Factitious Disorder Presenting as Atypical DKA

Factitious disorder imposed on self, formerly known as Munchausen Syndrome, is a challenging diagnosis with a variable clinical presentation. We present a case of factitious disorder presenting as repeated episodes of diabetic ketoacidosis but also incidentally discovered in a unique vignette. A 28 year old woman with uncontrolled Type I diabetes mellitus presented to the emergency department with complaints of severe abdominal pain but without vomiting. The patient had presented to the ED several times in the past month for similar complaints but had an inconsistent history of being admitted. Her blood glucose level was 335 mg/dL on admission yet the arterial blood gas demonstrated a pH of 7.40, CO2 of 40 and HCO3 of 24, not consistent with Diabetic Ketoacidosis. Urinalysis demonstrated a moderate amount of ketones, however the patient had also mentioned that she had not been eating for days due to the abdominal pain. However, the patient also did have an anion gap of 26 consistent with a high anion gap metabolic acidosis and a delta-delta gap that suggested a concomitant metabolic alkalosis. Incidentally, a medical resident was revisiting the patient and witnessed her performing self-induced emesis. The patient was asked about her actions and she initially denied her any attention seeking behavior. However, a patient care technician confirmed sighting of the same actions previously. The patient’s self induced emesis explained the concomitant metabolic alkalosis and regular-appearing ABG results. A diagnosis of diabetic ketoacidosis was made which was due to insulin non-compliance and self-induced emesis via factitious disorder imposed on self.
A Lethal Manifestation of a Vitamin Deficiency

Introduction
Although Vitamin B12 deficiency commonly causes macrocytic anemia and neuropsychiatric illness, in rare cases, complex lethal hematological abnormalities and prominent neuropsychiatric impairment can be observed.

Case
A 56-year old male was brought to ER with labored breathing and hematuria. He was found to have hypovolemic and septic shock. Labs showed severe acidosis, pancytopenia, megaloblastic anemia with hemolysis, PH 6.9, Pco2 41 mmHg, Po2 56 mmHg, Hco3 9 mEq/L, hemoglobin 1.4 g/L, MCV 137 fl, reticulocytes 0.4%, haptoglobin undetectable, Wbc 0.9 K/mcl, platelets 6 K/mcl, INR 3.5, B12 <150 pg/ml, MMA of 900 mcmol/L, homocysteine 61mcmol/L, LDH 1555 iU/L, D dimer 3480 ngFEU/ml, negative intrinsic factor antibodies, gastrin 818 pg/ml. Rt common femoral vein thrombosis and subsegmental pulmonary emboli were diagnosed. EGD showed gastritis with no sprue.

We initiated resuscitation, mechanical ventilation, antibiotics and transfusion of PRBCs, FFPs and platelets. B12 was administered intramuscularly. Anticoagulation was initiated once bleeding stopped and platelet count above 50. Patient improved and was extubated after 1 week with hematologic recovery. Due to aphasia and generalized weakness, he completed an intense rehabilitation program before discharge. Currently, he is following as an outpatient with complete clinical, hematologic recovery and complete resolution of thrombosis.

Discussion
We present rare serious manifestations of B12 deficiency which can be lethal. Besides severe neuropsychiatric changes, it can include severe pancytopenia (occurring in only 5% of patients), hemolysis due to ineffective erythropoiesis (occurring in only 1.5% of patients), and serious thrombosis through homocysteinemia present in B12 deficiency. B12 deficiency is commonly due to malabsorption. Although Intrinsic factor antibodies are frequently elevated, it is negative in 30% of patients with pernicious anemia. Early detection and appropriate treatment is crucial.
A Case of EBV-Induced Scleral Icterus, with VZV and HSV Cross-Reactivity

INTRODUCTION:
Epstein-Barr virus infectious mononucleosis (EBV-IM) traditionally presents with cervical lymphadenopathy, sore throat, and fever, whereas scleral icterus with conjugated hyperbilirubinemia is rare. This is a case of a patient who presented with scleral icterus that was found to have EBV-IM and demonstrated cross-reactivity for other human herpesviruses. This case highlights keeping a broad differential for acute scleral icterus in order to optimize treatment and minimize unnecessary hospitalization time, antibiotics, and procedures.

CASE DESCRIPTION:
A 24-year-old Caucasian immunocompetent male with a history of seborrheic dermatitis presented to the hospital complaining of new onset scleral icterus of one day’s duration. Symptoms began two weeks prior with intractable nausea and vomiting followed by poor oral intake, dark urine, fatigue, nasal congestion, sore throat, nonproductive cough, and cervical lymphadenopathy. He then developed scleral icterus which prompted him to come to the hospital. Patient denied recent travel or known exposures. He endorsed drinking four alcoholic beverages weekly. His last drink was two weeks prior. Physical exam revealed scleral icterus bilaterally, mild jaundice, and a benign abdominal exam.

Patient’s labs revealed leukocytosis (WBC: 20.7 k/mcL), transaminitis (ALP: 178 U/L; ALT: 530 U/L; AST: 682 U/L), and conjugated hyperbilirubinemia (total bilirubin: 11.3 mg/dL; direct bilirubin: 8.7 mg/dL). INR and alpha-1-anti-trypsin were within normal limits. He tested negative for viral hepatitis, HIV, COVID-19, attributable autoimmune markers, and other infectious etiologies. Abdominal ultrasound revealed no hepatobiliary pathology. CT Abdomen/Pelvis showed splenomegaly with retroperitoneal and inguinal lymphadenopathy.

Significant findings included positive heterophile antibody and EBV IgM with normal EBV IgG. He tested false-positive for IgM of VZV and HSV. Patient was diagnosed with acute EBV-IM and discharged home after two days of supportive care.

DISCUSSION:
Of its few published reports, scleral icterus in EBV-IM has mostly been from acalculous cholecystitis or hemolytic anemia, neither of which were present in this case. Documentation indicates high cross-reactivity with human herpesviruses, so HSV and VZV were deemed false-positive as the patient had no attributable history. By recognizing these less commonly discussed features, EBV-IM can be diagnosed quickly which results in decreased costs and unnecessary treatments (i.e., antibiotics or a cholecystectomy in a self-limited disease).
Lung Cancer: Dangers of Cigarette Smoking

Lung cancer is the most common cancer-related death in the United States. Non-small-cell lung cancer (NSCLC) accounts for about 85% of all lung cancer, with squamous cell carcinoma (SCC) being around 25-30%. Around 40% of patients are diagnosed in metastatic stage IV, which occurs via blood and lymphatic vessels. Screening with low dose CT begins in patients aged 50 and either current 20 pack year history or those who have quit within the past 15 years.

53 year old male with medical history of alcohol misuse, tobacco dependence, family history of lung and small intestinal cancer presented with a 3 week history of weakness, decreased appetite and confusion. He was initially stable, had mild hyponatremia and a lactic acidosis. In comparison to his benign labs 3 months prior, there was new hypercalcemia of 14, elevated PTHr, macrocytic anemia 11.9, and mixed cholestatic/hepatocellular pattern. He also had elevated PT/INR/PTT,LDH, CEA, and CA 19-9. He had a polypectomy for a benign tubular adenoma 1 month prior. CT head showed possible ventricular dilation, unlikely NPH. ID added Aspiration pneumonia coverage. CT showed hepatic low density masses, mildly prominent porta hepatitis, paraesophageal lymph nodes, and L3 lytic lesion. Thyroid Ultrasound was negative and CT chest showed bilateral small pleural effusions and lymphadenopathy, suggesting possible lymphoproliferative disorder. His admission included bilateral pulmonary embolisms, spontaneous bacterial peritonitis and decline in mentation. Ultimately, pathology showed NSCLC, favoring SCC. Unfortunately, he expired within 8 days of admission.

NSCLC includes adenocarcinoma, SCC, and large cell carcinoma. SCC most often originates at the tracheobronchial tree, however more recent cases have been diagnosed in the periphery of the lung. Approximately 90% of all lung cancers are caused by tobacco use. Other causes include alcohol use, secondhand smoke exposure, asbestos, radon, arsenic, chromium, nickel, and exposure to radiation therapy. Interestingly, the patient’s mother passed away from lung cancer at age 63 who also had tobacco dependence. Especially with a positive family history, perhaps early CT chest would have changed his outcome. However, it is unclear how the cancer accelerated since his labs 3 months prior to his SCC diagnosis were benign.
From the Throat to the Heart: A Case of Advanced Laryngeal Cancer with Cardiac Metastases

Laryngeal cancers have been noted to represent up to one-third of all head and neck cancers. Typically, these cancers tend to distally metastasize to lung, bone, and liver. The incidence of cardiac metastasis on the other hand is generally considered rare. To our best knowledge, approximately twenty cases of cardiac metastases from laryngeal cancer have been reported in the literature. This case seeks to advance and progress on that limited subject area.

We present a case of a 62-year-old male with a metastatic stage IV laryngeal squamous cell carcinoma, who presented to the hospital with generalized weakness. Imaging from the patient’s record 3 months ago showed a new cardiac mass centered in the inferior wall of the right ventricle, measuring approximately 3.8 x 3.9cm. During this hospitalization, patient underwent a follow-up echocardiogram which demonstrated a progressed 5.2cm x 4.4cm multilobular, fixed mass at the right ventricle, causing compression of apical and mid-septal wall into the left ventricle. Based on the mass's appearance and in light of patient's clinical history, a metastatic tumor from patient's laryngeal carcinoma was suspected. Cardiology team was consulted but given patient's poor baseline function and profound frailty, patient ultimately decided to pursue hospice care.

Given the rarity of cardiac metastasis from laryngeal carcinomas, there is very limited information on the pathophysiology and its management. It is generally thought that there are four ways a cardiac metastasis can occur: direct extension, hematogenously, lymphatically, or via intracardiac spread from the vena cava or pulmonary veins. In this case, we propose that the spread of neoplastic cells from a stage IV laryngeal carcinoma through the cardiac lymphatic network likely contributed to the cause of the right ventricular mass. As described in literature and seen in this case, metastasis to the myocardium and epicardium is usually via lymphatic spread. Lymphatic involvement can be especially problematic as neoplastic cells may obstruct nearby lymph channels, causing edema in the surrounding tissue which in turn, leads to myocardial dysfunction. Treatment is another area where information is scarce and management tends to be directed at the primary laryngeal cancer and symptomatic palliation.
A Rare Case ofSeptic Arthritis Caused by Haemophilus Parainfluenzae in an Immunocompetent Host

Introduction:
Septic arthritis is an infection in the synovial fluid and joint tissues, it is considered a rheumatologic emergency that may cause irreversible local joint destruction and impairment if left untreated. It has also been associated with systemic infection and increased mortality. It is most commonly caused by Staphylococci, Streptococci, Enterobacteriaceae, and Neisseria Gonorrhoeae. Haemophilus parainfluenzae is a Gram-negative bacilli, and a common commensal in the human oral cavity, respiratory, urogenital, and gastrointestinal tracts. We herein present a rare case of septic arthritis of a native joint due to this bacterium in an immunocompetent patient.

Case Presentation:
A 91-year-old caucasian female patient with a past medical history of knee osteoarthritis, atrial fibrillation on apixaban presenting to the hospital with chief complaints of right knee pain and swelling. Four days prior to admission she had dental work. She subsequently developed confusion, low-grade fever, myalgias, dry cough, nausea, and vomiting. Her baseline right knee pain was exacerbated with new decreased mobility, associated with swelling, and erythema. On arrival, vitals were stable, labs showed WBC 11.5, ESR 63, CRP 325.9. X-ray of the knee showed moderate to large right suprapatellar joint effusion, with no acute osseous injury demonstrated, and mild to moderate femorotibial osteoarthritis. Arthrocentesis was performed, synovial fluid was turbid, RBC 47,000mm3, WBC 181,568mm3, calcium pyrophosphate crystals, gram stain demonstrated gram-negative bacilli. Synovial fluid culture grew Haemophilus parainfluenzae. Blood cultures were negative. She was started on vancomycin, cefepime empirically, and later deescalated to ceftriaxone. Her knee remained swollen. Repeat arthrocentesis was performed and the culture was negative. TTE was negative for vegetation. The patient remained afebrile throughout the hospital stay, with a plan to discharge her with Ceftriaxone for four weeks.

Discussion:
Haemophilus parainfluenzae can be responsible for a variety of serious infections in immunocompromised patients such as endocarditis, bacteremia, and pneumonia. Septic arthritis due to H. parainfluenzae is an extremely rare entity, with only 17 case-patients reported in the medical literature. The presence of this pathogen should be suspected in patients presenting with chronic systemic diseases and oral, nasopharyngeal procedures alongside symptoms of septic arthritis.
A Case of Atypical Recurrent Thrombotic Thrombocytopenic Purpura Manifesting as a Large Ischemic Stroke

Introduction:
Thrombotic thrombocytopenic purpura (TTP) is a rare blood disorder with an incident rate of three per million adults per year. Female and black race are at increased risk. TTP resulting in thrombotic microangiopathy (TMA) caused by severely reduced activity of the von Willebrand factor-cleaving protease ADAMTS13. It is characterized by small-vessel platelet-rich thrombi that cause thrombocytopenia, microangiopathic hemolytic anemia, and end organ damage. Neurological manifestations are common, especially subtle changes such as confusion and headache. Transient focal neurological finding, seizure and coma could occur. Large ischemic strokes are very rare. We present a rare case of TTP presented as a large ischemic stroke without the characteristic schistocytes on a blood smear.

Case Presentation:
Patient is a 52 years old African American lady with a past medical history of hypertension, diabetes, tobacco use and stroke. She has previous episode of thrombocytopenia and TMA thought to be due to sepsis and disseminated intravascular accident. Presented this time with right sided upper and lower extremity weakness. Initial blood pressure was 200/110. Brain imaging showed a large subacute infarct in the right middle cerebral artery territory. Labs showed thrombocytopenia, anemia, elevated lactate dehydrogenase, normal haptoglobin, elevated fibrinogen, elevated fibrinogen split products and acute kidney injury. Peripheral smear showed only 1 schistocyte per high power field. Though her presentation was thought to be due to hypertensive emergency given her previous history, ADAMTS13 was checked. Hospital course was complicated by a second multifocal ischemic stroke. Final Diagnosis of thrombotic thrombocytopenic purpura was made when ADAMTS13 activity level came back less than 5%. Patient treated with two sessions of plasmapheresis and a high dose steroid. After treatment patients start to improve with resolution of thrombocytopenia and hemolytic anemia. Work up was negative for secondary cause and TTP regarded as idiopathic.

Conclusion:
TTP may present as a stroke without pathognomonic schistocytes which make the diagnosis challenging especially if other risks for stroke are present. Awareness of such an atypical presentation of TTP may help prevent delay in its diagnosis as Prompt recognition and treatment of this life threatening blood disorder is critical to avoid catastrophic outcomes.
Case Report: Metronidazole-Induced Acute Pancreatitis Presenting with Chest Pain

Introduction:
Medication-induced pancreatitis accounts for less than 5% of acute pancreatitis cases, and metronidazole has been reported to be a cause in multiple case reports with a positive rechallenge. There is an increased risk of acute pancreatitis within 30 days of exposure to oral metronidazole. This patient presented to our facility for a complaint of chest pain and subsequently was found to have acute pancreatitis secondary to metronidazole treatment for bacterial vaginosis.

Case:
A 38 year-old female presented with a chief complaint of chest pain. She stated that for the past 24 hours, she was experiencing constant, sharp, reproducible chest pain, 10 out of 10 at its worst, non-radiating and associated with two episodes of non-bloody, nonbilious vomiting and sweating. Further questioning revealed that the patient began taking metronidazole two days prior to the onset of her symptoms for bacterial vaginosis diagnosis. The acute coronary syndrome workup was negative, prompting further investigation into abdominal pain and vomiting. She was found to have an elevated lipase. The patient denied alcohol and illicit substance use. Laboratory results showed lipase level of 460 IUnits/L, triglycerides level at 94, autoimmune pancreatitis workup was negative.

Discussion/Conclusion:
Although rare, acute pancreatitis is a known side effect of metronidazole. Approximately 0.3-1.4% of the general population that is prescribed a medication with a known potential side effect of acute pancreatitis will subsequently develop acute pancreatitis. While metronidazole is a drug widely used to treat many infections, other reports have shown that a medication rechallenge should not be attempted for risk of a recurrent episode pancreatitis. It is unknown how metronidazole causes pancreatitis, which makes this an important topic to further expound upon to prevent future cases. It is imperative that pancreatitis is a differential in a chest pain and vomiting workup after other etiologies have been excluded.
Amoxicillin-Clavulonate Induced Antibiotic-Associated Hemorrhagic Colitis

Introduction: Antibiotic-associated hemorrhagic colitis (AAHC) is an uncommon diagnosis. Klebsiella oxytoca is thought to play a role, especially in patients receiving oral penicillins. AAHC typically resolves spontaneously after cessation of antibiotics; thus, recognizing the association is important for management.

Case Report: A 77-year-old woman presented to the emergency department with crampy abdomen pain and four days of diarrhea eventually becoming bloody, but no constitutional symptoms. She had no history of intestinal pathology, was not on anticoagulation, and had not traveled. Notably, ten days prior to admission she was prescribed amoxicillin-clavulanate for sinus discomfort and congestion. Additionally, her husband was recently diagnosed with COVID-19. She endorsed a dry cough but no dyspnea and no loss of taste/smell. She had completed only her primary mRNA vaccine series. Admission vital signs were normal, and an abdominal exam was benign. Rectal exam demonstrated large external hemorrhoids without active bleeding but with gross blood in the rectal vault. Laboratory results included a normal white blood cell count and differential, hemoglobin, PT/PTT, and INR. Clostridiodes difficile screen was negative. CT abdomen with contrast demonstrated fluid-filled small and large bowel suggesting possible sequelae of acute infectious or inflammatory enteritis and colitis, enhancement of the walls of the small bowel, and abnormal lumpy bumpy thickening of the walls of the transverse colon, possibly secondary to acute or chronic colitis. Stool culture grew Klebsiella oxytoca. The patient remained on room air, and diarrhea resolved with cessation of antibiotics. She was discharged with plans for outpatient colonoscopy.

Discussion: AAHC has been reported after penicillins, quinolones, and cephalosporins. Postulated mechanisms include allergic reaction, mucosal ischemia, and infection with K.oxytoca, which is not a common commensal and which produces a cytotoxin that induces cell death. COVID-19 has also been reported to cause hemorrhagic colitis but only rarely; given the positive stool culture and history of antibiotic use, AAHC seems more likely in our patient.

Conclusion: AAHC awareness can narrow the differential diagnosis and prevent further harm and unnecessary interventions and treatments.
Acute Aortic Dissection in a Young Female Using Amphetamines

Introduction:
Type B aortic dissections are defined as one in which the tear originates near the descending thoracic aorta. Risk factors for aortic dissection include hypertension, aortic aneurysms, and other lifestyle choices such as smoking and cocaine use. Here, we present a case of a 34-year-old female with a history of adderall use and tobacco use disorder presenting with a type B aortic dissection.

Case description:
A 34-year-old Caucasian female with a past medical history of attention deficit hyperactivity disorder (ADHD) on adderall 30 mg daily for the past one year and tobacco use disorder presents with sudden onset back pain. She suddenly was awaken with shearing, excruciating pain in the middle of back with radiation to the chest. Computed tomography angiography (CTA) at the time showed a type B Aortic dissection located distally to aortic arch all the way down the left common iliac artery. The patient was placed on multiple anti-hypertensive medications and was being monitored in the cardiac intensive care unit. During her clinical course, the patient’s hemoglobin dropped a total of 4 units and, repeat CT scan of the chest showed a large hemorrhagic pleural effusion on the left side. At that time, it was recommended by the patient be transfer to another tertiary care center that can manage these complex aortic dissection cases. The patient states she has been more stressed in the month, leading up to the event. Due to increased stress in her life, she has been smoking more, around 2 packs per day. She has also been taking adderall 30 mg every day for her ADHD. We attributed her dissection to her increased tobacco use combined with adderall as workup for rheumatological and genetic causes were all negative.

Conclusion:
Patients who are taking amphetamines and smoking cigarettes are at increased risk of aortic dissections secondary to hypertension. Patients should be evaluated at regular intervals for the need of amphetamines for their underlying mental health issues, especially if they have a significant social history that puts them at increased risk for other comorbidities.
GBS Tricuspid Valve Endocarditis Following Elective Abortion with Dilation and Curettage

Group B streptococcus (Streptococcus agalactiae) is a major pathogen associated with causing pregnancy related morbidity. However, infective endocarditis, is a rare manifestation of Streptococcus agalactiae causing invasive disease in the setting of recent elective abortion. We describe a case of group B Streptococcus tricuspid valve endocarditis from elective abortion.

A 25-year-old female with a history of asthma presented to the hospital with complaint of dyspnea. Patient states experiencing dyspnea for 1 week with associated hemoptysis and pleuritic chest pain. Patient mentioned undergoing elective dilation and curettage abortion 2 weeks ago. Initial findings performed in the ED included vital instability with tachycardia and tachypnea. Laboratory analysis showed leukocytosis 22 and anemia 7.7. Exam included bibasilar crackles and acute synovitis of bilateral wrists and left ankle. CT angiography of the chest showed bilateral patchy pulmonary infiltrates. Initial diagnosis made was community-acquired pneumonia and septic protocol was initiated. Blood cultures grew Streptococcus Agalactiae and she was continued on antibiotics. Transesophageal echocardiography was obtained showing 20 mm x 15 mm multilobed mass on the tricuspid valve, 7.6 mm x 3 mm mass attached to the larger mass and 3 mm mass attached to the previous mass with a thin stalk with associated mild pericardial effusion. Patient was continued on antibiotics with mild improvement. Rheumatological work-up was performed to evaluate contributory autoimmune process in the setting of inflammatory arthritis, bilateral lung infiltrates with pleural effusions, pericardial effusion, marked anemia with hepatosplenomegaly. Work-up showed elevated inflammatory markers, rheumatoid factor and lupus anticoagulant. Patient was started on steroids with significant improvement in overall status. Steroids were weaned off and antibiotics continued with resolution upon evaluation outpatient.

Our patient presented with group B Streptococcus endocarditis of the tricuspid valve and associated septic pulmonary emboli. Patient also presented with systemic inflammatory disease possibly autoimmune in etiology in the setting of significant response to IV steroids during hospital course. It remains unclear whether the auto inflammatory process was secondary to GBS endocarditis or a separate process in itself. However, this case emphasizes the importance of blood cultures and echocardiography in patients following elective abortion presenting with sepsis criteria.
Heparin-Induced Thrombocytopenia; Refractory and Progression of Thrombosis

Heparin-induced thrombocytopenia Type II is a significant syndrome due to autoantibodies complexed to platelet factor 4 and heparin. These antibodies can cause both arterial and venous thrombosis along with thrombocytopenia.

A middle aged adult female was discharged on Eliquis after Left Lower extremity thrombectomy and drug-eluting stent placement due to May Thurner syndrome. She presented again within 24 hours with fevers, abdominal pain and left thigh pain. She was diagnosed with poorly differentiated ovarian clear cell carcinoma and recurrent left lower extremity deep vein thrombosis. She was started on a Heparin drip to aim for another thrombectomy. She then developed significant thrombocytopenia. Heparin-associated antibody elevated at 1.83 Optical density. Serotonin release assay confirmed heparin-induced thrombocytopenia. Heparin was discontinued and argatroban was started. The patient remained to have persistent thrombocytopenia and further progression of thrombosis as evident by a new right lower extremity DVT. Argatroban initially continued to target a higher PTT level but without response. Two doses of 1 g/kg intravenous immunoglobulin with intravenous Solu-Medrol 125 mg were given followed by a prednisone taper. The patient’s platelet count improved drastically after. The patient was then started on Fondaparinux 7.5mg subcutaneously once daily due evidence of right ventricular thrombus and subsegmental pulmonary emboli.

This is another refractory heparin-induced thrombocytopenia with progression of thrombosis; a rare complication of heparin-induced thrombocytopenia where there are a handful of cases that reported successful treatment with intravenous immunoglobulin. Intravenous immunoglobulin carries a black box warning for thrombosis in hypercoagulable states but there has never been any reports of worsening thrombosis in heparin-induced thrombocytopenia. Also, current literature does not support corticosteroids for treatment of refractory heparin-induced thrombocytopenia. A clinical trial should be warranted to give us answers about the role of IVIG and Corticosteroids in the treatment of refractory heparin-induced thrombocytopenia.
A Full Heart: A Case Of COVID-19 Induced Cardiac Tamponade

Introduction: Cardiac tamponade results from a rapid accumulation of fluid within the pericardium. Viral etiologies are noted to cause pericardial disease. Here we present a case of likely COVID-19 induced pericarditis leading to tamponade.

Case Presentation: A 56-year-old female with a history of anxiety presented due to an episode of presyncope, exacerbated by bending forward. She was hypotensive, and had a lactic acid of 4 despite over 5 L of IV fluid resuscitation. She was unvaccinated, and did test COVID positive but didn’t require oxygen support. She was mentating appropriately. Over the course of the day, she developed new onset chest pressure. EKG showed sinus tachycardia, and was low voltage. A transthoracic echocardiogram was performed urgently, which revealed a moderate to large circumferential pericardial effusion around the RV, LV apex and distal left ventricular wall. There was chamber collapse with RV and RA involvement, suggestive of tamponade physiology. Patient became increasingly tachycardic, and eventually went into PEA arrest. During CPR, pericardiocentesis was performed with 100 cc of fluid removed. ROSC was achieved. Patient was intubated, and had a complicated ICU course with development of oliguric acute kidney injury requiring CRRT, unexplained hypotension requiring maximum vasopressor support, and acute right cerebellar hemorrhage that self resolved without intervention. Blood cultures, sputum culture, fluid studies from the pericardiocentesis, rheumatoid factor, ANCA, ANA, anti-smooth muscle, mixed connective tissue disease markers were all negative. Patient was extubated successfully, and now remains on room air. She currently has critical care myopathy, as well as dysphonia due to prolonged intubation, and is working towards rehabilitation.

Discussion: Cardiac tamponade remains a rare complication of COVID-19 infection. Incidence of cases is not currently available, and data mainly consists of case reports. Much like ours, these reported cases are limited by lack of COVID antibody testing in the pericardial fluid. There does appear to be more of an incidence in middle-aged men, as well as in black and ethnic minority groups. In patients with COVID-19 and a rapid cardiovascular decline, pericarditis and tamponade should be suspected. ECHO can aid in rapid and accurate diagnosis, and drainage results in rapid clinical improvement.
Multiple Pulmonary Artery Mycotic Aneurysms in a Patient with Infective Endocarditis

Introduction: Mycotic pulmonary artery aneurysm is a rare and life-threatening clinical entity. Only a few cases have been reported in the literature. We present a rare case of multiple mycotic aneurysms of the pulmonary artery with associated infective endocarditis.

Case: A 36-year-old female with a medical history notable for seizure disorder and daily Intravenous drug use presented with a complaint of fatigue, chills, fever, and painful swelling of the right knee started about two weeks ago. Vitals on admission were unremarkable other than sinus tachycardia. Physical examination revealed swelling, tenderness of the right knee, and a tricuspid regurgitation murmur. Labs were notable for HB:8.4 gm/dl, and the hepatitis C antibody was reactive. Blood cultures were obtained, and the patient was started on vancomycin and cefepime empirically for infective endocarditis. An echocardiogram showed large, 1.8 cm (W) x 2.4 cm (L), mobile vegetation on the anterior leaflet, and moderate tricuspid regurgitation. On day 3 of admission, the patient complained of worsening shortness of breath, and she started having frank hemoptysis. CTA revealed several segmental pulmonary artery mycotic aneurysm dilatations and multiple cavitary nodules throughout the lungs. Pulmonology evaluated the patient and recommended conservative management of aneurysms.

Discussion: Mycotic aneurysms are uncommon, but when they occur, they show preferential involvement for the aorta, peripheral arteries, and visceral arteries. Mycotic aneurysms arising from the pulmonary arterial system are rare, with only a few cases reported in the literature. IV drug use and bacterial endocarditis are two major known risk factors for developing a mycotic aneurysm. They carry high mortality and morbidity if left untreated. Timely diagnosis is critical to prevent life-threatening complications such as vessel rupture. CTA allows for timely diagnosis. Treatment strategies are diverse and include surgical interventions such as aneurysmectomy or embolization. Small mycotic aneurysms can also be treated conservatively.
Epidural Abscess and Bacteremia Due to Streptococcus gordonii

Introduction:
Streptococcus gordonii is a member of the viridans group of streptococci which may contribute to normal oral flora. It may create biofilms, causing dental plaque and can cause systemic infection including epidural abscess and bacterial endocarditis by entering the bloodstream after oral trauma. We report a rare case of epidural abscess due to S. gordonii.

Case presentation:
A 72-year-old male with co-morbid conditions including diabetes mellitus, lumbago and coronary artery disease with a recent transcatheter aortic valve replacement presents with progressive worsening low back pain, bilateral leg weakness, saddle anesthesia and urinary incontinence. He had a recent lumbar epidural steroid injection without relief. At presentation, he was hemodynamically stable and a soft systolic murmur along the left sternal border was appreciated. He had decreased lower extremity strength bilaterally. An MRI with gadolinium contrast on presentation showed discitis and osteomyelitis of L2-L3 and L4-L5 with epidural abscess. Diagnosis was consistent with epidural abscess with cauda equina syndrome. He underwent emergency laminectomy and evacuation of epidural tissue with aspiration of disc space. Initial blood cultures and intraoperative tissue culture grew penicillin susceptible S. gordonii. He was treated with intravenous ceftriaxone. Transthoracic and transesophageal echocardiogram was negative for any vegetations. Repeat blood culture on day 3 was negative. Patient’s weakness and neurological symptoms improved significantly after the surgery. The patient was discharged to complete a six week course of ceftriaxone.

Discussion:
While Staphylococcus aureus is the leading bacterial pathogen for epidural abscess accounting for about two thirds of the cases, members of the S. sanguinis group of the viridans streptococci can rarely cause bacteremia and epidural abscess following dental procedures. This patient did report a recent dental procedure several months before. Surgical decompression and drainage in addition to systemic antibiotic therapy is the treatment of choice.
Herpes Simplex Virus (HSV) esophagitis are usually found in immunocompromised individuals. We present a case of HSV esophagitis in a COVID 19 positive immunocompetent individual without a history of organ transplant or HIV infection.

Case presentation: The patient is a 57-year-old male with a history of schizophrenia presenting with altered mental status. He had a left facial droop, dysarthria, and tested positive for COVID-19. Brain imaging was normal. Transthoracic echocardiogram disclosed a left ventricular thrombus and heparin drip was started. Patient developed several bloody bowel movements and complaints of odynophagia. Colonoscopy showed several colon polyps, internal hemorrhoids and a rectal fissure. An esophagogastroduodenoscopy showed multiple esophageal and gastric ulcers. Biopsy from the esophageal ulcers was positive for Herpes simplex, while gastric biopsy was negative for H.pylori. HSV-1 IgG was positive. Patient was negative for HIV-1 Ab and HIV-2 Ab, and his CD-4 count was within normal limits. Patient was treated with acyclovir 400 mg three times daily.

Conclusion: COVID 19 may cause gastrointestinal symptoms such as diarrhea but have not been well associated with odynophagia. Additionally, HSV esophagitis is rare in immunocompetent individuals. This patient did not have typical risk factors of HSV esophagitis. The physiological stress caused by COVID-19 infection may have played a role in HSV reactivation. In patients with COVID-19 infections that have odynophagia, assessing for infectious causes of esophagitis may aid in faster diagnosis and prompt treatment.
Epiglottis Secondary to Thermal Injury from Cocaine Inhalation

Introduction: Since the 1970-80s, cocaine use, particularly crack cocaine, has skyrocketed in the United States. It is one of the most commonly used illicit drugs and even casual use of it can result in multiple cardiac and respiratory complications. There are many different ways patients can present to the emergency department after cocaine use, and a more unique presentation is acute epiglottitis after inhalation of crack cocaine. Epiglottis is often sudden in onset and without prompt treatment can progress to life-threatening respiratory obstruction. Patients with epiglottis from cocaine inhalation can have a more atypical presentation and may not exhibit signs of acute infection like fever or leukocytosis. Considering this entity as an emergent situation, healthcare providers should be aware that epiglottis from cocaine inhalation can occur and how it should be treated.

Case Report: The patient is a 49-year-old male with no past medical history who presented to the emergency department with progressively worsening shortness of breath that started 3 days prior. He was evaluated in the ED by ENT and was diagnosed with acute epiglottitis. After admission to the ICU and initial treatment with IV steroids and epinephrine, evaluation by infectious disease did not reveal any infectious cause for epiglottitis nor did the patient exhibit any signs of infection. Furthermore, ENT suggested that the laryngeal swelling was edema without any purulence unlike typical infectious etiology. However, urine drug screen was positive for cocaine. When further questioned, the patient admitted to frequently snorting and smoking crack cocaine as recently as 4 days prior to presenting to the ED. He was treated with empiric antibiotics with ceftriaxone 1 g per day along with IV Decadron and racemic epinephrine. Patient over his 24-hour stay showed resolution of his inflammation and symptoms and was discharged home.

Conclusion: In patients presenting with epiglottis without any identifiable cause, special consideration should be made for other non-infectious causes like cocaine use in order to prevent life-threatening complications and properly treat the patient.
Raising Awareness for Air Thromboemboli; Prevalence is Greater than Previously Predicted

Introduction:
Iatrogenic emboli during endovascular procedures occur more frequently than previously recognized. Greater awareness and postprocedural imaging have a role in identifying and preventing potentially fatal complications.

Case Report:
An active 84-year-old male with atrial fibrillation (CHADsVASc 4), hypertension, hypothyroidism and ischemic cardiomyopathy presented for elective Watchman implantation as an alternative to long-term anticoagulation. During catheterization, an air bolus entered the left atrium through the catheter, embolizing to the right coronary and left anterior descending artery, resulting in ST-segment elevation myocardial infarction, with brief cardiac arrest and restoration of flow after air aspiration. Posturing prompted a code stroke including evaluation by Neurology and Neuroendovascular. Timely hospital transfer for hyperbarics (i.e. within 6 hours of event) could not be arranged, prompting Trendelenburg positioning and 100% oxygen supplementation. MRI brain depicted severe anoxic brain injury related to diffuse cerebral microemboli without large vessel occlusion or stenosis. Without improvement in neurologic function off sedation and minimal potential of meaningful recovery, the family opted for compassionate extubation.

Discussion:
Ranging from central lines to pacemaker placement, endovascular procedures pose risk of iatrogenic embolic phenomena including hydrophilic polymers, calcified debris, displaced tissue, air, and foreign materials. Transcatheter left atrial appendage closures have particularly higher rates of air thromboembolism, with retrospective analysis proving acute ischemic injury is under-recognized and possibly fatal. For example, coronary air embolism has an incidence of 0.1% to 0.3% in cardiac catheterization. Cerebral air microemboli can cause global ischemia, edema, and petechial hemorrhages, which is more detrimental compared to phenomena like calcified debris, which result in classic strokes. Hickey et al explain prompt recognition of adverse events is equally important as reversal maneuvers are timely in cerebral air embolism. These rare but preventable events occur when catheters are inadequately prepared or deployed, validating proper device use during endovascular procedures is prudent in minimizing fatal outcomes. The occurrence of both iatrogenic embolic stroke and cardiac arrest in this case demonstrate the need for greater risk awareness and the low threshold for post-procedural imaging.
Not Just Certain, but Double-Positive You Haven’t Seen This Yet; Anti-GBM and ANCA Positive RPGN

Introduction
Rapidly Progressive Glomerulonephritis is a syndrome of progressive kidney function loss with classic morphological findings on histology. We present a rare case of late-onset RPGN secondary to double-positive anti-GBM/Antineutrophilic Cytoplasmic antibody disease.

Case Presentation
An 83-year-old female presents to the hospital by instruction of PCP after incidental discovery of an 8-fold increase in creatinine. Urine output and mentation significantly declined over the course of one week. Nephrology workup included testing for C3, C4, ANA, C-ANCA, P-ANCA, and anti-GBM; which were positive for the latter two. Subsequent renal biopsy demonstrated fibrocellular and cellular crescents encompassing 100% of her non-sclerotic glomeruli, with linear glomerular basement membrane staining of IgG. Cyclophosphamide, high-dose steroids, plasmapheresis, and eventually hemodialysis were initiated. While prognosis is historically guarded, we continue providing the patient and her family a fighting chance with continued dialysis.

Discussion
Anti-GBM disease has an incidence of less than 2 cases per million per year. Between 10-50% of patients with anti-GBM are also positive for P-ANCA, while only 10% of P-ANCA patients test positive for anti-GBM. While pulmonary involvement was not present in our case, alveolar hemorrhaging is not an uncommon finding with this disease. Prognosis drastically changes depending on percentage of glomerular crescent formation, with more favorable outcomes involving only 50% of glomeruli. While double-positive patients typically have worse renal biopsy results, ironically, they tend to recover more often than single-positive cases. Unfortunately, there are no improved mortality statistics in either group. 68% of patients are dialysis-dependent upon diagnosis, and none have achieved full renal recovery despite optimal treatment.

Conclusion
Anti-GBM/ANCA positive disease is thankfully not one commonly seen. Diagnosis is made through detection of anti-GBM antibodies in the serum, or deposition in the tissue. Renal biopsy can aid in prognosis, with more favorable outcomes presenting with <60% glomerular involvement. While no specific treatment represents the gold standard; Cyclophosphamide, plasmapheresis, steroids, and dialysis have given these patients years, when the disease wants only mere days.
Legionella in the Setting of TNF-Alpha Inhibitor Use

Legionella bacteria are gram-negative bacilli, transmitted via inhalation aerosols from contaminated water or soil. Legionnaires’ disease is historically known to have a seasonality of infection most common in late summer and early autumn.

Golimumab is a human monoclonal tumor necrosis factor blocking agent which is approved for use in rheumatoid arthritis as well as several other diseases. There are several cases reported of TNF-alpha inhibitor use in the setting of Legionnaires’ disease, with most of these involving infliximab, adalimumab, and etanercept.

The patient is an elderly female who presented to the ED in the winter with chief complaint of generalized weakness. Symptoms included shortness of breath, cough with sputum production and fever for one week prior to admission. Ten days prior to this admission, the patient was found to be Covid positive with a home test; the patient was fully vaccinated including a booster against Covid-19. Past medical history is significant for COPD, hyperlipidemia, CAD with stent, CKD, HTN, gout, and rheumatoid arthritis. Her rheumatoid arthritis was initially treated with methotrexate for approximately 3 months in which she achieved no response, followed by etanercept which was not affordable, and most recently hydroxychloroquine, prednisone and one dose of golimumab, which she received one month prior to this admission. During admission, given cough, fever, and right-sided consolidation on CT scan, patient was started on antibiotics for community acquired pneumonia. On day 2, patient was found to have positive urine antigen for legionella pneumonia. Patient received ceftriaxone and azithromycin for 5 days followed by levofoxacin for an additional 5 days. The patient was ultimately discharged to an inpatient rehabilitation program.

Golimumab works by interfering with endogenous TNF-alpha activity. This interference results in a reduced ability for leukocyte infiltration and ultimately a diminished activation of neutrophils and eosinophils, thus increasing the risk for serious infection. In patients on TNF-alpha inhibitors such as golimumab, a diagnosis of legionella should be considered, especially during the winter season when rates of legionella tend to be lower. In this case where the seasonality of acquiring legionella is uncommon, host risk-factors increasing susceptibility to this pathogen should be considered.
A Case of Apical Hypertrophic Cardiomyopathy

Apical hypertrophic cardiomyopathy (ApHCM) is defined as left ventricular hypertrophy predominantly involving the apex with a non-obstructive physiology. First described in 1976 by Sakamoto et al, 15-25% of Asian patients with HCM have ApHCM, but the prevalence in non-Asian population remains low at 1-3%. The inheritance is autosomal dominant, however, sporadic forms of acquired mutations in the sarcomere protein gene exists. Symptoms are more common in the fourth decade of life with male predominance. Most patients remain asymptomatic while others can develop arrhythmias (atrial or ventricular), angina or myocardial infarction. 51-year-old African American male with a medical history of hypertension presented to the hospital due to palpitations. Upon presentation, the patient was afebrile, blood pressure was 138/88 mmHg, heart rate was 150 beats per minute (bpm), respiratory rate was 20 and oxygen saturation was 98% on room air. Physical examination revealed an irregularly irregular rhythm. High sensitivity cardiac troponin T and TSH level were within normal limits. Electrocardiogram (EKG) showed atrial fibrillation with rapid ventricular rate of 143bpm, left ventricular hypertrophy and deep T wave inversions in the left precordial leads. Transthoracic echocardiogram (TTE) was completed to evaluate for potential valvular or other structural heart disease, and it demonstrated marked hypertrophy of the left apical ventricular apex along with spade-like configuration of the left ventricular cavity at the end of diastole. The echocardiographic finding of spade-like configuration of the left ventricular cavity along with the deep T wave inversions noticed on the EKG were characteristic of ApHCM also known as Yamaguchi syndrome. After management of the acute episode with diltiazem infusion, the patient was discharged two days later on metoprolol for rate control and apixaban for thromboembolic risk reduction. Recognizing and diagnosing this disease is key because recent data suggests that ApHCM carries a high mortality risk, approaching that of classic HCM. Furthermore, our case highlights that Yamaguchi syndrome must be considered in the differential diagnosis, when evaluating patients with T wave changes that may be mistaken as ischemia.
**HSV-2 Induced Chronic Inflammatory Demyelinating Polyneuropathy**

**Introduction**
Herpes simplex virus type 2 (HSV-2) is a leading cause of genital herpes in addition to neonatal encephalitis and meningitis. Rarely, HSV-2 can induce immune-mediated disorders of the peripheral nervous system such as chronic inflammatory demyelinating polyneuropathy (CIDP). CIDP is an acquired autoimmune disorder with progressive weakness.

**Case**
We report the case of a 56-year-old woman with a lengthy history of rheumatoid arthritis on immunosuppression who presented with worsening lower extremity weakness and pain for the last eight weeks. The patient was unable to stand and walk and had difficulty performing daily activities. Her review of systems was remarkable for significant weight gain of approximately 10 pounds and leg swelling. Her medications included Sarilumab for 2 years for rheumatologic disease. Physical examination was remarkable for bilaterally and symmetrically decreased motor strength in the lower extremities with areflexia and absent plantar reflexes. Blood tests showed normal levels of TSH, B12, folate, ESR, CRP and CPK. Cervical, thoracic, and lumbar MRI scans revealed no significant stenosis or neural compression. Brain CT scan revealed no acute processes. However, over the following days the patient became increasingly confused and was noted to have an ascending weakness to the upper extremities. Lumbar puncture showed no white blood count and mildly elevated protein (71). Repeat blood tests revealed mildly elevated ESR (50) and CRP (22). High-dose steroids (1 gm iv Solu-Medrol) was started. Autoimmune workup including RF and ANA was negative. With worsening encephalopathy, IV acyclovir was empirically initiated for possible encephalitis and steroids were discontinued. Meanwhile, brain MRI did not show any abnormalities. Subsequently, a polymerase chain reaction of CSF detected HSV-2. The suspected diagnosis was CIDP and IVIG 0.4mg/kg/d was started. The patient’s confusion and motor strength slowly improved and was transferred to subacute rehabilitation.

**Conclusion**
This case highlights the association between HSV-2 and CIDP, particularly in the setting of a patient with longstanding immunological disease. Awareness of this association, detection of HSV-2 through PCR of CSF and timely treatment with acyclovir may prevent distressing neurological effects.
Cannabis and Acute Coronary Syndrome: A Case Report Exploring Potential Links

Cannabis, or marijuana, is a commonly abused drug in the United States. Given its rapidly increasing use, it is paramount that physicians understand the risks of marijuana use that may impact patient safety, particularly its effects on the cardiovascular system.

A 43-year-old male with no significant past medical history presented to the emergency room with a chief complaint of chest pain. The patient reported intense substernal chest pain, which awakened him from sleep. He described the pain as a heavy sensation in his chest, rating the pain as 10 out of 10 in severity. The patient reported he had smoked marijuana prior to going to sleep. His social history was significant for smoking marijuana several times a day for the past fifteen years, occasional cigar smoking, and he was a social alcohol user. A twelve-lead electrocardiogram revealed ST-segment depression in leads V5 and V6 and T-wave inversion in leads III and aVF. Initial troponin was mildly elevated at 0.22 ng/mL. However, it continued to rise and ultimately peaked at 15.86 ng/mL. Urine toxicology panel was positive for cannabinoids only. The patient was treated as non-ST-elevation myocardial infarction and subsequently underwent coronary angiography and percutaneous coronary intervention. He was found to have 99% stenosis of the left circumflex artery. A drug-eluting stent was placed in the first OM as it was suspected to be the culprit lesion. He was initiated on dual-anti-platelet therapy consisting of Aspirin 81 mg daily and prasugrel 10 mg daily for an anticipated 12 months and discharged home.

There have been several seminal studies evaluating the impact of cannabis on cardiovascular health. Similarly, our patient had also been smoking marijuana shortly prior to the onset of his myocardial infarction. This case emphasizes that marijuana-induced acute coronary syndrome must be considered a cause of myocardial infarction, especially in young adults. Cannabis utilization continues to increase in both young and old adults. Since more people are consuming marijuana daily, it is imperative that we increase awareness about its detrimental effects.
Merkel Cell Carcinoma While on Rituximab - Is it a Coincidence?

A 79-year old woman with history of relapsed Stage II, grade III follicular lymphoma was treated with four cycles of rituximab and bendamustine with an excellent response. She was subsequently placed on maintenance rituximab infusions every eight weeks. Six months later, she presented to the dermatology clinic with a newly raised erythematous papule on her left cheek. Excisional biopsy confirmed a diagnosis of Merkel cell carcinoma which was resected with wide margins.

Merkel cell carcinoma (MCC) is a rare aggressive skin malignancy, with a dramatic increase in incidence in the past few decades. It grows and metastasizes rapidly and diagnosis in early stages is often missed. Almost half of the patients with MCC have another active malignancy, compromising their immune system. Most commonly reported were cutaneous neoplasms, followed by leukemia and lymphomas. MCC is most common in caucasion population. Most affected patients are above 50 years old. Sun exposure is a major risk factor responsible for mutations created by ultraviolet-A radiation. In 2008, a novel discovery of Merkel cell polymavirus (MCPyV) was detected in most patients with MCC that plays a role in oncogenicity. Most primary lesions are aysmtomatic. Pathology findings of tumor size, depth, and lymphovascular invasion are important prognosticating factors. Further imaging modalities (CT, MRI and FDG-PET scans) are essential for staging.

Treatment depends on the diagnosed stage. It varies from wide excision of the primary lesion followed by adjuvant radiation in early stages, to systemic chemo and immunotherapy.

There have been two reported cases in literature about significant progression of Merkel cell carcinoma in patients concomitantly receiving rituximab as part of treatment for another malignancy. Which raises questions for future investigation whether there is a direct association between rituximab use specifically, and the rapid growth of MCC, or is it due to an immunocompromised state in general.
Small Cell Carcinoma of the Anal Canal: A Rare Entity

Neoplasm of anal canal are <5% of the malignancy of gastrointestinal tract. Majority of the cancers are squamous cell and adenocarcinoma. Primary small cell carcinoma of the anal canal is a very rare diagnosis with only handful of cases reported in literature.

66-year-old female with multiple co-morbidities presented with rectal pain. She had initially noticed hemorrhoids six months prior to presentation. CT abdomen and pelvis showed bilateral inguinal lymphadenopathy and diverticulosis. Patient then had colonoscopy which showed mass in the anal canal, initial biopsy showed squamous cell carcinoma with basaloid features. Lymph node biopsy was also positive for metastatic cancer likely anal in origin. PET scan showed metastatic lesion in the lumbar vertebra. Review of slides was done before starting chemotherapy as component of small cell carcinoma was also seen. Due to diagnostic dilemma, patient was initially started on Carboplatin and Paclitaxel first cycle as it is effective against both squamous and small cell carcinoma. Repeat evaluation of the slides confirmed small cell cancer as the diagnosis and chemotherapy was changed to carboplatin and etoposide. Patient received 3 cycles of the carboplatin and etoposide after which atezolizumab was added. She received total of 6 cycles of the chemotherapy. Repeat PET scan showed partial favorable response.

Due to rarity of the disease, there was significant delay in diagnosis of the cancer. Diagnosis of small cell carcinoma in the anal canal relies on neuroendocrine differentiation. There is no formal evidence-based recommendation regarding treatment. They are considered equivalent to pulmonary small cell carcinoma and treated in similar manner.
A Rare Case of Segmental Arterial Mediolysis

Segmental arterial mediolysis (SAM) is a rare vasculopathy of unknown etiology characterized by disruption of the arterial layer, which can result in vessel dissection, hemorrhage and ischemia. Literature review has demonstrated a varied presentation ranging from benign abdominal pain to life threatening hemorrhage. We report a case of 58 year old female with past medical history of deep vein thrombosis/pulmonary embolism secondary to Protein C and S deficiency on Coumadin who presented with right flank pain for 3 days. Complete blood count and complete metabolic panel were within normal limits. CT scan of abdomen and MRI showed ischemia of segment 5 of liver secondary to intrahepatic arterial pseudo-aneurysm measuring 3cm with compression of anterior branch of right portal vein. Infectious workup and Antinuclear antibody(ANA) were negative. Antineutrophil cytoplasmic antibody(ANCA), complement level, myeloperoxidase antibody and PR3 antibody were negative. Hepatitis B was negative. ESR was 73 and CRP was 224. SAM was diagnosed on the basis of the patient’s clinical history, clinical examination, CT angiogram findings and ruling out of other etiology. She underwent embolization of hepatic pseudo aneurysm that improved the pain. After 5 days, she again reports abdominal pain. Repeat CT scan of abdomen showed large perihepatic hematoma with enhancing structure in right hepatic lobe near prior embolization site concerning for re-bleeding from known pseudo aneurysm. So, reembolization of gastroduodenal artery and hepatic artery was done. Despite reembolization, she developed gall bladder necrosis with rupture and underwent exploratory laparotomy with cholecystectomy. She developed worsening transaminitis. Liver transplant workup was done. She ultimately developed sepsis, and unfortunately we lost our patient.

Though segmental arterial mediolysis is rare, it is an important cause of unexplained vascular lesion. It should be considered when aneurysms, stenosis and occlusions are identified in medium and large vessel, especially when limited to one anatomical location. When segmental arterial mediolysis is suspected, multidisciplinary approach should be pursued.
Primary Carnitine Deficiency Induced Ventricular Fibrillation

Background: Children with primary carnitine deficiency (PCD) frequently develop cardiomyopathy; however it is not commonly seen in adults. Arrhythmias are less common and include forms of long QT-syndrome and ventricular fibrillation. ECG findings commonly involve peaked T-waves and short QT intervals that may resolve with supplementation of L-carnitine.

Case presentation: An 18-year-old female with a family history of sudden cardiac death (SCD) presented to the emergency department following a witnessed out-of-hospital cardiac arrest. She was initially noted to be in ventricular fibrillation (VF) and was subsequently defibrillated twice on presentation. Her rhythm converted to supraventricular tachycardia at which time adenosine 6 mg IV was administered twice with reversion to sinus rhythm. ECG on presentation demonstrated peaked T waves and short QT interval. She was admitted to the cardiac intensive care unit for targeted temperature management. Carnitine level on admission was subtherapeutic at 1 nmol/mL (normal: 34-78 nmol/mL) due to suboptimal dosing at home. She was loaded with L-carnitine 12 g/day with repeat levels improving to 41 nmol/mL. Echocardiogram revealed an ejection fraction (EF) of 35% with a dilated right ventricle and global left ventricular hypokinesis. The patient later regained consciousness and was transferred out of the cardiac intensive care unit. Repeat echocardiogram two weeks later revealed no significant change in EF. Her hospital course was complicated by methicillin-sensitive staphylococcus aureus pneumonia, for which ICD placement was delayed until completion of antibiotics. She was discharged home in stable condition with plans for 4 weeks of IV antibiotic therapy and close follow-up.

Discussion: Primary carnitine deficiency can present as ventricular fibrillation with or without pre-existing cardiomyopathy in adults. Strategies for primary prevention of SCD should be considered in these patients, especially in those with a known family history of SCD.
Diabetic Ketoacidosis Precipitated by COVID-19 Induced Acute Pancreatitis

Gastrointestinal manifestations of COVID-19 infection are increasingly being recognized, such as nausea, vomiting, and diarrhea. Research into the pathophysiology is ongoing. I present a case of a 52-year-old female without a history of diabetes who presented to the emergency room with progressive lethargy and altered mental status. Her test results were consistent with diabetic ketoacidosis with glycemia 1440 mg/dL, venous pH 7.23, beta hydroxybutyrate of 7.53 mmol/L, bicarbonate 14 mmol/L, and anion gap 32. COVID-19 (SARS-CoV-2) testing by nucleic acid amplification was positive on admission. She was admitted to the intensive care unit and started on an insulin drip. Her severe abdominal pain led to the diagnosis of acute pancreatitis with lipase >1200 U/L and computed tomography of the abdomen showed inflammatory changes around the head of the pancreas and second portion of the duodenum. Workup for the cause of her pancreatitis was unrevealing. A right upper quadrant ultrasound negative for evidence of cholelithiasis, common bile duct dilation or liver abnormalities. She denied alcohol and drug use, which was confirmed with a negative blood alcohol level and urine drug screen on admission. Furthermore, she was not taking any medications prior to arrival, specifically those known to cause pancreatitis. She was diagnosed with diabetes mellitus during this admission and DKA was the presenting situation precipitated by COVID-19 pancreatitis. She was treated with intravenous fluids and supportive care. The remainder of her hospital stay was unremarkable. She was discharged home with an insulin regimen, and her HbA1C two months later was 8.3%. Follow-up CT of the abdomen one month after discharge showed no abnormalities. Literature review revealed a study in China reported an elevated incidence of pancreatic injury with few exhibiting hyperglycemia, and without a diagnosis of acute pancreatitis (1). There are few cases of acute pancreatitis that are classified as being caused by COVID-19 infection (2). However, none of these reported a complication of DKA. This case demonstrates pancreatitis and DKA as rare, yet possible, complications of COVID-19 and emphasizes the importance of thorough history-taking and diagnostic evaluation.
A 45-year-old male presented to the ED with a complaint of right foot pain and nonhealing wounds. Patient was in sepsis, secondary to cellulitis of his right foot. A non-healing wound in his right 4th toe space was cultured to grow MRSA. In addition, multiple wounds were scattered on his bilateral upper and lower extremities, diffusely around the body, upper back, scalp and one in the perineal area. These erythematous ulcerations appeared gradually over the past month, were different sizes, usually circular in shape, and some were necrotic. Patient denied fevers, pruritis and joint pains. The distribution of the excoriations were peculiar, as they were in readily accessible areas to the patient. Infectious and autoimmune work-up came back negative. On further discussion, the patient claimed that picking at these lesions helped relieve the discomfort. He mentioned that he occasionally picks at his skin after getting a pruritic sensation and has subconsciously picked at his skin since childhood. After the scabs form, the patient felt the need to “relieve the pressure” by cracking the scabs. His medical history consists of generalized anxiety disorder, recurrent major depressive disorder, insomnia and hypertension. A diagnosis of dermatillomania was made. Treatment was initiated with N-acetylcysteine, citalopram and a referral for Habit Reversal and Cognitive Behavioral Therapy. Patient was placed in mittens, and the wounds continued healing during hospitalization. The patient was also advised to closely follow-up with his psychiatrist for anxiety management, and further evaluation for possible underlying cluster C personality disorder. Dermatillomania remains continuously difficult to treat and needs a personalized, multifaceted approach.

This case illustrates a four-decade undiagnosed dermatillomania resulting in a sepsis, and the importance of a complete history. Recognizing this uncommon pathological excoriation disorder is critical to initiate appropriate therapy and prevent severe complications such as cellulitis and bacteremia.
Nebulized Hydrogen Peroxide for COVID Pneumonia: Friend or Foe?

Hydrogen peroxide is widely used as a disinfectant and is an effective tool for disinfection of inanimate surfaces. With the recent increase in alternative medical therapies, a proposal has been made to administer nebulized hydrogen peroxide as a treatment for COVID pneumonia. This case will discuss the detrimental effects of such an alternative therapy and explore the mechanism of action behind this treatment.

A 62-year-old man, who was unvaccinated against COVID, presented to the hospital with one week of shortness of breath. Preceding admission, the patient reasoned that he had COVID due to recent exposure. He treated himself with ivermectin, erythromycin and nebulized food-grade hydrogen peroxide. Despite these remedies, his shortness of breath progressed, and he was admitted to our institution. He was diagnosed with COVID pneumonia and initially required 6L of supplemental oxygen. Despite treatment per protocol with high-dose steroids and tocilizumab, his clinical status continued to worsen – two days after admission he was on 15L and one week later he was BiPAP-dependent. His clinical course was complicated by sub-massive pulmonary embolism diagnosed by CT and he was treated with intra-arterial tissue plasminogen activator. CT thorax was repeated multiple times, showing rapidly progressing pulmonary fibrosis, coalescing multilevel blebs, and worsening acute respiratory distress syndrome (ARDS). Eventually, the patient was intubated, suffered cardiac arrest twice, and his family withdrew care after his second arrest.

This case highlights the potential risks of using alternative medical treatments, such as nebulized hydrogen peroxide. Hydrogen peroxide is physiologically produced by neutrophils in response to oxidative stress that causes auto-oxidative injury and damage to adjacent cells, which is an important molecular mechanism causing pulmonary fibrosis. Hydrogen peroxide also increases endothelial permeability, which is hallmark of ARDS – one of the major complications of COVID-19. It stands to reason that inhaling such a substance would cause further injury to epithelial cells and cause a cascade of inflammatory response within the tissue of the lung. This case emphasizes the fact that alternative therapies are not without risk. Considering the recent reluctance to trust evidence-based medicine, healthcare providers should familiarize themselves with these therapies and their potential adverse effects.
Granulomatosis with polyangiitis (GPA) is a rare vasculitis that mainly affects the ears, nose, throat, lungs and kidneys. This disease is exceptionally rare with the prevalence of 3 people diagnosed per 100,000 in the United States. GPA is associated with significant morbidity and mortality likely due to irreversible organ damage and side effects of intensive/extended use of corticosteroids and immunosuppressive agents.

A 58-year-old Caucasian woman presented to the hospital due to a one-week history of nausea and low-grade fevers. She had no significant past medical history as she had not seen a physician in many years. On admission she was found to be in acute renal failure with a creatinine of 11. Chest x-ray on admission was significant for bilateral opacities with bilateral small pleural effusion. She was eventually initiated on dialysis for her renal failure and antibiotics for presumed multifocal pneumonia. She was also found to have new onset hypertension and was started on 3 antihypertensive medications. Multiple screening tests were ordered given her acute renal failure and the patient was positive for C-ANCA. After one week of hospitalization, she suddenly went into hemorrhagic shock requiring transfer to the ICU for mechanical ventilation and vasopressor support. Despite adequate replacement of blood products, she continued to have acute blood loss anemia. Bronchoscopy done at bedside revealed alveolar hemorrhages. She was stabilized enough to obtain a CT chest/abdomen/pelvis which revealed a large 11 x 15 x 17 cm splenic hematoma. Interventional radiology performed an urgent splenic artery embolization which led to 60% of spleen devascularization. Her shock state did improve, and she no longer required vasopressor support. She was initiated on high-dose steroids and rituximab induction therapy for a presumptive diagnosis of granulomatosis with polyangiitis given +c-ANCA with renal and pulmonary involvement.

It is believed that splenic involvement in GPA is underreported and not uncommon. Few cases have been reported with patients ultimately requiring splenectomy. However, after initiating appropriate immunosuppressive treatment, outcomes tend to be favorable.
Pulmonary Embolism Despite Anticoagulation in COVID-19

It is widely known that COVID-19 infection causes a hypercoagulable state in patients. Current guidelines recommend initiating DVT prophylaxis for all hospitalized COVID-19 infected patients not already on anticoagulation for other disease states or where it is contraindicated. Presented is a case where a patient developed pulmonary emboli despite being on prophylactic anticoagulation.

An unvaccinated 59-year old male with no known past medical history presented to the ED with complaints of progressive shortness of breath. He was found to be COVID positive and admitted for acute hypoxemic respiratory failure and placed on 45L Optiflow. Based on the acuity of his respiratory failure and an elevated D-dimer, a CT was ordered to rule out pulmonary embolism; which at that time was negative. Duplex of his lower extremities was also negative for DVT. He was also found to have an NSTEMI for which he was placed on therapeutic enoxaparin for 48 hours and switched to prophylaxis dosing after. Consideration for superimposed bacteria pneumonia led to empiric treatment with cefepime and vancomycin. Despite treatment based on current COVID guidelines, the patient would continually require 45-50L O2 supplementation with no improvement. Two weeks after admission, his dyspnea worsened and he was moved to the ICU proper for further management. CT was repeated which now showed ARDS and a sub-massive saddle pulmonary embolism despite being on prophylactic anticoagulation. He was started on tPA and full-dose heparin. He continued to worsen, requiring intubation and vasopressor support. Despite being on four vasopressors and maximum ventilator settings, he did not recover. Eventually, the patient became progressively hypotensive and bradycardic, developing into PEA and unfortunately expiring shortly after.

This case demonstrates the hypercoagulability state that COVID inflicts upon patients may not be covered by our current anticoagulation/prophylaxis protocols. Further research and epidemiological studies need to be done to further refine our treatment guidelines. This case is also a reminder that, while we have learned much over the past two years of fighting COVID, we must continue to be vigilant and have a high suspicion for pulmonary emboli and DVT in patients receiving anticoagulation.
Acquired Angioedema as a Complication of Systemic Lupus Erythematosus

A 27 year old female with a past medical history significant for Systemic Lupus Erythematosus and Rheumatoid Arthritis presented for an episode of lip swelling, tongue swelling, and dyspnea in August of 2021. She stated that she had attended a bridal shower earlier in the day and eaten tacos, which she has tolerated in the past without any issue. After she got home she felt tired, took some Benadryl, and laid down for a nap.

Approximately one hour later she woke up with acute dyspnea, upper and lower lip swelling, tongue swelling, a globus sensation in her throat, and wheezing. She was taken to the emergency department where she was administered Epinephrine, Methylprednisolone, Benadryl, and Famotidine. Her lip swelling improved but did not completely resolve with these medications. She had been taking lisinopril, which was held from that point on. She reported prior milder episodes occurring since September of 2017. The episodes included bilateral eye swelling and mild lip swelling. They were noted to last for approximately 1-2 days at a time and to occur once every three months. The timeline and further history did not suggest an allergic or medication induced cause. She continued to experience recurrent episodes of lip swelling despite treatment with Loratadine 10mg BID PO and Famotidine 20mg BID PO for a three week period. Lab work was significant for low total complement of 35 U/mL (normal 42-95), low C4 of 6.8 mg/dL (normal 10-53), a normal C1 Esterase Inhibitor quantitative level of 27 mg/dL (normal 21-39), a normal C1 Esterase Inhibitor Function of 93% (normal > or + 68%), a low C1q of 3.6 mg/dL (normal 5 - 8.6), and a high C1Q Binding Assay of 6.4 ugEq/mL (normal 0-3.9) suggesting Acquired Angioedema. The decreased C1q distinguishes Hereditary Angioedema from Acquired Angioedema and the high C1q binding assay indicates Type 2 Acquired Angioedema secondary to autoantibodies against C1-INH. She was kept on prednisone 20mg daily for her SLE with a plan to initiate Lanadelumab subcutaneous injections every two weeks for prevention of further attacks of angioedema.
Guillain–Barré Syndrome Secondary to SAR-CoV-2: Two Patients with Two Different Outcomes

Guillain–Barré syndrome (GBS) secondary to severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), has been reported sparingly since the onset of the COVID pandemic. This acute, frequently severe, polyradiculopathy is autoimmune in nature. The onset of symptoms, CSF findings, and outcomes vary widely. We had the privilege of treating two patients that had two different outcomes.

Our first patient (Mr. A) was an unvaccinated relatively healthy 55-year-old man who came in two days after the onset of his symptoms. He initially had a cough, fever, and fatigue. His legs started to get weak the next night and he fell in the kitchen. He crawled to the couch and EMS was called. His weakness progressed rapidly. He couldn’t move his arms and started slurring his speech. He needed to be intubated shortly after his admittance. A lumbar puncture did not show albuminocytologic dissociation, but with the clinical presentation, he was still treated with IVIg. He was also treated with high doses of Solu-Medrol. He progressively improved clinically and 5 days after being admitted he walked out of the hospital.

Our second patient (Mr. H) was a vaccinated 75-year-old man, who came in two weeks after the onset of his symptoms, which consisted of a non-productive cough and diarrhea. He had comorbidities notable for hypothyroidism, oropharyngeal cancer status post chemoradiotherapy as early as 3 months prior. He progressively weakened after his symptoms started, stating it took him longer to get out of bed. He came in after collapsing in the bathroom unable to get himself up. During Mr. H's inpatient course a lumbar puncture did show albuminocytologic dissociation. Neurology promptly started IVIg. His lower extremity weakness at the hips and knees waxed and waned throughout his stay, but there was a gradual improvement in terms of strength. He was seen by a physical medicine and rehabilitation physician, who suggested inpatient rehab. The variability in GBS needs to be reported, so physicians do not miss the diagnosis. Two patients that came into the hospital for weakness secondary to COVID-19-associated Guillain-Barre syndrome had different onset of symptoms, CSF findings, and outcomes.
Periampullary Lipomas: A Rare but Important Differential for Cholangitis in the Elderly

Periampullary lipomas are rare, benign fatty tumors found in the small intestine. The prevalence in the population is unknown but they typically occur in men in their 7th or 8th decade of life. They are mostly asymptomatic but can appear on imaging as smooth well-marginated masses. These can rarely cause complications such as cholangitis as seen in this case.

An 83-year-old woman with a past surgical history of laparoscopic cholecystectomy presented to our institution for epigastric abdominal pain, nausea and vomiting after eating. She also reported dark amber urine for the last week. She is found to have a leukocytosis, mild hyperbilirubinemia and moderate transaminitis. A CT of the abdomen and pelvis showed 2 gallstones in the distal common bile duct distal to the ampulla and dilation of the common bile duct up to 1.7 cm. She was started on ceftriaxone and flagyl and she was scheduled to undergo an ERCP the following day. Initial ERCP showed an obstruction of the distal common bile duct with a lipoma situated just superior to the papilla as well as a small periampullary diverticulum. A second lipoma was also seen in the second portion of the duodenum, partially obscuring the en face view of the ampulla. The pancreatic duct was successfully cannulated and was normal. The patient subsequently was transferred to an outside facility for an ERCP with unroofing of the lipoma, sphincterotomy and stone extraction. She did well post procedure and was discharged in stable condition.

Although periampullary lipomas are relatively rare, it is important to recognize in patients with signs and symptoms of cholangitis, especially in this age group. This case shows the importance of complete history taking and broad differential diagnoses in order to arrive at the correct diagnosis and provide appropriate intervention and treatment.
Appendiceal Mucinous Neoplasm Masquerading as Recurrent Appendicitis

Appendiceal tumors are extremely rare with an incidence of 1-2 cases per 1 million people and account for 0.4%–1% of all gastrointestinal malignancies in the U.S. Histologically speaking, neuroendocrine tumors make up the majority of all appendiceal tumors at 65% followed by adenocarcinomas of the appendix which include mucinous, goblet and signet ring-cell tumors which account for 20%. Initial presenting symptoms can vary and are often nonspecific but many patients will present with RLQ abdominal pain similar to appendicitis. Appendiceal tumors are most often seen in the 6th decade of life and are occasionally identified incidentally on histology after an appendectomy.

A 77-year-old man with a past medical history significant for HTN, HLD and prediabetes presented to the hospital for RLQ abdominal pain with associated nausea and vomiting. A CT abdomen showed acute appendicitis with surrounding inflammation and small fluid collection consistent with possible perforation. The patient was treated with a 10-day course of Augmentin and advised to have a repeat CT scan for monitoring. The repeat CT showed a markedly thickened appendix with surrounding inflammation extending into the terminal ileum. Due to the abnormal CT, a colonoscopy was performed to rule out a cecal mass vs. inflammatory changes from acute appendicitis. Visualization of the cecum at that time did not appear to show mucosal abnormalities that would be consistent with neoplasm. No biopsies were taken. The patient states he was in his normal state of health until approximately one year later at which time he presented to the clinic for progressive RLQ pain with associated decreased appetite. A repeat CT abdomen showed a 17 mm enhancing appendix with moderate ascites and omental cake, suggestive of appendiceal malignancy with peritoneal carcinomatosis. The patient underwent a diagnostic laparoscopy and was found to have high grade mucinous adenocarcinoma of appendiceal origin.

Although appendiceal tumors are rare, this case highlights the importance of its consideration in the differential diagnosis particularly in elderly patients presenting with symptoms consistent with acute appendicitis.
SPICE Up Your Joint Infections

Joint replacement surgery is quite common in the US, with over 2 million cases performed in the past year. Of these, roughly 1% become infected representing over 200,000 infected joint replacements annually. Successful management of infected joint arthroplasties requires a multidisciplinary approach, including aggressive surgical debridement combined with appropriate antimicrobial support. Decision algorithms include a variety of factors, including duration and microbiology of infection, the status of bone stock, presence or absence of sinus tracts, host factors, and others. Approaches may include debridement and retention, and single vs. two-stage revision, depending upon these factors.

Our patient, a 53 year old woman with a medical history remarkable for diabetes mellitus and tobacco abuse, presented to the hospital with progressive knee pain after undergoing right total knee arthroplasty 4 months earlier at an outside hospital. Joint fluid revealed Serratia marcescens, and she was discharged from that facility with plans to receive a 42d course of ceftriaxone. She initially improved, but subsequently worsened, with increased pain, swelling, and tenderness, prompting her to present to our ED for evaluation. Fluid once again revealed Serratia marcescens, which was now reported as being resistant to ceftriaxone. She underwent revision with an antibiotic-impregnated articulating spacer and was discharged on a 6 week course of a carbapenem. She has completed that course of therapy and is being observed prior to revision arthroplasty to a permanent device.

Of importance, Serratia marcescens is a “SPICE” organism (Serratia marcescens, Providencia sp, Indole (+) Proteus, Citrobacter sp, and Enterobacter sp). As such, in clinical practice it may display inducible resistance to many extended penicillins and cephalosporins via de-repression of AmpC. Indeed, that did seem to be the case in our patient who developed resistance to ceftriaxone while on therapy. Recognizing this organism’s resistance tendency may have prevented her from suffering her initial treatment failure.
Left Ventricular Thrombus Formation in a Structurally and Functionally Normal Heart: Case Report

Introduction:
Virchow’s triad describes three factors that could explain thrombus formation: stasis, endothelial damage, and hypercoagulability. Most ventricular thrombosis occurs in the presence of impaired left ventricular function, like dilated cardiomyopathy, aneurysm, or following myocardial infarction. We report a left ventricular thrombosis (LVT) case in a patient with normal heart function and structure presenting with acute aortoiliac thrombosis.

Case Description:
A 54-year-old female presented to the ED complaining of progressive right leg pain and weakness that started 2 hours before her presentation. Her medical history is significant for uncontrolled type-2-diabetes (HbA1c 12.5) and 30-pack year smoking. She presented with high-anion-gap-metabolic-acidosis secondary to DKA. Physical examination detected tenderness over the right thigh with loss of sensation distal to the calf; right dorsalis pedis and posterior tibial pulse were absent. Her condition progressed quickly, and she developed livido reticularis in her right leg. Abdominal CT-angiogram revealed a near-complete occlusion at the aortoiliac bifurcation, bilateral common iliac arteries, and the proximal left internal iliac artery. She was started on a heparin drip and later underwent a bilateral aortoiliac embolectomy. Troponin trended up from 79ng/mL to 321ng/mL in the setting of a normal EKG. A transthoracic echocardiogram-TTE showed two mobile pedunculated echo-densities in the left ventricular apical region measuring 2.3cmx2.1cm and 1.0cmx0.5cm with a normal ejection fraction-EF of 55% and normal wall motion- suggestive of thrombosis, and later confirmed by a transesophageal echocardiogram-TEE. The patient underwent emergent cardiac catheterization-which showed a non-obstructive coronary disease- and a left ventricular mass excision. Pathology confirmed that the masses were consistent with a thrombus. Tumor investigation followed, and homocysteine levels came back unremarkable. Day-6, we discharged the patient on anticoagulation therapy comprising of Lovenox and Coumadin, with a plan to follow up with vascular surgery, hematology, and cardiac surgery teams.

Discussion:
Although very rare, this case highlights that LVT formation may occur in patients with a normal EF but with substantial risk factors conducive to Virchow’s triad, such as uncontrolled diabetes and heavy smoking. Here, LVT resulted in peripheral thrombus embolization and severe ischemic limb; anticoagulation or thrombolysis with or without surgical embolectomy remains the mainstay treatment.
Pasteurella multocida Bacteremia – The Story of Cat Man

Introduction:
Pasteurella multocida is a gram-negative coccobacillus that typically resides within the normal flora of the respiratory tract of domestic animals, especially dogs and cats. In humans, infections are associated with animal bites. Pasteurella infections without an animal bite are uncommon but can be life-threatening as they are more likely to manifest as invasive infections; usually presenting as soft tissue infections and bacteremia, possibly with cardiovascular complications.

Case Description:
A 77-year-old man presented with a fall preceded by dizziness and weakness without loss of consciousness. He was septic on arrival, with a lactic acid level of 6.0mmol/L. On examination, he had extensive chronic bilateral lower leg swelling with erythema and non-purulent ulcers. A CT scan showed bilateral, diffuse, subcutaneous soft-tissue swelling from the thigh to the foot. Blood cultures and labs were collected; meanwhile, vancomycin and cefepime was started for cellulitis from an unknown organism. Blood cultures later grew P. multocida, and his antibiotic regimen changed to ceftriaxone and doxycycline. Social history revealed that he enjoyed feeding feral cats and reported his cats licking his legs. He was discharged with instructions to complete 14 days of antibiotics, elevate his legs, and cover his wounds at home.

Discussion:
P. multocida bacteremia in humans is uncommon; untreated infection correlates with high morbidity and mortality. This patient had open wounds in his lower extremities, providing an excellent point of entry for the organism, leading to bacteremia. Management consisted of a tri-pronged therapeutic approach entailing treatment of current infection, preventing further infections, and improving healing. First line treatment for Pasteurella infections is penicillin-based antibiotics; however, this patient had a documented allergy, precluding its use. Oral doxycycline and IV ceftriaxone were utilized to establish adequate coverage. The second aspect of treatment was to prevent further infections by providing a mechanical barrier over his wounds to prevent his cats from licking the open wounds. Lastly, elevating the legs above heart level helped promote healing by encouraging vascular drainage. This case highlights the importance of social history for making a timely diagnosis of a life-threatening condition and determining a plan of treatment that fits with the values of the patient.
A Case of Ventricular Septal Rupture (VSR) Post-Myocardial Infarction: The Impending Doom!

Introduction:
Ventricular septal rupture (VSR) remains a rare, devastating complication following acute myocardial infarction (MI), typically occurring 3-8 days after an MI. Older age, female gender, prior stroke, ST-segment elevation MI, elevated cardiac markers, tachycardia, hypotension, and delayed/lack of reperfusion increase the likelihood of post-MI VSR and is highly fatal.

Case Description:
A 73-year-old female with a history of diabetes, hypertension, and hyperlipidemia presented to ED with shortness of breath for one week. She had a cough with frothy pinkish sputum and complained of nausea, vomiting, and fatigue. She also reported an episode of non-radiating chest pain with diaphoresis four days before admission, which resolved independently. Upon arrival, she had hypotension (72/54mmHg), tachycardia (103bpm), respiratory rate=18, and oxygen saturation 97% on room air. Pulmonary and cardiac exams were unremarkable. EKG showed diffuse new ST-segment elevations in leads II and V2-V5. CXR showed mild pulmonary vascular congestion. Labs were significant for troponin: 22,973ng/L, BNP: 958pg/ml. Transthoracic echocardiography-TTE revealed a reduction in left ventricle contractility with 35-40% ejection fraction. Emergent left and right heart catheterization detected VSD-ventricular septal defect and showed 100% stenosis of the left anterior descending artery (LAD). She underwent balloon angioplasty and eluting drug stent (DES) and insertion of intra-aortic balloon pump (IABP). The patient was started on norepinephrine drip and transferred to Surgical ICU. The patient’s clinical condition deteriorated, prompting Impella heart pump implantation. However, she developed ventricular fibrillation and cardiac arrest, requiring CPR and defibrillation in the OR. She was revived but went into asystole three separate times later in the day. The patient expired after the third event, as the return of spontaneous circulation (ROSC) could not be achieved.

Conclusion:
This case experienced a deadly combination of VSR post-ST-segment elevation MI, VSD, and LAD. Introducing primary percutaneous coronary intervention lowered VSR incidence to <1%; however, the mortality remains high and relatively constant, with reported 30-day mortality rates of 19–54%. The failed medical treatment makes surgical intervention the treatment of choice for VSR. Nonetheless, results of surgical repair are often suboptimal and are associated with high mortality owing to hemodynamic instability, tissue fragility, and cardiogenic shock.
A Rare Case of Lactobacillus Rhamnosus Ventriculitis Post-Craniotomy: When the Good Turns Ugly!

Introduction:
Lactobacilli are good gram-positive bacteria present in the normal flora of the oral cavity, gastrointestinal tract, and female genital tract. Lactobacillus rhamnosus GG-LGG strain is a commonly used probiotic known for its unique antimicrobial activity against harmful bacterial growth in the gut microbiota. While a microbiota-gut-brain axis exists and influences cognitive brain function, Lactobacilli rarely causes brain or CNS infections. However, there are few reports of LGG causing meningitis and ventriculitis (CSF infections) in the presence of an external ventricular drain or after recent neurosurgery. We present a case of Lactobacillus rhamnosus ventriculitis post-hemicraniectomy.

Case Description:
A 49-year-old female with a history of malignant cerebral edema endured a recent left MCA (middle cerebral artery) territory infarct requiring decompressive hemicraniectomy on a previous admission. She had a tracheostomy and PEG-tube placement. She got readmitted from an LTAC facility after developing purulent drainage from her incision site. On arrival, she was awake but obtunded, afebrile, tachycardic. Her craniectomy flap was draining purulent fluid. Initial labs were significant for leukocytosis (13.8x10⁹). CT-head revealed an interval-increase in the transcranial herniation size of the infarcted left frontal and parietal lobe through the left-sided craniotomy defect with associated ex-vacuo dilatation of left lateral ventricle and 3mm midline left shift. She was started on empiric vancomycin and cefepime for presumptive healthcare-acquired meningitis post-surgery. She had a ventriculostomy placed for communicating hydrocephalus. Intraoperative CSF was purulent, and analysis revealed elevated protein and a neutrophilic pleocytosis. CSF-culture grew Lactobacillus rhamnosus-LGG, which was resistant to vancomycin. Antibiotics were switched to daptomycin, high-dose meropenem, and intrathecal tobramycin for five days- later changed to ampicillin, clindamycin, and meropenem. Ventriculostomy removed, and repeat-CT showed persistent cerebritis and contralateral diffuse enhancement. Due to the patient’s significant co-morbidities and poor quality of life, her family decided on comfort care; she underwent palliative ventilator withdrawal and expired.

Conclusion:
Lactobacillus as a cause of ventriculitis is rare, with few reported cases of post-neurosurgical lactobacillus meningoencephalitis. LGG ventriculitis is associated with high mortality (30%), thus, requiring a prompt, comprehensive, multidisciplinary approach to care. A timely CSF sampling is essential to tailor antibiotics to the culprit of infection.
Anti-NMDA Receptor Encephalitis Masquerading as Acute Psychotic Disorder: A Case Report

Introduction:
Anti-N-methyl-d-aspartate (Anti-NMDA) Receptor Encephalitis, known as autoimmune encephalitis (AE), is an autoimmune syndrome characterized by antibodies to NMDA receptors in cerebrospinal fluid-CSF. AE causes prominent neuropsychiatric symptoms, including psychosis, seizures, and voluntary/involuntary movement disorders. Often, AE patients get mistakenly diagnosed and treated for psychiatric illness, while the underlying autoimmune process remains concealed. We present a case of anti-NMDA receptor encephalitis initially diagnosed as acute psychotic disorder.

Case Description:
A 38-year-old man was brought to ED with a complaint of bizarre behavior and hallucinations for three weeks. Two weeks prior, he experienced generalized convulsions and was admitted to another hospital and diagnosed with a seizure disorder and acute psychotic disorder. He received treatment in an inpatient psychiatric facility for six days and was discharged home on risperidone, lorazepam, and levetiracetam. However, upon discharge, he exhibited bizarre behavior depicted by pacing, profanities, and religious delusions; hence, the family petitioned and brought him to our ED. On arrival, he was febrile, tachypneic, and exhibited echolalia, nonsensical speech, and slowed motor movement. Initial CSF panel and blood cultures were negative. Brain-MRI showed a reduced mamillopontine distance-MPD - suggestive of occlusive hydrocephalus- and mild Low-lying Cerebellar Tonsils. EEG showed generalized waves slowing, with no epileptiform activity. On day-6, CSF showed NMDA receptor antibody yielding positive titers at 1:160-compatible with encephalitis; he received IV-SoluMedrol 1g and IVlg 0.4g/kg/day daily for five days. Unfortunately, he developed acute hypoxic respiratory failure secondary to massive saddle PE with right ventricular strain, detected on CT-thorax, for which he underwent thrombectomy. He also endured bowel obstruction with no identified point on exploratory laparotomy and experienced an ileus and omental repair. He was intubated requiring vasopressor support; later required a tracheostomy after multiple extubation attempts. Two months later, he sustained improvement in neurologic function and was discharged to a skilled nursing facility.

Discussion:
AE is uncommon, with atypical symptoms from other neurologic or psychiatric conditions. Such disjointed presentation can often lead to delayed diagnosis and treatment, adversely affecting outcomes, including the chance for a cure. Clinicians should pay attention to neuropsychiatric presentations unresponsive to treatment and investigate underlying autoimmune causes.
A Case of Chronic Abscess Post-Gluteal Augmentation Due to Mycobacterium Fortuitum: A Botched Attempt to Beauty!

Introduction:
Fat grafting has become a popular technique for gluteal enhancement, where fat is harvested by liposuction and transplanted to enhance the gluteal region. Cutaneous and soft tissue infections secondary to fat grafting are usually low and mainly caused by gram-negative bacteria and Staphylococcus aureus. However, rapidly growing mycobacteria (RGM) have become prominent causative agents for atypical skin infections leading to chronic abscess formation at the injection grafting site. Here, we present a unique case of Mycobacterium fortuitum causing a gluteal abscess post-grafting.

Case Description:
A previously healthy 26-year-old female presented to ED with a 5-day history of left buttock swelling and pain after undergoing a gluteal enhancement procedure 12-weeks prior to presentation. Bedside ultrasound showed no fluid collection; she was prescribed cephalexin for seven days and discharged home. However, she got readmitted five days later with worsening symptoms. She was afebrile and vitally stable but had a significant 9x9cm area of erythema and induration in the left gluteus. Labs showed no leukocytosis. CT-abdomen/pelvis revealed no fluid collection. We evaluated the surgical incision, drained 30ml of purulent fluid, and sent for culture. We started the patient on IV-vancomycin; gram-stain showed no organisms. We discharged her two days later on Doxycycline and Augmentin for 14 days. After nine days, the culture grew Mycobacterium fortuitum, therefore we recommended continuing doxycycline for three months. On Follow-up, the patient still had a residual discharge; re-cultured abscess fluid was sent for repeat-culture and sensitivity-later came back negative. We added moxifloxacin to Doxycycline for another two weeks. We lost the patient to follow-up, but she presented ten months later, and her injection site was infection-free.

Discussion:
RGM infection post-grafting, especially with M. fortuitum, should be suspected in delayed surgical wound healing and latent wound infection resistant to routine antibiotic therapy. A post-injection infection or abscess complex usually develops secondary to inadequate surgical equipment sterilization and unsterile water in wound irrigation and surgical solutions. M. fortuitum indolent cutaneous infections may require several rounds of surgical debridement and prolonged treatment with combination antibiotics, at least two agents, with in-vitro activity against the isolated bacteria for 3-4 months.
A Case of Phentermine-Associated Atrial Fibrillation: When a Diet Pill Tangoes with Cardiac Rhythm

Introduction:
Obesity is a significant risk factor highly-associated with cardiovascular complications. Besides diet and exercise, pharmacotherapy remains a popular option for managing obesity, with phentermine being the most commonly used drug. Phentermine (Adipex), a centrally acting appetite-suppressant and norepinephrine stimulant, is an adjunctive drug primarily prescribed for obesity (BMI>30) refractory to 3-6 months of lifestyle interventions. Short-term use is recommended because of the relative increase in sympathetic tone and adverse effects such as insomnia, dry mouth, dizziness, hypertension, and tachycardia. Of note, palpitation is a well-documented cardiovascular side-effect of phentermine. This case explores atrial fibrillation-AFib as a potentially severe adverse outcome of phentermine in an obese but healthy female.

Case Description:
A 49-year-old obese female without significant medical history, except for migraines, presented with acute onset of dizziness and palpitations. One day before presentation, she was started on Adipex (phentermine) 37.5mg for weight loss by her PCP. She only takes Excedrin for migraines. Her initial vitals showed hypertension (189/91mmHg) and tachycardia (200s beats/min). EKG revealed AFib with a rapid ventricular response-RVR. Labs were non-significant. Thyroid panel was unremarkable, and urine drug screen was negative. The patient was started on a diltiazem drip for rate control, later titrated to oral Cardizem 30mg Q6 hours. She achieved rhythm control with amiodarone drip and was anticoagulated with heparin. The AFib resolved, and she remained stable for the rest of the hospital course, maintaining a heart rate at 80-90s sinus rhythm. A 2-D echocardiogram showed a normal ejection fraction of 60-65%. She got discharged on metoprolol 25mg and aspirin 81mg and recommended discontinuing Adipex.

Discussion:
AFib is an unusual cardiovascular side-effect of phentermine that warrants clinicians' caution- especially in the absence of other triggers or structural heart diseases. AFib-RVR and phentermine-associated hypertension and tachycardia could be life-threatening. It remains unclear whether the caffeine in Excedrin may have exerted an additive, synergistic effect with noradrenergic high-dose phentermine to generate arrhythmia. Besides, caffeine, a widely-consumed CNS stimulant among dieters, could have added salt to injury to this potential paradoxical association, with hidden danger and fatal consequences. Perhaps, future cardiovascular outcomes studies may provide the right answers.
Exercise-Induced Vocal Cord Dysfunction or Exercise-Induced Asthma? Commonly Misdiagnosed Pathologies with Similar Presentation

Introduction:
Exercise is a common and well-known asthma trigger, especially high-intensity exercises, as seen in athletics. However, vocal cord dysfunction (VCD) can sometimes have a similar presentation with difficulty breathing and choking events, which might be misdiagnosed and treated as asthma.

Methods:
We conducted a literature review on PubMed and Cochrane databases using these keywords: “asthma,” “exercise-induced asthma (EIA),” “exercise-induced bronchoconstriction,” “vocal cord dysfunction (VCD),” “exercise-induced laryngeal obstruction (EILO),” and “Paradoxical vocal fold motion dysfunction (PVFMD).” We reviewed eight articles, including case-control, cross-sectional, retrospective, and prospective studies.

Results:
VCD is more prevalent in asthmatic patients and athletes than in the general population. A case-control study on 94 asthmatic patients and 40 control subjects showed that PVFMD prevalence was significantly higher in asthmatic patients with PVFMD (19%) compared to control group (5%) (p<0.001). Another retrospective study that referred 88 athletes for asthma workup showed that 35.2% had EILO, and 43.2% had a positive bronchoprovocation or bronchodilator reversibility test. However, a prospective study on 98 non-athlete individuals showed 7.5% EILO. One study on 100 patients used continuous laryngoscopy during exercise as a standard test and compared it to pre and post-exercise flow-volume loops. This study showed no significant association between laryngoscopic findings and flow-volume data. Another case-control study conducted on 16 patients using continuous laryngoscopy test during exercise showed that all these cases but not controls had positive findings, indicating that this test has diagnostic potential in VCD workup. While speech therapy remains the mainstay treatment for VCD, one study showed that 4/9 (44%) patients with VCD had rapid symptoms resolution after basic biofeedback. Another study demonstrated that hypnosis resolved VCD in 38% of the cases and improved symptoms in 31%.

Conclusion:
EIA and VCD are two different entities with very similar presentations. While asthma diagnosis is well-established, diagnosing VCD remains challenging. Treatment failure for EIA warrants considering VCD as an alternative diagnosis. Continuous laryngoscopy exercise test seems to be an effective diagnostic modality for VCD. Treatment for VCD seems to focus on behavioral therapy; more studies are needed to establish its definitive diagnosis and treatment and investigate novel therapies for refractory patients.
A Rare Case of Spontaneous Bacterial Pleural Empyema in the Absence of Pneumonia or Cirrhosis

Introduction:
Spontaneous Bacterial Pleural Empyema (SBEM) is an infection of the pleural fluid in the absence of pneumonia—a rare occurrence. Most cases described in the literature are in the context of the contiguous spread of the infected ascetic fluid secondary to cirrhosis, with limited reports on SBEM in the absence of cirrhosis or any other inciting factor. We present this rare case of SBEM without cirrhosis or hepatic hydrothorax involvement.

Case Description:
A 37-year-old African American female presented with worsening left-sided pleuritic chest pain and dyspnea that started one week prior. The patient denied any fever, chills, cough, or trauma to the chest wall nor any sick contacts, or prolonged immobility. Vital signs on presentation were significant for only mild fever. On physical exam, she showed moderate distress due to pain. Chest examination revealed dullness to percussion, and diminished breath sounds on the left side of the chest. Labs were significant for leukocytosis (WBC=24K). EKG showed sinus tachycardia without any acute ST changes. CXR showed loculated pleural effusion on the left lung, corroborated with CT-thorax, and left lung multiloculated effusion with associated atelectasis. We initiated broad-spectrum antibiotics and consulted cardiothoracic surgery. She underwent Left thoracotomy with epyemectomy, empyema drainage of 850cc of pleural fluid, and pleural biopsy. The pleural fluid culture was negative, but the pathology report indicated pleural abscess and inflammatory changes but no concerning signs of malignancy or TB. After surgery, the patient improved clinically and was discharged home after a short hospital stay.

Discussion:
This case highlights that SBEM is a diagnosis of exclusion in the absence of pneumonia or cirrhosis. SBEM criteria include transudative pleural fluid in the absence of a pulmonary source of infection, with polymorphs>500cells/ml or positive pleural fluid culture. Broad-spectrum antibiotics and thoracocentesis remain the mainstay treatment for SBEM, while epyemectomy with thoracotomy are other options if treatment fails. This case demonstrates that a bacterial pleural empyema has a variety of presentations, and in rare instances, it occurs without any apparent etiological cause. Clinicians should be vigilant not to miss such a diagnosis to initiate prompt management and avoid catastrophic outcomes.
The Incidence of Myocarditis: Weighing the Risks between COVID-19 Infection and Vaccination

Introduction: Myocarditis, cardiac muscle inflammation, an infrequent phenomenon caused by viral infections, has recently stolen the spotlight in COVID-19, with some cases being infection-induced and others being vaccine-related. This EBM case aims to compare myocarditis incidence rates post-COVID-19 infection against that induced by COVID-19 mRNA vaccines in young adults aged 18-45.

Methods: We performed a literature search on PubMed and PMC databases, using these keywords: “Myocarditis,” “Incidence,” “COVID myocarditis,” and “vaccine myocarditis.” We retrieved two systematic reviews and some population-based reports.

Results: A systematic review of 41 case reports on myocarditis in COVID-19 patients (median age 43.4, 71.4% males) reported hypertension and type-2-diabetes as significant predisposing comorbidities. Another systematic review of three studies showed that COVID-19 myocarditis incidence reached 12.5% in a Wuhan study, while in the US, 65 ICU departments reported an incidence of 0.1-2.5%, and another had a 0.25% incidence rate. Nevertheless, other studies have associated COVID-19 vaccines with myocarditis. A population-based cohort analysis from Israel included 5,125,635 individuals who received two doses of COVID mRNA-vaccine, of which 283 cases had myocarditis (0.003%). A similar US study reviewed forty hospitals documenting 2,000,287 individuals who received COVID-19 vaccines, 20 patients developed vaccine-related myocarditis and 37 pericarditis, making 57 patients (0.0028%). The Vaccine Adverse Event Reporting System-VAERS data (December 2020-August 2021, pre-Delta surge) showed that 192,405,448 individuals (aged 12-45) received two mRNA-vaccine doses; 1626 developed confirmed myocarditis within 7-days post-vaccination, the majority after the second dose (82%), and mostly among males <30. While myocarditis incidence for Pfizer vaccine alone was highest among adolescents aged 12-15 (70.7/million doses) and 16-17 (105.9/million doses), the incidence in 18-24 was 52.4 and 56.3/million doses in Pfizer and Moderna, respectively. One study showed that Pfizer–BioNTech had a higher myocarditis incidence of 6.70/million doses than Moderna's 4.98/million doses.

Conclusion: Despite being rare, our report has demonstrated a higher myocarditis incidence post-COVID-19 infection than post-vaccination, and the latter seems faster-occurring than latent post-viral infection, including COVID-19. However, with genuine concerns over underreporting real COVID-19 or vaccine-related myocarditis cases, weighing the risks and benefits of mRNA vaccine over COVID-infection becomes a leap of faith in young adults.
A Rare Presentation of a Life-Threatening Atypical Hemolytic Uremic Syndrome (aHUS) Following Acute Pancreatitis

Introduction:
Hemolytic Uremia Syndrome (HUS) is a subtype of thrombotic microangiopathy characterized by the triad: hemolytic anemia, thrombocytopenia, and acute renal failure. HUS is attributable to the deposition of platelet microthrombi in the microcirculation of various organs. 90% of HUS cases are due to Shiga-Toxin-producing-E. Coli (STEC-HUS) - mainly a childhood disease. Only 5-10% is atypical HUS (aHUS), with incidence of 1:500,000, resulting from genetic mutations or antibodies hyperproduction against complement proteins, thus, dysregulating the alternative complement pathways.

Case Description:
A 35-year-old male with a history of recurrent alcoholic pancreatitis, CKD-II, hypertension presented to ED complaining of acute constant dull upper abdominal pain after alcohol consumption. He was afebrile, hypertensive (160/80mmHg), bradycardic (50bpm), and denied diarrhea or oliguria/dysuria. He had a diffusely tender abdomen with no peritoneal signs. Labs showed elevated lipase >5000U/L and creatinine 1.5mg/dL (baseline 1.4mg/dL), with no signs of anemia, thrombocytopenia, or leukocytosis. CT-abdomen showed diffusely edematous pancreas. He was started on supportive management with IV fluids and pain control. Within 48-hours, his renal function worsened: creatinine increased 5.08mg/dL with new-onset oliguria, acute anemia (Hb=9.7mg/dL), and severe thrombocytopenia 16x10^9 platelets/L. Peripheral blood smear showed schistocytes. Subsequent labs showed hyperbilirubinemia (elevated indirect-bilirubin), low haptoglobin, and elevated LDH with normal PT/INR confirming intravascular hemolysis. After meeting this triad, we started him on emergent plasmapheresis for TTP-thrombotic thrombocytopenic purpura. STEC-HUS was unlikely in adults, and he had no fever, leukocytosis, or bloody diarrhea. Later, ADAMTS13 activity resulted in 102%, ruling-out TTP. He was started on Eculizumab (complement C5-inhibitor) for presumptive atypical HUS. Five days later, platelet count improved remarkably 254x10^9 platelets/L, and creatinine dropped to 3.39mg/dl and BUN to 29mg/dl. Complement work-up showed a reduction in C4 and factor-I, confirming aHUS diagnosis. The patient was discharged in a stable condition to follow up for further genetic testing.

Conclusion:
HUS presents a life-threatening but reversible episode with supportive treatment, unlike aHUS, a more lethal and often recurrent condition with unknown triggers. Timely recognition and treatment of aHUS dramatically slows progression to end-stage renal disease, the common complication seen in most recurrent aHUS. C5-inhibitor (Eculizumab) shows remarkable clinical improvement with near-complete response in days to weeks.
When NSAID Inflamed the Situation: A Complicated Case of Toxic Shock Syndrome Caused by Group-A Streptococcus Pyogenes

Introduction:
Group A streptococcus (GAS) is a gram-positive bacterium that causes pharyngitis or skin and soft tissue infections; in severe cases, it can cause invasive bacteremia, including Toxic Shock Syndrome (TSS). In the GAS setting, there is a possibility that a recent intake of NSAIDs would suppress the host immunity, disguise the signs and symptoms of this emerging invasive infection, and indirectly enhance TSS development. Here, we present a complicated case of TSS caused by GAS following acute NSAID intake.

Case Description:
A 45-year-old female with a history of hypertension presented to ED complaining of coffee-ground emesis, mild shortness of breath, generalized lethargy, and severe sore throat for a few days. She took five tablets of Ibuprofen (total=2g) 24-hours before the presentation. She became hypotensive, tachycardiac, tachypneic, hypoxic, and febrile upon arrival. Physical examination showed multiple bullae on the upper and lower extremities. Initial labs were significant for elevated creatinine, transaminitis, leukocytosis, significantly elevated CPK, thrombocytopenia, anemia, and lactic acidosis. CT-thorax showed bilateral multilobar pneumonia. The patient was intubated, admitted to the ICU, and started on vasopressors and antibiotics—azithromycin, ceftriaxone, and vancomycin. Blood cultures grew group-A Streptococcus Pyogenes within the first 24-hours, so we switched antibiotics to clindamycin and ceftriaxone and administered intravenous immunoglobulin—IVIg. EGD revealed esophagitis with esophageal ulceration and a small GIST tumor. However, a cascade of events complicated her hospital course: acute tubular necrosis-ATN (requiring renal replacement therapy); acute pancreatitis; epidermolysis; disseminated intravascular coagulation-DIC; and severe myositis (confirmed by left forearm biopsy, requiring debridement). She eventually developed dry gangrene resulting in bilateral below-knee amputation. After prolonged intubation, we placed a tracheostomy and sent her to Inpatient Rehab in stable condition.

Discussion:
The association between NSAID intake and increased risk of bacteremia has been controversial. Such plausible association focuses on NSAID’s role in inhibiting neutrophil function, suppressing fever, and augmenting cytokine release, delaying early diagnosis of GAS invasive infection, leading to TSS development and its catastrophic events. This case highlights the NSAID role as a potential culprit that induced GAS-TSS's convoluted course. IVIg treatment seemed promising and improved outcomes within the multidisciplinary management approach.
A Rare Case of Malignant Cerebral Edema After Fentanyl and Xanax Intoxication

Introduction:
Cerebral edema (CE) is associated with intoxication from opiates/opioid abuse and is mainly diagnosed post-mortem because of high fatality. Abuse of Fentanyl, the general anesthetic, is even more lethal than other opiates/opioid intoxication because of its 80-100 times increased potency than morphine on the CNS and brain opioid receptors leading to hypoxia-related brain injury and associated life-threatening respiratory depression, coma, or death. CE symptoms appear when ICP rises above 20cmH2O. There are few published reports of opioid-induced malignant cerebral edema; here, we present a rare case associated with fentanyl and alprazolam abuse.

Case Description:
A previously healthy 25-year-old male was brought to ED by EMS for a suspected drug overdose. He was hypotensive, tachycardic, tachypneic, and unresponsive. EMS administered 4mg intranasal Narcan for respiratory depression. He exhibited decerebrate posturing, pupils 3mm and sluggish, and GCS=4. He got intubated, and norepinephrine was initiated for BP support. Labs were significant for lactic acid=5.8, serum ammonia=74, serum osmolality=308, potassium=5.9, Cr=1.79, CPK=519, AST=62, WBC=18.7. Urine drug screening was positive for benzodiazepine, cannabinoids, nicotine metabolites, lidocaine metabolites, ketamine, and fentanyl. EKG showed sinus tachycardia. CT-head showed ischemic changes in bilateral cerebellar hemispheres- eliciting concerns over early transtentorial herniation, but CTA-head/neck was negative. While neurosurgery was inserting an ICP monitor, the patient's vitals declined, and his right pupil dilated, ICP measured >50. He was immediately transferred to OR for decompressive hemicraniectomy. He was weaned from sedation post-surgery and found alert and responsive with weakness in the right lower extremity. He was eventually extubated and discharged to physical rehabilitation. Five months later, he followed up as an outpatient for surgical clearance for cranioplasty; he was fully ambulatory, with some mild weakness of the right lower extremity remaining.

Discussion:
This rare case highlights the association between CE and non-fatal opioid intoxication by fentanyl and benzodiazepine overdose by alprazolam. Benzodiazepine overdose causes toxic leukoencephalopathy resulting in brain edema and herniation. Fentanyl abuse is lethal enough, and a combined intoxication with benzodiazepine overdose with Xanax (and other substances, such as ketamine) potentiated its effects to malignant CE and near-fatal herniation levels. Timely surgical and medical interventions prevented imminent death.
Multivalvular Infective Endocarditis in an Intravenous Drug User – A Case Report

Introduction:
Infective endocarditis (IE) is a life-threatening cardiac inner lining and valve inflammation and one of the most severe sequelae of injection drug use (IDU). It frequently involves the tricuspid valve, with Staphylococcus aureus being the most typical infecting organism and left-sided IE being more common in females. IE involving multiple cardiac valves is rare and is associated with more complications such as a higher incidence of heart failure and peri-valvular complications, which often require surgery for valve repair with or without replacement.

Case Description:
A 28-year-old African-American woman with an active intravenous heroin use history was brought to ED unresponsive. Upon arrival, her vitals were: BP= 105/77mmHg, HR=115beats/min, afebrile, RR=18/min, O2 saturation of 97% on RA. Tachycardia, S1 and S2 were normal, with no murmur. Physical exam was unremarkable, and skin exam was negative for track marks, Janeway lesions, or Osler’s nodes. Labs were significant for neutrophilic predominant leukocytosis (20.4k). Blood cultures grew methicillin-susceptible Staphylococcus aureus (MSSA), but CXR showed no acute bacteremia process. MRI-brain showed subacute infarcts in the right middle cerebral artery- MCA territory. TTE showed a thick mitral leaflet with mild to moderate mitral regurgitation. TEE showed extensive 2cm mobile mitral valve vegetation associated with leaflet perforation and moderate regurgitation and a left atrial clot with tricuspid valve vegetation and mild regurgitation. CTA-abdomen/thorax showed septic emboli to the lung, proximal superior mesenteric artery, spleen, and Kidneys. The patient was started on IV nafcillin and heparin. However, despite appropriate antibiotic therapy, she underwent mitral valve replacement and tricuspid valve repair due to ongoing systemic emboli and persistent positive cultures. After completing antibiotic treatment and negative repeat blood cultures, she got discharged home.

Discussion:
Left-sided valve IE poses a higher risk of neurological complications and embolization events, thus a higher mortality risk, requiring immediate treatment for favourable outcomes. IE Diagnosis relies on blood cultures and TTE/TEE; however, TEE had a better diagnostic advantage than TTE in detecting atrial clots and multivalvular vegetations; as shown in this case, TTE revealed mitral valve regurgitation but failed to detect the tricuspid valve involvement, which is more common in IDU.
A Case of Serotonin Syndrome: When a “Happy Hormone” Turns Lethal

Introduction
Serotonin syndrome, also called serotonin toxicity, is a life-threatening condition associated with increased serotonin concentration in the brain, mainly drug-induced. It usually involves a spectrum of clinical findings, including mental status changes, autonomic hyperactivity, and neuromuscular abnormalities. A diagnosis of serotonin syndrome is merited solely on clinical grounds, emphasizing a thorough history and physical examination.

Case Description:
A 20-year-old female patient with a history of depression was found unconscious and lying on the floor with empty medication bottles by her side; her mother called EMS to the scene and transferred her to the ED. The bottles included over-the-counter and prescription medications: acetaminophen (100 tablets), sustained-release bupropion 200mg (30 tablets), and sertraline [Zoloft] 50mg (30 tablets). The patient was non-compliant with her prescribed medications as there were plenty of tablets before attempting suicide by overdose. It was estimated that the patient took at least 25g of acetaminophen along with approximately 25 tablets of sertraline and bupropion. Physical examination revealed a diaphoretic and pale patient with widely dilated pupils, equal and reactive to light, with normal tone in upper extremities, but pronounced rigidity, tremor, and myoclonus in lower extremities- all consistent with serotonin syndrome. Twelve-lead EKG showed widening of QRS complex. She also had an acetaminophen level of 478mg/kg but without significant elevation in AST and ALT. The patient was intubated due to altered mental status, received benzodiazepines for serotonin syndrome, and was transferred to ICU. She was extubated later and discharged home.

Discussion:
Selective serotonin reuptake inhibitors [SSRIs] are the most commonly implicated medications associated with serotonin syndrome. Increased serotonin transmission could result from using a combination therapy of serotonergic medications. Monotherapy with bupropion, which inhibits dopamine and norepinephrine, does not increase serotonin but synergistically increases serotonin when co-administered with sertraline-Some case report suggest that Bupropion inhibits SSRI metabolism by affecting CYP2D6. Serotonin syndrome is diagnosed using Hunter toxicity criteria Criteria Decision rules; our patient has taken serotonin agents and exhibited the following: Spontaneous clonus; tremor plus hyperreflexia; inducible clonus and agitation; a. An accurate, timely diagnosis is life-saving.
A Unique Presentation of Adult-Onset Giant Cell Hepatitis Associated with Autoimmune Hemolytic Anemia

Introduction: We present a unique case of adult-onset giant cell hepatitis associated with autoimmune hemolytic anemia (GCH-AHA).

Case Report: A 29-year-old African American male presented with two weeks of progressively worsening scleral icterus, severe pruritus, fatigue, and dark urine. His medical history included autoimmune hemolytic anemia (AHA), eczema, and suspected infiltrative liver disease vs autoimmune hepatitis based on workup performed one year prior when the patient presented with similar symptoms, but a liver biopsy was not completed at the time. One year later, he was admitted for acute hepatitis. Abdominal ultrasound revealed no evidence of cholelithiasis or cholecystitis and a normal sized liver. He then underwent ultrasound guided liver biopsy, which revealed a pathologic diagnosis of chronic hepatitis with extensive hepatocellular injury and giant cell transformation (giant cell hepatitis) with extensive perisinusoidal and septal fibrosis, and extensive lobular Kupffer cells iron accumulation (presumably from hemolytic anemia). He was treated with Dexamethasone 40 mg intravenously daily for one week leading to complete resolution of clinical symptoms and significant improvement in liver enzymes upon discharge and instructed to follow-up for maintenance therapy.

Discussion: Adult-onset postinfantile giant cell hepatitis (PIGCH) cases are rare, with autoimmune hepatitis found to be the most prevalent etiology. Other etiologies include: infections, autoimmune, hematologic, hypoparathyroidism, sarcoidosis, and post-transplant. Giant cell hepatitis associated with autoimmune hemolytic anemia (GCH-AHA) is rare disease diagnosed in the infantile or young pediatric population, with only one case of postinfantile GCH-AHA in which the patient was age 12 years at time of diagnosis. There are no documented case reports of adult-age (> 18 years) onset GCH-AHA. The diagnosis requires a high index of suspicion in addition to: acute liver injury, detailed medical history, thorough serologic workup of all other liver disease etiologies, liver biopsy histologically confirming present of giant cell hepatocytes, and definitive diagnosis of AHA. Unique to this case was the presentation of adult-onset GCH-AHA, diagnosed by the following evidential workup: detailed medical history, liver disease workup that ruled out all other PIGCH etiologies except for AHA, diagnosis of AHA, and then a histologically confirmed diagnosis of giant cell hepatitis by liver biopsy.
What’s in a Sinus Infection? A Rare Case of Frontal Cerebritis from Contiguous Spread of Frontal Sinus Mucocele

Introduction:
Frontal sinusitis, a common condition affecting 15% of the adult population, can rarely lead to orbital and intra-cerebral complications, including meningitis or brain abscess, which could potentiate a seizure activity. We describe an interesting case of frontal cerebritis arising from the contiguous spread of mucocele secondary to chronic sinusitis- a rare occurrence.

Case Description:
An 83-year-old AA lady with a history of hypertension and chronic sinusitis got admitted after experiencing new-onset generalized tonic-clonic seizure for approximately 20 minutes requiring high doses of IV-benzodiazepine and Levetiracetam. CT-head was negative for hemorrhage and mass. CTA-brain showed probable 1.1cm meningioma in the superior right parietal region lateral to the falx cerebri without mass effect. MRI-brain showed findings highly suggestive of meningitis with cerebritis involving a portion of the anterior right frontal lobe. It also revealed mucosal thickening in the right frontal sinus and bone destruction involving the inferior aspect of the inner table of the right frontal sinus, immediately adjacent to the area of abnormality in the right frontal lobe- suggestive of sinusitis with an intracranial extension of the infection. CT-sinuses affirmed MRI findings, and Neurology concurred that the seizure was secondary to meningitis/cerebritis. EEG was unremarkable. Surgeon performed FESS-functional endoscopic sinus surgery. Histopathology showed respiratory mucosa with highly vascularized stroma, blood, and inflammation. The patient received empirical broad-spectrum antibiotics without antifungal coverage. She had an uncomplicated hospital course post-surgery and was discharged home for follow-up. MRI-brain at three-month showed resolution of meningitis/cerebritis. Six months later, she was seizure-free, and her CT-sinuses showed post-surgical improvement.

Discussion:
Frontal sinus mucocele is a benign, slow-growing pathology formed secondary to drainage block and retention of mucous secretions in the sinus that can erode the sinuses' posterior walls and extends to intracranial spaces leading to brain abscess/cerebritis and meningitis inducing seizures- as illustrated in this case- or meningoencephalitis. This case shows that frontal cerebritis requires prompt management to save the patient from intracranial complications and death once detected through diagnostic imaging. Nevertheless, earlier detection of cerebritis proves challenging as patients present with symptoms indicative of progression to abscess formation, such as headache, altered consciousness, and seizure.
Novel Case of a patient using the Minimed 770g System in Auto Mode While Taking Humulin U-500

BACKGROUND:
Patients with severe insulin resistance require more than 200 units of insulin a day on average. Despite this, these patients do not achieve adequate glycemic goals. With increasing prevalence of diabetes and obesity in the United States, severe insulin resistance has become more common, increasing the need for more concentrated insulins. Humulin R U-500 is a 5 times more concentrated form of Insulin that is used to control hyperglycemia in diabetics who are insulin resistant. Its use in insulin pumps is not FDA approved and has not been reported before as used in auto mode format.

CASE:
A 46-year-old female with Type 1 Diabetes Mellitus of 26-year duration, complicated by severe insulin resistance, Class 3 obesity, PCOS, OSA, hypertension, hyperlipidemia, and atrial fibrillation. The patient has a history of poorly controlled Type 1 diabetes mellitus with sequela of left eye retinal detachment, peripheral neuropathy, and peripheral arterial disease. She was seen in May 2016. During her initial visit, she was requiring 190 units of Lantus and 385 units of Glulisine. She was then switched to Humulin R U-500 Insulin at 350 units. She was later transitioned to insulin pump therapy in March of 2017. During her last routine visit in November of 2021, she was using the Medtronic MiniMed 770G pump with Humulin R U-500 in manual mode. Due to persistent fluctuating hyperglycemia, the patient was transitioned from Manual Mode to “Auto Mode” and began using the feature 93% of the time. At her 8 week follow up the patient’s total daily dose of insulin decreased from 350 units to 225 units daily, time in range was 76% compared to 48% in manual mode, with an average blood sugar of 173 mg/dl compared to 244 mg/dl previously. The patient improved her bolus amount per day from 31% to 63% in auto mode.

CONCLUSION:
This is the first case reported using Humulin R U-500 insulin with auto mode in insulin pumps and closed loop therapy.
Severe Pneumomediastinum in a COVID-19 Patient: A Rare Outcome of Survival

Pneumomediastinum is a rare condition where there is presence of air within the mediastinum. It is usually caused by trauma, chronic lung disease, or excessive physical strain. Pneumomediastinum associated with COVID-19 pneumonia is not an uncommon complication. Bilevel positive airway pressure (BIPAP) or continuous positive airway pressure (CPAP) support leading to pneumomediastinum in COVID-19 patients has been associated with worse outcomes. Surviving severe pneumomediastinum associated with COVID pneumonia is rare.

A 34-year-old woman presented to the emergency department with one day of cough, chills, and dyspnea. She was previously vaccinated with one dose of the Moderna vaccine a week prior to admission. She tested positive for COVID-19 in our ER and was admitted for acute hypoxic respiratory failure. She was previously diagnosed with lupus nephritis for which she has been on immunosuppressants and oral steroids prior to admission. Treatment was initiated with dexamethasone, remdesivir, and oxygen. Her oxygen requirements increased rapidly over the next five days requiring high flow nasal cannula and BIPAP support. Following use of BIPAP, the patient developed a severe pneumomediastinum resulting in intubation. She was maintained on lower tidal volumes and peak pressures on the ventilator in attempts to avoid progression of pneumomediastinum. Her extensive subcutaneous emphysema required multiple skin incisions and bilateral chest tube drains. She had a prolonged ICU course complicated by multi-organ failure. She was later transitioned successfully to tracheostomy for persistent respiratory failure. She was discharged and is currently at a long term acute care facility.

Our patient developed pneumomediastinum 24 hours within BIPAP use. Given the increased alveolar pressure and underlying lung injury in severe COVID-19 pneumonia, it is likely that intractable cough with positive pressure led to pneumomediastinum. Although pneumomediastinum with respiratory compromise in COVID-19 patients has shown overall poor outcomes, this is a rare case at our institution of a survival.
Post Myocardial Infarction Ventral Septal Defect in the COVID-19 ERA: How Fear Can Contribute to Delayed Presentations

We report one case of post myocardial infarction ventral septal defect following a delay in presentation during the COVID-19 era. A 72-year-old male presented to the emergency department eleven hours after he had begun to experience substernal chest pain and shortness of breath. The patient delayed his presentation due to a fear of exposure to COVID-19. EKG showed sinus tachycardia with ⅓ to 1 mm ST elevations in the inferior leads and reciprocal ST depressions in the anterior leads. High-sensitive troponin was also elevated at 7400.

He was taken to the Cardiac Cath lab where he received a total of four drug-eluting stents: distal RCA x1, early to late distal RCA x1, mid to proximal RCA x1, and proximal RCA x1. About eight hours post catheterization, a rapid response team (RRT) was called to the patient’s room for shortness of breath. On physical exam, a new grade 3/6 holosystolic murmur was noted in all cardiac listening posts. A stat chest x-ray showed worsening pulmonary edema. EKG showed left atrial enlargement with RBBB and inferior infarct. Echocardiogram revealed a post-myocardial infarction with moderate ventricular septal defect with clear left to right shunting. The patient was transferred to an outlying facility and underwent surgery eight days later for two vessel CABG and VSD repair. During this case report, we will highlight the significance of late presentation of myocardial infarction, as well as swift identification of post myocardial infarction complications.
Pancreatic Somatostatinoma Associated with Neurofibromatosis Type 1 - A Case Report

BACKGROUND: Somatostatinoma is a rare neuroendocrine tumor often associated with neurofibromatosis type 1, Von Hippel Lindau syndrome, and tuberous sclerosis. The incidence of somatostatinoma is estimated to be 1 in 40 million. Somatostatinomas have an insidious growth, and as a result, it presents in later stages as a malignant disease. Somatostatinoma presenting with “somatostatinoma syndrome,” which consists of diarrhea, diabetes, and gallstones, is uncommon.

CASE DESCRIPTION: We present the case of a 24-year-old female who presents with newly diagnosed diabetes, diarrhea, and abdominal pain. The patient’s family history consists of MEN (type unknown) syndrome in the patient’s father. Physical examination revealed extensive skin nodules (neurofibromas) and hyperpigmented patches (Café au lait spots), which raised concern for neurofibromatosis. Imaging demonstrated innumerable heterogeneous enhancing masses throughout the hepatic parenchyma and a hypodense lesion in the uncinate process of the pancreas, a stent in the biliary duct with a dilated pancreatic duct, enlarged retroperitoneal and mesenteric lymph nodes, and intraabdominal free-air suggestive of bowel perforation. Biopsy of the liver mass demonstrated neoplastic cells. The immunohistochemical evaluation showed cells that stained positive for AE1/AE3, KI67, Chromogranin, CD56, and CK7. The clinical presentation, radiological appearance, and immunohistochemical staining pattern support a diagnosis of somatostatinoma.

CONCLUSION: Although somatostatinoma presents at a later age given the insidious growth, it should not be missed in younger individuals who present with typical symptoms of somatostatinoma syndrome. Given its association with genetic disorders, effort should be made to distinguish sporadic tumors from those associated with genetic conditions.
An Unusual Presentation of Disseminated Histoplasmosis in an Immunocompetent Patient

Histoplasmosis is an endemic mycosis caused by Histoplasma capsulatum, a fungus that lives in the soil, especially in locations with a large amount of bird or bat excrement. Although it primarily causes pulmonary disease, disseminated infection can develop in rare cases. Immunocompromised patients are most at risk, but severe disease can also occur in immunocompetent patients.

A previously healthy 36-year-old male presented with 8-day history of fever, headache, neck pain, arthralgias, and photophobia. On the day of presentation, he developed a mild cough and shortness of breath. The patient worked as a landscaper and had recently worked at a shed with bat excrement present. Laboratory results were only significant for mild leukocytosis. There was no evidence of immunocompromise. Chest CT showed numerous scattered nodules in the lung with hilar and mediastinal lymphadenopathy. MRI of the brain demonstrated diffuse and patchy meningeal enhancement suggestive of inflammation. Comprehensive CSF and serum viral panels were unremarkable. Despite treatment with broad-spectrum antibiotics, the patient continued to have fevers and an intractable headache. The patient’s respiratory status rapidly declined. Fungitell was found to be mildly elevated. The patient was started on Amphotericin B due to a high clinical suspicion of fungal etiology. He showed clinical improvement 48 hours after the initiation of Amphotericin B. A bronchial biopsy showed necrotizing granulomas, and Histoplasma was confirmed by GMS staining. After a few days on Amphotericin B, the patient was switched to Itraconazole due to the development of hepatic dysfunction and hypokalemia. Notably, while on itraconazole, his pulmonary and CNS symptoms resolved despite the lack of CNS penetration of this medication. Histoplasmosis meningitis was suspected, in addition to pulmonary involvement, despite negative CSF findings due to clinical symptoms, MRI findings, and clinical course.

The diagnosis of histoplasmosis is often missed in immunocompetent patients because of its clinical resemblance to more common illnesses like bacterial pneumonia. Therefore, it is important to keep Histoplasma in the differential if the patient is exposed to a substantial number of spores or lives in an endemic area.
A Rare Case of Nocardia Otitidiscaviarum Infection

Introduction:
Disseminated nocardiosis is a rare opportunistic infection seen commonly in patients with impaired T-cell immunity. We present a case of disseminated Nocardia Otitidiscaviarum, of which less than 100 cases have been reported so far.

Case description:
A 72-year-old male with a history of renal transplant 20 years ago on chronic immunosuppression with prednisone, mycophenolic acid, and cyclosporin presented with shortness of breath, fever, and productive cough for two days. Initial chest x-ray showed consolidation, and patient was started on ceftriaxone and azithromycin. Treatment was escalated to methicillin-resistant Staphylococcus aureus (MRSA) and antipseudomonal coverage, but the patient became increasingly hypoxic. Bronchoalveolar lavage (BAL) revealed N. Otitidiscaviarum. The patient also developed an abscess on his right thigh; cultures from incision and drainage again showed N. Otitidiscaviarum. He also acutely developed visual hallucinations and showed multiple abscesses on a brain MRI. Patient was induced with meropenem, trimethoprim-sulfamethoxazole (TMP-SMX), and linezolid, following which he showed significant improvement. He completed maintenance treatment with minocycline and moxifloxacin for around a year with improvement in CT Chest and MRI brain.

Discussion:
Nocardia is an aerobic gram-positive microorganism found ubiquitously in the environment. Primary exposure occurs through inhalation of spores in soil. Opportunistic infection in immunocompromised hosts is the brunt of Nocardia cases. Nocardia predominantly affects patients with impaired T-cell mediated immunity, such as seen in solid organ or stem cell transplant, acquired immunodeficiency syndrome (AIDS), or patients on chronic glucocorticoids. N. Otitidiscaviarum is seen in 0.3 to 2.9% of all Nocardia infections. Pulmonary infection is the most common presentation with radiologic findings of nodules, consolidation, or cavitary lesions. Disseminated nocardiosis occurs from hematogenous or contiguous spread and presents with brain abscesses most commonly, along with soft tissue abscesses. Disseminated nocardiosis is clinically challenging to distinguish from aspergillosis, Mucormycosis, Mycobacterium tuberculosis, or Staphylococcus aureus with septic emboli. Diagnosis is made with tissue biopsy, modified acid-fast staining, and microscopy, which shows filamentous branching. TMP-SMX is the drug of choice, with alternatives such as minocycline and linezolid. Combination drug therapy is the standard of care and is continued for 6 to 12 months.
Importance of Video Capsule Endoscopy in Detecting Obscure Small Bowel Bleeds

Acute gastrointestinal (GI) bleeding is a serious medical condition and a common cause of admission with a mortality rate of 5-10%. It is estimated that a small fraction of GI bleeds is of obscure origin with a normal EGD (esophagogastroduodenoscopy) and colonoscopy. Small bowel bleeding accounts for 5% of all GI bleeding and is the most common indication for video capsule endoscopy (VCE). VCE is a novel procedure with high sensitivity to detect a possible obscure gastrointestinal bleeding.

The patient is a 51-year-old male with a history of pulmonary embolism treated with ongoing anticoagulation who presented for shortness of breath, weakness, and dizziness over the past couple of months. The patient's physical examination was unremarkable. The patient was found to have symptomatic iron deficiency anemia with a hemoglobin of 6.9 and heme-positive stool. He underwent an EGD and colonoscopy, which showed no significant abnormality to explain iron-deficiency anemia. Therefore, the patient was advised to undergo a VCE for his small bowel, which showed a bleeding polypoid lesion in the distal ileum. CT endoscopy showed a fat-containing polypoid lesion with peripheral enhancement and connected to ileal loops with a fat-containing stalk in the distal ileum. Imaging findings were consistent with Meckel's diverticulum. No other bowel lesions are noted. The patient was referred for surgical small bowel resection. Surgery revealed a long-stalked polypoid mass associated with an ulcerated inverted Meckel's diverticulum in the distal ileum.

In patients who have overt iron deficiency anemia, a colonoscopy and EGD should be considered. However, if the upper and lower endoscopy is negative, further investigation of the small bowel may be necessary. Capsule endoscopy is the preferred initial diagnostic test for small bowel evaluation in patients with overt bleeding. VCE can help detect sources of bleed in the small bowel such as malignancy. Early detection is important in the case of malignancy for optimal outcomes. In our case, the patient had a Meckel's diverticulum which is rare and usually clinically silent, but can ulcerate and become a source of bleeding. Furthermore, if a source of bleed is detected, it may be rectified by surgical intervention.
Pancreatic Somatostatinoma Associated with Neurofibromatosis Type 1 - A Case Report

BACKGROUND: Somatostatinoma is a rare neuroendocrine tumor often associated with neurofibromatosis type 1, Von Hippel Lindau syndrome, and tuberous sclerosis. The incidence of somatostatinoma is estimated to be 1 in 40 million. Somatostatinomas have an insidious growth, and as a result, it presents in later stages as a malignant disease. Somatostatinoma presenting with “somatostatinoma syndrome,” which consists of diarrhea, diabetes, and gallstones, is uncommon.

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CONCLUSION: Although somatostatinoma presents at a later age given the insidious growth, it should not be missed in younger individuals who present with typical symptoms of somatostatinoma syndrome. Given its association with genetic disorders, effort should be made to distinguish sporadic tumors from those associated with genetic conditions.
Systemic Lupus Erythematosus Mimicking a Cerebrovascular Accident

Introduction:
As the name implies, systemic lupus erythematosus (SLE) is a systemic autoimmune disease in which many different organ systems can be involved. This means that the disease varies in how it presents clinically, however the most common presentation tends to include vague symptoms such as malaise and fevers, along with some version of cutaneous and/or musculoskeletal findings. These nonspecific presenting symptoms can pose as a clinical challenge in arriving at a definitive diagnosis, as alternative diagnoses are often considered first based on which organ systems are involved at time of initial evaluation.

Case presentation:
We present a case of a 73-year-old male with a past medical history of remote rheumatoid arthritis (RA) and autoimmune hepatitis not currently being treated, presenting with confusion and expressive aphasia for a duration of one hour. Due to concerns for a cerebrovascular accident (CVA), he was started on a tPA infusion. Shortly thereafter, his lab testing revealed a platelet count of 4, and the tPA was immediately stopped. Extensive imaging of the brain was unremarkable for any signs of ischemia. With conservative management alone, our patient’s mentation completely resolved within two days of hospitalization. Despite four platelet transfusions and treatment for presumed immune thrombocytopenic purpura (ITP), his platelet count remained below 20 throughout his hospital stay. Upon subsequent lab testing for antinuclear antibody (ANA), anti-double stranded DNA (anti-dsDNA) and complement levels, a diagnosis of SLE was made with an ANA titer of 1:1280, as well as positive anti-dsDNA and low complement level.

Discussion:
Neuropsychiatric SLE can manifest in up to 75% of lupus patients, the most common symptoms including headache, mild cognitive dysfunction and mood disorders. There are, however, a number of ways in which neuropsychiatric SLE can present in the central nervous system (CNS). Given that extensive imaging of the brain parenchyma was unremarkable, our patient did not likely experience a true CVA or myelopathy, but rather a blend of cognitive dysfunction and acute confusional state. These CNS manifestations along with thrombocytopenia alone posed as a diagnostic challenge for our patient.
A Rare Case of Nodular Pulmonary Amyloidosis

Introduction:
Amyloidosis is a disorder caused by misfolding of autologous protein and its extracellular deposition as fibrils, resulting in vital organ dysfunction and eventually death. Pulmonary amyloidosis may be localized or a part of systemic amyloidosis. Nodular pulmonary amyloidosis is usually asymptomatic and has an incidental finding on chest radiography. Here we present a case of symptomatic pulmonary nodular amyloidosis.

Case presentation:
A 78-year-old lady presented to the Emergency Department complaining of worsening shortness of breath for the past few months not related to exertion. She is on lubricant eye drops for the past many years due to xerophthalmia. Physical examination and basic labs were unremarkable. Her Chest X-ray and CT scan showed numerous bilateral pulmonary nodules, including a suspicious spiculated 11 cm-sized nodule in the left lower lobe. PET-CT scan showed faint FDG activity in the lung nodules without any lymphadenopathy. Due to the suspicion of malignancy, she underwent right upper lobe wedge resection, and the biopsy showed evidence of nodular amyloidosis.

Due to that, an autoimmune workup was done, which showed positive ANA of 1:1280 (speckled pattern), strongly positive SS-A/Ro and SS-B/La antibodies (>8.0 Elisa Units), and elevated RNP antibodies (3.0 Elisa Units). Rest of the workup including complement levels, immunoglobulin levels, Anti Sm and ds-DNA antibodies were negative. Her monoclonal protein serum screen showed increased kappa (69.1 mg/L) and lambda light chains (31.5 mg/L). Serum protein electrophoresis was negative. The diagnosis of nodular pulmonary amyloidosis with AL/AH (mixed immunoglobulin light chain/heavy chain) was confirmed.

Discussion:
Nodular pulmonary amyloidosis usually represents localized AL (immunoglobulin light chain) or AL/AH (mixed immunoglobulin light chain/heavy chain). If multiple lung nodules are seen on imaging, it is imperative to obtain a biopsy for evaluation of malignancy amongst other pathologies. On confirmation of nodular amyloidosis, it is recommended to evaluate the presence of an indolent B-cell lymphoproliferative disorder such as extranodal marginal zone lymphoma or mucosa-associated lymphoid tissue (MALT lymphoma) due to its frequent association. As nodular pulmonary amyloidosis is usually localized, conservative excision is usually curative and the long-term prognosis is excellent.
Adjunctive Treatment to Restore Pegloticase Efficacy for Refractory Gout

Introduction:
Pegloticase infusions are an effective therapy for severe gout refractory to first-line agents but have a high discontinuation rate due to development of anti-drug antibodies drastically reducing its effectiveness and increasing the risk of infusion reactions. However, addition of immunomodulating therapies as a pegloticase adjunct can prevent the immunologic response and restore its efficacy.

Case Description:
A 56-year-old male with a history of chronic severe tophaceous gout, was treated with pegloticase infusions. Initially, low uric acid levels were achieved but again increased after repeated infusions demonstrating resistance to the medication. Adjunctive treatment with methotrexate 10 mg weekly along with halving the infusion rate restored pegloticase response and his uric acid levels remained undetectable for the remainder of the treatment.

Discussion:
Pegloticase is a recombinant polyethylene glycol-conjugated form of uricase which catalyzes uric acid into an easily excreted form. In patients on pegloticase therapy, plasma uric acid levels function as a marker to identify patients who have developed antibodies against the drug. Uric acid levels above 6 mg/dL on two consecutive occasions warrants discontinuation of the drug to prevent subsequent infusion reactions. The addition of immunomodulating therapies such as methotrexate have been shown to restore efficacy of pegloticase and prevent infusion reactions enabling sustained treatment and improved outcomes.
Primary Colonic B-Cell Lymphoma in a Young Patient

Introduction:
The development of extranodal NHL can be a diagnostic challenge despite the fact that 30% of the cases are extranodal and involve the gastrointestinal tract. The incidence of a primary colonic lymphoma is rare, especially in young patients.

Case:
A 21-year-old man initially presented to our emergency department (ED) with abdominal pain, weakness, rectal bleeding, and anemia. Two months prior to this admission, he presented to an ED in Colorado with rectal bleeding and abdominal pain. He was diagnosed with gastroenteritis and discharged with antibiotics. He was evaluated in Ohio for syncope and profound anemia (hemoglobin of 4.2) two weeks later. A CT scan of the abdomen and pelvis demonstrated right colon wall thickening. He was diagnosed with inflammatory bowel disease and discharged on prednisone.

At this presentation, he reported a 15-pound weight loss and CT imaging demonstrated significant right colon wall thickening with a 19cm “mass-like” lesion. A subsequent colonoscopy showed a large, ulcerated, partially obstructing right colon mass consistent with malignancy. Histology demonstrated a high-grade B-cell lymphoma that was CD20 positive by immunohistochemical staining. Unfortunately, the patient was discharged from our facility at his request before definitive therapy could be undertaken.

Two weeks later, he presented to a different ED with bloody diarrhea, abdominal pain, and vomiting and was found to have perforation of the cecum with free air. He underwent an exploratory laparotomy with a stormy postoperative course and eventually died from post-surgical complications.

Discussion:
Although a primary colonic lymphoma is exceedingly rare, especially in the young population, this case is instructive as it is common to overlook malignancy in the young that presents with gastrointestinal symptoms. The patient was seen in 2 separate hospitals and treated symptomatically even when he presented with profound anemia (hemoglobin of 4) and an abnormal CT scan of the right colon. Presentation of the disease can vary, however, should be considered and recognized in younger patients to avoid delays in proper management, which could lead to severe complications, as illustrated by this case. Given its rarity, no large trials have been conducted to evaluate optimal treatment.
Malignant Cardiac Tamponade: A Complication of Untreated Breast Cancer

Case Description: A 36-year-old woman presented to clinic with complaints of progressive shortness of breath for two weeks that was worse on exertion and in the supine position; leaning forward improved her symptoms. She had swelling of both legs but no chest pain, cough, sputum production, or fever. Her medical history is significant for invasive ductal carcinoma of the right breast that is locally aggressive with lymphatic and osseous metastases; this was complicated by recurrent right-sided malignant pleural effusion requiring routine thoracentesis. She received her cancer diagnosis two years prior and was recommended surgery, chemotherapy, and radiation. However, she declined treatment as it did not align with her health beliefs.

In clinic, she was tachycardic and tachypneic but not hypoxic. Her examination was remarkable for labored breathing, decreased bibasilar breath sounds, and distant heart sounds. Electrocardiogram demonstrated electrical alternans. Patient was sent for an urgent same-day echocardiogram, which demonstrated a large circumferential pericardial effusion; echocardiographic evidence of cardiac tamponade was seen with systolic and diastolic right ventricular wall collapse.

The patient underwent emergent pericardiocentesis with drainage of 800 ml of bloody fluid that was positive for malignant cells. She experienced significant relief of her symptoms post-procedure, however, the pericardial effusion re-accumulated within days. She underwent a right pericardial window with placement of an indwelling pleural catheter.

Impact/Discussion: Carcinomatous pericarditis is a rare complication of locally aggressive breast cancer by direct extension of malignant cells into the pericardium which creates a malignant pericardial effusion. Accumulation of pericardial fluid increases intrapericardial pressures, decreases cardiac filling, and eventually compromises cardiac output as seen in tamponade. Symptoms are typically nonspecific and include dyspnea, chest discomfort, and peripheral edema; patients also have tachycardia and tachypnea. However, since this effusion accumulates chronically, symptoms of muffled heart sounds, hypotension, and jugular venous as distension described in Beck’s Triad are not always seen.

Conclusion: Internists should maintain a high index of suspicion for malignant cardiac tamponade in at-risk patients. Although there is no definitive treatment, management is generally palliative to prevent sudden death from tamponade and improve the patient’s quality of life by relieving symptoms.
Strongyloides Hyperinfection Syndrome and Disseminated Disease After Heart Transplantation

Introduction: Strongyloides Hyperinfection Syndrome (SHS) is a rare, life-threatening condition that is usually reported in transplant patients and immunocompromised. Here we present a case of SHS with disseminated disease in a patient with prior heart transplant.

Case presentation: A 43-year-old female with recent heart transplant three months prior to presentation secondary to non-ischemic cardiomyopathy, presented to the emergency department with several complaints of dyspnea, fatigue, vomiting and poor appetite for 10 days. Three weeks prior to her current presentation, she was hospitalized for Escherichia Coli and Methicillin Resistant Staphylococcus Epidermidis Bacteremia for which she completed Ceftriaxone course and discharged on Vancomycin. On presentation, she was in shock of unclear etiology, all routine infectious workup was unremarkable including blood, sputum, urine, and stool cultures. Laboratory evaluation demonstrated evidence of diabetic ketoacidosis and pancreatitis which she was treated for; however, patient became hemodynamically unstable requiring mechanical ventilation and vasopressor support. Bronchoscopy with Broncho-alveolar lavage (BAL) was done which demonstrated Strongyloides parasites. On further investigation, another recipient from the same donor developed Strongyloides infection and the donor serology was also positive. Patient was started on oral Ivermectin, but she developed worsening hypoxemia requiring Veno-Venous Extra Corporeal Membrane Oxygenation (VV-ECMO). Subsequent Bronchoscopy showed increased burden of live Strongyloides, for which she was switched to Subcutaneous Ivermectin due to concerns of malabsorption secondary to severe ileus, and Albendazole was added. Repeat BAL after 2 weeks demonstrated eradication of Strongyloides. Her course was complicated by Vancomycin Resistant Enterococcus meningitis and Aspergillus Pneumonia for which she received appropriate treatments, and ECMO was successfully decannulated. Upon writing of this report, the patient remains ventilator dependent and encephalopathic, pending further workup, however she has poor prognosis overall.

Discussion: Strongyloides can have non-specific and unusual organ involvement in immunocompromised patients, particularly those with a history of transplant. It is usually suspected in patients from endemic country, in this case the donor was from Mexico. No guidelines recommend routine screening for Strongyloides, however clinicians should have a high index of suspicion for such infection in an immunocompromised patient who has signs of shock with no clear explanation.
Checkpoint Inhibitor Immunotherapy-Induced Colitis for BRAF V600E-Mutated Melanoma

Introduction: The rapid expansion of immunologic agents in targeted chemotherapy raises many questions as to how patients may respond when different combinations of immunologic agents are given. In the case of Cemiplimab plus Pembrolizumab (PD-1 inhibitors), a rare but serious side effect of severe immune mediated colitis has been reported.

Case Presentation: A 69-year-old female with stage IV BRAF V600E melanoma presented with a 11-day history of nausea, vomiting, abdominal pain and watery diarrhea with urgency. A few months prior to admission, she began a new immunotherapy regimen of Cemiplimab (6 cycles from 05/2021 to 08/2021) plus Pembrolizumab for 2 cycles (11/2021). Lab work up upon admission included infectious stool studies which were negative and stool calprotectin >3000 mg/kg. CT abdomen/pelvis noted liquified stool. Colonoscopy demonstrated erythematous, ulcerated and vascular-pattern-decreased mucosa at the terminal ileum, ileocecal valve, cecum, ascending colon, and rectum. Biopsies were later found to be consistent with checkpoint-inhibitor induced colitis. Treatment was initiated with solumedrol 2 mg/kg daily however she had persistent watery diarrhea requiring the initiation of infliximab.

Discussion: This case illustrates the continued need for side effect surveillance of immunotherapy, especially when used in combination. Furthermore, providers should be less hesitant to pursue steroid treatment when checkpoint-inhibitor induced colitis is suspected and infectious etiologies are ruled out.
CMV Pancreatitis: An Unseen Harbinger of Acute Leukemia

A 51-year-old male with no past medical or surgical history presented to the emergency department with a three day history of sharp epigastric pain. He had no history of cholelithiasis or alcohol use, and did not take any medications. His vital signs were within normal limits. His physical exam demonstrated epigastric tenderness to palpation. He had an elevated lipase of 137 IU/L and CT scan of the abdomen and pelvis demonstrated acute, uncomplicated pancreatitis. Additional laboratory workup demonstrated pancytopenia with a hemoglobin of 11.1 g/dL, WBC count of 0.9 k/uL (absolute neutrophil count of 0.13 k/uL), and platelet count of 51 k/uL. Serum triglyceride and calcium levels were normal, and an autoimmune workup was unrevealing. His infectious work up demonstrated both Cytomegalovirus (CMV) IgM and IgG positivity. CMV viremia via PCR testing was negative. Peripheral smear showed blasts (3%) and Auer rods were present. The subsequent bone marrow biopsy revealed a PML/RARA transfusion gene, confirming the diagnosis of APL. This clinical picture was consistent with a diagnosis of APL presenting as CMV pancreatitis.

There are no cases to our knowledge that have noted such a presentation of APL, particularly in non-transplant, treatment-naive patients. After a primary CMV infection (usually asymptomatic), CMV progresses to the latency period. In immunocompromised states, latent CMV infections may reactivate and cause significant symptoms. Given our patient’s positive IgM and IgG, CMV reactivation is a possibility as compared to a primary CMV infection. Patients with myeloid tumors have been noted to have a 3.9% rate of CMV antigenemia, which is notably lower compared to lymphoid tumors (13.6%). This further highlights our case’s unique presentation.
A Common Presentation of Acute Retroviral Syndrome with an Uncommon Presentation of Associated Rash

Over the past few years, the incidence of Human Immunodeficiency Virus (HIV) in the United States has consistently been around 40,000 cases diagnosed annually. In addition, an estimated 162,500 individuals are infected with HIV and remain undiagnosed. Initial presentation of acute HIV, known as Acute Retroviral Syndrome (ARS), consists of a dynamic constellation of mononucleosis-like symptoms ranging from cough and fever to fatigue and rash. Diagnostic testing results can be inconclusive due to the time-dependent process of viral inoculation to host immunological seroconversion. Taken together, this can make diagnosis of HIV difficult. Meanwhile, individuals with acute HIV infection pose a great transmission risk due to high plasma viremia and viral shedding.

Pityriasis Lichenoides et Varioliformis Acuta (PLEVA) is a rare dermatologic condition that manifests as a full body vesicular rash. Although the exact etiology of PLEVA remains unclear, PLEVA has been correlated with infectious insults, both viral and bacterial. In regard to HIV, PLEVA has only been reported with early and middle stage HIV, not late-stage illness. PLEVA is often a self-limiting condition, that tends to resolve after initiation of treatment for the primary infectious insult. For HIV, PLEVA skin lesions commonly clear within a few weeks of Acute Retroviral Therapy (ART).

We describe a case of a middle-aged man with a history of sickle cell trait who presented to the emergency department with acute flu-like illness and mucocutaneous vesicular rash involving the entire body, including the palms and soles of the hands and feet. He was subsequently diagnosed with ARS. Given his presenting rash, there was initial concern for disseminated illness in an immunocompromised patient, including varicella and molluscum contagiosum. The patient was started on intravenous acyclovir, in addition to antibiotic coverage for pneumonia. Ultimately, a skin biopsy demonstrated PLEVA. Initial treatment of antibiotics and acyclovir was stopped, and he was started on ART with resolution of his ARS symptoms and associated PLEVA rash. Recognition of PLEVA can lead to earlier testing, diagnosis and treatment of HIV, helping to slow the rate of transmission while improving clinical outcomes.
Newly Diagnosed Graves Disease with Congestive Heart Failure as the Initial Presentation

Hyperthyroidism produces its effects on the cardiovascular system predominantly through the effects of T3, which increases heart rate, myocardial contractility, cardiac output, and left ventricular mass. The objective of this clinical case report is to highlight the importance of having a high index of suspicion and warranting an extensive workup for thyrotoxicosis in the setting of a patient presenting with initial symptoms of congestive heart failure. The clinical case features a 61-year-old female with a past medical history significant for uterine cancer status post hysterectomy and salpingo-oopherectomy, who presented to the hospital with a complaint of worsening shortness of breath on exertion for the past month. Additionally she complained of wheezing, productive cough with clear sputum, bilateral lower extremity swelling, hoarseness in her voice, feeling hot all the time, and a 100 lb weight loss in the last year. On admission, the patient was hypertensive (BP 159/97), tachycardiac (HR 120), and had an O2 saturation of 93% on room air. On physical exam, the patient was tachycardiac with increased respiratory effort while lying supine, auscultatory crackles at the bases of the lung, and 2+ pitting edema in the lower extremities. Her thyroid was non-palpable, and no signs of lid lag or retraction were observed. Initial labs demonstrated hypokalemia, elevated BNP, anemia, and a negative troponin. EKG was significant for sinus tachycardia with evidence of supraventricular premature complexes. Chest x-ray revealed cardiomegaly, bilateral pulmonary vascular congestion, and bilateral pleural effusions. Echo was consistent with a non-ischemic cardiomyopathy. She was initially started on Metoprolol and intravenous Lasix. Further investigation revealed a low TSH, an elevated T3 and T4, and elevated TSH receptor antibodies consistent with Graves disease. In the following days, adjustments were made from Metoprolol to Carvedilol and from intravenous to oral Lasix. Additionally, she was started on Hydralazine, Aspirin, Spironolactone, Atorvastatin, and Methimazole for blood pressure and thyroid control. Upon discharge, the patient’s symptoms completely resolved. Although heart failure and thyroid control medications were the foundation for management, additional research needs to be conducted to measure the efficacy of these therapies.
An Atypical Coinfection of Pneumocystis jirovecii and Aspergillus fumigatus Following COVID-19 and Prolonged Steroid Use

INTRODUCTION: Pneumocystis jirovecii pneumonia (PJP) is a common AIDS-defining illness and is associated with glucocorticoid use in a dose and duration-dependent manner in non-HIV patients.

CASE REPORT: A 62-year-old male presented with 10 days of worsening shortness of breath, hypoxia, and nasal congestion. He was hospitalized for COVID-19 30 days prior and discharged on supplemental oxygen and a prolonged course of dexamethasone 6 milligrams (mg) daily continued till presentation. His past medical history included uncontrolled type 2 diabetes mellitus, hypertension, and obstructive sleep apnea. On examination, he was febrile, tachycardic, tachypneic, and hypoxic requiring 6 liters/minute oxygen. Diminished breath sounds were auscultated bilaterally. Chest x-ray showed diffuse bilateral patchy opacities, re-demonstrated on CT chest as ground-glass opacities, and two new cavitary lesions (largest axis 3.7 cm) in the left upper lobe. Vancomycin, cefepime, and metronidazole were started empirically. CT head was ordered due to concern for Mucormycosis given uncontrolled diabetes and nasal congestion, showing left ocular involvement without intracranial extension, prompting initiation of amphotericin B. Further, otorhinolaryngologic and ophthalmologic workup were not supportive for Mucormycosis. Finally, sputum PJP polymerase chain reaction (PCR) testing returned positive and bronchoscopy revealed PJP and rare Aspergillus fumigatus in the context of negative fungitell and galactomannan. Trimethoprim/sulfamethoxazole and voriconazole were started. Repeat CT chest at 6 weeks showed reduced cavities and voriconazole was extended an additional four weeks.

DISCUSSION: Judicious use of steroids for COVID-19 must be considered with initiation of PJP prophylaxis when at least 20 mg of prednisone equivalents are prescribed daily for four weeks. Although rare, PJP may present as cavitary lesions in AIDS. Additionally, fungitell has been extensively studied in HIV related PJP with scarce description in non-HIV patients. Mucormycosis was seen during the pandemic in India, typically with negative fungitell and galactomannan. COVID associated pulmonary aspergillosis has been documented with critical illness and cavitary lesions as 'halo' signs on imaging, not seen in our patient.

CONCLUSION: This case highlights an atypical post-COVID-19, prolonged steroid outcome. Further studies are needed to evaluate the role of fungitell and galactomannan in PJP and Aspergillus in this population of non-HIV, post COVID patients.
A Case of Oxybutynin Triggering an Acute Intermittent Porphyria Attack

Acute intermittent porphyria (AIP) is an autosomal dominant metabolic disorder characterized by a deficiency in heme biosynthesis, which leads to accumulation of heme precursors. AIP is specifically caused by heterozygosity for a mutation in the porphobilinogen deaminase (PBGD) gene leading to the accumulation of upstream, neurotoxic metabolites: porphobilinogen (PBG) and aminolaevulinic acid (ALA). Symptoms include abdominal pain, peripheral and autonomic neuropathies, neuropathic bladder dysfunction, and psychiatric manifestations. Our 50-year-old female with a medical history significant for acute intermittent porphyria (AIP) and hypertension presented with a 5-day history of headaches, abdominal pain, and nausea. Her blood pressure was found to be in the 200s/110s on admission. Patient had reported adherence to all of her home medications including her anti-hypertensives. She denied any known recent triggers to her AIP, which included stress and/or alcohol use in the past. Neuroimaging did not show an acute pathology. A few days later, her urine porphobilinogen and porphyrins were found to be elevated and she was diagnosed with an AIP attack. Clinical pharmacist reviewed the patient’s home medication list and noted that the patient had been recently started on oxybutynin by her primary care physician. The oxybutynin was discontinued, and the patient was started on a 4-day hemin infusion per hematology with resolution in her headaches, abdominal pain, and nausea. Triggers for AIP attacks include medications, starvation, stress, and alcohol. Most of the unsafe medications are hepatic cytochrome P450 or hepatic ALAS1 inducers such as some antibiotics, anti-epileptics, or barbiturates. Oxybutynin is reported very rarely in literature triggering AIP attacks; however, it is listed as unsafe in multiple AIP medication databases. The evidence for this is very limited with the consensus being that oxybutynin is metabolized primarily in the liver by CYP3A4 substrate. This case serves to alert clinicians to carefully review the medication list of patients presenting with an AIP attack, which plays a crucial role both in treating and preventing future attacks.
Ehlers Danlos Type II and Recurring Bouts of Pancreatitis - A Literature Review and Case Report

Introduction:
Ehlers Danlos Syndrome (EDS) is a group of inheritable disorders that results in decreased connective tissue function and assembly that can have a myriad of symptoms and multi-system involvement. Although commonly known to be associated with severe fatigue, hypermobile joints, elastic skin, easy bruising and bleeding tendencies, and aortic dissections, not much has been fully characterized about gastrointestinal syndromes in Ehlers Danlos patients. In this case report, we discuss a patient case presenting with a family and personal history of Ehlers Danlos and multiple bouts of pancreatitis. Few other cases have been reported showing the association of chronic pancreatitis in patients with Ehlers Danlos syndrome, and this case report serves to add an additional incidence of such association, potentially indicating another avenue of preventative medicine for Ehlers-Danlos patients.

Case report:
A 30-year-old male with a past medical history of EDS type II, and recurrent pancreatitis presented to our ER with severe nausea, vomiting, fever, and epigastric pain radiating to the back. He denied any recent alcohol use, hypertriglyceridemia, or known history of gallstones. He complained of severe pain in the epigastric region that radiated to the back; severe epigastric tenderness was noted on the physical exam. CBC showed white blood cells of 11,000/μL, ALT was 284 and AST of 161, lipase was elevated 138 IU/L. triglycerides was 251 mg/dL. RUQ u/s did not show any signs of gallstones or bile duct stones. Computed tomography of the abdomen obtained demonstrated inflammatory changes and scant fluid around the pancreas head, neck, and uncinate process. Diagnosis of acute on chronic pancreatitis was established. He was managed with IV fluids, pain, and nausea control. Liver function tests, lipase levels, and white blood cell counts were monitored for four days, and they improved and the patient was discharged after four days.

Discussion:-
Few case reports were found that showed a link between EDS and pancreatitis. In those cases and in our patient, no other causes for pancreatitis were seen. Though no current studies exist on the interplay of genetics between pancreatitis and Ehlers Danlos, this could be yet another avenue for further research in EDS patients.
Levetiracetam (Keppra) is one of the most widely used anticonvulsants due to its numerous benefits including high efficacy in both focal and generalized seizures, availability as an intravenous form, affordability over the last 15 years, relatively safe side effects, and advantageous pharmacokinetic profile. Keppra acts as a neuromodulator by reducing neurotransmitter release through binding to the brain synaptic vesicle protein SV2A (1). It is often the drug of choice in patients with hepatic impairment due to its renal clearance. A minute number of cases related to liver toxicity have been reported with the use of Keppra (2).

We hereby present a case of a 37 year-old male with a history of meningococcal meningitis at age 13, controlled asthma, no alcohol or drug abuse, presenting with new onset seizures and fever. Vitals showed normotension, tachycardia of 108, and fever of 103.0 F. Labs indicated leukocytosis of 20.3, elevated ammonia of 417, lactate of 22.8, and a negative hepatitis A, B, and C panel. The patient was alert and oriented times three and able to give a brief review of systems. Computed tomography of the head was negative for intracranial abnormalities and lumbar puncture was performed. Patient’s tonic clonic seizure in the emergency department was aborted with ativan 2 milligrams once followed by loading dose of levetiracetam 2 grams and maintenance dose of 500 mg twice daily. Patient was started on antibacterial, antiviral, and steroids for suspected meningitis and admitted to intensive care for close neurovascular monitoring. A few days prior to admission, patient’s liver enzymes were within normal range and became significantly elevated after levetiracetam with an alanine aminotransferase (ALT) of 408 and aspartate aminotransferase (AST) of 292 while gradually increasing during hospital course. Given the patient’s acute transaminitis, levetiracetam was discontinued and lacosamide 50 milligrams twice daily was initiated. Labs demonstrated a downtrend in aminotransferases thus confirming a rare suspicion.

This case highlights one of the rare side effects of levetiracetam causing transaminitis in a young individual with a fully functioning liver. Monitoring aminotransferases is not widely accepted when starting levetiracetam; however, this case presents hepatotoxicity as a major concern.
Pericardial Tamponade as an Initial Manifestation of Systemic Lupus Erythematosus

Acute pericardial tamponade has multiple causes including malignancy, uremia, post-myocardial infarction, iatrogenic, and infection. Systemic lupus erythematosus (SLE) can present with acute pericardial tamponade; however, it is rarely reported in the literature.

A 33 year-old Italian male with obesity, asthma, and essential hypertension presented with bilateral lower extremity edema, pleuritic chest pain, and dyspnea. He reported intermittent joint pains, fatigue, 60 pounds of unintentional weight loss, and anemia over the past year. Pertinent labs included BNP 165, troponin 18 > 19 ng/L, and normal thyroid levels. EKG showed sinus tachycardia and telemetry later showed electrical alternans. Chest x-ray showed no acute cardiopulmonary process. Transthoracic echocardiogram showed a moderate-sized pericardial effusion circumferentially around the heart with no evidence of cardiac tamponade. Physical exam was significant for persistent sinus tachycardia, pulsus paradoxus, jugular venous distension, and muffled heart sounds. He underwent a pericardial window with symptomatic relief, with fluid negative for malignant cells or infection. With elevated dsDNA titers (1:160), hypocomplementemia, anemia, serositis, and arthritis, the patient was diagnosed with SLE. He was managed with IV methylprednisone 60 mg every 8 hours for 5 days and discharged on prednisone 60 mg daily, azathioprine, and hydroxychloroquine. Prednisone 60 mg was tapered to 10 mg daily within 3 weeks but this caused him to have a recurrence of the pericardial effusion, requiring another pericardial window. He was continued on prednisone 60 mg daily with a slower taper and an increase in azathioprine, with no further recurrences.

This is a rare presentation of pericardial tamponade as an initial manifestation of SLE in a young male, when usually it is reported mostly in young females. Should our patient develop recurrent pericardial effusion despite dose adjustments in steroids, azathioprine, and hydroxychloroquine, literature review suggests tocilizumab as a possible successful treatment agent. Prompt recognition of SLE as a differential in those with pericardial effusion is needed in order to achieve targeted treatment to prevent recurrence, morbidity, and mortality. More research is needed regarding prognostic indicators of pericardial tamponade development and recurrence in order to determine optimal management.
Streptococcus Intermedius Hepatic Abscesses in the Setting of Previously Known Hepatic Cysts Without Periodontal Infection

Streptococcus intermedius is part of the Streptococcus anginosus group of bacteria that make up the normal flora found in the oropharynx, gastrointestinal tract, and genitourinary tract. It has proven to be one of the common pathogens associated with brain and liver abscess. Liver abscesses with Strep. Intermedius in immune competent patients is mostly seen with active periodontal infection. It is also seen in patients with associated comorbidities such as cancer and diabetes. Studies performed in 2014 demonstrated a correlation between dental manipulation and the seeding of Strep. Intermedius in the liver via hematogenous spread.

We present a rare case of multiple liver abscesses six months after a routine dental procedure. Our patient is a 64-year-old Caucasian female with a past medical history of hypertension, hypothyroidism and recent COVID-19 infection who presented with worsening two-day history of increased generalized weakness, fevers, chills, chest pain, shortness of breath, and epigastric abdominal pain with radiation to her right upper abdominal quadrant and back. Upon presentation, patient had hypotension, tachycardia, and low-grade fever. Labs showed elevated transaminases and leukocytosis. CT imaging revealed hypo dense smaller right and large left hepatic lesion and multiple small lesions in both lobes of the liver. She was started on broad-spectrum intravenous antibiotics. Interventional radiology performed a CT guided biopsy with drainage of the large left hepatic abscess, which grew streptococcus intermedius. Blood cultures came back positive for streptococcus intermedius. A transthoracic echocardiogram was inconclusive for any valvular vegetation; therefore, a Trans esophageal echocardiogram was done which was negative for endocarditis. During our focused investigation and history taking, we found that patient had regular dental cleaning procedure done six months ago. Patient initially received IV vancomycin and piperacillin/tazobactam for bacteremia and transitioned to IV Ampicillin/sulbactam for 6 weeks after sensitivities. She was discharged home safely with appropriate follow-up.

This case illustrates the importance of obtaining accurate history and timely management of liver abscesses, which can be fatal if missed. Our case differs from previously investigated cases, as the patient did not have an active dental infection, ongoing dental work, or recent dental procedures.
EBV Meningoencephalitis in an Immunocompetent Young Adult

Epstein-Barr Virus (EBV) infection is common among young adults, however neurologic manifestations are exceedingly rare. EBV infection of the CNS can present as meningitis, encephalitis, transverse myelitis, or acute disseminated encephalomyelitis (ADEM). Presentation of neurologic EBV infection is nonspecific, requiring CSF analysis. Though there have been reports documenting neurologic EBV infection, its rarity precludes large-scale studies, including meta-analysis.

We present a case of EBV meningoencephalitis in a 30-year-old female with a history of erythema nodosum (EN) following strep infection and infectious mononucleosis (IM). Eight days prior to presentation she developed bilateral lower extremity rash similar to previous EN followed by lower lip ulceration. Family reported she became confused and exhibited signs of photophobia. Initially, she was alert and following commands but confused. Hours later she was lethargic and minimally arousable. CT and MRI brain w/w/o were unremarkable. She developed a high-grade fever and was started on empiric meningitis coverage. LP showed elevated protein with mild lymphocytic pleocytosis. Patient’s mental status normalized after 6 days and rash had improved. Though pending at the time of discharge, EBV PCR of CSF resulted positive shortly after discharge. Four months later, she presented for continued cognitive fog and headaches, believed to be sequelae of EBV meningoencephalitis.

EBV CNS infections stratified by age and immunologic status warrant further investigation due to the prevalence of IM in young, immunocompetent adults. Majority of EBV meningoencephalitis cases have been documented in children or immunocompromised adults. Little is known about EBV meningoencephalitis in immunocompetent adults. A case series done at the Mayo Clinic analyzed six cases of adult EBV encephalitis from 2000-2012. Presentation was nonspecific and MRI was suggestive of HSV-1. Greater than 80% recovered without sequelae. Another case series by Fujimoto et al. of 10 cases of EBV-related CNS infections from 1984-2002 found mean age was 36 years. Immunologic status was not specified in either series. Though the little data that exist demonstrate long-term effects of EBV are not widely observed and treatment itself is supportive, inclusion of EBV CNS infection as a differential can play an important prognostic role, especially for young, otherwise healthy patients.
Atrial Septal Mass Presenting as Lambl's Excrescence

Most atrial septal masses are found to be atrial myxomas. However, we hereby present a case of an interatrial septal mass that turned out to be lambl's excrescence, which are rare filiform fronds that originate at site of valve closure, most commonly the aortic valve leaflets, in contrast to atrial myxomas which are commonly found along the interatrial septum (2). It is important to distinguish the two as management of the two diseases can be different.

A male in his 70’s with dyspnea on exertion, history of sarcoma, and history of DVT was found to have 9x9 mm mobile mass attached to the atrial septum on routine TTE. TEE confirmed a mobile spherical mass attached near the fossa ovalis at the base of the anterior mitral valve approximately 12x8 mm. Patient underwent resection of the mass and pathology report subsequently demonstrated lambl's excrescence. Patient reported improved symptoms after surgery. Though atrial septal mass is most commonly associated with atrial myxoma, lambl's excrescence should not be excluded from the differential since it is similarly associated with heart failure symptoms and embolic strokes (2).

Though complications are similar, management of atrial myxoma and lambl's excrescence are different. While atrial myxomas are commonly managed via surgical excision, asymptomatic lambl's excrescence can be closely monitored with medical management; if patient has history of thromboembolisms and symptomatic lambl's excrescence, they can be put on anticoagulation. However, if patient continues to have embolic events, they will need surgical excision of the lambl’s excrescence (1). Thus, this atypical location of lambl's excrescence informs clinicians to keep a broad differential for atrial septal mass and to consider early anticoagulation prior to surgery on a case-by-case basis.

Inhaled Nitrous Oxide Abuse in the Elderly Leading to Subacute Combined Degeneration

Nitrous oxide (NO) inhalants, commonly available as "Whip-it" canisters, are used recreationally by teenagers and young adults for euphoria and relaxation. NO has been known to cause profound neurotoxicity via B12 deficiency with recurrent chronic abuse. However, its use in the elderly population is not well documented. In elderly or malnourished individuals with suboptimal stores of B12, NO use can predispose to neurotoxicity even in acute users.

Presenting a case of a 72 year old male with past medical history of arthritis and Prostate Cancer post brachytherapy, who presented with 3-week duration of progressive bilateral lower extremity weakness and gait ataxia. The weakness started insidiously, first noticed 3 weeks prior when he moved a heavy carpet at home. He had associated lower back pain, radiating down his posterior thigh. No reported trauma, fevers, chills, recent viral infections, new medications, loss of bowel or bladder control. He felt unsteady on his feet, with frequent falls, requiring use of a cane to ambulate. Initial imaging showed L1-L5 diffuse disc bulge with hypertrophic ligament of flavum, and moderate to severe L3-L4 central spinal canal stenosis. Based on physical exam findings, there was suspicion of Vitamin B12 deficiency, which was confirmed to be low- 104.1. On further eliciting history, he admitted to inhaling NO regularly with his friend for recreational purposes for more than 10 years. MRI was ordered which showed T2/STIR hyperintensity in the dorsal column of the cervical and thoracic spinal cord segments consistent with SACD. He was started on IV B12 1000 mcg injections daily along with Physical therapy, following which he had mild to moderate resolution of symptoms. He was discharged to sub acute rehab and instructed to refrain from NO in the future.

The above case reiterates the importance of taking a good social history, even in elderly patients who we do not usually suspect as an age group for NO inhalation abuse. Providers need to be aware of NO’s effects on the CNS, myelination, B12 metabolism, hypotension, and Cardiac complications. Patients, teenagers, and family members must be educated about these side effects to increase awareness and help reduce NO abuse.
Atrioventricular Fistula a Rare Complication of an Infected Heart

Introduction:
Infective endocarditis, an infection of the heart involving the endocardium and its interior structures is not an uncommon phenomenon, but its potential complications can make it a difficult disease to manage and identify. The sequelae can have devastating effects and delayed recognition can lead to increased morbidity and mortality.

Case Description:
A 65 year old male with a past medical history of Diabetes, Hypertension, BPH and confusion for the past 2 months presented to the hospital due abdominal pain and a left foot wound. The patient prior to his declining mentation was fully functional and a business executive. Two months prior to admission the patient was seen at an outside hospital due to a left foot wound. He was found to have cultures growing GBS in the left foot wound, blood and urine which was treated with IV antibiotics. On physical exam the patient was markedly confused, orienting only to self and occasionally place. He had decreased but equal strength in his lower extremities and had an apparent aphasia. Auscultation of the heart revealed a systolic murmur heard in the cardiac apex with radiation to the axilla. A transthoracic echocardiogram revealed a possible mitral valve vegetation and evidence of severe mitral regurgitation. A transesophageal echocardiogram showed a severe amount of blood flow from the left ventricle into the left atrium through a ruptured abscess located on the posterior mitral valve leaflet. An MRI of the brain revealed diffuse multifocal lesions in the bilateral cerebral hemispheres which were likely embolic from the infective endocarditis.

Discussion:
This case illustrates the potential complications of Infective Endocarditis and discusses a rare complication involving abscess rupture causing an Atrioventricular fistula and embolic strokes. This rare complication mimics severe mitral valve regurgitation and greatly affects the hemodynamics of the heart requiring a surgical approach to valve repair/replacement. This case will discuss infective endocarditis, review literature on perivalvular abscesses and present this rare occurrence of an atrioventricular fistula as the result of an abscess rupture.
Spontaneous coronary artery disease (SCAD) currently accounts for less than 0.5% of all ACS cases that undergo coronary catheterization. Of the total reported cases of SCAD, 25% of SCAD patients have been reported in women less than 50 years old. Thanks to increasing number to heart centers around the country, there is likely to be an expected increase incidence of SCAD patients, which makes it crucial to study the mechanism and presentation of disease, as the secondary complications of SCAD can have a high mortality rate. We present a 43-yr female with a chief complaint of chest pain with a history of hyperlipidemia, anxiety, stage III angina, and hypertension. Patient underwent coronary catherization due to outpatient stress testing due to moderate-high risk for ACS. Patient underwent diagnostic heart catherization after stress testing revealed concerning findings on EKG and cardiac imaging. During heart catherization, patient underwent cardiac arrest, heart catherization was stopped, and was stabilized and transferred to the ICU—coronary artery evaluation afterwards revealed dissection of the distal LAD. The challenge of SCAD is the diagnosis is only made by heart catherization, which is also the iatrogenic nidus for dissection to occur. We present this case in order to increase awareness of SCAD, as well as to discuss the management, explain the currently understood pathophysiology—in hopes to expand possible future alternatives to diagnose patients who are thought to be high risk for SCAD if undergoing routine heart catherization and reduce possible morbidity and mortality.
Spontaneous Tumor Lysis Syndrome in High Grade Endometrial Carcinoma: A Case Report

Tumor lysis syndrome is a rare cause of morbidity and mortality most commonly occurring in hematologic malignancies being treated with cytotoxic therapy. Rarely, it can occur in solid tumors. It is even more seldomly found in endometrial carcinomas. We present the case of a 64-year-old woman with high-grade endometrial cancer who presented with nausea, abdominal pain, and hyperkalemia with acute kidney injury. She initially did not show metabolic derangements consistent with tumor lysis syndrome on presentation. However, during her hospital course, patient had sudden onset of unresponsiveness with shallow, agonal breathing. Laboratory values were consistent with tumor lysis syndrome and patient was initiated on IV Rasburicase with aggressive fluid hydration and hemodialysis. Although she regained consciousness and able to avoid intubation, her clinical course deteriorated rapidly, and she remained lethargic after initiation of treatment. The patient was transitioned to comfort care by family two days after start of treatment due to poor prognosis. This case highlights the importance of recognizing and maintaining a suspicion of tumor lysis syndrome in patients with gynecological cancers as prompt treatment can potentially be life-saving.
A Rare Case of Jejunal Small Bowel Intussusception in an Adult

Introduction:
Intussusception is a form of bowel obstruction when one segment of the bowel telescopes inside another. It is commonly seen in children and presents with abdominal pain, bloody bowel movements, and a palpable mass. Here we present a rare case where intussusception is seen in an adult with no complaints of abdominal pain and no physical exam findings. Intussusception in adults is commonly treated with surgery when malignancy is suspected.

Case Presentation:
67 year old male with a history of coronary artery disease and atrial fibrillation presented for fatigue and shortness of breath. Patient was found to have a hemoglobin of 6 units and MCV of 68 on admission. He denied any overt signs of bleeding. Patient was taking Eliquis, Plavix, and aspirin. Patient was up to date on colon cancer screening. He denied abdominal pain, nausea, vomiting, loss of appetite, or weight loss. Patient was transfused one unit of blood and gastroenterology was consulted. Patient underwent colonoscopy and esophagogastroduodenoscopy (EGD) without identification of any source of bleeding. CT enterography demonstrated proximal jejunal small bowel intussusception extending 5 cm. Patient had a repeat EGD using a pediatric colonoscope to visualize the jejunum which revealed a lesion 50 cm to the ligament of Treitz and biopsies were taken. Biopsy showed tubular adenoma with focal high grade dysplasia. Patient then underwent a robot-assisted small bowel resection of the mass.

Small bowel intussusception commonly seen in pediatric populations, adult cases account for a minority of cases. Here we describe a male who presents with non-specific symptoms and low grade blood loss found to have a jejunal intussusception, etiology attributed to tubular adenoma which is a precursor of adenocarcinoma. It is important that physicians recognize that small bowel intestinal tumors are a rare cause of intussusception in adults and have an incidence of 0.7–1.7/100,000 of all gastrointestinal tumors. Intussusception is most commonly seen in the ileocolic area of the bowel, but small intestinal tumors may lead to them occurring in the jejunum. Though small intestinal tumors are rare, current recommendations advise wide excision of the lesion and surrounding tissues.
Everolimus Induced Pulmonary Toxicity

Everolimus-associated pulmonary toxicity is very rare, let alone manifesting as a pleural effusion. Two cases reported everolimus induced pleural effusion; one was in a breast cancer patient (1), the other in a cardiac transplant patient (2). Both were chylous effusion. Patients on everolimus often have malignancy and are more prone to having pneumonia due to their immunosuppressed status. Thus, when everolimus using patient presents with pleural effusions, one may easily be tempted to narrow down the etiology to pneumonia and malignancy. However, we hereby report a patient with past medical history of recurrent thoracic spinal meningioma with recent history of everolimus treatment who presented with an exudative pleural effusion which was thought to be secondary to everolimus.

A 70-year-old Caucasian female with a past medical history of recurrent thoracic spinal meningioma with recent everolimus usage presented with new onset of shortness of breath for one week duration. Patient was found to be hypoxic, however she did not have any fevers or white count. Computed tomography of chest showed bilateral pleural effusions and bilateral pleural masses. Subsequent pleural studies showed exudative pattern but with pH of 7.89 and white blood cell count of only 389, it was determined that it was not a parapneumonic effusion. Pleural triglyceride level was only 29, thus ruling out chylothorax. Pleural culture and cytology were negative. Patient underwent a video assisted thoracoscopic procedure with pleural biopsy. Surprisingly, pleural mass biopsy showed acute and chronic necrotizing pleuritis with mesothelial hyperplasia, but no malignancy. Considering that biopsies were negative and pleural studies were negative for a parapneumonic pattern, it was thought that the pleural effusion could be attributed to everolimus.

This case demonstrates that everolimus induced pleural effusion should be in the differential when a cancer patient presents with an exudative pleural effusion. Though malignancy and pneumonia are most common etiology for exudative pleural effusion, clinicians should maintain a high index of suspicion when patient reports prior usage of everolimus since stopping the medication may lead to resolution of the effusion.
HIV Induced Cerebral Angiitis; A Rare Presenting Feature of HIV

Human immunodeficiency virus infection (HIV) causes multiple morbidities despite the introduction of highly active retroviral therapy (HAART) which significantly changed the outcome for HIV patients. Among these diseases, cerebral vascular accidents (CVA) are one of the significant complications and found to be higher in HIV-infected patients compared to the general population. Studies have shown that patients with HIV are at higher risk for CVA due to various mechanisms including coagulopathy, vasculitis, result of HAART regimen, and opportunistic infection. Stroke incidence has shown a 1.4-to-1.5-fold increase of stroke in HIV-infected adults when compared to non-HIV patient population (1). Here, we present the case of a 48-year-old man who presented with stroke as the first manifestation of HIV infection.

We present a 48-year-old male with a past medical history of type2 diabetes mellitus, previous tobacco use, and recent ischemic stroke involving the left cerebellar vermis two weeks ago, who presented to our hospital with dysarthria. On arrival, the patient was hemodynamically stable, and neurological exam revealed severe dysarthria and apraxia more on the left. Labs were unremarkable. CT head without contrast and CT angiography of the head and neck were negative for any acute process. Brain MRI showed acute infarcts in the left frontal lobe, right thalamus, and right cerebral peduncle. Standard stroke, immunological and hematological workups were all negative. Two days later the patient was complaining of numbness in his right leg, repeated MRI showed a new acute infarct within the left striatal capsular region. The patient then had a cerebral angiogram, which showed evidence of cerebral vasculitis at the terminal arterial of MCA/PCA/ACA, finding consistent with cerebral angiitis. Further workup including infectious etiology was positive for HIV-1, CD4 count 712. His symptoms improved and the patient was discharged home to follow up with infectious disease to start HAART.

HIV causes many complications and affects various organ systems. CVA is one of these morbidities. Our case emphasizes the importance of considering vasculitis as a cause of recurrent cryptogenic strokes especially HIV in the young population.
Pasteurella Multicoda Bacteremia Without Skin Puncture from Carrier Species

Immunocompromised individuals are at greater risk for infection with Pasteurella multicoda. We report a case of bacteremia in a patient with chronic obstructive pulmonary disease (COPD) and exposure only to the bodily fluids of a carrier species to expand the clinical presentation of this condition. A 70-year-old woman with a past medical history of COPD presented to the hospital with altered mental status and shortness of breath. A recent history of increased frequency of cough was described by a family member. The patient did not respond to conservative home treatment using inhalers. Vitals were significant for tachycardia and fever. On physical exam, the patient was unable to respond to questions due to confusion and respiratory distress. Mild expiratory wheeze could be heard bilaterally, with worsened aeration over the right lung compared to the left. Steroid and bronchodilator therapies were given to treat suspected acute exacerbation of COPD. Cultures were drawn due to the presentation fulfilling sepsis criteria. Laboratory studies demonstrated mild hyponatremia, normal white cell count, and acute kidney injury. Procalcitonin was noted to be elevated. Chest x-ray showed right lower lobe opacity consistent with lobar pneumonia. Empirical treatment of community acquired pneumonia was initiated using doxycycline and ampicillin-sulbactam. Follow up blood cultures returned positive for P. multicoda. Additional history obtained from the patient revealed ownership of a pet dog. The dog had recently become very ill with bladder incontinence and frequently shared a bed with the patient. After culture susceptibility returned, antibiotics were de-escalated to ampicillin-sulbactam only. The patient’s condition improved significantly, and switch to oral amoxicillin-clavulanic acid was made by hospital day five. The patient was discharged in stable condition with instructions to complete the remainder of antibiotic therapy. This case is an example of the increased risk patients with severe pulmonary disease may develop P. multicoda bacteremia via pneumonia. Although transmission is more commonly through skin puncture, a history of significant exposure to bodily fluids of carrier species should raise clinical suspicion. Identification of this risk factor in susceptible patients should help to expediate management and improve outcomes in the future.
Pulmonary-renal syndrome (PRS) is characterized by the combination of diffuse alveolar hemorrhage (DAH) and glomerulonephritis [1]. ANCA-associated vasculitis is a common cause. By presenting this challenging case we aim to enlighten the clinicians about atypical presentations this disease which can lead to catastrophic outcomes.

CASE PRESENTATION
A 73-year-old male with a past medical history of COPD, presented to the hospital with a few days’ history of progressive dyspnea, cough, and hemoptysis without a fever. On presentation, he was hypoxic, tachycardic with bilateral wheezes and rales, along with erythematous skin rash and edema in lower extremities. His workup demonstrated significant leukocytosis, anemia, and thrombocytopenia and acute kidney injury (AKI) with microscopic hematuria. The Respiratory Pathogen Panel was negative. Chest radiographs showed emphysematous and fibrotic changes of the lungs, in addition to patchy ground glass opacities. Blood cultures were sent, and broad-spectrum antibiotics were started. He was being treated for COPD exacerbation, his respiratory failure got worse requiring intubation and ventilatory support. Bronchoscopy showed diffuse alveolar hemorrhage. Further Infectious work up including tuberculosis and aspergillus testing in addition to blood cultures resulted negative, antibiotics were discontinued. He continued to worsen with increasing oral bloody secretions, developed macroscopic hematuria, and eventually required hemodialysis. Also, multiple platelets and blood transfusions were given, and hematology was consulted. He had a negative Anti-GBM, Myeloperoxidase, and proteinase-3-antibodies. ANA was positive, cardiolipin antibodies were slightly elevated. DsDNA antibodies, Anti-smith and Lupus Anticoagulant antibodies were negative as well. Intravenous pulse steroids were started with some clinical improvement. The plan was to follow steroids with Rituximab. But, upon relative hemodynamic stabilization, the family requested to transfer the patient to a higher-level tertiary care hospital. He passed away soon after transfer.

DISCUSSION
Nearly 60% of PRS cases caused by ANCA-associated vasculitides, while Goodpasture's Syndrome accounts for approximately 20% of the cases. Renal biopsy remains the gold standard for diagnosing glomerulonephritis; it wasn’t conclusive in our case, delaying the initiation of the appropriate treatment. The patient’s picture of AKI and pulmonary hemorrhage is highly suggestive of Granulomatosis with polyangiitis which would have benefited from the induction therapy with Rituximab as it is the standard of care [2].
HIV in a COVID-19 Patient with Neurosyphilis and HSV Encephalitis

Syphilis is a sexually transmitted infection (STI) with increasing incidence, particularly among individuals with human immunodeficiency virus and acquired immunodeficiency syndrome (HIV/AIDS). We present a case of neurosyphilis and herpes simplex virus (HSV) encephalitis with COVID-19 infection in a young male with history of HIV, Syphilis and non-compliance.

A 24 year old male with AIDS (CD4 of 10cell/ul and %CD4 <1), presented to the emergency department with altered mental status. On presentation, he was cachectic, unresponsive, afebrile, hypertensive, tachycardic, and saturating well on room air. Examination revealed perianal lesions. Work up revealed acute kidney injury (AKI), metabolic acidosis, positive syphilis antibodies and rapid plasma reagin (RPR) with a titer of 1:32. SARS-CoV-2 nasopharyngeal polymerase chain reaction (PCR) testing was positive. Additional work up including testing for: Cryptococcus, EBV, CMV and Toxoplasmosis were negative. Chest X-ray was concerning for right lung pneumonia. Magnetic resonance imaging of the head showed nonspecific findings suggesting encephalitis or other inflammatory processes. Lumbar puncture (LP) demonstrated protein of 392mg/dl, positive HSV-2 PCR and venereal disease research laboratory (VDRL) tests with VDRL titer 1:4, confirming HSV-2 and neurosyphilis. Patient was started on 14 days of IV penicillin for neurosyphilis and 21 days of acyclovir for HSV encephalitis/ perianal lesions.

Admission was complicated by 4 days of mechanical ventilation for difficulty protecting the airway, pancytopenia, and nephritic/nephrotic syndrome. Patient improved and was discharged on tenofovir alafenamide, emtricitabine, darunavir, and ritonavir together with dapsone for prophylaxis against Pneumocystis carinii pneumonia after testing was negative for G6PD deficiency and HLA-B*5701. Following discharge the patient was seen in the clinic. Although mentation was improving, perianal HSV had recurred. Antiretroviral was consolidated to symtuza and acyclovir was restarted. LP and RPR were planned to be repeated in 6 months.

This case demonstrates searching for diagnosis beyond COVID-19 in patients with atypical presentations, and the diagnosis and management of HSV encephalitis and neurosyphilis. It also illustrates the importance of maintaining a broad differential for encephalitis, particularly among those with HIV/AIDS, in whom STIs may be more common during this COVID-19 era.
A Rare Case of Livedoid Vasculopathy with Peripheral Neuropathy- Challenges in Pain Management

Introduction:
Livedoid vasculopathy (LV) is an extremely rare disease with an estimated prevalence of 1 in 100,000 cases in North America, predominantly in females. They present initially as painful, erythematous or purpuric plaques which may ulcerate eventually. Till date, there are no definitive treatment guidelines available. We present a case of Livedoid vasculopathy in a young male misdiagnosed and treated for cellulitis initially.

Case Presentation:
A 22-year-old male with history significant for uncontrolled type 1 diabetes mellitus (HbA1C 13.2%), and spontaneous pneumothorax presented to the emergency department with extremely painful bilateral lower extremity swelling of 2 weeks duration. On presentation, he was hemodynamically stable and afebrile, but was in severe distress secondary to pain. Examination revealed a tall young male with marfanoid features and erythematous bilateral lower extremity swelling extending up to knees, with blisters and violaceous lesions in the lateral aspect of the feet. Hyperesthesia was also noted. Initial work up revealed elevated ESR 36 mm/hr and CRP 53.9 mg/l without leukocytosis. Arterial and venous dopplers were unremarkable. He was empirically treated with multiple antibiotics for cellulitis without any clinical improvement. Further, a differential of vasculitis versus connective tissue disorder was considered, but serological work-up was negative. Hence, biopsy along with direct immunofluorescence was performed which revealed fibrin formation in small vessels positive for IgM and C3. Extensive workup for hypercoagulable states and autoimmune diseases remained negative. He was treated with aspirin, Pentoxifylline, Apixaban and Pregabalin along with Oxycontin and Oxycodone for pain management.
Patient was readmitted twice for pain crises which was treated with epoprostenol, amlodipine along with steroids to reduce inflammation initially and finally ketamine infusion, with transcutaneous electrical nerve stimulation with adequate pain control achieved

Discussion:
LV is dysfunction of coagulation with micro-thrombosis of the dermal capillaries. LV can be classified as primary (idiopathic 20%) and secondary, which is associated with hypercoagulable states, thrombophilia, autoimmune disorders or malignancies. Treatment with anticoagulants, antiplatelets, anabolic steroids, hyperbaric oxygen, intravenous immunoglobulin, thrombolytics, multivitamins, UV light have been tried with favorable outcomes. Acute management of peripheral neuropathy presenting as a pain crisis continues to be a clinical challenge and prompts for further studies.
Thrombotic thrombocytopenic purpura (TTP) is a serious life threatening condition that requires prompt diagnosis and therapeutic plasma exchange (TPE). So far in literature, cocaine has been described as a cause of Thrombotic Microangiopathy (TMA) in 6 cases. We present a case of recurrent TTP associated with cocaine abuse.

A 50-year-old male with a history of transient ischemic attack and recent cocaine abuse presented to the ED with left arm tingling and numbness associated with facial paresthesia. On assessment, his symptoms had resolved and examination was unremarkable. Workup revealed anemia, thrombocytopenia and acute kidney injury. Patient later developed fever and altered mental status, requiring mechanical ventilation for airway protection and ICU admission. CT head showed no acute intracranial pathologies. Further workup revealed intravascular hemolysis, and schistocytes on peripheral blood smear. With a PLASMIC score of 5, he was started on daily TPE and steroids for presumptive diagnosis of TTP. Pre-treatment ADAMTS13 activity was reported as low (<3%) with presence of an inhibitor. Patient also required dialysis for acute renal failure. Subsequently, he improved, was weaned off mechanical ventilation, and was discharged with a plan for outpatient TPE to minimize the chances of relapse. Patient never followed up and presented 3 years later with abdominal pain and nausea. Urine drug screen was positive for cocaine, and workup was consistent with TTP. Patient was started on TPE and steroids, and was discharged after a week once the platelet count improved. Two weeks later, workup at the clinic showed significant thrombocytopenia and he was hospitalized for a 3rd episode of TTP. Patient endorsed fatigue and recent cocaine use during the 3rd episode. Patient subsequently improved with TPE and steroids, and was discharged with a plan for outpatient TPE. In both recent admissions, ADAMTS13 activity was reported as low (<0.03%) with presence of an inhibitor.

To summarize, all 3 hospitalizations consistently demonstrated low ADAMTS13 activity and presence of an inhibitor. It illustrates that use of cocaine can cause recurrent TTP, hence further drug exposure should be avoided. The possible mechanisms of cocaine-induced TMA include vasoconstriction, endothelial damage, procoagulant activity and increased platelet activity.
Extraintestinal Clostridium difficile Infection- A Battle with Resilient Abdominal Wall Abscess

Introduction
Clostridium difficile infection (CDI) is the most common cause of healthcare associated infection with an estimated national incidence of healthcare associated CDI being 235,700 cases in 2017(1). Extraintestinal CDI is more rare; up to 0.61% of all CDIs, with most common infection in the abdominopelvic region(2). Here we present a case of abdominal wall abscess with monomicrobial C. difficile infection initially.

Case Presentation
A 61-year-old long-term nursing home resident with multiple comorbidities presented in shock and metabolic encephalopathy. Pertinent surgical history includes perforated diverticulitis 20 years ago status post colectomy with long term colostomy; history of three ventral hernias and strangulated parastomal hernia with small bowel perforation 6 months ago requiring partial ileal resection, and open abdomen with AbThera vac for 7 days before complete closure of abdomen and ostomy with mesh. On presentation, she had 7.9x5.2x12.0cm abdominal wall abscess superficial to the mesh, growing solely C.difficile. She was initially treated with catheter drainage and broad-spectrum antibiotics. She had multiple follow-up hospital admissions and a comprehensive summary of her physical examination, culture findings and antibiotic regimen are compiled in Table 1.

Discussion
The burden of extraintestinal CDI is likely underestimated owing to the fact that the organism is a fastidious anaerobe, and often presents with polymicrobial infection, drawing all attention to other notorious co-existing organisms(2,4,5). Our patient had multiple risk factors including bowel surgery following bowel perforation, long-term nursing home resident, and long-term proton pump inhibitor use. Our patient is unique in that she presented with monomicrobial infection 6 months following the inciting event, while most patients bear polymicrobial infections within 4 weeks. She was treated with nearly 17 weeks of broad-spectrum antibiotics despite being monomicrobial initially(2,5,6). Follow-up CT imaging and multiple hospital admissions not only consumed a vast amount of health care resources but resulted in decline of her quality of life.

Learning Points
-Although extraintestinal CDI are rare, they can cause serious morbidity.
-We recommend a prolonged duration of broad-spectrum antibiotics for monomicrobial CDI
-Healthcare workers should maintain hygienic protocols to prevent spread of infection; contact precautions and hand washing with soap and water
Unusual Case of Necrotizing Fasciitis Due to Urine Therapy!

Urine therapy is an old belief that dates back to the ancient times of Egypt, Rome, and Greece, where it was practiced for general well-being. As urine stays within its natural anatomical pass it is sterile and doesn't harm the body, but once it is excreted it is polluted with bacteria. Thus, ingestion or injection can lead to bad outcomes, like severe infections. By presenting this case we are trying to increase clinician awareness about this modality of alternative medicine.

CASE PRESENTATION
A 49-year-old female with no medical history presented with fever, swelling, and redness of the right thigh, of three days duration. She endorsed ingesting and self-injecting her stored urine onto different areas of the body, for the past month, upon reading about Urine therapy. Reported no recent travel, drug use, or contact with animals. Vitals signs were unremarkable except for tachycardia. Physical exam was significant for swollen, tender right anterolateral mid-thigh associated with rapidly expanding erythema, concerning cellulitis and abscess. Workup showed leukocytosis and elevated inflammatory markers. Computer Tomography revealed a large abscess in the anterolateral mid-thigh, measuring 6.8x3.5x7.5 cm with early signs of focal necrotizing fasciitis. Broad-spectrum antibiotics were started followed by Incision and Drainage with purulent output and a wound vac was left in place. Her clinical course improved, antibiotics were deescalated to Augmentin when cultures grew polymicrobial organisms including streptococcus anginosus, beta-hemolytic streptococcus, fusobacterium necrophorum and parvimonas micra.

DISCUSSION:
Urine therapy is used by many people to treat a wide array of medical conditions ranging from cancer to COVID-19 infection. It is practiced in multiple forms like drinking, applying to the hair or the skin, and injecting to the different parts of the body. Urine contains multiple toxins and microorganisms that can be highly resistant to antibiotics. Our patient was reluctant to seek medical attention immediately due to fear of embarrassment, however, she presented early in the course of her illness which lead to surgical intervention and prevented adverse outcomes. Our case recognizes the effect of social media in promoting alternative medicine which is largely unregulated from a medical perspective and helps promote awareness, among medical professionals, of such therapies.
Pancreaticopleural Fistula Presenting as Bilateral Pleural Effusions in the Setting of Acute Pancreatitis with Pseudocyst

Pancreaticopleural fistulas are a rare complication of pancreatitis, occurring in less than 10% of patients with pseudocysts. Persistent leakage of pancreatic fluid may cause malnutrition and infection, ultimately causing significant morbidity and mortality.

We present a 43 year-old male with a history of diabetes mellitus, alcohol use, and recent pancreatitis, who presented with epigastric pain radiating to the back with difficulty in breathing for several weeks. Lipase was elevated and computed tomography was consistent with acute pancreatitis with pancreatic pseudocyst and large bilateral pleural effusions. Thoracentesis with pleural fluid studies revealed an exudative process with amylase 3,900 IU/L. Concerned for pancreaticopleural fistula, patient was started on octreotide therapy and pancreatic duct was stented despite pancreatogram not showing fistula during ERCP.

Hospital course was complicated by post-ERCP pancreatitis, as well as several subsequent thoracentesis for continued fluid accumulation. A pleural drain was eventually placed and grew strep mitis, treated with intravenous ampicillin-sulbactam for empyema. Upon resolution of effusion and removal of pleural drain, repeat computed tomography confirmed communication between the peripancreatic fluid and pleural space via paraesophageal tract. Post-pyloric feedings were given with eventual improvement of abdominal pain prior to discharge.

Pancreaticopleural fistulas form due to disruption of the pancreatic duct or pseudocyst with tracking of pancreatic fluid into the pleural space, presenting as shortness of breath, cough, or chest pain. Diagnosis consists of pancreatic duct disruption via secretin-enhanced MRCP, or pancreatogram during ERCP showing extravasation of contrast or direct communication. Pleural effusions due to pancreaticopleural fistulas are distinguished from sympathetic pleural effusions in acute pancreatitis by being an exudative process with amylase levels >100,000 IU/L, and reaccumulating after therapeutic thoracentesis. Although the pleural effusion is almost always left sided, this patient showed large bilateral pleural effusions.

While initial management consists of reducing pancreatic output with octreotide and enteral nutrition with nasojejunal feeding, endoscopic transpapillary stenting with biliary sphincterotomy is often needed to promote internal drainage and subsequent reduction of flow through the fistula. If endoscopic therapy is not feasible, then surgical intervention or even percutaneous drainage may be considered.
Drug-Induced Liver Injury Secondary to Fluoxetine Use

Fluoxetine is a commonly prescribed antidepressant with generalized gastrointestinal side-effects that include nausea and diarrhea. While fluoxetine is metabolized in the liver, fewer than 1% of patients develop mild and self-limited transaminitis. Here, we present a unique case of clinically apparent drug-induced liver injury (DILI) soon after fluoxetine initiation.

A 28-year-old male with schizophrenia and bipolar disorder was admitted to the hospital due to a 3-day history of worsening right upper quadrant (RUQ) abdominal pain with a 2-day history of yellowing of the skin and eyes. A thorough history revealed that he was on chronic citalopram and olanzapine therapy but was started fluoxetine (20 mg) 5-days prior. Additionally, he denied alcohol, recreational drugs, herbal supplements, and prior liver disease. On admission, the patient was alert and oriented x4 with jaundice, scleral icterus, and asterixis. Laboratory work demonstrated WBC 15,000, platelets 235,000, ammonia 131, AST 3219, ALT 6574, ALP 181, total bilirubin 22.9, and INR 1.97. Acetaminophen and salicylate levels were unremarkable. Fluoxetine was held immediately, and the patient was given N-acetylcysteine. RUQ ultrasound was unremarkable. MRCP demonstrated fatty infiltration of the liver without nodularity or biliary dilation. A thorough workup including viral hepatitis (A, B, C, E), EBV, CMV, HIV, hemochromatosis, Wilson’s disease, and autoimmune hepatitis was unremarkable. A liver biopsy was eventually performed, demonstrating mixed-pattern DILI. With removal of fluoxetine, the patient’s liver function tests and clinical symptoms continued to improve, and on hospital day 10 he was discharged in stable condition.

Transaminitis from fluoxetine is typically mild, asymptomatic, and self-limited. Our case highlights a rare instance where fluoxetine precipitated DILI. It is unclear whether polypharmacy with his other psychiatric medications, or underlying hepatic steatosis, were predisposing factors to developing fluoxetine-associated DILI. Higher awareness for fluoxetine-associated hepatotoxicity is needed and caution should be used to consider current medications and underlying liver pathology when starting fluoxetine.
Chasing a Sweet Demon: An Unfortunate Case of Persistent Hypoglycemia and Metastatic Cancer

Introduction: Persistent hypoglycemia is a rare syndrome of repeated low blood glucose that requires prompt evaluation due to risk of seizures, confusion, and coma. The mechanism of low blood glucose is mediated by the pancreatic hormone insulin and can be affected by multiple factors including rare insulin-secreting tumors within the pancreas. In patients with intact insulin function and multiple episodes of hypoglycemia, these rare tumors must be ruled out as an etiology to prevent further debilitating symptoms. We present an unfortunate case of a young female with recently diagnosed metastatic cancer of unknown primary source who was persistently hypoglycemic.

Case: A 24-year-old African American female with no significant medical history presented to the emergency room for right upper quadrant abdominal pain and unintentional weight loss of six pounds in one week. Initial abdominal imaging showed a pancreatic mass and numerous lesions concerning for metastatic disease. She subsequently had a liver biopsy confirming adenocarcinoma, but the origin of malignancy was not identified. One week into her hospitalization, her blood sugar started to decrease as low as 30mg/dL. She was requiring frequent interventions of glucose administration. She was eventually started on a continuous glucose infusion at which point serum biomarkers for an insulin-secreting tumor were ordered per Endocrinology recommendations. Due to logistical problems with proper acquisition of these markers and laboratory delays, the results were deemed inaccurate. The labs were repeated and in the interim, she was started on intravenous octreotide and stress-dose steroids to maintain normoglycemia. The patient has received chemotherapy infusions but her primary source of cancer as well as the cause of her persistent hypoglycemia remains unclear. Due to her debilitating symptoms and lack of appetite, she has also been receiving total parenteral nutrition which has helped maintain normoglycemia.

Conclusion: This case highlights the rare occurrence of persistent hypoglycemia. The etiology of persistent hypoglycemia is difficult to confirm because the workup requires blood samples to be drawn at specific times of the day and while the patient is fasting. We emphasize that prompt evaluation of persistent hypoglycemia as well as immediate glycemic correction are necessary to prevent adverse outcomes.
A 55-year-old male presented to our outpatient clinic with acute onset sharp and burning pain in both hands and feet for a few weeks. The severity of his pain was limiting his ability to perform the essential duties of his job and effecting his sleep. He was recently diagnosed with diabetes mellitus type 2 with a HbA1c of 16.5. Medication included long-acting insulin, GLP-1 agonist, SGLT-2 inhibitor alongside ACEi and a statin for elevated ASCVD risk. His sugars on average dropped from 300s to 100s in a matter of days to weeks. He had been exercising regularly, losing weight, eating a diabetic diet. On exam, he had no focal deficits, no cranial nerve pathology, negative Tinnel and Phalen tests and no history of neck injury. His A1c on this visit was 7.7. He was started on gabapentin for symptomatic relief and an EMG/NCS was ordered to confirm the diagnosis. His diabetic regimen was continued and in the subsequent weeks he noticed significant functional relief of his symptoms.

First described in 1933 by Caravati, Treatment Induced Diabetic Neuropathy (TIDN), in a patient who developed symptoms of neuritis following rapid glycemic control with insulin; symptoms resolved with insulin withdrawal. Reported in both type 1 and type 2 diabetics treated with insulin or oral hypoglycemic agents with a history of poor glycemic control, pathogenesis of the condition and its associated pain is poorly understood. Proposed mechanisms including endoneurial ischemia, hypoglycemic microvascular neuronal damage, and regenerating nerve firing.

Gibbons and Freeman studied a cohort of 16 patients with TIDN, with a detailed description of autonomic symptoms, intra-epidermal biopsy results, and long-term follow-up. One proposed set of diagnostic criteria for TIDN, include: (1) acute onset of neuropathic pain or autonomic symptoms, (2) a decrease in HbA1c level of over 2% in 3 months, and (3) onset of neuropathic pain and/or autonomic symptoms within 8 weeks of the decrease in HbA1c level.

When suspected, TIDN should be confirmed with an EMG/NCS and treated like neuropathic pain. Occurrence of symptoms do not preclude treatment of diabetes mellitus. Timely recognition and treatment provide dramatic relief of symptoms.
Atypical Presentation of Myasthenia Gravis

Introduction:
Myasthenia gravis (MG) is one of the common neuromuscular junction conditions, characterized by progressive muscle fatigability, predominantly affecting the ocular and bulbar muscle groups, with relatively preserved sensory modalities. We present a case highlighting the non-classical presentation of myasthenia gravis with unique unexplained sensory deficits in addition to the classical motor deficits.

Case Presentation:
A 66-year-old with a past medical history of colorectal cancer presented with numbness and weakness in the jaw and tongue. He also reported that his symptoms had started 6 months ago with pins and needle-like sensations in the left thumb, followed by fatigue and trouble chewing. He also started to experience progressive numbness in his lower jaw, lips, chin, and tongue. He developed vertical diplopia 2 weeks later, followed by drooping of the left eyelid, first noticed by his wife. His symptoms worsened as the day progressed. Physical exam revealed partial ptosis on the left side, with no sensory or motor deficits. Studies included CBC, CMP, ESR, MRI brain w/wo contrast, CT head, and neck, which were negative for any acute process. Myasthenia panel revealed acetylcholine receptor blocking antibody of 30% and modulating antibody of 96%, the binding antibody was 19.60 nmol, consistent with the diagnosis of Myasthenia Gravis. EMG findings further confirmed the diagnosis. The patient was eventually started on pyridostigmine and prednisone, following which he had marked improvement in his symptoms.

Conclusion:
Myasthenia gravis is an autoimmune disease with a wide spectrum of clinical manifestations, with ocular and bulbar muscle weakness being the most common presentation. Sensory deficits can occur in up to 10% of patients and can be attributed to loss of myelinated and unmyelinated fibers. Sensory manifestations of myasthenia gravis improve along with motor manifestations when patients are started on acetylcholinesterase inhibitors and immunosuppressants, indicating that the abnormalities are part of the myasthenia gravis syndrome rather than being a coincidental factor. Therefore, to avoid investigative expenses and morbidity, clinicians should have a high index of suspicion for myasthenia gravis in patients who present with sensory-motor complaints.
May-Thurner Syndrome with Recurrent DVT, and Bilateral Pulmonary Embolism: A Case Report

May-Thurner syndrome (MTS), also known as iliac vein compression syndrome, Cockett syndrome, or iliocaval compression syndrome is caused by the compression of the left iliac vein by the right iliac artery, which increases the risk of deep vein thrombosis (DVT) in the left leg. We report a case of a 34-year-old man who presented with unprovoked recurrent deep vein thrombosis (DVT) complicated by bilateral pulmonary embolism (PE).

A 34-year-old Caucasian male with a past medical history of questionably provoked DVT 6 months ago, asthma, morbid obesity, PTSD, generalized anxiety, and panic disorder presented with breathing difficulty and chest pain. The Computed Tomography (CT) scan of the chest revealed bilateral pulmonary embolism and Doppler of lower extremities revealed acute DVT in the left leg. The Vascular, Haematology, and Pulmonology teams were consulted and the patient was started on IV Heparin. As per history, the patient had provoked left leg DVT 6 months ago after stamping on a metal rod followed by a long-distance drive. He was treated at Veteran’s hospital for DVT and his hypercoagulability workup was negative. Since it was a provoked DVT, the patient was advised to discontinue anticoagulation after a few months. A CT of the abdomen and pelvis during current hospitalization revealed compression of the left common iliac vein by the right common iliac artery worrisome for May-Thurner Syndrome. After 5 days of IV Heparin, anticoagulation was transitioned to oral Eliquis. Echocardiography revealed EF-65%, right ventricle borderline dilated with normal function. The patient was later discharged in a hemodynamically stable state on indefinite anticoagulation as recommended by vascular and hematology teams. He had an outpatient CT venography which revealed significant occlusion of the left iliac vein for which he underwent an angioplasty by the Vascular team. He follows outpatient with the vascular and hematology teams with serial Doppler ultrasound for monitoring.

Untreated MTS carries a risk of life-threatening complications such as PE, iliac vein rupture, retroperitoneal hematoma, or recurrent DVT. Proper identification of MTS carries a positive outcome in treating DVT and PE secondary to MTS. The clinical suspicion can be confirmed with CT and iliac venography.
Progressive Multifocal Leukoencephalopathy as a Manifestation of an Underlying B-cell Disorder, a Rare Presentation

Progressive Multifocal Leukoencephalopathy (PML) is a demyelinating disease of the central nervous system caused by the reactivation of the John Cunningham (JC) virus. It commonly occurs in patients with human immunodeficiency virus (HIV)/acquired immunodeficiency syndrome (AIDS) or those receiving immunosuppressive therapies. Rarely underlying primary immunodeficiency has been reported as the cause of reactivation, however, these patients have a prior history of symptomatic immune deficiency. Here we report a rare case of PML due to an underlying previously asymptomatic B-Cell disorder.

A 66-year-old female with a medical history of obesity, essential hypertension, and hyperlipidemia presented with a generalized tonic-clonic seizure. A progressive decline in memory, slow speech, and worsening mentation were reported for the past 2 months. Workup revealed T-2 Hyper intensity involving white matter in frontal and temporal lobes. Cerebrospinal fluid analysis was unremarkable, however, Cerebrospinal fluid JC virus titer was elevated (128 copies /ml). Diagnosis of PML was made. The patient tested negative for HIV. She had not received immunosuppressive therapy, recently or in the remote past. Extensive workup did not reveal evidence of hematological or solid organ malignancy. Extensive immunological workup revealed low levels of CD19 and CD20 cells and low IgG levels and suboptimal titers to recent vaccination; however, remote vaccination was preserved. She was diagnosed with B-Cell production and maturation disorder.

Around 80% of the US adult population is infected with the JC virus. Immunodeficiency causes PML through reactivation of the JC virus. Around 80% of PML cases are due to HIV/AIDS. Immunosuppressive therapies represent the second common cause of PML. Primary Immunodeficiency disorders are a rare cause of PML, and these patients usually have a prior history of recurrent infections. Our patient had a relatively unremarkable history, and PML was the first manifestation that led to the diagnosis of B-Cell disorder. To the best of our knowledge, this is the first case of this nature.
The Goat Cheese That Took Her Breath Away

Cow's milk allergy is one of the most common food allergies, and the second most common trigger for eosinophilic esophagitis (EoE). Cow's milk allergies are sensitized to either or both whey proteins and the casein fraction and many cannot tolerate goat's milk due to cross reactivity. It is very rare to be allergic to goat's milk without having an allergy to cow's milk as they have highly homologous proteins.

A 16 year old female with a past medical history of atopic dermatitis, allergic rhinitis, (EoE), intermittent mild asthma, food allergies (egg, peanut, tree nut, sesame, shellfish, coconut), presented with a reaction to goat milk cheese via specialty pizza. Within 5 minutes of consuming the pizza the patient started experiencing chest and back pain and shortness of breath. An epi-pen provided sudden relief and she went to the emergency department for further management. Follow up with her allergist consisted of testing the pizza ingredients and goat's milk cheese. A positive percutaneous confirmed an allergy to goat's milk, correlating with her initial exposure causing anaphylaxis. Interestingly, she has no allergy to cow’s milk and has always tolerated it and enjoyed it, despite a EoE and various other allergies in her brother, including cow’s milk. No further testing was done due to patient desiring diet change.

In an individual with various allergies including egg and EoE that often include cow’s milk allergy, it is particularly unique that there is no cow’s milk allergy or EoE trigger. It is suspected that there is a unique protein in the goat's milk that this patient is sensitized to besides the typical casein or whey. As scientist trained physicians, we often want more tests and work up to find the answers, however, we must weigh the time and suffering of the patient and costs of care in the shared decision making process. Further, when it comes to food allergies, physicians must evaluate the allergy diet change dilemma of balancing nutrition challenges while avoiding possible anaphylactic reactions.
Not Every Rhinorrhea is a Cold!

Persistent cerebrospinal fluid rhinorrhea can predispose to life-threatening meningitis by the oral flora. Although a history of head trauma is present in most such cases, CSF rhinorrhea rarely can occur in the absence of trauma, masquerading as an allergic symptom or sinusitis.

A 47 year old female noted significant, persistent rhinorrhea that became worse on leaning forward. Patient presumed it to be a cold and did not pursue evaluation. There was no reported head trauma, dental or spinal manipulation. Six weeks after the onset of nasal drainage, the patient presented to the ED with headaches, fever, and altered mental status. Physical examination showed signs of meningeal irritation. CSF analysis was typical for bacterial meningitis, and CSF cultures grew S.Oralis, a part of the normal oral flora. Beta-transferrin level of the nasal drainage was performed, which turned out to be positive. Empiric antibiotic regimen for meningitis was immediately initiated, including Ceftriaxone and Vancomycin. Overnight, the patient’s meningeal symptoms resolved, since S.Oralis was sensitive to Ceftriaxone.

CSF rhinorrhea with no history of recent head trauma can be easily mistaken for benign conditions such as sinusitis or allergies. Even in the absence of risk factors, persistent rhinorrhea that becomes worse on leaning forward should raise alarm for CSF leakage, as this predisposes the patient to life threatening bacterial meningitis. B-2 transferrin level of nasal drainage is a highly sensitive and specific test for CSF rhinorrhea, and it should be performed to rule out CSF leakage. Antibiotics should be started as early as possible to improve the outcomes in such cases.
Introduction: Toxic Epidermal Necrolysis Syndrome (TENS) and Steven Johnson Syndrome (SJS) are skin reactions characterized by blistering and skin sloughing at the level of the epidermis. TENS can be caused by underlying infection or the use of antibiotics, NSAIDs, and anticonvulsants. TENS and SJS both have high mortality rates; especially as cutaneous involvement increases.

Case Description: 75-year-old male presented with a diffuse, erythematous, blanching but not tender rash with bullae and swelling involving over 70% of his body. Patient had 9-11% epidermal detachment with taught, softball-sized bullae over lower extremities. Left hand was severely swollen with clear drainage from desquamated skin. Patient had taken his wife’s Keflex to treat what they believed to be cellulitis on his left hand after recovering from COVID-19 Pneumonia in subacute rehab. Patient has chronic diastolic heart failure and was still on 2 L of oxygen at baseline.

Blood cultures, MRSA screen, and biopsies of the rash were obtained; cultures were negative, and the biopsies were significant for actinically damaged skin. Patient was started on vancomycin and a high-dose prednisone taper. The rash improved daily, and wound care was heavily involved to manage the continued sloughing.

Patient was discharged when the extensive rash was vastly improved.

Discussion: With presumptive SJS or TENS it is vital to address the presumptive causal agent, rule out infection, and involve wound care early. It is also important to be mindful of ocular care, to maintain electrolyte balance, and manage the patient’s pain. This case was challenging given the patient’s chronic heart failure, as that complicated the fluid resuscitation. There are not currently set guidelines for use of corticosteroids in treatment; other treatment options for severe cases include IV-IG, cyclosporine use, or plasmapheresis. This patient’s hospital course was lengthy and complex; the patient experienced a type-1 myocardial infarct while admitted but refused catheterization. Unfortunately, the patient passed away within months of discharge due to severe multi-vessel disease and progressive kidney failure.
Pneumocystis Pneumonia in Cirrhosis: A Case Report and Literature Review

Introduction:
Pneumocystis jiroveci is a fungus that causes serious pneumonia in immunocompromised individuals most classically associated with AIDS or prolonged steroid use. Pneumocystis pneumonia (PCP) is not typically associated with cirrhosis as a common risk factor for PCP. We present a case of Pneumocystis pneumonia in a patient with cirrhosis and describe the limited but emerging literature of similar cases.

Case report:
A 59-year-old male with past medical history of alcohol abuse, cirrhosis, and COPD presented to the emergency department after gradual increasing generalized edema over 2 months. He was found to have severe alcoholic hepatitis. He initially developed septic shock attributed to spontaneous bacterial peritonitis (SBP) but clinically improved on ceftriaxone alone. He tested negative for HIV and viral hepatitis. Three days later he developed dyspnea, hemoptysis, and hypoxia. CT-angiogram revealed extensive ground glass opacities. His respiratory condition deteriorated and required mechanical ventilation. Bronchoalveolar lavage confirmed Pneumocystis jiroveci by PCR. 1,3-β-D-Glucan assay was elevated at 373pg/mL. Despite being HIV negative his CD4 count was reduced at 292 cells/mm^3. Treatment with trimethoprim/sulfamethoxazole was contraindicated due to severe renal impairment. Therapy was changed to clindamycin and primaquine before his continued declined ended in a transition to comfort care and his ultimate passing.

Discussion:
Our case was unique in that cirrhosis with alcoholic hepatitis was the only identified risk factor for immunosuppression. A review of the literature for PCP in the setting of cirrhosis revealed 14 described cases. Twelve were in the setting of alcoholic hepatitis, and nine of these received treatment with steroids. Commonly seen comorbidities included prior mechanical ventilation, leukopenia, and acute blood loss. Co-infection with other respiratory pathogens was common. In 12 out of 14 patients, the infection was fatal suggesting a very poor prognosis. In conclusion, this case and review are important reminders of the severe immunocompromised state caused by cirrhosis. Opportunistic infections such as PCP should be considered.
Covid 19 Associated Biventricular Failure

Patients with COVID-19 typically present with fever, dyspnea, acute respiratory failure and can develop various complications. COVID-19 patients who developed cardiac complications including stress cardiomyopathy (Takotsubo) and Myocarditis have been reported. We present a case of COVID-19 infection in a patient who developed biventricular failure.

This is a 44-year-old-man with comorbid hypertension, hyperlipidemia, drug abuse by injecting testosterone, presented to the ED with complaints of fever, dyspnea, hemoptysis, nausea, abdominal pain, diarrhea, severe body ache and fatigue for 3 weeks. On examination, the patient had decreased breath sounds with lower extremity pitting edema. COVID PCR was positive. Laboratory findings were significant for increased creatinine and BUN, along with elevated ESR, CRP and CK. Cardiac evaluation showed tachycardia with left ventricular hypertrophy on ECG, normal BNP, mildly elevated Troponin and D-Dimer. Initial TTE showed concentric hypertrophy, grade 2 diastolic dysfunction with moderate-severe LV hypokinesis with 34% EF. The patient had worsening oxygen demands and was intubated. IL 6 was elevated. Patient was given tocilizumab. Repeat TTE showed further decreased EF of 20-25%, global hypokinesis of LV, and RV enlargement which were not seen in the initial ECHO. The patient was diagnosed with dilated cardiomyopathy with biventricular failure and placed on GDMT and inotropic support. A repeat ECHO was performed after 10 days of initial which showed improvement in systolic function with EF 40% and mild global LV hypokinesis. The patient had a prolonged hospital stay due to multiple adversities including renal failure and hypoxic brain injury but maintained stable cardiac function on medications.

Our patient was diagnosed as dilated cardiomyopathy with biventricular failure secondary to Covid induced cytokine storm. There are case reports and few articles that reports acute biventricular failure due to Covid 19 induced cytokine storm and a few case reports of patients who developed takotsubo cardiomyopathy in covid-19 infection have already been reported. We believe the correlation between cardiomyopathy and Covid needs further exploration and this case will contribute to the growing literature.
Colorectal Cancer Associated with Fusobacterium Necrophorum

Introduction:
Fusobacterium Necrophorum is an anaerobic, non-spore forming pleomorphic Gram-negative rod which is considered a commensal of the upper respiratory and gastrointestinal tract. Besides being classically known as the main cause of Lemierre's syndrome, association between colorectal cancer and Fusobacterium Necrophorum has been described in literature. We present a case of Fusobacterium Necrophorum bacteremia with underlying colorectal cancer.

Case Presentation:
A 85-year-old male with a past medical history of recently diagnosed colon cancer presented with generalized weakness, fever and chills for 2 days. He denies upper respiratory tract symptoms, shortness of breath, cough, sore throat, otalgia, neck pain, abdominal pain, diarrhea or sick contact. Of note, he was recently diagnosed with adenocarcinoma of rectosigmoid colon less than 2 weeks ago with colonoscopy demonstrating an ulcerated rectosigmoid mass partially obstructing the colon. Upon presentation, he was febrile but vital signs were stable. On physical examination, the patient was septic looking but otherwise no neck swelling or tenderness, no cervical lymphadenopathy, throat was normal and abdominal exam was unremarkable. Initial labs were remarkable for leukocytosis with neutrophilic predominance and elevated CRP. CT Abdomen/Pelvis showed thickening wall of the sigmoid colon likely due to incomplete distension. He was started on broad-spectrum antibiotics for unknown source of sepsis. Subsequently, blood culture revealed Fusobacterium Necrophorum. Antibiotic was de-escalated to Metronidazole which the patient was treated with for a total of 14 days.

Discussion:
There is growing evidence which described the bidirectional association between colorectal cancer and Fusobacterium Necrophorum. Our patient with concomitant Fusobacterium Necrophorum bacteremia and colon cancer raises the question about a possible mutual synergistic relationship between these 2 entities. Fusobacterium Necrophorum may play an active role in oncogenesis of colon cancer via several proposed mechanisms which include activation of Wnt/β-catenin oncogenic pathway, tumor-infiltrating myeloid cells recruitment and inhibition of T-cell proliferation. On the other hand, colorectal cancer has been hypothesized to predispose to systemic Fusobacterium infection via several postulated mechanisms which include immunosuppression as a result of the cancer and disruption of colonic mucosa provides a portal of entry for systemic infection.
Sub-Massive Pulmonary Embolism (PE) Management in Lung Cancer with Brain Metastasis

Introduction: Pulmonary embolism is a common occurrence in lung cancer, with incidence of approximately 4%. Lung cancer survival rates are decreased when patients present concomitantly with PE and out of the most common cancer sites where thromboembolic events were evident, the lung leads at 17%.

Case presentation: A 66-year-old male with past medical history of chronic tobacco use (100 pack-years) who presented to the hospital with chief complaints of generalized weakness and dyspnea. On presentation, he was tachycardic, tachypneic, and had a SpO2 of 88% on room air. Lab work-up was remarkable for elevated lactate/CRP, elevated BNP and leukocytosis. CT imaging showed a large mass-like area completely filling the lower lobe of the left lung (11.9x10.2 cm). Initial differential included necrotic mass or lung abscess/empyema. Further imaging revealed moderate pulmonary embolism on the right involving most segmental branches with right heart strain. Transthoracic echocardiogram revealed abnormal inter-ventricular septal motion, D shaped during systole along with right ventricular systolic pressure of 42.1 mmHg. However, thrombectomy and tPa were not options. He was treated with Heparin drip. Patient was eventually treated for CAP and discharged on apixaban.

He presented again to the ED per instruction of his oncologist for biopsy of his lung mass. Ultrasound lower extremity at this time revealed bilateral DVT. Heparin drip was started. Biopsy of his lung mass revealed small cell lung cancer. MRI was ordered for staging work up and showed a hemorrhagic enhancing mass in the right frontal lobe with associated midline shift. He was started on dexamethasone, levetiracetam, prophylactic enoxaparin and referred for radiation oncology. He was given 3 rounds of chemotherapy and discharged on prophylactic apixaban to help prevent spread of PE but also prevent worsening of his hemorrhagic metastasis.

Discussion: Meta-analysis and prospective cohort studies regarding incidence of intracranial hemorrhage and major bleeding show that low-molecular-weight-heparin is an acceptable treatment option for primary and metastatic brain tumors with VTE (except in select high risk cancers). General treatment duration is 6 months or less. IVC filter placement in a limited study showed minimal comparative benefit and poorer prognosis.
Gonococcal Osteomyelitis: Case Report

Although gonorrhea is a very common sexually transmitted disease, only 0.5-3% are complicated with disseminated gonococcal infection. Here, we report a case of gonococcal arthritis complicated with osteomyelitis.

A 22-year-old male with a past medical history of asthma presented for a month-long history of pain and swelling of his left hand. He initially developed swelling of his right arm which resolved spontaneously but followed by swelling and pain of his left wrist and second finger. He denies any rash, fever trauma, insect bite, history of STDs, recent diarrhea or viral illness. Physical examination showed swelling of his left 2nd finger, mainly over 2nd metacarpophalangeal joint with reduced range of motion. Laboratory studies showed elevated ESR and CRP, positive urinary gonorrhea and chlamydia PCR. Otherwise, rheumatoid factor, anti-CCP, ANA and HLA B27 were negative. Xray of left hand and MRI confirmed diagnosis of osteomyelitis and septic arthritis. He underwent arthrotomy washout and synovial fluid sampling. However, synovial fluid PCR for Gonorrhea and Chlamydia were inconclusive. We made the diagnosis of septic arthritis of 2nd MCP joint complicated with osteomyelitis likely secondary to gonorrhea and/or chlamydia. The patient was treated with IV Ceftriaxone and PO Doxycycline for a total of 6 weeks course. Subsequent outpatients follow up revealed marked improvement of joint swelling/pain.

Gonococcal osteomyelitis is an extremely rare complication of disseminated gonococcal infection. On literature review, there were approximately 20 cases reported to date. The typical presentation includes a well-appearing individual with asymmetric joint pain in the small joints of hands and feet. Fever, rashes and constitutional symptoms may be present.

Historically, gonococcal osteomyelitis was not a specifically identified entity due to the difficulty on obtaining a confirmed diagnosis by PCR from samples. However, even without a positive culture or PCR, it is extremely important to have early recognition, close monitoring and prolonged treatment with antibiotics to guarantee complete recovery and prevent further complications or chronic arthritis.

Osteomyelitis caused by N. gonorrhoeae is a rare complication of its infection. Early recognition and appropriate treatment is imperative, especially with growing antibiotic resistance. Further research and trials are needed to help establish guidelines for proper management.
A Rare Case of Metastatic Medullary Thyroid Cancer in a Young Patient

Medullary thyroid cancer (MTC) is a rare neuroendocrine tumor of parafollicular C cells of thyroid gland. It accounts for 1-2% of thyroid cancers in the United States. 75% of these cases are sporadic in nature while the less common hereditary form as part of MEN2 syndrome presents at an earlier age.

A 24-year-old male with past medical history of ADHD presents to the hospital with a rapidly increasing swelling on the right side of his neck for the past 1 month associated with difficulty in breathing. He also reports intermittent facial flushing, and chronic diarrhea. Preliminary lab work including thyroid function tests was unremarkable. CT of neck and chest showed a large heterogeneous infiltrative right thyroid lobe mass crossing the midline and extending to superior mediastinum with mass effect of right internal jugular vein and common carotid artery. CT also revealed contiguous matted lymph node enhancing mass extending through the right levels 2, 3, 4, and 5 to the supraclavicular fossa suggestive of possible nodal metastasis. Calcitonin level and CEA were both elevated at 11633 and 160.6 respectively. FNAC of the thyroid mass was consistent with medullary carcinoma of thyroid. Plasma metanephrines were within normal limits. Patient followed up with oncology as outpatient. Genetic testing was negative for pathogenic mutations in 16 thyroid and PGL-Pheo related genes; including RET gene. MRI abdomen and pelvis was unremarkable. Patient was diagnosed with sporadic MTC, stage IVA (T4A, N1B, M0), and scheduled to follow up with surgery for thyroid resection and lymph node dissection.

Clinical behavior of Sporadic MTC is unpredictable. Although common in 4th-6th decades of life, it can also occur in younger population. Early diagnosis of sporadic MTC is important since age and stage at time of diagnosis are significant independent prognostic factors. Ten-year survival rates for patients with stages I, II, III, and IV MTC are 100%, 93%, 71%, and 21% respectively. Every patient with diagnosed medullary thyroid cancer should undergo genetic testing for the presence of RET mutation. Early diagnosis of patients with MEN2 associated MTC is important as it allows for screening of family members and thus prevention.
Rapid Progression of Pneumonitis Following "Natural" Mineral Oil Insecticide Zevo

Patients with underlying pulmonary disease should not be deceived by the word “natural” oil to describe commercial insecticides. The following case exemplifies how such exposure can lead to rapid progression of pneumonitis and multi-organ failure.

A 75-year-old female with past medical history of coronary artery disease, tobacco use and idiopathic chronic respiratory failure on 2L oxygen was admitted with severe shortness of breath. Initial chest x-ray showed mild bilateral patchy opacities. Computed Tomography (CT) chest revealed diffuse bronchiectasis and mixed pattern of ground glass opacities consistent with pneumonitis without visualized honeycombing. Extensive pulmonary workup including atypical pneumonia panel, MRSA, BAL cultures, AFB and COVID-19 was negative. The patient’s respiratory status declined throughout admission. IV steroids were started and broad-spectrum antibiotics were initiated. Bronchoscopy was inconclusive. The patient’s respiratory status continued to decline and on day ten, she was intubated. In the next 24 hours, she began to have multiorgan failure including worsening kidney function. Follow-up CT imaging to investigate the etiology showed perihepatic fluid and a pneumomediastinum. On day twelve of admission the patient deteriorated further and after discussion with the family the patient was transitioned to comfort care and soon after expired.

Additional history obtained from family revealed she began using a commercially available insect spray (Zevo) in her home prior to presentation.

The manufacturer’s safety card for Zevo is rated as safe for human exposure. The major carrier ingredient is mineral oil which can cause severe pulmonary problems in some patients including Lipoid Pneumonia and, in particular, Exogenous Lipoid Pneumonia. This is usually a diagnosis made by having a high index of suspicion as well as BAL findings showing lipid-laden macrophages.

Taking a good social history is often overlooked, however this case illustrates its importance amidst the COVID-19 pandemic. Although branding of products points toward a “natural” set of ingredients, chemical irritants remain a lethal cause of rapid decline especially in patients with underlying pulmonary disease. Patients should be made aware of the potentially fatal side effects of these irritants.
Myocardial Stunning: A Rare Complication of Cardioversion

Synchronized cardioversion (CV) is considered a safe, effective, and very reliable method in treating atrial fibrillation. We report a case of rare myocardial dysfunction and stunning following routine CV. A 71 years-old female with history of paroxysmal atrial fibrillation who presented for an elective Trans-esophageal echo (TEE) followed by CV. Transthoracic Echocardiogram (TTE) as an outpatient showed normal ejection fraction (EF) of 55-60% with no valvular disease and no left ventricular (LV) thrombus noted. TEE confirmed absence of LV thrombus, and CV with delivery of 200J was successful in restoration of sinus rhythm immediately. Within minutes, patient was noted to have significant drop in blood pressure followed by PEA cardiac arrest. After 3 rounds of epinephrine and continued chest compressions, ROSC was achieved. Patient was intubated and STAT EKG following the event revealed ST elevations in inferior and anterior leads. She was taken emergently to catheterization lab, where she had another, but brief, PEA cardiac arrest episode followed by ROSC. Coronary angiography revealed normal coronary arteries with no noted obstruction. Pulmonary angiogram was done and ruled out concerns for pulmonary embolism. Bedside TTE revealed significantly reduced biventricular function, with LV EF around 20-25%. Patient was diagnosed with myocardial stunning and cardiogenic shock secondary to that. LV Impella device was placed, and Milrinone and norepinephrine drips for inotropic and pressor support were started. Over the next few days patient showed significant improvement of cardiac function and was successfully extubated after removal of cardiac support measures. She was started on medical therapy and follow up as an outpatient revealed restoration of cardiac function. Our case raises vigilance to a rare but fatal complication to a routine cardiac intervention as synchronized cardioversion. Myocardial stunning is referred to a significant but often reversible dysfunction of the myocardium following an insult as ischemic, hypoxic, or obstructive events. Treatment is usually supportive, but in rare cases can require significant resuscitative measures, with notable restoration of cardiac function over time.
Prolonged Supratherapeutic Level of Phenytoin After Intentional Ingestion

Background:
Phenytoin is a commonly used anti-Seizure medication that has a narrow therapeutic index and its toxicity can be exacerbated by other drugs. Phenytoin’s half-life is known to be around 40 hours, but the clearance can be prolonged depending on each patient’s case. This clinical vignette presents a patient with supratherapeutic phenytoin level for 156 hours due to multiple factors.

Case Presentation:
A 66-year-old male with a past medical history significant for seizure disorder, hypertension, and history of subdural hematoma presented with complaints of altered mental status and hypothermia. The patient was found unresponsive by his family in the morning outside of his house with a big bottle of liquor and an empty bottle of phenytoin next to him. On presentation, the patient was hypothermic and hypotensive with a high alcohol level and supratherapeutic phenytoin level. The urine drug screen was negative. Imaging studies were unremarkable.

He continued to have severe ataxia for seven days. Phenytoin level continued to fluctuate above supratherapeutic level (>20ug/ml) for 7 days despite the initial phenytoin level being not too high to saturate liver enzyme. Bezoar formation was suspected, and an abdominal x-ray was taken. The abdominal x-ray was non-diagnostic but showed large fecal material. His prolonged course was possibly attributed to constipation because the phenytoin level pivoted after the resolution of constipation. His level was stable, and he was discharged home.

Discussion:
The narrow therapeutic index, the wide inter-individual variability in phenytoin metabolism, and the zero-order pharmacokinetics of the medication are responsible for the observed dose-related toxicity. This case illustrates a patient with phenytoin toxicity in which there’s a high suspicion of bezoar formation given the course of a prolonged supratherapeutic concentration with an initial decline and subsequent increase in serum levels. Our case highlights the importance of knowing the different changes in pharmacokinetics in patients using phenytoin with concomitant use of other medications or substances and considering other possibilities for an unexpected clinical course. It will better prepare physicians for similar scenarios to identify contributing factors and to decide when to resume phenytoin safely, which may improve morbidity and mortality in these patients.
Multiple Manifestations of Methadone-Induced Cardiotoxicity

Introduction:
Methadone is a medication commonly used in the treatment of opioid use disorder. Though effective, methadone does have risk of adverse events and has been associated with various forms of cardiotoxicity. This case details multiple manifestations of methadone-induced cardiotoxicity in a patient secondary to misuse of high dose, methadone maintenance therapy (MMT).

Patient Description:
A 30-year-old woman with past medical history of opioid use disorder on MMT presented to the emergency department with concern for 1-day of dyspnea and chest discomfort since a witnessed, syncopal event the day prior lasting 7 minutes with spontaneous resolution. The patient experienced a prodromal flushing sensation and urinary incontinence, though denied post-syncopal confusion. She did report recently self-administering higher doses of methadone than prescribed.

Upon presentation, the patient was hypotensive and had an elevated NT-proBNP and troponin. EKG showed normal sinus rhythm with pre-existing inferior lead T-wave inversions and prolonged, corrected QT interval (QTc) of 528ms. Transthoracic echocardiogram demonstrated severe left ventricular dysfunction with an ejection fraction (EF) of 18% and apical akinesis with preserved basal segment contractility.

Upon admission, methadone was held until QTc improved. A stress cardiac MRI (CMR) showed no evidence of late gadolinium enhancement though did demonstrate improved EF. The patient was diagnosed with methadone induced cardiac syncope likely secondary to polymorphic ventricular tachycardia (PMVT), as well as stress cardiomyopathy.

Discussion:
This case demonstrates rare manifestations of methadone-induced cardiotoxicity from both a rhythm and a structural perspective. Few similar pediatric cases have been reported. Our patient likely experienced cardiac syncope secondary to PMVT in the setting of prolonged QTc from high-dose methadone. Methadone is thought to affect QTc interval by direct effect on the resting membrane potential. Though there was some concern for possible seizure, the clinical history, clinical course, and prolonged QTc were consistent with arrhythmogenic cardiac syncope. In addition to an arrhythmia, our patient suffered stress cardiomyopathy, as indicated by confirmation of non-ischemic cardiomyopathy on stress CMR and improvement in the EF. This case demonstrates the potential for MMT misuse to lead to methadone-induced cardiotoxicity, which may manifest as arrhythmias and/or structural dysfunction.
Dietary Salt Substitute Causing Cardiac Arrest

Introduction
Congestive heart failure (CHF) is a growing epidemic and public health concern. Due to its increasing prevalence, patient education regarding lifestyle interventions and risk factor modification is paramount. Hypertension is a strong risk factor for the development of CHF. As a result, many patients are counseled on the importance of decreasing dietary sodium. We present a remarkable case of an individual who suffered from cardiac arrest as a result of dietary substitutes in an attempt to adhere to his dietary restrictions.

Case Presentation
A 79 year old man with a past medical history of hypertension and CHF presented to the emergency department (ED) after a witnessed, out of hospital cardiac arrest. In an effort to adhere to his dietary sodium restrictions, the patient had been utilizing the dietary substitute “NoSalt”. EMS was contacted due to dizziness and weakness, and found the patient to have collapsed due to pulseless electrical activity (PEA) arrest. Cardiopulmonary resuscitation (CPR) was initiated and return of spontaneous circulation (ROSC) was achieved. Upon presentation to the ED, his labs were notable for a potassium of 8.0 mEq/L and a creatinine of 2.28 mg/dL. EKG demonstrated a widened, bizarre QRS. The patient was begun on continuous renal replacement therapy (CRRT), and repeat laboratory findings later demonstrated significant improvement in the patient’s hyperkalemia.

Discussion
We present a remarkable case of excessive dietary salt substitute intake leading to PEA arrest. In an effort to reduce dietary “salt” intake, our patient turned to a seemingly safe alternative: “NoSalt”. NoSalt is a salt substitute consisting of potassium chloride. The patient reported consuming a generous amount of this NoSalt substitute with each meal, in addition to bananas and potatoes daily. The potassium content of these foods is approximately 16 mEq, 11 mEq, and 23 mEq of potassium, per serving respectively. This case demonstrates the importance of thorough patient education, as well as physician awareness of the potential harms of dietary supplements when used in excess. Nutritionist involvement should be considered to assist in navigating new dietary restrictions.
A Challenging Combination: Discoid Lupus, Limited Scleroderma, and Sarcoid Myopathy

Introduction:
Sarcoid myopathy is a rare manifestation of sarcoidosis in which granulomatous inflammation leads to muscle fibrosis and tissue injury. We present a case of a patient with chronic shoulder pain and proximal muscle weakness with new diagnoses of discoid lupus and limited scleroderma who was also found to have sarcoid myopathy.

Case Report:
A 47-year-old male with a past medical history of chronic right shoulder pain, arthralgias, and bilateral carpal tunnel syndrome presented to the rheumatology clinic for evaluation of joint pain. He had associated morning stiffness, Raynaud’s phenomenon, and NSAIDs helped his pain. On physical examination he was noted to have multiple discoid lesions on his face and scalp and sclerodactyly that did not cross his elbows. Laboratory work-up was significant for elevated inflammatory markers, positive ANA (1:1280 speckled), positive anti-RNP antibody, and positive SSA antibody. He was initially started on hydroxychloroquine and a prednisone taper, which did improve his symptoms. However, he continued to have right shoulder pain and developed bilateral upper extremity weakness. An EMG done with evidence of a diffuse, irritable "myopathic" process, an MRI of the shoulder was concerning for myositis, and he had an elevated serum creatine kinase. He ultimately underwent a left deltoid muscle biopsy which showed chronic active myopathy with non-caseating granulomas consistent with sarcoid myopathy. His diagnoses also included discoid lupus and limited scleroderma, with consideration for possible mixed connective tissue disease.

Discussion:
Sarcoid myopathy is a rare manifestation of sarcoidosis in which T lymphocytes, mononuclear phagocytes, and non-caseating granulomas accumulate in muscle tissue and can lead to muscle fibrosis and injury. Sarcoid myopathy can co-exist with rheumatologic conditions and can be difficult to distinguish. Therefore, muscle biopsy may be needed. Muscular weakness can be a clue that there is another process at play.
Cases have been reported of sarcoid myopathy coupled with other rheumatologic conditions such as systemic sclerosis, SLE, Sjogren’s syndrome, and psoriatic arthritis. However, our patient’s combined diagnoses of discoid lupus, limited scleroderma and sarcoid myopathy appears to be rare.
Did We Do That? A Case of Trimethoprim-Sulfamethoxazole Induced Liver Injury

Trimethoprim-Sulfamethoxazole (TMP-SMX) is frequently prescribed as a well-tolerated antibiotic. Common adverse effects involve skin rash, kidney injury, and gastrointestinal distress. A rare but serious adverse effect is drug induced liver injury. We present a case of a 58-year-old female who developed acute liver injury five days after the initiation of therapy with TMP-SMX. The patient presented with abdominal pain with positive murphy’s sign and jaundice. She had been started on TMP-SMX for a urinary tract infection five days prior to her presentation. She had taken TMP-SMX previously without any reported adverse effects. Patient was evaluated for gall bladder disease, and her TMP-SMX prescription was held on arrival. The patient rapidly improved without any additional intervention. Given negative workup, cholestatic pattern of liver injury in laboratory results, and quick improvement of symptoms following TMP-SMX discontinuation, the patient was diagnosed with drug-induced liver injury due to TMP-SMX with instructions to avoid TMP-SMX in the future. This presentation will review the specifics of the case as well as go through a review of the literature surrounding drug induced liver injury secondary to TMP-SMX. TMP-SMX hepatotoxicity is a well-known but rarely described phenomenon. By sharing our experience, we hope to raise awareness of this condition among the prescribing providers.
Capnocytophaga Canimorsus Bacteremia with Encephalopathy, Thrombocytopenia, and Hepatitis in an Immunocompetent Patient

Capnocytophaga is a gram-negative fastidious bacterium, a part of dogs' oral flora. Following a dog bite, immunosuppressed and particularly asplenic patients may develop various infections, ranging from mild cellulitis to life-threatening meningitis, endocarditis, and sepsis with multiorgan system failure. The severity of infection frequently correlates with the degree of immunosuppression. However, the literature lacks data about Capnocytophaga infection in immunocompetent patients. We describe the case of a 72-year-old healthy female presenting to the hospital for altered mental status. In the emergency department, she was febrile and tachycardic. Blood work revealed leukocytosis, profound thrombocytopenia, and hepatocellular injury. We started broad-spectrum antibiotics to provide coverage for possible meningitis while awaiting platelet recovery to perform a lumbar puncture. The response to antibiotics was dramatic. Mentation and liver enzymes were back to baseline within 48 hours. The patient received a platelet transfusion, in addition to antibiotics, and her platelet count started recovering after 72 hours allowing us to perform a lumbar puncture. Cerebrospinal fluid (CSF) analysis revealed mild pleocytosis with neutrophil predominance. CSF culture and bacterial polymerase chain reaction (PCR) were negative. Blood cultures drawn on admission grew gram-negative rods later identified as Capnocytophaga canimorsus. Capnocytophaga canimorsus was also detected in blood by bacterial PCR. A detailed physical exam revealed a minor superficial injury of her right index finger sustained while feeding her sick dog 2 days prior to the symptom onset. The patient completed 2 weeks of ceftriaxone with complete recovery. By describing this case, we hope to help better understand Capnocytophaga infection in immunocompetent patients and remind all providers that timely administration of antibiotics for dog bites, for both prophylactic and therapeutic purposes, can be lifesaving.
Inflammatory Arthritis Associated with IgA Vasculitis

Introduction
IgA nephropathy is a most common cause of primary glomerulonephritis in resource rich areas of the world, occurring most frequently in individuals of East Asian descent. It can be associated with systemic manifestations, including inflammatory arthritis.

Case Presentation
A 19-year-old male with end stage renal disease on peritoneal dialysis due to IgA Nephropathy, hypertension, anemia of chronic disease, and dilated cardiomyopathy presented with acute-onset left elbow pain and erythema without SIRS criteria. Physical exam revealed large effusion without evidence of abscess on MRI. Synovial studies found elevated neutrophil count without microorganisms or crystals and he was empirically treated for septic arthritis. He demonstrated no improvement with antibiotics, then developed abdominal pain, dactylitis, and painful knee synovitis. Extensive rheumatologic serology and infectious studies were negative. Hand X-rays showed periarticular calcifications secondary to renal osteodystrophy. His negative workup with history of IgA nephropathy was consistent with inflammatory arthritis associated with IgA vasculitis. Treatment with prednisone resulted in rapid improvement.

Discussion
IgA vasculitis can present with skin manifestations, kidney disease, arthritis and abdominal pain. Diagnosis should be suspected in patients presenting with renal impairment associated with arthritis with or without abdominal pain and purpura and can be confirmed with kidney biopsy. In patients with confirmed IgA vasculitis, presentation with synovitis should be worked up for alternative causes, but physicians should be aware of inflammatory arthritis associated with IgA vasculitis. These patients usually present with symmetric arthritis that may be associated with rash and abdominal pain. In cases with monoarticular joint pain, other causes such as septic arthritis and toxic synovitis should be ruled out. Joint aspiration can differentiate between infection and other causes of inflammation. Our patient was worked up appropriately for septic arthritis. His negative infectious and inflammatory workup, chronic course with subsequent polyarticular synovitis, and response to prednisone, support a diagnosis of inflammatory arthritis associated with IgA vasculitis.

Conclusion
Inflammatory arthritis associated with IgA vasculitis and nephropathy can present with arthritis involving few or multiple joints, usually in the lower extremities. It is important to consider this diagnosis even in patients presenting with mono-articular inflammatory arthritis.
Disseminated Mycobacterial Infection in Underlying Hematological Malignancy Presents as Neutropenic Fever of Unknown Origin

Introduction
Mycobacterium avium complex (MAC) is a non-tuberculous mycobacteria known to cause opportunistic infection in immunocompromised individuals. It can be isolated from multiple sites and causes disseminated infection affecting multiple organ systems. Patients often present with non-specific symptoms of night sweats, fevers, and fatigue in the setting of known immunodeficiency most commonly from advanced HIV and AIDS, but can also manifest in other immunocompromised states such as hematologic malignancy. We present a case of disseminated MAC in an HIV(-) patient preceding a diagnosis of B-ALL.

Case Presentation
A 38 year old male with a history of significant intermittent alcohol use presented with fatigue, fevers, chills, and night sweats after ceasing alcohol consumption five weeks prior. His initial exam and labs were consistent with neutropenic fever and bicytopenia. Thoracic imaging revealed mediastinal lymph node enlargement as well as a right upper lobe infiltrate. Bronchoscopy and endobronchial ultrasound with fine-needle aspiration of lymph nodes did not reveal any neoplastic changes, but did show necrotizing granulomatous inflammation and acid fast bacteria. He was started on guideline-based therapy for MAC infection. HIV PCR testing was negative multiple times. Bone marrow biopsy was positive for B-Cell acute lymphoblastic leukemia. He was started on induction chemotherapy. Further lymph and lung biopsies were able to isolate mycobacterium.

Discussion
The case highlights a diagnostic challenge of neutropenic fever without a definitive source, as well as an unusual presentation of disseminated MAC. Negative systemic initial bloodborne testing pointed toward a hypothesis of alcohol use causing immunocompromisation, leading to infectious presentation. Numerous potential sources of infection were considered as the driving force of fevers including cultured fungal pneumonia and perirectal abscess. However, fevers did not subside with over seven days of broad spectrum antibiotic and antifungal treatment. Clarity was brought to the clinical picture when MAC was cultured in lymph nodes, and even further when bone marrow biopsy was consistent with B-ALL.

Conclusion
Disseminated MAC can present in immunocompromised individuals with underlying hematologic malignancy. In HIV(-) patients with this infection, it is an important diagnostic consideration to include these neoplastic processes as a preceding cause of immunocompromisation.
Acute on Chronic Pulmonary Embolism Presenting with Complete AV Block

Introduction
Acute pulmonary embolism can present with a range of symptoms and clinical findings, including cardiac arrhythmias. Complete atrioventricular nodal blockade is a rare sequelae of pulmonary embolism. We present a case of a 71-year-old female presenting with recurrent syncope and palpitations, found to have acute on chronic pulmonary embolism and complete heart block.

Case Presentation
A 71-year-old female presented to the emergency room with a chief complaint of recurrent syncopal events, resulting in fall with head trauma, and progressive shortness of breath, for the past three weeks. Electrocardiogram showed complete heart block with a ventricular rate at 30 beats per minute. Initial lactic acid was elevated at 3.3 mmol/L and NT-Pro BNP of 16,519 ng/L. Computed tomography angiography showed extensive bilateral emboli involving main, lobar, and segmental vessels with evidence of right heart strain. Doppler ultrasound of the right lower extremity revealed deep vein thrombosis of the distal right femoral vein. Echocardiogram showed an estimated right ventricular systolic pressure (RVSP) of 95 mmHg, moderately dilated right ventricle, with systolic and diastolic septal flattening. Intermittent runs of mono and polymorphic ventricular tachycardia were noted on telemetry, confirmed by ECG. Our Pulmonary Embolism Response Team (PERT), including cardiology, interventional radiology, pulmonology, and cardiothoracic surgery discussed management options, and given the severity of her condition, the patient was taken for an emergent surgical embolectomy.

Discussion
Our patient presented with complete atrioventricular (AV) block with a massive pulmonary embolism. Pulmonary emboli are considered massive if they cause hypotension, cardiac arrest, or syncope. Recommended treatment options includes systemic thrombolitics, catheter-directed therapy, or surgical thrombectomy. Given that our patient had persistently elevated systolic blood pressure >180 mmHg and recent head trauma, fibrinolytic therapy was contraindicated. Catheter directed mechanical thrombectomy was contraindicated, as the patient developed polymorphic ventricular tachycardia with attempted placement of transvenous pacemaker. Therefore, the patient required emergent surgical embolectomy, as decided by the multidisciplinary team.

Conclusion
Pulmonary embolism can present with cardiac arrhythmias, with complete atrioventricular block being exceedingly rare. Treatment involves proper diagnosis, and management from a multidisciplinary team of specialists.
A Rare Presentation of Clostridioides Difficile Colitis with Hematochezia

Introduction:
Clostridioides Difficile (C. Difficile) is a spore-forming, toxin-forming bacteria that causes pseudomembranous colitis. There are approximately 450,000 C. Difficile infections every year in the United States making it one of the most common healthcare associated infections. Antibiotic use remains the greatest risk factor. Other risk factors include age>65, recent hospitalization, and use of proton pump inhibitors. C. Difficile infections are associated with high morbidity and mortality. The cardinal symptom of C. Difficile infections is watery or mucousy diarrhea. Fever and abdominal pain are also common. Infections may present with systemic signs of infections or toxic megacolon. Rarely, hematochezia related to ulcers or ischemic colitis has been described as the presenting complaint in the literature. Treatment depends on severity and is based around oral fidoxamicin, vancomycin and intravenous metronidazole.

Case:
A 62 year old male with heart failure, and atrial fibrillation on Xarelto, presented with a heart failure exacerbation. During the hospitalization he developed Enterococcus faecalis bacteremia secondary to an infected sacral decubitus ulcer. Transesophageal echocardiogram showed a vegetation on an ICD lead which was successfully removed. Broad spectrum antibiotics were narrowed to ampicillin and ceftriaxone with a plan for 6 weeks of therapy. The patient subsequently developed hematochezia while maintaining semi-formed stools. A flexible sigmoidoscopy revealed pseudomembranous colitis typical of C. Difficile infection. The hematochezia was thought to be due to friable colonic membranes. Based on the appearance of pseudomembranes the decision was made to start the patient on empiric oral vancomycin. C. difficile toxin stool assay was positive. His sepsis and hematochezia resolved shortly after initiation of vancomycin.

Discussion:
This case represents a rare presentation of C. difficile infection. Endoscopically, the patient displayed typical pseudomembranes with friable tissue which accounted for his hematochezia. Oral vancomycin resolved his sepsis and hematochezia further supporting that his hematochezia was due to his C. difficile infection. This case highlights how C. difficile infection should be on the differential diagnosis of gastroenterology related symptoms even in the absence of diarrhea, especially in the setting of the prolonged antibiotic use. It also highlights the value of direct visualization of the lumen during gastrointestinal bleeds.
Axillary Web Syndrome: A Confounding Presentation to an Already Confounding Phenomenon

Introduction: Axillary Web Syndrome (AWS) remains a poorly understood phenomenon. AWS is a cording of subcutaneous tissue arising in the axilla, extending distally. It is usually recognized in patients after breast surgery, particularly if coupled with axillary lymphadenectomy. AWS has proven difficult to appreciate on various imaging modalities, including ultrasound and magnetic resonance imaging. Based on few biopsies in the literature, cording lymphedema is primarily comprised of lymphatic and venous vessels with possible evidence of thrombosis and recanalization.

Case Presentation: A 73-year-old African-American female, with no recent surgeries or illnesses, presented to our internal medicine residency clinic for discomfort and swelling under her left axilla, which were more prominent with shoulder abduction. She denied any new constitutional symptoms, history of malignancy or breast surgery. She reported being a caregiver for someone in her home who required her total care for several years. She first noticed axillary swelling with uncomfortable tension when brushing her hair. On exam, the patient was afebrile with stable vitals. A cord-like subcutaneous band traversed her left axillary midline and was palpable to her medial forearm with minimal edema, distally. A left breast ultrasound and mammogram were unrevealing, as well as a left upper extremity duplex ultrasound to evaluate for venous thrombosis. She was referred to physical therapy for dedicated stretching and manual therapy.

Discussion: This AWS presentation is unique in that it is not associated with recent breast or axillary surgical procedures. Possible minor trauma in the setting of the patient’s heavy lifting triggered the development of lymphatic compression and potentially intra-lymphatic thrombosis. Like AWS itself, we are left with more questions than answers. As this syndrome is responsive to manual therapy, an accurate diagnosis is key for maintaining mobility and function.
A Rare Case of Coronary Thrombosis Due to Kawasaki Disease in an Adult

Introduction: Kawasaki disease is a mucocutaneous lymph node syndrome that occurs in the pediatric population and is seldom seen in adults. It presents as a self-limited illness with symptoms such as mucosal inflammation, acute vasculitis, rash, and cervical lymphadenopathy. Severe illness in childhood is known for its association with cardiovascular sequelae; most commonly coronary artery aneurysms. We report a rare case of Kawasaki disease associated with coronary artery aneurysms in an adult who presented with syncope and chest discomfort.

Case Report: A 43-year-old African American male with a history of hypertension and asthma exacerbation in childhood requiring intubation, presented to our institution after a syncopal episode and chest tightness. He admitted to COVID-19 infection 2 weeks prior with typical symptoms which had since resolved, ongoing intermittent chest pain for several years, and reported his father died of myocardial infarction in his 60’s. On admission, he was found to be tachypneic with peak troponin of 13,489. EKG showed inferolateral T-wave inversions. He underwent emergent heart catheterization which showed significant thrombotic occlusion to the distal RCA, requiring aspiration thrombectomy. He was noted to have aneurysmal coronary arteries consistent with Kawasaki disease. Upon further questioning, he denied symptoms of Kawasaki disease as a child; however, was unsure whether he had prolonged fevers in childhood. He was discharged on Aspirin, Plavix, and Eliquis with outpatient cardiology follow-up.

Discussion: Kawasaki disease is an acute febrile illness that presents as a self-limited vasculitis mostly affecting the proximal coronary arteries. This can lead to the development of coronary artery aneurysms which are associated with thrombosis. This rarely manifests in adults, as coronary abnormalities often regress after acute illness. There are limited reports of adults who presented with myocardial infarction attributed to persistent coronary aneurysms from presumed Kawasaki disease. In 2017, the American Heart Association published guidelines for the care of patients with Kawasaki disease. However, it did not discuss management of adults with symptomatic ischemic heart disease due to chronic aneurysms after childhood Kawasaki disease. We express the need to formulate guidelines regarding treatment of aneurysms, revascularization, and long-term surveillance in adult patients who present with Kawasaki disease.
Paraneoplastic Anti-LGI1 Limbic Encephalitis with Lymphoplasmacytic Lymphoma Causing Progressive Neuropsychological Decline

Background:
Paraneoplastic autoimmune encephalitides comprise a rare group of disorders that can be seen in conjunction with small cell lung cancers, testicular and breast cancers, or B-cell lymphomas. Within this group, anti-LGI1 mediated disease is even rarer, with prevalence rates being as low as 0.7 per 100,000.

Case:
A 67-year-old Caucasian male presented with progressively declining cognitive capacity for the past one year, with a prior admission for severe agitation requiring physical restraints and intravenous sedatives at the intensive care unit. Work up for underlying infectious meningitis/encephalitis and silent seizures at that time had returned negative. He was diagnosed with lymphoplasmacytic lymphoma with CNS involvement, that was being treated with methotrexate and rituximab. Flow cytometry revealed monoclonal B-cell lymphocytosis, with positive expression of CD5, CD11c, CD19, CD20, CD23, CD45, CD200 and kappa light chain. Despite appropriate treatment, the patient’s neurological symptoms persisted, with complaints of mild retrograde amnesia, bradyphrenia, tangential speech, depressed mood, and uncontrolled pain. Autoimmune encephalitis panel was positive for LGI1-IgG antibodies, consistent with autoimmune limbic encephalitis. Treatment was initiated with IVIG for a five-day course. Unfortunately, the patient’s symptoms remained largely unchanged; the patient and his family then elected to transition his care to hospice.

Discussion:
This case illustrates the poor neurological outcomes associated with autoimmune limbic anti-LGI1 encephalitis. Neurological symptoms can range from severe agitation to mild confusion and amnesia; corresponding psychiatric symptoms include severe depression and uncontrolled pain. Studies demonstrate improved neurological prognosis in patients with early initiation of immune therapy. Thus, the index of suspicion for paraneoplastic autoimmune limbic encephalitis in patients with certain cancers and neuropsychiatric manifestations should be high as prompt treatment carries a better prognosis.
An Unusual Presentation of Coxsackievirus B2

BACKGROUND
Viral infections of the heart are relatively common, and viral myocarditis is an important cause of dilated cardiomyopathy. Coxsackie B virus is one of the most important etiologies of viral myocarditis. We present a case of cardiac tamponade, followed by polymorphic ventricular tachycardia due to severe QT prolongation due to coxsackie virus associated with normal ejection fraction.

CASE PRESENTATION
A 39-year-old healthy woman presented to the hospital with chest pain and shortness of breath for 5 days. Physical examination showed elevated jugular venous pressure, hypotension, and muffled heart sounds. Laboratory evaluation: Normal except for elevated sedimentation rate and C-reactive protein. Electrocardiogram showed sinus tachycardia with low voltage QRS complexes. An urgent bedside echocardiography revealed a normal ejection fraction and moderate pericardial effusion with cardiac tamponade. Emergent pericardiocentesis was performed which yielded 405 ml of cloudy fluid. Autoimmune workup was negative. Fluid analysis was largely unremarkable other than a positive titer of 1:640 and 1:320 for type-1 and type-2 Coxsackie B virus, respectively. Coronary angiography was normal. Repeat echocardiogram showed resolution of effusion and she was discharged on a limited course of colchicine. Subsequently, the patient was readmitted for chest pain, dyspnea, and productive cough a week later. She was febrile and hypotensive on presentation. Computed tomography of the chest redemonstrated mild pericardial effusion. The hospital course was complicated by cardiopulmonary arrest secondary to prolonged QTc and polymorphic ventricular tachycardia. Cardiac MRI was obtained which did not reveal any myocardial infiltration, contraction abnormalities, reduction in ejection fraction or pericardial enhancement ruling out conditions such as sarcoidosis. Defibrillator was implanted for secondary prevention due to persistent QTc of 582msec.

CONCLUSION
Viral pericarditis in most cases follows a benign self-resolving course, but literature review showed that coxsackievirus can cause long-term damage to the myopericardium that leads to dilated cardiomyopathy in 25-40% of cases. In patients with acquired QTc prolongation of unknown etiology, inflammatory heart disease especially myocarditis should be considered. Experiments in mice with coxsackie virus induced myocarditis have shown that inflammatory infiltrate forms an important arrhythmogenic substrate. Treatments targeting immunological and inflammatory pathways may be an attractive therapeutic approach.
Post Infectious or Crystal Meth Induced? A Case of Glomerulonephritis Associated with Rectal Crystal Methamphetamine Use

Introduction
Amphetamines can affect the kidneys through different mechanisms and can cause glomerulonephritis, hypertension, and chronic kidney disease. We present a case of membranoproliferative glomerulonephritis that was possibly induced by crystal methamphetamine.

Case Description
A 47-year-old man with a past medical history of substance abuse presented to the hospital with abdominal pain, nausea, and dark colored urine. He was admitted prior to this presentation for proctitis with sepsis and was treated with antibiotics. On presentation, he was tachycardic and febrile with elevated WBC and creatinine. UA showed RBCs, WBCs, and protein. His CXR suspected pneumonia. CT scan of the abdomen showed persistent proctitis. He was started on IV fluids and antibiotics for sepsis due to pneumonia, urinary tract infection and recurrent proctitis. The patient’s kidney function did not improve, and he developed scrotal edema, ascites, and bilateral lower extremity edema. Further workup revealed nephrotic range proteinuria, with positive ANA and rheumatoid factor and Complement C3 level was slightly reduced. Renal biopsy showed proliferative glomerulonephritis with early membranoproliferative features and possible etiologies included cryoglobulinemia, post infectious, as well as autoimmune processes. The patient was started on steroids and underwent hemodialysis. Further workup was negative for hepatitis, HIV, double stranded DNA or anti-smith antibody. The patient admitted later on to injecting crystal methamphetamine into his rectum prior to the first hospitalization and that might have triggered the proctitis and glomerulonephritis eventually.

Discussion
This case represents one of the possible renal complications of drug abuse. Although rare, severe acute kidney injury with dialysis requirements can occur in patients using methamphetamine. A case series from South Africa reports that among patients with methamphetamine use, hypertension was found in 89% of the patients, and among patients who underwent renal biopsy, 58% had mesangiocapillary glomerulonephritis. This patient’s glomerulonephritis can be post-infectious following proctitis, but cultures were negative, though he responded to empiric antibiotic therapy. Sigmoidoscopy done during the admission for proctitis showed edematous rectal mucosa, but biopsy showed normal rectal mucosa without histological abnormalities. He remains on renal replacement therapy. Further research is needed to fully understand the renal effects of crystal methamphetamine.
McConnell’s Sign: Is it really a Pulmonary Embolus? Revisiting a Clinical Adage to Appreciate Mechanisms of Clinical Findings

INTRODUCTION:
Pulmonary embolism (PE) in the US has an incidence of 600,000 cases and accounts for 100,000 deaths annually. McConnell’s sign (right ventricular dysfunction with apical sparing) on echocardiography (ECHO) has long been described as a specific finding for PE. We present a case of dyspnea in a patient with idiopathic pulmonary fibrosis (IPF) and argue that this finding should be interpreted as acute pulmonary hypertension due to any cause, not just PE.

CASE:
The patient is a 70-year-old lady with IPF, COPD, obstructive sleep apnea, paroxysmal atrial fibrillation, hypothyroidism and COVID-19 pneumonia a year prior. She was discharged 3 days prior after receiving 3 units of packed red blood cells for a suspected GI bleed, and Apixaban was discontinued. She now presented to the ER with worsening dyspnea and was found to be hypoxic. Pulmonary examination revealed inspiratory rales, sibilant rhonchi and dullness to percussion. Pre-test probability for PE was high. ECHO revealed McConnell’s sign, and right ventricular (RV) systolic pressure of 47 mmHg. CTA did not reveal a PE, but did show extensive bilateral ground-glass and consolidative opacities and right-sided pleural effusion. Aggressive intravenous diuresis was commenced, resulting in marked clinical and radiologic improvement of pulmonary edema. On the 7th day decision was made to proceed with left and right heart catheterization. She had drug-eluding stents placed 2 coronary arteries, RV systolic pressure was 36 mmHg, and post-procedure ECHO was unremarkable. She was discharged home with significant symptomatic improvement.

DISCUSSION:
Idiopathic pulmonary fibrosis (IPF) is a disease with high morbidity and mortality, and is independently associated with increased risk of PE and bleeding. Reliance on the specificity of McConnell’s sign per traditional teaching exposed our patient to Heparin’s bleeding risk, created clinical complexity and delay in specific diagnosis. Our hypothesis is that pulmonary-edema-induced V/Q mismatch and hypoxic vasoconstriction caused acutely elevated pulmonary arterial pressure, which led to RV dyskinesis. Any process that acutely elevates RV afterload (like obstructive PE) can produce these findings. It is always important to interpret any clinical findings with context, and understanding mechanisms can help clinicians form better differential diagnoses and efficient therapeutic plans.
Central retinal artery occlusion (CRAO) develops typically in the mean age group of 60-70 years of age, primarily seen in these important associations: hyperlipidemia (particularly low-density lipoproteins), carotid artery disease, diabetes, and hypertension. Of these risk factors, carotid artery disease accounts for nearly 18% of all CRAO cases. The predominant mechanisms behind the development of CRAO in carotid artery disease involve plaque rupture and embolism, significant carotid artery stenosis, and the release of local vasoconstrictors. We present an unusual case of a young healthy gentleman with acute onset CRAO in the setting of carotid artery disease, who does not meet the typical risk factor profile for arterial disease.

A 44-year-old athletically built gentleman with history of well-controlled hypertension, nasopharyngeal carcinoma status post 8 rounds of radiotherapy and chemotherapy nearly 20 years ago, and iatrogenic hypothyroidism came with painless right supero-temporal loss of vision. On presentation, vital signs were normal. On visual acuity testing patient could perceive only hand movement in Right eye (OD) while 20/20 in the left eye (OS). Dilated fundus exam showed clear vitreous, normal cup-disc ratios bilaterally, Cherry red spot with macular edema OD. NIH was 1 on admission. Rest of the physical exam was normal. Lipid panel was normal. Blood counts and metabolic panel were within normal limits. Computed tomography (CT) of the head and MRI brain was unremarkable. CT angiogram showed significant calcified atheromatous plaques in common carotid with 50% stenosis bilaterally and an ulcerated plaque in right common carotid artery. He was started on DAPT and atorvastatin and continued on home dose of lisinopril and levothyroxine at discharge. Patients without traditional risk profiles for typical atherosclerosis should always raise suspicion for rare causes of vasculopathy. Given the patient’s history of radiation with concomitant chemotherapy, radiation-induced vasculopathy (RIV) fits right in. They typically present late, usually more than 10 years from radiation exposure, more frequently seen with concomitant chemotherapy exposure. Though there are established pathogenetic pathways involving chronic oxidative stress and NFkB pathways supporting RIV however diagnostic tools remain limited. Nevertheless, a high index of suspicion is necessary to prevent misclassification of such patients into traditional atherosclerosis.
A Case of Lhermitte Duclos Disease with Conversion Disorder

A 43-year-old Caucasian female presented to the emergency room with focal neurologic symptoms suspicious of stroke. MRI was negative for stroke, however, revealed a rare benign brain tumor called Lhermitte Duclos disease.

A 43-year-old Caucasian female with a history of assault with head trauma and strangulation and depression presented with intermittent slurring of speech, expressive aphasia, right-sided weakness and numbness for 3 days. The patient was assaulted by her boyfriend 2 months prior to her presentation and was having multiple life stressors following the assault. She was also hospitalized 9 years ago after being assaulted by her ex-husband. Initial neurological examination revealed right facial drooping, right shoulder shrug weakness, 4/5 strength in right upper extremity, 3/5 strength in right lower extremity, tingling sensation throughout the right side. Stroke protocol was activated, patient was outside time window for alteplase. MRI brain showed no acute ischemic changes, however revealed non-enhancing asymmetric enlargement of the right cerebellum which appeared hypointense on T1- with hyperintense signal on T2-weighted images, consistent with Lhermitte Duclos disease and were also demonstrated on a previous MRI from 9 years ago. Her symptoms persisted and conversion disorder was diagnosed.

Lhermitte Duclos disease is a rare benign brain tumor (dysplastic gangliocytoma) of the cerebellum. There have been less than 250 cases reported in medical literature to date. Majority of patients may start to present at the 3rd or 4th decade of life with cerebellar dysfunction or typical symptoms of increased intracranial pressure. To our best knowledge, this is the second reported case of LDD presenting as a psychiatric disturbance.

Any abnormal enlargement of the brain can disturb its general neuroanatomical structure leading to imbalances in neurotransmitters production, which can predispose the patient to psychiatric disorders. LDD also falls into this category. There is a demand for additional studies to be done about the neurochemical effects of brain tumor, that may shine a light on poorly understood psychiatric disorder such as conversion disorder. Furthermore, general practitioners should pay more attention to the psychiatric manifestation of rare brain tumors.
Antiphospholipid Antibodies and Vitamin D Levels in COVID 19 with and Without Arterial or Venous Thrombosis: A Case-Control Study

Introduction: Coronavirus disease-2019 (COVID-19) is associated with thromboembolism. Antiphospholipid antibody (APLa) formation is one of the mechanisms. Vitamin D deficiency has been associated with thrombosis in antiphospholipid antibody syndrome.

Objective: Measure APLa and vitamin D in hospitalized COVID-19 patients with and without thrombosis to evaluate if thromboembolism is associated with concomitant APLa and vitamin D deficiency.

Methods: Case-control study. Hospitalized COVID-19 patients with a thromboembolic event (ischemic stroke, myocardial infarction, deep venous thrombosis/pulmonary embolism, Cases (n=20) and Controls(n=20): Age, sex-matched without thromboembolic event. Patients with autoimmune disorders, antiphospholipid antibody syndrome, thrombophilias, anticoagulation therapy, prior thromboembolism, chronic kidney disease 3b, 4, end-stage renal disease, and malignancy were excluded. Literature shows that 50% of COVID-19 patients have at least one APLa and those with elevated partial thromboplastin time have a 90% prevalence of APLa. Based on this, assuming a difference of 40%, with alpha=0.05 and beta=0.20, we can detect a statistically significant difference with 20 patients in each group, using chi-square test. Anti-cardiolipin IgG/IgM, beta-2 glycoprotein-1 IgG/IgM, lupus anticoagulant, and vitamin D levels were measured in both groups.

Results: Cases were 5.7 times more likely to be vitamin D deficient (OR:5.7, 95% CI:1.3-25.6) and 9 times more likely to have at least one APLa (OR:9, 95% CI: 1.6-49.5). Patients with both APLa and vitamin D deficiency had significantly more clots compared to patients who were antibody positive without vitamin D deficiency (100% vs 47.4%; p=0.01).

Conclusions: Thrombosis in COVID-19 was associated with concomitant APLa and vitamin D deficiency. Future studies in COVID-19 should assess the role of vitamin D in reducing thrombosis.
Ciprofloxacin Induced Tumor Lysis Syndrome Associated with Serous Adenocarcinoma of the Female Genital Tract

Introduction:
Fluoroquinolone antibiotics have cytotoxicity effects given their ability to target eukaryotic topoisomerases and in elevation of p53 expression. We present here a case of tumor lysis syndrome in association with ciprofloxacin.

Case Presentation:
An 81-year-old woman with hypertension and hypothyroidism presented with generalized weakness, diarrhea and blisters in bilateral lower extremities for 5 days. She had been seen 9 days earlier for urinary tract infection and was prescribed Ciprofloxacin. She began to feel weak, started to have watery diarrhea and abdominal bloating, and blisters started to develop in her lower extremities. Physical examination: hemodynamically stable, moderate ascites and scattered blisters in bilateral lower extremities. Laboratory evaluation: Creatinine 4.49 mg/dL, Uric acid 16.7 mg/Dl, Calcium 8 mg/dL, Phosphorus 6.8 mg/dL and Potassium 5.8 mg/dL, consistent with Cairo-Bishop Criteria for tumor lysis syndrome. Computerized Tomography of abdomen revealed extensive peritoneal soft tissue nodularity, innumerable pulmonary nodules, and a lobulated soft tissue mass in posterior uterus. Paracentesis revealed metastatic serous carcinoma/adenocarcinoma. Immuno-histochemical staining revealed: CK7: strong positive, CK20: focal positive, Estrogen-negative, WT1: focal positive, p16: strong positive, p53: strong positive, PAX8: strong positive, consistent with serous carcinoma of either peritoneal, fallopian tube, or ovarian origin.

Discussion:
Tumor Lysis syndrome usually occurs after cytotoxic therapy which results in rapid cell destruction and release of intracellular ions, proteins, nucleic acids, and metabolites. Tumor lysis, especially spontaneous tumor lysis in solid cancers is rare, and some cases are reported due to initiation of steroids, chemotherapy and radiation. Fluoroquinolone antibiotics induce cytotoxicity by inhibiting eukaryotic topoisomerase Ila and elevation of p53 expression leading to induction of cellular apoptosis by Bax/Bcl-2 pathway. Ciprofloxacin has a potent anti-proliferative effect by activating caspases and causing mitochondrial depolarization. Fluoroquinolones have been found to augment the apoptotic effects of cancer drugs such as cisplatin.
The timeline of the presentation with tumor lysis syndrome after initiation of ciprofloxacin with no other medication changes indicated that ciprofloxacin is the most likely trigger of tumor lysis. There is demand for additional studies on anti-cancer effects of ciprofloxacin. Furthermore, general practitioners should be cautious when prescribing fluoroquinolones to patients with large tumor burden.
Severe Euglycemic Ketoacidosis in a Pregnant Woman with COVID-19

Introduction:
COVID-19 has been shown to be associated with ketosis or ketoacidosis in 6.4% of patients of which only 35% are diabetic. Pregnancy also predisposes to ketosis since it is a diabetogenic state. We present a case of severe euglycemic ketoacidosis in a pregnant woman with COVID-19 infection.

Case presentation:
A 27-year-old woman with a history of asthma presented at 36 weeks of gestation with shortness of breath at rest and pleuritic, mid-sternal chest tightness. She was COVID-19 positive. On examination: Afebrile, tachycardic (heart rate 130/min), tachypneic (respiratory rate 28/min), blood pressure: 113/81 mmHg, and saturating at 100% on 2 liters oxygen via nasal cannula. She was in respiratory distress with a gravid uterus. Laboratory studies were remarkable for bicarbonate 9 mEq/L, and blood glucose 107 mg/dL. Arterial blood gas: pH: 7.33, PCO2: 12, PO2: 157, lactate: 0.8 mg/dL consistent with anion gap and non-anion gap metabolic acidosis with respiratory alkalosis. Computerized Tomography of chest ruled out pulmonary embolism. Urinalysis showed 2+ ketones. Beta-hydroxybutyrate was elevated at 3.41 mg/dL. Serum osmolality and salicylate levels were normal. While the patient was being evaluated and managed, fetal heart rate showed decelerations, and an emergent cesarean section was performed. The patient immediately improved post-delivery, and bicarbonate improved to 20 mEq/L.

Discussion:
A few case reports have shown an association between pregnancy and COVID-19 infection resulting in euglycemic ketoacidosis. Pregnancy causes increased insulin resistance, enhanced lipolysis, and increased ketogenesis. Stress factors such as COVID-19 infection or starvation may predispose the pregnant woman to severe metabolic acidosis due to ketosis with normal blood glucose. The fetal mortality rate in patients with ketoacidosis can be as high as 70-90%. The mainstay of treatment includes fluid resuscitation, correction of ketosis with intravenous insulin, and expedited delivery.

Conclusion:
Pregnancy when associated with stress factors such as COVID-19 infection or starvation is a high-risk condition for euglycemic ketoacidosis, even in non-diabetic women. Close monitoring of mother and baby and treatment with fluids, intravenous insulin, and expedited delivery is important to reduce maternal and fetal compromise.
83-year-old Female with Fatal Pulmonary-Renal Syndrome Due to Microscopic Polyangiitis

Introduction
Microscopic polyangiitis (MPA) is one of the three ANCA-associated vasculitides. It is a multi-organ, necrotizing vasculitis predominantly affecting small-sized arteries. Rapidly progressive glomerulonephritis (RPGN) is a common renal manifestation of MPA, and diffuse alveolar hemorrhage is seen up to one-third of patients. Concurrence of these two is termed as pulmonary-renal syndrome which can be treated with immuno-suppressants. We present an 83-year-old female with pulmonary-renal syndrome due to MPA leading to death within one month of symptom onset.

Case Description
An 83-year-old, functionally independent white female with history of CKD stage III, hypothyroidism, and obesity presented with 2 weeks of worsening exertional dyspnea. Examination revealed tachypnea and bilateral crackles. Labs were significant for BUN of 31.2, creatinine of 2.20, and WBC count of 16.3. Chest X ray showed bilateral interstitial alveolar opacities. Patient was started on treatment for suspected CHF and pneumonia. However, her oxygen requirements continued to increase and CXR showed worsening infiltrates. Patient underwent bronchoscopy with BAL, which showed gross alveolar hemorrhage. Serological studies showed positive ANA, p-ANCA, anti-MPO and anti-PR 3 antibody. Her kidney function continued to worsen, and she developed severe oliguria. CT guided renal biopsy revealed focal, necrotizing, crescenteric glomerulonephritis, pauci-immune type. She received cyclophosphamide and high dose steroids. However, she was unable to tolerate hemodialysis due to cardiopulmonary instability. She remained on HHFNC for 2 weeks without any respiratory improvement. Given poor prognosis due to rapid renal and pulmonary decline, patient was made comfort care.

Discussion
MPA is rare, with prevalence estimated at 1 to 3 cases per 100,000 people in the US. The average age of onset is 50 years. Given that our patient was an 83-year-old female, MPA was not initially considered. Before patients develop RPGN, there is usually a prodromal phase which can last up to months. This patient was functionally independent at baseline, living by herself. Our case highlights that physicians should consider autoimmune vasculitis with renal and pulmonary failure, even in atypical age groups. It also highlights that atypical patients can present with rapid decline. Early recognition of signs and symptoms is important to start treatment as disease process is often fatal.
A Rare Case of Mediastinal Seminoma Superimposed by Stenotrophomonas Maltophilia Infection

Introduction:
Extragonadal germ cell tumors (GCT) account for 1-3% of all GCT and typically affect males between the ages of 20 and 35. Mediastinal seminomas (MS), an exceedingly rare GCT, usually occur in the anterior and superior mediastinum. Poor prognostic factors include age (≤ 33 years), metastasis with the number of sites, tumor size, and biochemical markers (AFP, β-hCG, LDH). Higher levels of AFP and β-hCG at the time of diagnosis are associated with poorer outcomes.

Case Description:
Patient is a 36-year-old previously healthy male who presented to the ED with acute onset chest pain, upper respiratory symptoms, and a 4-month history of 20 pounds unintentional weight loss. Chest CT angiogram showed a large anterior mediastinal mass measuring 11.6 x 0.4 x 8.1 cm. Laboratory work included high β-hCG-102, high LDH-332, and low AFP 6.5. CT guided mediastinal mass biopsy was suggestive of MS, with positive immunostaining for OCT3/4 and CD117. Due to concerns for post-obstruction pneumonia, the patient underwent bronchoscopy. Cultures from his bronchoscopy were positive for Stenotrophomonas Maltophilia (SM), which was treated with a 7-day course of Trimethoprim/Sulfamethoxazole and Metronidazole. Patient also underwent CT chest, abdomen, and pelvis which showed no evidence of metastatic disease, and scrotal ultrasound which showed no intratesticular masses. Patient was discharged and followed up with Oncology outpatient. He was started on Bleomycin, Etoposide and Platinum (BEP) chemotherapy with plans to follow up with CT chest, abdomen, and pelvis after 4 cycles of chemotherapy.

Discussion:
GCTs have the potential to become exceptionally large with obstruction of surrounding organs, resulting in cardiac and respiratory problems. Mediastinal seminomas are extremely sensitive to chemotherapy and radiation and are the first line for treatment therapy. Infection is a common complication of these tumors due to obstruction resulting in growth of SM species. SM is a multidrug-resistant gram-negative bacillus that is an opportunistic pathogen associated with high morbidity and mortality. With our case we intend to spread awareness regarding this rare but highly resistant pathogen which can be seen in MS patients with post-obstructive pneumonia.
Hypereosinophilic Syndrome (HES) is a heterogenous disorder with varying presentations. One such phenotype includes eosinophilic myocarditis which is characterised by eosinophilic infiltration of the myocardium, causing cell membrane damage, thrombosis and fibrosis. While most occurrences of eosinophilic myocarditis are idiopathic, rare cases have been associated with myeloid neoplasms. We discuss the case of a 34-year-old male with a 1-week history of an URI being treated with amoxicillin and steroids in the outpatient setting; who presented to our hospital with a truncal rash, cough, shortness of breath and bilateral lower extremity swelling. Labs revealed a total WBC count of 47.4, absolute eosinophil count 29.9 and BNP 927. CTA chest revealed large bilateral pleural effusions with pulmonary edema. TEE showed an ejection fraction of 44% and severe mitral regurgitation. The patient underwent bone marrow biopsy which showed evidence of some dysplastic eosinophils but no evidence of acute eosinophilic leukaemia, lymphoma or mast cell disorder. Cytogenetic revealed dysplasia of eosinophils with FISH analysis positive for FIPL1/PDGFR rearrangement. With this finding, the diagnosis of HES and eosinophilic myocarditis secondary to a myeloid neoplasm was made. He was initially treated with steroids which led to a rapid reduction in the absolute eosinophil count. Imanitib along with angiotensin receptor blockade/neprilysin inhibitor, beta blocker, mineralocorticoid receptor antagonist and loop diuretics were started leading to significant improvement in symptoms. While cardiac involvement in HES is rare, when present in carries a poor prognosis. Two subtypes of HES must be recognized; a lymphocytic variant and a myeloproliferative one. The majority of cases are of the myeloproliferative type involving rearrangement of the PDGFR gene, as was the case in our patient. This gene is responsible for the productive of an active protein-tyrosine kinase capable of transforming hematopoietic cells into eosinophil precursors which explains why our patient responded so well to Imanitib, a tyrosine kinase inhibitor. This case highlights the unique presentation of a myeloproliferative disorder leading to eosinophilic myocarditis along with the importance of early recognition and prompt treatment to prevent worsening clinical status.
Renal Infarctions as a Rare Manifestation of Amyloidosis

Introduction:
Systemic Amyloidosis is a group of clonal plasma cell disorders defined by tissue deposition of protein fibrils. The most common subtype is light-chain amyloidosis where kappa or lambda light-chains are misfolded into insoluble B-pleated sheets and accumulate throughout the body. Clinical features vary and largely depend on organs involved. Affected individuals can develop kidney injury usually due to acute tubular necrosis, autolysis, or cast nephropathy. However, renal infarcts are rare.

Case Description:
A 56-year-old male with a history of hypertension, hyperlipidemia, and alcohol use disorder presented with gross hematuria. Patient endorsed proximal lower extremity weakness bilaterally. He was tachycardic on examination. Lab work showed hyponatremic 117 and CK of 667. Urine chemistry showed proteinuria but no RBCs. CT Chest/Abdomen/Pelvis showed pleural effusions and multiple nodules in the lungs, liver, and kidney. Thoracentesis and fluid analysis showed transudate without malignant cells. CTA revealed bilateral heterogeneous renal lesions which resembled infarcts. Muscle biopsy was taken, given patient’s persistent muscle weakness and elevated CK. MRI spine showed mild lumbar degenerative changes. Kidney biopsy was not taken under the assumption that infarcts were due to atherosclerosis. Etiology of other symptoms were not determined, and he was discharged after 10 days. Muscle biopsy confirmed amyloid myopathy and denervation atrophy. Patient was hospitalized 1 month later for shortness of breath. CTA chest showed large bilateral effusions. CT Abdomen showed worsening renal infarcts. Multiple myeloma and MGUS were ruled out. Per Nephrology, bilateral renal infarcts were likely related to amyloidosis. Bone marrow biopsy confirmed loss of chromosome 13, associated with plasma cell myeloma. Patient eventually elected for hospice care due to poor prognosis.

Discussion:
Majority of amyloidosis cases present with a plethora of nonspecific symptoms including lower extremity swelling and shortness of breath, mainly from heart failure or proteinuria. Our case is unique since the patient’s chief complaints were muscle weakness and gross hematuria due to amyloid myopathy. He also had renal infarction diagnosed on CTA which is a very rare complication of amyloidosis. With our case, we intend to spread awareness about these not so common complications of amyloidosis.
Haemoptysis: A Potentially Fatal Complication of Chronic Pulmonary Aspergillosis in Patients with Sarcoidosis

Pulmonary aspergillosis is a known complication of pulmonary sarcoidosis and an estimated 24-37% of chronic sarcoidosis cases are complicated by aspergillosis in the Americas and Africa. Chronic pulmonary aspergillosis (CPA) is mostly seen in advanced stages of sarcoidosis and is associated with higher mortality. However, this mortality is likely due to advanced sarcoidosis rather than the infection. Hemoptysis is a common presenting symptom of patients with sarcoidosis and CPA, and at times it has proven to be a fatal complication. Here we report a case of pulmonary sarcoidosis with aspergillosis presenting with hemoptysis.

Case Description
A 49-year-old male with history of pulmonary sarcoidosis and pulmonary aspergillosis presented with hemoptysis. The patient had a diagnosis of pulmonary sarcoidosis for about 14 years, and pulmonary aspergillosis for five years. He had previously been treated with multiple courses of fluconazole; last treatment was completed four months prior to his current admission. After his last fluconazole course, it was decided to avoid further courses of fluconazole as the aspergillus was well contained and medical therapy would not infiltrate the aspergilloma. Over the past five years the aspergillus remained stable on CT. A bronchoscopy performed and localized the bleeding to the LUL. Biopsies and washings were obtained which demonstrated chronic inflammatory cells. Patient had continued hemoptysis. CT chest was performed showing sequelae of aspergillus disease and left a bronchial arteriogram and embolization of left bronchial artery were performed. Patient had continued hemoptysis requiring intubation and ICU admission. Subsequently, the patient was then transferred to a tertiary medical center for further care.

Discussion
Even though there are only a few clinical guidelines for the treatment of CPA, it is known that long-term treatment is needed for prevention of disease progression and complications. Long term anti-fungal medication is considered to prevent hemoptysis. Relapses are common after completion of treatment and surgical intervention is sometimes required. As with our patient who previously completed multiple courses of anti-fungal therapy the bleed was a relapse of aspergillosis. With our case, we wanted to spread awareness about this not so uncommon but potentially fatal complication of CPA.
Resident Poster #139
Program: St Mary Mercy Hospital
Program Director: David Steinberger, MD, FACP
Presenter: Sohaib Syed
Additional Authors: Victoria Reick-Mitrisin, Rahul Pansare, Amreet Sidhu, David Steinberger, Preeti Misra

Resident Coach: Improving Patient Satisfaction Scores on Medical Teaching Services

Patient's perception of healthcare received at hospital is reported by the Hospital Consumer Assessment of Healthcare Providers and Systems (HCAHPS) which is a national, standardized, publicly reported survey. Improving HCAHPS increases both patient safety and satisfaction. Public reporting was implemented in 2006 and did not show an increase in physician communication scores. The goal of this study was to determine how we can improve patient satisfaction scores on Medical Teaching Services (MTS).

Physician education and ongoing feedback have been successfully implemented to improve the Patient Satisfaction Scores (PSS). Banka et al showed increase of 8.1% in HCAHPS through physician education versus 1.5% in controls. We looked at ACGME competency “Interpersonal and Communication skills” and its role in improving patient satisfaction. Our objective was to evaluate implementation of resident coaching as an intervention to improve PSS on inpatient MTS in a community level academic-hospital. We compared HCAHPS score on Doctor Communication between MTS teams receiving Educational Intervention and without any intervention, both at baseline and after interventions, and assessed its impact on LOS, total cost of inpatient stay, ED re-visit within 7 days and readmission at 30 days.

2 MTS Teams C and D underwent Educational Intervention for Improving Physician Patient Communication. Weekly sessions between Coaches and the Teams were scheduled at each block and education was provided to improve communication. Issues were addressed by the attending physician as they occurred, and verbal feedback was obtained. The target was an achievable rise of 5% (+/-2%) in HCAHPS on the teams being coached. Datasets were acquired as part of Quality metrics from Michigan Data Analytics for both before and after implementation of the resident coach as well as post implementation period for all teams.

Outcomes of the statistical analysis showed there was only one statistically significant change in patient satisfaction scores with respect to how likely the patient was to recommend their physician. This showed that patients were 4.12 times more likely to give favorable recommendations if the physician had gone through the resident coaching program. Additionally, 7-day readmission rate significantly decreased (1.68% vs 5.85%, p=0.049). All other results showed no statistically significant difference.
The Dark Side of The Moon: A Case of Delayed Diagnosis of Infective Endocarditis in the COVID-19 Era

Clinical vigilance
A 52-year-old patient presented with shortness of breath who had a history of hypertension, dyslipidemia, and OSA on CPAP. Three months ago, he had episodes of fever and chills in addition to generally not feeling well followed by dyspnea initially with exertion, worsened later and became present at rest. He had associated orthopnea but no PND. He denied any chest pain; however he has been experiencing intermittent swelling on bilateral lower extremities. The patient was tested for COVID-19 five times, and each time it was reported to be negative.

On presentation, he was hemodynamically stable and saturating well on room air. An electrocardiogram showed sinus tachycardia, ST wave depression in II/III/V3-V6. Chest x-ray showed Bibasilar atelectasis, moderate right pleural effusion; however, no consolidation was noted. CT angiogram showed no pulmonary artery filling defect; however, it showed enlarged perihilar and subcarinal lymph nodes. It also showed moderate right and small left pleural effusion. Lab work showed elevated BNP at 2076, High sensitivity troponin was elevated at 80. CBC with differential showed WBC count within normal range. The COVID-19 PCR result was negative for the sixth time by this time. TTE demonstrated severe LV and RV dysfunction consistent with wide-open aortic valve insufficiency. A TEE demonstrated severe aortic regurgitation with thickened aortic cusps with mal-coaptation and pedunculated masses.

The patient was diagnosed with aortic valve endocarditis. Further questioning showed that he underwent a dental procedure prior to the onset of symptoms. Patient was started on antibiotics therapy. He later underwent surgical aortic valve replacement. The patient tolerated the procedure well and was discharged in good condition to complete six weeks of antibiotic therapy.

Discussion
In the case of this patient, before the COVID-19 era, he most likely would have been referred for a further evaluation immediately. The patient had a prolonged course of fever, chills, and generalized weakness in addition to heart failure symptoms; therefore, further investigations were warranted. The vigilance and precautions needed for patients suspected of having COVID-19 in no way should be relaxed, but fear should not preclude us from delivering appropriate care.
Acute Left Sided Visual Loss: Determining the Culprit Between Large Vessel Carotid Stenosis or LV Thrombus

Introduction: We present an interesting case of acute left eye blindness in a veteran patient with history of laryngeal cancer s/p chemoradiation and in the setting of a LV thrombus on anticoagulation resulting in a diagnostic challenge in determining the etiology.

Case Description/Methods: Our patient is a 61-year-old male with a history of heart failure with reduced ejection fraction, left ventricle thrombus, and h/o laryngeal adenocarcinoma status post chemoradiation who presented with sudden left sided visual loss. Home medications included apixaban, aspirin, and statin. Left eye was nonreactive to light with absent consensual pupillary response. Rest of the neurological exam was unremarkable. Head CT scan showed no acute intracranial abnormality with remote chronic ischemic changes. Neck and Head CTA demonstrated complete occlusion of the left vertebral artery from its origin to the cervical segment and complete occlusion of the internal carotid artery (ICA) at its origin with reconstitution at the petrous and cavernous segments. Brain MRI showed no acute infarct with presence of small old infarcts in the left occipital and frontal lobes. Fundoscopic exam demonstrated optic nerve edema and evidence of suspected ophthalmic artery occlusion. 2D echo demonstrated massive LV thrombus, unchanged from two months ago. The challenge was determining whether this presentation of ophthalmic artery occlusion was cardioembolic related to the LV thrombus and hence a failure of anticoagulation or related to occlusive carotid disease. During hospital course, patient developed transient sudden right sided weakness. CT perfusion demonstrated extensive ischemic penumbra in the left cerebral hemisphere consistent with acute decompensated chronic occlusion of the left ICA.

Discussion: In view of this development of contralateral hemispheric symptoms, it was retrospectively determined that the acute visual loss was related to carotid artery disease resulting as a late complication of localized radiation for laryngeal cancer. The patient was transferred to a tertiary facility for endovascular intervention. Hence, acute visual loss accompanied by contralateral deficits is obstructive carotid stenosis until proven otherwise. This case emphasizes the need for thorough patient centered evaluation on an annual basis thus providing an opportunity for early non-invasive or minimally invasive intervention.
Mimicking McConnell’s: Right Heart Strain in Acute Chest Syndrome

McConnell’s sign is an echocardiographic finding of right-ventricular free-wall hypokinesis with a hypercontractile apex. Though considered pathognomonic for pulmonary embolism (PE), we present a rare case of McConnell’s sign in a patient with acute chest syndrome. A 29-year-old woman with sickle cell disease was admitted 2 days post-partum for fever, tachycardia and hypoxia, saturating at 90% on 4 liters of oxygen. Chest X-ray showed a retrocardiac opacity. Transthoracic echocardiography (TTE) revealed McConnell’s sign. Her D-dimer 10.17 mg/L. Given her recent pregnancy and sickle cell disease, the main differential diagnoses included PE and acute chest syndrome. McConnell’s sign suggested a high likelihood of PE, however, a computed tomography for PE did not exhibit filling defects. Exchange transfusion along with antibiotics were initiated promptly. Subsequent TTE four days later revealed complete resolution of right ventricular dysfunction. McConnell’s sign can be present in other disease processes presenting with hypoxia. With increasing use of bedside ultrasound to aid in rapid decision making in critically ill patients, it is necessary to recognize a broader differential for this finding on TTE. A definitive diagnosis of PE should not be made based exclusively on the presence of McConnell’s sign.
Lemierre’s Syndrome in an Immunocompromised Individual

Lemierre’s Syndrome (LS) occurs when an infection from the oropharynx spreads into the neck causing septic thrombophlebitis of the internal-jugular-vein. Given its low incidence, there are a lack of controlled trials to guide therapy. LS typically affects young patients and can cause unilateral tenderness and swelling of the neck with concurrent fever and dysphagia. LS can develop one to three weeks after onset of pharyngitis. The most common inciting pathogen is Fusoform necrophorum. We present a rare case of LS occurring in an immunocompromised patient. A 41-year-old male with history of HIV presented with a two-day history of right neck and facial swelling associated with dysphagia. He endorsed a history of pharyngitis few weeks prior to admission. Physical exam revealed edema at the inferior angle of the mandible spreading inferiorly to the submandibular region. Maxillofacial CT scan showed a “filling defect within the right internal-jugular-vein with significant wall thickening and enhancement that was displacing the esophagus and trachea”, consistent with right internal-jugular-vein thrombophlebitis. Nasolaryngoscopy was performed and revealed a patent airway and no mass effect. It was determined that there was no drainable abscess. CT Thorax and TTE were negative for septic emboli. Anticoagulation and broad-spectrum antibiotics were started. The final diagnosis was LS in setting of recent pharyngitis. The patient was discharged on levofloxacin and metronidazole for four weeks and Apixaban for three months. This case illustrates a rare complication of pharyngitis, LS, and the importance of early diagnosis to avoid life-threatening outcomes. Our patient developed LS despite completing an empiric course of amoxicillin-clavulanic acid for preceding pharyngitis. Additionally, our patient was in an immunocompromised state in setting of his HIV. Special attention should be given to such patients with early testing and tailored treatment for tonsillopharyngitis causative organisms. Mainstay of treatment is a 6-week course of a beta-lactamase resistant beta-lactam antibiotic. Alternative therapies include metronidazole or clindamycin which should be considered in patients with beta-lactam treatment failure. There are no current guidelines on anticoagulation in the setting of septic emboli given insufficient evidence of reducing rate of embolus. Further studies on the benefits of anticoagulation are warranted.
Ischemic Monolemic Neuropathy Complicated by Carpal Tunnel Syndrome

Ischemic Monolemic Neuropathy (IMN) is a distinct clinical entity in which upper extremity neurologic symptoms present immediately following placement of a brachial artery arteriovenous (AV) access. The mechanism is thought to be secondary to transient reduction in blood flow to distal arm peripheral nerves. Neuropathy predominates over significant clinical ischemia.

A 33-year-old woman with end stage renal disease on hemodialysis since 2015 with left brachial AV graft for access presented for right brachial artery AV graft insertion. Immediately, following the procedure, she complained of intractable right upper extremity pain. Moreover, she complained of paresthesia in her first 3 fingers corresponding to median nerve distribution. She had motor block of those fingers. She could move her 4th and 5th digits. There was concern for dialysis access steal syndrome, compression neuropathy secondary to post-surgical swelling, wound hematoma, and iatrogenic injury from AV graft insertion. She was given IV narcotics and vascular surgery was consulted with doppler revealing palpable pulses in radial and ulnar arteries. Her symptoms worsened the next morning with paresthesia in all 5 fingers, radial pulse weakly palpable, and a cold right upper extremity. Vascular surgery was contacted. We advocated for our patient that imaging would delay surgery and cause further vascular compromise and neuropathy. The patient was taken for surgery with ligation of right AV graft with improvement but not complete resolution of her pain and neuropathy. She continued to have pain and right AV graft was removed 3 days after it had been created. Following AV graft closure, the patient along with daily physical therapy had significant recovery in her hand function. Neurology was consulted and electromyography was done which revealed swelling in median nerve not compressing the nerve but showed axonal loss in radial and ulnar distribution. It also showed carpal tunnel syndrome, likely chronic. Physiological study showed patent right brachial artery. IMN has an incidence of 0.3% to-3% and is often missed due to its non-specific symptoms. Any patient who complains of hand pain immediately following AV graft creation should be evaluated for IMN due to the potential for permanent neurologic loss if not recognized early.
Mutation Concordance Between Somatic Tumor DNA and Blood-Derived Circulating Tumor DNA in Pancreatic Cancer

Background: Treatment outcomes for pancreatic cancer are dismal often due to late presentation of disease. Pancreatic cancer diagnosis is made with endoscopic imaging and biopsy, and the latter can guide therapy when actionable tumor mutations are identified. However, anatomic location may limit the amount of tissue sampled, hindering somatic profiling. Circulating tumor DNA (ctDNA) derived from venous blood may address this obstacle as these mutations may reflect innate tumor biology and are much easier to obtain. Few studies have examined how similar mutations identified in ctDNA are to those identified in somatic tumor DNA, and some studies have been limited to advanced cancers.

Methods: Nineteen patients diagnosed with pancreatic cancer at Karmanos Cancer Institute with paired somatic tumor DNA and blood-derived ctDNA were identified for this study. Somatic tumor DNA and ctDNA were analyzed with clinical-grade next-generation sequencing (NGS), CARIS and Guardant, respectively. Number of alterations as well as concordance between identified somatic tumor DNA and ctDNA mutations were assessed.

Results: In our cohort, 8 (42%) patients had limited somatic tissue that hindered NGS analysis. Somatic tumor mutations of the KRAS gene were found in 14 (74%) and TP53 gene were found in 10 (53%) patients. The G12D mutation of the KRAS gene was the most frequently identified mutation, and this mutation showed high concordance between tumor DNA and ctDNA (80%). Mutations of other genes including SMAD3, ATM, CDKN2A, EGFR, ARID1A, BRAF, BRCA1, CDK4, JAK2, MYC, and NF1 were detected at lower frequency (5-16%).

Conclusions: Our study affirmed the potential of ctDNA as important measurable markers for pancreatic cancer, as ctDNA mutations showed high concordance with somatic tissue DNA mutations. The difficulty of obtaining sufficient tissue samples from tumors was evident in a large proportion of patients. Surgical tissue samples can be difficult to obtain due to anatomic location and/or surgical risks. Blood-derived ctDNA may help overcome these difficulties as it is easier to collect, associated with less risk, and can be collected multiple times for disease treatment and monitoring.
Corynebacterium spp Infection of Native Joints in an Immunocompromised Host

Mr. L is a 46-year-old man with history of hypertension, T2DM, perforated diverticulitis s/p left colon resection, rheumatoid arthritis, chronic DVT/PE, Charcot joint presented with AMS and increased swelling of right wrist and ankle. On exam, patient was febrile to 39.4, HR 158, hypoxic to 85% (quickly placed on nonrebreather), and frankly altered. Physical exam showed tender, erythematous right wrist and ankle with no overlying skin break. Lab work showed WBC 17 with neutrophilic predominance and prerenal AKI. MRI showed fluid collection in right ankle along with tenosynovitis, myositis, and cellulitis with underlying osteomyelitis not excluded. Patient was started on broad spectrum antibiotics and admitted to sepsis secondary to SSTI.

IR performed sterile aspiration of wrist abscess, podiatry performed OR I/D of right ankle joint; Intra-op cultures showed Corynybacterium afermentans from the ankle and Corynybacterium striatum in the wrist. The patient’s regimen was narrowed to linezolid due to the patient’s vancomycin allergy and the general susceptibility of Corynybacterium striatum to daptomycin, linezolid, and vancomycin. Review of patient’s previous admissions showed prior ulnar fracture, ligamentous disruption, and osteopenia as well as an admission earlier in the year for Corybacterium infection of the right ankle, sequelae of cellulitis. Patient was stabilized and discharged with Linezolid and close podiatry follow up.

Corynybacterium spp are gram positive bacilli that live as mucocutaneous commensals. Previously thought to be low virulence, but it is increasingly recognized as a pathogenic when isolated from intra-operative samples. Diabetes is a risk factor for pathogenicity. Joint infections from Corynybacterium tend to be linked to instrumentation and prosthetic implants, due to propensity for biofilm production with native joint infections a rarity. The poor glycemic control due to chronic steroid use and immunosuppressive medications caused a higher risk for pathogenic Corynybacterium infection in this patient overall. However, even though the affected areas were native joints, the patient’s neuropathic arthropathy and trauma predisposed those specific areas to be an infective nidus for a biofilm-forming agent like Corynybacterium, even without known direct inoculation.
Hypercalcemia Due to CLL

Introduction:
CLL is the most common chronic Leukemia in US adults. It is characterized by lymphocytosis, typically monoclonal in nature. Hypercalcemia has been described as a rare complication of CLL, typically in advanced disease states. We describe a patient presenting with a calcium level of 15.2 in the setting of CLL.

Case presentation:
69 year old female with PMH CLL presented complaining of Right Hip Pain. Imaging in the ED revealed fracture deformity of the right superior ischiopubic ramus described as subacute in nature, questionable for pathologic fracture. Routine labs showed a calcium level of 15.2, with ionized calcium at 12.8. PTH was within normal limits, vitamin D level was low. Hypercalcemia was attributed to active leukemia, as patient had WBC of 428,000 cells per microliter with lymphocytic predominance, in patient with previously diagnosed CLL.

Patient was given 2L Normal Saline boluses and started on aggressive fluid hydration 150 mL/hr, as well as diuresis with a one time dose of IV Lasix 20 mg to induce calciuresis. Patient was also given calcitonin and pamidronate, which was discontinued on second day of admission as calcium level had improved to 11.1. Calcium level continued to trend down to within normal limits, and patient was discharged to home after induction chemotherapy and cytoreduction.

Conclusion
Hypercalcemia is relatively common in cancer patients, however it is a less common finding in CLL. The mechanism of hypercalcemia in hematologic malignancies remains unclear. Further research is warranted to find the precise pathogenesis of hypercalcemia in CLL.
Dealing with Double Trouble! Emergent Cardiac Catheterization Complicated by Peri-Procedural Ischemic Stroke

Peri-procedural stroke is a rare but catastrophic complication of cardiac catheterization or percutaneous coronary intervention (PCI). If not recognized immediately it can lead to significant mortality and morbidity. Here we report an emergent cardiac catheterization complicated by an ischemic stroke.

A 72-year-old woman with a past medical history of hypertension presented with chest pain. Her EKG showed ST elevation in aVR and ST depression in the precordial leads. Emergent cardiac catheterization revealed almost complete occlusion of the LAD with a thrombus. Post thrombo-suction the patient developed facial droop, slurred speech, and left-sided weakness; initial NIHSS was 11 per stroke team evaluation. PCI was done with successful balloon angioplasty, stent was not placed as the use of dual antiplatelet therapy (DAPT) would worsen the possible hemorrhagic stroke. Due to hemodynamic instability during the procedure, an Intra-aortic balloon pump (IABP) was placed. Post-procedure, a stat CT head showed no acute intracranial process. CTA head showed stenosis of right M1. CT perfusion showed watershed ischemia in the right MCA region with no core infarct, suggesting an embolic source that confirmed a peri-procedural stroke. tPA was contraindicated as she required a heparin drip with the IABP. She received IV fluids to support intracranial pressure. Repeat CTs showed no hemorrhagic transformation. IABP was removed and heparin was discontinued after 48 hours. On discharge to rehab, her NIHSS was 2 with minimal left-sided weakness. She returned a month later for stent placement to LAD and was discharged on DAPT.

Managing peri-procedural stroke remains controversial; the approach is case and provider dependent. It is important for cardiologists to diagnose and act rapidly to provide the best management. A protocol should be planned beforehand at any institution with cardiac catheterization services. A stroke team, neurologist, and neuro-interventionalist should be involved early and work as a team for best outcomes. Further randomized controlled trials are needed for the safest and most efficacious treatment guidelines in cases of peri-procedural strokes.
Unbelievably High Hba1c in Asymptomatic Necrotizing Fasciitis

Introduction: Diabetes Mellitus is the most common co-morbidity associated with necrotizing fasciitis (NF). NF, especially early in the disease course, can mimic less severe skin and soft tissue infections. Patients prone to atypical presentations, such as the diabetic population, are more vulnerable to misdiagnosis of NF. If not recognized early and treated effectively, NF is associated with high mortality and morbidity. We report a patient with poorly controlled diabetes who was found to have asymptomatic NF.

Case: A 53-year-old male with insulin-dependent type II diabetes mellitus complicated by peripheral neuropathy and peripheral arterial disease presented with altered mentation. Per the family, he experienced fatigue and fecal incontinence for a few weeks and was noncompliant with insulin. On evaluation, he was hemodynamically stable and afebrile. The physical exam was unremarkable. Labs suggested diabetic ketoacidosis (DKA) and acute kidney injury. Hba1c was 20.3. WBC and lactate were 17 and 3, respectively. Urinalysis showed glucosuria, proteinuria, and ketonuria. CT head and CXR were unremarkable. DKA protocol was initiated. The following day, his mentation improved, and DKA resolved; however, blood cultures grew Enterococcus species. Detailed physical examination was repeated to determine the source of infection, which revealed a painless right-sided perianal abscess with foul purulent discharge. He was taken for incision and drainage. Surgical exploration exposed underlying necrotic tissue consistent with NF. After extensive debridement and appropriate antibiotic therapy, follow-up CT abdomen a week later showed the second site of NF in the left perianal region. Again, he had no pain, had no visible skin finding, was afebrile, and had no leukocytosis. He underwent further debridement, and he was discharged in a stable condition.

Discussion: Carrying a high level of suspicion for underlying NF in diabetic patients with abnormal skin and soft tissue findings is crucial. It is essential to remember that diabetic patients with complications such as peripheral neuropathy and arterial disease can have an indolent presentation of NF. A detailed history and physical examination are vital, especially in patients with altered mentation and diabetic ketoacidosis. Clinicians should have a low threshold for imaging in diabetic patients with skin or soft tissue infections.
A Case of Post-COVID Bell’s Palsy and Migraines

INTRODUCTION:
The COVID-19 pandemic resulted in a major burden on the healthcare system due to multiorgan complications including neurological problems. This may occur due to the virus having affinity to ACE2 receptors which are present throughout the nervous system. There are limited studies in regard to patients developing Bell’s palsy after having the infection. Here we present a patient with recurrence of migraines and development of Bell’s Palsy after being infected with COVID-19.

CASE:
Patient is a 61-year-old male who was fully vaccinated for COVID, presented to the hospital due to new onset drooping of his face. The patient has a history of migraines decades ago that are controlled off medication. He developed upper respiratory tract infection a month before admission, at that time he tested positive for COVID-19. The symptoms resolved within a week without hospitalization. Nonetheless, he soon developed migraines around his right temple radiating down his face. He woke up from a migraine and noticed he had drooping of right eye and mouth, so he came to the hospital. On admission, he was afebrile and hemodynamically stable. Neurological exam revealed incomplete closure of his right eye and right facial droop. He also reported sensitivity to sound. The patient had no other neurological findings and no skin findings. CT head showed no findings. MRI of the brain showed mildly asymmetric increased post-contrast enhancement of the right facial nerve. Neurology recommended to discharge the patient with prednisone 60mg daily for 10 days, topiramate 25mg daily and sumatriptan as needed.

DISCUSSION:
COVID-19 infection presented several challenges with treatment of acutely ill patients. The disease presents various novel complications especially in patients who have recovered from the infection and now enter the “post COVID” territory. Therefore, recognizing and preventing long term complications of COVID-19 infection has become crucial. This case represents a patient with recurrence of migraines and development of Bell’s palsy after the infection. Further studies are needed to understand the pathophysiology of why headaches occur post COVID and prevent neurological sequelae. Preventative strategies are needed to help with reducing migraines and immune complications that can lead to Bell’s palsy.
Acute Pulmonary Embolism in a Patient with Normal D-Dimer

Introduction:
Several studies on COVID-19 have helped us better understand the dynamics of this viral illness. Initially considered to be a respiratory disease, research later showed that it was the initiation of an aggressive systemic inflammatory response including a prothrombotic state. Clinicians have used inflammatory markers such as D-dimer as an indicator for underlying thrombotic state. We present the case of a pulmonary embolism (PE) despite normal D-dimer levels.

Case:
A 73-year-old female with a past medical history of hypertension and recent hospitalization for COVID-19 pneumonia. D-dimer on initial admission was 150, patient was treated for COVID-19 pneumonia and discharged home on 2L of O2 via nasal cannula. She returned to the hospital 1 month later with complaints of palpitations. EKG on admission showed sinus tachycardia, the patient was found saturating at 98% on 2L of oxygen, unchanged from time of discharge, otherwise vitally stable. Patients' wells score was calculated at 1.5 which pointed towards patients being low risk for PE, D-dimer measured at 645, was within normal limits when adjusted for age, indicating a low probability of VTE. Due to recent hospitalization and infection with COVID-19, CT Angiography was obtained and showed PE of the right main pulmonary artery extending into segmental right upper and lower lobe pulmonary arteries with no right ventricular strain. Patient was started on anticoagulation, and she was discharged home in stable condition.

Discussion:
It is now well established that COVID 19 infection causes a hypercoagulable state. Initial recommendations for management of patients with Covid-19 included measurement of serial D-dimers throughout the course of illness. This recommendation has since changed. In our case, despite the rise in inflammatory marker, the age-adjusted value was within normal limits. In addition, Wells Score, which is used to predict DVT and PE, did not serve to be a reliable scoring system. Trending laboratory markers like D-dimers from previous admissions should be used as a valuable tool when post COVID disease is suspected. Any increase in D-dimer even if below the cutoff for age-adjusted D-dimer should be an indicator for further evaluation with imaging to rule out underlying clots.
Introduction:
Subclinical Hypothyroidism is defined as an elevated serum thyroid-stimulating hormone (TSH) level with normal levels of free thyroxine (FT4) affecting up to 10% of the adult population. It is mostly commonly caused by Hashimoto’s thyroiditis. Here we present case of subclinical hypothyroidism in a patient with a history of Grave’s disease.

Case:
37 years old male with past medical history of Grave’s disease in remission. Presented with cold intolerance, constipation, and weight gain. Thyroid function was checked, and it showed an elevated TSH 8.9 with normal free thyroxine (T3 and T4) confirming the diagnosis of subclinical hypothyroidism. On further questioning about his Grave’s disease history, patient mentioned that he was positive for both Thyroid Stimulating Immunoglobulin (TSI) and thyroid anti-peroxidase antibodies (Anti-TPO) and was treated with methimazole for a total of 18 months. Since the patient was symptomatic, he was started on treatment with Synthroid with scheduled follow up with in 5 weeks.

Discussion:
Grave’s disease patients have a variable outcome after resolution of their symptoms. A study observing patients with Grave’s disease over a time of 20 years showed that about 62% of the patients developed recurrent hyperthyroidism, % had subclinical hypothyroidism, and 3% overt hypothyroidism related to TSH receptor blocking antibodies and thyroid peroxidase antibodies. Only 27% of the patients were in remission. Chronic thyroiditis or Hashimoto's disease, which occurs following the Graves' disease episode is likely due to extended immune response in Graves' disease. It includes the immune response to endogenous thyroid antigens, i.e. thyroid peroxidase and thyroglobulin, which may enhance lymphocyte infiltration and finally causes Hashimoto's thyroiditis.

Conclusion:
Pathogenesis for chronic thyroiditis following anti-thyroid drug treatment in patients with Graves' disease remains unclear. As Hashimoto’s thyroiditis is the cause of subclinical hypothyroidism, we believe the mechanism for prolonged immune response in patients with subclinical thyroiditis following Grave’s Disease may share a similar mechanism. Physicians and other healthcare providers should be aware of this complication and may consider continuing to follow thyroid function after patients are initiated on therapy and symptoms have resolved. Increasing awareness about long-term complications of thyroid dysfunction may lead to overall better outcomes for patients.
Inhaled Corticosteroids: An Overlooked Cause of Adrenal Insufficiency

Introduction
Inhaled corticosteroids (ICS) are used as a mode of treatment in patients with severe Chronic Obstructive Pulmonary Disease (COPD) who have repeated or severe exacerbations. Acute adrenal insufficiency (AI) due to ICS is a rare yet serious systemic adverse effect.

Case
A 64-year-old male with COPD, hypertension, arrhythmia-induced cardiomyopathy s/p ablation presented for recurrent presyncopal episodes, weight loss, muscle pain, and fatigue in the past two months. He reported prodromal symptoms of nausea and cold sweats. He denied chest pain or precipitating factors, such as prolonged standing, intense pain, palpitations, exertion, or association with standing up. He also denied decreased oral intake, recent infections, diarrhea, or vomiting. Blood pressure (BP) was 61/51 and pulse 118. EKG showed sinus tachycardia. Labs were significant for Na 126, WBC 16.4, lactate 1. The patient was given a 500 cc fluid bolus with an improvement of BP. A stat echocardiogram was done to rule out pericardial tamponade due to the patient's history of pericardial effusion after ablation. It revealed small pericardial effusion with no tamponade physiology and EF of 55%. Carotid duplex ultrasound was unremarkable. Further investigation of the home medication list revealed that the patient was on two high-dose inhaled corticosteroids for the past two years. The patient denied abrupt change or cessation of his inhalers or recent oral corticosteroids use. As the patient's symptoms were suspicious of adrenal insufficiency, morning cortisol was ordered, which was low. Cortisol levels after cosyntropin administration were suggestive of AI and serum ACTH level was low, confirming secondary AI. With other causes ruled out, this was attributed to long-term ICS use. The patient was treated with hydrocortisone which improved his symptoms on follow-up.

Discussion
ICS at high doses of 1.5mg/d is associated with marked adrenal suppression. Fluticasone demonstrated increased bioactivity compared to beclomethasone, budesonide, triamcinolone. Patients present with symptoms due to glucocorticoid deficiency, including poor weight gain, hypoglycemia, weakness, fatigue, and myalgia. They also present with hyponatremia due to increased vasopressin activity. This case report highlights the need for recognizing adrenal insufficiency and screening patients on higher ICS doses.
Streptococcus Constellatus Induced Necrotizing Pneumonia

Intro:
Streptococcus constellatus is a beta hemolytic cocci part of the natural microbiota of the oral cavity, genitourinary and respiratory tracts. This pathogen is the causative agent of purulent infections and abscess formations, however, few reports link it to necrotizing infections. We describe a case of a 68 year old female who developed ventilator associated pneumonia secondary to S. constellatus after surgical intervention.

Case Report:
A 68 year old female treated in our ICU after undergoing thrombectomy for acute limb ischemia. Attempted ventilator weaning on postoperative day 5 was not tolerated. Chest X-ray demonstrated extensive bilateral airspace disease, consistent with ventilator associated pneumonia. Bronchoscopy demonstrated copious respiratory secretions bilaterally with extensive mucus plugging of the right lower and middle lobes. The left lingula and lower lobe bronchus were found to be occluded with thick secretions. Bronchial washings were sent for microbiological evaluation. Beta hemolytic streptococci were seen on gram staining and empiric therapy with vancomycin and cefepime was initiated. Final cultures grew S. constellatus and antibiotics were changed to IV Unasyn. Consequent ventilator weaning trials failed due to increased work of breathing. CT chest was done and demonstrated findings consistent with necrotizing pneumonia. Due to limited clinical improvement in subsequent days, she was switched to a 7 day course of Zosyn.

Discussion:
S. constellatus is known to cause purulent infections and abscesses, however, necrotizing infections are rare. Studies have shown S. constellatus infections have been more commonly seen in immunocompromised patients and those with aspiration risk factors. Our patient was immunocompetent and developed a rapidly spreading necrotizing pneumonia after being ventilator dependent for 5 days. Tracheal intubation and mechanical ventilation served as her largest risk factors to aspiration of oropharyngeal contents, which was likely to include this pathogen. Patient was eventually transitioned to a tracheostomy site and clinical improvement was noted after 2 weeks of continued antibiotic therapy.

Conclusion:
Further guidelines are needed to determine if prophylactic antibiotic therapy should be administered to immunocompromised patients who undergo endotracheal intubation in order to prevent such life threatening infections. This case also demonstrates that Streptococcus constellatus infections should be considered in patients with significant aspiration risk.
Novel Case of Psychobacter pulmonis Bacteremia in Setting of UTI

Introduction: Psychobacter pulmonis (P. pulmonis) has yet to be reported as a pathogen causing bacteremia. Psychrobacter spp. are gram-negative coccobacilli with cryophilic characteristics belonging to the Moraxellacea family. We report a novel case of P. pulmonis bacteremia suspected to be caused by a urinary tract infection (UTI). Case Description: A 83-year-old male with history of atrial fibrillation and aortic valve replacement presented to our hospital with altered mental status (AMS). He was hemodynamically stable. He responded to painful stimuli and his abdomen was distended and tender. Labs revealed elevated creatinine (2.1 mg/dl) and leukocyte count (17.5x10^3/MCL), and low hemoglobin (9.9 g/dl). Bladder scan showed large volume retention; straight catheterization drained 1800cc of bloody urine with clots. Home medications were unremarkable for iatrogenic causes of AMS, urinary retention, or hematuria. Urinalysis was consistent with UTI. Blood and urine cultures were obtained. Broad-spectrum antibiotics and fluid hydration were started. CT head was done to rule out other causes of altered mentation and found negative for acute intracranial processes. For evaluation of obstructive uropathy, CT abdomen was done revealing bilateral hydronephrosis and distended urinary bladder with a large hematoma. Within 24 hours, the patient’s hemoglobin dropped to 6.8 g/dl requiring blood transfusion. His gross hematuria persisted therefore cystoscopy was done. Diffuse inflammatory changes in the bladder with oozing of blood were found, suggesting hemorrhagic cystitis. Final blood cultures grew pan-sensitive gram-negative rods in both vials. An external laboratory identified the bacteria as P. pulmonis. Urine culture was negative as antibiotics were started before sample collection. Continuous bladder irrigation was initiated. TTE and TEE showed no findings of endocarditis. After completing a course of appropriate antibiotics, the patient’s AMS and gross hematuria resolved. Discussion: Data regarding virulence factors or pathogenicity of P. pulmonis is lacking. Other species in this genus have been reported in cases of bacteremia, possibly associated with environmental exposure as Psychrobacter spp. is ubiquitous in marine environments. Our patient had P. pulmonis bacteremia and the only identifiable source of infection was UTI causing hemorrhagic cystitis. Although rare, more information on the pathogenicity of Psychrobacter spp. would aid clinicians in identifying sources of infection.
An Impelling Case of Cardiogenic Shock

Introduction:
The use of mechanical circulatory support devices has increased in cardiogenic shock and patients undergoing high-risk coronary interventions requiring hemodynamic support. The Impella device is efficient and safe, but its displacement can have catastrophic consequences. Here, we present a case of recurrent ventricular tachycardia caused by improper Impella device positioning, which resulted in mortality.

Case Summary:
A 77-year-old male with Diabetes Mellitus presented to ED for shortness of breath. Physical examination was unremarkable, and he was hemodynamically stable. Labs revealed an elevated troponin that trended up on subsequent draws; a D-dimer was normal. An ECG showed no acute ischemic changes. A Chest X-ray was unremarkable. He was placed on IV Heparin and was treated as NSTEMI. The following day he had worsening shortness of breath and became vitally unstable (hypotensive, tachycardic). Urgent cardiac catheterization showed triple vessel disease. Due to cardiogenic shock, an Impella device was placed. He had a cardiac arrest during the procedure, but return of spontaneous circulation (ROSC) was achieved. Shortly after, his telemetry alarmed for sustained ventricular tachycardia, ACLS protocol successfully stabilized the rhythm. Thirty minutes later, he was still going in and out of ventricular arrhythmias. He was started on an Amiodarone drip and required pressors due to worsening hemodynamic status. Impella device malposition was suspected and confirmed by stat Echocardiogram. Arrhythmias resolved after the pigtail catheter was correctly positioned with echocardiogram guidance, and hypotension improved. Unfortunately, the patient went into multiorgan failure and passed away the next morning.

Discussion:
Current guidelines state that the Impella inlet should be 3.5 cm below the aortic valve annulus away, and the Impella outlet above the aortic valve. Impella displacement is the most common complication and usually occurs during patient repositioning and transport. Displacement can result in direct irritation of adjacent cardiac structures leading to arrhythmias. Malpositioning of the Impella inlet can also lead to hemolysis. This case highlights the importance of timely recognition and assessment of device placement with bedside echocardiogram during the evaluation of new arrhythmia after Impella placement.
Challenges of White Coat Hypertension in Ambulatory Setting

The prevalence of WCH has been approximated to be as high as 40% in hypertensive patients who present in ambulatory settings. Furthermore, in patients with primary essential hypertension (PEH) who also have WCH, there are added challenges when evaluating high blood pressure in the clinic.

We report a case of a 40-year-old male with an established history of PEH who despite medication compliance and home logs showing controlled BP, consistently presented to the clinic with systolic BP (SBP) > 200mmHg. A 40-year-old male with PEH came to our office for a six-month visit. The patient followed regularly with our clinic and at every visit, a SBP > 200mmHg was noted. During previous visits, he had been referred to the hospital for hypertensive urgency. The patient was compliant with medications, maintaining home BP logs, and follow-ups. Vitals for this encounter were as follows: BP of 240/120mmHg, heart rate of 90 beats per minute, respiratory rate of 16 breaths per minute, and afebrile. On review of his home BP log, average SBP was 125mmHg. He denied headache, chest pain, difficulty breathing, or changes in vision. Physical exam was unremarkable. The patient was advised to go to the hospital for further workup due to concern for hypertensive urgency. However, he wanted to go home and check his blood pressure in a non-medical setting. He agreed to call the clinic with the home BP reading and said that should elevated BP persist at home, he would report to the hospital. After going home, the patient called the clinic and informed staff that his BP reading was 140/79mmHg. He was advised to continue his current medication regimen and regular BP monitoring.

This case highlights that patients who have BP readings high in office but normal at home are perfect candidates for 24 hour ambulatory blood pressure measuring. This will help in diagnosis of white coat hypertension & prevent unnecessary resource utilization of referral and decrease risk of hypotension or interactions with other medications. Underlying anxiety may contribute to WCH so in addition to medical therapy, early referral to psychologist or psychiatrist could be considered.
A Snare to the Heart: A Lead to Recurrent Pericarditis

Pericardial Effusion after Cardiac Device Implantation is a relatively rare finding. Pericardial Effusion, caused by myocardial perforation during pacing lead insertion is rare and usually occurs a short time after placement. We present a case of a patient who was readmitted to hospital for symptomatic pericardial effusion three months after pacemaker placement.

85-year-old female with PMH of ascending thoracic aortic aneurysm, sick sinus syndrome s/p pacemaker placement who presented to the Emergency Department for chest pain that woke her up at night. She had been admitted two months prior for similar symptoms. During her previous admission he was found to have a mild-to-moderate pericardial effusion which was one month after pacemaker placement. Due to the close timing of pacemaker placement and discovery of pericardial effusion, possibility of micro-perforation was considered, and pacemaker interrogation was initiated. At the time, she was treated conservatively with a 2-week course of Colchicine for suspected Pericarditis secondary to pacemaker implantation. On admission, EKG showed nonspecific T-wave changes. Chest CT Angiogram chest showed moderate-to-large pericardial effusion with Reflux into IVC and Azygos veins. Transthoracic Echocardiogram showed Large Circumferential Pericardial Effusion without Tamponade Physiology. Cardiothoracic Surgery performed a pericardial window, which led to resolution of symptoms. Pericardial fluid analysis was negative for malignancy and showed chronic pericarditis. ANA and TSH were negative at the time. Interrogation by Electrophysiology concluded that there was no pacemaker malfunction, and she was recommended to follow up outpatient for evaluation of recurrence and further management after a course of Colchicine and NSAID.

Although rare, Pericardial Effusions and Tamponade after Pacemaker placement can be dangerous or lethal to patients if not caught early enough. Clinicians must be vigilant in identifying Post-Cardiac Injury Syndrome within the Subacute Period of Cardiac Procedures. In this patient’s case, readmission to the hospital was necessary due to worsening pericardial effusion and pericarditis, and significant risk of Cardiac Tamponade despite outpatient management two months prior. Further studies regarding post-cardiac injury syndrome as a cause of recurrent pericarditis are encouraged to improve earlier identification and appropriate management of these patients, potentially leading to better patient outcomes and preventing hospital readmissions.
An Unusual Presentation of Pulmonary Embolism in a Patient Recovered from Asymptomatic COVID-19 Infection

Introduction:
COVID-19 infection is a known cause of hyper-coagulable state. The prevalence of any thromboembolic event could be as high as 43%, especially in critically ill COVID-19 patients. However, the true rate remains unknown in asymptomatic patients. We present a case of pulmonary embolism (PE) in an asymptomatic COVID-19 patient that presented with arrhythmia and was recognized after an abnormal test result.

Case Summary:
A 90-year-old male with diabetes and a recent asymptomatic COVID-19 infection one month ago presented with dizziness and palpitations that occurred at rest. Per initial evaluation by EMS, pulse was 170; blood pressure 98/58. EKG showed supra-ventricular tachycardia with no ischemic changes. The patient denied any symptoms suggesting an acute coronary syndrome. SVT and symptoms resolved after Adenosine administration. A COVID-19 antigen test was negative. Initial labs were remarkable only for a troponin of 2, which trended up to 3. An echocardiogram was done to rule out a cardiac cause of elevated troponin, which was unremarkable. With the patient recently having COVID-19, PE was one of the differentials as a cause of SVT and elevated troponin, despite Wells' score being 1.5. A CT angiography showed a right lower lobe segmental and sub-segmental PE; with no right ventricular strain. Upon further questioning, the patient denied any predisposing factors for a PE, and he did not require any treatment for COVID-19. The patient was initially treated with heparin and was discharged home on Apixaban the next day.

Conclusions:
COVID-19 is now accepted as an individual risk factor for DVT and PE, especially in critically ill patients. Multiple cases are also being reported about asymptomatic COVID-19 patients presenting with PE. It is crucial to keep PE high on the differentials for patients that recently had COVID-19 and have any sign/symptom/lab result that might suggest underlying PE, even if the signs are vague. As Well's criteria is mostly negative in these patients, a new scoring system is necessary for risk stratification.
Staphylococcus aureus Infection Leading to Endocarditis, Septic Arthritis, and Osteomyelitis Following Tocilizumab Infusion

Introduction:
Tocilizumab (TCZ) is a humanized IgG1 anti IL-6 receptor antibody that is commonly used in patients with Rheumatoid Arthritis (RA) who have not responded well to the disease modifying anti-rheumatic drugs (DMARDs). Most common side effects with its use are infections, including opportunistic and mycobacterial infections. We present one case of disseminated infection including endocarditis, septic arthritis, osteomyelitis, and myositis shortly after the patient received TCZ infusion.

Case:
A 39-year-old woman presented with fevers, fatigue, and right hip and leg pain. She has history of RA treated with prednisone 10mg/day and Systemic Lupus Erythematosus on hydroxychloroquine 200mg/day. She failed therapy with Methotrexate in the past year and therefore was started on TCZ infusion 2 weeks ago. Shortly after getting the infusion, she started feeling tired, nauseous, with intermittent abdominal pain. One week later, she developed high grade fevers up to 105°F, diarrhea, mild non-productive cough, and dyspnea. She also had worsening of her chronic right hip and knee pain. She was tachycardic, hypotensive, and diaphoretic on presentation. Her exam revealed a very tender right hip, knee, and ankle joints with restricted range of motion. She was pan-cultured and started on broad-spectrum antibiotics. Multiple imaging modalities including X-rays and MRIs of her lower back, hip, knee, and ankle joints were performed. They revealed findings consistent with septic arthritis and osteomyelitis of the hip and knee with arthrocentesis confirming the diagnosis. Blood and synovial fluid cultures grew methicillin - sensitive Staphylococcus aureus with persistent bacteremia despite being on appropriate therapy. Trans-esophageal echocardiography revealed two vegetations on the A2 and P2 segment of the mitral valve confirming endocarditis.

Discussion:
TCZ is an immunosuppressant drug which has been used in patients with RA as monotherapy or in combination with other DMARDs. The risk of infection ranges from 3.7 to 5.7 per 100-patient years. Widespread infection leading to endocarditis, septic arthritis, and osteomyelitis increases the patient’s morbidity and affects quality of life significantly. Therefore, a high index of suspicion for serious infections, and caution with their use should be maintained when considering such therapy.
Post-Transplant Lymphoproliferative Disorder; A Rare, but Important Complication of Transplant Immunosuppressive Regimens

Introduction: Post-transplant Lymphoproliferative disorder (PTLD) is the most common malignancy to affect organ transplant patients. However, the true incidence is unknown. 80% of cases are diagnosed within the first year of receiving organ transplant, making it an important complication to monitor for in post-transplant patients.

Case Description: We present the case of a 52-year-old woman who is being seen in the clinic 7 months post renal transplant in the setting of ESRD due to Sarcoidosis on Mycophenolate Mofetil, Tacrolimus, and Prednisone who is presenting with the chief complaint of shortness of breath and chest pain. On physical exam patient appeared well and in no acute distress with minimal rales noted in the left lower lung field. After review of records it was noted that her transplanted kidney did have a mass removed prior to implantation, pathology later revealed Renal Cell Carcinoma. CT scan of the chest was ordered which showed a large soft tissue like mass in the medial aspect of the left lower lobe contiguous with the left posterior hilum/pleura and encompassing/partially occluding the bronchus. Subsequent tissue biopsy initially showed possible Angiosarcoma, however after further review and FISH analysis the mass was consistent with Diffuse-Large B-Cell Lymphoma (DLBL) with a few cells weakly positive for EBV viral RNA consistent with likely PTLD. PET scan showed high uptake in the left lung mass, a mass in the spleen as well as T9. Oncology recommended discontinuation of Mycophenolate and observation. Follow-up surveillance PET scans to date have showed continual decrease FDG uptake and no new hypermetabolic areas consistent with remission of disease.

Discussion: This case demonstrates the importance of regular follow-up and evaluation of organ transplant patients as PTLD is quite prevalent in this population which may be picked up by physicians who are familiar with transplant medicine but may cause diagnostic difficulty in those not familiar especially in the primary care setting. Another interesting aspect in this case was resolution with stopping of Mycophenolate and no need to initiate cytotoxic therapy which is standard of care for DLBL in otherwise normal states of health.
Kratom Intoxication Presenting with Acute Toxic Metabolic Encephalopathy, Refractory Shock, and Respiratory Failure

Introduction
Kratom is a plant native to Southeast Asia and regions of Africa that, over the past years, has become more common in the US as an opioid alternative. It is sparsely regulated and has the potential to be used as a drug of abuse or cause unintentional intoxication due to unknown safe doses.

Case Presentation
We present the case of a 54-year-old woman with a history of depression and chronic pain, who presented to the emergency department by ambulance after her husband found her in bed unresponsive. Upon arrival, the patient was found to be hypoxic, hypotensive, and hypothermic. She had a Glasgow Coma Scale of 5 and was intubated for airway protection. Laboratory investigation was significant for elevated transaminases and alkaline phosphatase, mild leukocytosis, mild TSH elevation (6.65), lactic acidosis, and elevated procalcitonin. She was started on empiric antibiotics and admitted to the intensive care unit. Additional evaluation with chest X-ray, CT of the head, chest, abdomen, and pelvis was unremarkable. Lumbar puncture, urinalysis, blood cultures were also unable to identify an infectious source. Urine drug screen, acetaminophen, ethanol, and salicylate levels were all negative. EKG showed a prolonged QTC interval. ICU course was complicated by multiorgan failure including refractory shock, acute kidney injury, metabolic encephalopathy, and respiratory failure. She required vasopressor support, emergent hemodialysis, sedation, and mechanical ventilation. On hospital day 4 her condition had stabilized; she was extubated, and her mental status improved. She admitted she had been taking high doses (>10g/day) of kratom hoping it would help with her chronic pain.

Discussion
Case reports have linked kratom use to liver toxicity, cholestasis, seizures, and even death. Concomitant use with other drugs appears to substantially increase morbidity and mortality. Few cases have been reported linking kratom use to circulatory compromise and respiratory failure. In a PubMed search, we found 2 cases where patients presented with altered mental status and shock. Our case is similar whereas extensive laboratory and radiologic investigation for potential causes for persistent shock were negative and kratom intoxication was deemed as the likely culprit for the presentation.
Tuberculosis of the Lumbar Spine and Secondary Psoas Abscess in Immunocompetent Patient: An Uncommon Cause of Back Pain

INTRODUCTION: Though incidence of tuberculosis (TB) is low in the United States, it continues to affect certain groups. Extra-pulmonary cases of tuberculosis can present in a minority of patients. We present a case of tuberculosis of the lumbar spine complicated with an abscess of the psoas muscle in an immunocompetent male.

CASE PRESENTATION: A 39-year-old male with medical history of hypertension presented to the hospital due to a left psoas muscle abscess identified on outpatient work-up. He had a two-year history of worsening back pain and was eventually referred to neurosurgery due to intermittent paresthesia. Previous x-ray had showed mild lumbar spondylosis. He was initially treated with physical therapy and analgesics, which did not result in symptom improvement; magnetic resonance imaging of the lumbar and thoracic spine revealed loss of disc height on L4-L5 and destructive changes with acute marrow edema, raising concern for discitis/osteomyelitis, in addition to ventral epidural extension involving the left psoas and iliopsoas musculature with abscess formation. Patient then visited the emergency department, where he endorsed progressive lumbar pain and denied fevers, cough, fecal or urinary incontinence and focal motor deficits. Upon questioning, patient mentioned to have successfully completed 6 months of isoniazid treatment for latent tuberculosis while in prison almost two decades prior. HIV test was negative. Chest x-ray was unremarkable. Urine and blood cultures were negative. Around 60 ml of purulent fluid were drained from left psoas abscess and acid-fast bacilli smear was positive. Fluid culture grew Mycobacterium tuberculosis (MTB). Combination treatment of isoniazid, rifampin, ethambutol and pyrazinamide was initiated.

DISCUSSION: MTB infection of the lumbar spine with associated psoas abscess has been reported in a small subset of individuals. Upon literature review, most available reports involve patients with some degree of immunosuppression who have no history of TB treatment. In our case, patient had remote exposure to MTB and completed a course of treatment for latent TB at that time. He tested negative for HIV and had no underlying cause for immunosuppression such as malnutrition or chronic steroid use. Our patient was started on standard TB treatment and symptoms subsided shortly afterwards.
Spontaneous Coronary Artery Dissection, a Rare Cause of ACS

Background: Spontaneous coronary artery dissection (SCAD) is an uncommon cause of acute coronary syndrome (ACS) which is typically associated with fibromuscular dysplasia, rheumatological and genetic diseases. Here we present a case of a young female, who presented with unrelenting epigastric pain found to have SCAD.

Case: A 48-year-old African American female presented to the ER with multiple episodes of intermittent epigastric pain which radiated down her left arm and was accompanied by heartburn, nausea, dizziness, and diaphoresis. These episodes lasted for 20-30 minutes and resolved on their own. Medical history was significant for obesity, hypertension and COVID-19 pneumonia 5 months prior, treated as an outpatient. Upon arrival, patient was hemodynamically stable. Initial EKG showed T wave inversions in the inferior and lateral leads. Serial troponin was 4.22 ng/ml and 4.34 ng/ml (0.00 – 0.04 ng/ml) respectively. Patient underwent left heart catheterization due to ongoing pain and NSTEMI, which revealed extremely tortuous coronaries and SCAD in the mid distal segment of left anterior descending artery. Workup for autoimmune conditions, fibromuscular dysplasia and systemic inflammatory disease was negative. CT angiogram of the head, neck, chest, abdomen and pelvis was also unremarkable. She was discharged on medical management with anti-anginal and dual anti-platelet therapy along with close cardiology follow up.

Decision making: This case highlights that SCAD can mask itself as epigastric pain and can be mistaken as GERD. Patient had a low likelihood of coronary artery disease considering her age, non-anginal chest pain and minimal risk factors for atherosclerotic disease. Due to EKG changes along with elevated troponin and multiple episodes of pain, decision was made to pursue invasive management. Timely catheterization helped us reach the right diagnosis and potentially saved the patient from deadly complications.

Conclusion: SCAD is a rare and fatal cause of ACS. COVID-19 tropism to the heart is well known, causing thromboinflammation. A handful of cases of COVID-19 associated with SCAD have been reported however causality is yet to be defined. It is unclear whether her previous viral infection had any relation with her actual disease and further studies need to be done to better elucidate the pathophysiology.