breaths per minute, with an oxygen saturation of 99% on room air. General physical exam revealed jugular venous distention, bilateral pitting edema to the mid shin bilaterally. Lower extremities were warm with mild tenderness to palpation.

Labs revealed a b-type natriuretic peptide level of 338.3, elevated TSH 91.4 uIU/mL, and low free thyroxine 0.54 NG/DL. Echocardiogram showed an estimated left ventricular ejection fraction of 20%, diffuse hypokinesis, large pericardial effusion, without evidence of tamponade. Chest CT confirmed a large pericardial effusion with bilateral pleural effusions. Pericardiotenesis was performed and 600 mL of clear serous fluid was aspirated, with an additional 300mL draining over the ensuing 3 days. Fluid analysis revealed an exudative, non-hemorrhagic effusion with negative cultures and cytology. Pericardiotenesis combined with thyroid hormone replacement and an optimal heart failure treatment regimen led to significant clinical improvement.

Discussion
New onset heart failure is rarely attributed to hypothyroidism. Pericardial effusions solely attributed to hypothyroidism are relatively uncommon. Earlier studies showed prevalence’s ranging from 30-80%. Kabadi et al. found only 2 out of 30 patients with primary hypothyroidism to have pericardial effusions. (9) This large difference was attributed to older studies looking primarily at patients with severe and longstanding hypothyroidism. Thus pericardial effusions may be a frequent manifestation in myxedema or severe hypothyroid states, but is rarely seen in mild or well controlled hypothyroidism. Patients with new onset heart failure must be screened for hypothyroidism given the elevated morbidity and mortality that is seen with those not treated appropriately. (4)

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**A RARE CASE OF GUILLIAN BARRE SYNDROME IN THE SETTING OF INFLUENZA VACCINATION AND VELOLIZUMAB INFUSION**

Background:
Guillain Barre Syndrome (GBS) is a rare complication of influenza vaccine administration. Studies on different influenza vaccines have not shown any significant association with GBS other than the 1976 swine influenza vaccine. Vedolizumab is a monoclonal antibody used for Crohn’s disease and there have been no case reports of GBS associated with its use. We report a case of GBS in the setting of recent induction therapy with Vedolizumab.

Case Summary:
We present a case of a 32-year-old man with past medical history of Crohn’s disease who presented with lower extremity weakness and paresthesias in his upper and lower extremities for 2 weeks. He was administered influenza vaccine for the first time in his life 4 weeks prior to the onset of his symptoms. He received 2 induction doses of Vedolizumab before his symptom onset, prior to which he was on Infliximab.

On physical examination, he was found to have absent reflexes, decreased muscle strength and decreased sensation to light touch in his lower extremities. Extensive work up including electrolytes, magnesium, serum thyroid stimulation hormone, vitamin B12 level, autoimmune work up, serum protein electrophoresis, HIV, Lyme serology, hemoglobin A1c and MRI of the cervical spine were all within normal limits. CSF studies revealed increased protein without increased white cell count. He was then admitted to the hospital and treated with intravenous immunoglobulin. His weakness and paresthesias were improving and he was discharged home to follow up with his neurologist. He underwent an electromyogram and nerve conduction studies which showed motor polynueopathy with mixed pattern of axonal and demyelinating features.

Discussion:
Both Crohn’s disease and influenza vaccination have been rarely associated with GBS. In our patient, the Crohn’s disease was not active when GBS presented making this an unlikely cause; significantly less likely a cause would be the recent influenza vaccination, with GBS felt to occur about once per one million vaccinations.

We postulate another scenario: that the influenza vaccine and Vedolizumab may have interacted to increase the risk for GBS. In such cases one would have to weigh the pros and cons of vaccination and the potential interaction with this monoclonal antibody, considering his high risk for recurrence of GBS after re exposure, the need for this medication for the treatment of his Crohn’s disease and the potential risks of developing post influenza complications (would he ever get the influenza vaccine again?)

To our knowledge this is the first case report of GBS with this combination of medications.
Resident/ Fellow Clinical Vignette

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**Turbulent Forces Within Abnormal Cardiac Anatomy May Be a Cause of Profuse Bleeding. A Case Discussion of Acquired Qualitative Defect of Von Willebrand Factor Due To Severe Mitral Valve Regurgitation**

**Introduction**  
Von Willebrand Factor (vWF) also known as Von Willebrand Factor Antigen (vWFAg) is a 2050 amino acid protein, released from endothelial cells essential in normal homeostasis. Multimers of vWF can be extremely large, more than 20,000 KDa. Functional activity of vWF is measured by Ristocetin Cofactor Assay in vitro (RCoF). Von Willebrand Disease (vWD) is a hematologic disorder which is a most common hereditary coagulation abnormality however can be acquired in several medical conditions. Acquired Von Willebrand Syndrome (AvWS) is characterized by structural and functional defects of vWF. The condition can be seen in autoimmune, lympho/myeloproliferative and cardiovascular disorders, as well as solid malignancies. Mechanism of the development of the disease can be various including: antibody mediated clearance or functional interference; adsorption of vWF into surface of transformed cells or platelets; increased shear stress and subsequent proteolysis and destruction of vWF multimers. The latest mechanism is well known in patients with valvular heart disorders and in patients with left ventricular assisted device.

**Case Report**  
41 y/o female with recent history of SOB on exertion, fatigue and episodes of profuse vaginal bleeding was referred to a cardiology and hematology clinic for work-up and evaluation. Physical examination finding were consistent for a systolic murmur. Platelet count was within normal range. Coagulation panel revealed normal PT/PTT time, normal levels of Factor VIII and Von Willebrand Factor Antigen (vWFAg), decreased multimers of vWFAg and decreased activity to Ristocetin in vitro. Echocardiogram revealed severe Mitral Saddle Valve Regurgitation (MVR) amenable to Mitral Valve Annuloplasty (MVA). Preoperatively patient responded to Desmopressin challenge, resulting in increased activity to RCoF and cessation of episodes of vaginal bleeding. Patient successfully had the MVA and was followed up in cardiology and hematology clinic in three months. Bleeding episodes stopped post operatively. Follow up lab work, revealed normal levels of Factor VIII and vWFAg accompanied by normal response to Ristocetin and normal level of multimers.

**Conclusion**  
Acquired Von Willebrand Disease may be seen in many diseases and may have different mechanism in its pathophysiology. Acquired vWD in MVR is believed to be in result of increased clearance of largest vWF multimers due to turbulent forces within abnormal cardiac anatomy. Direct proteolysis of largest multimers of vWF consequently resulting in abnormal qualitative defect in coagulation was studied in this case. Symptoms of bleeding due to qualitative defect of vWFAg may be normalized after correction of valvular abnormality such as seen in his patient.

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**A FIVE MINUTE LIFE SAVING TEST**

**Introduction:**  
Acute febrile illness is a common presentation to a primary care physician or to the ER, with a wide differential diagnosis. These cases illustrate the significance of early diagnosis and treatment of Babesia by reviewing a peripheral blood smear.

**Case 1:** A 56-year-old woman with diabetes presented with 1 week of high fevers, sore throat, nausea, vomiting, abdominal and joint pains, not relieved by Motrin given by PCP. There was no recent travel. On admission T 104 F, tachycardic, hypotensive, petechial rash above the ankles. WBC 4.6, platelets 59K, T bili 2.3, normal CXR, hepatosplenomegaly on sonogram. The patient was admitted as a case of sepsis and started with IV Vanc and Zosyn; the same date of admission patient was reevaluated for fever of 105F by the resident on call who did the five minute life saving test: A peripheral blood smear examination which revealed ring forms and a maltese cross c/w Babesia. The patient was then started on Atovaquone azithromycin and Doxycycline. The patient shown clinical improvement with resolution of parasitemia.

**Case 2:** A 69-year-old man with HTN and diabetes present with 3 weeks of high fever, abdominal pain, vomiting and dark urine. He was seen by his PCP, given symptomatic treatment. No travel history. On admission, BP 80/50 mmhg, pulse 120, T 102.2 F, pale, icteric, hepatosplenomegaly, WBC 4.6, platelets 59K, T bili 2.3, normal CXR, hepatomegaly on sonogram. The patient was admitted as a case of sepsis and started with IV Vanc and Zosyn; the same date of admission patient was reevaluated for fever of 105F by the resident on call who did the five minute life saving test: A peripheral blood smear examination which revealed ring forms and a maltese cross c/w Babesia. The patient was then started on Atovaquone azithromycin and Doxycycline. The patient shown clinical improvement with resolution of parasitemia.

**Discussion:** Both patients presented with fever and sepsis syndrome. Both were seen by their PCP prior to admission. Babesia is transmitted by Ixodes tick bite, with cases also having coexisting Lyme and/or anaplasmosis. Doxycycline was added to empirically treat coexistent parasites. Delays in diagnosis can lead to fatal complications with renal failure, ARDS and DIC. With automated differentials being done by labs, fewer smears are fully manually reviewed; Due to the suspicion of the house staff, the smear was fully reviewed, making the diagnosis of Babesia possible. The review of peripheral blood smears should still be considered a routine examination in patients presenting with febrile illness.
Resident/ Fellow Clinical Vignette

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**Dietary history in the diagnosis of Peripheral Neuropathy of unclear etiology: A Case of Mercury Toxicity**

Mercury toxicity and its consequences are well described. This diagnosis can be easily missed if the history is not taken in detail about occupational exposure or dietary habits. We describe a patient that presented with osteomyelitis and neuropathy. Further questioning of the patient led us to the diagnosis of chronic mercury poisoning.

Patient is a 42 year male with no significant past medical history, resident of New York, came with right 3rd toe and left 1st toe pain, deep wounds, swelling, and erythema in the plantar area that extended to the dorsum of foot for 3-4 weeks. Symptoms were associated with numbness, weakness, and tingling of lower extremities, depression, anxiety, and irritability for 6 months. He denied any fever, chills, nausea, vomiting, dyspnea, chest pain, cough, skin rash, forgetfulness or suicidal thoughts. On physical exam, the patient was found to have deep wounds with malodor but no active discharge of right 3rd toe and left 1st toe. The right 3rd toe had dry gangrene as well. He also had glove and stocking sensory loss extending to mid legs bilaterally with decreased deep tendon reflexes, mild muscle weakness, but with a normal gait. He also had normocytic anemia. Empirical antibiotics were started and patient underwent amputation of right 3rd toe. His blood and bone cultures were positive for Methicillin Susceptible Staphylococcus Aureus. Since there was no obvious etiology for patient’s neuropathy, more detailed history was obtained, and patient admitted to a diet that composed almost entirely of fish and other seafood. Patient’s mercury level was then collected. The blood mercury level was 20 mcg/L (normal < 5 mcg/L) but urine mercury level was normal. He was diagnosed with chronic mercury poisoning with irreversible neurological complications. There is no effective treatment for toxic organic mercury exposure. He was asked to avoid consuming fish and other seafood. Mercury toxicity can be caused by elemental mercury, inorganic mercury salts, or organic mercury. Chronic consumption of organic mercury compounds have caused severe epidemics of poisoning in Japan in the 1940s and Iraq in 1971. Exposure is mostly via consumption of mercury-contaminated fish. In this poisoning, patients may present with neuropsychiatric symptoms such as depression, anxiety, or psychosis and neuropathy. The preferred test for organic mercury toxicity is a whole blood mercury level. Organic mercury is eliminated by the fecal route so urine mercury testing is not reliable. Thus, having a high index of suspicion is important in patients with peripheral neuropathy of unclear etiology, to diagnose mercury poisoning in order to prevent irreversible damage to the nervous system.

**Introduction**

CT Angiography has been the gold standard for diagnosing pulmonary embolism with sensitivities and specificities of greater than 90%. Alternative imaging techniques - including magnetic resonance angiography - have been described to diagnose PE. We present a case of pulmonary embolism in an 83 year old woman, diagnosed by magnetic resonance angiography, in whom CTA was contraindicated.

**Case Report**

An 83 year old woman with history of late stage idiopathic pulmonary fibrosis on 4L oxygen presented to the ED with complaints of new, continuous chest pain. Other medical history included a provoked pulmonary embolism diagnosed by V/Q scan and left lower extremity peroneal DVT in 2014 for which she received three months of warfarin, and an anaphylactic reaction to iodine-based CT contrast agents.

The chest pain was initially described as substernal, pressure-like, non-radiating, and associated with shortness of breath and increased oxygen requirements. The patient was also tachypneic and tachycardic. Troponin peaked at 2 ng/mL with multiple ST depressions and T wave inversions on EKG. A D-dimer was greater than 1000 ng/mL. Due to patient’s history of anaphylaxis to iodine-based contrast it was decided to pursue a V/Q scan. The V/Q scan revealed a large perfusion defect in the right lower lobe consistent with high probability for pulmonary embolism. However, it was felt that a baseline V/Q scan for a patient with late stage IPF would demonstrate intermediate probability PE at baseline and therefore a high probability reading had substantially reduced specificity in these circumstances.

There was a question of whether or not to pursue lifelong anticoagulation for an unprovoked PE with only a suboptimal confirmatory V/Q study. Our team resorted to a seldom used technique: Gadolinium enhanced magnetic resonance angiography. MRA revealed a 4.2 x 2.2 cm right main pulmonary artery embolism. The patient subsequently elected to follow recommendations for chronic anticoagulation.

**Discussion**

This case serves as a reminder of the difficulty in making a diagnosis of pulmonary embolism in patients with underlying lung disease. According to one study of thirty consecutive patients by Meaney et al, MRA provided a sensitivity of 100%, specificity of 95%, positive predictive value of 87%, and negative predictive value of 100% when directly compared with CT angiogram. In the larger multicenter, prospective PIOPED III study with 371 adults, a technically adequate MRA had sensitivity of 78% and specificity of 99%. Sensitivities were greater for emboli located in the lobar and segmental vessels as compared to the subsegmental vessels. In conclusion, MRA remains a promising diagnostic modality for diagnosing pulmonary embolism, especially in patients with severe underlying lung disease or in whom CT angiography is contraindicated.
Resident/ Fellow Clinical Vignette

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Percutaneous Coronary Intervention at Centers with and without On-Site Surgical Backup: A Meta-Analysis and Meta-Regression

Percutaneous Coronary Intervention at Centers with and without On-Site Surgical Backup: A Meta-Analysis and Meta-Regression

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Methods:
We conducted electronic database searches in PubMed, CENTRAL, and EMBASE. The Cochrane Register, Google Scholar databases, and the scientific session abstracts were searched for eligible studies. Risk ratios and 95% confidence intervals were computed with the Mantel-Haenszel method. Fixed-effect models were used; if heterogeneity (I²)>25 was identified, effects were obtained with random models. Meta-regression analyses were performed to determine whether the effects of PCI without on-site CS were modulated by pre-specified study-level factors.

Results:
Twenty-seven studies were included with total n=8,558,618 patients. No significant difference was observed for all-cause mortality (RR 1.02, 95% CI 0.86-1.21, p=0.82, I²=97.2%), cardiovascular mortality rates (RR 1.18, 95% CI 0.93-1.50, p=0.17, I²=2.98%), myocardial infarction rates (RR 0.89, 95% CI 0.62-1.29, p=0.55, I²=88.5%), repeat revascularization (RR 0.87, 95% CI 0.43-1.76, p=0.69, I²=98.8%), stroke (RR 1.28, 95% CI 0.56-2.91, p=0.55, I²=98.8%), shock (RR 0.76, 95% CI 0.43-1.35, p=0.35, I²=93.7%), mechanical circulatory support (RR 0.83, 95% CI 0.46-1.50, p=0.53, I²=99.8%), bleeding (RR 0.88, 95% CI 0.43-1.81, p=0.73, I²=99.6%), and emergency CABG (RR 0.97, 95% CI 0.64-1.46, p= 0.87, I²=84.1%). In a meta-regression analysis, the effect of PCI without on-site CS, baseline clinical features did not affect the long-term all-cause mortality outcome.

Conclusion:
There was no significant difference in complications rates, and clinical outcomes for PCI performed at centers without on-site CS compared to centers with on-site CS.

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Acute Aortic Dissection manifesting as Paraplegia

Aortic dissection is the leading cause of death among aortic pathologic conditions. It is a catastrophic disease and requires very prompt treatment. The mortality rate is about 50% within 48 hours of onset. Patients typically present with sudden onset of severe chest pain, but in about 10% of patients, dissection is painless. Painless paraplegia as a presenting manifestation of aortic dissection is exceedingly rare and limited to a few case reports in the literature. A 60 year old Caucasian female who presented to our emergency room due to fall and inability to walk. She had a sharp throat pain and fell due to sudden onset of pain, weakness and numbness of the legs. She denied any chest pain or back pain except for intermittent abdominal pain. Her medical history included hypertension, depression and was on metoprolol and venlafaxine. Her vital signs were as follows: blood pressure 82/42 mmHg, pulse 94 bpm & regular, respiration 16 breaths/min, temperature 96.5 F, and saturation 99% on room air. She appeared mildly lethargic but answered questions appropriately. Her cardiac and respiratory systems on exam were normal. Distal pulses were very weak and both legs were pale and cool to touch. Neurologic exam revealed cranial nerves II to XII intact. Deep tendon reflexes were absent in patient’s legs. She had motor deficits in lower extremities bilaterally. Intravenous fluid boluses were given with continuous infusion. EKG showed sinus rhythm at 95 bpm. Chest X ray revealed mild vascular congestion, no mediastinal widening. Complete blood count was normal and complete metabolic panel revealed renal insufficiency. Due to high suspicion of aortic dissection, Chest CT angiography was done revealing aortic dissection with hemorrhage and cardiac tamponade with dissection extending into the abdominal aorta. Contact for transfer to tertiary institution for surgical management was made. Blood pressure was not responding to intravenous fluid and dopamine was initiated with improvement in blood pressure (90/60 mmHg). EMS arrived within 20 minutes of order for transfer, but patient seized and lost pulses. CPR was initiated, she was intubated and was successfully resuscitated, and transferred to tertiary institution. Follow-up revealed patient underwent a 16.5-hour procedure for repair. Day 1 postoperative, patient was still comatose. In acute aortic dissection, neurological manifestations are not uncommon. Occasionally, aortic dissection may extend into the iliac, femoral, or superficial femoral arteries and cause leg ischemia. In our patient, the presentation of fall due to paraplegia is a result of dissection extending and involving the iliac and femoral arteries. Our patient complained of intermittent abdominal pain but no chest pain. The team maintained a very high index of suspicion which led to early diagnostic studies and transfer to a tertiary institution for surgical management.
Resident/ Fellow Clinical Vignette

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**Calcium-phosphate deposits in tumor lysis syndrome with reversal of oliguric renal failure with early hemodialysis.**

Tumor lysis syndrome can lead to acute kidney injury by two different mechanisms: (a) uric acid crystallization in the kidney tubules and (b) deposition of calcium-phosphate product. We are presenting a 54 year old female with lymphoma that developed tumor lysis syndrome and oliguric acute tubular necrosis. The patient had low calcium levels and severe hyperphosphatemia of >15 mg/dl, meeting the remote criteria of calcium-phosphate product of >70 mg2/dL2 for intermittent maintenance hemodialysis. CT without contrast of the abdomen and pelvis demonstrated evidence of several calcium-phosphate deposits and nephrocalcinosis (figure 1). Subsequently, with hemodialysis and gentle hydration with normal saline for prevention of further crystal deposition in the renal tubules and interstitium she recovered her renal function to the baseline with normalization of all electrolytes including phosphorus in three days. To our knowledge, we are presenting the first case of complete reversal of oliguric renal failure from calcium-phosphate deposition with our unique approach of early hemodialysis initiation based on hyperphosphatemia and elevated calcium phosphate product >70 mg2/dL2. These deposits, containing calcium and phosphate ions, precipitate as hydroxyapatite crystals which leads to nephrocalcinosis and irreversible kidney damage (chronic kidney disease). On the basis of this finding, one might postulate that early initiation of hemodialysis and clearing the phosphate load may prevent acute kidney injury and halt the ongoing kidney damage. We suggest that all patients with tumor lysis syndrome should be considered for early hemodialysis with isolated hyperphosphatemia and renal failure, even in the setting of normal uric acid and potassium levels to prevent further worsening of renal parameters.

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**Thrombocytopenia and Hemodialysis - The membrane matters**

Introduction:
Substantial activation of platelets can occur in the course of hemodialysis (HD). Typically, the platelet count decreases slightly during the first hour of dialysis, but mostly returns to initial values by the end of dialysis. We present a patient with End stage renal disease (ESRD) who developed recurrent thrombocytopenia following dialysis, which resolved after changing the type of the dialyzer membrane.

Case:
A 55 year old female with a history of hypertension, diabetes mellitus and chronic kidney disease stage 4 presented with shortness of breath on exertion and bilateral pedal edema. She had stopped her diuretics 6 weeks ago and noted worsening symptoms and decreased urinary output. Vitals were stable and physical examination revealed facial puffiness, bibasilar crackles and pedal edema. Laboratory studies on admission showed WBC 2.5, Hb 5.4, platelets 75, BUN 96 and creatinine 6.4. Six months ago her creatinine was 3.1, with Hb 7.3 and WBC 3.1. She was given IV diuretics, with worsening of the renal function. One unit of platelets was transfused and a dialysis catheter was inserted. HD three times per week was started with a polysulfone membrane dialyzer. The platelet count dropped after the first HD from 76 to 43. Thereafter platelet count was noted to drop by 10-20x10^9/L after every HD. Heparin locks were switched to saline locks in the dialysis catheter. Heparin-PF4 antibody assay was negative. Bone marrow biopsy was unremarkable and serum immunoelectrophoresis revealed no monoclonal gammopathy. Hemoglobin and bilirubin remained stable. Platelet nadir was 12x10^9/L without any hemorrhagic complications. The dialyzer membrane was changed to a cellulose triacetate membrane and the post dialysis drop in platelet count stopped. She was discharged with a stable platelet count of 54x10^9/L.

Discussion:
Our patient was initially dialyzed with a polysulfone membrane, which is sterilized by electron beam radiation (EBR). The exact mechanism of thrombocytopenia is unknown but it has been hypothesized that exposure to EBR may change membrane structure leading to platelet activation, aggregation or adsorption. EBR of dialyzer membranes can also produce intermediary substances, which could cause platelet activation. The thrombocytopenia resolved upon switching the dialyzer membrane to a cellulose triacetate membrane sterilized by gamma radiation. The relatively lower platelet aggregation with this membrane was likely due to decreased activation of bound glycoprotein IIb/IIIa. Dialyzer hypersensitivity symptoms are infrequently associated with a fall in platelet count. Most recent cases of dialysis-associated thrombocytopenia have been reported with polysulfone membranes. It is important for clinicians to be aware that in patients undergoing HD, polysulfone dialyzer could lead to thrombocytopenia. Switching to a cellulose membrane dialyzer could be a better option in these patients.
Introduction:
Influenza and Mycoplasma are two common respiratory pathogens well documented to cause co-infections and super-infection. Pancreatitis is a rare documented complication of both infections. We report a case of acute pancreatitis associated with Influenza and Mycoplasma co-infection.

Case:
A 50 year old obese female with past medical history of hypertension and hypercholesterolemia presented to the emergency with complaints of multiple episodes of nausea and bilious vomiting for 3 days. It was associated with a dull and persistent epigastric pain, 5/10 in intensity, radiating to the back. She also noticed positional dizziness and decreased urine output for 2 days. She denied any respiratory complaints. She never consumed alcohol and had no prior history of gallstones or abdominal surgery. On presentation, temperature was 36.6 °C, blood pressure was 72/54 mmHg, heart rate was 116/minute and respiratory rate was 29/minute with oxygen saturation of 95% on room air. Patient was dehydrated, had feeble peripheral pulses and mild epigastric tenderness. Laboratory studies revealed leukocytosis of 40.9 x 10^9/L with 87% neutrophils, acute kidney injury (AKI) and an elevated lipase of 1127 U/L. Blood gas analysis showed mixed respiratory alkalosis and metabolic acidosis with an increased lactic acid. Transaminases, alkaline phosphatase, bilirubin, calcium and triglycerides were normal. A left basilar infiltrate was noted on chest X-Ray. Abdominal imaging showed diffuse enlargement of the pancreas with surrounding edema and fluid with no gallstones, biliary sludge or duct dilation. Molecular nasal swab was positive for Influenza A viral RNA and Enzyme Immunoassay was positive for Mycoplasma pneumoniae. Blood and urine cultures were negative. She had an APACHE score of 19, Alveolar-arterial gradient of 70 mmHg with a PaO2/FiO2 ratio of 350. She was admitted to the intensive care unit for acute pancreatitis with hypovolemic shock and AKI. She was treated with aggressive fluid resuscitation and broad-spectrum antibiotics, which were later switched to Azithromycin and Oseltamivir. The patient showed hemodynamic improvement. Her leucocytosis and AKI also improved. Lipase eventually trended down to 176 U/L. She responded appropriately to the treatment and made a full recovery.

Discussion:
Acute pancreatitis associated with mycoplasma infection is rare and its presentation ranges from painless pancreatitis to severe necrotizing pancreatitis. It is hypothesized that mycoplasma causes local cytokine production in the tissue either from direct effect of bacterial lipopolysaccharides or from altered immune modulation. Also, the systemic hypercoagulable state with infection may lead to vascular occlusion to the organ. Meanwhile, acute pancreatitis is also a rare manifestation of influenza infection. In vitro experiments have demonstrated the ability of influenza virus to infect human pancreatic cell lines. Hence, it is important for clinicians to be aware that mycoplasma and influenza co-infection could be associated with pancreatitis with varying severity.
Severe Pulmonary Hypertension Medically Optimized Prior To Successful Heart Transplantation

Introduction
Pulmonary hypertension (PH) is a common complication of left heart disease. Irreversible PH with an elevated pulmonary vascular resistance (PVR) > 5 Woods units or transpulmonary gradient (TPG) > 15 is a contraindication to orthotopic heart transplant (OHT) because of an increased risk for right heart failure (HF) and death. We present a case of initially severe and fixed PH in a patient with advanced HF.

Case
Our patient is a 33-year-old Chinese man with a history of severe Pulmonary Hypertension. He had a history of autoimmune disease, liver disease, HIV, thromboembolic disease, diet drug use, or a family history of HF. Right heart catheterization (RHC) revealed severe PH with a pulmonary artery pressure (PAP) of 86/40 (59) mmHg, an elevated pulmonary capillary wedge pressure (PCWP) of 17 mmHg, a TPG of 42, and a PVR of 19.12 Woods Units (Figure). Dobutamine lowered the PCWP but increased PAP. Nitric oxide improved PVR but both the PVR and TPG remained too high for OHT candidacy. Treatment with dobutamine, furosemide, bosentan 125 mg twice a day, and sildenafil 20 mg three times a day was started and hemodynamics followed daily. After 1 month of treatment, the PAP improved gradually but the TPG remained above 15. An intra-aortic balloon pump (IABP) was placed with the plan for left ventricular assist device (LVAD) insertion as bridge to transplant. Within 24 hours, the mean PAP decreased significantly, proving reversibility of PH. He successfully underwent OHT 1 month later without any post-operative right HF.

Conclusion
Irreversible PH secondary to left sided HF is a contraindication to OHT. This case shows that initially severe, fixed PH could be improved with optimization of HF and PCWP, off-label pulmonary artery therapies, and an IABP. An aggressive strategy of medical therapy in select OHT candidates with PH may obviate the need to implant an LVAD.

Resident/ Fellow Clinical Vignette

A Rare Case of Extramedullary Leukemic Infiltration of Bilateral Breasts as a First Sign of Relapse in AML

INTRODUCTION
Extramedullary spread of AML to CNS, lungs, and pericardium is commonly described, however breast infiltration is rare. More commonly, we see a primary leukemic infiltration of the breast called a granulocytic sarcoma before bone marrow involvement with AML. Breast infiltration in AML patients remains a rare occurrence, especially in patients that have been treated into remission. This case describes an extramedullary leukemic infiltration of bilateral breasts as the first sign of systemic relapse in AML. A prompt diagnosis changes staging, management and signifies a poor prognosis compared with AML without extramedullary breast involvement.

CASE
A 24-year-old woman was diagnosed with AML, 96% blasts on bone marrow biopsy, negative molecular markers, and a normal FISH. Initial treatment was 7+3 induction (cytarabine & daunorubicin), complicated by malignant pleural effusions. She then underwent consolidation chemotherapy with FLAG-IDA, and a subsequent bone marrow biopsy showed complete remission. Two months later she presented to clinic with complaints of progressive bilateral breast changes: the right breast had enlarged, was firm to touch with everted nipple and had a stuck-on appearance to chest wall; the left breast had grown three times in size with everted nipple with crustig, was edematous, and had peau d’orange texture. At time of new presentation, medical history included thalassemia trait, depression, CHF, and SVC syndrome, for which pt was on cymbalta, metoprolol, lasix, and xarelto; lab work-up showed WBC 6.84 K/uL, Hgb 9.6 g/dL, lymphocytes # 2.33, neutrophils # 2.76, monocyte 11.4 %, eosinophil 8.5 %, atypical lymph 2 %; CMP was unremarkable. MRI showed non-specific adenopathy with enhancement throughout right breast and in upper half of left breast, appearance consistent with fulminant mastitis vs diffuse leukemic infiltrate. Repeat bone marrow biopsy showed normocellular marrow with 1% blasts. Right breast core biopsy showed a diffuse infiltrate of immature myeloid blasts (confirmed by flow cytometry), with destroyed ductal structures. A sibling allogeneic stem cell transplant with curative intent was held due to relapsed AML and she underwent salvage Clobarabine/Ara-C chemotherapy.

DISCUSSION
Leukemic breast involvement constitutes only 0.25% of all breast tumors. A review article described only 105 cases of AML with breast involvement in the last 45 years. Of those, only 31 developed breast involvement after initial treatment, and of those only 6 patients were in the 20-29 age group. Bilateral presentation is rare, as in 85% of the cases, there is single breast leukemic infiltration. In any patient with malignancy, an enlarging breast mass must be biopsied. Regardless of whether the marrow demonstrates remission, a breast infiltration signifies systemic leukemia. These lesions are more resistant to therapy and prompt aggressive salvage therapy. The prognosis is worse compared to patients without extramedullary involvement with no reports of long disease-free survival.
Resident/ Fellow Clinical Vignette

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What makes the lungs tick?

Introduction: Atypical pneumonia secondary to Anaplasma is an uncommon presentation for the pathogen and a rare etiology of pneumonia. The following case presents a patient with pneumonia, elevations of liver function tests (LFTs), leukopenia and thrombocytopenia.

Case Description: A 64-year-old woman with history of osteoporosis presented to the hospital with 4 days of fever, whole-body aches, nausea, vomiting and poor appetite. One-week prior, she reported having a runny nose and cough. She endorsed recent travel to upstate New York where she went hiking but had no recalling of tick bites nor skin rashes. On admission, the patient was feeling short of breath, weak and distressed. Her temperature was 100.5 F, heart rate of 92 bpm, respiratory rate of 18 bpm, blood pressure of 98/48 mmHg and SpO2 of 93% on room air. On physical exam, was found to have crackles at both lung bases, with no other positive findings. A chest x-ray showed left lower lobe alveolar opacities. Laboratories were pertinent for pancytopenia, WBCs 4.0 K/uL, platelet count 53,000 K/uL, AST 179 and ALT 169. A clinical diagnosis of atypical pneumonia was made and the patient was treated with ceftriaxone and azithromycin. Despite the initial antibiotic regimen, the patient developed hypoxic respiratory failure that required high flow oxygen therapy. Due to a high suspicion of a tick-borne illness and lack of clinical improvement, azithromycin was changed for IV doxycycline. Anaplasma, Borrelia and Ehrlichia serologies were all initially negative. Due to the high suspicion of a tick-borne illness, azithromycin was changed for IV doxycycline. Over the course of the next 72 hours, the patient showed significant clinical improvement and resolution of the hypoxic respiratory failure. However, the patient persisted with leukopenia, thrombocytopenia and elevated LFTs. The patient was discharged on oral doxycycline and was followed as outpatient where laboratory abnormalities resolved and titers of IgM and IgG for Anaplasma phagocytophilum became positive at 1:256 and 1:1280 respectively, confirming the suspect diagnosis.

Discussion: This case illustrates the importance of maintaining a high degree of clinical suspicion in a patient with a clinical scenario suggestive of a tick-borne illness despite negative serologies. As it was done in this patient with pneumonia, worsening hypoxic respiratory and failure of improvement to the initial antibiotic regimen. A. phagocytophilum is an uncommon cause for pneumonia but should be considered in the setting of appropriate travel history and elevation of LFTs, leukopenia and thrombocytopenia. Supportive treatment with high flow oxygen therapy was important in the management of this patient possibly avoiding mechanical ventilation and should its usage should be considered more often in these circumstances.

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A Rare Case of Drug-Induced Liver Injury Following Prophylactic Dose of Unfractionated Heparin

INTRODUCTION:
Administration of unfractionated heparin (UFH) has been associated with elevated liver enzymes in patients undergoing anticoagulation treatment. Case studies have reported transaminitis, particularly in intravenous heparin use. Rarely, however low dose administration of subcutaneous injection has been linked to this side effect. UFH is now a standard practice for deep venous thrombosis (DVT) prophylaxis in hospitalized patients at higher risk for thromboembolic events. Considering the importance of both treatment modalities in patient medical care, physicians need to monitor liver function to prevent acute liver injury.

CASE DESCRIPTION: A 58 year old man with a past medical history of vestibulopathy of unknown etiology presented with lightheadedness of 15 minutes duration and was admitted for syncope work-up. Prior to admission his daily medications included aspirin, atorvastatin (low intensity 10mg), finasteride, lisinopril, metoprolol, omeprazole, and tamsulosin. The initial physical exam, electrocardiogram, chest x-ray, complete blood count and comprehensive metabolic panel were all unremarkable. The only new medication added to his regimen was subcutaneous heparin 5000 Units every 8 hours for DVT prophylaxis. In the course of the hospitalization elevation of liver enzymes was observed. By 72 hours post-admission, alanine aminotransferase (ALT), aspartate aminotransferase AST and alkaline phosphatase (ALP) had reached 393, 219, 188, respectively, as compared to ALT 14, AST 15 and ALP 80 at admission. Also of importance, his total bilirubin reached a peak of 1.8 as compared to his baseline value 0.5.

DISCUSSION: Approximately 5% of patients receiving UFH will develop transit elevations of serum transaminases within the first 4 days of treatment. The proposed pathophysiology includes direct toxicity to hepatocytes, hepatocyte membrane modification, and immune-mediated hypersensitivity reactions. Elevated ALT and AST levels are reported to be more common than elevated ALP. Enzyme levels typically normalize within 2 weeks following discontinuation of heparin. While abnormal bilirubin levels are rarely identified in previous case studies, our patient demonstrated not only elevated AST, ALT, ALP, but increased total bilirubin as well. In this particular case, the transient transaminases returned to baseline within 2 weeks of heparin discontinuation.

CONCLUSION:
We recommend close monitoring of these specific lab values in patients receiving UFH. Heparin discontinuation typically results in normalization of liver function. A cautious use of UFH is warranted, particularly in patients with additional risk factors for liver injury, including but not limited to alcohol abuse or simultaneous treatments with therapy known to be hepatotoxic. In addition, awareness that the acute liver injury can be a side effect of UFH can prevent unnecessary testing for evaluation of liver conditions and thererfor reduce prolonged hospitalization and healthcare cost.
A RARE CASE OF HYPERTHYROIDISM PRESENTED AS SEVERE HYPERCALCENIA

Hypercalcaemia secondary to thyrotoxicosis has been attributed mainly to the thyroid hormone mediated an increase in osteoclast activity. A methodical approach to hypercalcaemia generally leads to a diagnosis of the underlying pathology. Yet a physician can not let a step by step approach deter them from intellectual curiosity. In this case an otherwise healthy female was admitted for sever symptomatic hypercalcaemia which turned out to be impending thyroid storm.

A 49-year-old female with no significant past medical history came to the Emergency Department for recent unexplained weight loss, altered mental status, and lethargy. Three months prior to her arrival, her husband passed away, and she was diagnosed with bereavement and depression. For the past two months the patient had progressively become debilitated with lethargy and abdominal pain. We were unable to obtain a reliable history due to lethargy and severe discomfort. Laboratory value was significant for an elevated corrected calcium level of 14.2 mg/dl. With recent unexplained weight loss and hypercalcaemia the patient was admitted to inpatient service for management of symptomatic hypercalcaemia. The patient was started on intravenous hydration, bisphosphonates, and calcitonin. The following day her PTH returned with a decreased value of 7.47 ng/L. Evaluation of malignancy with tumor markers and imaging was unremarkable. The morning post admission, the patient started to deteriorate. She developed a fine tremor, vomiting, and worsening of her mental status. TSH was found decreased (0.01 &#181;U/mL), with an elevated T4 (5.76 ng/dl). Utilizing the Burch Worthington grading criteria, we determined she was in a state of impending thyroid storm with a score 25. The patient was immediately started on Methimazole, Propranolol, Iodide, Hydrocortisone, and was transferred to the intensive care unit (ICU). Within hours upon transfer to the ICU, the patient developed tachycardia, fever, nausea, vomiting, and diarrhea, indicating thyroid storm with a Burch Worthington score of 40. Within 36 hours of starting proper treatment for thyrotoxicosis the patient stabilized and mental status had improved. The patient was further managed on the floor for intractable diarrhea that caused decreased phosphate and potassium levels. After a week of treatment the patient’s calcium level had normalized, and she was able to be discharged with her physical and mental health intact. Hyperthyroidism can cause mild hypercalcaemia in 20 percent of thyrotoxic patients. The patient in our case had severe hypercalcaemia and the diagnosis of thyrotoxicosis was delayed. If missed, the consequences of thyroid storm are fatal. Only a small percentage of patients with hyperthyroid develop storm, but if left untreated has a mortality rate of 50-90 percent. With early recognition and treatment the mortality can dramatically decrease to less than 20 percent.
Developing rapidly progressive ILD. ILD associated with this characteristic rash and ILD. Further serology can confirm the myopathy which can be initially misleading. A high index of dermatomyositis (shawl rash), without the inflammatory amyopathic dermatomyositis with predominant skin and lung dysfunction. The patient is showing improvement in her Forced Vital Capacity (1.24L to 1.51L) and oxygen saturation was started on rituximab and after her second treatment, antibody which is specific. She was treated as an outpatient with prednisone for intractable fever and pancytopenia with hemolysis. Two months earlier she had developed a rash across her upper chest and a bilateral palmar erythematous rash with nodularity and blistering. Outpatient biopsy showed a mild perivascular lymphocytic dermatitis with epidermal hyperplasia and hyperкерatosis, which was non-specific. She was treated as an outpatient with prednisone for the rash. It had been tapered off just prior to admission, when the rash. She was initially treated for cough and fatigue. She denied fever, chills, chest pain, joint pains or proximal muscle weakness. She was initially treated for possible pneumonia with broad spectrum antibiotics. A CT Chest revealed bilateral ground-glass opacities without honeycombing and with central and traction bronchiectasis. Given her rash and CT chest findings, an autoimmune panel was ordered. Rheumatology and pulmonology were consulted. Blood work was notable for a mildly positive ANA (1:40) and elevated ESR of 40, but negative for CPK, anti-dsDNA, anti-Jo, ANCA, anti-Ro, anti-La, anti-U1RNP, anti-Smith, and aldolase. The patient was started on high-dose steroids for suspected ILD. An extended myositis panel was positive for anti-MDA5 antibody which is associated with dermatomyositis. The patient was started on rituximab and after her second treatment, mycophenolate was added. At three month follow-up, the patient is showing improvement in her Forced Vital Capacity (1.24L to 1.51L) and oxygen saturation. The steroids are being tapered and she is now maintained on mycophenolate. Discussion: Our case outlines a striking presentation of clinically amyopathic dermatomyositis with predominant skin and lung findings. Our patient had subtle characteristic features of dermatomyositis (shawl rash), without the inflammatory myopathy which can be initially misleading. A high index of suspicion is warranted in patients presenting with a characteristic rash and ILD. Further serology can confirm the presence of anti-MDA5 antibody and increased risk of developing rapidly progressive ILD. ILD associated with this subtype portends a graver prognosis, so early detection is key to initiate prompt treatment.

Discussion: Babesiosis is a zoonotic disease caused by the protozoa Babesia. Babesia infects humans via the ixodes tick and can cause lysis of the host’s red blood cells. Often times a co-infection with Lyme Disease can be seen as the disease is transmitted by the same tick vector. While Lyme Disease in the New England area is common, there has recently been a rise in Babesia co-infection in the region. We present a case of Babesiosis co-infected with Lyme in an immunosuppressed patient that had recently been to Upstate New York.

Case Description: A 50 year old female with a past medical history of Rheumatoid Arthritis (on Methotrexate since 2015) presented with a 1 month history of diffuse myalgia, fatigue, anorexia, and fever (max temperature 102°F unresponsive to Tylenol), after travel to a farm in Upstate New York 1 month prior to symptom onset. The patient reported that she had initially been given a 10 day course of Cephalexin for cellulitis by PCP which briefly improved symptoms, however they returned soon after. The patient did not note seeing any tick bites on herself. On admission the patient met SIRS criteria (fever and tachycardia); labs during admission were significant for pancytopenia with an elevated LDH of 312 indicating hemolysis. Subsequent microbiology workup showed positive Hemacolor Stain for Babesia with 1% Parasitemia and positive Lyme IgM and IgG serology. The patient was given Azithromycin and Atovaquone for 7 days for Babesiosis and Doxycycline for 14 days for Lyme - symptomatic improvement was seen within 24 hours. Subsequent 2 week follow-up showed resolution of fevers, with follow-up CBC for pancytopenia improvement pending and follow-up Babesia PCR to be done in 3 months to verify parasitemia clearance.

Discussion: Babesia microti is the predominant species to infect individuals in the U.S. This patient’s symptoms presented 1 month after visiting an endemic area, which is consistent with the median incubation interval for B.microti (37 days). The disease is subclinical however in cases of immunosuppression (as in this patient who was on chronic methotrexate) it can manifest with fever, malaise, myalgia, anorexia, as well pancytopenia with hemolysis. Often times, a concomitant rash will herald Lyme co-infection: not seen here, however. As observed, response to Azithromycin, Atovaquone, and Doxycycline can be rapid. While Babesia is rare, there has been a rise in its incidence in the New England area presenting as co-infection with Lyme, as can be seen in this case. Clinical symptoms may be significant in those that are immunosuppressed, however response to antibiotic therapy is prompt. We recommend high suspicion for Babesia in patients with recent visits to areas endemic for Ixodes tick in New England who present with intractable fever and pancytopenia with hemolysis.
**Resident/ Fellow Clinical Vignette**

<table>
<thead>
<tr>
<th>Nafia Sayedy MD</th>
<th>Imran Sayeedi MD</th>
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<tr>
<td>M. Sayedy MD; D. Kagolanu MD; P. Anand MD; C. Wankhade; R Seidman MD</td>
<td>Beekman, K; Kapoor, A; Mufuka, NH; Aijaz, H; Ferdosian, B; Qureshi, R; Petrossian, R</td>
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<td>Nassau University Medical Center</td>
<td>Flushing Hospital Medical Center</td>
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<td><strong>Statin Induced Autoimmune Myopathy</strong></td>
<td><strong>MASKING PRESENTATION: ATYPICAL GUILLAIN-BARRÉ SYNDROME</strong></td>
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<td>Myopathy associated with statin therapy is one of the most reported adverse effects leading to discontinuation of the medication. Necrotizing myopathy is a rare but a serious disease that can be caused due to statins. It is seen in 1 out of every 100,000 persons. We present a case of Statin Induced Autoimmune Myopathy (SINM), which progressed over many years after the cessation of statin use. This disease lead our capable patient to become wheelchair bound and prevented her from being able to perform activities of daily living.</td>
<td><strong>ABSTRACT</strong></td>
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<td>A 75 year-old Caucasian female with a history of hypertension, arthritis, hypothyroidism, migraines and depression presented with worsening bilateral weakness of both upper and lower extremities. She was prescribed Lipitor 20mg daily for hypercholesterolemia 7 years prior. She stated that recently while driving her vehicle, she was unable to lift her right foot from the gas pedal to brake and was forced to use her right hand to help lift her right leg/foot onto the brake pad to safely stop her vehicle. As her lower extremity muscle weakness progressed, she was advised to discontinue Lipitor after an electromyogram (EMG) revealed myopathic changes with myotonia in lower extremities strongly suggesting statin induced myopathy. Three months after stopping Lipitor, she had a right arm muscle biopsy due to worsening symptoms and persistently elevated creatinine kinase (CK) levels around 4,000U/L, which helped reaffirm concern for immune mediated necrotizing myopathy secondary to statin therapy.</td>
<td>Diagnosis of Guillain-Barré Syndrome (GBS) is dependent upon high degree of clinical suspicion. Late recognition can delay treatment, negatively impacting recovery and prognosis. We discuss a case of unilateral weakness, urinary incontinence, and normoreflexia with non-diagnostic initial work-up. Findings suggestive of GBS on electromyography (EMG) eventually prompted initiation of proper treatment. This case highlights the importance of clinical suspicion despite atypical presentation.</td>
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<td>Our patient is a 19-year-old female presenting with left lower extremity weakness, associated tingling, and decreased sensation for four days. Physical exam showed decreased proximal left lower extremity motor strength and sensation with distal tendon reflexes (DTRs) intact. She reported inability to urinate, requiring catheterization. Initial work-up and imaging were unremarkable, including MRI of the brain, cervical and thoracic spine. Lumbar puncture was performed with normal cerebrospinal fluid (CSF) analysis. Intravenous steroids were given for two days with subjective improvement of symptoms, after which the patient left against medical advice. However, she returned one week later with worsened left lower extremity motor strength, left foot drop, and absent DTRs. EMG was performed demonstrating severe peripheral neuropathy and clinical diagnosis of GBS was made. Confirmatory lumbar puncture was deferred and intravenous immunoglobulin (IVIG) administration resulted in complete symptomatic resolution.</td>
<td><strong>CASE PRESENTATION</strong></td>
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<td>She was prescribed Lipitor 20mg daily for hypercholesterolemia 7 years prior. She stated that recently while driving her vehicle, she was unable to lift her right foot from the gas pedal to brake and was forced to use her right hand to help lift her right leg/foot onto the brake pad to safely stop her vehicle. As her lower extremity muscle weakness progressed, she was advised to discontinue Lipitor after an electromyogram (EMG) revealed myopathic changes with myotonia in lower extremities strongly suggesting statin induced myopathy. Three months after stopping Lipitor, she had a right arm muscle biopsy due to worsening symptoms and persistently elevated creatinine kinase (CK) levels around 4,000U/L, which helped reaffirm concern for immune mediated necrotizing myopathy secondary to statin therapy.</td>
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<td>GBS has an incidence of 1 to 2 cases per 100,000 annually, with typical symptoms of progressive symmetrical muscle weakness accompanied by depressed or absent DTRs in 90% of patients, however reflexes may be intact early in presentation. Paresthesias may occur with mild sensory symptoms. Dysautonoma occurs in 70% of patients, presenting as tachycardia, incontinence, blood pressure dysregulation, arrhythmia, and ileus. Posterior reversible leukoencephalopathy syndrome has been reported in patients with dysautonomic hypertension. Lumbar puncture normally shows elevated CSF protein and normal white blood cell count (i.e. albuminocytologic dissociation) in more than 60% of patients one week from onset of symptoms. EMG studies can show demyelination or an axonal picture, further classifying variants of GBS. Nerve conduction studies in GBS may not become positive until two weeks after onset of symptoms. MRI may show enhancement of intrathecal spinal nerve roots and cauda equina. Treatment includes supportive care, IVIG, and plasmapheresis. Patients may require closer monitoring in an intensive care unit for increasing weakness, respiratory depression, and dysautonoma.</td>
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<td><strong>CONCLUSION</strong></td>
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<td>Antibodies against HMG-CoA reductase are the likely cause for SINM. As SINM is newly documented, it is crucial to recognize the unique clinical and histological profile differentiating SINM from other myopathies. In doing so, as with the case above, it is our hope that clinicians will be more adept at recognizing this diagnosis and treat as earlier as possible.</td>
<td>Although rare, atypical presentation of GBS can hinder diagnosis. Typical features of symmetrical weakness, hyporeflexia, and albuminocytologic dissociation may be absent, particularly early in disease course. Diagnosis should not be excluded based on atypical findings, and clinical suspicion should remain to prevent progression of a potentially fatal disease.</td>
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Background: Nafcillin induced acute liver injury is a rare and potentially fatal complication that has been known since the 1960s but inadequately studied. At this time, the only proven treatment is early discontinuation of the drug. Because of the high prevalence of nafcillin class antibiotic use in the United States, it is important for clinicians to have a high clinical suspicion for this diagnosis. Our case highlights the importance of early detection and recognition of the signs and symptoms early in the disease course.

Case Presentation: A 68-year-old male with a history of methicillin-sensitive staphylococcus and L3/L4 osteomyelitis on antibiotic therapy presented with painless jaundice after starting intravenous nafcillin therapy. Prior to starting the medication, the patient had liver enzymes within normal limits and normal bilirubin levels. At the time of presentation, the patient’s lab work exhibited a bilirubin/direct bilirubin of 9.4/8.2 mg/dL; alkaline phosphatase of 311 IU/L; AST/ALT of 109/127 IU/L. The patient was switched to IV vancomycin given concern for drug-induced liver injury. Imaging did not show obstruction of the hepatobiliary or pancreaticobiliary trees. Serology was unremarkable for viral etiology, autoimmune processes, Wilsons disease, and hemochromatosis. A liver biopsy showed findings consistent with drug-induced liver injury. The patient’s liver function tests peaked at day seven of admission and trended towards normal levels with cessation of nafcillin therapy. The patient was discharged with a diagnosis of nafcillin induced acute liver injury.

Conclusion: Nafcillin-induced liver injury is a rare process that is associated with high levels of morbidity and mortality. We present a case of liver failure attributable to nafcillin use that necessitated discontinuation of the drug. Our case highlights the importance of early recognition of the diagnosis and careful monitoring of liver function when nafcillin is employed in the clinical setting.

Introduction: Enterocutaneous fistula (ECF) represents one of the most protracted and difficult problems with substantial morbidity and mortality rates. For successful management of ECF, it is imperative to pinpoint their exact location; however, in patient with complicated anatomy, radiological techniques such as fistulogram or CT abdomen may not be very helpful. Herein, we demonstrate a novel technique of using oral contrast mixed with methylene blue dye (MB) to help us better confirm the presence of fistula.

Case: A 76 years old woman with past medical history of diabetes mellitus and atrial fibrillation was admitted for right lower quadrant (RLQ) abdominal pain. The site of pain revealed a cutaneous opening with associated serosanguineous discharge. CT abdomen and pelvis was done which revealed a possible abscess in the lower abdominal wall and an equivocal communication with the small bowel. Fistulogram was done without any clear evidence of ECF. Hence, it was presumed that the likely source of the discharge is from the sinus opening associated with abdominal wall abscess. During her hospitalization, the discharge became foul smelling with some feculent material; however, in patient with complicated anatomy, management of ECF, it is imperative to pinpoint their exact location; however, in patient with complicated anatomy, radiological techniques such as fistulogram or CT abdomen may not be very helpful. Herein, we demonstrate a novel technique of using oral contrast mixed with methylene blue dye (MB) to help us better confirm the presence of fistula.

To elucidate the anatomy and confirm the presence of fistula, we performed a CT enterography (CTE) with gastrografin oral contrast. To help with visual confirmation, we mixed 10 cc of MB in 1100 cc of gastrografin. CTE revealed findings compatible with ileo-cutaneous fistula. Visual confirmation was attained with blue stained contrast draining from the fistulous opening in the RLQ. Patient had a retrograde double balloon enteroscopy which revealed a necrotic area with fistulous opening in the ileum. Biopsy results of the necrotic area revealed no evidence of malignancy. An exploratory laprotomy revealed a small previously placed mesh eroding into the small bowel and the abdominal wall. The infected mesh was extracted with enterotomy into small bowel that was resected with primary anastomosis of small bowel.

Discussion: Management of ECF is dependent upon localizing the exact etiology and location. In our case with help of MB stained gastrografin, we demonstrated both visual and radiological confirmation of the presence of ECF. Barium was not used in our patient due to the risk of leakage induced chemical peritonitis. We recommend use of diluted MB for visual confirmation of fistulas in patients with complicated gastro-intestinal anatomy.
# Resident/Fellow Clinical Vignette

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**MITRAL STENOSIS WITH VENTRICULAR TACHYCARDIA IN PREGNANT PATIENT: AN UNUSUAL PRESENTATION**

**INTRODUCTION:**  
Rheumatic mitral stenosis has classically been associated with left atrial arrhythmias. The incidence of ventricular arrhythmias in rheumatic heart disease has rarely been described. We present a uniquely challenging case of a pregnant female with rheumatic mitral stenosis and ventricular tachycardia.

**CASE DESCRIPTION:**  
A 34 year old female in her fourth pregnancy without any prior obstetric complications presented with transient loss of vision of the right eye in her 33rd week. She had rheumatic fever in her teenage years and diagnosed with moderate rheumatic mitral stenosis one year ago and has remained asymptomatic. She underwent an initial workup including EKG showing normal sinus rhythm, cardiac echocardiogram revealing normal left ventricular systolic function with an ejection fraction of 70%, moderate mitral stenosis with a valve area of 1.5cm², mean gradient of 20mmHg and moderately elevated pulmonary artery systolic pressure of 48mm Hg. MRI/MRA of the head and neck showed normal and patent vessels with no occlusion, narrowing or clots in the vessels of the head and neck. She was diagnosed with Amaurosis Fugax and a 24-hour Holter monitor was placed which revealed 9 episodes of ventricular tachycardia the longest duration of which was 226 beats during which she remained asymptomatic. After discussing with her the risks and benefits of initiating anti-arrhythmic drug therapy during pregnancy, she was started on Sotalol and monitored on telemetry with no recurrence of ventricular tachycardia.

**DISCUSSION:**  
Ventricular tachycardia is an uncommon rhythm during pregnancy and management is complicated due to the toxicities associated with drug therapy. The key goal of medical management of mitral stenosis is rate control to maximize diastolic filling time of the left ventricle. This patient presenting with Amaurosis Fugax in her third trimester with moderate mitral stenosis, moderate pulmonary hypertension and episodes of ventricular tachycardia requires multispecialty coordination between obstetrics, cardiology and cardiac electrophysiology and this combination has never been described in the literature. Sotalol is one of the least teratogenic anti-arrhythmic drugs which is effective in ventricular tachycardia in pregnancy and can also help with beta-blocking properties and heart rate control for mitral stenosis and proved to be invaluable in this challenging case.

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**CATAMENIAL PNEUMOTHORAX: A RARE CASE OF THORACIC ENDOMETRIOSIS**

Thoracic endometriosis is a rare condition in which endometrial tissue grows in and around the lung. It can present as pneumothorax, hemotherax, hemothysis, or lung nodules. Spontaneous pneumothorax occurs in 1.2-6 women out of 100,000 with endometriosis. We present a rare case of a large, spontaneous pneumothorax requiring pleurodesis, thoracoscopy, thoracotomy, and hormonal therapy with medroxyprogesterone.

A 43-year-old female initially presented with chest pain and difficulty breathing for one day prior to admission. A right sided pneumothorax was diagnosed and a chest tube was placed. The patient complained of pleuritic chest pain occurring monthly during menstruation for a year. CT scan revealed several right sided pleural nodules. The clinical impression was that of catamenial pneumothorax. A right VATS revealed multiple parietal pleural implants along the pleura and diaphragmatic surface. The implants contained chocolate colored fluid. Pleural biopsy and talc pleurodesis were performed. Pathology of the same was consistent with right pleural endometriosis.

Over the following months, patient had recurring right pneumothoraces requiring repeated chest tubes. Of note, patient was unable to get hormonal therapy due to insurance issues. In an attempt to control her symptoms, patient underwent a partial oophorectomy, a second VATS procedure and finally a right pleural decortication. Patient presented to her last clinic visit with mild shortness of breath and local pain related to the thoracotomy. Patient was without clinical symptoms of a pneumothorax. She is currently on hormonal therapy with medroxyprogesterone injection and norethindrone acetate without further complications.

Although thoracic endometriosis is still rare, it is the most common form of extra-pelvic endometriosis. It is often misdiagnosed, with symptoms recurring for months before the correct diagnosis is made. This case illustrates the importance of early recognition of the symptoms of thoracic endometriosis in any woman of reproductive age. This condition can be treated through surgery, medical treatment, or hormone therapy.
Resident/ Fellow Clinical Vignette

Trisandhya Sharma
Laxmi Upadhyay MD, Prakash Acharya MD, Bernard Gitler MD, Stephen Jesmajian MD
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SCIMITAR SYNDROME: A SWORD WITH MULTIPLE EDGE

Introduction:
Scimitar syndrome (SS) is characterized by partial or total anomalous pulmonary venous drainage of the right lung or left lung to inferior venacava (IVC). On imaging, the abnormal pulmonary vein resembles a Middle Eastern sword or scimitar. We present a case of SS with multiple clinical manifestations.

Case presentation
A 44 year old female with history of paroxysmal atrial fibrillation (AF) not on anticoagulation due to low CHADS2VASc score and gastrointestinal bleeding presented with palpitations which lasted fifteen minutes. She reported chronic mild dyspnea on exertion. She did not have chest pain, orthopnea, paroxysmal nocturnal dyspnea or lower extremity swelling. Vital signs - blood pressure: 98/56 mmHg; heart rate (HR): 130/minute, irregularly irregular; respiratory rate 12/minute. Cardiac examination revealed split S2 with loud P2 component. Electrocardiogram showed AF at 128/minute with no acute changes. Complete blood count, serum chemistries, cardiac enzyme and chest X-Ray were normal. Intravenous metoprolol for HR control and a heparin drip for anticoagulation were started. Transthoracic echocardiogram showed normal left ventricular function with dilated right atrium (RA) and right ventricle (RV). Estimated RV systolic pressure was >40 mmHg. After twelve hours, patient developed epigastric pain radiating to the chest and back. Lipase and lactic acid were normal but D-dimer was 923 ng/ml. No evidence of pulmonary embolism or aortic dissection on computed tomography (CT) angiography of chest. An anomalous pulmonary venous drainage of the right lung with pulmonary veins (PV) draining into the junction of the RA and IVC was seen. The right lower lung was hypoplastic and the inferior mediastinum was shifted to the right hemithorax. The findings were suggestive of SS. Retrospective reevaluation of the chest X-Ray showed anomalous pulmonary vein forming the shape of a scimitar. Abdominal CT showed bilateral renal infarcts. Pain was controlled with morphine. Anticoagulation was bridged to warfarin and the patient was discharged.

Discussion
Common presentation of SS in adult is slow onset chronic shortness of breath (SOB), chronic or recurrent AF and recurrent respiratory infections. Excessive PV return to the right side of the heart causes RA and RV dilatation, resulting in atrial remodeling predisposing to AF. Pulmonary hypoplasia can cause reduction of the vascular bed which combined with increased flow into the RA from the anomalous PV leads to pulmonary hypertension which contributes to the chronic SOB.

Conclusion:
Even though SS typically presents in infancy, it can be a rare presentation in the adult population. Therefore, in a young adult presenting with recurrent AF or chronic SOB without obvious pulmonary or cardiac cause, possibility of SS should be considered.

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Ehizode Udevbulu
Brookdale University Hospital

NORSE as Initial Presentation in Lupus Cerebritis

A 19 year old man was admitted to hospital because of seizure episodes at home. Twenty four hours before admission, patient was complaining of joint pain. On the morning of admission, he woke up with periorbital rash/edema and generalized itching. Later that afternoon, he had 2 episodes of generalized tonic clonic seizure lasting for 1 min. He was postictal until arrival to hospital. In emergency room, he continued to have generalized tonic clonic seizures, intubated for airway protection. He has no known past medical history, no illicit drug use and no known previous seizure episodes. He has not has any prior surgeries. Medications were only Aleve occasionally. He has an allergy to aspirin.

On examination, there was orbital swelling and redness around his eyes. GCS of 6 , hemodynamically stable however febrile with temp of 39 degrees Celsius. There was high clinical suspicion for bacterial meningitis and he was treated with antibiotics and high dose methylprednisolone. LP was performed however findings were nonspecific, wbc of 15 cell/ul. Initial CT head unremarkable. Labs findings demonstrated pancytopenia wbc 2.9 10x9/l with ANC of 2.1, platelets of 110 x9/l and hb of 11.5 g/dl. Chemistry demonstrated acute kidney injury bun/cr 42mg/dl/1.5 mg/dl, transaminitis 325/665 U/L Alcohol level <10, negative urine toxicology and negative troponin. Hepatitis C antibody was positive however RNA PCR was negative. Negative HIV test. Electrocardiogram showed normal sinus rhythm. Hematology evaluated the patient for suspicion of TTP however peripheral smear was not indicative and it was ruled out. Infectious disease recommended continuing empirical treatment for suspected viral meningitis with acyclovir. Patient was admitted to MICU for status epilepticus and treated with continuous midazolam Infusion. MRI brain was performed and was unremarkable. At this point, decision was made to transfer the patient to a hospital with continuous EEG monitoring. Patient was transferred to outside hospital with continuous EEG monitoring capacity; Further testing revealed ANA positive with high titers >=1:1280, DS - DNA+, Low Complement levels. Diagnosis of lupus was made, he was weaned off of midazolam, treated with high dose steroids, and has shown consistent clinical improvement.

In conclusion, the meningoencephalitis with seizures, pancytopenia, microscopic hematuria in setting of positive ANA with high titers, DS-DNA +, with low complement levels, diagnosis of lupus cerebritis was made.
Resident/ Fellow Clinical Vignette

**Oral Protein Supplement Induced Hyperkalemia**

**Introduction**

Protein supplements are commonly used over the counter medications. Hyperkalemia is defined as serum potassium level above 5 mEq/L. We present a case of hyperkalemia induced by use of high dose oral protein supplement containing arginine.

**Case Presentation**

A 61 year old female presented with complaint of gradual onset generalized weakness for one week. She had poor appetite for the past few weeks and had lost 30 lbs weight in the previous two months. Her past medical history included hypothyroidism, chronic kidney disease (CKD) stage 3 and recently suspected liver carcinoma based on imaging and blood testing. She had declined further workup of liver lesion with liver biopsy. She was on multiple nutritional supplements and had started a new protein supplement 2 weeks back. On presentation, patient had stable vitals. Examination revealed nodular and enlarged liver. Laboratory studies showed sodium: 129 mEq/L, potassium: 6.9 mEq/L, BUN 57 mg/dL, creatinine: 1.41 mg/dL, glomerular filtration rate (GFR): 38.6 ml/min, AST: 184 U/L, ALT: 31 U/L, Alkaline phosphatase: 229 U/L. Labs from three weeks ago, before the protein supplement was started, showed serum potassium of 5 mEq/L with similar renal and liver function test. She was treated with intravenous regular insulin, dextrose and Kayexalate. Her protein supplement was found to contain 500 mg of Arginine per capsule. She was taking 6 tablets 3 times a day. The patient did not take the medicine since her hospital admission and her K level decreased and stayed between 4-5 mEq/L. Patient was advised to stop the protein supplement and have biopsy of the liver lesion as an outpatient.

**Discussion**

Hyperkalemia is frequent in advanced stages of CKD but in the early stages secondary causes need to be ruled out. Arginine (a cationic amino acid found in most proteic supplements) increases the efflux of K through H+/K+ pump causing hyperkalemia independent of serum Ph. It also increases the excretion of K when normal renal function but significant hyperkalemia can occur in patients with poor renal function. Also decreased metabolism in the liver causes high circulating amounts of arginine that can further increase the hyperkalemia. There are documented cases of hyperkalemia in patients with deranged liver function receiving I.V arginine but none secondary to ingestion of oral supplements.

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A 61 year old female presented with complaint of gradual onset generalized weakness for one week. She had poor appetite for the past few weeks and had lost 30 lbs weight in the previous two months. Her past medical history included hypothyroidism, chronic kidney disease (CKD) stage 3 and recently suspected liver carcinoma based on imaging and blood testing. She had declined further workup of liver lesion with liver biopsy. She was on multiple nutritional supplements and had started a new protein supplement 2 weeks back. On presentation, patient had stable vitals. Examination revealed nodular and enlarged liver. Laboratory studies showed sodium: 129 mEq/L, potassium: 6.9 mEq/L, BUN 57 mg/dL, creatinine: 1.41 mg/dL, glomerular filtration rate (GFR): 38.6 ml/min, AST: 184 U/L, ALT: 31 U/L, Alkaline phosphatase: 229 U/L. Labs from three weeks ago, before the protein supplement was started, showed serum potassium of 5 mEq/L with similar renal and liver function test. She was treated with intravenous regular insulin, dextrose and Kayexalate. Her protein supplement was found to contain 500 mg of Arginine per capsule. She was taking 6 tablets 3 times a day. The patient did not take the medicine since her hospital admission and her K level decreased and stayed between 4-5 mEq/L. Patient was advised to stop the protein supplement and have biopsy of the liver lesion as an outpatient.

**Discussion**

Hyperkalemia is frequent in advanced stages of CKD but in the early stages secondary causes need to be ruled out. Arginine (a cationic amino acid found in most proteic supplements) increases the efflux of K through H+/K+ pump causing hyperkalemia independent of serum Ph. It also increases the excretion of K when normal renal function but significant hyperkalemia can occur in patients with poor renal function. Also decreased metabolism in the liver causes high circulating amounts of arginine that can further increase the hyperkalemia. There are documented cases of hyperkalemia in patients with deranged liver function receiving I.V arginine but none secondary to ingestion of oral supplements.
A case of Well Differentiated Papillary Mesothelioma of the peritoneum in a male presenting with LGIB.

Introduction
Well-differentiated papillary mesothelioma (WDPM) is a rare variant of epithelial mesothelioma. WDPM predominantly affects young females and is most often an incidental finding. The peritoneal cavity is the most common site affected; however, WDPM of the pleura, pericardium and tunica vaginalis have also been reported.

Case Presentation
We report a case of a 50-year-old Mexican male who was referred from the emergency department for rectal bleeding. He was discharged on antibiotics for presumed colitis after abdominal and pelvis CT scan revealed rectal thickening. The patient continued to have rectal bleeding. Colonoscopy revealed a large circumferentially partially obstructing mass in the rectum. Biopsy revealed well-differentiated adenocarcinoma. Subsequent CT scan of the abdomen and pelvis demonstrated this rectal mass with perirectal lymph nodes. Patient received neoadjuvant chemotherapy before laparoscopic evaluation with low anterior resection surgery. Frozen sections of peritoneal implants initially revealed chorionic placental tissue of unknown origin. In view of the peritoneal lesions, it was decided to do a peritoneal lavage with the fluid for cytology and definitive surgery was delayed until there was clarification on the origin of these peritoneal masses. Pathology revealed papillary mesothelial proliferation. This was verified by an independent pathologist to be well-differentiated papillary mesothelioma by immunohistochemical staining. Treatment strategies included lower anterior resection followed by hyperthermic intraperitoneal chemotherapy. However, patient was lost to follow up could not be reached for further plans of management.

Mycotic Prostatitis: Prostatic Aspergillus in an Immunocompetent Patient

Mycotic prostatitis is a rarely encountered condition. Candida, histoplasmosis, aspergillus, coccidiodes, cryptococcus, and blastomyces are all possible sources of the infection, and can be seen in both immunocompromised and immunocompetent hosts. The symptoms of mycotic prostatitis can resemble benign prostatic hyperplasia, bacterial infection, or neoplastic growth.

A 79 year old male was admitted for acute renal failure. His medical history is significant for acute kidney injury, benign prostatic hyperplasia, hypertension, diabetes mellitus type II, coronary artery disease, and hypothyroidism. At presentation, the patient complained of urine retention and frank hematuria. Physical exam revealed an agitated and afebrile patient with normal cardiac rate and rhythm with left sided pacemaker, equal air entry bilaterally with no adventitious breath sounds, and bowel sounds were present with no abdominal pain on palpation. Examination of the lower extremities was significant for 1+ pitting edema. A Foley catheter was present. On admission, his BUN was 85 and creatinine was 4.0. White blood cell count was 7.8. IV fluids were started, and urology was consulted. They recommended the patient undergo a transurethral resection of the prostate. The patient was medically cleared for the procedure, and it was performed. The procedure was successful, and biopsy of the prostate was obtained and sent to pathology. Upon review of the biopsy, pathology noted the presence prostatic stromal and adenomatous hyperplasia, urothelial hyperplasia, prostatic calculi, and severe mycotic prostatitis with abundant fungal aggregates consistent with aspergillus.

Though rare, it is important that clinicians be mindful of the possibility of mycotic prostatitis in patients presenting with symptoms consistent with benign prostatic hyperplasia, even in the absence of an immunocompromised state. Some cases of mycotic prostatitis may be incidentally discovered upon prostate biopsy for surgical treatment of benign prostatic hyperplasia or prostatic carcinoma. Other incidents of mycotic prostatitis may require systemic antifungal therapy in addition to prostatectomy if the infection is not localized to the gland.
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<th>Vikrant Tambe MBBS</th>
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<td>Christy Ann Gilman, MD; Bashar Sharma, MD; Gaganjot Singh, MD</td>
<td>Hung-I Liao MD, Obed Adarkwah MD</td>
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<td>SUNY Upstate Medical University Hospital</td>
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**CEFTRIAXONE FAILURE IN PATIENT WITH NEUROSYPHILIS AND PENCILLIN ALLERGY**

**Report:** Penicillins are the agent of choice in patients with Neurosyphilis. There are some case reports that exhibit clearance of the organism with Ceftriaxone in a patient with severe penicillin allergy. We present a case of treatment failure with Ceftriaxone and subsequent success with treatment of intravenous penicillin post desensitization.

**Case:** 34-year-old male was admitted with complaints of increased headaches and back pain. He had recently moved to Upstate from Florida where he was diagnosed with Neurosyphilis with RPR titer of 1:32 in February 2017 when he attempted to donate blood. He was subsequently treated with 2 weeks of IV Ceftriaxone therapy as he was allergic to penicillin and he reportedly improved with this regimen. He had moved to Syracuse two months after in April.

He, later, started having recurrence of his headaches and blurriness of vision so he presented to outside hospital for further evaluation. He was evaluated with LP and RPR titer was found to be 1:8. He was transferred to our hospital for further management. Upon presentation, he reported that his headaches and blurry vision were worse and was started on IV Ceftriaxone in ER. He did not have any overt focal neurological defects and MRI did not show any acute disease process. His LP was performed and his CSF VDRL titer was reactive and hence a decision to change antibiotic to IV penicillin was taken despite the four-fold fall in the RPR titer with Ceftriaxone. Since he was allergic to penicillin, measures for quick resuscitative measures were placed and he was challenged with 1/100th dose of IV penicillin and then 1/10th dose and finally full dose without any observable reaction. He showed improvement 2 days post initiation of this therapy. He was discharged with the same regimen of IV penicillin 4 million units every 4 hours for 2 weeks, then weekly injection of Benzocaine penicillin for 3 weeks and started on long term weekly injections of IV penicillin.

**Discussion:** There are very few case reports of successful treatment of meningitis in patients who are HIV negative with Ceftriaxone. It achieves levels well above the MIC for Treponema pallidum of 0.0006 mg/ml with a dose of 1 gm per day. CDC (2015 STD guidelines) recommends it as an alternative agent for management of neurosyphilis with a dose of 2 gm a day in patients with meningitis for a duration of 10-14 days. We highlight one of the rarely conducted Ceftriaxone trials and point to the importance of being aware of incidence treatment failure and early institution of desensitization and treatment with intravenous penicillin in patients with penicillin allergy. More research is needed in CSF penetration and treatment efficiency with Ceftriaxone is required before it is a valid recommendation.

**Introduction**

The African root, Ibogaine, has been reported to be one possible solution to drug addiction. According to the NIH, substance abuse across the nation costs approximately $740 billion due to criminal activity, health care, and decreased work productivity. Solutions to drug addiction are sought every day, one of them being Ibogaine. However, ibogaine comes with unknown subsets of side effects. Here, we present a case of ibogaine that was associated with QT interval prolongation without electrolyte abnormalities.

**Case Presentation**

A 22-year-old female with no significant past medical history except IV heroin use presented to the emergency department with altered mental status. She reported that she was trying to quit heroin use and was recommended by friends to use Ibogaine. On review of systems, she denied chest pain, dyspnea, and palpitations. Physical examination was remarkable for a diastolic murmur and altered mental status. CSF studies, laboratory testing and radiological imaging were performed to evaluate for sources of infection. Laboratory values were significant for BUN of 29 mg/dl, urine specific gravity >1.30, WBC 19.9 x 109/L with a neutrophil differential of 92.3%. No electrolyte abnormalities were noted. She remained afebrile throughout hospitalization, and the leukocytosis resolved within 1 day of admission with rehydration with normal saline. EKG was significant for QT prolongation 535 msec. Transthoracic echocardiogram revealed a thickened mitral valve. Cardiology was consulted for further evaluation and recommended transesophageal echocardiogram (TEE) and blood cultures to rule out infectious endocarditis in light of the EKG changes, leukocytosis and a murmur. TEE and blood cultures were negative for infection. At this time, the likelihood of infectious endocarditis was low. Repeat chest x-ray, EKG, and troponins were all within normal limits without obvious abnormalities. EKG also showed normalization of the QT interval 412 msec, as compared to four days prior. The patient’s symptoms resolved with IV hydration, and the patient was hemodynamically stabilized for transition of care, with referral to join Narcotics Anonymous to treat her addiction.

**Discussion**

Currently, ibogaine is not approved for medical use in the United States. Ibogaine has been used as a detoxification agent in other countries; however, there are inherent risks in its use. Current literature demonstrates that ibogaine is associated with cardiotoxicity and possibly sudden cardiac death. This patient presented with QT prolongation likely secondary to ibogaine use, since there were no notable electrolyte abnormalities. Ibogaine causes QT prolongation by blocking hERG (human Ether-a-go-go-Related Gene) potassium channels in the heart. QT prolongation can progress to tachyarrhythmias such as torsades de pontes or even death. It is pertinent to carefully monitor each patient taking this herbal medicine, and it is imperative to consider ibogaine use as a differential diagnosis in recovering IV heroin users.
### Resident/ Fellow Clinical Vignette

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<th>Nitin Tandan MD</th>
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<td>Ruby Maini; Obed Adarkwah, MD; Hung-I Liao, MD; Nilay, Shah, MD</td>
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<td>New York University School of Medicine</td>
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**An unusual presentation of Primary Progressive Multiple Sclerosis with underlying Neurofibromatosis**

**Introduction**

As medicine evolves, we continue to improve our understanding of multiple sclerosis. It is usually characterized by neurological changes that occur at different points in time. Current literature suggests a rare, questionable association of multiple sclerosis with neurofibromatosis. Here, we present a case of a young female without classic neurological symptoms but with suggested diagnosis of multiple sclerosis.

**Case Presentation**

A 27-year-old female presented to the emergency department with vertigo for two days. She denied any history or current symptoms of weakness, numbness, and tingling in any body parts. She reported blurry vision secondary to astigmatism that improved with glasses but denied any vision changes and double vision. Her symptoms transiently improved by taking a warm shower. Past medical history is significant for lattice degeneration in the eyes and neurofibromatosis, for which she follows her private neurologist annually. She denied any family history of neurological conditions. Physical examination was significant for caf&deg;3; au lait spots along the back and throughout the abdomen and normal strength and sensation in the upper and lower extremities. Reflexes were 2+ throughout. Cranial nerves II-XII were grossly intact. Laboratory values revealed normal TSH, free T4, B12, 25 OH vitamin D and folate levels with negative serologies for HIV, TPA IgG, and RPR. Head CT showed a suspicious lesion near the ventricles and radiology recommended a follow-up MRI with gadolinium. MRI of the brain with gadolinium demonstrated bilateral periventricular white matter lesions and with parietal and frontal white matter lesions, suggestive of multiple sclerosis. An MRI of her C-spine showed that the patient had a 3x3 mm white matter lesion on her spinal cord, also consistent with multiple sclerosis. These lesions suggested a diagnosis of multiple sclerosis based on the 2010 McDonald criteria; subsequently, the patient was started on high dose methylprednisolone for five days. She responded well to the steroids and was safely discharged home with instructions to follow up with her neurologist.

**Discussion**

A history of neurofibromatosis is not often included as a predisposition to demyelinating disease, particularly multiple sclerosis. This case report is unique as compared to current literature regarding the association of neurofibromatosis and multiple sclerosis in that there were no "classic" symptoms of weakness, changes in sensation or changes in her vision prior to presentation of vertigo. The only other symptom that she presented with is symptomatic relief when she took hot showers. While the literature describes a rare association between the two conditions, it is important to note that the two conditions may coexist and presentations may vary significantly secondary to underlying disease processes.
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Nassau University Medical Center

Expect the unexpected- An unusual case of recurrent cystic swelling near the knee joint

Introduction-
Tuberculosis (TB) of appendicular skeleton is an uncommon infection. It constitutes 1-3% of all forms of TB and 7-15% of extrapolunary cases. The knee joint is the third most commonly affected joint in tuberculosis after spine and hip.

Case presentation-
58 year old Bangladeshi male presented to ER upon request from department of health. He had swelling in his left knee joint and recurrent cystic swelling in his left leg, posterior and lateral to the knee since last 2 years. The joint was aspirated couple of times at another facility. His last aspiration was 5 weeks ago. Symptoms resolved after aspiration. Records showed that the cyst fluid culture was positive for pan sensitive Mycobacterium tuberculosis. He denied fever, chills, recent travel, sick contacts, cough, chest pain, dyspnea and weight loss.

He was afibrile with stable vitals. Systemic exam was benign. There was no lymphadenopathy or rash. He had a cystic swelling posterior and lateral to the knee joint, 8 cm x 5 cm, non-tender and warm to touch. CXR, CBC and CMP were normal. MRI of the knee joint showed thickened synovium with osseous erosions suggestive of osteomyelitis. Fluid cell count and differential were not significant. Rheumatologic workup was negative. Sputum acid fast bacilli smears and cultures were negative. Anti-TB drugs INH,B6,RIF,PZA and EMB were initiated. Due to extensive joint damage patient had radical synovectomy. Repeat synovial joint fluid culture was again positive for M. tuberculosis which was pan sensitive. Histopathology of the synovium showed necrotizing and non-necrotizing granulomatous inflammation.

Discussion-
Tuberculous arthritis tends to occur in hip or knee and is usually monoarticular. The joint is generally "cold" (signs of acute infection are usually absent). Tuberculous synovitis is diagnosed by microscopy, culture of the synovial fluid and histopathological examination. Tuberculous osteomyelitis typically occurs at a single site. Tuberculous osteomyelitis frequently presents as "cold abscess" with swelling, modest erythema or pain, and little or no local warmth. MRI can be useful to detect the extent of the damage. Although extrapolunary TB may be treated for 6 months, the treatment for bone and joint disease often requires 12-18 months, depending upon the extent of damage. Surgical management is indicated when there is extensive bone involvement or cold abscess formation. Pigmented villonodular synovitis and monoarticular rheumatoid arthritis are important clinical mimickers.

Conclusion-
This case highlights the uncommon presentation of tuberculous arthritis. Patients may present without constitutional symptoms. Pulmonary involvement is seen in only one third cases of skeletal TB which may lead to diagnostic delay. Tuberculous synovitis is often overlooked during clinical examination. It is necessary to increase clinical awareness of joint TB to ensure early diagnosis and treatment.

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ROCHESTER GENERAL HOSPITAL

ISOLATED CNS VASCULITIS MANIFESTING AS RECURRENT STROKE

BACKGROUND
Primary angiitis of the central nervous system is an uncommon inflammatory disease with an incidence of 2.4 cases per 1000,000 person-years. It is characterized by small vessel vasculitis limited to the central nervous system without other systemic manifestations. It occurs twice as commonly in males and is usually diagnosed in the 5th decade of life. We report the case of a young male who was diagnosed with primary angiitis of the CNS after a three year struggle of recurrent hospitalizations and exhaustive clinical work up for recurrent hemiparesis.

CASE: A 23 year old male with no past medical history presented after a witnessed seizure at home and was found to have extensive venous thrombosis of the superior sagittal sinus, left transverse venous and straight sinus on MRI venogram. Thrombophilic work up was negative and he was discharged on anticoagulation. He was admitted a few months later with sudden onset right sided hemiparesis, left sixth nerve palsy and left sided Horner’s syndrome. MRI brain was consistent with widespread cerebral edema secondary to venous congestion involving the brainstem, right medial thalamus, pons, midbrain and right hippocampus. CT chest, abdomen & pelvis and ultrasound scrotum to rule out paraneoplastic syndromes, were negative. Lumbar puncture and EEG were unremarkable. Empiric dexamethasone resulted in rapid improvement in symptoms.

On detailed questioning, the patient recalled that he may have had scrotal ulcers as a teenager. Suspicion for undiagnosed Behçet’s syndrome sparked an extensive rheumatologic work up including ANA, ENA, RF, anti-CCP, ANCA, anti-SSA and SSB, C3 and C4, ESR and CRP which was completely unremarkable. Repeat MRI showed improvement in cerebral edema and patient was discharged home after 10 days of intravenous steroids. He returned 8 weeks later with left sided hemiparesis and left facial droop. Labs revealed elevated inflammatory markers with an ESR and CRP of 48 and 97.2 respectively. MRI brain showed multiple enhancing lesions within the midbrain and middle cerebral peduncle along with 2 punctate foci of abnormal restricted diffusion within the right thalamus and left internal capsule. He responded to pulsed solumedrol. A right temporal lobe biopsy was consistent with acute and chronic inflammation with granulomatous vasculitis.

CONCLUSION: Diagnosis of isolated CNS vasculitis requires presence of unexplained neurologic deficits lasting longer than 6 months, demonstration of classic angiographic or histopathologic features and no evidence of systemic vasculitis, all of which were present in our patient. Untreated CNS angiitis has a high mortality and results in death from recurrent cerebral infarctions, but aggressive immunosuppressive therapy with prednisone and cyclophosphamide has shown to improve clinical outcomes. Detailed history should be obtained in all patients with inconsistent neurologic signs and symptoms which can prompt early work up and diagnosis of this potentially fatal disease.
New York Chapter ACP
Resident and Medical Student Forum

Resident / Fellow Research
Resident/Fellow Research

Assessment of Factors Influencing Poor Medication Adherence in Hypertensive and in Ethnically Diverse Patient Diabetes Patients Population

Background: Diabetes and Hypertension are the two most prevalent chronic diseases in the United States of America. Non-adherence to medications that are otherwise effective in treating these diseases can lead to significant morbidity, mortality and staggering high healthcare costs. The purpose of this study was to determine modifiable factors that influence adherence to diabetic and hypertensive medications, in ethnically diverse patient populations at Richmond University Medical Center community clinic (RUMC-clinic).

Methods: A cross-sectional IRB approved prospective study survey was administered to 253 patients with diabetes and hypertension at RUMC-clinic. Patients 18 years and older, who had clinical diagnoses of hypertension and/or diabetes were randomly approached and informed consents were obtained. Adherence was measured by using the standardized Morisky Medication Compliance Questionnaire. Further, demographic and socio-economic information such as age, sex, marital status, education level, depression, psychological stress, financial stress level and the number of dependents at home were collected. Pearson’s chi-squared, and Kruskal-Wallis ANOVA tests were used and a binary logistic regression model was created for statistical analysis. When Kruskal-Wallis test yielded a significant result we utilized Dunn’s test to achieve more granularity. Statistical significance was considered at P<0.05.

Results: Out of a total of 253 participants, 226 fully completed the survey. 59.7% were female and 40.3% were male. The mean age of the participants was 56 years; ranging 22-86. From the surveyed patient population, the majority self-identified as Black or African American (43%) and Hispanic (25.3%), followed by White or Caucasian (16.7%), Asian (9.9%), Native American or Alaskan Native (0.9%) and other (3.16%). Data analysis found that the greatest factors influencing medication adherence are gender, with females more likely to adhere than males (P = 0.04212), depression (P = 0.001577), psychological stress (P = 0.04212), and financial stress (P=0.02).

Conclusions: The rate of adherence observed in this study was low, with only 24.7% of participant’s adherent to their treatment. Significant predictors for poor adherence are male gender, depression, psychological stress and financial stress. Three of these predictors are potentially modifiable and logical targets for intervention. Healthcare professionals should be aware of these factors, especially when prescribing long term medications and therapy. To improve medication adherence, we recommend screening patients with depression; educating patients about federally funded resources to improve financial stress; and providing education on a wide spectrum of techniques to minimize psychological stress.

PREDICTORS OF NON-CALCIFIED PLAQUE AMONG PATIENTS WITH OBSTRUCTIVE CORONARY ARTERY DISEASE ON CORONARY COMPUTED TOMOGRAPHY ANGIOGRAPHY

Purpose of the study: Although there are significant similarities in the risk factors for coronary artery disease (CAD) and calcification of the coronary intimal layer, the number of patients with non-calcified obstructive CAD is not negligible. In this study we examined differences in the distribution of cardiac risk factors between patients with calcified and non-calcified obstructive CAD detected on coronary computed tomography angiography (CCTA). Methods: We retrospectively evaluated consecutive patients greater than or equal to 18 years old with no known CAD, negative initial cardiac bio-markers, and non-ischemic electrocardiogram referred for CCTA between June 1, 2013 and December 31, 2014, for evaluation of chest pain in a tertiary care academic center. A cohort of 207 patients had obstructive CAD defined as greater than or equal to 50% stenosis on CCTA. We compared risk factors of patients with zero calcium score to patients with calcified disease.

Results: In a univariate analysis of the 207 patients with obstructive CAD, there were no statistically significant differences between patients with calcified and non-calcified plaque with respect to race, gender, hypertension, hyperlipidemia, smoking status, obesity, and family history of premature CAD. Patients with non-calcified plaque compared to those with calcified plaque tended to be younger (mean age in years 53.3 & 95% confidence interval 49.8-56.8; 60.2 & 95% confidence interval 57-63.4). Patients with non-diabetic (9.4% vs 25.1% for diabetic status, p=0.05). In a binary logistic regression analysis, age and gender were found to be independent predictors of non-calcified plaque (Odds Ratio: 1.08, 95% Confidence Interval 1.03-1.13, p =0.001 for age; 0.381, 95% Confidence Interval 0.16-0.89, p=0.026 for gender), while a statistical trend was observed for diabetes status (Odds Ratio: 0.29, 95% Confidence Interval 0.08-1.05, p =0.059).

Conclusion: Among traditional CAD risk factors, young age and female gender are strong independent predictors of non-calcified plaque among patients with obstructive CAD, while non-diabetic status shows a statistical trend as an independent predictor. Our findings suggest that calcium scoring should not be used to detect obstructive CAD in young, female, and non-diabetic patients who present with chest pain and have negative initial cardiac biomarkers with non-ischemic findings on electrocardiogram.
## Effect of Vitamin C on Reactive Oxygen Species Formation in Erythrocytes of Sickle Cell Patients

**Background:** Sickle cell patients produce more reactive oxygen species (ROS) than healthy individuals, leading to increased cell membrane damage. Theoretically, reducing ROS formation would preserve red cell membranes of sickle cell patients. Vitamin C is a powerful anti-oxidant capable of inhibiting ROS formation in a variety of situations, by functioning as an electron donor to reduce molecular oxygen. This study aimed to determine whether Vitamin C reduced ROS formation in sickle red cells.

**Methods:** 27 homozygous (HbSS) patients were recruited from the outpatient clinics of Lagos University Teaching Hospital, Nigeria, and annex at the Sickle Cell Foundation, Lagos, Nigeria. Demographic information and EDTA patient blood samples were collected. The test group were red cells preincubated in 80uM and 100uM Vitamin C concentrations before stressing with tertbutylhydroperoxide. These were compared to stressed matched controls preincubated in phosphate buffered saline. Cell staining was done with CellRox Orange followed by flow cytometry to quantify ROS. Results: ROS count for Vitamin C pre-treated red cells was significantly lower than matched controls (p=0.001). Average ROS count for 80uM test samples was 27.5/ul (95% CI, 17.5 to 72.5) and for 100uM 3.9/ul (95% CI, 1.9 to 5.9). Male gender was significantly associated with elevated baseline ROS count (p=0.03).

**Conclusion:** Vitamin C reduced ROS formation in HbSS cells. Future studies should focus on a role for Vitamin C as a safe, cheap addition to maintenance therapy of sickle cell patients.

## Telephone Interpreter Services Patient Perspectives: A Qualitative Analysis of Patient Experiences with Telephone Interpreter Based Clinical Encounters

**Background:** Racial and ethnic minorities in the US, a large and growing population, experience higher incidence of and greater morbidity from chronic disease. Limited English proficiency (LEP) is a known contributor to these health disparities. The Culturally & Linguistically Appropriate Services standards promote health equality through the incorporation of interpreter services (IS). Currently IS are provided by ad-hoc interpreters, in-person interpreters, and telephone interpreters. IS have shown improved quality of care, but limited data exists on patient perspectives of IS; particularly telephone interpreter services (TIS). Better understanding patient experiences with TIS could elucidate ways of enhancing patient-centered care for this patient population, potentially improving health outcomes.

**Study Purpose:**

To assess the experiences of Spanish-speaking, LEP patients with TIS, and to identify facilitators and barriers for use of TIS during clinic visits.

**Methods:**

Participants were self-identified, Spanish-speaking, LEP individuals who utilized TIS during a clinic visit within the past 12 months. They completed a semi-structured questionnaire and participated in a one-hour, audio-recorded focus group. The focus group facilitator completed field notes after each focus group. Audio recordings were transcribed and analyzed by content analysis and grounded theory.

**Summary of Results:**

A total of 10 individuals participated in 3 focus groups. Participants were female, ages 33-70 years (mean 53), from Mexico (n=5), Ecuador (n=2), and the Dominican Republic (n=3), and had resided in the US for an average of 22 years (range 13-38). Seventy percent reported poor self-rated English language ability (20% fair, 10% not reported). Participants reported feeling gratitude for TIS, that TIS increased access to healthcare, and preference for TIS to family members interpreting. Barriers to the use of TIS were insecurity about confidentiality, lack of "calor humano" (human warmth), and limited "desenvolvimiento y amplitud" (engagement and breadth) in telephone interpreter based encounters. Facilitators to the use of TIS were positive physician attitude toward TIS, and physician framing of TIS as a tool for providing quality care. Preference for language-congruent providers over TIS varied; some prioritized access to care, while others prioritized human connection not easily facilitated by TIS.

**Conclusion:**

Telephone interpreter services (TIS) are generally accepted by Spanish-speaking, LEP patients because they increase access, and are more anonymous than in-person interpreters or family member interpreters. Patients seem to balance the need for access to healthcare with the discomfort of compromised privacy in interpreter-based encounters. Positive physician attitudes toward TIS facilitate the use of TIS. Healthcare providers should be aware their attitude toward TIS can affect patient experiences and acceptance of TIS. For patients, relationship building is an important component of language-congruent encounters that is lacking in TIS-based encounters. This suggests a need for training for both patients and physicians on relationship building in utilizing TIS.
Ovarian cancer is one of the most fatal and difficult to treat gynecologic malignancies. Data from the American Cancer Society places five-year survival rates of the disease at 45%. Standard treatment for ovarian cancer entails surgical resection, followed by combination paclitaxel and carboplatin chemotherapy. While the response rates to chemotherapy are high, over 80% of patients have recurrent disease. Recent studies indicate that the high rates of relapse are associated with an immunosuppressive environment characterized by tumor infiltration of CD4+ regulatory T cells (Treg). Conversely, tumor infiltration by CD4+ Th17 cells is associated with prolonged survival. Modulation of the p38 MAP kinase-signaling pathway in dendritic cells (DC) has shown great promise in modulating this Treg/Th17 balance. Specifically, cytokine-matured DC with a combination of IL-15 and p38 MAP kinase inhibitor (p38i) has been shown to redirect CD4+ cells towards a Th17 response, resulting in a concomitant increase in ovarian tumor antigen-specific CD8+ cytotoxic T lymphocyte responses. In our studies, we investigated outcomes of p38i and IL-15-treated DC vaccination versus standard cytokine-matured DC vaccination in murine models. Mice were IP injected with epithelial ovarian cancer cells (ID8) and subsequently placed into the following treatment groups with Sp17 ovarian tumor antigen-loaded DC: IL-4/GM-CSF (antigen-free), IL-4/GM-CSF/Sp17 (standard antigen-loaded DC), p38i/IL-15 (antigen-free), and p38i/IL-15/Sp17 (antigen-loaded Th17-inducing DC). The end-point was length of post-treatment survival. The p38i/IL-15/Sp17 mice had an increased survival time (83 days) compared to the IL-4/GM-CSF group (58 days). A second experiment tested p38i/IL-15-treated DC as adjuvant therapy with cisplatin. ID8 ovarian tumor-bearing mice were treated as follows: no treatment, cisplatin, cisplatin+P38i/IL-15 DC, and cisplatin+P38i/IL-15/sp17. Again, the cisplatin+ p38i/IL-15/sp17 had longer post-inoculation survival times (150+ days), compared to both the non-treatment (50 days) and cisplatin (100+) groups. Overall, these results confirm the significant promise of combined p38i/IL-15/sp17 matured DC vaccines in the treatment of ovarian cancer.

**References:**


**Acknowledgments:**

This study was supported by grants from the American Cancer Society and the National Institutes of Health (NIH). The authors would like to thank the patients who participated in the study and the physicians who referred them.

**Disclosure:**

The authors declare no conflicts of interest.
Objective: Chronic Myeloid Leukemia (CML) is a clonal disorder of the hematopoietic stem cells associated with an oncogenic, reciprocal translocation t (9; 22)(q34;q11), resulting in the Philadelphia chromosome. Imatinib or Glivec (STI571), the targeting agent for ATP binding sites of tyrosine kinase enzyme is used commonly as a first line treatment modality for CML. This is one of the first studies from Southeast Asia to report the hematological response and side effects of Imatinib in CML patients.

Method: This cross sectional study was conducted at outpatient departments of Civil Hospital Karachi and Doctor Plaza Clinic in Karachi, Pakistan. Sampling technique used was convenient sampling. The duration of the study was from August 1st, 2015 until August 31st, 2016. By using Open Epi, an open source calculator, the minimum sample size was calculated to be 14 (with Confidence Interval 95%). Newly diagnosed CML patients with Positive bcr-abl gene hybrid evident by either Fluorescence in Situ Hybridization or Polymerase Chain Reaction, with no prior therapy (hydroxyurea, interferon or other tyrosine kinase inhibitors) were recruited. A total of 80 patients were recruited from the outpatient departments. Out of 80, 11 were excluded on the basis of inclusion/exclusion criteria and remaining 69 newly diagnosed and untreated patients were started on a 400-mg dose of Imatinib once in a day. Those were then followed closely to measure the duration till complete hematologic response was achieved, confirmed by physical examination and complete blood. In regular follow-ups, patient-reported signs and symptoms were noted and drug toxicities were observed through clinical examinations and various lab tests.

Results: Commonest clinical presentation of patients with CML was found to be splenomegaly (72.5%), followed by fatigue (53.7%), hepatomegaly (33.33%), fever (24.6%), bleeding dysfunction (14.5%) and others (5%). A complete hematological response was achieved by 92.5% of the individuals within three months of starting treatment with a 400mg dosage of Imatinib once a day (Range from 1 week to 12 weeks); while another 7.2% of the patients only achieved complete hematological response at 6 months of therapy. Commonest side effects observed were weight gain (42%), followed by skin toxicities (36.2%), gastrointestinal symptoms (33.33%), deranged complete blood count (anemia, neutropenia, thrombocytopenia and leukopenia) (14%) and abnormal Liver function Tests (3%).

Conclusion: In a South East Asian population, our study clearly demonstrates that Imatinib is effective in the treatment of CML. We found increased incidence of weight gain, skin toxicities and GI symptoms compared to historical data, likely attributable to other environmental factors. These results show that Imatinib is quite effective and tolerable amongst patients with CML in South East Asia, long term data will be needed to better define its safety profile.

Keywords: Chronic Myeloid Leukemia, Imatinib, Hematological response, Side Effects
Background: tAML, compared to de novo AML, has higher adverse features and a shorter overall survival (OS). The use of chemotherapy and hematopoietic cell transplant (HCT), and OS of tAML outside of clinical trials has not been studied well. Current study was designed to identify the epidemiology, treatment patterns and OS of tAML based on a national database. Methods: A total of 1,611 cases of tAML were identified between 2001-2011 using the National Cancer Database (NCDB). Data on age, race, gender, income, insurance and educational status, Charlson comorbidity index (CCI), receipt of chemotherapy and HCT were abstracted. Log-rank test was used to test equality of survivor function among the variables. Factors that attained statistical significance during bivariate analysis were factored into multivariate analysis using Cox Regression model. Results: Median age at diagnosis was 63 years (range 18-90), with 54% < 65 years, 59% females and 80% Caucasians. 67% underwent chemotherapy (20% single agent, 45% multiple agents and 2% unknown). 19% received HCT. Median OS was 6.7 months (m) with 1-year OS of 33%. Median OS was lower among patients with higher comorbidity burden (7.8m for CCI of 0, 6.0m for CCI of 1, and 3.8m for CCI of 2; p < 0.001), without versus with treatment (3.7m vs 8.3m; p < 0.001) and in Medicare insured compared to Private/Managed care/Medicaid insured. Cox regression model showed the predictors of OS to be: receipt of HCT (relative risk, RR 0.36); use of multiagent chemotherapy (RR 0.80); age > 65 years (RR 1.25), higher comorbidities (RR of 1.21 for CCI of 1, and 1.45 for CCI of 2) and diagnosis on or after 2008 (RR of 0.81). Conclusions: Over half of patients with tAML are younger adults (< 65 years), however, the receipt of chemotherapy and HCT is relatively low. OS is poorer in general but improves with the use of multiagent chemotherapy and HCT. OS is worse in older patients and those with comorbidities. Given a dismal prognosis, older patients should perhaps be managed by leukemia team with expertise in geriatric oncology and should participate in clinical trials of novel therapies.

Introduction: The annual rate of colonoscopies in geriatric population (age > 65) is on the rise. The accuracy, yield, safety, and utility of a colonoscopy is dependent upon its quality of bowel preparation. Age is a known factor influencing poor bowel preparation in elderly patients. A number of other factors including co-morbid conditions, and bowel preparation regimens have been implicated in poor preparation for colonoscopy, however, an often overlooked aspect, especially in the geriatric population, is polypharmacy. To date, no prior study has assessed the role of multiple daily medications on the quality of bowel preparation in this age group. We intend to evaluate the correlation and effect of polypharmacy on the quality of bowel preparation in the geriatric population. Methods: We conducted a retrospective analysis of patients >65 years of age, seen by the gastroenterology service between January 2009 and December 2015, who underwent a colonoscopy examination for screening/surveillance or diagnostic purposes, all utilizing the same bowel preparation regimen consistent of 4 liters of Polyethylene glycol 3350. Basic demographics and underlying comorbidities were noted. Total number of daily medications were recorded. Bowel preparation (good vs poor) was correlated to the total number of daily medications, using a multivariate logistic regression model, thereby adjusting for covariates. Results: 3178 patients (predominantly Black and Hispanic) who underwent colonoscopy were studied, of which 72.5% had good preparation vs 27.5% that had poor colon preparation. There was a statistically significant association between the number of daily medications and the odds of having a poor colonoscopy outcome. A patient taking 5 or more medications on a daily basis had a 27% higher risk of having a poor preparation, compared to a patient taking no medications, with a p-value of <0.001. Overall, the number of comorbidities also predisposed to poor preparation, however, this residual association was entirely explained by the increase in the number of medications as the number of comorbidities rose. By itself, each comorbid condition did not cause a statistical significance in a univariate analysis, except for diabetes mellitus. Interestingly, other than opioids, medications known to cause constipation like iron, anti-cholinergics and psychotropics did not affect the quality of bowel preparation. Conclusion: We have been successful in identifying polypharmacy as a risk for poor quality of bowel preparation for colonoscopy in elderly patients. This translates to a higher rate of missed malignancies and an increased rate of repeat procedures, which adds to the financial burden on our healthcare system. It is imperative that such an influential factor is identified beforehand. We suggest that prior to elective colonoscopies, primary care physicians should consider tailoring medications to only the most necessary ones.
Complications associated with esophageal stenting. A Review article and Meta-analysis.

Introduction:
Self-expandable metal stents are being used to treat benign and malignant esophageal conditions. There is wide variation in rates of complications reported in the literature. The purpose of this meta-analysis is to evaluate the frequency of stent-related complications.

Keywords: esophageal stenting, SEMS, complications.

Material and Method:
A literature search was performed and data was gathered from 45 studies (14 retrospective, 21 prospective & 10 RCTs), after the exclusion of 14 studies. Inclusion criteria: (i) only SEMS (ii) publication year 2000 or later Exclusion criteria: (i) non-English language (ii) plastic or Biodegradable stents. Data extraction: Data was extracted and reviewed by 2 investigators and results were obtained by calculating the weighted average.

Results:
The data included outcomes of 4310 patients among the 45 studies. Dysphagia improvement (Score: 0-4) was reported in 1845 malignant cases to improve by 1.8 scores. Stent migration rate was reported 16.25% in benign and 9.51% in malignant esophageal cases. An average difference noted between FCSEMS and partially covered stents was 15.56% and 8.34% respectively. Chest pain after the stent placement was reported in 31% of the benign and 24% of the malignant cases. The procedural hemorrhage (minor-major) was estimated to be 2.5% in benign and 5.77% in malignant cases. Stent removal secondary to stent complication was 21% and 16.85% in benign & malignant conditions respectively. Stent-related mortality was 3.75% in benign and 4.16% in malignant diseases. The major cause of mortality was hemorrhage i.e. 48.7%, followed by aspiration (21.95%) and perforation (14.64%).

Discussion:
Esophageal stents can be very effective in palliation of malignant dysphagia as well in the treatment of benign esophageal conditions. However, esophageal stents can also be associated with significant risk of complications and rarely be associated with mortality. The careful benefit-risk ratio should be considered in every case.
Liver abscess (LA) has been described as a predictor of the presence of Colon cancer and colonic polyps. Several case reports have described association of cysticogenic liver abscess and identification of occult colonic tumors. However, very few studies have elaborated the relationship between the two.

Methods:
Using the National Inpatient Sample 2014, we identified admissions associated with liver abscess. We calculated the rate of colon cancer and colonic polyps in patients with and without liver abscess. Chi square test and Logistic regression were used to estimate difference in rates and Odds ratios. aim of our study was to look into association of liver abscess with colonic tumors including colon cancer and colonic polyps.

Results:
A total of 5,644,925 admissions of adult patients (=18 years) were reviewed. Of those 3,605 admissions related to the diagnosis of liver abscess (LA) (0.06%) were identified with a median age of 63 years, males = 59.3%. On comparing the presence of liver abscess on the basis of gender, males were more likely to have LA 0.09% vs female 0.04 % (P<0.001). Stratification based on race revealed that the majority were white patients (65.3%), however patients of Asian - Pacific descent had higher odds of having liver abscess (0.15%) (P<0.001).

Other factors found to be associated with higher incidence of LA were chronic liver disease and cirrhosis (0.12%), intestinal perforation (0.84%) and a slight increase with diabetes (0.07%) (P<0.001 for all).

On comparing the rate of colon cancer in patients with or without LA, we found colon cancer was significantly higher in patients with liver abscess 2.64% vs 0.56% (P<0.001) with an Odds ratio = 4.79 [95% CI 3.9 - 5.87]. Colonic benign tumors were also noted with higher rate (1.4%) and Odds ratio: 2.5 [95% CI =1.89 - 3.29] (Relative Risk of 4.68).

Patients with liver abscess and colon cancer were younger than those without abscess (median 61 vs 68 year-old) (P<0.001).

Conclusion:
Liver abscess is associated with colonic masses especially colon cancer with odds ratio of 4.79. This supports previous case series. Our study highlights the fact that in patients presenting with LA, physician should consider screening for colonic mass to rule out colon cancer.

Liver abscess is associated with colonic masses especially colon cancer with odds ratio of 4.79. This supports previous case series. Our study highlights the fact that in patients presenting with LA, physician should consider screening for colonic mass to rule out colon cancer.
New York Chapter ACP
Resident and Medical Student Forum

Resident/Fellow /Medical Student
Quality, Advocacy & Public Policy
Resident/Fellow/Medical Student Quality, Advocacy & Public Policy

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Improving the Rate of Annual Ophthalmologic Exams for Patients on Hydroxychloroquine for Chronic Rheumatic Diseases.

Background: Antimalarials are commonly used for the long-term management of a variety of chronic rheumatic diseases including Systemic Lupus Erythematosis (SLE) and Rheumatoid Arthritis (RA). Hydroxychloroquine (HCQ) is one of the most commonly prescribed antimalarial agents in the United States. Long-term use of HCQ may lead to potentially blinding retinal toxicity. The retinopathy is not reversible and, at present, there is no therapy. Hence, recognition at an early stage is important to prevent central visual loss. The American Academy of Ophthalmology recommends initiation of specialized testing at 5 years after starting HCQ for low-risk patients with normal baseline funduscopic exams. For high-risk patients on HCQ, annual eye examination is recommended.

Methods: This study was conducted in two academic centers in New York, which serve a mixed population. The IT Department helped us identify 72 patients who were given prescriptions of HCQ in either the rheumatology subspecialty clinics of these hospitals or associated practices within the past year. A chart review was performed to evaluate how many of them had either an ophthalmology visit or referral within a year of their visit, with the more aggressive goal of annual ophthalmology evaluation regardless of risk factors. Once baseline data was collected, there was an intervention to improve this rate. This included systematic documentation, annual alerts in EMR, educational flyers and educating patients about the risk of toxicity and the importance of annual screening. Post intervention data was analyzed to evaluate whether there was any improvement in the rates of ophthalmology visits or referrals.

Results: An analysis of the baseline data revealed a rate of ophthalmology referral or visit of 84.7% for patients on HCQ at two centers and the post intervention data shows an increase in the ophthalmology referral or visit rate to 90% at these two hospitals.

Conclusion: The rates of ophthalmology screening at the academic centers were comparable at baseline. Based on the pre and post intervention data collected, the rate of ophthalmology referrals of patients on HCQ improved after a multi-centered, systemic quality improvement intervention suggesting that this is a promising avenue to improve quality of care for rheumatology patients.

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Improving Colorectal Cancer screening rates with the introduction of fecal immunochemical testing (FIT)

Purpose:
Colorectal Cancer (CRC) is the second leading cause of cancer death. In ECMC’s Internal Medicine Clinic (IMC), baseline screening rates were <30% in eligible individuals aged 50-75 seen at least once over 18 months. The purpose of this investigation is to increase the rate of CRC screening by 10% in one year with the introduction of FIT testing for eligible patients aged 50 to 75 in the EMC IMC patient population.

Methods:
This QI study worked to identify barriers regarding, requesting, ordering and completion of FIT testing. The Plan-Do-Study-Act (PDSA) cycles were developed to identify patient, provider, systematic and testing barriers. We used S.M.A.R.T. objectives in addition to the Institute of Medicine’s 6 Aims of Changing the Healthcare System. An electronic patient registry was used to identify baseline CRC screening rates in this patient population and to track FIT ordering. A process flow map was created for standardization between providers and other clinic staff. Outcome measures were to improve CRC screening rates by 10% over one year, and the return of FIT kits with follow-up of positive results. Process measures were identifying patients eligible for screening, and providers ordering the correct screening test with tracking of FIT kits. Balancing measures included a backlog in colonoscopy scheduling from increased referrals and an increase in cycle time for clinic staff education.

Results:
The number of FIT kits ordered since the initiation of this QI study increased each month with each successive PDSA cycles. There was no significant change in CRC screening with colonoscopy, but there was a linear trend for CRC screening with FIT testing, resulting in an overall improvement in screening. There was a 43.1% return rate of FIT kits in February and 40.5% in March. Overall, 40% of patients were up to date with CRC screening by the end of March. An unforeseen limitation was ECMC’s EMR becoming unavailable in early April. Overall screening rates remained at 39% for April and May. Nearly triple the amount of FIT kits were ordered in June when EMR was fully active. In June, 42% of patients were up to date with CRC screening with either colonoscopy or FIT testing

Conclusion:
There are various obstacles with the introduction of FIT testing in order to improve baseline rates of CRC screening in our patient population. Future PDSA cycles aim to further determine patient and provider barriers to eventually reach the ultimate goal of ≥80% by 2018 Pledge. For average-risk patients, FIT testing is the preferred method of screening for our patient population.
Background: Rising healthcare cost is one of the biggest problems facing the United States today. This includes wasteful spending. Urine cultures are often done without indications or without looking into urinalysis results first before being ordered. Additionally, treating asymptomatic bacteriuria is a prime example of inappropriate antibiotic use and expenditure. Purpose: To determine the criteria for reflex cancellation of unnecessary urine cultures using urinalysis results.

Methods: The design was a retrospective study of patients in a community-based teaching hospital. All patients ages 12 years and older who had a urinalysis and urine culture done at the same time from January 1, 2016 to March 31, 2017 were included in the study.

Positive urine culture was defined as cultures with growth of more than 10,000 colony-forming units/mL of any bacteria. Multiple bacterial growths deemed as contamination by microbiology laboratory were considered as negative results. The cut-offs for variables in urinalysis considered as positive were white blood cell count of more than 10 per high-power field, any leucocyte esterase and nitrite other than negative, and any bacteria on microscopy. A urinalysis was considered high-risk if at least one of the variables was positive. A low-risk urinalysis was defined as a urinalysis which was negative on all the variables mentioned.

Results: There were 2,995 patients included in the study. Majority were female (60%, n=1789) and the average age was 65 years old (range 12-105). Majority of tests were ordered by medicine (74%, n=2210), followed by emergency medicine (16%, n=490). 74% (n=2203) of cultures were positive while 26% (n=792) were negative.

Among the four variables, presence of bacteria was the most sensitive in predicting a positive urine culture (88%). Nitrite had the highest specificity of 97%. Leucocyte esterase and bacteria had the highest negative predictive value (90%). All four had dismal positive predictive value, highest was nitrite (69%).

There were 2,203 high-risk urinalyses (74%) and 792 low-risk urinalyses (26%). The sensitivity of a high-risk urinalysis was 94% while the negative predictive value of a low-risk urinalysis was 94%.

There were 46 cases of false negative urinalysis. Only two cases had indication for treatment. Two other cases were treated with antibiotics but without indication. The rest were not treated.

Conclusions: Using the variables noted, urine cultures can be automatically cancelled if the urinalysis is deemed low-risk. In our study, this would have resulted in a 27% reduction in urine cultures with an estimated savings of $8,000. False omission rate was low (6%). By removing cases of asymptomatic bacteriuria, only 0.25% (n=2) would have been falsely omitted. We suggest that specimens for urinalysis and culture be collected at the same time but cultures only be processed if urinalysis identifies high-risk features.
Resident/Fellow/Medical Student Quality, Advocacy & Public Policy

Muhammed Rajib Hossain MD
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CLINICAL DOCUMENTATION IMPROVEMENTâ€”A PROBLEM AND SOLUTIONS- INTERNAL MEDICINE RESIDENT’S PERSPECTIVE SURVEY

Objective: To determine the internal medicine residents’ perceptions of the problems related to clinical documentations and possible solutions to the problems.

Introduction: Clinical documentation plays a critical role in patient care as it helps in coordination between interdisciplinary team members involved in care, to avoid mistakes, duplication of investigations and create an appropriate plan of care. We sought to understand the perspective of medical residents regarding time spent on clinical documentation, challenges to improvement and possible solutions.

Method: A cross-sectional online survey questionnaire was provided to all internal medicine residents at our institution. Residents voluntarily completed the survey. Questions were structured to elicit their perceptions of current practices, challenges and solutions.

Results: A total 87 residents (PGY1-33, PGY2-23 and PGY3-31) responded to the survey, 67% of them spent 15 to 30 minutes on each note and less than 15 minutes seeing each patient at bedside (53%). They spent more than 2 hours on daily progress notes (63%) whereas less than a total of 2 hours at beside of all their assigned patients (62%). There were no significant differences in these findings on the resident’s post-graduate year level. Half of the respondents (49%) always copied and pasted their notes but nearly all participants (95%) edited their notes properly.

Some of the perceived challenges to good documentation included the overly detailed nature of progress notes required for regulatory, billing and legal purpose (67%), burn out or stress (72%), and extra time spent on other clerical documentations (84%), ICD coding (55%).

Most residents responded that shortening the length of clinical documentation (77%) and upgrading EMR system (80%) were possible solutions. Other solutions to improve quality of clinical documentation were using a checklist to prevent propagation of outdated or inaccurate information in the patient chart (79%), upgrading EMR system (80%) and more editing and feedback from faculties and senior residents (75%).

Discussion: Survey demonstrated that residents spent large portion of their time in documenting daily progress notes and in contrast, spent less time in direct patient care. Most residents copy and paste documents from previous notes which could pose a risk to document integrity. But almost all respondents spent time to edit their notes. Disabling copy paste function will not help clinical documentation. They believe that using checklist will help in better documentation. Although clinical documentation improvement is crucial, no published articles to date have systematically reviewed strategies to improve clinical documentation. Further studies on interventions are needed.

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Organ Transplantation and the Need for a Basic Minimum of Healthcare

Relying purely on the altruism of potential organ donors has not produced the amount of organs required to significantly impact the transplant waiting list. Based on current data, there are over 116,000 people on the organ donation waiting list, and it continues to grow. Every ten minutes an individual is added to the list, while a daily average of 20 people die awaiting donations. The gap that divides organ donors from organ recipients is widening. With an average wait time of five years to receive transplantation, the kidney is the most transplanted organ. This time spent waiting on dialysis, limits an individual’s opportunity for a better quality of life. Our current system of organ donation must be re-evaluated. My assertion is that a new system for organ donation will lead to an increase of organs donated, resulting in a shorter wait period for recipients, and thus improving basic minimum of healthcare for individuals.

A literary review was conducted of existing literature and the potential systems concerning organ donation were investigated. Utilizing John Rawls’ Theory of Justice, along with Norman Daniels’ essay â€œJustice, Health, and Healthcare,â€ I contend that individual liberty, and therefore individual opportunity, must be maintained through upkeep of a basic minimum of healthcare. Rawls and Daniels establish the right of individuals to a basic minimum of care. I believe these rights extend to include interventions that improve individual health outcomes and decrease wait time for an organ recipient. The limits placed on organ donation are limits placed on individuals in need, and these restrictions deny citizens of their basic rights to care.

Inspection of various donation systems reveals potential alternatives to the current altruistic-only system. Donors and their family are the only ones not benefitting from the system; doctors, nurses, hospital staff and OPOs all get paid while the recipient gains a better health status. Accordingly, a Reuters-NPR Health Poll determined 60% of participants believe some sort of compensation should be awarded. The duty of beneficence requires alternate approaches to raise the number of healthy organs put into the system, with financial incentives being a consideration. Through donation, organ recipients can increase their quality adjusted life years, and by including donor compensation, the system does not incur added burdens. In creating an organ sharing system inclusive of donor-vendors, the cost of remuneration of the donor would offset the current or projected cost of caring for the ill-recipient continuing in an ill state. Increasing organ donation provides for those in greatest need of care, moreover improving their basic minimum of healthcare. Remuneration of donor-vendors creates a system that adequately meets the needs of an organ-sharing network by maximizing the benefit to donors and recipients.
A QUALITY IMPROVEMENT PROJECT TO IMPROVE OUTPATIENT OSTEOPOROSIS SCREENING RATES IN WOMEN OVER 65 USING A TEAM-BASED HUDDLE

Purpose: To improve the compliance of ordering dual energy x-ray absorptiometry (DXA)-based osteoporosis screening in all female patients age 65 and older who did not have a DXA ordered in the 2 years prior to a regularly-scheduled clinic visit to the internal medicine resident practice from September 2016 to March 2017.

Methods: The U.S. Preventive Services Task Force recommends screening for osteoporosis in women aged 65 years and older since interventions can mitigate the risk and morbidity of low-trauma fractures. DXA is the most widely used method for screening for osteoporosis since it is widely available, offers precise measurements of bone mineral density at various sites, and can be used to monitor response to treatment. The Northwell Improving Patient Access Care Cost Through Training (IMPacCT) program is a grant-funded inter-professional training model that incorporates a team-based multidisciplinary approach to patient care through the use of daily small-team huddles prior to each resident clinic session. With the assistance from the clinic’s medical assistant, women age 65 and older who qualified for DXA screening were identified prior to our daily small-team huddle sessions; this was done by reviewing the electronic records for previous osteoporosis-screening prior to each clinic session. DXA screening status was then highlighted during the huddle and an order was placed for that patient’s visit. Manually reviewing the electronic record for each patient for previous DXA screening took minimal time and was considered sustainable to implementation efforts by the housestaff and the medical assistant.

Results: From September 2016 to March 2017, a total of 52 individual patients were identified as being female, over age 65 at the time of their visit, and eligible for osteoporosis screening; of these, 21 (40.4%) were already up-to-date with their osteoporosis screening by the time of their clinic visit, and a referral was not needed nor provided. In 36 of the remaining patients that were eligible for a DXA scan, an order was correctly placed in 14 of their visits; the other 22 visits where orders where not placed were considered to be "missed." By the end of the study period, 37 of the eligible 52 women had obtained DXA scans and been screened for osteoporosis, raising the clinic practice’s prevalence of appropriate screening in this cohort from 40.4% to 71.2%.

Conclusion: Incorporating explicit inclusion criteria for screening into team-based huddles to identify eligible patients improved the osteoporosis screening rates in our clinic, compared to usual care which relies on the provider to remember to identify which patients are eligible for screening at the time of the clinic visit. The results show that utilizing team members such as medical assistants in a huddle-based approach can help improve health maintenance screening in resident practices.
RESIDENT/FELLOW/MEDICAL STUDENT QUALITY, ADVOCACY & PUBLIC POLICY

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DOES MONEY BUY HAPPINESS? - RELATIONSHIP BETWEEN SALARY, HOURS WORKED, AND SATISFACTION IN MEDICINE SPECIALTIES

Purpose: Internal medicine residents are faced with making career choices with little information on work-life balance and satisfaction by specialty, with a relative paucity of reports that explore the impact of physicians work hours and salary on their overall career satisfaction. We assessed the twelve most commonly sought internal medicine subspecialties to determine the relationship between career satisfaction, hours worked, and salary.

Methods: The most recent (2015) MGMA DataDive, (2011) AAMC, and (2009) BMC Health Services data reports were utilized to determine the relationship between provider compensation, hours worked, and career satisfaction by specialty.

Results: Twelve internal medicine subspecialties were included in the study. The highest median annual salary and hourly wage were noted among gastroenterologists ($529,233 and $181.25, respectively). The lowest median annual salary and hourly wage were noted among ambulatory internal medicine ($233,404.00 and $83.99, respectively). The hours worked per week ranged from 57.5 hours (cardiology: non-invasive) to 48.5 hours (endocrinology). Allergy/Immunology reported the highest career satisfaction (0.50), while the lowest satisfaction score was noted amongst pulmonary/critical care physicians (0.01). The primary specialties of hospital medicine had higher mean satisfaction score compared to ambulatory (0.40 vs. 0.24) - generalist fairied between these two specialties with respect to compensation but had lower mean satisfaction score (0.19). Hours worked per week had a negative correlation with satisfaction (r = -0.41) i.e., the more hours were correlated with worse satisfaction. In contrast, hourly wage had little correlation with career satisfaction (r = 0.03).

Conclusions: We report satisfaction among internal medicine specialists is related to the work hours rather than compensation. The paradoxical finding may be due to better work-life balance that may be a better predictor of career satisfaction than physician compensation. Prior studies have explored work hours and income separately. Consistent with our findings, prior studies among all specialties (including surgical and non-medicine) show that specialties with what may be considered a better lifestyle (i.e. lighter work hours and call schedules), may be more satisfied with their current professional life. In contrast, other reports have suggested that higher income is associated with an increased likelihood of being very satisfied, along with a decreased likelihood of being dissatisfied across all specialties.

Our findings indicate that overall compensation has little bearing on a physician’s satisfaction, while work hours seem to have an impressive role – suggesting the significant impact of work-life balance among clinicians. Though correlation does not mean causality, our findings raise important question whether money buys satisfaction, or more generally happiness - our study reports not to be the case. These findings may be useful for employers, graduate medical educators, and physicians - particularly internal medicine residents and their advisers in making an informed decision regarding specialties as a career.

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YOUTUBE & HEPATITIS C: WHAT A PATIENT CAN HEAR AND SEE

According to internal calculations, the popular video sharing service known as YouTube has reached 88 countries in 76 different languages. Almost one-third of internet users are on YouTube, and viewers watch over a billion hours of video per day. Various genres and content can be found in these videos, including educational models. Thus, technologically-literate patients often turn to this tool to shed light on common medical issues affecting them and their loved ones. For example, they can access media for advice, support groups, expected outcomes and common therapies.

According to the Centers for Disease Control and Prevention, Hepatitis C affects an estimated 2.7-3.9 million Americans. This represents a sizable population of Americans that no doubt look for guidance from their peers. As a contagious disease that mainly affects the liver, Hepatitis C is transmitted most commonly via blood products, specifically via IV drug use and blood transfusions before the 1990’s, although there are cases of transmission via bodily fluids.

The first 50 videos that came up on YouTube upon inputting “Hepatitis C” were viewed, evaluated, and graded using the Global Quality Scale. The videos were chosen based on the search input, and without filters, to mirror the experience a patient would undergo when searching the term themselves. The scale uses a score that ranges from 1 to 5; 1 indicates a poor quality video missing important clinical information, while a score of 5 indicates a valuable video covering clinically useful information for patients. Videos were considered misleading if the material provided by the video contained content unrelated to clinically proven facts.

The results of the study were such that, on average, a video scored 4.114 on the quality scale, lasted 456 seconds long, was viewed 72,457 times, and had been online for 1,112 days. 94% of the videos were meant to educate the public, 38% were examples of personal experiences, 10% were drug ads, and 6% were labeled misleading with clinically ambiguous evidence. These results demonstrate the vast potential of media in educating the public about Hepatitis C. 94% of the videos had educational components to them, while over a third of the videos had personal anecdotes included. Patients can access these videos in preparation for their doctor visit, or they can utilize them after the visit to find more information regarding the physician’s recommendations. As there is no regulatory oversight for the videos posted, physicians must be wary of and educated regarding online content, and stress the possible falsehoods that must be checked with a licensed healthcare professional. With tremendous strides in treatment to bring the cure rate of Hepatitis C close to 100%, it is imperative that healthcare professionals assist in directing our patients towards safety.