Decreasing Inappropriate Use of IV Levofloxacin in a Hospital Setting

Fluoroquinolones have a unique pharmacological aspect, bioavailability is same for both IV and PO forms. Despite this, prescribers have favored using IV form of levofloxacin during inpatient hospitalizations, compromising patient safety and increasing cost. Our aim is to reduce inappropriate IV levofloxacin use by 50% in non-indicated settings and encourage early IV to PO conversion. Institute of Healthcare Improvement model was used for this quality improvement project. An interdisciplinary team approach was utilized which included physicians, pharmacists and EMR specialists. Inclusion criteria consisted of inpatient diagnosis of pneumonia or COPD exacerbation who were tolerating oral diet.

Our project completed 3 PDSA cycles summarized here.

PDSA 1: Didactic sessions to physicians were provided. Post intervention review of charts showed a reduction from 46% to 26%.

PDSA 2: In collaboration with the P&T committee and the antimicrobial committee, an automatic switch from IV to PO Levofloxacin after 48 hours of hospitalization was implemented. Post intervention showed a reduction to 14%.

PDSA 3: Project was extended to 3rd cycle to educate new class of residents and monitor sustainability. Post intervention data analysis revealed 6% inappropriate IV levofloxacin prescriptions.

Use of IV levofloxacin in a non-indicated setting increases length of hospital stay, diminishes already limited resources and increases health care costs. We used a multidisciplinary approach to address this problem. Through education and EMR changes, our team was able to achieve favorable outcomes and we plan to monitor sustainability through periodic analysis of data.
Post Operative Euglycemic Diabetic Ketoacidosis

Introduction: Sodium-glucose cotransporter-2 (SGLT-2) inhibitors are newly approved antihyperglycemic medications which have been associated with a life-threatening complication, euglycemic diabetic ketoacidosis.

Case Description: 53 years old male with history of Type-II DM presented with fever, shortness of breath and abdominal pain on post-operative day-2 status-post laparoscopic appendectomy. On presentation, he was febrile, tachycardic, tachypneic and hypoxic. Physical examination was unremarkable. His medications including canagliflozin were held prior to surgery. Initial labs revealed increased anion gap metabolic acidosis with elevated ketones, normal lactate, normal blood glucose, renal function and electrolytes. Urinalysis revealed glucosuria and ketonuria. CT abdomen and chest were essentially unremarkable. Patient didnot respond to initial treatment with antibiotics. Further questioning revealed that patient resumed canagliflozin prior to readmission. Treatment with intravenous dextrose and regular insulin was started for euglycemic ketoacidosis. By next day, patient’s clinical symptoms and metabolic acidosis improved.

Discussion: Euglycemic ketoacidosis is a rare but life-threatening condition associated with SGLT2-inhibitors. Absence of hyperglycemia makes the diagnosis more challenging. Current guidelines suggest stopping these drugs 24hrs pre-operatively, but there are no guidelines suggesting when to resume. Our patient presented with euglycemic ketoacidosis even though canagliflozin was held prior to surgery and resumed 48hr postoperatively. Interestingly, our patient persisted to have glucosuria (urine glucose> 1500mg/dl) even after 72hours (> 6 half-life) from his last dose. This can be explained by non-time dependent pharmacokinetics of Canagliflozin leading to its accumulation in plasma after multiple doses. Thus, It is challenging to decide when to safely resume canagliflozin post-operatively.
A Rare Cause of Diffuse Alveolar Hemorrhage

Introduction: The incidence of diffuse alveolar hemorrhage induced by new oral anticoagulants has not been well elucidated. We present a case of this rare complication.

Case: 75-year-old man with history of COPD, CAD, atrial fibrillation (on apixaban) presented with cough, fever and exertional dyspnea. He denied other symptoms including orthopnea and leg swelling.

On examination, he had reduced air entry with wheezing in bilateral lower lobes. In emergency department, patient was found to be in severe sepsis with fever (Temp: 101), leukopenia (WBC: 3.4) and hypotension with systolic blood pressure in the 70s which improved to 90s after receiving fluid resuscitation. Chest X-ray revealed bilateral lower lobe infiltrates and CT chest showed patchy airspace disease in the lower lobes, both concerning for pneumonia.

He was started on broad-spectrum antibiotics for treatment of sepsis most likely due to pneumonia and steroids for COPD exacerbation. Patient failed to respond and worsened clinically. Eventually, bronchoscopy was done which revealed diffuse alveolar hemorrhage. Coagulation, Infectious, autoimmune workup was negative. He clinically improved after stopping apixaban. A follow-up Chest x-ray, 2 months after stopping apixaban was normal.

Discussion: Apixaban induced diffuse alveolar hemorrhage is a rare complication and presents mostly with hemoptysis. The absence of hemoptysis and presentation mimicking pneumonia makes the diagnosis more challenging, as in our case. We encourage cautious use of new oral anticoagulation especially in patients with underlying chronic lung conditions. Further studies are needed to reassess the risk versus benefit of anticoagulation in above described clinical setting.
Introduction:
Herpes Simplex Encephalitis (HSE) is a devastating central nervous system infection that if untreated is almost universally fatal. Even if properly managed, HSE carries a near certainty of permanent neurological dysfunction. Early antiviral therapy is critical in optimizing management to improved outcomes and requires prompt clinician recognition of defining clinical features. Although encephalitis often presents with easily identifiable phenotypes, particular diagnostic challenge arises when encephalitis is encountered in the setting of atypical parenchymal manifestations.

Case:
A 39-year-old male reported to the hospital with debilitating headache and fever. Exam demonstrated overt meningismus, however extensive neurologic exam was unremarkable. He was febrile and bradycardic with significant leukocytosis. Lumbar puncture revealed lymphocyte predominate pleocytosis, and indices consistent with viral pathogen. He was hospitalized for empirical antimicrobial therapy, including Acyclovir. Over the first 48 hours, his clinical condition worsened. He developed profound bradycardia consistently in the 20’s, however serial neurological exams remained unchanged. The following day he began exhibiting severe neuropsychiatric disturbances, lethargy, and loss of gustatory function. CSF testing confirmed HSV-2 by PCR. Patient ultimately responded to Acyclovir therapy in the following days, with parallel resolution of neuropsychiatric symptoms and bradycardia, however had persistent gustatory changes.

Discussion
Encephalitis concerns should extend to atypical signs and symptoms representing brain tissue disfunction, and thus underlying encephalitis. HSE shares the target insult location, the temporal lobes, with the established epilepsy induced Ictal Bradycardia Syndrome, and suggests bradyarrhythmia as both a primary temporal lobe neuronal insult, and previously unrecognized encephalitis defining syndrome.
Linezolid-Induced Interstitial Lung Disease

Introduction
Common side effects of linezolid include peripheral neuropathy, thrombocytopenia and serotonin syndrome. Drug induced interstitial lung disease (DILD) as an adverse effect has not yet been established.

Case
A 34-year-old male with a history of injection drug use, hepatitis C, and seizures presented to the emergency department for a syncopal episode with associated urinary incontinence thought to be due to seizure. The patient reported that he had developed a nonproductive cough and dyspnea two weeks prior with no associated fever, chills or sick contacts. In addition, he had been taking linezolid for three weeks for right lower extremity infection. At presentation, he was tachypneic, tachycardic and hypoxic. On examination, he appeared tachypneic, his lungs were clear and his right lower extremity demonstrated excoriations without active soft tissue infection. His WBC was 13.9 with predominant neutrophils and chest xray demonstrated diffuse interstitial changes bilaterally. His linezolid was held and two days later, the patient’s cough, hypoxia and dyspnea had resolved, WBC had normalized and repeat CXR showed significant improvement.

Discussion
Linezolid has been associated with DRESS syndrome but has not been linked to DILD. DILD can be caused by numerous medications including antibiotics such as isoniazid, nitrofurantoin, and sulfasalazine. Diagnosis is made clinically and is mainly based on excluding other causes. Eosinophilia may or may not be present. Treatment involves discontinuation of the offending agent and in acute cases, symptoms tend to resolve within 24 to 48 hours. Clinicians should be aware of this potential side effect of linezolid.
Bifidobacterium longum: A Rare Case of Severe Bacteremia in Adults

Introduction:
Mostly known for inhabiting the gastrointestinal tract of humans and used as a probiotic, bifidobacterium spp. is generally regarded as a benign organism of the gastroenterointestinal flora. Rarely seen as a sole etiology of invasive human infections, we present a surprising case of bifidobacterium longum causing severe bacteremia in the setting of possible colonic source.

Case Description:
A 66 year old female presented to the ED with progressive dyspnea, purulent cough and fevers. She was admitted for acute hypoxic respiratory failure requiring BiPAP; subsequent sepsis workup to identify respiratory etiology was negative. Her respiratory status worsened with the development of severe sepsis ultimately requiring intubation. Additional sepsis workup to evaluate other sources of sepsis revealed rectum thickening on CT Abd/Pelvis with positive blood cultures for enterococcus and bifidobacterium longum. Further investigation of history suggested a suspicion for an infectious GI source. Inpatient endoscopy was recommend but due to her critical respiratory status, it was unable to be performed. Following a course of IV Unasyn, she eventually was extubated and later transferred to LTAC for continuing care.

Discussion:
Our case would be the first documented case of bacteremia due to bifidobacterium longus in an adult. Not only an unsuspected source of sepsis, but prior cases have only been reported in pediatric patients, and this would be the first known adult case of Bifidobacterium longum causing bacteremia. In such cases, a gastroenterologist should be consulted and endoscopic evaluation should be employed to identify the orogastroentestinal tract as the leading source.
INTRODUCTION
Posterior Reversible Encephalopathy Syndrome (PRES) is an increasingly recognized and reported condition thought to be due to disordered cerebral autoregulation, commonly from hypertensive crisis, preeclampsia or cytotoxic immunosuppressive therapy. Management includes controlling underlying settings and antiepileptic therapy. Prognosis remains vast, with most patients recovering within two weeks.

CLINICAL CASE
A 53 year old Female with a history of Bipolar, COPD and hypertension presented to the ED due to acute toxic encephalopathy from polypharmacy. Initial CT Head without contrast depicted no acute abnormalities. On the fourth day of admission, she began having new-onset myoclonic seizures, localized to her right upper extremity. She was transferred to ICU due to status epilepticus requiring midazolam drip and eventually intubation. Repeat CT and MRI imaging showed acute hypoattenuation involving bilateral cerebral white matter of occipital and parietal lobes, compatible with PRES. Of note, she had uncontrolled hypertension throughout early admission, likely precipitating PRES. Requiring multiple antiepileptics and nicardipine drip for hypertension control, her epilepsy resolved and she was extubated. Follow-up imaging showed improvement of cerebral hypoattenuation. Unfortunately, her mentation did not return back to baseline within two weeks. Enduring an almost 8 week hospital course, she was eventually discharged to a subacute rehab for further care.

DISCUSSION
This case depicts a unique clinical condition likely as a result of uncontrolled hypertension. This case represents a classic underlying etiology of PRES, however, our patient challenges the notion that the prognosis generally resolves within two weeks.
Acute Tianeptine Triple Toxicity

Tianeptine Sodium is a non-FDA approved atypical tricyclic antidepressant marketed in the US through online vendors as a treatment of major depressive disorder. We report a case of Tianeptine toxicity presenting with severe signs and symptoms of serotonin syndrome, TCA toxicity, and opioid intoxication, followed by withdrawal signs post treatment.

Clinical Case: A 23-year-old man presented with multiple new onset seizures after consuming substantial doses of Tianeptine Sodium. Upon arrival he was obtunded with constricted pupils. He responded to intravenous naloxone, however, he became agitated and restless. Vital signs were significant for hypertension, tachycardia and tachypnea. Physical exam revealed diaphoresis, horizontal nystagmus, mydriasis, resting and intentional tremors, hyperreflexia with spontaneous myoclonus, and bilateral Babinski signs. ECG demonstrated sinus tachycardia with a QTc of 569ms. Urine drug screen was negative for drugs of abuse. His was treated with IV lorazepam, oral cyproheptadine and sodium bicarbonate. His condition worsened with fevers, muscle rigidity and acute kidney injury, requiring intubation and dantrolene. He gradually improved but then experienced auditory and visual hallucinations which later subsided. Subsequently, he developed flu-like symptoms, nausea, vomiting, watery diarrhea, and anxiety. No sources of infection were found. Tianeptine Sodium is described as having novel neurochemical profiles different from other TCAs and is increasingly recognized to have remarkable addictive properties. It is important to recognize effects of this drug to appropriately provide treatment and support.

With few published cases of tianeptine toxicity, this is first to elucidate such multiple, simultaneous syndromes induced by its intoxication and withdrawal.
A Swampy Cause of Cholangitis

Ascending cholangitis is a life threatening condition. It occurs due to an obstruction of the biliary duct system. Common pathogens found in acute cholangitis include Escherichia coli and Enterococcus species. However, it is very unusual to see Edwardsiella tarda as a cause of the infection. Here we discuss a case of ascending cholangitis with this very unusual pathogen.

90-year-old Caucasian male with an extensive medical history presented with a three-day history of fever, abdominal pain, and generalized weakness. He had recently returned from a six month stay in Florida. Liver enzymes were elevated on admission. A CT of the abdomen revealed acute cholecystitis with a calculus in the neck of the gallbladder. Blood and gallbladder fluid cultures grew both, E. coli and Edwardsiella tarda. A penrose drain was placed to drain the gallbladder and he was treated with broad spectrum antibiotics.

Edwardsiella tarda is a rare but serious food and waterborne infection that results in high mortality. It can cause mild infections like gastroenteritis to more severe infections like ascending cholangitis, peritonitis, and meningitis. E. tarda is not a well documented infection in humans but should be suspected in patients, presenting with GI symptoms, who have exposure to fresh or brackish water in states surrounding the Gulf of Mexico. A high clinical suspicion for unusual pathogens, based off a well-taken history can decrease time to diagnosis and improve mortality in these deadly conditions.
Can Social Stress Break the Heart Twice?

Introduction:
Stress cardiomyopathy (takotsubo cardiomyopathy, Broken Heart Syndrome) is a syndrome characterized by transient regional systolic dysfunction of the left ventricle, mimicking myocardial infarction, but in the absence of angiographic evidence of obstructive coronary artery disease or acute plaque rupture. There are few cases documenting recurrent Takotsubo cardiomyopathy.

Case:
69-year-old female presented with one day history of chest pain with elevated troponins. Cardiac catheterization revealed normal coronary arteries but an ejection fraction of 10% compared to normal EF in January 2017. Her hospitalization was complicated by cardiogenic shock requiring impella device. After a prolonged hospitalization she made a full cardiac recovery.

She was diagnosed with Takotsubo cardiomyopathy on 2016. At that time she had similar presentation with EF of 5-10%, and also achieved full cardiac recovery. Both episodes were triggered by social stressors.

Discussion:
Stress cardiomyopathy was first described in 1990 in Japan. Stress cardiomyopathy occurs in approximately 1 to 2 percent of patients presenting with troponin-positive suspected acute coronary syndrome (ACS) or suspected ST-elevation myocardial infarction. Pathogenic mechanisms include catecholamine excess, microvascular dysfunction, and multivessel coronary artery spasm. A physical or emotional trigger is often but not always present.

Diagnostic criteria include presence of transient regional wall motion abnormalities, absence of angiographic evidence of obstructive coronary disease, presence of new electrocardiographic abnormalities or modest troponin elevation, and absence of pheochromocytoma or myocarditis.

Conclusion:
Takotsubo cardiomyopathy is rare, but should always be suspected when patients present with chest pain without any precipitating conditions, specially with history of Takotsubo.
The First Reported Case of Pasteurella Canis Osteomyelitis Without Animal Bite Exposure

Pasteurella is a group of gram negative coccobacillus that are commonly found in the oropharynx of animals. These organisms can cause a variety of infections that are a result of animal bites. Pasteurella canis is less commonly reported in literature and rarely causes osteomyelitis. Currently there are only two cases of osteomyelitis from Pasteurella canis following an animal bite in literature. We report the first documented osteomyelitis from Pasteurella canis without associated animal bite or elevated acute reactive agents.

A healthy 23-year-old woman presented to the urgent care center complaining of worsening swelling of the right hand. The pain began after she fell while walking her canine. She denied any animal bite or systemic symptoms. Weeks later, erythema, warmth and edema were noticed. Vitals were normal but swelling of the fourth digit was evident. Initial laboratory testing displayed normal WBC, ESR and CRP. Blood cultures were negative. Initial radiograph showed an irregular lucent lesion involving the fourth digit of unknown significance. MRI confirmed osteomyelitis of the middle phalanx and biopsy revealed the rare Pasteurella canis.

Pasteurella is known to cause zoonotic infections in humans. Pasteurella canis is an unusual cause of osteomyelitis. Currently, there are only two case reports of Pasteurella canis osteomyelitis following an animal bite and zero documented cases of Pasteurella canis osteomyelitis without animal bite exposure. This case highlights an unusual presentation of osteomyelitis and emphasizes the value of proper history documentation and physical examination in arriving at the correct diagnosis despite normal laboratory values.
A Case of Infectious Mononucleosis Complicated by Multiple Neck Space Abscesses

Infectious mononucleosis (IM) is characterized by lymphadenopathy, fever and tonsillar pharyngitis that may persist for several weeks. Persistence of symptoms may delay identification of life threatening complications.

A 24-year-old female with a recent diagnosis of IM presented to the hospital with complaints of odynophagia associated with worsening dysphagia, lymphadenopathy and fever for two weeks. On physical exam, she had bilateral cervical lymphadenopathy and enlarged non-draining tonsils. Lab work revealed leukocytosis, hyperbilirubinemia, transaminitis and positive heterophile test. On the third day of admission, she developed respiratory stridor and fever. CT scan of the neck showed peritonsillar abscess descending into the left parapharyngeal space. She underwent urgent incision and drainage (I&D) of the left parapharyngeal abscess and IV vancomycin and clindamycin were initiated. The patient remained intubated and was transferred to the medical intensive care unit. Repeat CT scan showed a new left multilocular abscess. The patient underwent a second I&D and was extubated and improving. Wound cultures grew Eikenella Corrodens so she was started on IV ampicillin/sulbactam. Patient then developed swelling of the right neck. CT scan showed right parapharyngeal abscess that required another I&D. Given the patient’s severe presentation, she was tested for HIV and was negative.

Review of literature revealed a few case reports describing IM resulting in peritonsillar abscess. However, to our knowledge, this is the first case report describing multiple deep neck abscesses secondary to IM in an immunocompetent patient. Identifying complications associated with IM may be challenging given the relatively long course of this acute infection.
Mesenteric Leiomyosarcoma: A Case Report

Mesenteric leiomyosarcoma is a very rare mesenteric solid tumor, usually misdiagnosed as a gastrointestinal stromal tumor (GIST). Leiomyosarcoma typically presents with nausea, vomiting, weight loss, and a palpable mass. Surgical resection is the treatment of choice for this tumor. Leiomyosarcoma has a less favorable outcome compared to GIST.

Case Report: A 68-year-old female with a history of hypertension, diabetes mellitus and latent tuberculosis presented with decreased oral intake secondary to nausea, vomiting, and diarrhea for two days. She had lost 10-pounds over the previous two months. On examination, there was mild right upper quadrant tenderness. Computed tomography (CT) with contrast revealed a large right-sided solid mesenteric tumor, causing extrinsic compression of the proximal and midportion of the right ureter with hydronephrosis. CT guided biopsy showed smooth muscle neoplasm consistent with leiomyosarcoma. A right ureteric stent was inserted, followed by explorative laparotomy. The tumor was encasing the right ureter. Excision of the tumor with the midportion of the right ureter was done with anastomosis of the upper and lower portion of the right ureter. The patient improved postoperatively and after six months there is no recurrence.

Conclusion: Leiomyosarcoma is a very rare mesenchymal tumor of the gut. A biopsy is mandatory to differentiate it from GIST. Nausea and vomiting with weight loss are the usual presentation, as the tumor is often extramural without mechanical obstruction of the gut. Treatment usually is by surgical resection with frequent imaging follow-up.
When Statins Strike Back: A Rare Case of Statin-Associated Autoimmune Myopathy

Statins are used daily in the primary and secondary prevention of cardiovascular disease. Statin-associated autoimmune myopathy (SAM) is a rare side effect of statin use attributed to a discrete autoimmune entity within the statin induced myopathy spectrum.

70-year-old male with a history of hyperlipidemia presented with proximal muscle weakness and myalgias after viral illness 8 weeks prior. Outpatient workup revealed transaminitis and medium intensity statin therapy was discontinued without improvement. Initial exam revealed 3/5 strength in proximal muscles, tenderness to palpation, preserved neurologic function without dermatologic findings. Laboratory analysis revealed persistent transaminitis, elevated CPK greater than 16,000 and myoglobinuria. Treatment with IV fluids for presumptive diagnosis of statin induced rhabdomyolysis was initiated. His symptoms progressed to dysphagia requiring mechanical feeding. Persistently elevated CPK with negative inflammatory myopathy workup prompted us to obtain HMG CoA reductase antibody leading to the diagnosis of SAM. Muscle biopsy was obtained which showed necrotic fibers varying in size with foci of mild endomysial inflammation with increased capillary staining for membrane attack complex, no other features of dermatomyositis were present confirming the diagnosis. The patient was treated with steroids, IVIG, azathioprine and inpatient rehabilitation with significant improvement.

Despite an incidence of approximately 1 in 100,000, SAM must be considered in patients presenting with a clinical picture consistent with statin induced rhabdomyolysis especially if symptoms persist despite stopping the statin. This autoimmune phenomenon must be diagnosed and treated promptly with immunosuppression otherwise it can be self-perpetuated resulting in progressive significant debility.
A Case of Delayed Torsade After 2 weeks of Loperamide Ingestion. Implications for Sudden Cardiac Death Prevention

Introduction: Loperamide is increasingly being used as an alternative to heroin drug abuse. We report a case of torsades de pointes after an excessive Loperamide ingestion.

Case presentation: A 27-year-old male presents with exertional presyncopal episodes with palpitations. EKG showed sinus bradycardia with QTc interval of 571, left axis deviation and Brugada pattern. He was placed in CVICU for monitoring. The patient was a previous heroin abuser and had recently been taking 150 capsules of loperamide at once to achieve euphoria. Serial EKGs showed gradual shortening of the QTc interval but later he developed TdP and ventricular fibrillation requiring defibrillation. A loop recorder was implanted due to QTc lengthening beyond 500 msec for >14 days after last reported ingestion. A stress test was performed which showed appropriate QTc shortening during exercise. He was then discharged with a Life Vest. Subsequent genetic testing for long QTc variants was negative.

Discussion: Published literature suggest that Loperamide does not cross the blood brain barrier (BBB), which make it preferred for anti-diarrheal use as a “safe” OTC medication. Recent case reports show that it might not be as safe as we believed and it might cross the BBB to produce the desired mood changes per abusers. Serious and life-threatening effects, including cardiac arrhythmias, might occur after Loperamide abuse. Our case highlights the risk of delayed cardiac arrhythmias and torsades even after 2 weeks of drug ingestion. Loperamide is believed to prolong Qtc by affecting potassium currents related to HERG protein.
Anchoring Leading to the Delay of Diagnosis of Malignancy

Introduction
Anchoring is a form of bias that leads clinicians to adhere to an initial diagnosis and rationalize information as it comes to make it fit the clinical picture of the initial diagnosis. This can be dangerous and lead to serious diagnoses being missed or diagnosed too late.

Case Presentation:
A 70-year-old male presented with non-radicular back pain and decreased urine output. He was recently admitted obstructive nephropathy presumably caused by his well-known history of BPH. He presented in the same fashion with the addition of back pain and hypercalcemia with appropriate suppression of PTH. His PTHrp was significantly elevated. MRI showed possible osteomyelitis with possible underlying abscess. Neurosurgery drained the paraspinal fluid collection and biopsy showed squamous cell carcinoma. He underwent laminectomy. Patient was then found to have bladder mass on CT. He underwent cystoscopy with biopsy which showed invasive urothelial carcinoma of high grade with squamous differentiation.

Discussion:
Hypercalcemia is a well-known manifestation of malignancy. However, this malignancy could have been diagnosed earlier. This patient was admitted one month prior to the admission in which he presented with back pain, hypercalcemia, and obstructive uropathy. His presenting symptoms one-month prior was also for obstructive uropathy, however no cystoscopy was performed and no workup for other causes of obstruction were pursued, likely because of the patient’s past history of BPH with bladder outlet obstruction status post core-therm ablation. It is possible that anchoring on the initial diagnosis led to a delay in his diagnosis of malignancy.
Hyperuricemia and Coronary Artery Disease - A Systematic Review of the Literature and Meta-Analysis

Introduction: For 5 decades, the independent role of hyperuricemia in the etiology of coronary heart disease (CHD) has been a question. Studies to date have shown inconsistent findings. We conducted a meta-analysis to investigate this relationship.

Methods: Using PubMed, a systematic review of the literature was conducted. From 35 studies, we included 13 studies with these characteristics: prospective, more than 1 year of follow-up, subjects free of CHD or renal disease at the outset, used multivariable analysis to control for other risk factors, included serum uric acid as a risk factor and coronary artery disease (CAD) as the outcome. A meta-analysis was conducted with the 8 studies that provided an hazard ratio (HR). Data were analyzed with NCSS v. 12.0 and the variance was calculated per Cochrane guidance.

Results: The non-directional zero, directional zero and heterogeneity hypotheses were rejected (p<0.0001). From the random effects model, a summary HR of 1.17 (95% CI: 1.05, 1.30) was computed, indicating that hyperuricemia increased the risk of CAD by 17%.

Discussion: Uric acid has been proposed to cause atherosclerosis via endothelial dysfunction and by causing inflammation through the production of interleukins, TNF-alpha and Chemokine Monocyte Chemoattractant Protein-1. From the meta-analysis, hyperuricemia appears to be an independent risk factor for CAD. Thus, treatment of hyperuricemia may help to decrease the risk of CAD.

Conclusion: We found that hyperuricemia is an independent risk factor for CAD. Future studies should address the role of detection and treatment of hyperuricemia in the prevention of CAD.
Unmeasurable Concern: The Extremes of Tacrolimus Toxicity

Tacrolimus is an immunosuppressive medication used in preventing organ transplant rejection. It has a very narrow therapeutic window and needs to be monitored for dose adjustments. Blood levels can be affected by drug to drug interactions, infection, and persistent diarrhea. Here we present a classic case of tacrolimus toxicity with an initial tacrolimus level that was alarmingly too high to quantitatively measure.

A 69-year-old African American male with pMHx significant for heart transplant on tacrolimus 1.5mg BID was sent over by his nursing home for abnormal labs. Patient did not have any new complaints but mentioned diarrhea. After further observation, patient became encephalopathic and was anuric. Initial labs revealed BUN 68, Creatinine 4.62 with baseline around 2, CO2 12, AG 16, and a tacrolimus trough level > 60 ng/mL. Patient’s kidney function did not improve and required dialysis. The tacrolimus level remained > 60 for three days and did not drop to < 10 ng/mL until eleven days later despite tacrolimus being held. Patient was provided supportive care and his neurologic status returned to baseline within 7 days, although he continued to require dialysis. This case illustrates not only the effects of tacrolimus toxicity, but the importance of close monitoring and dose adjustments. It is unclear how the patient got to unmeasurable levels, but persistent diarrhea may have been a component. Clinicians should be aware of this symptom, potential drug-drug interactions, and nonspecific signs of toxicity such as altered mental status and decreased urine output when prescribing this medication.
Clostridium septicum Infection Presenting as Cerebral Edema and Pneumocephalus

Background: Clostridium septicum is a rare infection typically noticed in immunocompromised patients with gastrointestinal malignancies. This infection presents in a fulminant manner with a high mortality rate preventing early recognition. Clinical suspicion in the right setting can assist in the selection of optimal empiric therapy.

Case presentation: A 60-year-old female patient with metastatic breast cancer developed a sudden onset of epigastric pain, foul-smelling bloody diarrhea, nausea, vomiting, fever, and fatigue one day prior to admission. The patient received appropriate supportive care including IV vancomycin, cefepime, and flagyl. The patient’s condition deteriorated with severe mental status changes and she was put on a ventilator. Key findings of the CT scan demonstrated diffuse loss of gray-white matter demarcation, pneumocephalus, new diffuse sclerotic changes, bilateral temporal subcutaneous emphysema and evidence of a right frontal/parietal scalp contusion. Abdominal CT showed signs of ascending colitis. The patient failed to respond to therapy and died. C. septicum was confirmed in blood cultures after the patient’s death.

Conclusion: Bacteremia with sepsis is a rare disorder associated with C. septicum but carries a 60% mortality rate, often within 24 hours. Metastatic lesions and necrotizing skin and soft tissue infection are seen but the acute presentation with central nervous system involvement and pneumocephalus is even more unusual. The early clinical suspicion and adding penicillin-G/clindamycin to the antibiotic regimen can be helpful in reducing mortality.
High Dose Insulin: When Epinephrine Is Not Enough

Introduction: Suicide rates in the US are increasing, and intentional poisoning is among the top three methods of attempted suicide. Overdose on commonly prescribed medications is frequently encountered. This case highlights management of overdose on antihypertensive agents, specifically, beta blockers and calcium channel blockers.

Case Presentation: A 60 year old male presented to the ER after being found unresponsive with empty bottles of metoprolol, amlodipine, lisinopril, and melatonin. Upon arrival, the patient was minimally responsive, hypotensive, and bradycardic. He was given multiple injections of glucagon, atropine, and calcium without any improvement. Patient was intubated secondary to increasing lethargy. Central venous access was obtained, and dopamine, glucagon, and epinephrine were titrated to maximal doses with no improvement in blood pressure or heart rate. High dose insulin (HDI) therapy was started and titrated to 10 units/kg/hr, and hemodynamics improved. He was able to be weaned off pressure support, and HDI was titrated down and discontinued. The patient was extubated, and after a prolonged hospital recovery, and after being cleared by psychiatry, he was discharged to a rehabilitation facility.

Discussion: This case demonstrates the effectiveness and safety of HDI therapy in the setting of beta blocker and calcium channel blocker overdose. In high concentrations, insulin has positive inotropic properties, increases myocardial uptake of carbohydrates, and overcomes the relative insulin deficiency that is present in calcium channel blocker overdose. In addition, HDI improves response to catecholamines and aids the reversal of metabolic acidosis by accelerating the oxidation of lactate in muscle cells.
Concomitant Primary HIV and Cytomegalovirus (CMV) Infection

Introduction: Co-infection of HIV and CMV is common but acute concomitant HIV and CMV infection is rare. We report a patient presenting with acute symptomatic co-infection due to HIV and CMV.

Case: A previously healthy 25 yo male presented with one week of nausea, vomiting, abdominal pain, diarrhea and cough. On examination, he had a Tm 103°F, HR 95/minute and appeared weak and had mild abdominal distension. Initial tests were as follows: amylase 246, lipase 172, ALT 167, AST 261, and chest Xray demonstrated possible right middle lobe infiltrate. HIV testing was performed after he admitted to a new unprotected sexual contact three weeks earlier. He was hydrated and treated with IV ceftriaxone and azithromycin. HIV antibody was positive, CD4 100 and HIV RNA was > one million copies. He had progressive bilateral pulmonary infiltrates and worsening oxygenation despite antibiotics and a CMV PCR was 26,100 IU/mL and beta-D-glucan was negative. His antibacterial agents were discontinued and IV ganciclovir and he had gradual improvement. He was discharged home on oral valganciclovir and trimethoprim/sulfamethoxazole prophylaxis. He was seen in the office two weeks later, HIV therapy was initiated and he completed a one month course of anti-CMV therapy.

Discussion. Acute HIV infection is associated with a wide range of symptoms and may be associated with opportunistic illnesses such as Pneumocystis pneumonia, thrush and CMV reactivation. Concomitant primary HIV/CMV co-infection is rare but should be considered especially in patients with pneumonia unresponsive to routine antibiotics and concomitant pancreatitis.
The Clinical Features of Pneumonia Caused by Legionella Pneumophila vs. Streptococcus Pneumoniae: A Retrospective Study

Introduction: Legionnaires’ disease is a serious pneumonia that is caused by Legionella bacteria. Clinical features may include extrapulmonary clinical manifestations, such as the presence of acute confusion, diarrhea, fever up to 104 °F, and hyponatremia. These clinical manifestations may however be associated with other bacterial causes of pneumonia. Since mortality from untreated Legionella is high (15-50%), clinical clues may permit an earlier diagnosis leading to correct antimicrobial selection.

Objective: To determine the clinical features that differentiate (CAP) caused by Legionella versus S.pneumoniae; and to assess outcomes caused by both pathogens.

Methods: This was a retrospective study of 106 adult patients admitted to St. John from 1,1, 2013 to 10, 31, 2017 for pneumonia caused by either for Legionella or S. pneumoniae. Data collected included demographics, comorbidities, month of admission, laboratory values and vital signs. Outcomes included length of stay, need for intubation, septic shock, readmission within 30 days, and death.

Result: Legionella was more prevalent in men (OR 18.2, P <0.0001) and had lower mortality than S. pneumoniae (OR 7.4, P 0.02). The CWIC score was higher in patients with S. pneumoniae (OR 0.57, P 0.004). Diarrhea on presentation was a statistically significant factor for Legionella ( OR 3.7, P 0.05); however, hyponatremia was not (mean for legionella was 130.2 vs. 132 with P 0.5). The increase in incidence of Legionella in the summer months has been well established, however, the higher prevalence in men suggests the need for further study of why men may be at higher risk.
Nothing Works! Amegakaryocytic Thrombocytopenia Diagnosed After Refractory Idiopathic Thrombocytopenia Purpura

About 20-30% of idiopathic thrombocytopenia purpura (ITP) are diagnosed incidentally (usually as isolated thrombocytopenia). When symptoms are present, they usually including mucocutaneous bleeding, ecchymosis, and petechiae. After thorough review of the patient’s history, physical exam, and peripheral smear, ITP is a diagnosis of exclusion. We cared for a 39-year old healthy female whose incidental thrombocytopenia was suspected to be from ITP. On physical exam, petechiae was noted on her extremities. Peripheral smear revealed rare platelets without clumping or leukoerythroblastosis. After a couple of days on dexamethasone, she was started on IVIg due to the lack of response. This too was ineffective and she was given one dose of rituximab with minor improvements in platelets. Due to the refractoriness, she underwent CT-guided bone marrow aspiration biopsies showing a cellular marrow with progressive trilineage hematopoiesis and markedly decreased megakaryocytes. FISH studies were negative for any myelodysplastic syndromes. She was started on a thrombopoietin mimetic agent, eltrombopag, with improvements in platelet counts. Although our patient’s history and physical exam were stereotypical of ITP, her refractory nature prompted further evaluation leading us to the diagnosis of amegakaryocytic thrombocytopenia (AMT). Severe thrombocytopenia in the presence of decreased megakaryocytes with preservation of hematopoiesis in other lineages in the bone marrow characterizes this rare disorder. The proposed pathogenesis of AMT involves antibodies against thrombopoietin resulting in T-cell mediated megakaryocyte destruction. Clinically, it is difficult to distinguish ITP from AMT – the differentiating factor is bone marrow findings.
A Tricky Tempest – A Case of Refractory Thyroid Storm

Thyroid storm is a rare endocrine emergency. It manifests as life-threatening hypermetabolic multiorgan derangements, hyperpyrexia, and encephalopathy. Mortality rate is high if untreated. Early detection is crucial and relies on clinical suspicion before diagnostic tests. It is imperative to initiate appropriate early therapy for best prognosis.

A 53-year-old female with uncontrolled hypertension presented with progressive dyspnea, nausea (without emesis) and epigastric pain for 2-3 days. She had bilateral pitting edema of hands for which she took her mother’s furosemide. She developed acute respiratory failure requiring mechanical ventilation and ICU admission. She had persistent diaphoresis and febrile episodes. She had significantly low TSH and markedly elevated free T3/T4. Thyroid receptor antibody was positive for Graves’ Disease. Burch-Wartofsky scale for thyroid storm was 80-90 points (> 45 highly suggestive of thyroid storm). She was started on methimazole and hydrocortisone. T3/T4 normalized within 1 week with TSH lagging behind without any clinical improvements. In the interval, she developed mild transaminitis likely secondary to methimazole. Methimazole was discontinued and low-dose SSKI was started. Transaminitis resolved and clinical condition started to improve.

Treatment of thyroid storm involves blocking: 1) production/release of thyroid hormone, 2) peripheral conversion of T4 to T3, and 3) sympathetic activity. Thionamides, such as methimazole and PTU are used to block production. Methimazole is more commonly chosen due to the rarity of hepatotoxicity when compared to PTU. If thionamides result in no clinical improvement, the clinician should consider intolerance due to hepatotoxicity. In such cases, iodine therapy should be considered.
Hidden Hepatitis C

It is well known that hepatitis C can cause extra-hepatic complications such as autoimmune disorders, cryoglobulinemia, renal disease, and skin manifestations; the most common being essential mixed cryoglobulinemia. This presents a particular challenge in testing for hepatitis C because large amounts of cryoglobulins in the blood can lead to a false negative result with PCR testing. We present a 37 year old woman with a medical history significant for IV drug use, a recent diagnosis of psoriasis without laboratory testing, and hepatitis C exposure who presented with left thigh abscess, sensory changes to the left lower extremity, lower extremity palpable purpura, and arthritis of the left ankle. She was found to have a positive rheumatoid factor and C-reactive protein. Complement levels of C3 were normal, while C4 was at the lower limit of normal. Skin biopsy of her lower extremity lesions revealed leukoclastic vasculitis. Antibody testing for hepatitis C was positive while quantitative testing was negative. Work-up for other forms of vasculitis with ANCA, myeloperoxidase antibodies, and serine protease antibodies was negative. We were unable to finish our work up due to the patient leaving against medical advice. However, with known exposure to hepatitis C and a clinical picture suggestive of cryoglobulinemia we concluded her presentation was due to chronic hepatitis C infection.
One and a Half the Trouble: Ischemic Event Presenting as Dizziness, Transient Diplopia and an Oculomotor Nerve Palsy

This patient is a 68 year old female with a history of hypertension, end stage renal disease, diabetes mellitus and ischemic stroke. After a dialysis session, she reported double vision and dizziness with ambulation. Neurological examination was remarkable for weakness of abduction and adduction of the left eye and weakness of adduction of the right eye. CT of the head was notable for chronic ischemic changes and heavy calcifications. MRI of the brain revealed an acute lacunar infarct in the posterior aspect of the pons likely involving the medial longitudinal fasciculus. The involvement of the MLF is responsible for the patient’s ocular palsy known as One and a Half Syndrome. Her diplopia resolved but the cranial nerve III palsy persisted until discharge. She was discharged on dual antiplatelet therapy and a statin.

One and a Half Syndrome is a disorder of lateral gaze palsy looking toward the side of the lesion and internuclear ophthalmoplegia looking away. The syndrome is caused by a lesion in the paramedian pontine reticular formation and medial longitudinal fasciculus. The PRPF controls the ipsilateral abducens nucleus responsible for the lateral rectus. The nucleus also emits the MLF controlling the contralateral oculomotor subnucleus and the medial rectus. One and a half syndrome is exceedingly rare accounting for 10.8% of all reported cases of internuclear ophthalmoplegia. As in our case, the most common presenting symptoms are diplopia and dizziness. Prognosis is generally positive in One and a Half Syndrome with 49% having complete recovery at 6 months.
Think Beyond the Numbers: A Case of Lithium Toxicity with Normal Therapeutic Level

Introduction
Lithium is the mainstay treatment for bipolar affective disorder. Due to its narrow therapeutic range, careful monitoring is required to prevent toxicity that can manifest as neurological, gastrointestinal, and cardiac symptoms. However, reports of toxicity despite lithium levels within therapeutic range have been reported.

Case presentation
A 52-year-old woman with bipolar disorder presented to the hospital reporting a two-week history of nausea, vomiting, weakness, dizziness, blurry vision, gait instability, and tremor. She has been managed on lithium 300 mg twice daily for eleven years, with a dose increase to 600 mg twice daily three weeks before presentation. Laboratory findings revealed normal serum lithium level of 1.0 mmol/L (recommended maintenance range is 0.4-1.0 mmol/L). Thyroid function tests, electrolytes, and renal function were normal, and infectious workup was negative. Patient’s symptoms improved after holding lithium and providing intravenous hydration.

Discussion
Our case demonstrates an example of lithium toxicity despite therapeutic serum level. This diagnosis was confirmed by the presentation of typical symptoms of lithium toxicity, improvement of symptoms after holding lithium, and exclusion of other possible diagnoses. This supports the hypothesis that serum lithium levels do not reflect the intracellular concentration. Furthermore, this draws question to whether the therapeutic range may be too aggressive, considering toxicity reported at the upper end of the normal therapeutic range in some patients. Therefore, we suggest conservative initiation dose, slow dose increases, and close monitoring of symptoms in addition to lithium levels. We believe that more data should be studied in a systematic manner.
Pseudogout in a Knee Joint Post- Total Knee Replacement: A Diagnostic Dilemma!

Acute crystal arthropathy in a prosthetic joint is a rare occurrence, with very few confirmed cases of pseudogout. Nevertheless, it should be considered in the differential diagnosis of a patient presenting with a swollen, painful, and erythematous joint after total knee arthroplasty (TKA) to prevent unnecessary surgical debridement and prolonged courses of antibiotics.

An 81-year-old man with a history of bilateral TKA for osteoarthritis developed bilateral knee swelling, worsening pain and erythema accompanied with high-grade fevers and chills. Blood work showed an ESR of 107mm, CRP of 14mg/l, and a neutrophil predominant WBC of 9500 cells/mcL. X-rays showed no evidence of implant loosening. Right knee aspirate showed: WBC 13,510 cells/mcL with 65% neutrophils and calcium pyrophosphate dihydrate (CPPD) crystals. Left knee aspirate showed: WBC 18,050 cells/mcL with 68% neutrophils and no crystals. Synovial fluid and blood cultures returned negative. The patient received bilateral knee arthrotomy with irrigation, debridement and liner exchange, and a plan for four weeks of IV antibiotics. Ultimately, it was determined that his diagnosis was most consistent with pseudogout, his antibiotics were later discontinued, and he was referred to Rheumatology for the further management of this peculiar case.

This case demonstrates the diagnostic dilemma that exists in differentiating between crystal arthritis and septic arthritis in patients with a history of joint replacement and inflammatory joint symptoms. The diagnosis is supported by the presence of positively bi-refringent rhomboid shaped CPPD crystals in the synovial fluid. NSAIDS are the drugs of choice for its treatment with an excellent prognosis.
Hypothyroidism is a well-established side-effect of amiodarone, however severe hypothyroidism presenting with torsades is an underappreciated complication. We present a rare case of amiodarone-exacerbated primary hypothyroidism manifested by bradycardia-induced torsade de pointes.

69 y/o female with hx of hypothyroidism (on levothyroxine) and paroxysmal atrial fibrillation s/p cardioversion about a year ago (on amiodarone for rhythm control, digoxin and metoprolol for rate control) presented to ED with episode of syncope. On arrival her EKG showed junctional bradycardia at the rate of 18 bpm with QT of 744 ms. Initial labs revealed K: 3.6 mg/dl, Mg: 1.9 mg/dl, Trop neg x2, TSH: 76.59 uIU/ml (was 5 prior to amiodarone initiation), digoxin level: 2.3 ng/ml, Cr: 3.1 mg/dl. Patient had episodes of VT/Torsades requiring cardioversion. Transvenous pacemaker was placed and IV amiodarone was started.

She did not have any more episodes of VT however she continued to be pacemaker dependent even after digoxin level normalized therefore permanent pacemaker was placed. Amiodarone was discontinued prior to discharge due to concerns of reliable follow up for monitoring of the thyroid function.

In our case initially there appeared to be multiple reasons for bradycardia resulting in QT prolongation and subsequent Torsades. After repleting electrolytes and normalization of digoxin level, it became apparent that patient remained pacemaker dependent.

This highlights the role of hypothyroidism-induced bradycardia as a cause of life threatening arrhythmia and the impact of amiodarone in aggravating pre-existing thyroid disease. Our case highlights the importance of avoiding amiodarone in patients with preexisting thyroid dysfunction and if needed then close monitoring of the thyroid function is warranted.
Refractory Ascites in a Patient with Severe Preeclampsia

Introduction:
Preeclampsia refers to the new onset of hypertension and proteinuria or hypertension and end-organ dysfunction with or without proteinuria after 20 weeks of gestation. We present a case of refractory ascites in a pregnant woman with severe preeclampsia.

Case:
A 32-year-old female at 27 weeks gestation came to the hospital for worsening dyspnea and abdominal distention. There is significant medical history of preeclampsia. Blood pressure was 168/102 mmHg and urine protein/creatinine ratio 0.6. Her serum biochemistry was normal, albumin: 2.6 g/dL. She underwent emergency cesarean section and 4 liters of ascitic fluid was drained from peritoneal cavity. Post-surgery patient re-accumulated ascitic fluid which was drained. Ascitic fluid analysis negative for infection and cytology was negative for malignancy. Calculated serum ascitic albumin gradient (SAAG) was >1.1 suggestive of transudative fluid. Liver cirrhosis, portal vein pathology and cardiomyopathy were excluded by ultrasonography. She was treated with albumin, lasix and spironolactone which resolved the ascites and prevented re-accumulation.

Conclusion:
Ascites as a manifestation of preeclampsia is very rare and is associated with worse outcomes. The mechanism of ascites in preeclampsia is a combination of endothelial dysfunction, vascular hyper-permeability and low serum albumin. We managed our patient with delivery, paracentesis, diuretics and albumin replacement. It is prudent for physicians to keep a high index of suspicion in patients with preeclampsia and early intensive management to prevent fetal and maternal complications.
Catastrophic Antiphospholipid Syndrome Complicated by Intracranial Hemorrhage

Catastrophic antiphospholipid syndrome (CAPS), characterized by diffuse thrombotic microangiopathy and rapidly progressing multiorgan failure, represents less than 1% of all antiphospholipid syndrome (APS). As opposed to large vessel thrombosis seen in APS, diffuse small vessel thrombosis dominates the clinical picture of CAPS.

A 38-year-old female with the past medical history of APS, spontaneous abortions and recent lower extremity deep vein thrombosis on Enoxaparin presented with nausea, vomiting, and fever for ten days. Labs were significant for thrombocytopenia, chronic anemia, acute kidney injury, and elevated transaminases and troponins. Given her symptoms, CT abdomen/pelvis was done which was unremarkable. A broad infectious workup was non-revealing as well. On day 3, she developed confusion, left arm weakness and seizures, and was transferred to the ICU. CT head revealed right parenchymal and subarachnoid hemorrhages. Hematology testing showed elevated PT, aPTT and D-dimer. Disseminated intravascular coagulation and thrombotic thrombocytopenic purpura were ruled out the help of with normal fibrinogen and negative schistocyte. Anticardiolipin and Beta-2-glycoproteins antibodies were noted to be elevated. With a suspicion of CAPS, high dose IV Steroids and IV Ig were started. The patient gradually improved and was started on Rituximab.

CAPS has a high mortality of 50%. Although thrombosis is the major manifestation, bleeding can occur in 10% of patients. Definite diagnosis of CAPS requires biopsy proven small vessel thrombosis in addition to positive APL antibodies and evidence of acute multisystem involvement. However, treatment should not be delayed where there is high clinical suspicion as early treatment can be life-saving.
Hepatitis A is a common viral infection with a benign course but in rare cases can progress to acute liver failure. It usually presents with abdominal pain, nausea, vomiting, diarrhea, jaundice, anorexia or asymptptomatically but it can also present atypically with relapsing hepatitis and prolonged cholestasis. In addition, extrahepatic manifestations have been reported including urticarial and maculopapular rash, acute kidney injury, autoimmune hemolytic anemia, aplastic anemia, acute pancreatitis, mononeuritis, reactive arthritis, glomerulonephritis, cryoglobulinemia, Guillain-Barre syndrome and pleural or pericardial effusion. A rare manifestation of hepatitis A is acute myocarditis. We report a case of a young female who presented with “flu-like symptoms” and was found to have severe elevation of liver enzymes due to acute hepatitis A infection. On her 3rd day of admission the patient developed chest pain and nonspecific electrocardiographic changes. Her troponins rose to 16.4 ng/mL, and a transthoracic echocardiogram revealed global hypokinesis and a depressed ejection fraction at 30%. A CT angiography showed no evidence of significant coronary artery disease. The patient was managed supportively, and symptoms and laboratory findings slowly improved over the next 7 days. Her chest pain resolved and follow up echocardiogram showed improved ejection fraction to 45%.
Not so Cryptic! An Unusual Case of Cryptogenic Organizing Pneumonia Diagnosed as Primary Biliary Cholangitis

INTRODUCTION: Lung–gut axis is well established with inflammatory bowel disorders causing diverse pulmonary manifestations including cryptogenic organizing pneumonia (COP). We present an unusual case of biopsy-proven COP, who was eventually diagnosed with primary biliary cholangitis (PBC) after three years of unremitting symptomatology.

CASE PRESENTATION: A 61-year-old female with a history of non-specific arthralgia was evaluated three years ago for dyspnea and easy fatigability. A CT scan of the chest showed multiple consolidations in the right lung. She was treated with oral antibiotics for pneumonia. Subsequent follow-up CT scans demonstrated shifting consolidations bilaterally with a peripheral predilection. A bronchoscopy with biopsy and culture was negative. The patient underwent surgical lung biopsy, and a diagnosis of organizing pneumonia was made. An extensive autoimmune workup was negative. The patient continued to have extreme fatigue and was hospitalized 2.5 years into presentation with anemia and febrile illness, requiring a blood transfusion. Another elaborate autoimmune workup was negative except for an elevated IgM and C-reactive protein. A repeat High-resolution CT-scan showed improvement in lung opacities. However, she continued to have extreme fatigue and serial liver function studies over the next 6 months revealed elevation of alkaline phosphatase. An anti-mitochondrial antibody was strongly positive, and a diagnosis of PBC was made.

CONCLUSIONS: Previous reports of PBC associated lung disease are seen with an established diagnosis of PBC. It is exceptional to see such a long latency, and this case illustrates the need for heightened clinical suspicion for recognition of hepato-pulmonary autoimmune axis.
Lymphomatoid Granulomatosis; A Rare Differential Diagnosis of Granulomatosis with Polyangitis

77 years old male with PMH of HTN, CLL, recurrent sinusitis presented with chief complaint of cough, fatigue, fever, weight loss and shortness breath for 6 weeks. He completed multiple courses of antibiotics and prednisone as outpatient with no improvement in his symptoms. He was hypoxic and febrile on presentation to ER. Infectious work up including blood and sputum cultures, fungal serologies and quantiferon were negative. CT chest showed innumerable pulmonary nodules. On day 3 of hospitalization, he developed purple skin nodules. Skin biopsy showed granulomatous inflammation in dermis involving vessels and adnexal structures. A diagnosis of Granulomatosis with Polyangiitis (GPA) was strongly considered. However, autoimmune serologies including ANA, ANCA, anti-MPO, anti-PR3, RF, CCP were negative. He continued to have high grade fevers despite being on broad spectrum antibiotics. He eventually underwent VATS with wedge biopsy which revealed diffuse large B cell lymphoma with features of lymphomatoid granulomatosis along with positive EBV staining. He was started on R-CHOP regimen for treatment of his lymphoma.

Lymphomatoid Granulomatosis is a rare lymphoproliferative disorder in the family of EBV associated B cell lymphoma. Histologically it shows areas of transmural lymphoid infiltrates of vessels and focal necrosis. Clinically it presents very similarly to GPA with constitutional symptoms, skin rash/nodules, pulmonary and ENT involvement. Lymphomatoid Granulomatosis is treated as diffuse large B cell lymphoma and generally has poor prognosis compared to GPA. Biopsy should be obtained as soon as possible in these cases to distinguish between the two as treatment and prognosis are very different.
A Quality Improvement Project to Increase Compliance with Diabetes Measures in an Academic Outpatient Setting

Rationale: Medical residents are first-line primary care providers for patients with diabetes mellitus. American Diabetes Association (ADA) sets annual guidelines on preventative measures that help delay the onset of more severe complications of diabetes. However, previous studies describe suboptimal diabetic preventative care in resident clinics.

Aims: The aim of our quality improvement (QI) project is to improve diabetic care, with HbA1c value as primary outcome and other core measures as secondary outcomes.

Methods: Our resident clinic at Beaumont Hospital, Royal Oak consists of 76 residents divided in 8 teams. In November 2016, baseline data on core ADA measures was obtained on over 500 diabetic patients. A root cause analysis was conducted. 5 teams developed a QI intervention plan to improve their diabetes care and 3 teams served as controls. In November 2017, post-intervention data was collected.

Results: The change in HbA1c value was +0.09 in intervention and +0.322 in control groups (p=0.174). With secondary outcomes, the change in outcome measures were as follows: eye examinations (+5% in intervention vs. -7% in control, p<0.01), foot examinations (+13% vs. +5%, p=0.09), lipid panel (+7% vs. -5%, p<0.01), microalbumin (+4% vs. +1%, p=0.03), and HbA1c (+8% vs. +5%, p=0.24).

Conclusions: We noticed a paradoxical slight increase in HbA1c levels post-PDSA cycle. However, there were significant improvements in the secondary outcome measures in the QI intervention groups. Further conclusions will be determined from final data analysis of this study.
Extrahepatic Pancreatobiliary Obstruction Causing Recurrent Symptoms - Just Another Diverticulum?

A case of rare entity called: Lemmel Syndrome is presented, where clinical picture of pancreaticobiliary obstruction is present, in the absence of underlying choledocholithiasis. In addition we present case images of endoscopy, CT scan and fluoroscopy confirming an obstructive process caused by duodenal diverticula. This is a 90-year-old woman who presented with clinical picture concerning for cholangitis and acute pancreatitis. She has past medical history of recurrent pancreatitis, without identified underlying cause. Imaging revealed incidental presence of duodenal diverticula without presence of biliary stones. Similar findings were noted on endoscopic retrograde cholangiopancreatography (ERCP). Sphincterotomy was performed with improvement of symptoms. The biliary obstruction resulting in cholangitis and pancreatic injury was caused by obstructive process from the adjacent duodenal diverticula, consistent with Lemmel Syndrome.

Lemmel syndrome was first described in 1934 by Lemmel and is defined by obstructive jaundice in the absence of gallstones due to a periampullary duodenal diverticulum. Biliary obstruction in Lemmel Syndrome can be caused by different mechanisms. Current proposed etiologies include: direct mechanical irritation of periampullary diverticula that causes chronic inflammation and fibrosis of the papilla, sphincter of Oddi dysfunction or direct mechanical compression caused by duodenal diverticula. Conservative management is recommended in periampullary diverticula that are asymptomatic and do not cause laboratory abnormalities. ERCP with sphincterotomy is usually considered as an initial approach in symptomatic periampullary diverticula. Surgical excision is another option if there is lack improvement and clinical deterioration, however it may be associated with significant mortality.
Hemophagocytic Lymphohistiocytosis (HLH), a Rare yet Potentially Fatal Disorder in Adults

HLH was thought of as a pediatric disorder, here we present a case of HLH in a 71 year old female. This was a 71 year old Caucasian female with a 3-month history of a low grade fever and weight loss who presented for dyspnea. The patient was found to be pancytopenic. Initial workup for anemia revealed elevated LDH and a ferritin of 2655 μg/l. Chest CT PE revealed bilateral hilar lymphadenopathy, innumerable lung nodules and rib fractures bilaterally. The patient subsequently underwent a bone marrow biopsy which revealed hemophagocytosis with CD25 expression, raising the concern for HLH. Further workup revealed an elevated triglyceride and IL-2 receptor levels. The patient met the diagnostic criteria for HLH defined by HLH-2004 trial and the diagnosis was established. She was immediately started on steroids and etoposide. Unfortunately, she developed fulminant fungemia, DIC and multi-organ failure and eventually expired. Autopsy was performed and revealed a necrotic infiltrate involving bone marrow, suspicious for necrotic malignant neoplasm of hematolymphoid origin and hemophagocytosis. Interestingly, the patient’s mother and sister also died of fever of unknown origin and her 36-year-old daughter had also been experiencing fevers. This case illustrates the importance of prompt diagnosis and treatment of this rapidly progressive, life-threatening, hyper-inflammatory syndrome which is rising in incidence in adults. Patients with a genetic predisposition are more likely to relapse on treatment and have a high risk of a lethal outcome and thus haematopoietic stem cell transplantation becomes of utmost importance to these patients and affected family members.
Double the Trouble: Dual Paraneoplastic Syndromes in a Patient with Small Cell Lung Carcinoma

Paraneoplastic syndromes occur in patients with certain malignancies, however the occurrence of two or more simultaneously is rare. We report the case of a patient with small cell lung cancer (SCLC) presenting with the syndrome of inappropriate antidiuretic hormone (SIADH) and Cushing’s syndrome due to ectopic ACTH production. A 58-year-old woman with a history of rheumatoid arthritis and 40 pack year smoking history presented with dyspnea and cough for a few weeks duration, with 15lbs unintentional weight loss. Chest x-ray revealed a collapsed left lower lobe for which she was directed to the emergency department. Upon presentation, her blood pressure was elevated at 177/81 mmHg otherwise stable vitals. Physical exam revealed diminished breath sounds at the left base and negative otherwise. Labs revealed a sodium of 125mmol/L, potassium 2.9mmol/L, chloride 87mmol/L, serum osmolality 269mOsmol/kg, urine osmolality 706mOsmol/kg and urine sodium of 158 mmol/L. A diagnosis of SIADH was established, requiring use of hypertonic 3% saline, with the eventual initiation of Tolvaptan given persistent hyponatremia. She presented with Cushing’s syndrome as well, manifesting with severe new onset hypertension and hypokalemia. Work up revealed failure to suppress cortisol levels with high dose dexamethasone testing, confirming the diagnosis of ectopic ACTH production. She was started on Ketoconazole. Further imaging revealed a 6.3 cm left infrahilar mass with subcarinal adenopathy and multiple hepatic lesions. A liver biopsy revealed pathology consistent with metastatic small cell lung carcinoma. After initiation of chemotherapy, her labs normalized, all medications were discontinued, and imaging revealed improvement in her malignancy.
Sjogrens Syndrome Presenting as Type 1 RTA

Renal Tubular Acidosis (RTA) is a relatively uncommon, and often unrecognized disorder. Many times, RTA can be the only presenting sign of a systemic process, specifically autoimmune diseases. Distal (Type 1) RTA is characterized by impaired distal acidification in the cortical collecting duct of the nephron, and it is seen in up to 25% of patients with Sjogrens Syndrome. A large proportion of these patients also have polyclonal hypergammaglobulinemia. The pathophysiology behind this association is unknown.

A 68-year-old female without significant past medical history presented with three months of fatigue, malaise, and xerostomia. Physical exam revealed multiple dental caries, but was otherwise unremarkable. Chemistry panel revealed marked hypokalemia (2.1 mmol/L), a non-anion gap metabolic acidosis, an elevated total protein (8.5 g/dL) with an elevated globulin level (4.2 g/dL) and hypergammaglobulinemia (2.2 g/dL). ECG showed flattened T waves and a pseudo-prolonged QT interval. Autoimmune workup was significant for gross elevations in Sjogren SS-A and SS-B antibody levels. Potassium supplementation resulted in partial resolution of her symptoms, and she was discharged home with outpatient rheumatology follow up. This case illustrates the importance of a thorough evaluation of the etiology of laboratory abnormalities that may seem "idiopathic" at first glance. This diagnosis is critical because if left untreated, severe electrolyte imbalances can occur and precipitate fatal complications. Distal RTA is a rare disorder; however, perhaps this is due to underdiagnosis. In a patient who presents with unexplained RTA or hypergammaglobulinemia, autoimmune diseases (specifically Sjogren’s Syndrome) should be considered.
Delayed Development of Complex Regional Pain Syndrome Following a Non-Hemiplegic Stroke

Complex regional pain syndrome (CRPS) presents with pain, swelling, limited mobility, vasomotor instability, skin changes with patchy demineralization of the distal extremity. In stroke patients, it usually occurs in the upper extremity in the hemiplegic side in about 4-6 weeks. We present a case of CPRS which occurred 3 years after a non-hemiplegic stroke.

51-year-old female with history of right-sided stroke with no residual deficit, known stable right frontal meningioma presented with left arm pain and swelling for a week without any history of trauma. Examination revealed extreme tenderness, swelling with limited range of motion of wrist and arm without erythema or warmth. X-rays of upper extremity were normal. She had no improvement despite empiric IV antibiotics for possible cellulitis. CT head and MRI brain revealed stable meningioma with no recurrent stroke. Extensive rheumatologic and infectious workup including, ESR, CRP, uric acid, RA, CCP, ANA, HIV, VZV, were normal. Finally, triple phase bone scan revealed asymmetric flow of radiotracer with delayed uptake in distal joints on left wrist; consistent with CRPS. She was treated with naproxen, gabapentin and physical and occupational therapy with resolution of symptoms after 5 months.

Cases of CRPS have been well documented following hemiplegic stroke. However, to the best of our knowledge, there has been no report of CRPS following a stroke without hemiplegia. In this population, if symptoms are consistent, workup for CRPS may aid in early diagnosis and initiation of appropriate therapy while minimizing unnecessary treatments like antibiotics or opioids.
Unmantle that Lymphoma

Introduction: Mantle Cell Lymphoma (MCL) is one of 70 subtypes of non-hodgkins lymphoma (NHL). Incidence of NHL in USA is approximately 71,000 per year and 6% of those are MCL. It is most common in Caucasian men in their 60s. Typically the disease is indolent and diagnosed at advance stages.

Case Description: 79-year-old female who presented to with weakness, fatigue and diffuse abdominal pain worsening for 2 weeks. CT of the abdomen showed enlarged spleen extending into left pelvis. She also had hyperkalemia, anemia, thrombocytopenia and elevated INR. She required multiple transfusions and hyperkalemia was corrected. Bone marrow biopsy performed was non-diagnostic for any malignancy or myeloproliferative disorder. BCR-ABL was negative therefore CML was ruled out. Patient eventually had a splenectomy after multiple platelet and PRBC transfusions since it was thought to be consumptive coagulopathy. Pathology report showed features of mantle cell lymphoma (translocation of 11;14) which had transformed into diffuse large B cell lymphoma. Plan was to start patient on first-line therapy with R-CHOP.

Conclusion: MCL is an aggressive disease that occurs due to translocation of 11;14 which results in over-expression of Cyclin D1. There has been extraordinary progress in treatment of MCL in recent years and survival rate has increased to around 5-7 years however there is no “cure” found yet. Patients have progression after that and blastoid version of the disease usually worsens with chemotherapy. Further studies are needed to determine markers that may help with early diagnosis and treatment and lead to better outcomes.
A Curious Case of the Chilblains

Chilblains or Perniosis is a vasospastic cutaneous eruption that occurs on exposure to cold. It is an inflammatory condition, characterized by violaceous painful blisters. We present a case of a patient who underwent an extensive workup finally to realize that it was a detailed history that led to the diagnosis.

An 84-year-old healthy male presented to the emergency room with sudden onset of multiple painful blisters on both hands and neck. On exam, he was afebrile; vitals within normal limits; the blisters were firm, fluid-filled; some broke open, discharging clear, non-foul smelling fluid. Infectious disease was consulted; the working diagnosis was Bullous Pemphigoid vs Toxic/Infectious bullous lesions. He was started on Vancomycin for MRSA skin infection. Extensive laboratory workup ordered including IgE, IgG, Complement, ANA, CRP, and ESR; all of which were normal. Skin biopsy was planned. Additional history revealed the patient had shoveled snow for 30 minutes wearing thin gloves. Superficial wound cultures remained negative, with no new lesions seen. A diagnosis of frostbite induced blistering skin disease made, patient educated to protect from the cold and he was discharged home.

A detailed history is always the cornerstone in getting to a diagnosis. Despite extensive evaluation, the crucial piece of history clinched the diagnosis. The phenomenon of cold-induced cutaneous lesion can easily be mistaken for a myriad of diseases causing patients to undergo unnecessary and painful diagnostic procedures. As physicians, we must always remember that medical history is the first diagnostic tool and often the most important one.
"Bug on the Back" - A Rare Case of Salmonella Osteomyelitis of the Vertebra in an Otherwise Healthy Young Adult

Introduction:
Salmonella is a rare causative agent of osteomyelitis about 0.45%, is predominantly seen in patients with hemoglobinopathies, and is typically seen in diaphysis of long bones.

Case Report:
A 24 year old Caucasian farmer with no significant risk factors, presented with a 2 month history of back pain. He went to chiropractor and PCP multiple times with no relief with massages or multiple pain medication. He does livestock farming and has almost 200 chickens and 7 pigs at home. He cooks and consumes the pigs but never the chickens. Denies fever, chills, night sweats, paralysis, bowel, bladder incontinence, trauma, weight loss, recent travel, diarrhea or constipation. He had some cough but no phlegm production. XR- lumbar spine showed osteoarthritis at L3-L4 level. CT chest showed irregularity and destruction of end plate at T9-T10 level, representing underlying osteomyelitis. Triple phase bone scan revealed intense uptake at T9 vertebra. MRI revealed discitis/osteomyelitis of T8 and T9 vertebral bodies. All fungal and TB workup, HIV testing, blood cultures, CD4 count and bronchoscopy with BAL culture was all negative. CT guided aspiration showed serosanguinous fluid collection and cultures grew salmonella enteritidis. It was sensitive to Rocephin and patient was discharged with 6 weeks antibiotics.

Conclusion:
The diagnosis of native vertebral osteomyelitis is challenging and delayed most of the time to an average of 2-4 months as idiopathic neck pain and back pain are very common symptoms and could initially be misdiagnosed. Cultures are the key to diagnosis.
Opioid Prescribing for Chronic Pain and Screening for Mental Health Co-morbidity in an Academic Internal Medicine Clinic

Patients who have an undertreated or untreated mental illness tend to have co-occurring pain conditions. An academic internal medicine program implemented an ambulatory curriculum to assess whether the educational content on addressing mental health (MH) co-morbidity in chronic pain (and opioid use) would translate into clinical practice. After conducting a needs assessment, patients taking any opioid were referred for enrollment in an IRB-approved 3-month feasibility study. The goal is to determine whether the curriculum makes a difference in MH outcomes in patients with chronic pain.

The chart review (n=300) showed that 44.2% patients with a co-morbid MH condition had opioids prescribed as compared to 28.2% having a non-opioid prescription (p=0.007). After logistic regression adjusting for age, gender, and ethnicity, patients with MH conditions are 2.2 times more likely to have an opioid prescribed than patients without mental health conditions (OR=2.15, p=0.019). For the feasibility arm (beginning June 2018), an interim analysis of patients enrolled-to-date revealed that 29% met criteria for MH co-morbidity with an average PHQ-9 of 17 and GAD-7 of 12. There is a high proportion of opioids prescribed, which is more prevalent in patients with co-morbid MH conditions. Data analysis will occur in early October to ascertain whether mental health screening scores improve with interleaving lessons on treating chronic pain. This study may highlight that improved education in residency for safely and effectively managing chronic pain and addressing MH co-morbidity are needed to help curtail a tragic opioid epidemic.
Persistent Watery Diarrhea in Presence of Pseudomembranes—Not Always Clostridium difficile!

Introduction:
Collagenous colitis (CC), a subtype of microscopic colitis, is an inflammatory gastrointestinal condition, where chronic, intermittent, watery diarrhea is the leading symptom. Alternatively, pseudomembranous colitis (PC) is almost exclusively associated with infectious etiology—Clostridium difficile infection (CDI). Herein, we present an interesting case of pseudomembranous CC in absence of CDI.

Case description:
41-years-old female presented to hospital with persistent diarrhea and diffuse abdominal-pain for two-weeks. Diarrhea was described as watery, non-bloody, occurring every 30mins-1hour. Patient was treated for CDI 4-months ago with multiple-rounds of antibiotics due to reoccurring watery diarrhea. Upon admission, lab-workup revealed mild leukocytosis. CT abdomen noted fluid levels in colon, consistent with gastroenteritis. Initial diagnosis was presumed to be CDI due to recent-history, hence, oral vancomycin was started empirically. However, stool-studies were negative for CDI among other infectious etiologies. Colonoscopy was done which revealed yellowish-plaques (pseudo-membranes) throughout the colon with mild-inflammation of underlying-mucosa. However, biopsies revealed benign-colonic-mucosa with surface-ulceration, marked collagen deposition of lamina-propria—consistent with severe CC. Patient was started on budesonide therapy, which significantly improved her symptoms.

Discussion:
Our patient was diagnosed with rare-variant of CC called “pseudomembranous collagenous colitis”—a relatively new entity with only a handful of reported cases. Endoscopically, CC has normal-appearing colonic-mucosa, with minimal changes reported in upto 20% cases. Nevertheless, formation of pseudo-membranes in otherwise typical CC is extremely uncommon. Hence, clinicians need to be aware that prior-history of CDI and presence of pseudo-membranes does not preclude diagnosis of CC, as it completely changes the face of management.
Unusual Duodenal Perforation in Behçet’s Syndrome

Case Description:
A 56-year-old-female - with past medical history significant for Behcet syndrome- presented to the ED encephalopathic. The patient decompensated on the 2nd day of admission with respiratory failure and septic shock secondary to suspected aspiration pneumonia. An abdominal computed tomography was done and revealed pneumoperitoneum. Emergency laparotomy was performed for suspected perforation of the GI tract and patient was found to have a large anterior perforated duodenal ulcer with active bleeding.

Discussion:
Gastrointestinal involvement in Behcet’s disease (BD) is characterized by ulceration which can involve any area from the oral cavity to anal canal. The most common sites of involvement in the gastrointestinal system in Behcet's disease are the terminal ileum, cecum, and ascending colon secondary to large vessel vasculitis i.e. mesenteric vessels, not in the duodenal region like this patient. The gastrointestinal bleeding in patients with Behcet’s disease is usually from colonic ulcers or ruptured esophageal varices. Our patient had bleeding from a duodenal ulcer which is quite rare and seldom previously reported in Behcet’s disease.

Significance:
The number of operations reported of intestinal involvement in Behçet’s disease has increased rapidly, but perforated intestinal Behçet’s disease is still rarely reported. Colonic perforation is an unusual complication of BD and may occur in any part of the colon, however, it most commonly affects the ileocecal region. Recognition and awareness of this syndrome and its complications are critical to the institution of appropriate therapy and timely surgical intervention, as it is associated with significant morbidity and mortality.
Anti-NMDAR Encephalitis: A Case Report

The N-methyl-D-aspartate receptor (NMDAR) is a kind of glutamate receptor that contributes to memory and learning through the signal transmission in the central nervous system, antibodies against this receptor is associated with limbic encephalitis. Anti-NMDAR encephalitis initially described in 2007 as autoimmune/paraneoplastic encephalitis. We present a case of anti-NMDAR encephalitis in a young Caucasian woman. A 35 years old female with past medical history of depression, bipolar disorder presented with acute onset confusion. She didn’t have any fever, chills, headache, neck stiffness, rash, focal neurologic deficit, recent travel, or camping. Patient’s initial lab showed mild leukocytosis, lymphocytic pleocytosis and mild elevation of protein in the CSF. Head CT and MRI were normal. Lyme serology and HSV-PCR were negative. TSH, UDS and Alcohol levels were WNL, EEG didn’t reveal any seizure activity in the brain. She was treated with acyclovir empirically which was discontinued with the negative lab results. Patient’s encephalopathy continued with fluctuating psychomotor agitation, insomnia, impaired memory, and behavioral changes. Autoimmune panel including ANA, ESR, thyroid ab, ENA and NMDA were sent and was started on high dose IV steroid 500mg TID empirically, symptoms continued to improve on steroids. ANA and NMDA serology came positive, she was discharged on steroid and Azathioprine.

Symptoms of anti-NMDAR encephalitis are very nonspecific which often result in misdiagnosis and/or underdiagnoses of this entity. We present this case with a review of literature to enhance awareness about the disease in the medical community for a prompt diagnosis and treatment of the disease to decrease mortality and morbidity.
Minimal Change Disease as a Presentation for Hodgkin’s Lymphoma

A number of glomerulopathies, tubulopathies and vascular renal diseases can signal the presence of an underlying cancer, including, nephrotic changes as paraneoplastic syndromes in lymphoid malignancies. In particular, the association of nephrotic syndrome (NS) with Hodgkin's Lymphoma (HL) is rare, however we present a case where minimal change disease preceded the diagnosis of HL.

A 21 year old female presented to the ER with a 3 week history of nausea, vomiting, epigastric pain and diffuse anasarca. Initial testing documented significant proteinuria prompting a CT-guided kidney biopsy, which showed minimal change disease. She was initiated on prednisone and diuretics, which improved her symptoms initially however, she returned to the emergency department in 3 months with dyspnea and abdominal pain and persistent proteinuria. Imaging showed mediastinal and supraclavicular lymphadenopathy, biopsy proven to be Hodkin’s Lymphoma, nodular sclerosing type. She is currently on ABVD therapy which has reversed proteinuria, suggesting association and raising question for early diagnostic workup for solid organ malignancy. The occurrence of nephrotic changes as manifestation of HL, suggests that some immunological abnormalities may play a role in the pathogenesis of this association, and may be the initial presentation for HL. The etiology remains elusive however, studies have suggested that autoimmune disorders of T-lymphocyte function and natural killer cell deficiency may lead to nephropathy in HL. The course of NS does not always run parallel to that of the lymphoma, however it should be a differential for persistent proteinuria, as timely treatment has shown to reverse the nephropathy.
Nitrous Oxide Abuse a Big NO-NO!

Intro: Nitrous Oxide abuse can have varying side effects. One such debilitating adverse reaction is inactivation of vitamin B12 causing functional subacute combined degeneration.

Case: 22 year old female presenting after a fall 1 month ago with complaints of diffuse numbness, weakness and electric shock like sensations through her lower extremities. Also reports 2 episodes of urinary incontinence, 20 lb weight loss in 2 months. On examination she had bilateral C7-C8 and L3-L4 dermatomal numbness, reduced vibration sense, increased tone in distal lower extremities, and DTRs slightly brisk throughout (except ankles). She admitted to recreational nitrous oxide use. CBC, CMP, B12, Folate levels unremarkable. MMA elevated at 0.49 and Homocysteine elevated at 30. MRI showed longitudinal hyper-intense lesion in anterior cervical and upper thoracic spinal cord. She was treated with parenteral vitamin B12 replacement while admitted and discharged to subacute rehab.

Discussion: Nitrous oxide is a less commonly known recreational drug, accessible in grocery stores “Whippit” containers. A debilitating side effect can be functional subacute combined degeneration. This occurs as nitrous oxide irreversibly oxidizes the cobalt ion of cobalamin preventing methylcobalamin from acting as a coenzyme in the production of methionine and S-adenosylmethionine. This is necessary for methylation of myelin sheath phospholipids resulting in decreased myelin formation. This is a functional cyanocobalamin deficiency, hence traditional methods of diagnosis are unreliable and a high degree of suspicion is necessary. Once nitrous oxide abuse is stopped and cyanocobalamin is replaced enzyme activity recovers in 3-4 days, cyanocobalamin is administered till clinical resolution.
The Elusive Differential: Non-Small Cell Cancer of the Lung with Elevated BhCG Levels in a Young Female Patient

Diagnosing malignancy is becoming more unique in both clinical and biological manifestation. Here, we present a case of over expression of BhCG in the setting of adenocarcinoma of the lung.

41-year-old female with a history tubal ligation presented with abdominal pain and weight loss. CT abdomen/pelvis demonstrated multiple nodular lesions in the right lung with pleural thickening, loculated pleural effusions, and soft tissue mass at T12. She was found to have an elevated BhCG on presentation (86 mIU/mL). Due to her elevated BhCG and abdominal pain, choriocarcinoma was suspected. In the past, she had multiple pleural effusions with many negative diagnostic thoracentesis. The patient then underwent a VATS with pleural biopsy; which was negative. Subsequently the paraspinal mass at T12 was biopsied. Pathology was most consistent with poorly differentiated TTF-1 negative adenocarcinoma of the lung.

In the above case presentation, the primary malignancy was adenocarcinoma of the lung with expression of BhCG. This marker is measured in the setting of germ cell tumors and choriocarcinoma for staging, prognosis and response to treatment. Normally tumor markers that are expressed in non-small cell carcinoma of the lung include ALK gene, cytokeratin fragment 21-1 or EGFR. Further research is needed to establish a correlation between BhCG and non-small cell lung cancer. At which time, BhCG could be used as a tumor marker to assist in early diagnosis, response to treatment of adenocarcinoma of the lung; as well as a prognostic indicator.
Late Onset Polyglandular Autoimmune Syndrome

Introduction:
Type 3 PAS is characterized by a combination of autoimmune thyroiditis, type 1 diabetes and other autoimmune processes.

Care presentation:
A 93-year old male with CAD, CKD, HTN presented with a fall. Patient was found to be in DKA with blood glucose of 805 and beta-hydroxybutyrate of 6.04. Patient has no known history of endocrinopathy. His blood sugar was normal three weeks prior when he had an URI. Diagnosis of latent autoimmune diabetes of adults was supported with elevated GAD antibodies and low C-peptide of 0.8. Further work-up revealed TSH of 15.71, free T4 of 0.6 and a positive Thyroperoxidase antibodies suggesting autoimmune thyroiditis. Patient did have episode of hypotension. However, morning cortisol level was not suggesting of adrenal insufficiency. Patient was diagnosed with type 3 PAS.

Discussion:
Environmental exposure or intrinsic changes in the gene were hypothesized as the triggers for PAS. Patient did have a URI which could trigger PAS if he is genetically susceptible. However, type 3 PAS typically happens in the fourth decade of life. A diagnosis at age 93 was considered a very late onset for LADA and type 3 PAS. It was not clear if the patient has type 2 PAS. Cortisol was collected after possible steroid use for hypotension workup, which can cause falsely elevated cortisol. A 21-hydroxylase antibody was collected for further workup of adrenal insufficiency, which could classify him as type 2 PAS.

Conclusion:
When a patient develops one autoimmune endocrinopathy, it is important to screen for other endocrinopathy.
Hydralazine Induced Diffuse Alveolar Hemorrhage in Drug Induced Lupus

Hydralazine, commonly used to treat hypertension, is most notorious for its' several variable and debilitating side effects including drug induced lupus erythematosus (DILE). Not only is DILE associated with Hydralazine, but there have been some case reports linking Hydralazine to ANCA-associated glomerulonephritis and vasculitis. The concern is that this vasculitis can cause a serious complication known as Diffuse Alveolar Hemorrhage (DAH) which has a very high mortality rate. We report a case of Hydralazine induced vasculitis presenting as DAH in a patient with known systemic lupus erythematosus (SLE) and recent diagnosis of lupus nephritis. She was treated with hemodialysis (HD) and plasmapheresis as well as a course of pulse-dose steroids. Immunosuppressive therapy was not initiated during her hospital course due to pneumonia and herpes epiglottitis. She was found to be myeloperoxidase (MPO) antineutrophil cytoplasmic antibody (ANCA) and anti-histone antibody positive. We concluded that Hydralazine induced MPO-ANCA vasculitis can rarely present with DAH in a patient who had been taking Hydralazine for years and requires prompt cessation of the medication. Treatment modalities are variable and involve hemodialysis, plasmapheresis, and even immunosuppressive therapy which may be needed for complete resolution of symptoms.
**Large Saddle Embolism in Coronary Vasculature Leads to Flash Pulmonary Edema - A Rare Case Report**

**Introduction**
Coronary artery embolism (CE) is a rare cause of acute myocardial infarction (AMI), with an increased risk of death. Although flash pulmonary edema (FPE) may present following acute coronary ischemia, it has not been reported in the setting of an AMI from a large saddle embolism. To our knowledge, this is the first reported case in literature.

**Case Report**
An 86-year-old Caucasian female with hypothyroid and dementia presented to ED in acute respiratory distress. Vital signs demonstrated tachycardia and hypoxemia. Physical examination revealed bilateral crackles. A 12-lead electrocardiogram showed sinus tachycardia with ST-segment depressions in leads V2 to V5. Labs revealed a troponin-I of 0.38 ng/mL which increased to 64.50 ng/mL. Treatment was instituted for FPE secondary to NSTEMI. Transthoracic echocardiography showed moderate mitral valve regurgitation and stenosis. Subsequent left heart catheterization revealed a large saddle embolism involving two vessels of non-dominant circumflex coronary artery without associated stenosis. Unfortunately, she sustained cardiopulmonary arrests which lead to her death.

**Discussion**
CE is associated with poor long-term survival and increased risk of death. Diagnosis is corroborated by angiographic findings and thromboembolic risk factors in the presence of an AMI. The underlying cause of coronary artery embolism in our patient may be attributed to mitral valvulopathy. Emergent percutaneous intervention may necessitate both thrombus aspiration and stent placement.

**Conclusion**
The presentation of an AMI from a saddle CE is rare; however it can prove fatal as in our case report. Early recognition and appropriate therapy should be instituted early to prevent further decline.
Encapsulated Fat Necrosis Associated with Precipitous Weight Loss

Encapsulated fat necrosis (EFN) was first described by Przyjemski et al in 1977. Lesions commonly present on lower extremities and can be solitary or multiple (up to ten). Though most are mobile, they can be fixed and range from 1 to 35 mm in diameter; however, lesions as large as 18 cm have been reported. Lesions are typically caused by trauma that compromises blood supply, causing necrosis of adipocytes. Around 40% of patients do not recall a precipitating event nevertheless. Subsequent fibrous encapsulation hinders resorption and isolates the mobile mass from surrounding tissue.

A 50-year-old female presented with numerous small subcutaneous masses on her thighs and abdomen. These lesions became noticeable after an unexpected 60 pound weight loss. The patient’s medical history was significant for autoimmune hepatitis, Hashimoto’s thyroiditis, Sjogren’s syndrome, fibromyalgia, and polycystic ovaries. Clinical examination revealed hundreds of pebble-like subcutaneous masses on her anterior thighs and right lower abdomen. These lesions were freely mobile, non-tender, and firm on palpation. Gross examination showed three pieces of yellowish spherical soft tissue, 3 to 8 mm in diameter. Microscopic examination revealed circumscribed adipose tissue with necrosis and micro-cyst formation, surrounded by a fibrous capsule. Lipo-membranous changes or significant calcification were not identified.

This patient differs from the previously reported cases in several ways: the vast number of lesions present, the relation to precipitous weight loss, and finally the association with several autoimmune disorders. Interestingly, isolated autoimmune conditions, such as scleroderma, have been reported in relation to encapsulated fat necrosis.
Ectopic Hamartoma Tyyoma with Epithelial Atypia (Rare and Unique)

Introduction: Ectopic hamartomatous thyoma is a benign soft tissue tumor usually occurring in the lower neck in middle-aged adults. It is extremely rare, with <80 reported cases. Although the term thyoma is used, the entity is not related to mediastinal thymomas but likely represents a benign mixed tumor with features of branchial anlage.

Case discussion: A 63-year-old man had an enlarging soft tissue mass near the left clavicle, associated with mild pain for 2 months. The swelling measured ~1 x 2 cm in the greatest dimension, soft without any overlying erythema. Imaging showed degenerative changes in the acromioclavicular joint without an acute process or focal mass. The mass was surgically excised, measuring 3.0 x 2.8 x 2 cm. Histopathological analysis showed biphasic elements, with both spindle cells and acinar components weakly positive for cytokeratins (AE1/AE2) and CD-99, with some staining for EMA mainly in the acinar components and CK7 staining in both components. Stains for desmin and S100 protein were negative, with the latter showing nonspecific adluminal staining in the acinar component. Vimentin stain was positive within the spindle cell component. Some areas of epithelial atypia were noted, so clinical interval follow-up was recommended. The patient had not yet had any new recurrences or swellings.

Conclusion: This rare case of ectopic hamartoma with epithelial atypia adds to the literature on the tumor's clinical and pathological characteristics. Given its benign course and distinctive histopathological identity, it is important to recognize the tumor, and surgical excision is the treatment of choice.
Cerebral Nocardiosis: A Rare Cause of Brain Abscess - How Easy Is It to Diagnose?

Introduction:
Nocardia is aerobic, partially acid-fast, gram-positive rod, causes mild to severe pulmonary infections. Rarely, causes cerebral abscess. The standard of diagnosis for brain abscess is staining and culture. Our case signifies the need to use molecular assays as standard for diagnosis.

Case summary:
72-year-old female with history of endometrial carcinoma and diabetes mellitus presented with headaches and right sided weakness. MRI brain showed multiple bilateral ring enhancing lesions. Cerebral abscess was diagnosed. Patient started on I.V vancomycin and I.V meropenem. Gram stain showed beaded gram-positive rods. AFB-stain was negative. Cultures showed actinomyces. Antibiotics were switched to I.V penicillin G. Specimens sent to state-lab for confirmation. There was no improvement clinically. Few days later state-lab results showed nocardia species. AFB-stain was positive. I.V trimethoprim was started, patient condition deteriorated and she expired.

Discussion:
Risk factors for cerebral nocardiosis are immunosuppressive drugs, HIV, DM, chronic infections and cancers. Nocardia abscess is often misdiagnosed as pyogenic abscess/primary/secondary tumor. Here it was misdiagnosed as actinomyces. Actinomyces is acid-fast negative. Nocardia is usually acid-fast positive to a varying degree, depending upon the mycolic acid content of the cell. Initial acid-fast was negative, which led to wrong diagnosis. Early and accurate diagnosis are crucial because of high mortality (20%-66%) and increasing resistance pattern. With molecular assays like 16S ribosomal DNA polymerase chain reaction, we can diagnose nocardia early and accurately.

Conclusion:
Use of molecular diagnostic assays might have prevented the adverse outcome and they should be made standard for diagnosis.
VZV Angitis in an Immunocompetent Patient Treated with Steroids

VZV-angitis is a rare manifestation of latent VZV-activation. VZV- related vasculopathy is extremely rare in immunocompetent patients.

A 68 YO M with PMH of systolic CHF, ESRD, PVD presented with shingles in the right V1 distribution which progressed to pre-septal cellulitis and opthalmitis with marked swelling of the eyelid and non-reactive pupil on the right side with blurred vision. Culture grew MRSA and he was started on IV Acyclovir, Vancomycin and oral prednisone. A day later he became acutely encephalopathic and had an acute generalized seizure. LP and CSF analysis was positive for VZV by PCR. CTA showed calcifications in Right ICA but no inflammatory changes, MRI brain was performed which showed areas consistent with possible acute infarction vs vascular inflammation in Right centrum semiovale and posterior parietal cortex which were consistent with VZV-angitis. Risks/benefits of using IV methylprednisolone 1G for 3 days were weighed and then first dose of this high strength Steroid regimen was given and the following day he was awake, alert and more oriented. After 3 days of methylprednisolone he was transitioned to Prednisone taper. During hospitalization his mentation wax and wane but he was ultimately discharged to LTACH in a stable condition.

In the case above, in addition to standard treatment with acyclovir, high dose steroids were given and it rapidly improved the clinical condition of the patient. Role of steroids is unclear but the rapid improvement in clinical condition of this patient can be attributed to anti-inflammatory properties of steroids.
Gastric Kaposi Sarcoma Discovered in an Immunocompromised Renal Transplant Patient

Introduction - Kaposi sarcoma (KS) is an angioproliferative tumor caused by human herpesvirus 8. GI tract lesions are the most common extracutaneous location and can even occur in the absence of cutaneous lesions. However, these tumors are often clinically silent. Only with tumor growth can the patient present with abdominal pain, nausea, vomiting, anemia, digestive bleeding, mechanical obstruction, intussusception, and/or perforation. The purpose of this case study is to describe a case of an incidentally discovered gastric KS to help illustrate clinical presentation and endoscopic description of this rare finding.

Case description - 61 year old male with extensive PMH including ESRD post renal transplant presented from the renal transplant clinic due to worsening swelling and lethargy despite the recent addition of furosemide. Labs revealed hemoglobin at 6.8. Due to anemia and need for 1 unit of blood, GI was consulted and he underwent EGD, which showed a large friable mass. Biopsy of the gastric mass was positive for Kaposi Sarcoma. Extensive opportunistic infection workup was negative. CT chest was performed which showed pulmonary nodules, mediastinal and axillary lymphadenopathy. He underwent biopsy of the right axillary lymph node, which was also positive for Kaposi Sarcoma.

Discussion – Considering the GI tract is the most common extracutaneous site involved in KS, endoscopists should be alert for this diagnosis. Endoscopic appearance of KS lesions includes ulcerated, flat, polypoid/nodular, and volcano-like lesions. In immunosuppression-associated KS, withdrawal or reduction of immunosuppressants is advised. Endoscopic examinations could be useful for staging and evaluating therapy outcomes.
Colchichine Toxicity: A Case of Cancer Deception

Medication-induced injury of the gastrointestinal (GI) tract is increasingly common but generally under-recognized. Colchicine is an antimitotic alkaloid that causes variable injury. Colchicine toxicity can produce diagnostic morphologic features in gastrointestinal mucosal biopsies mimicking high-grade dysplasia (HGD) and adenocarcinoma.

A 58 years old male with a past medical history of colon adenoma, myocardial infarction, coronary artery disease status post stents and gastroesophageal reflux disease underwent coronary artery bypass graft surgery (CABG) and was hospitalized for 11 days. Intraoperatively, there was an incidental finding of suspicious lymph nodes and PET scan was ordered. Post-op recovery was complicated with significant pleuritic chest discomfort. Diagnosis was consistent with postpericardiotomy syndrome. He was started on colchicine 0.6mg BID on day 6 of surgery. PET scan was positive for pancreatic head mass with lymphadenopathy. GI was consulted and he underwent upper EGD with EUS on day 10. He had irregular GE junction and biopsies were performed. Patient was discharged on day 11 with GI follow up. Histopathology was consistent with intramucosal carcinoma. He underwent endoscopic mucosal resection. Pathology report stated that esophageal mucosa had abundant ring mitoses which was consistent with colchicine toxicity.

Current literature shows colchicine effects mostly in gout patients. Further date and studies are needed to recognize these changes. It is interesting to note these findings in a patient with short course of colchicine therapy. Multidisciplinary team meetings should also be held with expert pathologist and endoscopists to decide on best course of action.
Cytoreduction Methods to Prevent the Progression of Hyperleukocytosis in a Newly Diagnosed CML Patient

Hyperleukocytosis is a laboratory abnormality that is variably defined as a total white blood cell count greater than 50x10^3 or 100x10^3. When symptomatic it is referred to as leukostasis, a fatal complication mainly affecting the central nervous system and lungs. Incidence of hyperleukocytosis and its complications vary depending on the type of leukemia and patient affected. Cytoreduction is the mainstay treatment for this phenomena, it can be achieved by 2 methods: chemical; hydroxyurea treatment or induction chemotherapy treatment, and physical; leukapheresis.

31 year old male with no significant past medical history presented to his family physician with the complaints of fatigue, night sweats, decreased appetite, and a 60lb weight loss that started 6 months prior to presentation. CBC showed: WBC 356.7x10^3, Absolute Neutrophils 181.91%, Absolute Basophils 39.42%, Platelets 659x10^3, Hemoglobin 8.7g/dL. He was admitted to the hospital. Bone marrow biopsy was performed and showed CML-Chronic phase. While awaiting the results of the BMB, cytoreduction was immediately initiated using Hydroxyurea and Leukapheresis to prevent progression to Leukostasis. The patient underwent 7 days of cytoreduction as inpatient, at discharge his WBC was down to 37.3x10^3. The patient followed up with Oncology and was started on Dasatinib when his WBC reached <15x10^3.

Hyperleukocytosis can progress to affect patients with vast complications if not treated promptly. Lowering the WBC with the above stated methods is crucial for survival, in addition to supportive measures to prevent and/or treat complications such as anemia and Tumor Lysis Syndrome.
The Membrane That Can Lead to Stroke

Introduction:
Out of the many sources of cardio-embolic strokes, Cor triatriatum sinister is an uncommon one. Echocardiography is an invaluable tool in detecting such an anomaly.

Case description:
A 78 years old female with no past medical history came to the ED complaining of a sudden onset of facial drooping and weakness over her right side. She denied history of embolic events or similar symptoms. vitals were WNL. On physical exam she had significant weakness of the Rt side. Subsequently, stroke alert was called and CT head showed no acute abnormality. MRI revealed a small focus of lacunar infarction involving the left thalamus with diffusion restriction consistent non-hemorrhagic stroke. Patient was started on low dose aspirin and efforts were direct to locate the potential source of embolization. A bilateral carotid ultrasound showed less than 50 percent occlusion. A TTE showed a linear echo density membrane like was noticed in the left atrium in the apical four chamber view just above left atrial appendage (LAA) consistent with Cor triatriatum sinister.

Conclusion:
In a nutshell, this case shades the light on a rare congenital anomaly however can lead to devastating consequences. Cor triatriatum sinister is an uncommon cause of stroke and very few cases have been previously reported. The underlying mechanism of stroke in Cor triatriatum sinister is the low flow state generated between the membrane like fibromuscular tissue and LAA. It is worth mentioning that Cor triatriatum sinister should be differentiated from supra mitral membrane.
Autoimmune Hepatitis: An Uncommon Cause of Acute Hepatitis

Introduction: Autoimmune hepatitis (AIH) is a rare cause of acute hepatitis with a reported incidence of about 0.9-2 per 100,000 cases per year. We report a case of acute hepatitis due to AIH.

Case Summary: A 40-year-old female presenting with complaints of nausea, vomiting, dull right upper abdominal discomfort, yellowish discoloration of her eyes and skin for 10 days. Physical examination revealed conjunctival icterus, yellow skin, with mild tenderness at the right upper epigastric region. Labs were remarkable for elevated AST 574 U/L, ALT 1,021 U/L, total bilirubin 7.4 mg/dl, direct bilirubin 6.6 mg/dl, ALP 415 U/L and Gamma-GT 520 U/L. Urine was positive for bilirubin and urobilinogen. Abdominal CT and US abdomen did not show evidence of acute cholecystitis. Infectious hepatitis panel was negative. Serum ceruloplasmin, ferritin, and alpha-antitrypsin were within normal range. However, anti-nuclear, anti-smooth muscle antibodies titers and Total serum IgG were elevated (1:320, 1:20 and 1,780 mg/dl respectively). Liver biopsy showed acute on chronic inflammation with cellular necrosis and ballooning of hepatocytes. Prednisone 60 mg daily was started, following which there was remarkable improvement in symptoms and progressive decline in serum AST, ALT, total and direct bilirubin. On discharge, she was started on azathioprine for maintenance.

Discussion: AIH is an uncommon but treatable cause of hepatitis. It shows excellent response to steroids, immunosuppressant’s and a majority of patients achieve remission. This case highlights the importance of having a high index of suspicion for AIH in patients with acute hepatitis whose infectious workup come back negative.
Hemodynamically Stable Pulmonary Embolism with Right Ventricular Thrombi: A Management Dilemma

Introduction: Right ventricular thrombus (RVT) is an uncommon complication which occurs in approximately 4% of patients with pulmonary embolism (PE). It carries higher 30-day mortality risk compared to acute PE patients without RVT. The management guidelines for PE with right ventricular thrombus are not well established and has been a matter of debate.

Case: 84 years old female presented to the emergency department with complaints of shortness of breath and chest pain. Her vitals were remarkable for tachycardia and tachypnea; however, her blood pressure was within normal limits. Due to AKI Ventilation-perfusion scan was performed which showed a high probability of pulmonary embolism with multiple mismatched perfusion/ventilation defects. An echocardiogram showed RVT without evidence of right heart strain. The next day she had an episode of vomiting following which her O2 saturation dropped to 70's. An emergently obtained echocardiogram demonstrated disappearance of the previously observed RVT suggesting its migration to cause a massive recurrent PE along with right heart strain with severely elevated right ventricular systolic pressure. An emergent Computed Tomography Angiography of the chest was immediately ordered to determine the location of the PE to perform catheter-directed thrombolysis. However, she lost her pulse; there was no ROSC with 35 minutes of CPR she was pronounced dead.

Conclusion: Even though the current guidelines recommend standard anticoagulation without reperfusion therapy for hemodynamically stable PE with right heart thrombus, the outcome of our case, however, suggests otherwise.
IgA Nephropathy, Underrecognized Common Cause of Glomerulonephritis and CKD

Introduction: IgA Nephropathy is the most common cause of glomerulonephritis world-wide. It can lead up to ESRD in 20-30%. We describe a case of a young African American Male in North America who presented with CKD and poorly controlled hypertension who was treated with combination of steroid and ACEI.

Case Description: A 28 year old African American Male who presented with 3 weeks of progressively worsening frontal headache. He presented with blood pressure of 209/121 as well as eGFR of 40. The patient denies history of gross hematuria. The patient’s blood pressure was difficult to control and ultimately required 3 maximum antihypertensive therapy. Urine protein/creatinine ratio was 2.72g/day. Diagnosis of IgA Nephropathy was confirmed by renal biopsy; which showed IgA nephropathy with proliferative and sclerosing features (oxford score: M1, E1, S1, T2, C1) as well as findings consistent with malignant hypertension. The patient was started on prednisone oral 100mg once followed by 50mg once daily as well as lisinopril. Lisinopril was up-titrated to 20mg until the patient’s urine protein was undetectable by 4 month follow up. Once the proteinuria was controlled, the prednisone was tapered weekly until prednisone 10mg once daily. Renal function was minimally improved.

Discussion: Despite the high prevalence of IgA Nephropathy world-wide, there is large discrepancy in the prevalence between different regions; particularly affecting Asia more than North America or Europe. Due to the strong regional and ethnic bias in IgAN, it still proves to be a difficult disease to diagnose and treat in North America.
Enoxaparin Induced Iliopsoas Hematoma and Subsequent Bilateral Hydronephrosis and Severe Acute Renal Failure

Introduction: The definition of ilio-psoas hematoma (IPH) refers to a traumatic or spontaneous retroperitoneal blood collection involving the ilio-psoas muscle. IPH incidence for patients on anticoagulation has been reported as 0.1 to 0.6 %. IPH can rarely lead to bilateral hydronephrosis and acute kidney injury (AKI).

Case description: A 60 year-old woman with a history of deep vein thrombosis (DVT) and pulmonary embolism, presented with acute left lower extremity swelling, pain and redness. Venous duplex ultrasound showed acute DVT of the left popliteal and peroneal veins, and so she was started on Enoxaparin. On day 6 she started to have abdominal pain, tachycardia, fever, leukocytosis and she became lethargic. Her hemoglobin dropped dramatically: 12 to 4 g/dL and she developed AKI. Abdominal CT showed a left psoas hematoma extending to the iliopsoas region causing left kidney hydronephrosis and hydroureter; a repeat CT showed small infracolic region mass causing right kidney hydronephrosis. She required bilateral ureteral stents. Her mentation, along with her kidney function improved significantly afterwards. She did not need any hemodialysis sessions and the hematoma resolved after stopping anticoagulation. She was discharged with instructions for outpatient follow up with urology for stent management.

Conclusions: Spontaneous IPH in patients on anticoagulant is uncommon; however it can be associated with multiple devastating complications including; pain, nerve palsy, muscle dysfunction and hydronephrosis. Approaches to therapy vary from conservative, interventional and open surgery, depending on the cause and volume of the haematoma, timing of diagnosis, degree of neurological impairment and haemodynamic stability.
Indications and Complications of Midlines

Background: Peripheral venous access is challenging in patients with poor vasculature and those who need IV access for longer durations. Midlines have gained popularity in some hospitals as they can be kept in place for up to two weeks. However, there is no established evidence of benefits and risks.

Methods: We performed a retrospective chart review to assess the indications and prevalence of Midlines' complications in our institution. Descriptive statistics were conducted using SPSS version 22.

Results: A total of 440 Midlines were placed for 6 months, with 81% being ordered due to poor vascular access. Most lines were placed from first attempt (78%). The lines were inserted in the right upper extremity in 55% cases. The duration of the lines had a mean of 3 days. Seven patients developed deep vein thrombosis (DVT) and 5 patients had bacteremia when Midline was in place, while 9 had a DVT event and 1 had bacteremia after line removal. Six patients had a line site infection. 88% of Midlines had no complications with the most common complications being leaking and edema. The lines clogged in 2 patients.

Discussion: Our data shows that Midlines exhibit benefits, but they are not without risk. There were only a very few clogged lines, however this seems to be due to under-documentation. In the end, Midlines would likely be preferred over central lines due to their wide range of benefits. However, their limitations and complications should be considered in order to best care for diverse patient populations.
When the Good Becomes the Bad: A Case of Lactobacillus Rhamnosus Septicemia Unrelated to Probiotic Use

Introduction: Lactobacilli are gram positive, facultative anaerobic bacilli that live as commensals in the gastrointestinal and genitourinary tracts. They are promoted as probiotics. Although they are considered non-pathogenic, there are case reports of Lactobacillus-related serious infections in immunosuppressed hosts - mostly associated with probiotics use. We present a case of Lactobacillus septicemia unrelated to probiotic use.

Case Description: A 64-year old male presented with complaints of jaundice and an unintentional 40 pounds weight loss. Physical exam revealed a cachectic male with whole-body jaundice and scleral icterus with epigastric tenderness. The patient and family denied any probiotic use. Laboratory tests suggested an obstructive jaundice. CT-abdomen revealed a mass in the head of the pancreas with intra and extra hepatic biliary dilatation. He underwent ERCP followed by percutaneous biliary drainage. His post-operative course was further complicated by sepsis due to fungemia. While on Micafungin (and having completed a course of Meropenem and Vancomycin, empirically for cholangitis), his sepsis remained unresolved. Repeat blood cultures grew Lactobacillus Rhamnosus (4 sets) with resistance to Vancomycin (MIC1:256). In view of multiple comorbidities and recent antibiotic use, he was treated with Daptomycin and repeat cultures after 72 hours were negative with improvement in his sepsis.

Conclusions: It was hypothesized that the recent instrumentation along with the tumor eroding the vessels and ductal system of the pancreas resulted in the translocation of Lactobacillus into the bloodstream. This case recognizes Lactobacillus as a potential pathogen and that blood cultures with susceptibility testing are important in guiding treatment.
Hyperammonemic Encephalopathy, a Rare Presentation of Valproic Acid Toxicity in the Absence of Liver Impairment

Introduction: Hyperammonemia can occur due to a broad range of etiologies and is commonly found in the setting of liver impairment. However, the absence of liver pathology presents a diagnostic challenge. Valproic acid (Valproate) toxicity has been associated with hyperammonemic encephalopathy. It is a rare occurrence and can be overlooked.

Cases:
1) A 28-year-old woman presented with 3-days of altered mentation, elevated ammonia level and normal liver function. The patient’s mentation improved with Lactulose therapy and was subsequently discharged. She had a readmission shortly thereafter with the same presentation. Patient was on Valproate for epilepsy treatment. Valproate levels were elevated, but discontinuing Valproate removed the inciting cause of encephalopathy. She improved with no further hospitalizations.
2) A 57-year-old woman presented with 2-hours of altered mentation after intentional overdose. Patient had elevated ammonia and Valproate levels without hepatic impairment. Discontinuation of Valproate and L-carnitine therapy improved patient’s encephalopathy.

Discussion: These cases represent a rare complication of Valproate use. Its effect on urea cycle is thought to be one possible explanation for hyperammonemia. Management includes discontinuation of the drug and symptomatic treatment with Lactulose. We also highlight L-carnitine as proposed treatment for such toxicity. It is important for clinicians to be mindful of both hepatic and non-hepatic causes of hyperammonemia. These patients remind us to act with benevolence, utilizing a stepwise approach to diagnosis and treatment in order to improve patient outcomes. In retrospect, in our first case such consideration would have prompted earlier discontinuation of Valproate, potentially avoiding hospital readmission.
Diabetic Myonecrosis: An Unusual yet Debilitating Complication of Uncontrolled Diabetes

Introduction: Diabetic myonecrosis is a rare complication of long-standing, poorly controlled diabetes mellitus (DM), with around 200 published cases. It usually occurs in patients with microvascular complications.

Case description: A 43-year-old man presented with 4 week history of worsening left thigh pain. His past medical history included DM (glycated hemoglobin level 17.6%), diabetic neuropathy and microalbuminuria. He had no family history of muscle disorders and was not on statin. He had presented 4 weeks ago for the same complaint. At that time X-Ray of the left femur showed no fracture. Lower extremity arterial and venous Doppler ultrasound imaging were normal. He was discharged with analgesics. On this admission the left thigh was firm, erythematous and tender. CT-scan of left lower extremity showed hyperenhancing vastus medialis suggesting myonecrosis. TSH was normal and urine drug screen was negative. Patient improved with conservative management, analgesics, optimized glycemic control and low-dose aspirin.

Conclusions: Although self-limiting, diabetic myonecrosis often closely mimics cellulitis and deep venous thrombosis. Increased awareness of this rare complication can prevent delayed or missed diagnosis leading to increased morbidity, inappropriate treatment and unnecessary investigations. Diabetic myonecrosis should be considered in patients presenting with acute muscle pain and in whom other causes of thigh pain such as bacterial infection, immune myositis, venous and arterial thrombosis are ruled out. MRI is the test of choice and muscle biopsy need to be considered for atypical cases. Pain can persist over days to weeks. Treatment consists of glycemic control, antiplatelet therapy and bed rest.
A Case of Head Trauma Triggering ACEI Induced Angioedema

Introduction: In the United States, Angiotensin Converting Enzyme Inhibitors (ACEIs) are the main culprit of drug-induced angioedema. Patients most commonly present with swelling of the face or tongue and rarely with intestinal angioedema. A few cases in recent literature have identified a possible link between local trauma caused by spinal surgery or head and facial injuries and ACEIs induced angioedema. We present a case of ACEIs related angioedema in a patient with local head trauma.

Case Description: A 66 y/o male with history of hypertension and on Lisinopril for 10 years presented with tongue swelling after a head injury. The patient took 40 mg of Lisinopril early morning and a couple hours later slipped and hit his head. Immediately, the patient started experiencing tongue swelling. CT maxillofacial showed significant tongue enlargement. ENT performed a laryngoscopy which was negative for any masses. The patient received intravenous dexamethasone and diphenhydramine. Swelling resolved 2 days after discontinuing Lisinopril.

Conclusions: A literature review identified a few reported cases of neck surgery and facial trauma as potential causes of angioedema in patients on ACEIs. Some studies indicate that local inflammation causes the levels of bradykinin to increase; that, in combination with ACEIs induced inhibition of bradykinin breakdown causes an excessive level of bradykinin which then causes tissue edema. In our patient’s case, we think the head trauma from the fall initiated the sequence of events leading to angioedema. Besides systemic steroids, the ultimate treatment is stopping Lisinopril.
Cryptococcal Meningitis Presenting as Diplopia

Introduction: Cryptococcal meningitis is a potentially fatal fungal opportunistic infection affecting immunocompromised patients. It affects 957,900 people worldwide every year. Most common symptoms are fever, malaise, headache, photophobia, and vomiting. Diplopia, as one of the presenting features, is not as commonly seen as these other symptoms.

Case Description: A 37 year old female with known history of HIV, not on HAART, was admitted with complaints of double vision, headache, photophobia and sinus congestion. On examination the patient had left sided deviation on right eye lateral gaze. Magnetic resonance venography ruled out cavernous sinus thrombosis. Lumbar puncture was done with CSF analysis which was positive for Cryptococcal meningitis. The patient received Liposomal Ampho-B 5mg /kg IV q 24 hrs + 5-Flucytosine 25mg/kg PO q 6 hrs for 14 days, followed by 6-8 weeks of high dose Fluconazole, and secondary prophylaxis. Her symptoms improved after the initiation of antifungals.

Conclusion: Diplopia is not one of the common presenting features of Cryptococcal meningitis. This was an interesting case due to its rare presentation with this symptom. Complications arise due to failure to recognize symptoms of raised intracranial pressure and start treatment in a timely manner. The mechanisms behind raised intracranial pressure are not clearly defined although clogging of arachnoid villi by Cryptococcus and subsequent reduction in resorption of CSF is thought to be one of the reasons.
Biopsy Showing Plasma Cell is Not Always a Plasma Cell Neoplasm

Introduction: Plasmablastic lymphoma (PBL) is a difficult to diagnose aggressive B cell lymphoma. The diagnosis is challenging because tumor cells share resemblance to plasmablastic myeloma. It is seen most commonly in patients with HIV, immunosuppression and organ transplant.

Clinical Case: A 59 year old African American female, with recurrent ventral hernia in the past, presented with acute on chronic abdominal pain. CT abdomen described possible incarcerated ventral hernia. At the time of emergent laparotomy, a soft anterior abdominal wall soft mass was found. Initial pathology report was consistent with plasma cell myeloma (PCM). Multiple myeloma workup was pursued. Serum protein electrophoresis was polyclonal. Bone marrow biopsy showed 8% plasma cells. Bone survey showed multiple lucency in left femoral shaft. She was initially treated for PCM, however repeat pathology report from tertiary center showed atypical cells positive for MUM1, CD138, CMYC and negative for CD20, CD3 which was consistent with PBL. Thereby therapy was changed to cover PBL with Etoposide/Phosphate/Prednisone/Vincristine Sulfate/Cyclophosphamide/Doxorubicin Hydrochloride (EPOCH).

Conclusions: Our case is unique in that it was initially diagnosed as PCM but instead turned out to be PBL. PBL remains a diagnostic challenge due its similarities with PCM, but the treatments for the two conditions are different. In the process of B cells becoming plasma cells, centrocytes transform to plasmablasts before becoming mature plasma cells, which is thought to be the pathogenesis for this similarity. As clinicians it is imperative to investigate further patients with suspected PCM but with atypical immunohistochemistry.
Tearing Down the Gallows: May-Thurner's Syndrome and Duration of Anticoagulation

May-Thurner's syndrome, also known as iliac vein compression syndrome is an anatomical and pathological condition associated with compression of venous outflow as a result of obstruction in the iliocaval venous territory. The level of compression may vary leading to symptoms ranging from being completely asymptomatic to complete venous occlusion with or without thrombosis. It is particularly very common in young women.

A 61 year old male with past medical history significant for hypertension and chronic kidney disease presented to the Emergency department with acute left leg swelling and pain. All labs were within normal limits. Bilateral lower extremity Doppler showed extensive acute DVT. CT venography showed extensive thrombus with narrowing of the left common iliac vein caused by compression by the right and left common iliac arteries consistent with May Thurner's syndrome. Patient was treated with catheter directed thrombolytic therapy with stent placement in the iliac vein. Patient was discharged on apixaban for 3 months.

Limited data exists on the duration of anticoagulation for patients with iliac compression vein syndromes. This begs the question: Are DVTs in patient with MTS without other risk factors classified as a provoked or unprovoked DVT? What should the duration of anticoagulation be? The current VTE guidelines do not address the question on duration of therapy in patients with Iliac vein compression syndromes. This case demonstrates the importance of further research into this not-so-common but significant cause of DVT.
A Silver Lining

An 86 year old man with a long history of hypertension and osteoarthritis presented to nephrology clinic for elevated creatinine. Examination revealed a blue discoloration to the proximal aspect of his fingernails bilaterally. He reported the fingernail discoloration occurred 15 years prior following supra-therapeutic dosing of colloid silver. Creatinine was 1.93 mg/dl and estimated glomerular filtration rate of 33 ml/min, his urine protein to creatinine ratio was 0.2 g/g. Renal biopsy was performed for diagnostic purposes as well as to provide information for his daughter. She continued to ingest colloidal silver supplements despite recommendations she discontinue it to avoid renal toxicity. Renal biopsy showed global glomerulosclerosis and dark granular glomerular deposits consistent with renal vascular disease and silver toxicity.

Argyria is a well described complication of silver toxicity with the characteristic grey-blue discoloration of the skin and eyes. Deposition of silver into tissues, including the kidney, has been seen on both biopsy and autopsy. Association of silver exposure and increased creatinine has been noted in case reports and a small cross sectional study on workers manufacturing precious metals, however, causation was not established due to confounding cadmium exposure. Animal models suggest renal toxicity following silver exposure.

For our patient, his long standing hypertension along with his colloidal silver ingestion likely contributed to his kidney disease. These biopsy results were enough to convince his daughter to stop ingesting colloidal silver products giving a silver lining to this case after all.
A Case of Acne Induced Heartache

Propionobacterium acnes is an unusual cause of infectious endocarditis and is associated with prosthetic valves. It is difficult to diagnose because often it is a contaminant and when not, requires prolonged incubation. Here we examine the case of a 55 year old man who was diagnosed with propionibacterium acnes, prosthetic-valve endocarditis. 55 year-old man with past medical history significant for aortic valve insufficiency with aortic valve repair in 2003, AVR re-do with a bovine valve in 2013 after valve leaflet rupture presented with the chief concern of fevers, chills, sweats, and abdominal pain of four months. Concurrent to this, patient had odontogenic issues requiring tooth extraction. Four months prior to admission, patient was prescribed several courses of antibiotics and had one negative set of blood cultures. Patient was admitted to hospital for intermittent fevers, abdominal pain, and leukocytosis. On admission 3 sets of blood cultures were drawn. Patient was diagnosed with acute cholecystitis. Cholecystostomy tube was placed as temporizing measure and on 9th day culture results grew Propionibacterium acnes. Transesophageal-echo revealed a 1.6x1.1 cm vegetation on the prosthetic valve that was causing LVOT obstruction. Consequently, cardiothoracic surgery replaced aortic valve. Cholecystectomy, drain removal done as outpatient. This case illustrates the need for increased suspicion of infectious endocarditis in a patient with history of prosthetic valve that presents with infectious symptoms (intermittent fevers); an adjunct lesson accentuates the importance of prolonged incubation periods of blood cultures in evaluating for causative organism in a patient with prosthetic valve.
Ipilumumab and Nivolumab Associated Tenosynovitis in Metastatic Renal Cell Cancer (RCC)

Introduction: Immune checkpoint inhibitors, which increase antitumor immunity by blocking intrinsic down-regulators, are currently at the forefront of clinical studies. Unfortunately, by increasing the activity of the immune system, immune checkpoint blockade can lead to immune-related adverse events (IRAEs). This case illustrates a rheumatic IRAE.

Case Description: A 67 year-old-male with RCC presented with severe enterocolitis causing diarrhea after his third cycle of Ipilumumab and Nivolumab. Hospital course was complicated by bilateral ankle swelling and pain, left greater than right. Ultrasound of the left ankle revealed effusion and bursitis. Antinuclear antigen, rheumatoid factor, and anti-CCP antibodies were negative. He was diagnosed with immune-related tenosynovitis and treated successfully with steroids, resulting in resolution of both tenosynovitis and enterocolitis.

Discussion: Musculoskeletal IRAEs are rare, and the pathophysiology is still unclear. They are reported in 6-8% of patients treated with a single immune agent, and 10% in patients treated with dual immunotherapy. No defined guidelines currently exist for grading severity and treatment of rheumatic IRAEs, however most have been reported to be steroid-sensitive, and resolved within 6-12 weeks with prolonged courses of high-dose steroids. Physicians should be aware of IRAEs and recommended treatment. Clinical significance: As the use of immunotherapy grows, internists will be encountering more frequent cases of IRAEs. Prompt recognition of IRAEs will lead to appropriate and timely treatment.
Unique Recurrence of Oropharyngeal Cancer

Secondary cardiac tumors are common, but squamous cell carcinoma (SCC) is typically not the one to metastasize to the heart. 59-year-old male with past medical history of sarcoidosis and stage-IV tongue SCC treated with adjuvant radiation and surgery, in remission for a year presented to the hospital with symptoms concerning for superior vena cava (SVC) syndrome. A recent follow up contrast CT-chest showed stable mediastinal, hilar and neck lymphadenopathy. Those were biopsied earlier, and pathology was consistent with sarcoidosis. Admission contrast CT-chest showed SVC occlusion with 3.8x3.7 cm filling defect at right atrial appendage. It was difficult to characterize this as a thrombus versus a mass. Inpatient PET-CT showed an increased uptake at right atrium as well as hypermetabolic mediastinal and hilar adenopathy. Upon discussion with nuclear radiologist, lymphadenopathy uptake could be due to either sarcoidosis or metastasis. Both vascular and thoracic surgery services recommended against biopsies of lymphadenopathy given highly engorged collateral veins and high risk of bleeding complications. Pulmonary and oncology were also consulted. IR-guided biopsy revealed only thrombus. A repeat biopsy under guidance of intravascular ultrasound helped identifying the location of the echogenic mass. Results were consistent with metastatic SCC. Patient was referred to the multidisciplinary cancer clinic for further management. Meanwhile, he was started on anticoagulation with some improvement in his symptoms within 48 hours of treatment. This case sheds light on the importance of multidisciplinary approach to complicated cases and the important role of personal communications with specialists to expedite reaching diagnostic and therapeutic plans.
Impact of Auditing Blinded Discharge Summaries on Hospitalists' Performance

Introduction:
In 2007, a systematic review recognized deficits in discharge summaries with missing test results (33-63%), treatment or hospital course (7-22%), discharge medications (2-40%), and results pending at discharge (65%). In 2009, ACP's policy statement suggested a standard communication format for discharge summaries. We studied audit using standardized checklist as a teaching tool.

Methods:
Trained medical record reviewers reviewed discharge summaries completed by 15 index hospitalists for accuracy and completeness. For the intervention, these hospitalists conducted an audit of their peer's discharge summaries using a standardized checklist from October to December 2016. After the intervention, trained medical record reviewers audited the discharge summaries completed by the 15 index hospitalists. Weighted mean and standard deviation were calculated and two sample t-tests were used to compare the two groups with a 2-tailed p-value cutoff of 0.05.

Result:
Medical record reviewers audited 51 discharge summaries before and 70 after intervention. Mean accuracy improved from 92.27% to 97.03% and completeness improved from 81.53% to 86.61% after intervention. Change for both accuracy and completeness by 4.76% (P<0.001) and 5.08% (P <0.001), respectively, were statistically significant.

Conclusion:
Using standardized checklist to audit discharge summaries completed by peers is an effective teaching tool that can significantly improve the accuracy and completeness of the participating hospitalists' own discharge summaries. The clinical significance of our study is highlighted by the literature emphasizing gap in care during the transition process. Going forward, our aim is to implement this checklist and audit as teaching tool for the internal medicine residents.
Rare Case of Thyroid Storm Masked by Diabetic Ketoacidosis

Diabetic ketoacidosis (DKA) can be triggered other clinical conditions. In this case we describe uncommon presentation of DKA induced by thyroid storm.

82-year-old woman with history of type II diabetes presented with two days of falls, abdominal pain, nausea and vomiting. She was noted to have hyperglycemia with high anion gap metabolic acidosis and positive serum and urine ketones suggesting DKA. Investigations were negative for triggering infections. DKA resolved with intravenous fluids and Insulin. However, she remained consistently tachycardic. Tachycardia work up on the second day of presentation including TSH, free T4, free T3 were suggestive of thyrotoxicosis. Thyroid peroxidase antibodies and thyroid stimulating immunoglobulins were also positive. Thyroid ultrasound showed several thyroid nodules. Methimazole and Prednisone were initiated with good response. On day 10 and 70 from initial presentation, patient showed significant clinical and laboratory improvement.

Literature review shows several similar case reports of newly diagnosed thyrotoxicosis manifested by DKA in known type I and type II diabetic patients. Some of these cases were obscured by other confounding conditions including pericarditis, flu syndrome, psychosis and cellulitis ultimately delaying diagnosis and treatment of thyroid storm. Fortunately, none of these cases were associated with adverse outcomes.

While very uncommon, thyroid storm and DKA can simultaneously occur. DKA is mostly diagnosed earlier than thyrotoxicosis given standardized initial laboratory testing for DKA. By presenting this case we emphasize on including thyroid function test as part of the initial work up for DKA to avoid delayed diagnosis and preventable adverse outcomes.
Giant Cell Myocarditis and the Role of Early Endomyocardial Biopsy

Introduction
Giant Cell Myocarditis (GCM) is a rare and deadly disease seen in relatively young individuals. It is known for its frequently missed diagnosis until autopsy/transplant and its association with other autoimmune conditions in 20% of cases.

Case
A 44-year-old male with a history of rapidly progressing cerebellar ataxia and vitiligo, presented with chest pain, shortness of breath and had multiple episodes of ventricular tachycardia (VT) in the emergency room. Blood work revealed elevated troponins, Erythrocyte sedimentation rate and C-Reactive protein. Electrocardiogram showed Q waves in lead 1, aVL without ST changes. Echocardiogram showed moderate global hypokinesis with an ejection fraction of 33%. Cardiac catheterization revealed unobstructed coronary arteries. Cardiac MRI showed minimal late gadolinium uptake, severely reduced global systolic function and cardiac edema. Lack of definite late gadolinium enhancement swayed suspicion away from GCM, thus delaying endomyocardial biopsy (EMB). Patient’s hospital course was complicated by intermittent runs of non-sustained VT and cardiogenic shock requiring intra-aortic balloon pump support. He ultimately succumbed to cardiac arrest on day 5 of admission and was found to have GCM on autopsy.

Discussion
Untreated GCM is rare and fatal with significant improvement in long-term survival if treated appropriately, thus underlining the paramount importance of timely diagnosis. Cardiac MRI is inferior to endomyocardial biopsy in diagnosing GCM, with the gold standard being a surgical biopsy. Therefore, in the right clinical scenario, escalating investigation rapidly to include an EMB despite a benign MRI, may be lifesaving.
Recurrent Muscle Cramps in Cirrhotic Patients - Rare Cause

There is an increased prevalence of chronic muscle cramps in patients with cirrhosis; cause is incompletely understood. Rifaximin, used for hepatic encephalopathy prophylaxis, also causes muscle cramps, rarely associated CPK elevation.

Case presentation
A 27-year-old African American female with Liver Cirrhosis presented with severe generalized muscle cramps for a few days, especially in hands, abdomen and legs. She had a similar episode 1 month ago, managed at a different facility. PMH was significant for autoimmune hepatitis diagnosed twenty years, cirrhosis, s/p TIPS and recurrent hepatic encephalopathy on Lactulose/Rifaximin maintenance therapy. On presentation she was in severe pain, with tachycardia, mild ascites, muscle soreness without weakness. No asterexis seen. Labs showed elevated lactic acid, creatinine 1.46, negative UDS,UA with 3+ blood but no RBCs, positive urine myoglobin and CK of 5866. Patient was treated for rhabdomyolysis with intravenous fluids and analgesia. Her diuretics were held. Despite hydration, patient continued to have severe cramps with muscle soreness and the next day the CK was 7105. At this stage Rifaximin was discontinued. The very next day, the muscle cramps improved and CK was down to 473. Patient was asymptomatic at the time discharged and was instructed not to take rifaximin.

Conclusion:
The link between patients with liver cirrhosis and rhabdomyolysis while being treated with Rifaximin has been documented in the past, but it may not be readily recognized. Muscle cramps in cirrhotics on Rifaximin should not be presumed to be due to their liver disease and CK levels should be checked.
Elevated Serum Triglycerides Interfere with Serum Lipase Assay and Confound the Diagnosis of Acute Pancreatitis

INTRODUCTION
The sensitivities of serum amylase and serum lipase for diagnosis of acute pancreatitis are reported to be 0.72 and 0.79 respectively. In patients with a high suspicion of pancreatitis, normal levels do not exclude the diagnosis. We present a CT confirmed case of acute pancreatitis and markedly elevated triglycerides with a normal serum lipase.

CASE
A 34-year-old gentleman, with a past medical history of alcohol abuse and family history of hypertriglyceridemia, presented to the emergency department with sudden onset of severe epigastric abdominal pain. The pain was 8/10, constant and sharp in nature. He had nausea and one episode of bilious, non-bloody vomiting. He drinks five to six beers daily for more than 15 years. His lipase level was 68 units/L (reference range 22-51 units/L) total cholesterol 383 mg/dL; serum triglyceride >5,000 mg/dL; and HDL 25 mg/dL. CT abdomen with contrast revealed peripancreatic inflammation and fat stranding at the pancreatic body consistent with acute pancreatitis. The patient was managed conservatively and his symptoms resolved.

DISCUSSION
Medical literature reports the estimated negative predictive value of serum lipase was between 94% to 100%. Normal serum lipase in the setting of acute pancreatitis is extremely rare. Our hospital employs the Random Access Lipase reagent which utilizes a photometric method to determine pancreatic lipase activity. Hyperbilirubinemia, free hemoglobin and hypertriglyceridemia exceeding 500 mg/dL have been reported to interfere with this lipase determination method. Normal lipase levels do not exclude the diagnosis of acute pancreatitis, especially in the setting of hypertriglyceridemia.
An Unusual Eruption of an Upper Respiratory Infection

INTRODUCTION
Acute Generalized Exanthematous Pustulosis (AGEP) is an acute eruption of numerous nonfollicular, sterile pustules on erythematous, edematous skin, accompanied with fever and leukocytosis that develops within hours of exposure to antibiotics in one in a million cases. Very few cases triggered by viral infections have been reported.

CASE
A 62-year-old female presented with a one week history of sore throat, fever, and non-painful chest redness. The rash initially began on her face, without ocular or oral manifestations. The rash progressed to involve her trunk and extremities with skin sloughing, pustules and fever (105.6°F). Physical exam revealed exquisite dermal tenderness, diffuse erythema with facial edema, innumerable pinpoint pustules, with patchy desquamation of skin. Nikolsky sign was negative. She was thrombocytopenic (90,000 per μL), anemic (10.2 gm/dL), and leukopenic (3400 cells/mm3). Serology did not reveal acute infections with parvovirus B19, Epstein Barr virus, or HIV. She was started on clindamycin and topical hydrocortisone. Culture of pustular exudate was negative. A skin biopsy showed intraepidermal and subcorneal, pustular dermatosis with large neutrophil aggregates and scant eosinophils which confirmed AGEP. Her pancytopenia resolved and the skin lesions improved.

DISCUSSION
The onset of AGEP was prior to initiation of antibiotics and accompanied by pancytopenia, possibly triggered by a viral respiratory illness. Prior reports have implicated Parvovirus and EBV as the triggers which was also not the case. Clinicians should be aware of atypical presentations of rare diseases like AGEP with pancytopenia triggered by a viral infection.
Recurrent Pancreatitis due to Celiac Disease

INTRODUCTION
Celiac Disease (CD) is a disorder characterized by small bowel mucosal inflammation, villous atrophy, and crypt hyperplasia that occurs with exposure to dietary gluten. CD has extraintestinal manifestations such as pancreatico-biliary disease. Postulated mechanisms include reduced gallbladder emptying due to impaired cholecystokinin release and pancreatitis due to malnutrition. We present a case of recurrent pancreatitis due to underlying CD.

CASE
24-year-old lady presented with epigastric abdominal pain radiating to the back, associated with nausea and vomiting. Physical examination revealed exquisite tenderness in her epigastrum. Her lipase was 1,417 U/L. Ultrasound of the abdomen showed a dilated pancreatic duct. She underwent magnetic resonance cholangiopancreatography which showed an enlarged pancreas with peripancreatic inflammation. She subsequently underwent esophagogastroduodenoscopy and duodenal biopsy which showed duodenitis with intraepithelial lymphocytes and villous blunting. Tissue transglutaminase (TTG) IgA antibody level was 77 U/mL. She instituted a gluten free diet, and her symptoms resolved. Follow up 3 months later showed continued resolution of her symptoms. Her TTG-IgA antibodies decreased significantly after one year.

DISCUSSION
A number of epidemiologic studies have reported an increase incidence of pancreatitis in CD patients when compared to the general population. CD should be suspected in patients with pancreatitis in the absence of other common risk factors such as alcohol intake, medications, cholelithiasis, trauma and hypertriglyceridemia. CD should also be considered in the etiology of papillary stenosis.
Amiodarone Induce Lung Injury Presenting as a Lung Mass

INTRODUCTION
Amiodarone-induced pulmonary toxicity (AIPD) is serious and third most common side effect with incidence of 3-15%. Pulmonary adverse effects are usually observed with dose of 400mg or more daily usage for 6-12 months. Although, diffuse interstitial disease is the common presentation with incidence of 26.7%. But, we present a rare case of focal amiodarone lung disease mimicking malignancy.

CASE
An 80-year-old male with remote history of smoking, prostate cancer, Paroxysmal A fib on amiodarone for 18 months was admitted with L1 vertebrae fracture due to fall. Patient denied any cough, fever, and dyspnea. Investigations showed WBC 15.4 thous/MCL. Chest X-ray revealed posterior left lobe consolidation with pleural effusion was confirmed with CT Chest. Subsequently, antibiotic therapy for pneumonia was commenced. During hospital stay patient underwent Kyphoplasty. Repeat CXR did not exhibit any change in consolidation. There was even worsening of pleural effusion after completing antibiotics. Patient also endorsed 20 lbs weight-loss in last 2-3 months. Thoracentesis demonstrated transudate fluid without any malignant cells. Finally, CT guided biopsy from lung parenchyma confirmed amiodarone-induced lung injury.

Discussion:
AIPD should be among differential diagnosis if any patient on amiodarone presents with new infiltrates or pleural effusion which is usually exudate but transudate in our case. There are only limited case reports and case series reported in literature with focal amiodarone-induced lung injury. Most patients responded well to the withdrawal of amiodarone and steroid therapy. It often takes several months for radiological findings to resolve due to longer half-life of amiodarone metabolites.
Twiddler Doo-Daa, Twiddler Ouch! My Oh My, the Shocks Are in My Armpit!

Introduction
Implantable cardioverter defibrillator (ICD) is an effective tool that reduces the risk of sudden cardiac death due to arrhythmias. ICD implantation has late complications rates of 2 to 3%. Lead dislodgement occurs in about 4% of patients with late complications that present as arrhythmia, perforation or inappropriate pacing of adjacent structures. Physicians should obtain a left sided chest x-ray in patients with ICDs who come in with a new complaint of localized seizure like muscular activity or unilateral paresthesia.

Case Presentation
A 54 year old gentleman with atrial fibrillation and ischemic cardiomyopathy presented to us with irregular heart rate averaging 140BPM, tremors, paresthesia and electric shocks when he lays on his left side. He had been treated outpatient for possible neuropathy without relief. In-hospital chest x-ray identified an ICD. Close inspection revealed the device had shifted in position and leads were wrapped around the generator. Cardiology referral for device re-implantation was obtained.

Discussion
Twiddler syndrome is a rare cause of ICD malfunction that can result from deliberate or accidental manipulation of the device by patients with resulting retraction of the lead into the implantation pocket. It results in failure to sense or treat arrhythmia as was noted in this case.

Conclusion
Patients with ICDs who report new localized abnormal neuromuscular activity in the chest, neck or upper limbs or arrhythmia could have lead dislodgment secondary to Twiddler syndrome. A simple chest x-ray can quickly identify this, mitigating associated morbidity.
Massive Fatal Intraperitoneal Bleeding from Gastrohepatic Ligament Following PEG Tube Placement: An Extremely Rare Complication

The insertion of a PEG tube is a safe procedure with few complications. Immediate mortality after the procedure is less than 1%. Intraperitoneal bleeding is extremely rare and a few cases have been reported associated with the hepatic laceration and spleen avulsions. An 84-year-old male admitted for pneumonia and found to have dysphagia and the family decided for PEG tube placement for further feeding. PEG tube was placed in the first attempt without any obvious complication by an experienced gastroenterologist. 5 hours post-procedure, the patient was found unresponsive and pulseless. Resuscitation was started following ACLS protocols and ROSC was obtained within 4-5 minutes and he was transferred to MICU. He was started on vasopressors. Bedside abdominal ultrasound showed massive fluid in the intraperitoneal cavity. Labs showed a significant drop in hemoglobin to 6.3 from 10.6. The gastroenterologist was called, and emergent EGD at bedside did not show any signs of bleeding in the stomach. The patient was then sent to the operating room for emergent laparotomy for intraperitoneal bleed. Active bleeding from the gastrohepatic ligament was found. 1400 ml of blood was drained. The source of bleeding was controlled. Despite multiple blood transfusions and high dose norepinephrine and vasopressin infusion, the patient remained hemodynamically unstable. The patient could not sustain the acute conditions and expired. PEG tube placement is a safe and a very common procedure. Massive intraperitoneal bleeding is extremely rare. Physicians should be mindful of immediate massive bleeding from gastrohepatic ligament after Russell PEG procedure.
A Rare Case of Bilateral Achilles Tendon Rupture Secondary to Levofloxacin

Tendon rupture secondary to Levofloxacin use is rare, estimated to be 1 in 1.6 million prescriptions. Bilateral involvement occurs in only 50% of tendon rupture cases associated with Fluoroquinolone use.

An 89-year-old male with history of COPD and CAD presented to the ED complaining of weakness in his legs. Two weeks prior he was discharged on Levofloxacin and Prednisone for Pneumonia and COPD exacerbation. He fell the night prior to admission after he developed stiffness in his ankles. The following morning he was unable to bear weight on his legs and had bilateral heel pain. On initial exam, protuberant masses and tenderness was noted near the Achilles tendon bilaterally. Initial creatinine was 1.2 and X-ray of the Ankles were normal. MRI was positive for bilateral full thickness Achilles tendon tears. Orthopedics was consulted and he was deemed not a surgical candidate given multiple comorbidities. Patient was discharged to inpatient rehabilitation for weight bearing exercises.

Risk factors for tendinopathy with Fluoroquinolone use include high dose, prolonged use, age greater than 60 years, concurrent steroid therapy, history of organ transplantation, renal dysfunction with steroid. The latter triples the odds of tendon rupture. Age and steroid use were risk factors in our case. Levofloxacin prescription should be avoided in those on steroid therapy if possible, particularly in the elderly population, to decrease tendinopathy risk. Patients should be educated about the risk of tendon rupture, identification of tendinopathy symptoms including tendon pain early in the course and to seek medical attention if symptoms develop.
A Series of Unfortunate Events Following Silicone Injections

A 50-year-old man presented to the ED with a chief complaint of progressively worsening severe left hip pain and fevers for two weeks along with hematochezia for two days. Upon initial exam, he was febrile, his left proximal thigh was tender and his left hip movement was severely restricted, multiple sinus tracts draining pus were noted on both his breasts. Upon further questioning, he explained that he purchased an unknown dermal filler online and self-administered it into his bilateral pectoral region around two months prior to presentation. He only noted the purulent breast drainage a few days prior to presentation. Initial labs showed moderate leukocytosis, severe normocytic anemia and mild lactic acidosis. Empiric IV antibiotics were started. A left hip MRI showed multiple intramuscular abscesses and possible left hip septic joint which were drained. Breast wound and blood cultures grew MSSA. During his hospitalization, he required emergent angiography for severe hematochezia, which revealed right colonic angiodysplasia. Emergent colonoscopy failed to achieve hemostasis requiring right hemicolecotomy. He then underwent bilateral breast debridement for source control. Surgical pathology report revealed silicone material and granulomas. Orthopedics recommended outpatient total left hip arthroplasty. Patient was discharged on IV Cefazolin for 6 weeks.

Excessive NSAID use for pain control possibly could have contributed to GI bleeding in this patient.

Injectable cosmetics are easily obtainable products, but inappropriate use may lead to life-threatening complications as illustrated by our case; this highlights the need for heightened regulations and increased awareness of the risks of injectable cosmetics use.
Coexisting Virchow and Irish Nodes - A Rare Case of Metastatic Adenocarcinoma of the Pancreas

Introduction: The left supraclavicular lymph node (Virchow's node) is a common site of metastasis in breast, lung, and gastric cancers but represents a rare site of metastasis in pancreatic cancer with only 11 reported cases. The left axillary lymph node (Irish node) is a rare site of metastasis usually associated with advanced gastric cancers. There are no reported cases of Irish node metastasis from pancreatic cancer and the coexistence of both nodes together has been reported only once in a cancer that was of gastric origin.

Case Description: 67-year-old female presented with abdominal bloating. CT scan revealed a mass in the pancreatic body. Biopsy confirmed the presence of a pancreatic adenocarcinoma. Whipple procedure was performed and this was followed by multiple sessions of chemotherapy, as well as radiation and biological therapy. Over the course of the following two years, the patient had developed stage IV pancreatic cancer with metastasis to the liver and lung that required further resection. She later on developed left supraclavicular adenopathy which was biopsy proven to be metastatic carcinoma originating from the pancreas. Subsequently, she started to develop a hard nodule in her left axilla. Imaging revealed a new left axillary lymphadenopathy. She passed away three weeks later due to complications of metastatic pancreatic cancer.

Discussion: Virchow's and Irish nodes represent a very uncommon site of metastases in pancreatic cancer and carry clinical significance in prognosticating and possibly altering the course of therapy.
Novel Treatment Therapy for Delayed Post-Hypoxic Leukoencephalopathy due to Recreational Drug Overdose

Introduction: Delayed post-hypoxic leukoencephalopathy (DPHL) is a rare condition that follows an anoxic brain injury, such as carbon monoxide poisoning and recreational drug overdose. A lucid period follows for 1-3 weeks with an abrupt onset of neuropsychiatric symptoms often mistaken as a primary psychotic illness.

Case description: A 28-year-old female with a history of IV drug use presented with agitation after recent respiratory failure from a recreational drug overdose. She was doing well at home for 2-3 weeks after the event with only short-term memory loss. At presentation, she had acute onset agitation with paranoia, hallucinations, delusions, minimal sleep requirements and depressed affect. A complete workup for infectious causes was negative. Arylsulfatase A level was normal and metachromatic leukodystrophy was ruled out. MRI demonstrated a symmetric abnormal hyperintense T2 signal abnormality involving the bilateral caudate nuclei, anterior lentiform nuclei and globus pallidus compatible with leukoencephalopathy. EEG was unremarkable. Several medications were trialed without relief of symptoms, including levetiracetam, topiramate, olanzapine, and trazodone. A regimen of quetiapine with lorazepam was effective, in addition to propranolol. Upon discharge, she had significantly decreased agitation.

Discussion: Our patient had the classical presentation of DPHL following a recreational drug overdose with classic hyperintensity on MRI imaging. Our patient did not have pseudodeficiency of arylsulfatase A as a predisposition for DPHL, although this is not always present. The literature does not discuss recommended treatment therapies for managing a patient's neuropsychiatric symptoms. Therefore, this case report will add to the literature already present on DPHL.
Posterior Reversible Encephalopathy Syndrome After 5-FU Therapy for Colon Cancer Presenting as Status Epilepticus

Posterior Reversible Encephalopathy Syndrome (PRES) associated with chemotherapy is reported to have an incidence of 0.02 to 0.1% but is generally described with Bevacizumab. 5-Fluorouracil (5-FU) associated cerebral toxicity is rare, presenting as multifocal inflammatory leukoencephalopathy which is distinct from PRES. We report an unusual case of PRES associated with 5-FU therapy presenting with Status Epilepticus (SE).

A 43-year-old woman with stage IV rectal adenocarcinoma on the 5th cycle of 5-FU continuous infusion presented with new onset generalized tonic clonic seizures. This was preceded by confusion since the previous day. Initial BP was 151/109 mmHg and physical exam was pertinent for ongoing generalized convulsions. Labs revealed normoglycemia with creatinine elevation of 1.3 mg/dl, mild hypercalcemia and mild hypomagnesemia. IV Lorazepam and Phenytoin failed to control seizures necessitating intubation and initiation of Propofol drip. MRI brain showed bilateral multifocal cortical and subcortical T2/FLAIR hyperintense signal in occipital lobes suggestive of PRES. There was no evidence of intracranial mass, infarction or hemorrhage. CSF analysis revealed normal cell count and cytology. On Day 3, EEG showed cessation of seizure activity, thus propofol was discontinued, patient extubated and discharged to inpatient rehabilitation. Repeat MRI brain after 8 days showed resolution of prior abnormalities. PRES risk factors including fluid overload, increment of mean BP 25% above baseline and creatinine greater than 1.8mg/dl were absent in our case, hence, favoring 5-FU as a likely etiology. Our case also highlights the significance of recognising confusion as early signs of PRES.
Hemodynamic Recovery Following EKOS-Catheter Directed Thrombolysis for Saddle Pulmonary Embolus

Most deaths in acute pulmonary embolism (PE) result from right ventricular (RV) pressure overload and subsequent failure. EKOS-catheter directed thrombolysis is increasingly used for treatment of massive and submassive PE. Various parameters are being considered to assess thrombus resolution and hemodynamic recovery post procedure. We demonstrate early resolution of RV dysfunction with normalization of heart rate and recovery seen on transthoracic echocardiogram (TTE).

A 70-year-old male presented with dyspnea and pleuritic chest pain. On exam, heart rate was 105 bpm with a loud S2 and respiratory rate of 30. Blood pressure was normal. Blood work showed troponin I level of 1.01ng/mL. Chest computed tomography (CT) showed bilateral saddle pulmonary emboli. Transthoracic echocardiography showed severely dilated and hypokinetic right ventricle. Patient was treated with intravenous heparin and underwent bilateral catheter directed tPA via EKOS. Alteplase was delivered to the EKOS catheters at a rate of 2 mg/hour for 4 hours then 1 mg /hour for 12 hours. Within a short period after the procedure, tachycardia and dyspnea improved, indicating improvement of the right ventricular strain following EKOS. The basal RV diastolic diameter on TTE reduced from 5.10 cm to 4.43 cm two days post procedure. Catheter directed thrombolysis; including EKOS, can rapidly restore normal pulmonary artery perfusion and improve pulmonary arterial pressure and right ventricular strain in patients with large clot burden. Various parameters are being studied to evaluate post procedural success of EKOS; our case highlights the use TTE and resolution of tachycardia as early indicators of recovery.
Severe Hypomagnesemia, Hypokalemia, and Hypocalcemia with Tremor and Gait Instability due to Proton Pump Inhibitor Use

Magnesium deficiency is associated with a number of additional electrolyte derangements including hypokalemia and hypocalcemia. Severe deficiency can potentially lead to lethal sequelae, including cardiac arrhythmias and seizures. Current evidence suggests gastrointestinal losses of magnesium as the primary mechanism of hypomagnesemia in the setting of proton pump inhibitor (PPI) therapy. The use of PPI therapy is highly prevalent worldwide and often times PPIs are inappropriately utilized both in the outpatient and inpatient settings. In this report, we present the case of a 67-year-old male with a history of chronic PPI use due to severe esophagitis in the setting of gastroesophageal reflux disease with hiatal hernia who presented with symptoms of generalized weakness associated with lightheadedness, tremors, gait instability and falls. The patient was found to have severe hypomagnesemia with concomitant severe hypokalemia and hypocalcemia, that began to improve following exchange of PPI for Histamine H2-receptor antagonist with aggressive intravenous electrolyte replacement.
Atrial Septal Aneurysm as the Etiology for a Cryptogenic Stroke

We report a 43-year-old woman with a medical history of scleroderma, pulmonary hypertension, Lupus, and recurrent deep vein thrombosis (DVT) on apixaban who was admitted for community acquired pneumonia. Around 5:00 am the morning after admission, she developed sudden onset left sided hemiplegia, and left sided homonymous hemianopsia. Stroke work up included, a MRI revealing right sided frontal and parietal acute infarctions, as well as a transthoracic echocardiogram with a positive bubble study. With patient’s history of recurrent DVTs, cryptogenic stroke became the working diagnosis. Transesophageal echocardiogram did not reveal the suspected atrial septal defect (ASD) or patent foramen ovale (PFO); however did show an atrial septal aneurysm (ASA) as well as a pulmonary arteriovenous malformation (AVM). Upon further history, patient revealed she had been non-adherent with anticoagulation therapy. Patient’s visual symptoms resolved, however at time of discharged she had residual left sided weakness.

ASAs occur in up to 2.4% of patients, and can be congenital or arise secondary to increased chamber pressures. ASAs have 2 mechanisms for cardioembolic events. One is via a commonly comorbid interatrial shunt, such as a PFO or ASD. The second mechanism is the aneurysm itself can act as a nidus for thrombus formation in the left atrium. Our case is unique because the ASA was accompanied by a pulmonary AVMs, instead of the usual PFO or ASD. If our patient were to have a second cardioembolic event, consideration must be given to repairing the pulmonary AVM.
An Incidental Finding of Streptococcus Salivarius Bacteremia with Associated Endocarditis: A Case Report

Streptococcus salivarius is a facultative anaerobic bacterium that commonly inhabits the gastrointestinal tract. It is described as a commensal organism that occasionally causes opportunistic infections. It rarely causes bacteremia and data regarding this organism causing significant pathology is limited. Only 5–15% of blood culture isolates of Streptococcus viridans are S. salivarius. Bacteremia is uniquely associated with a mucosal breakdown in the gastrointestinal tract (i.e. due to cancer) and noncolonic malignancies. We present a case of native aortic valve endocarditis due to this rare organism, penicillin-resistant S. salivarius, in the absence of occult malignancy.

A 60-year-old male with a medical history remarkable for Barrett’s esophagus, tobacco and alcohol use disorder was admitted to our hospital for a complicated urinary tract infection. He had no presenting cardiac symptoms. Blood cultures grew S. salivarius with intermediate resistance to penicillin with an MIC of 0.5 ug/mL, but susceptible to ceftriaxone. A transthoracic echo demonstrated severe aortic regurgitation with multiple vegetations. The patient underwent emergent surgical aortic valve replacement and was treated with ceftriaxone and gentamycin for four weeks. Workup did not reveal a malignancy. Nonetheless, the patient had poor oral dentition with multiple cavities and a history of Barrett’s esophagus. We hypothesize that the organism could have translocated into the bloodstream via these areas of mucosal breakdown. The patient was set up with gastroenterology for periodic screenings of the upper and lower gastrointestinal tract.
Eosinophilic, Neutrophilic, and Monocytic Leukemoid Reaction in a Woman with Adenocarcinoma of the Lung

A leukemoid reaction represents an elevation in white blood cell counts with a characteristic left shift along the leukocyte differentiation pathway. This reaction can be a sign of an underlying infection, hematologic malignancy or a medication side effect. The causes of such a reaction are sometimes difficult to identify. Paraneoplastic syndromes are common in lung malignancies, however, a combined eosinophilic, neutrophilic and monocytic leukemoid reaction in patients with lung adenocarcinoma patients has been rarely described.

A 57-year-old female with adenocarcinoma of the lung metastatic to liver and bone, presented with confusion of one day duration. A complete blood count revealed a white blood count of 95100/ mm3, eosinophils of 27100/ mm3, monocytes of 1700/ mm3 and neutrophils of 38400/ mm3. Peripheral blood smear showed numerous neutrophils with vacuoles, many eosinophils, and monocytes. Very few myelocytes and pro-myelocytes were seen. Blood and urine cultures were negative. A chest X-ray was negative for new infiltrates and a brain MRI showed no signs of metastasis that could explain her confusion. Polymerase chain reaction for BCR-ABL gene mutation was ordered to rule out a rare variant of Chronic Myeloid Leukemia and was still pending to date.

The leukemoid reaction in this patient was attributed to a paraneoplastic response from the production of hematopoietic growth factors such as granulocyte–macrophage colony-stimulating factors, stimulating all cell lineages. Even though no other causes for her eosinophilia, neutrophilia, and elevated monocytes were identified, patients with these conditions should be also evaluated for rare hematologic malignancies.
The Disguise Artist: A Case of ANCA Vasculitis with Atypical Symptoms

Anti-neutrophil cytoplasmic antibody (ANCA) associated vasculitis is a spectrum of conditions related to the production of autoantibodies, specifically anti-neutrophilic cytoplasmic antibodies, that lead to downstream damage to blood vessel endothelium and tissues. ANCA-associated vasculitis (AAV) is typically associated with a constellation of symptoms such as fatigue, weight loss, arthralgias, cough, rhinosinusitis and neurologic dysfunction. Epidemiologically, affected patients are predominantly elderly males with 90% of cases affecting renal function. However, the diagnosis and management of AAV may be especially challenging when faced with an atypical presentation.

We present a case of a 22 year-old male with a history of congenital hydrocephalus, seizure disorder, and chronic headaches presenting with one-week history of hypothermia, unsteady gait and altered mental status. Extensive neurological, hematological and infectious work-up did not yield any results. Incidental development of hematemesis, and consequent CT imaging of the lungs showed bilateral pulmonary disease with interstitial lung disease. Subsequent lung biopsy confirmed the diagnosis of isolated pulmonary microscopic polyangiitis (MPA), a type of AAV. The patient was treated with steroids and azathioprine, along with rituximab. The patient’s clinical symptoms and mentation improved drastically after initiation of therapy. Our case highlights the importance of considering vasculitis on the differential even when the most common symptoms and clinical features are not present. The presentation of AAV can range from single to multi-organ involvement, with varying degrees of organ damage. Ultimately, the prompt diagnosis of AAV is imperative as early initiation of therapy leads to improved outcomes.
Laughing Gas Is No Joke

Introduction
Chronic abuse of N2O causes multiple hematologic and neurologic disorders. It interacts with vitamin B12 resulting in selective inhibition of methionine synthase and folate metabolism. Neurologic findings usually include polyneuropathy, ataxia and psychosis.

Case Presentation
A 24-year-old male with history of poly-substance abuse including N2O, presented to the outpatient clinic for chronic numbness and tingling of 2 months duration, started in his palms and soles and extended upwards to his thighs. He also experienced diminishing fine motor skills like writing, without loss of motor power. On physical exam, patient was alert and oriented with intact cranial nerves. Sensation was diminished in a symmetric fashion over bilateral hands, sparing the dorsal aspect, extending to the distal third of the forearms bilaterally, along with soles and ankles of the feet, sparing dorsal aspect and extending to the medial thighs. Proprioception and vibratory sensation were also diminished bilaterally in both hands and feet. Neurologic exam showed a slightly positive Romberg’s sign. Laboratory work-up showed normal electrolytes and Hemoglobin, with slightly elevated MCV and low vitamin B12 level. Patient was started on weekly vitamin B12 injections for possible nitrous oxide toxicity. Symptoms gradually improved over the course of 4 weeks.

Discussion
Neurotoxicity secondary to chronic nitrous oxide abuse, manifesting in a similar fashion to subacute combined degeneration syndrome, is potentially reversible with vitamin B12 supplementation and abstinence from N2O use. In case of marginal B12 stores, nitrous oxide use could actually, acutely cause B12 deficiency and neurotoxicity.
A Young Adult Without Joint Pain - An Unusual Presentation of Adult-Onset Still's Disease

Adult-onset Still's disease (AOSD) is a rare inflammatory condition hallmarked by daily fevers, a salmon-colored maculopapular rash, and inflammatory arthritis. This case presentation illustrates a patient who did not present with typical inflammatory arthritis symptoms and yet was diagnosed with AOSD.

Our patient is a 36-year old gentleman who presented to the ED with the acute complaint of malaise, myalgias, and sore throat for five days. He was febrile on presentation prompting a full infectious workup which was negative. He continued to have daily fevers accompanied by a rash and his clinical course was complicated by the development of myopericarditis, pleural effusions, and a new normocytic anemia. During his hospital stay the patient denied having any joint pain, and no joint erythema nor deformities were noted. A ferritin level was checked and was greater than 7500ng/ml. A peripheral smear and bone marrow biopsy did not reveal malignancy or hemophagocytosis. A diagnosis of AOSD was established and he was transferred to a tertiary care center for a rheumatology consult. He was started on anakinra and his symptoms significantly improved.

AOSD is a diagnosis of exclusion with non-specific symptoms requiring an exhaustive diagnostic workup to rule out infection, malignancy, autoimmune conditions, and drug reactions. Interestingly, the patient did not present with the typical joint pain and therefore the diagnosis was delayed. Thus, it is important to consider this diagnosis in your differential when a patient presents with generalized multi-organ inflammation even when typical arthritis symptoms are not present.
Are We Missing Diabetes Mellitus Type 3c?

Diabetes Mellitus Type 3c (T3cDM), also called pancreatogenic diabetes is associated with underlying pancreatic pathology, most commonly chronic pancreatitis.

A 66-year-old man was hospitalized for dehydration and syncope secondary to hyperglycemic hyperosmolar state. He was discharged on 70 units insulin detemir (LAI) daily. A week later, he presented with altered mental status, non-bloody diarrhea and 20lbs weight loss. On physical examination he was dehydrated and disorientated. His finger stick glucose was 31 mg/dL and hemoglobin A1c was 18.5. After hypoglycemia was corrected with supportive management, LAI was started at a lower dose (40 units) daily. His LAI had to be titrated down due to hypoglycemic episodes. He eventually required only 5 units a day, which was 7 percent of his home dose.

Further workup revealed low C-peptide level, negative anti-glutamic acid decarboxylase and anti-islet cell antibodies. Vitamins A, D and K levels were also low. Imaging studies demonstrated signs of chronic pancreatitis. His diarrhea improved with pancreatic enzymes supplement.

Patients with T3cDM can be at varying stages of insulin deficiency and sensitivity, depending on the course of pancreatitis. This can lead to higher risk of developing hypoglycemic and hyperglycemic complications. Insulin requirement to achieve and maintain glycemic control in such patient may be significantly less than other insulin-dependent patient.

There should be a high clinical suspicion for T3cDM in the patients with underlying pancreatic pathology. Insulin therapy should be initiated with caution in such patients. Alternating stages of insulin sensitivity and resistance can predispose patients to hypoglycemic and hyperglycemic complications.
Chylothorax: A Rare Complication of Upper Extremity Deep Venous Thrombosis (DVT)

A 24-year-old female presented with acute, progressively worsening shortness of breath, left sided neck and axillary pain with swelling, and temperature of 101.1°F. Physical exam revealed decreased breath sounds, dullness to percussion, and egophony in the right lower lung field. Imaging of the chest with an X-ray and subsequent computed tomography showed a moderate-sized, right-sided pleural effusion. Given the elevated concern for thrombosis, Doppler ultrasonography was performed of left upper extremity and showed an extensive DVT involving the jugular and subclavian veins. A right-sided thoracentesis was performed, with about 600 mL of cloudy, yellow fluid drained. Pleural fluid analysis confirmed a chylous effusion, with triglycerides of 564 mg/dL and 2371 nucleated cells with 97% lymphocytes. As her current DVT was unprovoked, an evaluation for thrombophilia was initiated, with results revealing heterozygosity for Factor V Leiden gene mutation.

Chylothorax is caused by disruption or obstruction of the thoracic duct or its tributaries, with increasing lymphatic pressure leading to leakage of chyle into the pleural space. On rare occasions, deep venous thrombus of the jugular – subclavian junction can obstruct the thoracic duct and its tributaries, causing chyle to leak in pleural space. As illustrated by our patient, left upper extremity DVT in the setting of thrombophilia, lead to obstruction of the lymphatic vasculature, leading to chylothorax. It is important to maintain a high index of suspicion for chylothorax in patients presenting with upper extremity DVTs and shortness of breath.
A Case of Systemic Sarcoidosis Presenting as Hypercalcemia-Induced Pancreatitis

Introduction: Sarcoidosis is a granulomatous inflammatory disease which can involve multiple organs and can have varied presentation. We present an interesting case of sarcoidosis masquerading as acute pancreatitis.

Case: A 42-year-old African-American-female presented with acute onset abdominal pain associated with nausea. She also complained of fatigue and dry cough for 3 weeks and dry mouth for a year. Physical examination was unremarkable except for abdominal tenderness without guarding and aphthous ulcers in mouth. Labs were significant for lipase of 879 U/L consistent with clinical diagnosis of pancreatitis. CT scan of abdomen/pelvis was significant for extensive retroperitoneal lymphadenopathy and hepatosplenomegaly. Workup for pancreatitis was unremarkable except for total calcium of 15.4 mg/dL confirming hypercalcemia as etiology for pancreatitis. Further evaluation revealed reticulations in the pulmonary apices and bilateral paratracheal lymphadenopathy which were confirmed with CT chest which showed upper lobe fibrosis and bulky mediastinal lymphadenopathy. Right supraclavicular lymph node biopsy was positive for non-caseating granulomas confirming the diagnosis of Sarcoidosis. She was started on Prednisone therapy.

Discussion:
Hypercalcemia occurs in 10-20% of patients with sarcoidosis however, hypercalcemia-induced-pancreatitis in such conditions is a rare occurrence. Hypercalcemia in sarcoidosis occurs due to increased 1, 25-dihydroxy-Vitamin D production by the sarcoid granulomas and often responds well to steroids. This highlights the importance of identifying the underlying systemic process leading to acute pancreatitis as the management differs.
Autoimmune Hepatitis: The Importance of Diagnosis and Differentiation from Lupus Hepatitis

Introduction
Autoimmune hepatitis (AIH) is a chronic liver disease predominantly affecting women with an incidence of 0.8 to 3 per 100,000. Presentation ranges from asymptomatic to fulminant liver failure.

Case
A 50 year old African American female presented with anasarca and dyspnea for three months with progression over the last two weeks. She endorsed painful swelling of her abdomen and legs, denied fevers, arthralgias and rash. Her exam demonstrated pitting edema of bilateral lower extremities and abdominal wall with hepatomegaly. Lab studies demonstrated: pancytopenia; AST 200; ALT 128; antinuclear antibody (ANA) 1:640; smooth muscle antibody (ASMA) positive; IgG 3,190 and anti-double stranded DNA positive. A biopsy of the liver confirmed cirrhosis with histopathology suggestive of autoimmune hepatitis.

Discussion
Our patient was diagnosed with AIH using the International Autoimmune Hepatitis Group scoring system, which accounts for ANA/ASMA, IgG level, absence of viral hepatitis and liver histology. She was treated with standard therapy of prednisone and later azathioprine with near normalcy of her liver function tests one year later. This case is important because AIH and systemic lupus erythematosus (SLE) hepatitis share clinical and serologic presentations, however can be distinguished based on histopathology. The key distinction is the difference in treatment and prognosis. Untreated AIH can progress to cirrhosis and has a 5 year survival rate of 25%. In contrast, SLE does not progress to cirrhosis and is treated with primarily with hydroxychloroquine. Knowing how to differentiate these two entities will improve clinical outcomes and optimize prognosis.
Unprovoked Thromboembolism in the Setting of Nephrotic Syndrome

Introduction: Nephrotic syndrome is characterized by heavy proteinuria, hypoalbuminemia, edema, and often thrombotic disease. Nephrotic syndrome is an important consideration in patients with unprovoked thrombotic events.

Case: A 36-year-old Caucasian male with past medical history of polysubstance and alcohol abuse presents with gradually worsening dyspnea and lower extremity edema for three months. He admits to orthopnea and a 20-pound weight loss during this time. On presentation, he was noted to be hypertensive and tachycardic, cardiopulmonary exams were unremarkable, 1+ pitting edema noted in bilateral lower extremities with tenderness in bilateral calves. Bilateral lower extremity ultrasound was negative for acute DVT, CT angiography of the chest revealed bilateral pulmonary emboli.

Creatinine 0.7 mg/dL, GFR >60 ml/min, albumin 1.5 g/dL. Urinalysis was significant for 3+ protein, and 24-hour urine protein resulted as 15.3 grams/day. ANA, C3, C4, SPEP with immunofixation, light chains, hepatitis B and C testing were all negative. Renal biopsy revealed diffuse glomerular basement membrane thickening, and anti-PLA2R antibody resulted as positive fitting the diagnosis of idiopathic membranous glomerulopathy. The patient was initiated and continued on anticoagulation, he underwent addiction rehabilitation prior to immunosuppressive therapy.

Discussion: Of the various nephrotic syndromes, membranous glomerulopathy poses a significant risk for thromboembolic events, and uniquely prophylactic anticoagulation is considered based on bleeding risk and severity of hypoalbuminemia. The etiology of hypercoagulability in nephrotic syndrome is not well understood, but is found in 10-40% of patients. Serum anti-PLA2R antibody has provided advancements in diagnosis and monitoring of membranous glomerulopathy.