Transient Enophthalmos in NF1 Complicated by Lumbar Puncture

Neurofibromatosis type-1 (NF1) is an inherited autosomal dominant disorder, affecting 1 in 3000 population. NIH diagnostic criteria of NF1 includes distinctive bony lesions which are sphenoid dysplasia and pseudoarthrosis. They can occur with enlargement of the middle fossa resulting in herniation of the intracranial contents into the orbit and proptosis. Here, I present a case of a NF1 patient experiencing enophthalmos from decreased intracranial pressure status post lumbar puncture (LP).

A 19 year old female with a history of NF-1 presented to ED with the complaints of blurred vision, right-sided headache and eye pain about 30 hours post LP. As a part of work ups, LP was performed in her previous admission. The physical exam was significant for right enophthalmos, tearing, right eye pain with extraocular movement and vision 20/50 right, 20/25 left. CT Head and Orbit revealed right globe migrated 12.4 mm posteriorly and new right lacrimal gland enlargement compared to pre-LP scan in addition to right sphenoid wing dysplasia and right orbit deformity with absence of medial and superior orbital wall posteriorly. The patient was given IV methylprednisolone along with ampicillin/sulbactam for possible dacryoadenitis. Day 3 repeat MRI Orbit demonstrated a slightly proptotic right globe. Her headache and eye pain improved and she was discharged home with ophthalmology follow up.

This case is a rare presentation of transient enophthalmos complicated by LP in the setting of absence of greater wing of sphenoid occurring at a frequency of about 4% in NF1.

References
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Severe Hypothermia Requiring Modified ACLS and Venous- Arterial Extracorporeal Membrane Oxygenation (VA ECMO)

Introduction:
Hypothermia is defined as a core temperature less than 95 F. It can be classified as mild (90-95 F), moderate (82-90 F), or severe (< 82 F). Hypothermia causes almost 1500 deaths in the United States. Presentation and management differs depending on the severity. Etiologies include outdoor cold exposure, cold water submersion, and medical conditions such as hypothyroidism, sepsis, and medications. We present a case of hypothermia requiring advanced life-saving measures and benefiting from VA ECMO.

Case Presentation:
A 40 years old male was found down with a core temperature of 77 F. At presentation, the patient was unresponsive, hypothermic, and in PEA arrest. He was intubated, and ACLS protocol was initiated. Hypothermia protocol was also initiated including chest tubes placement, warm IV fluid resuscitation, and external warming. Patient did not respond to initial management and developed ventricular fibrillation. Per modified ACLS for hypothermia, CPR was continued by mechanical CPR machine and shocks were delayed until rewarming was achieved. Pertinent labs noted venous pH 7.01, PCO2 76, lactate 11.5, creatinine 1.8, and troponin of 1.03. Patient was then started on VA ECMO requiring cardiac shocking for ventricular fibrillation. Patient was maintained on VA ECMO for 2 days and exhibited a full cardiac and neurologic recovery. seven days later, he was discharged home walking without assistance.

Discussion:
This case illustrates the importance of early recognition of severe hypothermia, the application of modified ACLS guidelines, and the benefit of VA ECMO in severe hypothermia treatment. Moreover, national guidelines for the management of hypothermia are needed in order that all patients with severe hypothermia would benefit from VA ECMO.
Unusual case of a Loculated Pleural Effusion Due to Chronic Pancreatitis

Introduction: Large exudative pleural effusions are most commonly caused by infections and malignancy. Less commonly, other etiologies such as pancreatitis, or chronic Boerhaave's syndrome should be considered.

Case description: A 65-year-old male with a history significant for prior retroperitoneal fluid collection and alcohol abuse presented with left sided chest pain of 2 weeks duration. He denied abdominal pain, fever, cough, vomiting, or shortness of breath. Physical exam was significant for decreased breath sounds on the left lung base, he had no abdominal tenderness. Chest X-ray showed a large left sided pleural effusion. Two sets of blood cultures were sent, and the patient was started on broad spectrum antibiotics. Thoracentesis revealed exudative fluid with elevated amylase>6000. Repeat CXR showed persistent effusion. Bronchoscopy and VATS were performed without evidence of a bronchial lesion, or mass. Loculated cloudy fluid was drained and a chest tube was placed. Blood and pleural fluid cultures were negative. CT scan suggested possible pancreatic pseudocysts with fluid seen near the tail of pancreas. Serum Lipase was mildly elevated at 146. EGD with EUS was performed to evaluate for pancreatitis versus Boerhaave’s syndrome. EGD revealed normal esophagus, however, fluid and debris was seen around the tail of pancreas, suggestive of acute pancreatitis. Pleural fluid analysis showed elevated lipase>20000. Patient was discharged and was advised outpatient follow up for repeat CT and possible ERCP in 2 weeks.

Discussion: Massive pleural effusion is a rare complication of chronic pancreatitis. Large effusions are likely due to the presence of a pancreaticopleural fistula which has an incidence of 0.4%. Another mechanism is through transdiaphragmatic lymphatic blockage. This case aims to increase awareness of these rare etiologies of massive pleural effusion and possibly help establish better guidelines for diagnosis and treatment which appear underexplored currently.
COVID-19 Vaccination. A Stressful Conundrum

Introduction
Adrenal crisis is a life-threatening condition which can be precipitated by internal or external factors. We present a case of adrenal crisis following administration of the COVID-19 vaccine.

Case Presentation
A 33-year-old male with history of adrenal insufficiency following TBI presented with hypotension, syncope and hypoglycemia after administration of COVID-19 vaccination. The first dose was well-tolerated. Following the second dose he reported fevers, generalized weakness and syncopal episodes. He presented to the emergency department with a BP of 93/46, fever of 101.9 °F, tachypneic and tachycardic. Laboratory findings revealed a blood glucose level of 63 mg/dL and lactic acidosis. No electrolyte abnormalities were noted. He received 3 L of IV fluids, 1 amp of dextrose and 100 mg dose of IV hydrocortisone. Vital signs improved and hypoglycemia resolved with dextrose. He was discharged the following day on his usual steroid regimen and educated of the importance of doubling his home steroid regimen with future vaccinations or illnesses.

Discussion
The hallmark presentation of adrenal crisis is shock. Patients also present with significant electrolyte derangements including hyponatremia, found in 70 to 80% of patients, and hyperkalemia secondary to mineralocorticoid deficiency. Acute infectious processes may often be the precipitant of adrenal insufficiency. However, reportings of adrenal crises precipitated by vaccinations appear under reported. A previous report revealed severe adrenal crisis triggered by administration of 3 vaccines in a single healthcare appointment.

Conclusion
Routine vaccinations are known to cause systemic symptoms. This is no more evident than with the administration of COVID-19 series. Injection site pain remains the most commonly reported reaction. Systemic symptoms nearly double in reporting following the second dose, likely owing to the more robust second immune response. It may be reasonable to suggest patients with adrenal insufficiency receive stress dose steroids with administration of the 2 part COVID-19 vaccine to avoid adrenal crisis.
Introduction: Hypercalcemia is a common feature of malignancies. The cause of hypercalcemia includes bony metastasis or endogenous hormone production. Humoral hypercalcemia is a well-known paraneoplastic syndrome of lung cancer, and is uncommonly described in other malignancies. Other cancers known to cause humoral hypercalcemia are ovarian and breast cancer. It is a rarely described phenomenon in bladder cancer. In this clinical vignette, we describe a patient with metastatic invasive papillary urothelial cell carcinoma of the bladder presenting with humoral hypercalcemia of malignancy.

Case Presentation: A seventy-nine year old male patient with a history of papillary transitional cell carcinoma of the urinary bladder presented to the hospital with weakness, altered mental status and complaints of lower back and left thigh pain. Physical exam was significant for confusion. His calcium level was 18.3 gm/dl on laboratory analysis. Further lab work up showed low PTH, phosphorus, and vitamin D levels. He was treated with IV sodium chloride, IV zoledronic acid, and calcitonin with improvement in his calcium to within normal limits after three days of therapy. His metabolic encephalopathy resolved. The patient was also noted to have hypophosphatemia which was resistant to treatment. The patient had known metastasis to his bones, however, alkaline phosphatase was not elevated. Subsequently, PTHrP was ordered and returned elevated at 8.6 pmol/L confirming paraneoplastic humoral hypercalcemia of malignancy.

Discussion: This case illustrates an uncommon cause of humoral hypercalcemia of malignancy from papillary urinary bladder cancer. This case highlights the importance of keeping a broad differential when approaching clinical situations with other obvious causes such as bony metastasis are present. Humoral hypercalcemia of malignancy should be included in the differential diagnosis of urinary bladder cancer especially when other laboratory findings are discordant with bony metastasis.
**Fever of Unknown Origin: Histoplasmosis, Sarcoidosis, or Both?**

Fever of Unknown Origin: Histoplasmosis, Sarcoidosis, or Both?

Introduction: Pulmonary histoplasmosis is typically asymptomatic but may present with respiratory symptoms, including cough, chest pain, and fevers. Sarcoidosis has a similar clinical presentation. Moreover, histoplasmosis often mimics sarcoidosis on imaging, and immunosuppressive therapy used to treat sarcoidosis can further exacerbate histoplasmosis, leading to disseminated infection. We present a case of pulmonary sarcoidosis with underlying disseminated histoplasmosis.

Case Presentation: The patient is a 48-year-old African American male with past medical history of pulmonary sarcoidosis diagnosed eighteen years prior, remote six pack-year tobacco use history, and COPD, who presented with recurrent nightly fevers. The patient denied any respiratory symptoms, including cough, dyspnea, or wheezing, as well as no recent travel history. CTA of the chest showed lymphadenopathy in the right superior mediastinum, right hilum and subcarinal region, findings consistent with known sarcoidosis. HIV, tuberculosis, and syphilis screen were negative. Bronchoscopy with lavage was initially negative for any infectious etiology, or malignancy. An excisional biopsy of the right supraclavicular lymph node was performed which demonstrated both non-necrotizing granulomatous inflammation, as well as small oval budding yeast forms. Fungal culture from bronchoscopy was positive for Histoplasma capsulatum. The patient was initially treated with amphotericin, with transition to itraconazole, and prednisone for his underlying sarcoidosis. The patient’s fevers finally resolved with antifungal treatment.

Discussion: Even in patients with atypical symptoms of sarcoidosis, disseminated histoplasmosis must remain as differential diagnosis since immunosuppressive therapy, including steroids, can further exacerbate fungal infections. In the rare cases where both are present concomitantly, both must be treated appropriately with clinical judgement.
Primary Squamous Cell Pancreatic Cancer: A Unique Challenge

Introduction:
Squamous cell carcinoma of the pancreas is exceedingly rare; large case series have reported that squamous cell cancers make up approximately 1% of pancreatic ductal carcinomas. Theories regarding the development of squamous cell pancreatic carcinoma center on antecedent acute or chronic pancreatitis-caused inflammation to induce metaplasia. Here, we present a case of primary squamous cell carcinoma of the pancreas in a patient without any history of pancreatitis, acute or chronic.

Case Presentation:
The patient is a 59-year-old Caucasian male with a past medical history of hypertension and type II diabetes mellitus who had presented to the hospital with persistent abdominal pain and unintentional weight loss. A CT of his abdomen and pelvis showed a lobular soft tissue density toward the superior margin of the pancreatic head, with encasement of the nearby vasculature; biopsy of the pancreatic mass revealed primary squamous cell carcinoma. Further investigation revealed no history of diagnosis of either acute or chronic pancreatitis at any point. As he was a poor surgical candidate, the patient was started on palliative chemotherapy with several changes made to the regimen based on poor response on follow-up imaging.

Discussion/Conclusion:
Various studies have theorized a link between the development of primary SCCP with antecedent inflammation like that seen in pancreatitis; one study showed pancreatitis in 49% of 100 patients with pancreatic cancer. However, in our patient, there was no history of diagnosed pancreatitis, nor any subjective history as such. Even with the very rare incidence of primary pancreatic squamous cell cancer, we believe this case represents both an opportunity to further define this disease entity, as well as a complex challenge as for treatment. Further studies regarding molecular markers in primary squamous cell pancreatic cancer will be necessary to formulate treatment pathways of this rare but aggressive disease.
Symptomatic Bradycardia and Hypotension from Wild Honey Intoxication

Introduction
Wild honey intoxication is a rare presentation. It is also known as mad honey due to its intoxicating effects. Poisoning occurs after consumption of honey-containing grayanotoxin, a toxin derived from different species of Rhododendron found in specific regions of the globe.

Case presentation
A 46-year old female presented with sudden onset of vomiting, generalized weakness, sweating, and dizziness after ingestion of 4 teaspoonfuls of wild honey brought from the hilly region of Nepal. On examination, the patient was ill looking, bradycardic with heart rate of 42 beats per minute, and hypotensive with blood pressure of 80/50 mmHg. A 12-lead ECG revealed sinus bradycardia with a heart rate of 39 beats per minute. Other labs including total leukocyte count, hemoglobin, electrolytes, troponin were unremarkable. Provisional diagnosis of wild honey intoxication was made. Patient was given 0.6 mg of atropine along with 1 L of 0.9% normal saline. Second dose of atropine 1.2 mg was given intravenously. After 15 minutes her heart rate improved to 92 and blood pressure was 100/70 mmHg. Patient was observed for 24 hours after which she was discharged from the hospital.

Discussion
Wild honey has been used for a long time in various parts of the globe for its recreational and medicinal values including hypertension, coronary artery disease, diabetes, dyspepsia, indigestion, peptic ulcer, flu, cough, and also as an aphrodisiac. Wild honey is different in comparison to commercial honey. It contains grayanotoxin, which acts on the sodium channels on muscles and nerves, muscarinic receptors, and causes stimulation of the vagal nervous system, simulating cholinergic toxicity. Cumulative effects lead to bradycardia, hypotension, altered mental status, drowsiness and gastrointestinal side effects. Symptoms may mimic the clinical picture of organophosphate poisoning. Treatment is symptomatic. Hypotension and bradycardia improves with intravenous fluids and atropine.
Left Ventricular Free Wall Rupture: A Rare Case Resulting from Delayed Presentation Due to Covid-19 Fear

Introduction:
Left Ventricular free wall rupture (LVFWR) is an increasingly rare complication of acute myocardial infarction (AMI), which is usually associated with catastrophic results and high mortality rate.

CASE:
76 year old female with history of hyperlipidemia, hypertension who initially experienced typical chest pain but delayed seeking medical attention by several hours due to SARS-CoV-2 fears. Was found to have ST elevation myocardial infarction in the inferolateral leads and underwent subsequent angioplasty requiring drug eluting stent placement to the Left Circumflex. Three weeks later she presented with syncope episode and mentioned experiencing pleuritic chest pain. As part of syncope workup, echocardiograph was obtained that revealed approximately 16mm defect in wall extending 13 mm deep and a slit like penetration in the free wall consistent with LVFWR with hemoperricardium. Patient was vitally stable and transferred to Cardiac Intensive Care Unit and obtain emergent cardiac magnetic resonance imaging which confirmed LVFWR with partially contained hemorrhagic pericardial effusion. After evaluation by cardiothoracic surgery patient elected for conservative management and follow up.

Discussion:
LVFWR is an increasingly rare complication of AMI as a result of early intervention with angioplasty or thrombolitics. Recently the incident of LVFWR after AMI was reported at 0.14% and usually fatal or catastrophic requiring urgent surgical intervention. Even with surgical intervention the mortality rate is as high as 35%. This patient’s delay in initial presentation with chest pain due to fear of SARS-COVD-2 resulted in delayed intervention, which is associated with increased incidence of AMI complications. Furthermore, contained LVFWR with hemodynamic stability is an even smaller subset of the rare catastrophic complications following AMI. This case emphasizes the importance of early presentation and appropriate intervention in reducing the complications associated with AMI.
Lung Cancer Presenting as Necrotizing Pulmonary Mass in a Patient with AIDS

Lung Cancer Presenting as Necrotizing Pulmonary Mass in a Patient with AIDS
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Introduction: Necrotizing lung masses are more commonly associated with infectious processes in HIV/AIDS. However, other etiologies must be excluded including opportunistic infections, endobronchial lesions and malignancies.

Case Presentation: A 65-year-old transgender female with history of COPD, tobacco abuse, and HIV non-adherent to HAART presented with dyspnea, 3-month duration of productive cough, and hypoxia. CT scan identified a necrotic right lobar consolidation, necrotic lymphadenopathy and pleural effusions. Given a CD4 count 192/mm3, AIDS was diagnosed. Actinomyces was clinically suspected, yet all infectious etiologies were unrevealing. Subsequent thoracentesis, bronchoscopy and fine needle aspiration revealed malignancy of indeterminate type. Unfortunately, the clinical course worsened after bronchoscopy necessitating BiPAP with subsequent cardiac arrest leading to her passing.

Discussion: Pulmonary adenocarcinoma is accepted as the most common non–AIDS defining malignancy, occurring 2–4 times higher than the general population. It develops at a younger age with a more aggressive course and prognosis suggesting associated causative factors of immunosuppression, recurrent pulmonary infections, and possible HIV oncogenesis. However, the most accepted hypothesis remains a higher rate of tobacco abuse in HIV patients. Necrotizing lung masses are more commonly associated with infectious processes in HIV/AIDS. However, other etiologies must be excluded including opportunistic infections, endobronchial and parenchymal lesions. The association of HIV with malignant processes has been well documented, yet with few case reports on necrotizing adenocarcinoma. Thus, it is increasingly important to implement screening spiral CT scans and further studies on necrotizing lung cancer to determine its clinical significance with regard to this population subtype. In general, necrotizing adenocarcinoma is rare and its occurrence in HIV-positive patients is particularly noteworthy.
Sarcoma of The Brachial Plexus: A Case Report

Introduction:
Brachial plexus neuropathy due to malignant nerve sheath tumors are rare, with an incidence rate of 0.001%. In patients with neurofibromatosis type 2, data indicates that increased risk is associated with patients with a history of radiation, although it continues to be exceedingly rare, with limited treatment options. We thus present a case of neurofibromatosis-associated malignant peripheral nerve sheath sarcoma without a known pre-existing plexiform neurofibroma.

Case:
A 44-year-old female with past medical history of neurofibromatosis type 2, and morbid obesity who presented with a chief complaint of left shoulder and upper extremity weakness and paresthesia, which had begun approximately one month prior to presentation. MRI of the brachial plexus revealed a large lesion with central necrosis involving the inferior aspect of the left brachial plexus, as well as fusiform thickening of the C7 nerve root, with anterior and superior extension and an overall bulky area measuring 5 cm. Multiple attempts at IR biopsy were unsuccessful, and so neurosurgery was consulted for resection of the tumor. Patient underwent subtotal resection, histopathology confirmed diagnosis of malignant peripheral nerve sheath sarcoma. The patient was referred to a sarcoma center, as the main treatment backbone would likely involve complete resection of the tumor with eventual limb amputation followed by radiation therapy.

Discussion:
The overall prognosis for this condition is poor, with a five-year survival rate of between 34-64% in various studies. Continuing to identify cases with prompt imaging studies and then biopsies, and consideration of enrolling patients into clinical trials such as the one being conducted at sarcoma centers, will only help to bolster our understanding of this disease, and the treatment modalities and adjunctive therapies that may improve patient mortality.
Hepatic Vein Pylephlebitis Resulting in Bilateral Pyopneumothorax

Pylephlebitis is a rare condition characterized by infective thrombosis of the portal vein generally associated with intra-abdominal infections. We highlight a unique presentation of hepatic vein pylephlebitis stemming from complications of diverticular perforation. Pyopneumothorax has not been reported as a complication of this infection.

A 36-year-old African American male with a history of sigmoid diverticulitis with complications of perforation and abscess formation presented with fever, dyspnea, cough and epigastric abdominal pain. He was tachycardic, had decreased breath sounds on the right side and an initial WBC 47,000. CT scan of the abdomen, pelvis, and thorax showed hepatic vein opacification indicative of thrombosis, along with multiple pulmonary and hepatic lesions indicative of septic emboli and hepatic abscess. Blood cultures grew positive for Streptococcus anginosus. The hepatic abscess was drained and wound and anaerobic cultures grew positive for Escherichia coli, Streptococcus constellatus, and Fusobacterium necrophorum. Empiric antibiotic therapy with ceftriaxone and metronidazole was initiated. The patient developed bilateral hydro pyoneumothoraces, the fluid was drained repeatedly by interventional radiology. Fluid cultures grew S. anginosus. The patient slowly improved and the patient was subsequently discharged on long term IV antibiotic therapy for four weeks. Pylephlebitis is a condition that is rarely reported in the literature. Pylephlebitis is generally caused by intra abdominal and pelvic infections such as diverticulitis and appendicitis. Diagnosis requires demonstration of portal vein thrombosis which is commonly accompanied by bacteremia. Bacteremia is generally polymicrobial, with common isolates including Bacteroides fragilis and Escherichia coli. Associated complications include pyogenic hepatic abscess along with septic pulmonary emboli as was observed in this case. The development of pyopneumothorax was a consequence of metastatic spread of the streptococcal infection as members of the S. milleri group (including S. anginosus, constellatus) have been associated with this complication. Clinicians should be aware of this potential complication.
Renal Failure and Hemoptysis, A Unique Presentation, A Unique Diagnosis: Microscopic Polyangiitis

The combination of pronounced renal failure and hemoptysis is a common presentation of antineutrophilic cytoplasmic antibody (ANCA)-associated vasculitis. Microscopic polyangiitis (MPA) is a rare, systemic, necrotizing, pauci-immune, small vessel vasculitis that typically presents in this manner and is associated with anti-myeloperoxidase (MPO) specificity in a vast majority of patients. It affects approximately 20 per 1 million European and North American people per year. Diffuse alveolar hemorrhage is a common pulmonary pathology associated with MPA, but chronic interstitial fibrosis is another pulmonary manifestation. Kidneys are typically slowly affected, leading to end-stage renal disease, but can also present with rapidly proliferating renal failure. Remission is often achieved with chemotherapeutic agents and immunosuppression, but 5-year mortality can approach 25% with increasing age and escalating respiratory or renal involvement.

A 69-year-old female with past medical history of hypothyroidism, myxedema heart disease, and aortic insufficiency presented with symptoms of body aches, chills, shortness of breath, and cough with mild hemoptysis. The cough started 2 months prior and the patient never had any previously documented renal disease. Her presenting creatinine was 5.4, hemoglobin 5.9. CTA chest was negative for pulmonary embolism but did show bilateral ground-glass opacities. COVID-19 test was negative. Renal ultrasound showed increased echogenicity within the renal cortices, intrinsic renal disease, but no hydronephrosis. Microscopic analysis of urine showed dysmorphic RBCs and presence of granular and RBC casts. The patient was found to be P-ANCA positive with predominant MPO antibody. Renal biopsy was performed and showed evidence of crescent formation without granulomas. Life-saving diagnosis of microscopic polyangiitis was made, followed by urgent plasmapheresis, steroid taper and rituximab infusion which led to improvement of clinical status. This prevented intubation and need for dialysis.

In patients with hemoptysis and renal failure, especially with rapidly proliferating renal failure, a diagnosis of MPA should be considered.
Hydro-Pneumopericardium Following Pericardial Drain Removal

Introduction: Pneumopericardium is a rare clinical finding that is characterized as air accumulation in the pericardial space. While pneumopericardium is an uncommon clinical finding, it can have life-threatening consequences if undetected. Tension pneumopericardium may develop causing cardiac tamponade, and subsequent hemodynamic instability. Causes of pneumopericardium can be categorized as traumatic, iatrogenic, fistula formation with adjacent structures, infection, barotrauma from positive pressure ventilation, and spontaneous.

Case Presentation: A 43-year-old female with past medical history of moderate pericardial effusion, uncontrolled hypertension, insulin-dependent diabetes mellitus, chronic kidney disease, and diastolic heart failure presented with dyspnea and abdominal pain. In the emergency department, her blood pressure was 219/126 mmHg and oxygen saturation was 100% on 4L nasal cannula. Labs were significant for elevated troponin and pro-hormone B-type natriuretic peptide. An electrocardiogram noted sinus rhythm, and chest X-ray showed cardiomegaly with pulmonary edema and bilateral pleural effusions. An echocardiogram revealed a large pericardial effusion without tamponade physiology. Given the increase in size of the effusion, a pericardiocentesis was performed, and initially drained 760 cc while a pericardial drain was left in place. The patient's drain fell out, so a repeat echocardiogram was performed that raised concern for pneumomediastinum. A chest CT scan showed hydropneumopericardium. The patient was hemodynamically stable, so no further interventions recommended and the patient was discharged home. The patient returned ten days later and a repeat echocardiogram showed large free flowing effusions with signs of early tamponade requiring repeat pericardiocentesis.

Discussion: A few cases of pneumopericardium have been reported in the literature following removal of a pericardial drain. This case serves to highlight that iatrogenic pneumopericardium is a rare, but possibly life-threatening consequence of invasive thoracic procedures.
Predicting QT Interval Prolongation in Patients Diagnosed with the 2019 Novel Coronavirus Infection

BACKGROUND
2019 Novel Coronavirus (COVID-19) patients frequently develop QT interval prolongation that predisposes them to Torsades de Pointes and sudden cardiac death. Continuous cardiac monitoring has been recommended for any COVID-19 patient with a Tisdale Score of seven or more. This recommendation, however, has not been validated.

METHODS
We included 178 COVID-19 patients admitted to a non-intensive care unit setting of a tertiary academic medical center. A receiver operating characteristics curve was plotted to determine the accuracy of the Tisdale Score to predict QT interval prolongation. Multivariable analysis was performed to identify additional predictors.

RESULTS
The area under the curve of the Tisdale Score was 0.60 (CI 95%, 0.46-0.75). Using the cutoff of seven to stratify COVID-19 patients had a sensitivity of 85.7 % and a specificity of 7.6%. Risk factors independently associated with QT interval prolongation included a history of end-stage renal disease (ESRD) (OR, 6.42; CI 95%, 1.28-32.13), QTc ≥ 450 ms on admission (OR, 5.90; CI 95%, 1.62-21.50), and serum potassium ≤ 3.5 mmol/L during hospitalization (OR, 4.97; CI 95%, 1.51-16.36).

CONCLUSIONS
The Tisdale Score is not a useful tool to stratify hospitalized non-critical COVID-19 patients based on their risks of developing QT interval prolongation. Clinicians should initiate continuous cardiac monitoring for patients who present with a history of ESRD, QTc ≥ 450 ms on admission or serum potassium ≤ 3.5 mmol/L.
A Rare Presentation of COVID-19 Associated Thrombotic Thrombocytopenic Purpura; Therapeutic Challenges

Introduction:
TTP is a potential fatal clinical syndrome that can be acquired by infections like COVID-19. Prompt diagnosis and initial treatment are critical to decreasing morbidity and mortality. A 49-year-old African American male with only a past medical history of hypertension and hyperlipidemia presented to the hospital initially with rectal bleeding and hematuria for a few days. He was found to have COVID-19. Laboratory works up showed anemia, Thrombocytopenia, elevated LDH, and normal renal function. Physical exam was only positive for left lower quadrant abdominal tenderness. CT abdomen and pelvis were unremarkable. He was presumed to have ITP related to COVID-19 infection but TTP was also in the differential diagnosis. The patient's hematuria and rectal bleeding were resolved, and he was stable for discharge. He was discharged on steroids. After 3 weeks, the patient re-presented to the ED with vomiting and confusion. His labs showed hemoglobin 6.4 g/dl, platelet 12 billion/liter, Cr 4.45 mg/dl, LDH 1959U/L, reticulocyte count 9.5%, Haptoglobin <8 mg/dl, with blood smear, demonstrated an increased number of schistocytes with associated nucleated red blood cells and left myeloid shift was noted which was consistent with microangiopathic destruction of RBCs. ADAMTS13 was sent in the patient’s prior admission but resulted after the patient had been discharged. The value was <5%, consistent with TTP. Other viral markers were unremarkable. Urgent plasmapheresis started. The Patient’s encephalopathy improved after the first plasmapheresis. He received 6 days of plasmapheresis. The complete blood count was rechecked every day and showed significant improvement after plasmapheresis.

Discussion:
TTP can be triggered by viruses that may include COVID-19. Plasma transfusion or exchange is the most effective treatment for TTP patients in the setting of COVID-19. As we continue to learn about the COVID-19 our case serves as an example of how severely this virus can present.
A Case of Massive Hemothorax Leading to Obstructive Shock

Introduction
Obstructive shock is one of the rarest types of shock caused by the reduction of cardiac output despite normal intravascular volume or cardiac function. Classically, it has been known to be caused by tension pneumothoraces, cardiac tamponade and massive pulmonary emboli. The speed at which the state of this shock progresses must be kept in mind in order to proceed with an immediate life-saving intervention.

Case
74-year-old female, presented to the emergency department with dyspnea. Contrast Tomography (CT) scan of the chest showed a large necrotic left upper lobe mass with multiple pulmonary nodules and mediastinal lymphadenopathy with metastatic disease involving the brain. Biopsy of the mass showed poorly differentiated adenocarcinoma. During her hospital course, she developed worsening dyspnea, hypotension, requiring intubation and pressors for hemodynamic support. Decision was made to evacuate large pleural effusion with the idea of relieving the tamponade effect on the heart. Thoracostomy tube was inserted, with evacuation of 2 liters of old-dark bloody effusion and immediate resolution of obstructive shock. Repeat CXR was obtained that revealed improvement of lung field aeration. Due to guarded prognosis, decision was made to terminally wean. Time of death was announced the following day.

Description
There have been a number of rare cases such as obstructive shock in the setting of massive pleural effusions, acute inferior cava (IVC) obstructions due to IVC filter placement that have fortunately been able to be decompressed and subsequently have led to significant improvement hemodynamically. Our case demonstrates a very rare cause of obstructive shock with mediastinal shift due to a massive left-sided hemothorax, relieved by a thoracostomy tube. This case report highlights the importance of considering an immediate life-saving intervention in a timely manner to prevent further increase in morbidity and mortality.
The Role of Diuretics in COVID Pneumonitis

There is not enough data supporting diuretic use in COVID ARDS, when there is normal BNP and no sign of fluid overload. Multiple studies, have demonstrated that conservative fluid management, fluid restriction and use of diuretics in ARDS patients can decrease lung injury and increase ventilator-free days by reducing intravascular volume and lung edema, without significant hemodynamic consequences. In COVID-19 pneumonitis as a result of diffuse alveolar damage and increased capillary permeability we have pulmonary edema similar to ARDS. The pulmonary edema worsens as intravascular volume increases. Therefore, it is reasonable to consider diuretic therapy for COVID ARDS requiring high flow oxygen as well. Conservative fluid management is for patients who are adequately resuscitated and shock-free. It means 1) No maintenance fluids 2) Diuretics, as tolerated, to normalize volume status until off the ventilator or oxygen 3) Holding diuretics for rising creatinine and/or active urine sediment. We applied Conservative fluid management and diuretic therapy on more than 20 patients with COVID pneumonitis, who required high flow oxygen despite being on steroid and/or Remdesivir, and were able to successfully wean them off of oxygen with daily diuresis. Here we present one of these cases.

65 y.o. female was admitted for shortness of breath due to COVID pneumonitis. She was started on steroid, Remdesivir, and Lovenox. She was saturating 80% on 55 L of high flow O2. There were no signs of fluid overload on exam. Daily Diuresis was started to decrease lung congestion, with close monitoring of patient’s blood pressure and creatinine level. She was off oxygen after 3 weeks of diuresis.

This case demonstrate the importance of considering diuretic therapy for COVID patient’s who require high flow oxygen despite not being fluid overload due to heart, liver or kidney dysfunction. More investigation needs to be done regarding the role of diuretics and fluid restriction in treatment of COVID ARDS.
“NUT” An Anatomical Malignancy! A Solid Tumor Characterized by a Genetic Translocation

NUT midline carcinomas are rare poorly differentiated tumors driven by t(15,19) rearrangements and clinically characterized by rapid progression and high mortality. A 49 year-old male with no comorbidities, presented with acute worsening of chronic cough, new onset hemoptysis and left sided chest pain for 2 weeks. Workup revealed NUT midline carcinoma of the lung as confirmed by next-generation sequencing that revealed the presence of a NUTM1-BRD4 fusion. He was diagnosed with NUT midline lung cancer clinical stage IIIB. The tumor was not amenable to surgical resection, therefore, he was started on concurrent chemoradiation with weekly carboplatin and paclitaxel for 5 weeks. Follow up CT scan showed partial response and the patient was started on maintenance durvalumab. Two months later, he presented with a metastatic site in the posterior muscle compartment of the left arm and was treated with local radiotherapy (RT). He tolerated maintenance durvalumab until he had pulmonary recurrence four months later. Durvalumab was then stopped, and he was started on BET inhibitor, molibresib 120 mg daily. Nearly 3 months after being on molibresib, he presented with brain metastasis for which he had a craniotomy with tumor resection and gamma knife to solitary metastatic lesions. The patient was then started on chemo-immunotherapy with carboplatin plus pemetrexed and pembrolizumab. After two cycles, his disease progressed and he succumbed to it. The patient survived for a total of 18 months. An optimal treatment regimen for this rare malignancy has not yet been established. Current treatment approaches are aimed at surgery, chemotherapy and radiation, in varying combinations based upon location and staging. Despite these treatment efforts, remission is generally brief, followed by recurrence of further metastasis, and ultimate mortality. Data regarding the role of target therapies like bromodomain and extra-terminal domain protein (BET) inhibitors are still in early phases of clinical trials.
A Broken Heart: A Case of Stress Induced Cardiomyopathy Precipitated by Community Acquired Pneumonia

Takotsubo cardiomyopathy or stress-induced cardiomyopathy is a syndrome characterized by a transient reduction of systolic cardiac function generally in response to emotional or physical stressors. Stress-induced catecholamine excess is believed to be the main factor driving myocyte dysfunction. Most patients are female and postmenopausal.

We present a case of a 62-year-old Caucasian male with a past medical history of hypertension who presented to the ED with the chief complaint of squeezing chest pain followed by multiple episodes of productive cough and epigastric discomfort. ECG showed sinus tachycardia with premature supraventricular complexes. Chest X-ray showed severe pulmonary vascular congestion. Labs showed elevated WBC to 26.8 bil/L, elevated troponins to 5.81 ng/ml, elevated Lactic acid to 4.6 mmol/L, elevated Procalcitonin to 9.8 ng/mL, and elevated serum creatinine to 2.58 mg/dL. 2D Echo showed EF of 15% with global wall akinesis. He was admitted to the medical Intensive care unit. He was started on broad-spectrum antibiotics, IV heparin. A central line was placed for hemodynamic support. His respiratory status has improved without the need for intubation. Two days later, continuous rhythm monitoring showed new onset Atrial Fibrillation. Labs showed down-trending WBC and serum creatinine with an appropriate response to antibiotics. One week later, a repeat 2D Echo showed a normal EF of 60% with no wall motion abnormalities. He underwent coronary CTA which showed non-obstructive coronary artery disease. The patient was discharged on Aspirin (81 mg once daily), Eliquis (5 mg twice daily), Coreg (6.125 mg once daily), Entresto (24-26 mg twice daily), and Atorvastatin (80 mg once daily).

Takotsubo cardiomyopathy is a reversible but potentially life-threatening condition that carries high morbidity and mortality. Treatment of the underlying cause improves outcomes, and generally conservative supportive care will lead to an improvement in cardiac function in due course.
Immune Thrombocytopenia in COVID Pneumonia: A Case Report of the Oldest Patient

Introduction: COVID infection has shown to cause thrombocytopenia with multiple different mechanisms including the destruction of platelets with immune system stimulation similar to ITP with little evidence for optimal management. Here, we present a COVID infected patient presented with idiopathic thrombocytopenia with a partial delayed response to the treatment.

Case Presentation: A 92-year-old man with a past medical history of coronary artery disease status post remote coronary artery bypass graft, hypertension, diabetes mellitus presented with petechiae, epistaxis, and was found to be profoundly thrombocytopenic with a platelet count of 1x10^9/L. He also had an asymptomatic COVID 19 infection, a normal coagulation profile, and hemolysis markers. There were adequate megakaryocytes in the marrow with no evidence of dysplasia or thrombotic microangiopathy. There was no splenomegaly. He was treated with IVIG, steroids, and platelet transfusion. The platelet sluggishly responded to 106 X 10^9/L on day 23rd. Platelet again started to decrease with the nadir of the 60 X 10^9/L. Prednisone was tapered and Rituximab was added, platelet plateaued at 50-60 X 10^9/L. Meanwhile, the patient was bed-bound, developed a sacral ulcer, later passed away with sepsis.

Discussion: This is the oldest patient known so far to have ITP from COVID infection. Despite IVIG, steroids, and Rituximab the response to treatment was delayed. This has also been observed in other reported cases with suggested several mechanisms of ITP in COVID infection. Also in all COVID cases, a low platelet count has been associated with a fatal outcome suggesting that COVID infection may alter the response of immunomodulation in ITP. This case will educate the clinician for the optimization of treatment on older patients with thrombocytopenia secondary to COVID infection.
Rare Case of Recurrent Hyperhemolysis Syndrome (HHS) and Methemoglobinemia in a Patient with Sickle Cell Anemia

Hyperhemolysis syndrome (HHS) is a rare but serious and potentially life-threatening complication of red blood cell (RBC) transfusion and has been described in both sickle cell disease and non-sickle cell disease patients. It is characterized by destruction of both donor and host RBC. Methemoglobinemia is another rare condition leading to cyanosis, tissue hypoxia and potentially death. We present a middle-aged female with sickle cell anemia who developed methemoglobinemia and hyperhemolysis after a surgical procedure.

41 yo female with sickle cell anemia (SC type) was hospitalized for elective right total hip resection arthroplasty. Patient received 3 units blood transfusion during the surgery. Post operatively patient developed cyanosis and was found to have methemoglobinemia. One week after surgery hemoglobin dropped precipitously from 8.4 gm/dl to 2.5 gm/dl within 24 hours. Lab investigation showed LDH >4500 U/L, haptoglobin <8 mg/dl, bilirubin 12.1 mg/dl, AST 2672 U/L. Coombs test was negative. Reticulocyte count was elevated 202 bil/L. Peripheral smear showed marked normocytic normochromic anemia with an absolute reticulocytosis and moderate anisopoikilocytosis including occasional spherocytosis. G6PD testing showed normal enzyme level. Methemoglobinemia resolved with use of methylene blue. Hyperhemolysis was treated initially with IVIG and Toculizumab without improvement. Patient then underwent splenic embolization and plasmapheresis. She was also treated with Rituximab. Despite aggressive treatment patient continued to have hemolysis and developed multi organ failure. Ultimately family decided to pursue comfort care and patient subsequently died.

Hyperhemolysis syndrome is a rare transfusion related complication that can occur in patient with sickle cell disease. It can pose a significant therapeutic challenge and can unfortunately have devastating consequence including death.
Acute Renal Failure and an Acid-Base Dilemma

Introduction: Acute renal failure (ARF) is well-recognized to result in metabolic acidosis. The acidosis of ARF is associated with decreased ammoniagenesis and anion excretion. Alkalosis in the presence of ARF presents diagnostic uncertainty and therapeutic challenges. This vignette describes a case of ARF resulting in several electrolyte imbalances and metabolic alkalosis.

Case Description: A 54-year-old female with past medical history of small bowel resection with colostomy presented for nausea and vomiting for two weeks. On presentation, she was noted to be tachycardic and lethargic. Her workup revealed a serum creatinine of 6.8 mg/dL, blood urea nitrogen of 147 mg/dL, bicarbonate (HCO3-) of 35 mEq/dL, chloride of 57 mEq/dL, potassium of 2.1 mEq/dL, and sodium of 122 mEq/dL. Her venous blood gas revealed a pH of 7.34. She was given aggressive fluid resuscitation and started on continuous renal replacement therapy for uremic encephalopathy with improvement of her mental status and electrolyte derangements.

Discussion: Alkalosis is an unusual and rare finding in patients with ARF. Normally, as GFR falls, the kidney is less able to excrete the daily acid load, and HCO3- declines as chloride increases producing a hyperchloremic metabolic acidosis. As GFR falls further, anion excretion becomes affected resulting in an anion gap metabolic acidosis. ARF with metabolic alkalosis can occur either in volume depletion with endogenous HCO3- retention, or a gain of exogenous HCO3. In this case, vomiting lead to volume depletion and a loss of gastric secretions containing potassium, and hydrogen chloride. The net effect is intestinal retention of endogenous HCO3-, tubular hydrogen excretion, and intracellular shifts of hydrogen. Occult retention of anions also occurs in renal failure and explains the lack of dramatic pH changes. Recognizing the interplay between history taking and the various electrolytes can give clues to the keen clinician in discerning cause and effect.
Chemotherapy for AKI? A Case of Lymphoma Induced AKI Requiring Hemodialysis

Introduction: Mantle cell lymphoma is an aggressive and rare lymphoid neoplasm that usually presents in advanced stages. Kidney involvement is very rare and can occur by either direct invasion or paraneoplastic glomerulonephritis. There are only a few reports of paraneoplastic GN in MCL. We present a rare case of paraneoplastic glomerulonephritis-induced AKI requiring hemodialysis.

Case presentation: A 73-year-old lady with a history significant for recently diagnosed MCL, presented with generalized weakness. She was found to have stage III AKI. She initially maintained UOP, therefore dialysis was held off. Due to significant proteinuria and hematuria, a kidney biopsy was pursued, which revealed infiltration of lymphoma with Crescentic GN. Her UOP declined throughout her hospital course requiring HD eventually. She received CHOP chemotherapy and was discharged to continue HD and chemotherapy outpatient.

Discussion: MCL’s most common sites of extranodal involvement include the bone marrow, spleen, GI tract, and liver. Kidney involvement is rare and creates a challenge for clinicians; as chemotherapy agents are generally nephrotoxic and can further worsen renal function. Chemotherapy is generally held until AKI improves. MCL is very aggressive and without chemotherapy only palliative options are available. For our patient, chemotherapy was initially held due to her significant AKI but later pursued based on biopsy results.

Conclusion: We present a rare case of MCL and even rarer renal involvement manifesting as lymphoma-induced GN. This case highlights a paradox, as chemotherapy was used to help treat AKI when it is usually avoided due to its nephrotoxicity. Clinicians should keep this in mind when faced with individuals with MCL with renal involvement as the timing for initiating chemotherapy can be detrimental to prognosis.

VIPoma and End-Stage Renal Disease (ESRD): A Unique Mechanism of Encephalopathy

INTRODUCTION: Encephalopathy is characterized by global cerebral dysfunction without primary structural brain disease encompassing delirium and acute confusion, possibly triggered by an underlying metabolic etiology.

CASE: A 37-year-old female with past medical history of ESRD on hemodialysis (HD) and chronic diarrhea secondary to VIPoma presented to the hospital after she was found unconscious by the caregiver. The patient used octreotide for her chronic diarrhea. On examination, she was severely cachectic (BMI=13 kg/m2) with a blood pressure of 85/60 mmHg, heart rate 110/min and was noted to be severely dehydrated. She received 2 litres of normal saline bolus after which she became responsive; however, she was not alert or oriented. Laboratory investigations revealed anion gap metabolic acidosis with a pH of 7.02, bicarbonate 10mEq/L, anion gap 21 mEq/L and lactic acid 3.7 mg/dL. The patient had lost large quantities of fluid through her gastrointestinal tract despite octreotide use. Upon further investigation, it was noted that since last year the patient had been receiving 1 liter fluid pre-dialysis without volume removal during dialysis due to her fluid balance. After eliminating other possible causes of encephalopathy especially infections, medications and stroke, a diagnosis of encephalopathy secondary to severe metabolic acidosis due to bicarbonate wasting was established. She was given 2 ampules of bicarbonate and 5% dextrose in water which led to clinical improvement. She subsequently underwent dialysis with bicarbonate bath and was discharged home.

DISCUSSION: VIPoma presents with secretory diarrhea (>3 litres) in 70% of the patients. ESRD and VIPoma can lead to low serum bicarbonate. We discuss a unique case of encephalopathy secondary to metabolic acidosis due to a synergistic effect of VIPoma and ESRD leading to bicarbonate wasting. Our patient, despite oliguria never required fluid removal. Instead, the losses due to chronic diarrhea were replaced with 1 liter of fluid pre-dialysis.
Astronomical Hypertriglyceridemia Years After TBI

Introduction
There are many side effects of total body irradiation (TBI). Even though rare, some patients are at an increased risk of developing hypertriglyceridemia years after TBI. Here we present a similar case with a massive hypertriglyceridemia.

Case Presentation
A 34-year-old male, with medical history of Hodgkin’s lymphoma s/p bone marrow transplant and whole-body irradiation in 2011, diabetes mellitus on insulin, recurrent hypertriglyceridemia, presented with severe abdominal pain and vomiting. Abdominal pain was epigastric and associated with vomiting. On examination, the abdomen was tender on palpation, no guarding or rigidity. Laboratory investigations revealed lipase of 422, triglycerides of 17618, glucose of 453, anion gap of 19, HCO3 of 13 and HbA1c 12.7. CT abdomen revealed inflamed pancreas with necrosis. Diagnosis of acute pancreatitis secondary to hypertriglyceridemia and diabetic ketoacidosis was established. He was admitted to ICU and treated with IV insulin and fluids. Plasmapheresis was done for the hypertriglyceridemia. Eventually the patient's DKA resolved and triglycerides came down to 384. Patient was discharged with outpatient endocrinology follow-up. Patient eventually was started on weekly plasmapheresis for recurrent hypertriglyceridemia

Discussion
Review of literature showed cases with metabolic derangements in association with TBI but no case has described such severe hypertriglyceridemia as seen in our patient. The patient’s diabetes worsened 8 years after TBI. He currently requires 200 U basal and 200 U bolus insulin every day. Hypertriglyceridemia also started 8 years after TBI, found during his first admission for pancreatitis. He had a total of 8 admissions for pancreatitis in the last 1 year. Other differential in this case includes acquired lipodystrophy as it can present with similar metabolic abnormalities. To conclude, even though rare, complications of metabolic syndrome like insulin resistance and hypertriglyceridemia can occur years after TBI and it is important to evaluate for these complications when managing such patients.
Wellens Syndrome as a Determinant of Coronary Artery Stent Restenosis in Myocardial Infarction (MI) Induced DKA

Wellens syndrome is a characteristic ECG pattern that is specific for critical high-grade proximal stenosis of the left anterior descending (LAD) coronary artery. The classic T-waves are present in the precordial leads, particularly V2-V3, and are described as either deeply inverted or biphasic.

A 31 year-old female with a history of hypertension, hyperlipidemia, coronary artery disease status post drug eluting stenting 7 months ago, end-stage renal disease, and type I diabetes presented to the emergency department for a 2 day history of intractable nausea and vomiting and thus has been non-adherent with her insulin regimen. She denied chest or abdominal pain. Serum glucose was 577 mg/dL and β hydroxybutyrate was elevated. Diabetic ketoacidosis protocol was initiated with an insulin infusion. ECG showed biphasic t waves in V1, V2 and V3. Troponin I was 0.17 ng/mL initially, heparin was initiated and she was transferred to the ICU. Twenty four hours later, Troponin I levels increased to 6.26 ng/mL, prompting coronary catheterization. A 99% occlusion of the previously deployed stent in the proximal LAD was observed, which was revascularized with two drug eluting stents. The patient was maintained on aspirin and clopidogrel after the procedure.

Patients with Wellens syndrome present with mildly elevated cardiac enzymes and characteristic T wave abnormalities in the acute setting without chest pain. However, this sign has only been reported once in patients with in stent restenosis of the LAD. The approach to treatment is a high suspicion for T wave abnormalities in the precordial leads, particularly V2-V3, without any delay in urgent cardiac catheterization. Pharmacologic therapy has no role in the management of Wellens syndrome and cardiac stress testing is an absolute contraindication.

Patients in whom Wellens syndrome is suspected should undergo urgent cardiac catheterization despite having a previous history of myocardial revascularization with minimal troponin elevations.
Sepsis and Hemophagocytic Lymphocytosis: A Case Report Highlighting a Similar Presentation with Different Treatment

Introduction: Hemophagocytic Lymphocytosis (HLH) is a rare life-threatening condition characterized by over-activation of the immune system resulting in hypercytokinemia, multiorgan failure and systemic inflammation. Secondary HLH usually presents in adults without genetic defects potentially triggered by malignancy and sepsis. (1) We present a septic patient who met criteria for HLH.

Case Presentation: 57-year-old lady from Ghana presented with suspected sepsis. She was found to have Klebsiella in the urine and blood. She was initially treated with pressors and IV antibiotics. Imaging obtained for persistent abdominal pain revealed massive splenomegaly. She acquired anemia and thrombocytopenia. TTP was ruled out. Further lab abnormalities such as coagulopathy, profoundly increased LDH, AKI, elevated liver enzymes and ferritin levels along with multisystem involvement raised suspicion for HLH. Bone marrow biopsy did not reveal any hemophagocytosis but CD25 levels were found to be profoundly elevated. Criteria for HLH was met and dexamethasone was started with improvement in labs and overall condition.

Discussion: Differentiating between HLH and sepsis is imperative as treatment for HLH is not in the sepsis guidelines. Hyperinflammation is present in both states giving an over-lapping clinical picture. (2) Clinical features of fever, splenomegaly, cytopenias, hypertriglyceridemia or hypofibrinogenemia, hemophagocytosis in bone marrow or spleen or lymph nodes, low/absent NK cell activity, ferritin > 500, soluble CD25 receptor > 2400 U/mL should raise suspicion for HLH as 5 of 8 criteria are required for diagnosis. Bone marrow biopsy for hemophagocytosis is neither sensitive nor specific therefore not required for diagnosis. Treatment includes dexamethasone, etoposide and in more severe cases with relapse, stem cell transplant. (1).

Conclusion: Clinicians should suspect HLH in septic or “septic-like” patients meeting diagnostic criteria. Prompt recognition is critical as treatment differs. This can be a challenging diagnosis as HLH can be complicated by culture positive sepsis as evidenced by our case report.
Absent Left Main Coronary Artery (LMCA) in a Patient Presenting with Chest Pain

Introduction:
Anomalous coronary arteries can be benign or life threatening with reported incidence from 0.3% to 5.6%. Absent left main coronary artery (LMCA) is a rare congenital cardiac malformation with reported incidence of 0.41%. Anomalous coronary anatomy can lead to severe complications such as myocardial ischemia, arrhythmias and sudden cardiac death.

Case report:
We report a 68-year-old male with history of Hypertension, diabetes mellitus and atrial fibrillation who presented with an atypical chest pain of acute onset. EKG showed sinus rhythm, left axis deviation, right bundle branch block and associated ST segment depressions in leads V1-V3. Initial Troponins were elevated at 0.529. Aspirin 325, atorvastatin 80 mg were given following that heparin drip was started. Transthoracic echocardiogram showed ejection fraction of 40-45% with no regional wall abnormalities and grade two diastolic dysfunction. Cardiac catheterization showed tortuous right coronary artery, absent LMCA with left anterior descending and left circumflex arteries having separate origins with no obstructive disease. No significant obstructive disease was found hence no intervention as balloon angioplasty or PCI were done. Patient was discharged home with 3 months of dual antiplatelet therapy with risk factor modification.

Discussion:
Congenital coronary artery anomalies are second most cause of sudden death in young athletes due to premature coronary artery disease. These are diagnosed incidentally by CT coronary angiography, interventional coronary artery angiographies and in rare cases by transthoracic/transesophageal echocardiography. Though, congenital coronary artery anomalies are quite rare, they can result in acute coronary syndrome and may cause technical difficulties during percutaneous coronary interventions.

Conclusion
Our presentation highlights the need to keep possible diagnosis of congenital coronary anomalies in mind while managing patients with acute coronary syndrome.
An Eroding Cholesteatoma Proved Lethal: A Rare Cause of Tension Pneumocephalus

Pneumocephalus is defined as the presence of air inside the cranial vault. Benign and tension pneumocephalus are different ends of the same disease spectrum. Tension pneumocephalus leads to the formation of a pressure gradient, requiring emergent surgical decompression to prevent herniation of the intracranial structures. In this report, we present a rare case of tension pneumocephalus with essentially benign radiological findings secondary to a ruptured cholesteatoma. The patient was a 64-year-old woman with a history of end-stage renal disease on hemodialysis and hypertension. She presented to the emergency department (ED) with acute-onset weakness and decreased mentation. Physical exam findings were consistent with a cerebrovascular accident (CVA). CT scan and CT angiogram (CTA) were unremarkable for ischemia or hemorrhage but showed signs of free intracranial air, consistent with the diagnosis of pneumocephalus. After the activation of the code stroke, neurosurgery and neurology were consulted. Worsening respiratory status led to a decision to proceed with emergent intubation, but it was held based on the family’s decision to proceed with comfort measures. The patient’s status declined further within minutes and she died. Afterward, the case was discussed with the radiologist, who interpreted the cause as a cholesteatoma that had eroded through the temporal bone.
Sinus Venosus Atrial Septal Defect

Introduction:
Sinus venosus atrial septal defects (SVASD) are rare congenital heart defects contributing to approximately 4-11% of atrial septal defects (ASD). If large defects are not closed, they may result in complications such as pulmonary hypertension, dysrhythmias and stroke. We present a patient with recurrent TIA/Stroke that was found to have a SVASD. (1)

Case presentation:
72-year-old lady with stage III lung cancer presented with transient right-sided hemiparesis and paresthesia. Imaging was unremarkable. Transthoracic echocardiogram was negative for thrombi or shunts. EKG and telemetry were without atrial fibrillation. She was diagnosed with a cryptogenic TIA and discharged on aspirin and statin. She presented months later with global aphasia. TPA was administered as she was a candidate. CT Angiogram head and neck showed an M2 occlusion of the left MCA. Due to improvement of symptoms, as evidenced by a decreased NIHSS, mechanical thrombectomy was not performed. Recurrent cryptogenic vascular events prompted Transesophageal Echocardiogram evaluation (Bubble study done via both arms). This was negative for intra-atrial, ventricular thrombi or valve vegetations but bubbles were observed in the left followed by the right atrium, raising suspicion for anomalous pulmonary venous return and SVASD. This was confirmed by cardiac CT angiography which showed a SVASD measuring 12 mm in diameter. She was recommended to follow-up with a tertiary center; this was declined due to her poor cancer prognosis.

Conclusion:
Diagnosis of SVASD can be challenging with a negative transthoracic echocardiogram, however, having a high suspicion, such as recurrent cryptogenic TIA/Stroke, should prompt evaluation with a transesophageal echocardiogram as it is more sensitive. Further imaging with cardiac CTA or MRI can be helpful in quantifying the size of this rare defect as defects greater than 1 cm are likely to require intervention especially if it is complicated by a TIA/Stroke.
Endoscopic retrograde cholangiopancreatography (ERCP) is utilized in clinical practice for the diagnosis and treatment of biliary and pancreatic disease. We describe a case of splenic hematoma as the outcome of a patient requiring ERCP for obstructive jaundice secondary to atypical pancreatic cancer.

A 56-year-old female presented to the emergency department with acute onset diffuse abdominal pain and weakness of one-day duration. The patient was recently diagnosed with atypical pancreatic cancer and had an ERCP with stent placement done for obstructive jaundice that day. On presentation, she was afebrile, hypotensive, tachycardic, and tachypneic; physical examination showed jaundice, and diffuse abdominal tenderness with maximum tenderness at the left upper quadrant. Complete blood count showed hemoglobin 7.3 g/dL with white blood cell count 14.1x10^3/ micro L. Comprehensive metabolic panel showed total bilirubin- 13.3 mg/dL, direct bilirubin 12.0 mg/dL, ALT- 96 U/L, AST – 86 U/L, ALP – 285 U/L. Serum lactate on presentation was 10.3 mmol/L. A repeat hemoglobin showed a drop from 7.3 g/dL to 5 g/dL; she received two units of PRBC and two units of FFP. After stabilizing the patient, a computed tomographic (CT) scan of the abdomen and pelvis was done with contrast showed hemoperitoneum around the spleen, and subcapsular splenic hematoma. Interventional radiology was consulted, followed by successful embolization of the distal splenic artery.

Splenic injury after ERCP is a lethal outcome and has not often been described in the literature. The exact mechanism of injury to the spleen during ERCP is not known, it should be considered if there is abrupt abdominal pain, hypotension, or a drop in hemoglobin/hematocrit following ERCP. Management options can vary from conservative management with serial hematocrit monitoring, angiographic embolization as a bridge therapy to splenectomy. A surgical approach involving splenectomy is indicated in hemodynamically unstable patients, whereas splenorrhaphy can be considered in stable patients.
A Rare Case of Chronic Pyelonephritis: Not Just One but Two!

Introduction: Xanthogranulomatous pyelonephritis (XPN) is an uncommon condition that results in destructive chronic inflammation of the kidneys, where granulomatous tissue with lipid-laden macrophages replaces normal renal parenchyma. It often occurs in the setting of staghorn calculi and is rare. Bilateral XPN is even rarer.

Case Description: A 65-year-old African-American male was referred for admission by his PCP because of abnormally low hemoglobin (6.6g/dL). Physical exam showed diffuse abdominal discomfort and distension. Labs showed microcytic anemia, acute renal injury (Creatinine: 4.67mg/dL), elevated AST (49U/L), and CRP (>160mg/L). Urinalysis showed pyuria, and urine culture grew Proteus mirabilis. Renal ultrasound revealed bilateral polycystic kidneys with parenchymal thickening and staghorn calculi, as confirmed later by CT-abdomen. Renal calyces were dilated with a multiloculated appearance ("Bear Paw's Sign"). Staghorn calculi, enlarged polycystic kidneys, and worsening kidney functions warranted bilateral XPN diagnosis. Bilateral nephrostomy tubes were placed, and a fistula with the duodenum was noted during the procedure. CT-Abdomen/pelvis one-week later showed persistent bilateral XPN and staghorn calculi; a fistula between the abscess and the proximal transverse colon was also observed. Right nephrectomy was performed, and the histopathological study confirmed inflammatory processes, although the pathologist could not detect lipid-laden macrophages.

Discussion: XPN affects less than 1% of chronic pyelonephritis cases. It is a predominantly unilateral disease, which makes this bilateral case even rarer. It is often associated with chronic suppurative infection from staghorn calculi. Concomitant infections with E. coli, Proteus mirabilis, or Enterococcus faecalis are common. Presenting symptoms are non-specific: flank pain, fever, and weight loss. Laboratory evaluation may show anemia, elevated CRP, abnormal liver function, and renal failure. Radiologic imaging may depict more specific findings, such as the Bear's Paw Sign. Histopathological study with lipid-laden macrophages can support diagnosis, though not required. Curative treatment in this patient is bilateral nephrectomy and subsequent life-long hemodialysis.
IgA Nephropathy in an African American a Rare Entity

Introduction:
IgA nephropathy, or Berger's disease, is the most common primary glomerulonephritis secondary to inflammatory IgA deposits in the glomeruli, leading eventually to end-stage renal disease (ESRD) and renal failure. It is uncommon in the African American population, with a prevalence of only 3%.

Case Description:
A 43-year-old morbidly obese, African American male with stage-IV chronic kidney disease (CKD), uncontrolled hypertension, and one episode of microscopic hematuria presented to ED with fatigue and bilateral leg swelling (occasional). Physical examination was benign except for elevated BP-188/115, conjunctival pallor, and bilateral pedal edema. Laboratory studies revealed the following: hemoglobin= 5.2ng/L; BUN= 120mg/dL; creatinine= 28.52mg/dL; GFR= 2mL/min; Urinalysis- blood +2, protein +3, RBC 2-5/HPF, protein/creatinine ratio 2.6g/day; and negative hepatitis panel. Autoimmune workup came back negative for ANA, ANCA, Myeloperoxidase Ab, Serine Protease 3 Ab, with normal complement levels. Further investigation unveiled severe renal disease on sonography and IgA nephropathy with collapsing glomerulonephropathy on renal biopsy. The patient received three-units of packed red cells transfusion, and hemodialysis was initiated.

Discussion:
Few cases of IgA nephropathy have been reported among African Americans; still, IgA nephropathy should be considered a differential in the setting of persistent microscopic hematuria and/or proteinuria, including the presence of risk factors such as hypertension. IgA nephropathy diagnosis can only be confirmed by kidney biopsy. Therefore, access to appropriate medical care and timely referral to a nephrologist, plus monitoring of urinalysis in patients with early-stage CKD, can play an essential role in making this diagnosis and in slowing progression to ESRD.
Concurrent Acute Kidney Injury and Hepatic Injury Associated with Pneumonia: Something in the Air?

Introduction:
Legionnaires' disease (LD) classically presents as unilateral lobar pneumonia with low-grade fever, watery diarrhea, elevated hepatic transaminase levels and hyponatremia. Additionally, rhabdomyolysis and renal injury may be present. Legionella pneumonia can be difficult to differentiate from other forms of pneumonia, as the classical features are often absent. LD may occur as a community outbreak after inhaling aerosols from contaminated water or soil, as happened in the Flint Michigan water crisis in 2014-2016.

Case Description:
A 55-year-old lady presented to ED with low-grade fever, tachycardia, and watery diarrhea. Labs were significant for hyponatremia of 119mmol/L, AKI (BUN 60mg/dL, Cr 1.41mg/dL), and elevated liver transaminases (ALT 173, AST 221). Chest-x-ray showed left lower lobe interstitial airspace opacities. She was empirically started on ceftriaxone and doxycycline. When Legionella urine antigen came back positive (serogroup 1), antibiotics were changed to azithromycin, the drug of choice for LD. AKI and hepatic enzyme elevation responded well to treatment with azithromycin with resolution of AKI and decrease in transaminases (Cr 0.56; BUN 7; ALT 113; and AST 96).

Discussion:
AKI is a rare complication of LD, ranging from mild to severe kidney injury. Elevated liver enzymes have also been reported in LD. However, to the best of our knowledge, concurrent AKI and elevated hepatic transaminases due to Legionella infection have not been reported in the literature. Physicians should consider the possibility of LD if a patient has pneumonia, AKI and elevated transaminase levels on presentation. LD can be fatal (up to 30%) - delaying diagnosis and prompt antibiotic treatment may increase mortality.
Isolated Native Tricuspid Valve Endocarditis in an HIV Patient Due to Streptococcus Pneumoniae: A Rare Clinical Phenomenon

Introduction:
Pneumococcal infective endocarditis (IE) is responsible for <1% of IE. Isolated native tricuspid valve endocarditis is rarely seen in the absence of common risk factors like intravenous drug use (IDU) or congenital heart disease.

Case Presentation:
A 47-year-old female presented with complaints of fever and left-sided pleuritic chest pain for a month. She has HIV but had not been compliant with the treatment. She had no history of alcohol or IDU. Vital signs were significant for temperature of 39°C, a heart rate of 110 and blood pressure of 130/85 mmHg. On physical exam, she had grade II/VI systolic murmur in the left lower sternal border, which was new compared to the previous admission examination, and crackles mainly on the left lower side. Laboratory evaluation revealed mild leukocytosis (12.8 k/cumm), CD4 counts of 499 cells/mm3, HIV viral load of 2,730 copies/mL, and negative urine drug screen. A chest X-ray showed left lower lobe pneumonia. She was empirically started on ceftriaxone and azithromycin for community-acquired pneumonia. Blood cultures were positive for Streptococcus pneumonia that was pan-sensitive. Transthoracic echocardiography (TTE) showed 1.9 x 1.5 cm echo density attached to the tricuspid valve leaflet, ejection fraction was 55-60%. Transesophageal echocardiography (TEE) revealed large tricuspid valve vegetation and a perforated posterior tricuspid leaflet with severe regurgitation. The patient refused any surgical intervention, her clinical conditions improved and was discharged on ceftriaxone 2 gm to complete a four-week course.

Conclusion:
Pneumococcal endocarditis is rare and has a poor prognosis. A high index of suspicion should be applied in all patients especially those with alcohol abuse, immunocompromised, previous valve disease, or cardiac anomalies. What made our case interesting is that our patient had right-sided endocarditis, which is much less frequent than left side, and the fact that she denied IDU and had no cardiac anomalies.
Introduction: Wernicke encephalopathy (WE) is a threatening, acute neurological condition caused by thiamine (vitamin B-1) deficiency and requires immediate management to prevent irreversible neurological damage or even death. WE is characterized by the triad of encephalopathy, ophthalmoplegia, and gait ataxia and can progress to Korsakoff syndrome (KS) if untreated, resulting in permanent memory impairment. KS is an extension to WE and reflects the chronic condition with more pronounced neurological involvement, mainly mental deterioration and amnesia. When WE and amnestic KS coexist, they form the Wernicke-Korsakoff syndrome (WKS).

Case Description: A 40-year-old African American female with a history of Crohn's disease and chronic alcoholism presented to ED with altered mental status. She was encephalopathic, febrile, hypotensive, and tachycardic. Labs revealed leukocytosis and high inflammatory markers, hypernatremia, acute kidney injury, and lactic acidosis. Toxicology screen was negative. CT-head was unremarkable. The initial presumptive diagnosis was meningoencephalitis, but CSF analysis only showed elevated protein. Septic workup was negative. CT-chest and abdomen demonstrated multiple lower neck, mediastinum, and retroperitoneal lymphadenopathy. The patient remained encephalopathic despite correction of electrolytes, AKI, and improvement in SIRS criteria. Autoimmune and paraneoplastic workup was negative. A presumed diagnosis of WE was made, which was later supported by head-MRI showing atrophy of mammillary bodies. The patient was started on thiamine 500mg IV Q8H. Treatment partially improved her encephalopathy. A left supraclavicular lymph node biopsy was suggestive of sarcoidosis.

Discussion: Physicians should be attentive to suspect thiamine deficiency, especially in patients with chronic alcoholism, as WE's classical neurological presentation may be absent or masked by other concomitant infectious or metabolic conditions and progression to KS could be evitable with thiamine administration. Early recognition of WE and early administration of thiamine- a safe, inexpensive, and effective treatment, could reverse WE and halt KS, a permanent neuropsychiatric condition.
Introduction: Ethanol causes cardiac injury in a dose-dependent manner leading to dilated alcoholic cardiomyopathy (ACM). ACM can develop from any alcoholic beverage, and the amount of alcohol consumption directly influences the natural course of ACM. Any reduction in alcohol consumption quantity may improve cardiac health, hence this report.

Methods: We searched PubMed and Cochrane databases using the following keywords: "Alcoholic cardiomyopathy," "strategies to reduce alcoholic intake," "alcoholic cardiomyopathy reversibility" We evaluated 20 studies, including systematic reviews and meta-analysis, and observational studies.

Results: Most patients who develop ACM have been drinking >90 g/day of alcohol for >5 years. Habitual alcohol drinkers develop progressive impairment in cardiac function and structure, causing repeated congestive heart failure-CHF episodes, arrhythmias, and progression to death (mortality rate of 10%/year). End-stage ACM with coexistent liver cirrhosis or neurological damage from ethanol has a mortality of >30%/year. Moreover, binge drinking episodes are associated with acute effects such as arrhythmias leading to sudden death and refractory CHF. The most useful measure to control the natural course of ACM is complete abstinence. Controlled drinking of ethanol <60g/day can also achieve significant cardiac function improvement but to a lesser extent than complete abstinence. Even a daily low-dose ethanol consumption coincides with an increased risk of cancer, neurological brain damage. At the same time, high-risk of addiction leads to a lifetime accumulated dose of ethanol that accelerates the attenuation of threshold level needed to develop ACM. Thus, ACM is likely to develop in populations sensitive to ethanol's toxic effects on the heart, like women, and in patients with ethanol-induced cirrhosis, malnutrition, or neurological damage. Hence, the only safe ethanol dose for the heart is zero.

Conclusion: Abstinence from alcohol is the most crucial fundamental solution for reversing alcoholic induced cardiomyopathy with improvement in systolic function and prevention of heart failure and arrhythmias.
Necrotizing Granulomas in a Condition Resembling Sarcoidosis

Introduction: Necrotizing granulomas can be caused by infections, vasculitis, and malignancy. Biopsy and staining for microorganisms are necessary to determine the etiology. Most of the time, infection is the culprit; however, some necrotizing granulomas remain unexplained. In rare cases, necrotizing granulomas could be attributed to sarcoidosis.

Case Description: A previously healthy 29-year-old male presented to the hospital complaining of non-painful masses in his neck and testicles. He denied any travel outside of the State, weight loss, night sweats, or appetite change. There was diffuse non-tender cervical, axillary and inguinal lymphadenopathy on physical exam, which led us to the following differential diagnoses: sarcoidosis, malignancy, histoplasmosis, mycobacteria, or HIV. Scrotal ultrasound showed several hypoechoic masses within the testicles. CT findings revealed mediastinal, hilar, and inguinal lymphadenopathy suggestive of sarcoidosis. Quantitative HCG, alpha-fetoprotein, HIV Ag/Ab combo, syphilis EIA screen, gonorrhea, and chlamydia screen were all negative. Lymph node biopsy showed necrotizing granulomas. Acid-Fast Bacillus and Grocott's methenamine stains were negative for mycobacterial and fungal microorganisms, respectively. There was no morphologic evidence of malignancy. Hence, a presumptive diagnosis of sarcoidosis was postulated. The patient got discharged with a plan to follow-up with Urology and Rheumatology, at which point, he was lost to follow up.

Discussion: There are several causes for necrotizing granulomas, most important of which are: histoplasmosis, tuberculous and nontuberculous mycobacteria, rheumatoid arthritis, granulomatosis with polyangiitis (Wegener), malignancy, and rarely sarcoidosis. Although sarcoidosis usually presents with noncaseating granulomas and rarely presents with necrotizing granulomas, if other causes are ruled-out, sarcoidosis can be proposed as a diagnosis of exclusion. Sarcoidosis is mostly self-limiting, and most patients have spontaneous resolution of symptoms, and at times, a corticosteroid is the recommended treatment. If this patient were to develop worsening symptoms, cultures should be drawn to investigate rare fungi' potential presence as an etiology.
Acute PE and Stroke from Paradoxical Embolization, a Management Paradox: Damned if You Do, Damned if You Don’t!

Introduction: VTE occurs in 0.066% of pregnancies worldwide. The arterial thromboembolism risk is increased from 3 to 4-fold and the risk of venous thromboembolism is five times higher in a pregnant that in a non-pregnant woman [1]. These complications are closely related to physiological changes during pregnancy [2].

Case Description: 37-year-old female at 6 weeks gestation presented with a right MCA stroke, however, she was out of the tPA window. CT-thorax with contrast demonstrated saddle PE with right heart strain. Paradoxical embolization was suspected and supported by a 2D echo bubble study. Additionally, a bilateral venous duplex ultrasound was negative for DVT. IV heparin drip was discontinued on day 4 after a repeat CT head revealed hemorrhagic conversion. Heparin was restarted after hemorrhage stabilized on day 6. Seven days after admission, repeat bilateral venous duplex revealed acute DVT. She acutely deteriorated into cardiac arrest and expired after ACLS.

Discussion and Conclusion: The management of PE with stroke from paradoxical embolization is especially challenging in pregnancy. The risk of intracerebral hemorrhage from anticoagulation must be carefully balanced with the risk of hemodynamic collapse from massive PE. This presentation is not uncommon in pregnancy and claims 70,000 lives per year[3] worldwide. Unfortunately ischemic strokes are as prevalent as hemorrhagic strokes in pregnancy adding another layer of complexity[4]. This case intends to explore the possibility of early intervention with closure of PFO or placement of an IVC filter.
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Concomitant Intravascular Volume Expansion and IV Diuretics to Reduce Complications and Improve Outcomes of Acute Heart Failure

Introduction: Acute heart failure (AHF) is associated with significant in-hospital morbidity and mortality. Intravenous (IV) diuresis is required in acute heart failure; however, it is challenging in the setting of hypotension due to low cardiac output. Current guidelines indicate use of short term inotropic agents with diuretics, but are associated with adverse effects such as cardiac arrhythmias. We performed an evidence based review to identify outcomes of concomitant IV diuretics and IV fluid administration in acute heart failure.

Methods: A literature search was done using Pubmed and Cochrane databases from 2011 to 2021. Any articles studying fluid resuscitation with loop diuretics as an alternative therapy to only loop diuretics fit the inclusion criteria. The keywords used were “acute heart failure”, “diuretics”, and “normal saline”. 5 studies were selected for review after applying inclusion and exclusion criteria.

Results: Overall, 3 systematic reviews and meta-analyses and 2 prospective cohort studies were included. All 5 studies found a statistically significant shorter length of hospital stay in patients treated with both hypertonic saline solution (HSS) and furosemide versus furosemide alone. All 3 systematic reviews and meta-analyses reported a statistically significant lower mortality, decreased hospital re-admissions, lower rise in serum creatinine, and a higher weight loss in the HSS group compared to controls. One systematic review also reported a statistically significant higher increase of natriuresis in the HSS group compared to controls and did not report a significant difference in BNP levels between both groups.

Conclusion: According to our evidence based review, concomitant use of HSS as an agent for intravascular volume expansion and IV diuretics has shown significant improvement in length of stay, mortality, and cardio-renal syndrome in patients with acute heart failure. Further RCTs establishing similar outcomes and studies comparing the administration of HSS with other current advanced heart failure therapies are required.
Pure Autonomic Failure: A nightmare of Blood Pressure Control!

Introduction: Pure Autonomic Failure (PAF), a neurodegenerative disorder caused by α-synuclein accumulation in the brain and is characterized by isolated autonomic nervous system dysfunction in the absence of other neurological abnormalities. Similar autonomic dysfunction with α-synuclein accumulation can be present in Lewy bodies’ dementia, Parkinson's disease, multiple system atrophy, and rarely in Multiple Sclerosis. PAF is challenging as it is a diagnosis of exclusion. Orthostatic hypotension is the hallmark of PAF but is often consorted by supine hypertension; concurrent conditions require adequate symptomatic relief to prevent complications, as there is no cure or reverse for this rare condition.

Case Presentation: A 31-year-old AA female with a history of uncontrolled hypertension since childhood leading to end-stage renal disease presented with an elevated BP of 240/140mmHg, despite reported adherence to home medications and regular dialysis. She also reported episodes of pre-syncope on standing. She denied tremors, gait disturbances, bowel or urinary incontinence. Physical examination was positive for orthostatic hypotension. Workup for secondary hypertension, including angiotensin/renin ratio, renal artery scan, and thyroid and adrenals functions, were performed in the past and were unremarkable. Pre-syncope workup was never pursued. Her supine BP remained uncontrolled and Head-up Tilt Testing showed severe orthostatic hypotension symptoms. Vasodilatory antihypertensives were discontinued, and her supine hypertension was controlled with clonidine, labetalol, and lisinopril. She was kept in reverse Trendelenburg position while sleeping and lying down. Midodrine PRN was prescribed to prevent symptomatic orthostatic hypotension during the daytime.

Discussion: Managing PAF is challenging, primarily because it seeks to achieve BP control to prevent supine hypertension and orthostatic hypotension. Management includes dietary modifications, cross-leg maneuvers before standing, and lower limb compression stocking. Daytime medications such as midodrine, fludrocortisone, and pyridostigmine are recommended for postural hypotension and non-vasodilatory antihypertensives at night to manage supine hypertension along with reverse Trendelenberg position when sleeping.
**Silent Gangrenous Cholecystitis in an Untreated Diabetic Patient**

**Introduction:**
Gangrenous cholecystitis (GC) is a deadly complication of progressive acute cholecystitis, characterized by inflammation leading to ulcerations or ischemic necrosis of the gallbladder wall. It has an atypical presentation in diabetic patients and puts them at risk for rapid clinical deterioration (sepsis or wall perforation) and high mortality.

**Case Presentation:**
A 65-year-old African American female with a history of untreated diabetes mellitus presented to ED complaining of dizziness, and non-radiating heavy chest pain of a few minutes duration. Lab workup showed elevated troponin (805ng/L) and elevated D-dimer (5.56pg/mL). CT- Chest was done to rule-out pulmonary embolism and revealed an incidental hepatic cyst, measuring 9cm. The daily abdominal examination was benign; however, further testing revealed an abnormal liver panel, elevated WBC count, and high triglycerides. Blood culture turned positive for Streptococcus anginosus. Magnetic Resonance Cholangiopancreatography-MRCP was done to find the septic focus revealed gangrenous cholecystitis with multiple gallstones and a hepatic abscess. Due to multiple comorbid conditions, cholecystectomy was not recommended. The patient underwent therapeutic decompression with image-guided percutaneous cholecystostomy and hepatic drainage tube placement. The fluid culture showed few gram-positive cocci, for which the patient received IV Unasyn for 6-weeks.

**Discussion:**
To our knowledge, this is a decidedly rare case of GC presenting with a benign abdomen in a patient with untreated diabetes. As clinicians, it is imperative to have a high index of suspicion for gangrenous cholecystitis in a patient with leukocytosis, abnormal liver panel, and more importantly, with untreated diabetes as it has a high mortality rate of 22%.
Could Fluconazole Induce Mastitis in a Non-Lactating Woman?

Introduction:
Mastitis refers to inflammation of the breast tissue with or without infection. It can be lactational or non-lactational, and it can occur postmenopausal. Fluconazole is known to cause some notorious side effects such as alopecia, and GI symptoms-nausea, vomiting; and to a lesser extent, breast pain in some cases. We present a rare case of fluconazole induced mastitis in a middle-aged woman.

Case Description:
A 46-year-old African-American woman with a medical history of type-1 diabetes presented to the hospital complaining of multiple vomiting episodes. On exam, she was hypotensive. Labs showed glucose 859mg/dL, Anion gap 36mEq/L, WBC 36x10⁹/L consistent with the diagnosis of diabetic ketoacidosis-DKA. She was treated with IV-insulin and empiric antibiotics for possible Boerhaave’s syndrome with pneumomediastinum and subcutaneous emphysema due to hyperemesis induced by DKA. During her hospital stay, she was evaluated for melanotic stool. Her hemoglobin dropped from 11.6g/dL to 8.2g/dL. EGD showed severe monilial esophagitis with pseudo-diverticulosis formation. The patient was started on Mycostatin and fluconazole 200mg PO. After one dose of fluconazole, she developed bilateral breast pain, worse on the right. The right breast was swollen, erythematous, warm, and tender, but no galactorrhea, nipple discharge, or palpable lumps. The left breast was mildly swollen and tender. A pregnancy test was negative. Fluconazole was discontinued, and IV voriconazole was started. The patient’s breast symptoms improved following discontinuation of fluconazole.

Discussion:
Initiation of fluconazole was associated with mastitis signs and symptoms, which resolved following discontinuation of fluconazole. Mastitis is rare in non-lactating women, but some literature noted that diabetic women could be susceptible to developing mastitis outside the lactation or postmenopausal setting. As clinicians, it is essential to rule out fluconazole induced mastitis in patients who develop breast symptoms after initiation of fluconazole.
Acute Limb Ischemia in a COVID-Positive Patient

Introduction:
Patients with Coronavirus disease-2019 (COVID-19) mainly present with acute respiratory dysfunction but can cause complications across multiple organ systems. Covid-19 is also associated with a hypercoagulable state leading to an increased incidence of thromboembolism. We are presenting a case of acute limb ischemia in a patient with COVID-19 despite therapeutic anticoagulation.

Case Description:
A 73-year-old female, with a medical history of chronic obstructive pulmonary disease, stage 4 chronic kidney disease, hypertension, and hyperlipidemia, presented with a 1-week history of intermittent fevers, dry cough, and shortness of breath. She was found to be in acute hypoxic respiratory failure and tested positive for Covid-19. She was started on heparin infusion empirically given her high D-dimer but was switched to prophylactic enoxaparin for a day as it was speculated that the elevated D-dimer was likely reactive. The patient’s respiratory status and renal function deteriorated through the hospital stay, necessitating mechanical intubation. She was switched back to therapeutic anticoagulation after one day. Slight discoloration of her right leg was attributed to being on vasopressors, but the creatinine phosphokinase (CPK) trended up. The entirety of her right leg became mottled with loss of palpable pulses while the CPK rose to >8000. An arterial duplex showed extensive arterial and venous occlusions of both legs. Since she was considered a poor candidate for surgical intervention, the family decided to change the goals of care to the comfort of the patient, and she passed shortly after.

Discussion:
Covid-19 leads to a hypercoagulable state, which can trigger arterial and venous thromboembolism. A high index of suspicion is warranted for acute limb ischemia in patients with Covid-19. Evaluation and management of the hypercoagulability in these patients is a critical component as it has a significant impact on the morbidity and mortality of these patients.
Propofol Induced Torsades to Pointes and Ventricular Fibrillation Arrest

Introduction:
Propofol has been shown to cause QT corrected (QTc) prolongation. However, it has been rarely reported to cause significantly prolonged QTc inducing fatal arrhythmias.

Case:
We present a case of a 45-year-old gentleman with a past medical history non-ischemic dilated cardiomyopathy with reduced ejection fraction (secondary to alcohol abuse), thoracic aortic aneurysm, hypertension and hyperlipidemia who presented to the ED intoxicated and complaining of abdominal pain. EKG at presentation showed a QTc of 475ms. The patient was extremely agitated, with acute hypoxic respiratory failure and was ultimately intubated. He was sedated with propofol and dexmedetomidine. EKG obtained 24 hours and 48 hours after initiation of sedation, showed a QTc of 520ms and 760ms respectively. Overnight, the patient went into polymorphic ventricular tachycardia and subsequently into ventricular fibrillation (V-fib) arrest. The patient was defibrillated and resuscitated successfully. The Propofol drip was discontinued. EKG after 24 and 48 hours showed a QTc of 660ms and 476ms respectively. During in hospital stay the lowest recorded values for potassium, magnesium and calcium were 3.0, 2.3 and 8.8 mg/dl.

Discussion:
Propofol has been implicated in causing QTc prolongation. However, only one case of propofol induced V-fib arrest has been reported in the literature. Propofol is considered a relatively safe drug and is very commonly used in the intensive care unit setting. Nonetheless, it has been shown to cause QTc prolongation especially in patients with a borderline prolonged QTc at baseline. Though no threshold value exists, significantly prolonged QTc interval can lead to Torsades de Pointes and ultimately V-fib. Further research is still required to evaluate the clinical significance and a cause-and-effect relationship between propofol and QTc prolongation. Closer QTc interval monitoring in patients on propofol should also be considered. Moreover, a cutoff value is needed to guide when to avoid anesthesia with propofol.
Trastuzumab Emtansine-Induced Pulmonary Toxicity: A Diagnosis Not to be Missed!

Antibody-drug conjugates (ADCs) such as trastuzumab emtansine (T-DM1) have been an exciting advent in oncology, targeted therapy that can discriminate between healthy and pathologic tissues. Besides their efficacy, these agents can also generate immune-mediated adverse events. Pulmonary toxicity can be a life-threatening sequela of these agents that clinicians need to be aware of as early recognition and management may be lifesaving. Herein, we report a case of a 68 year old female with history of ER/PR positive and HER-2 positive breast cancer on the sixth cycle of T-DM1 who presented with sudden-onset fevers and shortness of breath. She was hemodynamically stable but subsequently developed a fever of 103 F and became hypoxic requiring 5 L/min oxygen supplementation by nasal cannula. Chest radiograph reported diffuse interstitial markings. Her blood count and chemistries was notable only for thrombocytopenia and lymphopenia. Inflammatory markers including D-dimer were elevated. COVID and influenza A/B swabs were negative. Chest computed tomography pulmonary angiogram demonstrated peripheral and basilar patchy airspace and ground glass opacities. Bronchoscopy was unremarkable. Given the negative infectious workup, there was a high clinical suspicion for T-DM1-induced pulmonary toxicity. High-dose intravenous corticosteroids were administered which provided symptomatic relief along with marked improvement of her hypoxia. She was discharged on a prednisone taper with oncology follow up to discontinue T-DM1.

There are few cases of T-DM1-induced interstitial pneumonitis reported in the literature. With the increasing popularity and use of T-DM1, T-DM1-induced pulmonary toxicity is also on the rise. Awareness of radiological patterns and clinical manifestations of these adverse events is crucial as early recognition and management with CT scan and bronchoscopy, along with administration of high-dose intravenous corticosteroids may be lifesaving.
Rare Case of Coexisting Thin Membrane Basement Disease with Minimal Change Disease

Introduction: Thin basement membrane disease (TBMD) is characterized by diffuse thinning of the glomerular basement membrane and minimal proteinuria. We present a case of TBMD with concurrent nephrotic-range proteinuria as part of minimal change disease (MCD).

Case Description: A 20-year-old female presented with facial and peripheral edema, progressing to anasarca, and weight gain of 10 pounds within a few days. She had 3 g of proteinuria, microscopic hematuria, and normal serum creatinine. Her only medication was ibuprofen. Given nephrotic range proteinuria, NSAID-induced vs. idiopathic minimal change disease (MCD) was in the differential. The patient was started on steroids before the biopsy was done. The patient went into remission in 4 weeks; therefore, steroids were rapidly tapered. Immunological testing, including anti-PLA2r antibody, was negative. Three months later, the patient had recurrent proteinuria > 2 g with bilateral peripheral edema. The patient underwent a repeat renal biopsy, which showed diffuse foot process effacement, consistent with MCD, and a diffusely thin basement membrane consistent with thin basement membrane disease (TBMD). The patient was then initiated on a 16-week course of prednisone, with a slow taper over six months.

Discussion: Our patient represents a unique case of TBMD with heavy proteinuria due to MCD, which there have only been about three cases of so far. Persistent hematuria in the absence of other comorbidities should prompt a differential, including TBMD. The presence of concurrent nephrotic-range proteinuria should raise concern for FSGS and MCD. Patients should undergo a renal biopsy before treatment with glucocorticoids since MCD is a steroid-responsive disease and can be missed after a short course of steroids.
A Case of Gemella bergeri Endocarditis and Vertebral Osteomyelitis from Dental Caries

Introduction:
Gemella bergeri is a Gram-positive coccus which is an oral and upper respiratory tract commensal. There have been only a few case reports of it causing endocarditis. We report a case where the infection seeded to the vertebrae causing diskitis and osteomyelitis (OM).

Case Presentation:
A 63-year-old male presented with a 3-week history of worsening lower back pain and lower extremity weakness. He also reported chills, night sweats, and fatigue. Physical exam was significant for carious teeth, splinter hemorrhages in his fingernails, a diastolic murmur over the aortic area, and tenderness over the lumbar spine. Rectal tone was normal. Magnetic resonance imaging of the lumbar spine showed discitis, OM, and epidural abscess at the L4-L5 vertebral levels. Blood cultures were drawn and the abscess was aspirated. A transesophageal echocardiogram demonstrated mobile echodensities on the mitral and aortic valves and a flail aortic leaflet. The blood and abscess fluid cultures isolated Gemella bergeri. The patient was discharged on ceftriaxone for a total of six weeks from the first negative blood culture. Before the course was complete, he was readmitted for an acute exacerbation of heart failure and required transfer to a tertiary care facility where he underwent mechanical aortic valve replacement and mitral valve patch repair. Prior to surgery, he underwent tooth extraction because of concerns of odontogenic Gemella infection. He was switched to oral linezolid for the rest of his antibiotic course.

Discussion:
Gemella bergeri is an uncommon cause of endocarditis. Damaged or abnormal cardiac valve morphology, poor dentition, and intravenous drug use are considered to be risk factors. Gemella is susceptible to penicillins, cephalosporins, and aminoglycosides. To our knowledge, this is the first reported case of diskitis and vertebral OM from G. bergeri.
INTRODUCTION: Relapsing polychondritis (RPC) is a rare disease with potential of multi-organ involvement and a variable disease course. This is a case of a patient with the rare disease of RPC in conjunction with Sweet Syndrome and rheumatoid arthritis (RA).

CASE: The patient is a 58-year-old male who presented with an erythematous, pruritic palmar rash, ear swelling, and red eyes. He had a history of seropositive RA with no prior treatment, hidradenitis suppurativa, pancytopenia, COPD and hypertension. Cephalexin and prednisone did not offer relief. Physical exam identified bilateral conjunctival injection consistent with episcleritis, bilateral pinna swelling, and erythematous patches of the hands. Basic lab work found macrocytic anemia (Hg 7.5, MCV 113.9) and normal renal function. Extensive rheumatologic and inflammatory workup was done, and the patient was found to have elevated CRP (5.6), ESR (94), rheumatoid factor (25), IgG (2,107), and IgA (680). Myositis and vasculitis workup was unremarkable. Myelodysplastic syndrome with RPC is associated with a poorer prognosis, thus SPEP was done which did not show evidence of monoclonal gammopathy. Skin biopsy of the palms revealed neutrophilic dermatosis. This finding, along with the auricular chondritis and episcleritis raised suspicion for relapsing polychondritis. The hospital stay was complicated by pneumonia and respiratory distress. Therefore, a computed tomography of the airway was ordered, which revealed a Saber Sheath trachea. The patient was successfully treated with systemic and topical (ocular) glucocorticoids, as well as antibiotics for the pneumonia.

DISCUSSION: This is a rare case of combined relapsing polychondritis, neutrophilic dermatosis and rheumatoid arthritis. This patient had auricular, ocular, and tracheal involvement, the latter of which likely played a role in development of pneumonia. Neutrophilic dermatosis (Sweet Syndrome), is also associated with RPC. Relapsing polychondritis should be suspected with pain, redness, and swelling of the auricle, without signs of infection.
Secondary Plasma Cell Leukemia: A Rare Presentation

Introduction:
Plasma cell leukemia (PCL) is a rare variant of multiple myeloma (MM). Historically, primary PCL has been far more common than secondary PCL. Presenting signs can include those seen in MM and those seen in other leukemias. Extramedullary involvement may also occur and usually presents as subcutaneous masses.

Case:
Our patient is a 55 year old with past medical history of IgG lambda multiple myeloma diagnosed 4 months prior who is receiving weekly CyBorD and presented with bilateral eye swelling. He stated that he first noticed swelling around both eyes two weeks prior. Lab work up revealed beta-2-glycoprotein elevated at 6.9, LDH elevated at 2069. Monoclonal protein evaluation confirmed IgG lambda monoclonal protein and free lambda monoclonal protein (1.6 and <0.1 g/dL) present in the mid and fast gamma regions. Free light chain evaluation revealed 0.7 kappa light chain, 1765 lambda light chain, FLC ratio of 0. A CT of the showed enlarged extraocular muscles and biopsy showed highly atypical plasmacytoid population which was plasmablastic. Flow cytometry showed a clonal population of CD19 negative, CD56 positive plasma cells. Bone marrow biopsy was notable sheets of plasma cells occupying 90-100% of the bone marrow. He was diagnosed with refractory IgG Lambda plasma cell leukemia with extramedullary involvement and was started on Decadron 40mg IV daily. He also received VTD-PACE with palliative radiation.

Discussion:
Secondary PCL is a rare manifestation of progressive disease. The original diagnostic criteria for PCL required 20% plasma in the periphery. Our patient had only 13% but given his other manifestations, immunohistochemical staining, and recent chemotherapy this diagnosis was made. Involvement of the extraocular muscles by PCL is also a particularly uncommon extramedullary manifestation, with a prevalence of just 0.3% and carries a poor prognosis. Our patient unfortunately passed away 5 months after this diagnosis despite aggressive treatment.
Anti-Glomerular Basement Membrane Disease, Should We Treat or Not Treat?

Anti-GBM (Goodpasture) disease is a rare small vessel vasculitis characterized by antibodies directed against the basement membrane of the alveoli and glomeruli (1). The possibility of rapidly progressive Glomerulonephritis and Acute renal failure makes it imperative to diagnose and treat Anti GBM.

A 66-year-old man with psoriasis and hypertension presented to our institution with a two-week history of fevers, chills, and decreased urine output. Physical exam showed lower extremity edema. Lab work revealed an acute kidney injury with a creatinine of 11 from a baseline of 1, hyperkalemia, and metabolic acidosis. Urinalysis was positive for hematuria and mild proteinuria. He was urgently initiated on hemodialysis for fluid overload and solute removal. Further workup revealed elevated anti-glomerular basement membrane antibodies (anti-GMB ab) at a level higher than 680 IU/ml, and a negative autoimmune and infectious workup. Kidney biopsy with light microscopy showed diffuse sclerotic glomeruli with cellular crescents and fibrinous necrosis. Immuno-histology staining was positive for linear deposits of Immunoglobulin-G and complement (C3) consistent with anti-GBM glomerulonephritis with a diffuse crescentic and necrotizing pattern. He received high dose steroids, five sessions of plasmapheresis, in addition to ongoing HD. There were no signs of renal recovery on discharge. However, a two-week course of oral cyclophosphamide was added on discharge along with a steroid taper. Fortunately, he was deemed to be a transplant candidate, and pre-transplant workup was initiated.

Although treatment includes plasmapheresis and immunosuppression. Treating patients who present with dialysis-dependent kidney failure without pulmonary hemorrhage remains debatable due to the low likelihood of renal response (2). However, a short trial of plasmapheresis and immunosuppressive therapy was applied in our patient despite the extent of crescentic GN and dialysis given the acuity of his presentation, which might predict injury reversibility, lack of comorbidities, and ability to tolerate immunosuppression to maximize the chance of renal response(3).
A 55-Year-Old Male Presenting With a Lower Extremity Rash: A Case of IgA Nephropathy

Introduction:
IgA nephropathy is mesangial deposition of IgA, for which is established by kidney biopsy, and may either present in primary or secondary forms related to other medical conditions. Later in its course, patients may present with decreased glomerular filtration rate (GFR) and proteinuria. Leukocytoclastic vasculitis is a specific type of inflammatory small-vessel vasculitis that may present secondary to IgA. This report will discuss a case of secondary IgA nephropathy as well as concomitant leukocytoclastic vasculitis in a patient with reactivated hepatitis C.

Case Presentation:
A 55-year-old male with a history of hepatitis C status post treatment with Harvoni and alcoholic cirrhosis was transferred to our hospital for a bilateral lower extremity rash, as well as progressively worsening kidney function. He was found to have IgA nephropathy, as seen on kidney biopsy as well as skin biopsy showing leukocytoclastic vasculitis. Further investigation revealed the patient’s Hepatitis C RNA to be significantly elevated and serum cryoglobulin levels to be positive, suggestive that a reactivated hepatitis C that was previously in sustained virologic response was the cause of the IgA nephropathy and possibly the leukocytoclastic vasculitis.

Conclusion:
We present a rare case presentation of secondary IgA nephropathy with concomitant leukocytoclastic vasculitis in the setting of recurrent Hepatitis C infection. We hypothesize that this patient had a secondary type of IgA nephropathy and a leukocytoclastic vasculitis that was due to reactivation of Hepatitis C and underlying cirrhosis.
Mysterious Hiccups: An Unusual Manifestation of Acute Appendicitis

Acute appendicitis is a clinic diagnosis. It typically presents with periumbilical pain that migrates to the right iliac fossa, and is associated with anorexia, nausea, and vomiting. When the diagnosis is delayed, it can lead to complications such as perforation. Our case involves a 50-year-old male with a history of GERD, who presented with a three-day history of epigastric pain. The patient initially presented three days prior where his evaluation was unremarkable except for a liver hemangioma, found on a CT Liver Protocol. The patient was discharged on antacids and ranitidine, but re-presented the following day with persistent hiccups along with recurrent pain. Of note, the patient was repeatedly self-inducing vomiting to temporarily relieve his hiccups. This time, labs were significant for leukocytosis (18 K/uL) and elevated AST (52 IU/L). His hiccups were aborted with a Valsalva maneuver and he was managed symptomatically with IV fluids, ondansetron, and famotidine. The following day he presented a third time with right lower quadrant abdominal pain and a physical exam that was concerning for peritoneal irritation. Imaging was suggestive of a perforated tip appendicitis and he was emergently treated with a laparoscopic appendectomy and IV antibiotics.

Our patient had an atypical presentation of perforated appendicitis that was diagnosed after his third presentation to the emergency room. On initial presentation, the evaluation was unremarkable. The patient’s presentation progressed to include hiccups and a leukocytosis. Hiccups can result from any irritation of the vagus or phrenic nerves, which are part of the efferent limb of the hiccup reflex. Irritation of this pathway leads to diaphragmatic contraction. In this case, it was a manifestation of an intra-abdominal process. Therefore, it is important for clinicians to maintain a broad differential in patients that present multiple times with similar symptoms.
A Suspected Primary Lung Cancer Turns Out to be a Rare Pleural Epithelioid Hemangioendothelioma

Introduction:
We are reporting a case of Epithelioid hemangioendothelioma (EHE) that manifested as a pleural effusion. Our patient ultimately was diagnosed late in her course, had a broad negative work up, and required a pleural biopsy to obtain the diagnosis.

Case:
Our patient is a 48-year-old female with medical history significant for hypertension who presented multiple times with recurrent intractable chest wall pain. Her history was significant for loss of appetite and weight loss of about 20 lbs in the last 2 months. She had an unremarkable laboratory and imaging work up and eventually had thoracentesis performed, which was significant for an exudative, non-malignant pleural effusion. She had a bronchoscopy with biopsy performed which was negative for malignancy. Due to recurrent symptoms a PET scan revealed a hypermetabolic 1.9 cm x 3.9 cm left lingular pulmonary mass and metastatic nodular pleural thickening in the left hemithorax with small left pleural effusion concerning for a lung malignancy with pleural metastatic disease. A pleural biopsy revealed EHE. Due to the unrespectable tumor, she was started on palliative radiotherapy for five sessions and was later started on adriamycin, ifosfamide and mesna. Unfortunately our patient passed away.

Discussion:
EHE is a rare vascular tumor with multiple subtypes. Only 96 cases of EHE have been reported, of which 5 presented with pleural effusions. This tumor can arise in the bone, liver, soft tissue, and pleura. Although etiology is not well understood, it has been reported that over 90% of EHEs express oncogenic WWTR1-CAMTA1 fusion. This results in a transcriptional program that is resistant to anoikis and oncogenic transformation. We would like other internists to be aware of this diagnosis: presentation, poor prognosis, treatment.
A Rare Case of Hemophagocytic Lymphohistiocytosis

Hemophagocytic Lymphohistiocytosis (HLH) is severe hyper-inflammatory syndrome emerging from a deregulated immune response. It is classified as primary or secondary, with the latter being most commonly associated with malignancy. HLH carries a high rate of mortality and understanding early clinical findings are imperative for diagnosis, early treatment, and aggressive supportive care to prevent further complications. This report highlights a rare case of primary HLH in an adult male with the most common initial symptoms. A 48-year-old male with past medical history of iron-deficiency anemia presented to the hospital with intermittent fevers, weight loss, and fatigue that had been ongoing for six months. He was febrile to 39.4°C. Physical examination revealed cachexia and non-palpable purpuras on the palmar surfaces of his bilateral hands. Laboratory findings with significant for pancytopenia, elevated inflammatory markers (ferritin 83363, lactate dehydrogenase 3499), cholestasis (direct bilirubin 0.3, alkaline phosphatase 234), and elevated triglycerides 313. Imaging and peripheral smear were unremarkable. Bone marrow biopsy was pursued with evidence of the classical pattern of hemophagocytosis from macrophages. Patient was started on induction therapy with eight weeks of etoposide and dexamethasone with improvement in symptoms.

Primary HLH is increasingly rare in adults and early suspicion based on clinical findings is important in decreasing mortality. Per the HLF-2004 trial, fever (>38.5), pancytopenia, hypertriglyceridemia, elevated ferritin (>500), and hemophagocytosis on bone marrow should raise concern. Per the H-score, this carries 93-96% probability of the disease. Induction therapy consists of etoposide and dexamethasone with improvement in symptoms.
Recurrent Symptomatic Anemia with Arteriovenous Malformations and Epistaxis

Hereditary hemorrhagic telangiectasia (HHT) is a rare autosomal dominant condition in which telangiectasias and arteriovenous malformations (AVMs) are located in multiple organ systems. This case presents a 52-year-old woman with a 10-year history of recurrent symptomatic anemia requiring biweekly transfusions who presented with an increasing amount of fatigue and dyspnea for one week. She has a history of intermittent, self-resolving epistaxis, duodenal AVM bleeds, menorrhagia, and blood loss during labor requiring multiple transfusions. She is unaware of her paternal medical history and mothers was unremarkable for hematologic conditions. Her exam was significant for gingival and lower lip telangiectasias, pale inner eyelids and fingertips, and a holosystolic murmur. Her CBC showed microcytic anemia with elevated red cell distribution and initial hemoglobin level 3.1 that responded appropriately to 3 units of packed red blood cells. Upper endoscopy revealed multiple non-bleeding angiodysplastic lesions in the duodenum and jejunum and 27 individual AVMs were treated with argon plasma coagulation. Transthoracic echocardiogram revealed a small patent foramen ovale with predominantly right to left shunting across the atrial septum. Based on her history of recurrent epistaxis, telangiectasia, and AVMs, she has clinically been diagnosed to have HHT based on 3 of the 4 Curaçao criteria. Her management consisted of genetic testing, biweekly hemoglobin monitoring and iron supplementation, and follow-up with Hematology, Gastroenterology to screen for AVMs, and Cardiology. Her right to left shunt bypasses the pulmonary capillary bed and increases her risk of developing paradoxical emboli to the brain which can lead to stroke. Given the rarity of HHT and the multi-organ AVMs, patients benefit from a multi-system approach to manage systemic complications of HHT. Furthermore, their family members should be screened for AVMs.
Flash Pulmonary Edema and Cardiac Tamponade in Scleroderma Renal Crisis

INTRODUCTION
Scleroderma renal crisis is a life-threatening manifestation of systemic sclerosis and can result in serious complications such as pericardial effusion, tamponade, and flash pulmonary edema.

CASE
A 39-year-old man with history of discoid lupus and scleroderma formerly treated with plaquenil and methotrexate presented with generalized myalgia, abdominal pain, and nausea. He was hypertensive on admission with elevated creatinine to 3.23 from a baseline of 0.80. Urinalysis showed microalbuminuria and renal ultrasound revealed increased echogenicity in the left kidney suggestive of medical renal disease. Given oliguria and AKI, renal biopsy was performed and showed thrombotic microangiopathy with proliferative endarteritis and chronic glomerulopathy consistent with scleroderma renal crisis. Patient was started on captopril therapy and intermittent hemodialysis was initiated. Subsequently, he developed worsening shortness of breath with evidence of pericardial effusion with tamponade physiology on echocardiogram and required pericardiocentesis. He was readmitted a week after discharge with evidence of flash pulmonary edema and persistent hypertension requiring urgent dialysis. We present a case of severe systemic sclerosis presenting with rapid onset life-threatening complications including scleroderma renal crisis, pericardial effusion, and flash pulmonary edema.

DISCUSSION
Scleroderma renal crisis is rare yet life-threatening complication found in 5-20% of diffuse systemic sclerosis. The incidence of renal crisis is higher earlier in the disease and carries with it the risk of acute kidney injury requiring renal replacement therapy. There are few case reports of scleroderma renal crisis concurrently complicated by pericardial effusion with tamponade physiology as both are very rare complications of systemic sclerosis. Though known to be potential complications of systemic sclerosis, early identification and treatment of pericardial effusions, flash pulmonary edema, and acute renal failure may prevent significant morbidity and mortality in these patients.
A Case of Acute Renal Failure Associated with Diffuse Infiltrative Lymphocytosis Syndrome

CASE
A 39-year old female with a history of HIV adherent to HAART for three years presented with a 2-week history of lower extremity edema. On presentation, she had significant proteinuria (112 mg/dL) with acute renal failure (creatinine 3.6, GFR 21). Urine light chains were consistent with a glomerular proteinuria and imaging showed diffuse lymphadenopathy. CD4 count was 727. She was diuresed with improvement in edema, but kidney function continued to worsen. Renal biopsy showed a lymphocytic infiltrate with associated lymphocytic tubulitis composed of predominantly CD8 (+) T-cells consistent with a diagnosis of diffuse infiltrative lymphocytosis syndrome (DILS). She was started on steroid therapy in addition to HAART. We present an atypical case of diffuse infiltrative lymphocytosis syndrome causing acute renal failure in an HIV-positive patient.

DISCUSSION
Diffuse infiltrative lymphocytosis syndrome (DILS) is a rare multisystemic syndrome that can manifest in untreated HIV-positive patients and those with normal CD4 counts. It is a CD8 (+) T-cell lymphocytosis associated with CD8 (+) T cell infiltration of multiple organs. DILS has been described as a Sjögren-like disease associated with sicca, lymphadenopathy, bilateral parotid gland enlargement/parotiditis, and extra-glandular organ involvement (including the nervous system, kidneys, gastrointestinal tract, and lungs). There have been rare cases of acute interstitial nephritis. The renal manifestation has been described with tubulo-interstitial disease, mild-grade proteinuria, and enlarged kidneys. Renal biopsy is often diagnostic. Treatment consists of steroid therapy (with improvement of renal failure) when organ infiltration is suspected as well as HIV anti-retroviral therapy.

CONCLUSION
Although there are many causes of acute renal failure in HIV-positive patients, careful attention should be paid to history and other possible syndromes in such patients. There are a few reported cases of renal manifestations of DILS; therefore, clinical suspicion should remain high in HIV-positive patients with acute renal failure regardless of CD4 count.
An Atypical Presentation of CJD Masquerading as Hashimoto Encephalopathy

Altered mental status, or toxic metabolic encephalopathy, is a frequently used term in the inpatient setting and can encompass mental status changes in all forms. One important cause of altered mental status is rapidly progressive dementia.

A 73-year-old male with a history significant for hypertension, hyperlipidemia and distant history of alcohol abuse presented with sub-acute on chronic mental status changes and progressive weakness. His symptoms first presented as confusion and memory changes that progressed to gait instability and inability to perform activities of daily living. On presentation to the hospital, patient was altered, A0x0, and unable to speak. Initial labs remarkable for TSH 56 uIU/mL, free T4 0.26 ng/dL, + antiTPO and anti-thyroglobulin. Patient’s mental status changes were originally attributed to primary hypothyroidism/Hashimoto’s encephalopathy and he was treated with levothyroxine, IVIG and steroids. However, patient did not improve with those treatments. Physical exam notable for encephalopathy and aphasia with startle response, decreased strength in all extremities and unable to perform coordination exams. MRI revealed mild asymmetric cortical restricted diffusion involving the left medial and posterior parietal cortex. Lumbar puncture was done which revealed positive RT-QuIC, positive 14-3-3 protein and positive T-tau protein. These findings are associated with >98% likelihood of prion disease. Throughout his hospitalization, patient continued to decline and patient was discharged to home hospice per his family’s wishes.

Our patient presented with findings of rapidly progressive dementia with a positive Creutzfeldt-Jacob (CJD) panel. These correspond within the probable classification of CJD based on the CDC guidelines. Those guidelines include having a neuropsychiatric disorder plus the positive RT-QuIC in CSF. CJD is a type of rapidly progressive dementia that can present in a variety of ways. This disease is caused by prion infection and the diagnosis can often be missed due to the wide differential diagnosis.
Covid Catastrophes: A Rare Presentation of Catastrophic Antiphospholipid Syndrome

Catastrophic antiphospholipid syndrome (CAPS) is a rare but life-threatening complication of classic antiphospholipid syndrome consisting of multi-organ thromboses, microthrombosis, and hematologic manifestations. A high index of clinical suspicion is crucial for the timely initiation of treatment and reduction in mortality. Here we present an interesting case of a 62-year-old male with a history of antiphospholipid syndrome on anticoagulation, and a prior CVA presenting with shortness of breath and fever for one week following a known COVID-19 contact exposure. Physical examination was significant for tachypnea and diminished breath sounds. Imaging revealed multifocal pneumonia. Treatment for community acquired pneumonia was initiated. Later in the day following admission, patient began to experience hemoptysis along with black stools. Patient was transferred to ICU for hemodynamic monitoring. Concerns for abdominal bleeding prompted repeat blood work which revealed newly elevated coagulation studies with normal platelet count. Given the patient's known history, anticoagulation was held. Despite this, patient continued to have episodes of bleeding throughout the hospitalization. Of note, the patient was found to be COVID positive.

Hematology was consulted and recommended initiating plasmapheresis. Patient also received IVIG and was stabilized over the next several days. Because CAPS was on the differential early on, an appropriate treatment plan was promptly initiated, likely preventing any major complications. It was concluded that the COVID-19 viral pneumonia was the trigger of this patient’s dormant disease. Literature review shows that CAPS is a highly fatal, and often a difficult to diagnose condition given its overlapping features with other thrombotic microangiopathies.
The Fine Line Between Watermelon Stomach and Heyde’s Syndrome

Watermelon stomach also known as gastric antral vascular ectasia is a rare cause of upper gastrointestinal bleed (UGIB). It’s characterized endoscopically by the appearance of watermelon stripes within the stomach, accounting for up to 4% of nonvariceal UGIB. Another obscure GIB disorder, known as Heyde’s syndrome, involves aortic stenosis (AS), angiodysplasia and acquired von Willebrand disease. This is explained by high shear stress at the AS, causing unfolding of von Willibrand multimers leading to coagulation abnormality and GIB. We present a rare case of UGIB in the setting of watermelon stomach and Heyde’s syndrome.

An 88 year-old male with medical history of severe AS, atrial fibrillation on apixaban who presented to ED with angina and dyspnea for three days and 1 episode of melanotic stool. Upon arrival he was hypotensive. Labs were significant for hemoglobin 5.4 with baseline of 12. Anticoagulation was discontinued, 2 units of PRBCs given, and pantoprazole drip. Gastroenterology performed an upper endoscopy revealing a large hiatal hernia, small amount of watermelon-appearing stomach in the antrum and a duodenal arteriovenous malformation which showed evidence of fresh blood and active ooze. Epinephrine was injected and 4 hemoclips were placed and hemostasis was achieved. Patient tolerated procedure well. After 48 hours, anticoagulation was resumed, although he remains high risk for rebleed. Cardiology was also following, but patient was reluctant to consider aortic valve replacement or left atrial appendage intervention. Close follow up was advised.

GIB patients have a high propensity for labile hemodynamics and require very close monitoring. Especially in patients whose benefits outweigh the risks for anticoagulation. This case represents a patient with an unusual concomitant of GIB disorders and challenging management. The treatment options between watermelon stomach and Heyde’s syndrome involves a multidisciplinary approach of medical therapy, endoscopic interventions, and colon or cardiac surgery.
A Rare Case of EBV Cholestatic Hepatitis

Epstein-barr Virus (EBV) is a herpesvirus that commonly causes fever, fatigue, and splenomegaly. Jaundice is thought to be present in up to 5% of cases, due to hemolysis or cholestasis. We present a rare case of EBV-induced cholestatic hepatitis.

A 25-year old female with history of weekend binge drinking presented with fatigue, headache treated with acetaminophen 6000 mg daily for 3 days, fever of 38.9* centigrade, and jaundice. History was negative for ingestion of illicit drugs, herbal supplements, and mushrooms. Urine drug screen, ethanol, pregnancy, and hepatitis screen were negative. Lab work revealed a white blood cell count of 4.5 K/uL (6% atypical lymphocytes, 13% bands, and 1% myelocytes), hemoglobin 13.1 g/dL, platelet count 105 K/uL, AST 173 IU/L, ALT 236 IU/L, total bilirubin 13.6 mg/dL, direct bilirubin 8.5 mg/dL, alkaline phosphatase 183 IU/L, INR 1.08, Prothrombin time 14.4 seconds. Acetaminophen level was 12.6 ug/mL and ferritin was 829 ng/mL. Abdominal ultrasound revealed splenomegaly and a normal biliary tree. She was started on NAC therapy for concern of acetaminophen toxicity. However, further investigations showed a positive EBV Capsid antibody IgM and EBV DNA quantitative 38,694 IU/mL.

Pathogenesis of EBV-induced cholestatic hepatitis likely involves CD8+ lymphocytes activating an immune response that causes hepatocyte injury. This case highlights the importance of early EBV recognition in order to prevent splenic rupture, malignancy, and liver failure. We also took into account a family history of hemochromatosis. She tested positive for the H63D heterozygous hemochromatosis mutation. Studies have associated this mutation to cholestatic liver injury in presence of alpha 1 antitrypsin deficiency, risk of hepatic cancer in cirrhotics, and fatty liver disease. Patient was conservatively managed and advised follow-up, contact sports avoidance, and genetic counseling.
Double-Positive Anti-GBM, Anti-MPO: A Documented but Largely Uninvestigated Rarity

Introduction
Anti-GBM disease and ANCA-associated vasculitis (AAV) in themselves are rare conditions. Though presentation of the two conditions are similar, their disease processes unfold in significantly different ways. Anti-GBM disease, while treatable, confers a significantly low chance of renal survival. In contrast, AAV has a much higher incidence of renal recovery but almost always relapses. When the two coexist, a disease process with intermediate renal survival and relapse is seen.

Case Presentation
A 55-year old, previously healthy female presented for vomiting, decreased urine output, hematuria, and right-sided flank pain for 1 day. She had no personal or family history of renal disease. Labs showed stage 3 acute kidney injury, proteinuria, and anemia. Autoimmune work up showed positive anti-myeloperoxidase antibody and low-positive anti-GBM antibodies. Steroids and plasmapheresis were initiated. Kidney biopsy confirmed features of the two disease entities with proliferative, necrotizing, sclerosing crescents and linear GBM IgG staining. In light of confirmation of AAV, she was started on Cytoxan. Kidney function initially improved but began to worsen despite initiation of the appropriate therapies. Of note, the patient maintained independent kidney function throughout her initial hospital course but unfortunately progressed to end-stage renal disease requiring dialysis approximately one and a half months after diagnosis, concerning for relapse, and therefore more suggestive of AAV-type course.

Discussion
Several studies have investigated this double-positive disease, but only a handful of large studies exist. These studies have universally shown that double-positive patients have disease courses that are an intermediary between the two. As such, a number of questions remain to be answered: what is the optimal treatment regimen for these patients? Does anti-GBM promote the development of AAV, or vice versa? Though rare, the coexistence of anti-GBM and AAV is clearly documented and requires additional investigation.
Pneumocystis jirovecii Pneumonia in Setting of COVID Induced Immunosuppression and Chronic Steroid Use

Hypoxic respiratory failure secondary to COVID-19 pneumonia is routinely treated with glucocorticoids. However, the immunosuppressive effect of long-term steroids can potentially make a patient susceptible to opportunistic infections such as Pneumocystis jirovecii pneumonia (PCP). Previous cases have shown co-infection with COVID-19 and PCP in an HIV positive patient as well as HIV negative patients. We present a patient with no history of HIV who tested positive for PCP after completing a month-long steroid course for the diagnosis of COVID-19 pneumonia.

A 74 year old female with past medical history of recent COVID-19 infection, paroxysmal atrial fibrillation, hyperlipidemia, and obesity presented with a chief complaint of worsening dyspnea. Prior to this, she tested positive for COVID-19 and completed 7 days course of dexamethasone and 5 days of Remdesivir. She was repeatedly re-admitted for additional episodes of hypoxia and ultimately treated with additional steroids leading to a total of 30 days of treatment. Notably she was continually lymphopenic. Due to prolonged steroid treatment and lymphopenia, the possibility of an opportunistic infection was entertained. PCP was then confirmed by bronchoalveolar lavage. She was treated with a 21-day course of trimethoprim and sulfamethoxazole leading to complete resolution of her hypoxia and symptoms.

COVID 19 infection is associated with significant lymphopenia, reductions in CD4+ T, CD8+ T, NK and B cells. The combined immunosuppressive effects of COVID 19 and systemic steroids could predispose patients to develop secondary opportunistic infections such as PCP. Increased awareness of this predisposition should lead clinicians to aggressively pursue additional testing such as bronchoscopy, generally reserved for patients with significant immunosuppression in patients with persistent and progressive respiratory manifestations following COVID 19 infection.
SARS-COV-2 Causing Collapsing FSGS

SARS-COV-2, the causative virus for Covid-19 which is predominantly thought to cause respiratory illness ranging from a mild cough to ARDS and multisystem organ failure. It is suspected to have atypical manifestations; we describe such a case in which a confirmed covid-19 patient developed collapsing FSGS.

72-year-old African American female with a past medical history of essential hypertension, hyperlipidemia, coronary artery disease and stage 2 CKD, not on any nephrotoxic drugs develop acute oliguric renal failure after being diagnosed with Covid-19. At the time of presentation, she was noted to have a Creatinine of 10.44 and a GFR of 4, baseline 0.9 and 74 respectively. She was oliguric with nephrotic range proteinuria and progressively worsening renal function which eventually required hemodialysis. Extensive evaluation, including autoimmune, imaging, hepatitis panel, SPEP revealed no clear etiology for her acute renal failure, subsequently a renal biopsy was performed, which showed evidence of collapsing FSGS.

Much is yet to be discovered about SARS-COV-2 and clinicians should be vigilant for phenotypically diverse presentations of covid-19 to aid in the optimal management of such patients. There is a growing body of evidence to suggest that covid-19 has a wide spectrum of disease activity not limited to the respiratory system and further research is needed to know about the true disease spectrum of covid-19. We propose that covid-19 should be considered in the differential for the patient with acute renal failure, nephrotic range proteinuria and collapsing FSGS, albeit seemingly rare, possibly occurring in genetically susceptible individuals and we should be aware of potential long term sequalae of infection with the SARS-COV-2 virus.
Pulmonary Embolism Masquerading as Decompensated Congestive Heart Failure

Introduction: Heart failure secondary to Pulmonary embolism remains under-diagnosed. It can be potentially life threatening as massive and sub massive Pulmonary embolism can result in Pulmonary Hypertension which can eventually lead to death due to sustained hypotension and cardiogenic shock from Right Ventricular failure.

Case Description: A 65-year-old female presented as a case of New onset Decompensated Congestive Heart Failure on the background of severe anemia due to gastrointestinal bleeding. She had acute hypoxic respiratory failure, which did not improve significantly on initiation of diuresis. McConnell’s sign, defined as right ventricular free wall akinesia with sparing of the apex, was visualized on the Echocardiogram. The presence of McConnell’s sign triggered a workup for Pulmonary Embolism. Treatment of pulmonary embolism in this case proved to be a challenge in view of severe anemia caused by a yet unknown source of gastrointestinal bleed.

Discussion: Pulmonary Embolism has a wide spectrum of presentation from syncope to sudden cardiac death. Pulmonary Embolism should be suspected in patients with Decompensated Heart Failure who fail to respond to appropriate treatment. As Pulmonary Embolism with heart failure has a higher overall mortality than those without heart failure, early treatment is vital. Treating Pulmonary Embolism requires appropriate risk stratification. Multiple interventional procedures like catheter directed thrombolysis, percutaneous mechanical thrombus fragmentation, or percutaneous/ surgical thrombectomy have been devised to capture the benefits of decreasing the clot burden and reduce risk of bleeding from systemic anticoagulation. But clinical experience and accessibility to these novel interventional strategies acts as a limitation.
The Monsters Inside Me

Aortic root abscess is a life threatening complication of aortic valve endocarditis, which increases morbidity and mortality. This occurs more frequently in prosthetic valves than native ones. Successful management requires early diagnosis, antibiotic therapy, and possible surgical debridement or replacement of the aortic root.

A 65-year-old Caucasian male with history of hypertension, diabetes mellitus, paroxysmal atrial fibrillation, PAD, and aortic stenosis s/p TAVR presented to the emergency department with encephalopathy and left foot ulceration. He was being treated for a chronic lower extremity wound and had a skin flap performed 1 week prior. His podiatrist diagnosed him with osteomyelitis. Inpatient blood cultures grew Enterococcus faecalis, and he was treated with daptomycin, cefepime, and metronidazole. TTE was unrevealing, and a subsequent transesophageal echocardiogram revealed TAVR with multiple vegetations and an aortic root abscess at the mitral-aortic intervalvular fibrosa. Brain imaging did not reveal any embolic phenomena. Due to his aortic root pathology, endocarditis, and severe PAD with occluded left popliteal artery, he necessitated transfer to a tertiary center. His antibiotics were changed to ampicillin and ceftriaxone and he underwent an AKA due to gangrene. He was a poor surgical candidate for aortic repair and was treated conservatively with 8 weeks of antibiotics.

Aortic root abscess is frequent sequelae of aortic valve endocarditis and should raise suspicion in patients who fail to improve on appropriate antibiotics. Conduction abnormalities and arrhythmias are common developments. Urgent antibiotics and surgery are recommended due to increased morbidity and mortality with delayed intervention.
**Methylene Blue in Treatment of Acute Metabolic Encephalopathy Secondary to Ifosfamide Toxicity**

Ifosfamide toxicity is rare with approximately 10-30 percent of patients developing an encephalopathy. The decision to treat is often based on additional risk factors for toxicity as the encephalopathy can improve spontaneously. A 74 year old female with a past medical history of Large B Cell Non-Hodgkin Lymphoma refractory to R-CHOP therapy, currently undergoing chemotherapy with RICE presented with acute encephalopathy. She was being observed overnight after receiving her regular RICE infusion therapy earlier in the day, when nursing noticed that she was repeating her own name in response to questions, unable to state her age, and unable to follow simple commands. A stat CT Head was performed and showed no evidence of acute intracranial hemorrhage and a small left sided meningioma. An MRI Brain was also performed which showed an aneurysm of the right carotid terminus, questionable punctate foci scattered throughout both cerebral hemispheres, and no identified mass effect. An infectious etiology of the encephalopathy was ruled out as she was afebrile and had no focal neurologic deficits, headache, or meningismus. Her blood cultures showed no growth at that time. Her creatinine on admission was 1.47, indicating acute kidney injury. Her baseline creatinine was approximately 0.48. There was concern for Ifosfamide neurotoxicity and she was transferred to the ICU and started on methylene blue infusions at 50mg IV Q4. She received twelve doses of methylene blue with continued improvement in her mental status back to baseline within two days. This case illustrates the clinical decision making that should be used in treating acute encephalopathy secondary to Ifosfamide toxicity. Decision to treat with methylene blue should be made by assessing the severity of the patient’s encephalopathy and other risk factors the patient has for delayed metabolism or excretion of the drug.
Not Exactly Infective Endocarditis: A Case of Cardiac Papillary Fibroelastoma

The incidence of primary cardiac tumors is less than 0.1%, and less than 10% of these tumors are determined to be cardiac papillary fibroelastomas (CPF). A vast majority of CPFs are found on heart valves, while the rest are found throughout the cardiac chambers. Most commonly, CPFs are diagnosed in the setting of acute stroke due to embolization of the tumor itself or a thrombus. Otherwise, they may present as myocardial infarction, sudden cardiac death, angina, syncope, heart failure, or other systemic embolic events. About 30% of CPFs are found incidentally on echocardiogram or autopsy. The objective of this clinical case report is to showcase an unusual presentation of this cardiac tumor.

A 79-year-old female with a recent diagnosis of infective endocarditis (IE) presented to the hospital with complaints of sharp left-sided chest pain, for which she had previously been hospitalized multiple times. Recently, patient was hospitalized in Florida with a complicated pelvic abscess and septicemia. At that time, she was noted to have a tricuspid valve (TV) mass presumed to be a vegetation and long-term antibiotic therapy with linezolid and piperacillin-tazobactam was initiated. At our facility, repeat blood cultures were negative and transthoracic echocardiogram revealed a small filamentous structure on the left ventricular aspect, unable to rule out vegetation. Therefore, transesophageal echocardiogram was obtained and displayed a 0.7cm x 1.1cm pedunculated, solid, mobile mass on the posterior leaflet of the TV that appeared to be a CPF. Antibiotics were then discontinued, and IE was ruled out.

This case highlights an exceedingly rare condition that may be overlooked in the setting of a much more common and potentially fatal disease such as IE. In making an accurate diagnosis, the patient was spared long-term intravenous line placement and broad-spectrum antibiotic therapy, both of which have their own plethora of potential complications.
Isolated Iliac Artery Aneurysm and Dissection Leading to Hydroureter and Hydronephrosis

Iliac artery aneurysms are relatively uncommon and account for approximately 10-20% of all AAAs. Solitary or isolated iliac artery aneurysms which do not involve the aorta account for less than 2% of all abdominal aneurysms. Of these, 70% of cases involve the common iliac. Most iliac artery aneurysms can be routinely monitored unless they are larger than 3.5cm.

A 73 year-old woman with a past medical history significant for hypertension and a 45-pack year smoking history presented after experiencing acute onset left lower quadrant pain radiating to the left flank. The patient denied trauma to the area, or changes in bowel or bladder function. All other review of systems were negative. Initial imaging revealed a left iliac artery aneurysm with evidence of a small 2.5 x 2 cm focal dissection as well as left hydronephrosis with notable ureteral caliber change at the level of the aneurysm. The patient was taken to the OR for placement with a ureteral stent to allow for patency of ureter. Two days later, she underwent placement of a Vibahn stent for her iliac artery dissection. There were no reported complications with either of her surgeries and her hospital stay was relatively uneventful. She was seen in clinic two weeks after and was noted to have resolution of her left-sided hydronephrosis.

This case brings to light that although most iliac artery aneurysms are relatively asymptomatic if they are less than 3 cm, some patients may experience symptoms if the aneurysm is large enough to cause a mass effect on surrounding structures. In conclusion, this case highlights the need to keep broad differentials when evaluating patients with abdominal pain as this patient did not fit the typical demographic that we would expect to see to be presenting with an iliac artery aneurysm and dissection.
Take My Breath Away: A Case of Vape-Associated Lung Injury

Acute lung injury from e-cigarettes has been a growing problem in the United States surging its way into mainstream news in late 2019. Multiple studies have attempted to identify a single common ingredient in these popular devices that could be the cause of such harmful pulmonary injury—vitamin E and THC being the leading culprits thus far. We have a case of a 29-year-old medical resident with a past medical history of asthma who presented to the hospital with progressively worsening shortness of breath, fever, and malaise. The patient was found to have bibasilar pneumonitis and patchy infiltrates on chest imaging. After further questioning, he admitted to 1–2-year daily use of use e-cigarettes with both nicotine and cannabis. Acute hypoxic respiratory failure due to vape-associated lung injury required intubation and mechanical ventilation for 12 days. After ruling out high DRIP community-acquired pneumonia and asthma exacerbation, it was concluded that his daily use of e-cigarettes ultimately led to his hypersensitivity pneumonitis. This case should serve as an example of the danger of e-cigarettes and how they are not, in fact, necessarily a “safer” alternative to cigarettes as advertised especially for younger patients.
Rhombencephalitis in an Elderly Female Presenting with Fever and Altered Mental Status

Rhombencephalitis is a rare infection that affects the brainstem and cerebellum. It is imperative that empiric treatment not be delayed as it can result in neurological damage and possibly death, even if causative or definite etiology is not found.

A 76 year old Caucasian female with a history of coronary artery disease and hypertension presented with severe back pain. On admission, she was confused with incoherent speech. She was tachycardic, tachypneic, hypoxic and febrile. Her neurologic exam was without focal neurological deficits but with expressive aphasia and tremulous upper extremities. Lumbar puncture showed lymphocytic pleocytosis with elevated glucose and protein. MRI of the brain revealed Rhombencephalitis. EEG was negative for seizures and CSF was unremarkable for any bacterial/viral etiology. She was started on empiric Acyclovir, Ampicillin, Ceftriaxone, Vancomycin and Dexamethasone. Subsequently Vancomycin, Ceftriaxone, Ampicillin and Dexamethasone were discontinued due to negative lab results. Additionally, HSV 1 and 2, ANA, ENA, ANCA, C3/C4, RF, Myeloperoxidase antibody, IGRA, Quantiferon TB and NMDA receptor antibody and serine protease 3 antibody were unremarkable. Ampicillin was restarted for a total of six weeks, due to persistence of Listeria infections with culture negative results and Acyclovir was continued for 21 days.

Rhombencephalitis is a rare and grave infection that affects the brainstem and cerebellum. The etiologies of Rhombencephalitis falls into three main categories: infectious, autoimmune and paraneoplastic. The most common infectious causes include Listeria Monocytogenes, Herpes Simplex Virus and Echovirus. Definitive diagnosis is made by CSF culture and brain biopsy with histopathology. MRI is imaging of choice. It is imperative that prolonged course of empiric treatment should not be delayed as it can result in neurological damage and possibly death, even if causative or definite etiology is not found.
Profound Metformin-Associated Metabolic Acidosis and Dialysis

Metformin-associated lactic acidosis is a rare but life-threatening condition with often high mortality rates. Critical patients often require extracorporeal measures (dialysis) to ensure survival. We present a unique case of severe acidosis with pH= 6.72 and lactic acid of 26 mmol/L with favorable sequelae after swift initiation of continuous renal replacement therapy (CRRT).

A 61-year-old AA woman with type 2 diabetes mellitus presented to the emergency department 3 hours after ingestion of an unknown amount of metformin as a suicide attempt. She was confused after waking up from a nap which prompted her husband to bring her in. Physical exam showed her to be moderately distressed, moaning in bed. She had slurred speech but was able to follow simple commands. Laboratory workup showed profound metabolic acidosis: arterial blood gas with pH of 6.728 pCO2 of 10.2 mmHg, pO2 of 170 mmHg. Actual bicarbonate was <5 mmHg, anion gap “unable to be calculated” and lactic acid of 19.9 mmol/L. In addition to the above, her potassium was 6.3 mmol/L, and creatinine of 11.8 mg/dL. The patient was admitted to the intensive care unit where she decompensated and was intubated with vasopressor support initiated. Nephrology was contacted and began hemodialysis. The patient’s repeat arterial blood gas showed a pH of 6.74 with worsening lactic acidosis of 26 mmol/L. The patient became unstable and was switched to CRRT. She tolerated CRRT well with normalization of her acidosis and was successfully extubated on day 6. She was discharged in favorable condition on day 7.

This case illustrates the importance of timely initiation of extracorporeal measures to remove metformin metabolites and prevent acute clinical decompensation, especially with pH < 7.1. Recognizing this initiative is critical and likely was a life-saving measure in this case of profound metabolic acidosis.
For the last year, COVID-19 has run rampant with cases involving bilateral infiltrates leading to severe acute respiratory failure due to SARS-CoV-2 coronavirus. While much investigation regarding the initial infection has been studied, post-COVID-19 infection sequela are currently still being studied. It is initially thought to include minor symptoms including fatigue and persistent dyspnea as well as long term organ complications involving cardiac, respiratory, renal, and neurological systems. Here we present a case of a rather healthy 51-year-old male who contracted COVID-19 shortly before Christmas and was hospitalized for a few days due to acute hypoxic respiratory failure. He received MATH+ protocol treatment including steroids and required 2 L supplemental oxygen upon discharge. He presented to the ED two weeks later and was found to have a left apical pneumothorax subsequently requiring hospitalization and chest tube placement for several days prior to discharge home. Ten days later, the patient returned to the ED with dyspnea and worsening hypoxia. He was found to have a large right spontaneous pneumothorax on imaging requiring hospitalization and chest tube placement once again. During this hospitalization, the patient had a negative autoimmune and connective tissue disease workup. He was evaluated by cardiothoracic surgery, who recommended further imaging. CT- chest imaging revealed a 5 cm right lower lobe abscess with an air-fluid level noted, suggesting necrotizing pneumonia and a resolving pneumothorax. He was evaluated by infectious disease and discharged home with appropriate antibiotic therapy. COVID-19 viral pneumonia complications can manifest in various clinical presentations. Our case demonstrates yet another rare complication of 2 unilateral pneumothoraces in a healthy patient without any history of chronic lung disease or mechanical ventilation-associated barotrauma. It is important to keep a spontaneous pneumothorax as a differential on a patient with a history of COVID-19 infection who presents with dyspnea and hypoxia.
Flood syndrome is a rare complication of ascites and liver cirrhosis with the development of an umbilical hernia. The formation of the hernia is caused by the increased intra-abdominal pressure due to the increasing ascites combined with the decreased wound healing effects from cirrhosis. As a result, the hernia spontaneously ruptures and is followed by a sudden and significant rush of ascitic fluid. Ulceration or necrosis of the hernia normally precedes the spontaneous rupture. We present a case of a 63-year-old male who initially presented to the emergency department with complaints of generalized weakness leading to a mechanical fall. Patient has a medical history significant for known alcoholic cirrhosis. Prior to patient’s admission, he undergoes biweekly paracentesis, but missed his scheduled appointments due to the Christmas and New Year holidays. Patient’s previous paracentesis was performed twenty-one days before his admission. During his admission and due to his increasing abdominal girth with associated pain, an abdominal ultrasound was performed which confirmed a large amount of ascites in all four quadrants of the abdomen. With the increasing intra-abdominal pressure and increased ascitic burden multiple complications arose including but not limited to electrolyte derangements, acute kidney injury, metabolic acidosis, and worsening shortness of breath. Interventional radiology was consulted to perform paracentesis to remove the ascites. Of note, patient had been developing a thinning of the overlying skin as well as worsening necrosis around his umbilical hernia site with fistula formation. On day three of patient’s admission and prior to his scheduled paracentesis, patient’s umbilical hernia and fistula spontaneously ruptured and approximately 12-13 liters of straw colored ascitic fluid drained onto the patient’s bed and floor, which is consistent with Flood Syndrome. Patient subsequently underwent repair of his hernia and further evaluation and treatment of his cirrhosis.
Anti-NMDA Encephalitis: New-Onset Seizures and Bizarre Psychiatric Manifestations

Background: Anti-NMDA Encephalitis is a rare syndrome first described in 2007. Less than 200 cases have been reported. The condition is a result of paraneoplastic syndrome, most commonly seen in young females. Patients commonly present with seizures and wide-ranging psychiatric manifestations.

Case: A 27-year-old female presents with new-onset seizures and worsening psychiatric manifestations. The patient was hospitalized for her first seizure three days prior and discharged home on oxcarbazepine. While at home, the patient had continued worsening psychiatric manifestations. During her stay, the patient continued to have auditory hallucinations and waxing and waning mental status. CT and MRI brain were within normal limits. 24 hour EEG showed no epileptic activity. Extensive blood work showed no apparent abnormalities. Lumbar Puncture protein, WBC, and glucose were within normal limits. However, the Paraneoplastic CSF was positive for anti-NMDA antibodies. CT abdomen revealed a right ovarian teratoma. A thyroid ultrasound also relieved a 0.8x0.8cm nodule. FNA of the nodule revealed papillary thyroid carcinoma.

Decision-making: The patient was given scheduled Ativan, Seroquel, and PRN Zyprexa hallucinations. Due to concern of encephalitis, the patient was started on acyclovir and antibiotics until CSF results. The patient was given five days of 1g solumedrol without success. The patient then started five days of IVIG, again with minimal improvement. Once the teratoma was discovered, OB/GYN was consulted and removed the teratoma. However, after removal, the patient had minimal improvement. Because of resistance to past treatments, oncology started Rituximab.

Conclusion: The patient had some improvement in her clinical course after treatment and teratoma removal; however, she was not at baseline. The patient was going to follow up outpatient for thyroidectomy per surgery recommendations. The patient was maintained on scheduled Ativan and Seroquel. No seizures were seen during her stay, and she was continued on her previous oxcarbazepine dose.
Thin Zinc: Acquired Acrodermatitis Enteropathica

Acrodermatitis enteropathica (AE) is a rare autosomal recessive form of zinc deficiency, whereas acquired acrodermatitis enteropathica may occur due to decreased absorption or excessive zinc loss. Zinc is absorbed in the jejunum and conditions such as malabsorption syndromes involving the jejunum, total parenteral nutrition (TPN) dependence, burns or surgery may result in zinc deficiency.

A 55-year-old African American female with a history of chronic debility and short gut syndrome requiring TPN after multiple small bowel obstructions and resections presented to the hospital with concerns of a progressive rash. The desquamating rash initially involved 5-7% of total body surface area that subsequently encompassed over 90% of the total body surface area. The rash had a hyperpigmented and leathery appearance with associated superficial sloughing of the skin. The initial concerns were for Steven Johnson Syndrome (SJS). On further chart review, she was found to have severe zinc deficiency of 32 ug/dL (normal 70-120 ug/dL). A punch biopsy was significant for superficial epidermal reticular degeneration with associated parakeratosis, consistent with necrolytic erythema. These biopsy findings, in conjunction with the patient’s clinical picture, was most consistent with acquired acrodermatitis enteropathica. Due to the extensive skin changes and severity, the patient was transferred to a burn unit. Nutrition was optimized through a parenteral which showed symptomatic improvement in the patient’s condition. She was later discharged home.

The classic triad of AE is diarrhea, alopecia, acral and periorificial dermatitis. The diagnosis of AE is established by clinical features, biopsy, and serum zinc levels. Resolution of dermal lesions with zinc supplementation is the gold standard in establishing the diagnosis. Clinical improvement may occur within days and is often dramatic after initiating therapy. Acrodermatitis enteropathica is an important diagnosis to consider, especially as early diagnosis and zinc supplementation can assist in the complete resolution of the symptoms.
Introduction – Plasmacytomas are neoplastic proliferations of monoclonal plasma cells occurring as single lesions or as part of a systemic disease known as multiple myeloma. Solitary plasmacytomas of the bone are very rare, consisting of 5% of plasma cell disorders. The spine is most frequently affected, but scapula, clavicle, and rib involvement together account for 20% of all plasmacytoma lesions. This case discusses the initial evaluation, diagnosis, and treatment of an isolated acromial plasmacytoma.

Case description - The patient is a 61-year-old female with history of arthritis and unspecified thyroid cancer status-post thyroidectomy who presented to the emergency department for left shoulder pain after sustaining a fall. She was noted to have an anterior dislocation of her shoulder, a large Hill-Sachs lesion, anterior instability with a large rotator cuff tear, and a lytic lesion of her acromion. The patient received a left shoulder arthroscopy and core biopsy of her acromial lesion, as well as repair of all additional injuries. The patient did suffer a pulmonary embolism 5 days following the surgery, and was subsequently anticoagulated. This prompted concern for underlying malignancy. The biopsy was consistent with plasmacytoma. Further workup for additional lesions and multiple myeloma was negative. The patient is currently receiving radiotherapy and being closely monitored by hematology oncology.

Discussion – This case highlights the necessity for early recognition and evaluation of lytic lesions. Skeletal pain and pathological fractures are the most frequently presenting symptoms, and should be evaluated while maintaining a broad differential. Radiotherapy significantly reduces rates of progression into multiple myeloma and should be started as soon as possible.
Blood is Thicker than Water: Polycythemia Vera-Induced Heart Failure

Polycythemia vera (PV) is a myeloproliferative disorder characterized by increased hematocrit and hyperviscosity. Although PV patients classically present with vasomotor symptoms of erythromelalgia, pruritus, fatigue, and headache, heart failure is a rare and poorly documented complication. We present an extraordinarily rare initial presentation of PV as acute decompensated heart failure.

A 70-year-old female, with no past medical or family history, presented to the hospital with dyspnea. She had bilateral lower extremity edema, bibasilar crackles, tongue swelling, and diffuse erythroderma. Initial laboratory studies revealed a hemoglobin 23.7g/dL, hematocrit 69.1%, and NT-proBNP 34,040pg/mL. Transthoracic echocardiogram revealed a new ejection fraction of 25%. She underwent a left heart catheterization demonstrating no significant epicardial coronary artery disease. Further studies revealed an LDH 352U/L, uric acid 12.9mg/dL, erythropoietin 1.61mIU/mL, haptoglobin 92.7mg/dL, CALR Exon 9 mutation-negative, MPL Exon10 mutation-negative, and a JAK (V617F) mutation of 74% (normal <0.1%); consistent with polycythemia vera. CT thorax, abdomen, and pelvis were negative for any masses, lymphadenopathy, or thromboembolism. Furosemide, aspirin, metoprolol tartrate, lisinopril, and hydroxyurea were initiated. During her hospital course, the patient had multiple phlebotomy sessions with subsequent improvement in symptoms. After two months with continued phlebotomy, the patient’s hemoglobin and hematocrit normalized to 13.9g/dL and 39.1%, respectively, and repeat transthoracic echocardiogram showed an ejection fraction of 50%.

This case highlights a unique presentation of hyperviscosity in polycythemia vera. Increased oxygen consumption from the hypermetabolic state of myeloproliferative disorders causes decreased systemic vascular resistance, and high-output heart failure ensues. Moreover, hyperviscosity slows blood flow in the coronary microcirculation and leads to microinfarcts with subsequent ventricular dysfunction. Our patient’s PV treatment through phlebotomy, with a target hematocrit <45%, and hydroxyurea allowed for a decreased thrombosis risk and improvement in left ventricular function. Overall, clinicians should always investigate the etiology behind heart failure in order to appropriately manage every patient.
Unusual Presentation of Mycoplasma Mucositis: A Case Report

Introduction – Mucositis is an inflammatory condition of the mucous membranes which can be caused by a wide variety of etiologies, including both infectious and non-infectious. In Mycoplasma pneumoniae, a known cause of pneumonia, a minority of patients develop extrapulmonary manifestations described as Mycoplasma pneumoniae-induced rash and mucositis (MIRM). This case highlights the broad differential associated with mucositis, the variable presentations associated with MIRM, and the appropriate management.

Case description - The patient is a 23 year old male without any significant medical history who presented to the emergency department for complaints of fever, congestion, myalgias, and oral mucosal swelling for the past 8 days. The patient was diagnosed with community-acquired pneumonia and was sent home with amoxicillin clavulanate. He returned to the emergency department with worsening oral mucosal irritation, cough, fevers, ocular discharge, posterior cervical lymphadenopathy, and vesiculobullous lesions of the penile shaft and scrotum. CT of the thorax and abdomen revealed changes consistent with pneumonitis and borderline splenomegaly, and the patient was admitted for further monitoring and treatment of impending sepsis. The patient was started on acyclovir as the genital lesions prompted concern for an atypical presentation of herpes simplex 2, and azithromycin was added for mycoplasma pneumoniae coverage. The patient’s mucositis and fevers continued to improve during his hospitalization. The patient received an extensive workup for infectious and autoimmune etiologies, revealing only elevated IgM and IgG titers to mycoplasma pneumoniae. The patient was discharged with a 21 day course of azithromycin and had complete symptom resolution.

Discussion – The presence of mucositis should prompt a thorough evaluation for both infectious and non-infectious etiologies. This case highlights the importance of appropriate antibiotic use, recognition of variable disease presentations, and reevaluation of illnesses that do not improve with initial management.
A Case Report of Sick Sinus Syndrome as an Initial Presentation of Primary Amyloidosis

Introduction
Primary light chain amyloidosis (AL amyloidosis) rarely presented as sick sinus syndrome (SSS). We presented a case of AL amyloidosis presented with SSS as the initial presentation to highlight the importance of early recognition by ECG and TTE to improve the patient's quality of life.

Case presentation
A 79-year-old African American female presented with shortness of breath. ECG and telemetry monitoring demonstrated SSS with sinus bradycardia, pauses up to 3 seconds, and atrial fibrillation (AF) with a rate of 130/min. And the patient underwent urgent pacemaker placement to prevent further sinus pauses. Ten days later, the patient had complained of dysphagia and difficulty swallowing. Esophagogastroduodenoscopy revealed esophageal and duodenal ulcers, and biopsy revealed small columnar pieces of mucosa and collections of eosinophilic, amorphous material demonstrated on Congo red staining. The Congo red stain contained one fragment with blood vessels that exhibited mural deposition of amyloid. Two months later, the patient presented with persistent, progressive shortness of breath. Transthoracic echocardiogram (TTE) demonstrated an ejection fraction of 60%, a small pericardial effusion, left ventricular posterior wall diastolic thickness 1.4 cm (0.6-1.0/0.6-0.9 cm), right ventricular systolic pressure 56 mmHg, and mild to moderate biatrial enlargement. Kappa/Lambda ratio was 74.1. A renal biopsy was performed, which confirmed the diagnosis of primary amyloidosis. Due to the terminal nature of her condition, the patient was discharged with comfort measures to hospice care.

Discussion
Conduction system abnormalities are a potential complication of amyloidosis due to amyloid deposits' infiltration of the conduction system. ECG findings include low voltage QRS (<5mm in height in limb leads) with poor R-wave progression in the chest leads, first or Second or third-degree atrioventricular block, AF, and Ventricular tachycardia. TTE features that support diagnosis includes increased left ventricular wall thickening, low ventricular volumes, biatrial enlargement, mildly thickened valves, and pericardial effusion.

Social stigma and neglect Post-COVID-19 and self-quarantine can be associated with Brief Psychotic Disorder. A 53-year-old African American man with no significant past medical history was brought to the emergency department with symptoms of persecutory delusions post COVID-19 and self-quarantine. His symptoms included false beliefs that other people were plotting to kill him, especially his wife. This led him to be combative at work and home. His symptoms worsened so his wife brought him to the hospital. He was treated with Haloperidol 5 mg in the ED. The Clinical Health Psychology and Psychiatry team diagnosed the patient with Brief Psychotic Disorder (BPD) as per Diagnostic and Statistical Method of Mental Disorder Fifth Edition (DSM-5). During follow up visit in outpatient clinic in a week, we found him back to his baseline without any delusional thoughts. Increased stressors post COVID-19, neglect at home and social stigmatization at work of getting COVID-19 along with his individual vulnerability appeared to be the cause of his delusions but various other mechanisms may exist. Our case raises the question: Does social stigma and neglect Post-COVID-19 and self-quarantine matter?
A Novel Case of Atypical Hemolytic Uremic Syndrome Associated with a Previously-Unreported Mutation in the Complement Factor CFH

Hemolytic uremic syndrome (HUS) is caused by Shiga toxin-producing E. coli (STEC) in more than 90% of cases, and rarely occurs in adults. When HUS is driven by a complement-mediated process rather than Shiga toxin-induced endothelial injury, it’s called atypical HUS (aHUS). It is a rare phenomenon related to mutations in genes coding for complement proteins and complement factors. Here, we present a novel case of aHUS associated with a mutation in the complement factor H related 4 (CFHR4) gene.

A 30-year-old Down Syndrome male presented with diarrhea and jaundice after consumption of beef. Workup revealed hyperleukocytosis, rapidly progressive renal failure that necessitated dialysis, thrombocytopenia, pulmonary embolism requiring tPA, and microangiopathic hemolytic anemia. Differentials including thrombotic thrombocytopenic purpura, thrombotic microangiopathy of leukemia or lymphoma, and STEC-HUS were ruled out with normal ADAMTS13, flow cytometry, bone marrow biopsy, and stool cultures. The diagnosis of aHUS was made and the patient was started on weekly eculizumab, an anti-C5 humanized monoclonal antibody. Patient improved and was discharged to inpatient rehabilitation, and ultimately home, where he continued to improve on eculizumab. The genetic testing results returned; although none of the known mutations associated with aHUS were detected, a mutation in the CFHR4 gene was found that has the potential pathogenic effect of decreasing the efficacy of complement factor 3b and thus dysregulating the alternative complement pathway, consequently causing aHUS. This mutation has never been reported in literature before, and a diagnosis of exclusion of CFHR4-associated aHUS was made.

The rarity, complexity, and rapidly progressive nature of aHUS makes it a challenge. As in this previously unreported case of CFHR4-associated aHUS, the condition remains a diagnosis of exclusion that needs to be acted upon quickly. High index of suspicion and early intervention with plasma exchange or eculizumab is required to prevent fatal complications and recurrences.
Seize the Diagnosis: A Case of Hashimoto Encephalopathy

Hashimoto’s encephalopathy is a rare disorder associated with elevations in TPO and anti-TG antibodies with clinical features of altered level of consciousness, seizures and myoclonus. We present a case with significant features consistent with Hashimoto’s encephalopathy.

A 66 year old female with history of hypothyroidism presents to emergency department after being found to be minimally responsive at home. On physical exam patient exhibited only spontaneous movements with withdrawal from pain. Initial CT head imaging was negative. Patient had two seizure episodes. Neurology recommended EEG which was inconclusive. Toxicology screens were negative. Lumbar puncture showed a mild lymphocytic pleocytosis, negative HSV and viral encephalopathy panel. Interestingly, blood cultures were positive for Enterococcus faecium, no source was identified. Patient continued to decline with development of acute respiratory failure requiring ICU admission and intubation.

Post extubation, patient exhibited physical exam findings of functional quadriplegia. MRI, MRA and MRV imaging were nondiagnostic. Due to uncertain etiology of encephalitis, Rheumatologic work up was pursued and found to be positive for Anti-GAD antibody. The patient was started on IVIG for five days with no clinical improvement. Additional studies showed elevated Anti-TPO antibody and anti-TG antibody, elevated TSH with normal T4. Due to concern for Hashimoto encephalopathy, the patient was started on solumedrol with improvement in mental status.

Hashimoto’s encephalopathy exhibits lymphocytic pleocytosis on CSF and nonspecific EEG and MRI brain findings and is often responsive to steroids; as seen in our case. It is rarely reported in literature which may be due to underdiagnosis and under recognition. Additionally, this disorder is an enigma in it’s pathology in relation to its elevation of anti-thyroid antibodies and subsequent neurologic and mentation changes. This case raises questions regarding the recognition of Hashimoto’s encephalopathy and highlight that its presence should be evaluated in patients presenting with encephalopathy and anti-thyroid antibodies.
Chronic Lymphocytic Leukemia (CLL) is inherently associated with increased risk of Squamous cell carcinoma (SCC) because of immune dysregulation. Additionally, there is increasing evidence recognizing the association of Ibrutinib therapy, used in CLL, with Non-melanoma skin cancer (NMSC). We present a case of Cutaneous Squamous Cell Carcinoma in a patient with CLL, which resolved after discontinuation of Ibrutinib therapy.

A 87-year-old male on successful treatment with Ibrutinib for relapsed CLL, for a year, presented with a new, large, ulceroproliferative lesion on the scalp (15 cm X 10 cm X 5 cm). Biopsy of the lesion revealed Squamous Cell Carcinoma. Patient had history of Cutaneous Squamous cell carcinoma and Basal cell carcinoma in the past, after he was diagnosed with CLL. However, previous lesions were small (1-3 cm) and responded well to surgery. Patient did not develop any new skin lesions after achieving complete response and during relapse, prior to starting Ibrutinib therapy. Patient’s current skin lesion responded minimally to radiation therapy. Due to his non-impressive response to radiation, association of SCC with Ibrutinib was sought and decision to hold Ibrutinib was made. After discontinuation of Ibrutinib, there was near resolution the lesion with only mild skin discoloration left.

Ibrutinib is an oral Bruton’s tyrosine kinase inhibitor used for multiple B-Cell Lymphoproliferative disorders including CLL. A retrospective study, analyzing the outcomes of 691 patients with CLL treated with Ibrutinib, presented at the 2019 American Society for Clinical Oncology (ASCO) Annual Meeting, showed 7.8%, 15.8% and 23.3% of patients at 1, 3, and 5 years of follow-up, respectively, had developed non-melanoma skin cancer. Association of Ibrutinib with SCC should be identified as it may warrant discontinuation of Ibrutinib. With the expanding use of Ibrutinib, clinicians need to be aware of the different presentations of Ibrutinib-associated complications, including cutaneous SCC.


COVID-19 Associated Pulmonary Aspergillosis

Introduction
Coronavirus disease -2019 (COVID-19) is caused by severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) infection. Coronavirus disease–associated pulmonary aspergillosis (CAPA) has been reported since the spread of COVID-19 in the world in late 2019.

Case Description
59-year-old-female with history of alcohol-induced dementia, chronic obstructive pulmonary disease, type 2 diabetes mellitus and rheumatoid arthritis presented to the emergency department from a nursing home with difficulty in breathing. Physical examination was remarkable for oxygen saturation in the mid-70s by pulse oximetry and bibasilar decreased breath sound with rales in bilateral lungs. She required intubation with mechanical ventilation and critical care. She tested positive for SARS-CoV-2 by polymerase chain reaction and subsequently developed acute respiratory distress syndrome. She received treatment with dexamethasone for 10 days. Unfortunately, after failing multiple spontaneous breathing trials tracheostomy was performed on day 14. Despite treatment with vancomycin and cefepime for possible hospital acquired pneumonia, the patient remained febrile. Chest x-ray revealed increased infiltrate with bilateral pleural effusion. Work up for other cause and deep vein thrombosis resulted negative. Respiratory culture grew Aspergillus Fumigatus for which she received Voriconazole. Later switched to amphotericin B because of elevated liver enzymes. She was transferred to the general floor on day 29 where she remained mechanically ventilated.

Discussion
Aspergillus can cause mixed pathology in COVID-19 patients ranging from airway inflammation to bronchial invasion. In patients with severe COVID-19, Studies have shown that it impairs CD4+ T cells function including reduced interferon-gamma production. Virus mediated CD4+ T cell dysfunction along with concomitant steroid use, hypoxia, ARDS, etc. further compromising the host immunity are the common predisposing risk factor for aspergillosis in COVID patient. CAPA should be considered in patients not responding to conventional treatments. A positive result for CAPA is commonly seen after 14 days of COVID-19 symptoms.
Meta-Analysis of Left Atrial Appendage Closure Versus Oral Anticoagulation in Non-valvular Atrial Fibrillation

Left atrial appendage closure (LAAC) devices are an alternative therapy in non-valvular atrial fibrillation (NVAF) patients with contraindications to oral anticoagulation (OAC). However, there are limited data about the clinical outcomes of LAAC devices compared to medical treatment. Comprehensive research for studies comparing LAAC devices and OAC for patients with NVAF was performed from inception to January 1, 2021. A meta-analysis was performed using a random effect model to calculate odds ratios (OR) with 95% confidence intervals (CIs). Five studies were eligible that included a total of 4,778 patients with a median-weighted follow-up period was 2.6 years. Compared to OAC, the LAAC device arm was associated with a lower risk of the composite of stroke, systematic embolism, and cardiovascular death (OR 0.72; 95% CI 0.53-0.98, p=0.03). LAAC device arm was also associated with a lower risk of all-cause mortality (OR of 0.61, 95% CI 0.47-0.80, p=0.0004), hemorrhagic stroke (RR 0.19; 95% CI 0.08-0.49, p=0.0006), and major bleeding (OR of 0.62, 95% CI 0.43-0.88, p=0.007). There was no significant difference in ischemic stroke and systematic embolization between the two groups. In conclusion, our meta-analysis showed lower all-cause mortality, hemorrhagic stroke, major bleeding, and the composite of stroke, systematic embolism, and cardiovascular death in the LAAC device arm when compared to OAC. However, the risk of ischemic stroke, systematic embolism were similar between the two groups.
Abnormal Liver Enzymes in Homosexual Male: Could be Syphilitic Hepatitis?

Introduction
Syphilis is a sexually transmitted spirochetal disease which can present as obstructive hepatitis knowns as syphilitic hepatitis.

Case Description
70 year-old-male with history of psoriasis and hypertension presented with mild abdomen pain for one month which got worse for a day. The pain was generalized crampy, 4 out of 10 in intensity and associated with melena. He denied use of acetaminophen, herbals or any drugs. He used to drink a glass of wine per week. Physical examination revealed mild generalized abdomen tenderness and macular rash on trunk and extremities with scales. Laboratory test revealed normal findings except alkaline phosphatase(ALP) 656, alanine aminotransferase(ALT) 314, aspartate aminotransferase(AST) 133, total bilirubin(TB) 5.3mg/dL, direct bilirubin 4.2mg/dL and positive guaiac test. Serological tests were negative for viruses and autoimmune liver disease. Imaging of abdomen and pelvis was unremarkable. Esophagogastroduodenoscopy(EGD) showed esophagitis. Liver biopsy revealed cholesteric disease of unknown etiology. Patient was discharged home on acid suppression therapy but returned after twelve days with six episode of melena, worsening jaundice and new rash on trunk. He revealed this time that he was sexually active with a man for last 10 years and tested for syphilis in between. Physical examination was notable for generalized maculopapular rash on trunk including his palms and soles. Abnormal blood test again were ALP 878, AST 97, ALP 180, TB 14.3, DB 13.1 and GGT (gamma-glutamyl transferase) 757. Syphilis antibody (IgG) and RPR were reactive with RPR titer 1:64. EGD performed showed esophagitis with eight small ulcers. He received benzathine penicillin and liver enzyme came to almost normal in a week.

Discussion
Syphilitic hepatitis presents with disproportionately elevate ALP, GGT and TB. It should always be considered in homosexual men with abnormal liver enzyme levels, serological evidence for syphilis, exclusion of other causes of liver diseases and liver enzyme levels returning to normal after antimicrobial therapy.
Psoriasis is a disorder of immune dysregulation previously thought to exclusively affect the skin, but as of recent, has come to be better understood to affect multiple organ systems. A 49-year-old male with psoriasis with psoriatic arthritis and stage two CKD presented to the ED with bilateral lower extremity swelling. One month prior to presentation, the patient was hospitalized for psoriasis flare up. His vital signs were within normal limits except for HR of 102 beats/minute. His physical exam showed generalized well-demarcated, erythematous plaques with thick scales. Laboratory workup at that time was significant for acute on chronic kidney disease with hyperkalemia. The urine sediment was positive for dysmorphic red blood cells and white blood cell casts. His autoimmune, rheumatic and infectious workup were unremarkable. Renal biopsy was obtained and showed glomeruli obsolescence and cellular crescents. Immunostaining of his kidney biopsy revealed predominance of IgA. He was started on intravenous methylprednisolone 1 gram daily for 3 days followed by 60 mg prednisone daily for one month. He was discharged improved and stable. On current presentation, the patient had progressively worsening leg edema and difficulty breathing. Initial laboratory tests showed stable kidney function from previous admission and normal heart failure workup. Nephrology was consulted and added cyclophosphamide for a more aggressive immunosuppressive regimen. The patient was discharged improved and stable with outpatient nephrology follow up.

Immunoglobulin A nephropathy (IgAN) is a commonly observed comorbidity in psoriasis. Rapidly progressive, crescentic IgAN in psoriasis is rare and has not been widely studied. The KDIGO guidelines recommended treating IgAN with lifestyle modifications and maximum tolerated ACEI/ARB. In the case of crescentic IgAN, the therapy recommended should be is immunosuppressive therapy. Unfortunately, crescentic IgAN has not been extensively studied therefore not much is known regarding long-term outcomes and factors affecting kidney function recovery.
Lights Seem Bright and Everything Else is Lighter – A case of Digoxin Toxicity

Background
Digoxin is a commonly used cardiac glycoside that is used in the management of cardiovascular disorders like Atrial Fibrillation (AF). However, it has a narrow therapeutic range and its' intoxication results in cardiac, neurological, digestive, and hematological adverse effects.

Case Presentation
A 69-year-old female with medical history of type 2 DM, HTN, HLD, AF on metoprolol succinate, warfarin, and digoxin 0.125 mg orally every 72 hours since 4,2020 and end-stage renal disease on hemodialysis since 9,2020, who presented with one week history of chest pain, nausea, abdominal pain and visual disturbances including chromatopsia, specifically xanthopsia. EKG demonstrated junctional RBB, prolonged QRS. Troponin level was indeterminate. CBC showed Hb of 10.8 g/dL, platelet count of 19x10^3/uL. Electrolytes were within normal limits. Digoxin intoxication was confirmed with serum digoxin level of 7.4 ng/mL (ref range 0.8–2 ng/ml). She denied recent changes in her medications or digoxin doses. Patient was admitted to ICU with continuous cardiac monitoring. She received 5 doses of Digfab. After stopping digoxin, chest pain and GI symptoms resolved in 48 hours. EKG showed NSR, however; visual complaints persisted which warranted ophthalmology consult who attributed them to digoxin intoxication. Thrombocytopenia gradually improved and hematology team attributed it to digoxin toxicity as it is a rare but known side effect of the drug.
Patient significantly improved afterward. Cardiology recommended complete avoidance of digoxin indefinitely and consider alternatives for rate control.

Conclusion
This case is a reminder of adverse effects of digoxin treatment to be considered by treating physicians. Close level monitoring is mandatory. In general, when facing a new health complaint in a polymorbid patient, it is crucial to elicit a complete history, including medication history, and to include drug adverse reaction in the differential diagnosis.
Impact of Patient Feedback to IM Residents on HCAHPS Scores

Physician communication is integral for improving patients’ experience and clinical outcomes. Hospital Consumer Assessment of Healthcare Providers and Systems (HCAHPS) scores are used by Centers for Medicare & Medicaid Services (CMS) to assess the quality of care provided to the inpatient Medicare beneficiaries’ and are tied to the value-based purchasing pay for performance metric. Residents of an academic program primarily deliver the majority of patient care, we designed a study assessing the impact of structured, timely, and individualized patient feedback to residents on the teaching attending’s HCAHPS scores. We conducted random surveys (n=668) of hospital admitted patients in our Internal Medicine (IM) Residency from March 2018 to March 2020. The study was approved by the institutional review board and a CMS exemption was obtained. The objective anonymous survey consisted of 11 questions with answers rated on the Likert scale. There were two open-ended questions at the end of the survey for qualitative information sharing. We included any adult patient, who remained admitted to the IM teaching service for two midnights, had decision-making capacity, and was able to provide written informed consent. We excluded patients with any of the following: altered mental status, critical sickness, or worsening clinical status on the day of the survey, or refusal to give informed consent. The survey responses were sent electronically within 48 hours to the resident and the supervising attending. By comparing the pre- (2016-2018) with post-intervention (2018-2020) median HCAHPS scores, we found a consistent and sustainable nine percentage point increase (from 64% to 73%) in HCAHPS scores. This study demonstrates that timely, structured, and individualized resident feedback results in a consistent and sustainable increase in the HCAHPS scores. The results suggest similar interventions can be used in academic programs at a national level to provide real-time resident feedback to improve communication skills and patient experience.
Stauffer Syndrome Leading to a Diagnosis of Renal Cell Carcinoma in a 64-Year-Old Male

Renal Cell Carcinoma is the most common solid renal malignancy; clear cell and papillary carcinoma are the two subtypes of renal cell carcinoma. Renal cell carcinoma’s classic triad of flank pain, hematuria, and palpable mass is the presenting scheme in a minority of patients estimated to be less than 15%. However, renal cell carcinoma has been associated with multiple paraneoplastic syndromes including but not limited to paraneoplastic erythrocytosis, hypercalcemia, hypertension, and Stauffer syndrome which might lead to the diagnosis of an underlying renal cell malignancy.

Stauffer syndrome was first described in 1961 as an entity of nonmetastatic hepatic dysfunction. Laboratory evidence includes elevated alkaline phosphatase, gamma-glutamyl transferase, aminotransferases, prolonged prothrombin time, and thrombocytosis. The exact pathogenesis of Stauffer syndrome is not clearly identified, but it has been proposed that IL-6 secreted by tumor cells affects hepatocytes and leads to the constellation of laboratory findings and symptomatology.

We hereby present a case of a 64-year-old white male who presented with bilateral lower extremity weakness leading to falls. Further hospital course, imaging, and laboratory workup showed elevated aminotransferases, alkaline phosphatase, low albumin, normal total bilirubin, prolonged prothrombin time, and thrombocytosis with an underlying incidental diagnosis of renal cell carcinoma, clear cell pathology possibly pointing out to Stauffer syndrome given no other clear etiologies to the hepatic dysfunction. The patient was eventually referred to a genitourinary oncologist and general surgeon for further evaluation and management. We believe that this case adds to the literature that hepatic dysfunction with an unclear inciting etiology should prompt further workup that should aim at diagnosis paraneoplastic syndromes with a driving malignant process which may allow early diagnosis and prompt medical/surgical management; expectantly improving disease recognition and providing a better quality of patient care.
Ischemic Colitis in Severe COVID-19 - An Unforeseen and Catastrophic Gastrointestinal Complication

Introduction: COVID-19 was declared a global pandemic in March 2020. Initially, it was thought to be primarily a respiratory disease but a wide array of manifestations and complications have been reported in the course of the disease, especially in patients who are critically ill. Of these, gastrointestinal complications are increasingly being recognized.

Case Presentation: A 60-year-old gentleman with a past medical history of diabetes mellitus presented with shortness of breath associated with non-productive cough for 2 weeks. He was hypoxic requiring oxygen supplementation. Chest x-ray showed bilateral ground-glass and patchy areas of consolidations. COVID-19 PCR assay was positive. He was admitted to the medical floor for treatment of COVID-19 pneumonia. He completed 5 days of Remdesivir, 10 days of Dexamethasone, and received convalescent plasma. However, he developed worsening respiratory failure and was intubated on day 20 of hospitalization, requiring ICU care. In the ICU, his condition continued to deteriorate with hypotension, fever and worsening leukocytosis, requiring vasopressor support and treated with broad spectrum antibiotics. On Day 5 of ICU stay, examination revealed distended abdomen and CT Abdomen showed dilation of the ascending, transverse, and proximal descending colon, pneumatosis, associated inflammatory changes along posterior wall of cecum/ascending colon and free fluid in the right lower quadrant. He underwent emergency exploratory laparotomy and subtotal colectomy which showed ischemic bowel from ascending colon to mid descending colon. Unfortunately 2 days later, the patient developed multiorgan failure and expired briefly after he was transitioned to comfort care.

Discussion: Life-threatening gastrointestinal complications of COVID-19 infection such as ischemic bowel remains a diagnostic challenge especially in intubated and sedated patients. Multiple factors have been described in the pathogenesis. These include COVID-19-associated coagulopathy and direct invasion of gastrointestinal tract by COVID-19 due to the ACE-2 receptors which are highly expressed in the gastrointestinal tract.
Severe Epistaxis Secondary to Dabrafenib and Trametinib Use in Non Small Cell Lung Carcinoma

BRAF mutations are estimated to be present in 2-4% of non small cell lung carcinoma (NSCLC). BRAF inhibitor (Dabrafenib) and MEK inhibitor (Trametinib) are currently approved in the treatment of NSCLC harbouring BRAF V600E mutation. However, the use of this new combined targeted therapy can be associated with serious and life threatening toxicities. Here we report a 77 year old male with a recent diagnosis of lung adenocarcinoma with metastasis to the brain and small bowel. Genomic analysis showed BRAF V600E mutation and he was started on dual therapy with Dabrafenib and Trametinib one month ago. Patient presented to the emergency department with a sudden onset of epistaxis over the last 6 hours. He developed respiratory distress due to profuse bleeding and was intubated for airway protection. The bleeding stopped with nasal packing and nebulized tranexamic acid (TXA). After 24 hours of intubation, and with his Epistaxis being controlled with the balloon pack, he was extubated. Shortly after extubation, he started coughing large amounts of dark clotted blood, developed stridor and was re-intubated. Using video assisted laryngoscopy, a large mass-like clot was noted to be partially occluding the vocal cords during intubation. A flexible fiberoptic bronchoscopy was done emergently with retrieval of a large clot extending from oropharynx down into the distal trachea (approximate length about 12 inch). There was no evidence of acute bleeding within the lung segments after the clot removal.

Life threatening bleeding has been reported as a side effect of the combination therapy with Dabrafenib and Trametinib in NSCLC and metastatic melanoma. In the clinical trial BRF113928, 3.2% developed grade III or IV hemorrhage. Our case aims to raise the awareness of physicians to one of the side effects of the new targeted therapy.
Fatal Recurrent Biventricular Takotsubo Cardiomyopathy

Background: Takotsubo cardiomyopathy (TCM) is characterized by ventricular wall motion abnormalities most commonly in the form of left ventricular (LV) apical ballooning. Although less frequent, other variants of left ventricular TCM are not uncommon. Nonetheless, concurrent right ventricular involvement is rarely reported and can easily be overlooked. We present a unique case of recurrent biventricular TCM with a drastic outcome.

Case Summary: A 63-year-old female presented in March 2020 with nausea, vomiting and abdominal pain. She rapidly progressed to respiratory failure requiring intubation. Electrocardiogram demonstrated sinus tachycardia and < 1 mm of inferolateral ST segment elevation. Troponin I and BNP levels were elevated. Chest CTA demonstrated pulmonary edema and small nonocclusive segmental pulmonary emboli. A 2-D echocardiogram (ECHO) findings suggested biventricular TCM with LV ejection fraction (LVEF) of 20%. With aggressive medical therapy, she subsequently improved and went home on carvedilol, lisinopril, atorvastatin, and apixaban. After 7 weeks, regadenoson-Technetium 99-m myocardial perfusion study demonstrated normal perfusion and function. Nine weeks post-hospitalization, ECHO demonstrated complete resolution of the biventricular wall motion abnormalities with LVEF of 60%. In October 2020, the patient presented again with recurrent symptoms. She was hypotensive and developed respiratory failure requiring intubation and vasopressors. ECHO was consistent with biventricular TCM recurrence with severe biventricular dysfunction. Despite aggressive medical therapy, multiorgan failure developed. As her condition continued to deteriorate, her family requested comfort care only. The patient died shortly after withdrawing life support.

Conclusion: Patients presenting with TCM, right ventricular involvement need to be carefully looked for. Early recognition can help anticipate complications and necessitate closer monitoring and special management considerations. Spreading awareness among providers on the frontier is essential. Incidence and impact of recurrence of biventricular TCM are yet to be determined. Further research is needed to help understand the underlying pathophysiology and discover therapy targets and treatments.
Rothia Mucilaginosa, Foe to the Immunocompromised

Rothia Mucilaginosa is a gram-positive aerobic cocci, part of the oral microbiome. It is extremely infectious in immunocompromised patients, causing bacteremia, endocarditis, peritonitis, osteomyelitis, and central nervous system infection. Difficult to grow in-vivo, it is sensitive to penicillin and vancomycin, with resistance to fluoroquinolones. We present a case of a devastating infection in an immunocompromised patient.

A 30-year-old female with a history of acute lymphoblastic leukemia with central nervous system involvement was admitted to the hospital for pyrexia associated with diarrhea. Her last hospital admission was for a febrile neutropenia from an unknown source. Her labs demonstrated hemoglobin 12g/dL, white blood cell count 0.00x10^3/uL and platelets 19x10^3/uL. She was started on aztreonam, acyclovir, trimethoprim-sulfamethoxazole and voriconazole. Diagnostic workup demonstrated Clostridium difficile colitis, and Rothia mucilaginosa bacteremia. CT chest showed probable invasive fungal disease, thus antifungal was changed to amphotericin B. A week into her admission, she was noted to be confused with a bidirectional nystagmus. Imaging of her brain was concerning for infarcts in the splenium of the right corpus callosum and striatocapsular region. Spinal fluid cultures grew Rothia Mucilaginosa, with no malignant cells. She developed hyperleukocytosis which was treated with hydroxyurea and steroids. She ultimately needed mechanical ventilation for airway protection. During her ICU stay, she was found to be completely unresponsive with fixed dilated pupils. CT brain revealed intra parenchymal hemorrhage, with midline shift and transtentorial herniation, attributed to her severe thrombocytopenia. No surgical intervention was done and later her code status was changed to comfort only.

This case demonstrates how the oropharyngeal microbiome can serve as a threat to immunocompromised individuals. Meningitis being the most fatal of all the infectious spectrum. Intravenous antibiotics are the usual treatment, but intrathecal administration can be done if the patient’s clinical status is appropriate.
Disseminated Histoplasmosis in a Patient Taking Budesonide

Disseminated histoplasmosis is not common in patients who are non-AIDS or non-transplant recipients. Recognizing manifestations of disseminated form, having appropriate diagnostic tests for high suspicious patients in endemic areas, and involving infectious disease service early if confirmed diagnosis are key points.

Case Presentation; A 58-year-old female with a past medical history of Crohn’s disease on Budesonide 9 mg daily presented with chief complaints of worsening weakness and intermittent fevers for one month. On admission, the patient was febrile, tachycardic, and required oxygen by Nasal Cannula. Physical exam revealed coarse crackles in both lung bases. Laboratory showed pancytopenia, elevated transaminases, ferritin, CRP, procalcitonin, lactate, LDH. Peripheral smear was negative for immature cells. Extensive infectious workup was unremarkable except for positive urine Histoplasma. CT angiogram Chest demonstrated mild patchy opacities of lower lobes, tree-in-bud opacities of bilateral lungs, small lymph nodes in bilateral hilar, moderate pericardial effusion. Disseminated histoplasmosis was confirmed by bone marrow biopsy. Liposomal Amphotericin B was started. After a long stay in the hospital, the patient was recovered and discharged with Itraconazole to a Long-term acute care hospital.

Discussion: Histoplasmosis is the most prevalent endemic mycosis in the United States, especially in the Ohio and Mississippi River Valleys. Most cases of disseminated histoplasmosis developed in adult patients with HIV/AIDS or organ transplant recipients. It is not common when patients are only on immunosuppressive medication of steroids. The manifestations of disseminated histoplasmosis are constitutional, respiratory, and gastrointestinal symptoms, hepatosplenomegaly, elevated transaminases, pancytopenia. Diagnostic methods include blood fungal culture, cytology or histopathology, antigen tests (eg, serum, urine, CSF, Bronchoalveolar lavage). Differential diagnosis includes viral or other fungal infections, endocarditis, military tuberculosis, HIV/AIDS, hematologic malignancies, rheumatologic diseases. The recommended treatment of moderate and severe cases would be Amphotericin B 2 weeks followed by Itraconazole 12 months and monitor serum Itraconazole concentrations.
Cutibacterium Acnes Pericarditis in the Setting of Cardiac Metastasis

Introduction:
Patients with malignancy who present with pericarditis and pericardial effusions should undergo a full work up to elucidate the cause of their pericarditis.

Case:
A 62-year-old individual with significant past medical history of high-grade neuroendocrine cancer, with metastasis to cardiac tissue, and known pericardial effusion presented to the hospital complaining of shortness of breath and intermittent chest pain. Following examination, he was found to be vitally stable. A Transthoracic echocardiogram was performed and was pertinent for a large pericardial effusion. Inspiratory variations were seen in both tricuspid and mitral valves, suggesting tamponade physiology. Given the history of malignancy, cardiovascular surgery was consulted to determine if the patient would benefit from a pericardial window. Due to the presence of cardiac metastasis and since the individual was set to start chemotherapy, it was determined that pericardiocentesis would be the preferred route. The patient underwent pericardiocentesis which resulted in the drainage of 1.1 L of red turbid fluid. The pericardial fluid was sent for cytology and culture which was negative for malignancy. Pericardial fluid culture came back positive for Cutibacterium Acnes. Over the course of 6 days, the patient’s drain output decreased (Total output >2L), limited echocardiogram (Day 6) revealed trivial pericardial effusion and, as a result, the pericardial drain was removed. Infectious disease was consulted and they recommended a 5 week course of IV ceftriaxone.

Discussion:
Purulent pericarditis is an important diagnosis that should not be overlooked. Cutibacterium Acnes is a rare cause of purulent pericarditis and as it can take 5-7 days to grow in cultures, it can lead to reduced detection. It is essential that patients with a history of malignancy have a full workup for their pericarditis and pericardial effusions, as cases such as this could have been simply ruled as neoplastic pericarditis, leading to improper medical treatment.
Amiodarone-Induced Torsades de Pointes

Amiodarone is an effective antiarrhythmic medication with several mechanisms of action that typically reduce its pro-arrhythmic potential, however it can result in prolongation of the QT segment on EKG. In the setting of R-on-T phenomenon, this can result in a polymorphic ventricular tachycardia known as Torsades de Pointes (TdP).

A 68-year-old female with a history of mildly prolonged QTc (479 ms), nonischemic cardiomyopathy (LVEF 40%), and mitral valve repair, presented to the ED with syncope 23 days after starting amiodarone for PVC suppression. While at a doctor’s office, the patient experienced two sequential syncopal episodes without significant prodrome. EMS documented a wide-complex tachycardia, corresponding to episodes of unresponsiveness. In the ED, vital signs were normal. EKG revealed normal sinus rhythm, a QTc of 764 ms (when measured and calculated manually) and a left-bundle branch block. The patient continued to experience episodes of near syncope and flushing, corresponding with intermittent polymorphic ventricular tachycardia (PMVT).

The patient was treated with IV magnesium and started on a lidocaine infusion. A transvenous pacer was placed for overdrive pacing and amiodarone was discontinued. Laboratory workup revealed a therapeutic amiodarone level at 1.0 mcg/mL, electrolytes were within normal limits. With lidocaine infusion and atrial pacing, QTc sequentially shortened on serial EKGs to 502 ms over the next 5 days. During this time, the patient had no further episodes of PMVT or presyncope. PMVT was thus attributed to R-on-T phenomenon in the setting of prolonged QTc. Given the reversible iatrogenic cause of her PMVT, an implantable cardiac defibrillator was deferred. The patient was transitioned to mexiletine and discharged with a wearable cardiac defibrillator.

This case highlights the QTc prolonging effects of amiodarone that can result in rare, but serious complications. The manual measurement of QTc is necessary to quantify the severity of QTc prolongation and thus patient risk for development of TdP.
Myocardial Infarction Seen But Coronaries Clean: A Case Of MINOCA

Myocardial infarction (MI) with non-obstructive coronary artery disease (CAD), a syndrome referred to as MINOCA, occurs in approximately 6% of patients with acute MI. Classical cardiac risk factors are often absent, with a disproportionately high prevalence noted in females <55 years old. Despite absence of obstructive atherosclerosis, current data suggests similar morbidity and mortality outcomes as MI secondary to obstructive CAD. Diagnosis requires exclusion of nonischemic etiologies of cardiac enzyme elevation.

Case Presentation: A 56-year-old female with poorly-controlled dyslipidemia presented with acute substernal chest pain concerning for angina. High sensitivity cardiac troponin T enzymes were elevated and increased to a peak of 1,997 ng/L (normal <14 ng/L). Her EKG demonstrated sinus bradycardia with early repolarization. Transthoracic echocardiogram demonstrated inferior wall hypokinesis with preservation of systolic function. CT angiogram of the thorax was negative for pulmonary embolism or aortic dissection. Management for type 1 NSTEMI was initiated, including full dose aspirin, clopidogrel, metoprolol tartrate, IV heparin and high-intensity statin. Coronary angiography demonstrated no obstructive CAD. Cardiac MRI with IV gadolinium contrast revealed evidence of a transmural myocardial infarction in the inferior wall. Though not definitive on imaging, spontaneous coronary artery dissection (SCAD) was suspected given troponin elevation and MRI findings. She was discharged in stable condition on aspirin, clopidogrel, beta blocker, high-intensity statin and recommended to complete cardiac rehabilitation without recurrent chest pain.

Discussion: MI in the absence of underlying obstructive coronary atherosclerosis (MINOCA) is an uncommon finding but has important clinical implications. Potential MINOCA etiologies includes SCAD, viral myocarditis and stress-induced myocarditis. Evaluation is aimed at identifying the underlying etiology both for prognostication and targeted therapy. Presently, consensus on long-term management has not been established, but it is clear that MINOCA is not a benign condition. Raising awareness of this uncommon syndrome is important to foster appropriate counseling and management.
Statin-induced Drug Reaction with Eosinophilia and Systemic Symptoms (DRESS syndrome)

Drug reaction with eosinophilia and systemic symptoms (DRESS) is a rare, potentially life-threatening, hypersensitivity reaction characterized by skin eruption, eosinophilia and organ involvement typically 2-8 weeks post culprit drug exposure. This condition can have a prolonged course with relapses despite discontinuation of the culprit medication. Most commonly implicated medications include sulfonamides, allopurinol, antipsychotics and anticonvulsants. DRESS syndrome with statins has not been described in literature to date. An 87 year old female with hypertension and dyslipidemia presented with fatigue, arthralgias and bilateral lower extremity rash. The patient was started on atorvastatin prior to the onset of symptoms. Vital signs and cardiopulmonary exam were unremarkable. Skin exam showed a dark, non-blanching rash on both lower extremities. Laboratory examination showed significantly elevated eosinophil and leukocyte counts along with increased liver transaminase, creatine kinase, sedimentation rate and CRP levels. Work up for hematological malignancy including bone marrow biopsy was unremarkable. Skin biopsy revealed perivascular and interstitial inflammatory infiltrate with abundant eosinophils and neutrophils consistent with DRESS syndrome. Atorvastatin was discontinued and patient was treated with prednisone. Her symptoms and laboratory abnormalities including eosinophilia improved. Prednisone was discontinued and statins were deemed contraindicated for this patient. Statins are commonly prescribed in the United States, with an estimated 35 million Americans taking them. There has been a substantial increase in use of statins in line with cholesterol treatment guidelines based on risk factors. While adverse events such as myalgias and transaminitis are well documented, statin-induced DRESS syndrome is a rare, potentially serious condition which should be considered in patients with new onset rash and eosinophilia while on statin therapy.
Spontaneous Coronary Artery Dissection Precipitated by Intercourse

Introduction: Spontaneous Coronary Artery Dissection (SCAD) describes an intracoronary intimal separation of arterial supply which can precipitate acute coronary events [1]. SCAD provokes between 0.1-0.4% of acute coronary syndromes, most commonly inflicting women with risk factors that include multiparity, connective tissue disorders, inflammatory conditions, and hormonal therapy [2]. Herein, we present a case of intercourse induced SCAD in a gravid female.

Case Presentation: A 24-year-old female gravida 5 para 3 in 1st trimester of pregnancy with a history of asthma presented with acute onset retrosternal chest burning with radiation to her shoulders and arms initiated during sexual intercourse. Physical exam demonstrated normal S1/S2 without murmur, pulses were equal, and blood pressure was normotensive, with no contributory findings. Electrocardiogram discerned no signs of ischemic change, however initial high sensitivity troponin was 139 with two hour delta of 172 increased to 311. Transthoracic echocardiogram displayed distal anterior wall hypokinesis. Patient was admitted for suspicion of SCAD and underwent coronary angiography which demonstrated normal major coronary arteries, however with distal lesions in both the obtuse marginal branch and diagonal artery and resulting in no intervention. The patient was started on aspirin and metoprolol succinate with resolution of symptoms and was discharged with follow up with maternal fetal medicine and cardiology.

Discussion: Previous case reports suggest that intercourse has been a precipitating agent of SCAD as well as other arterial dissections including aortic, carotid, vertebral, and cerebral arteries [1]. Mechanism between the relationship between intercourse and dissection remains unknown, although additional intravascular stress is thought to be contributory. Complications of SCAD can include ventricular arrhythmia, myocardial ischemia, and death, thus clinical awareness is important to recognize and treat patients as indicated. Pregnancy holds many inherent gynecologic risks, but it is also imperative to understand the life-threatening cardiac dangers of pregnancy including SCAD.
Disseminated Cryptococcosis in the Setting of IPF Can Be a Fatal Complication of Long-Term Immunosuppressive Therapy

Background: Cryptococcosis is a systemic fungal infection with rapidly fatal sequelae that primarily affects immunocompromised individuals. Previous studies have shown that about one-third of Cryptococcus cases occur in the setting of long-term corticosteroid therapy. This case report highlights the importance of initiating anti-fungal agents at a lower threshold in patients on long-term immunosuppressive therapy.

Case Presentation: We present a 76-year-old man with IPF who presented to the hospital with confusion and was admitted for work-up of acute encephalopathy. Of note, due to ongoing progression of his IPF and multiple admissions for respiratory failure, his Pulmonologist was unable to wean down his daily high dose steroids. A Brain MRI revealed acute infarcts in the cerebral hemispheres bilaterally and in the left cerebellar vermis. EEG was performed due to episodes of right-sided fixed gaze but revealed no epileptiform activity. He later developed another episode of right-sided fixed gaze, followed by unresponsiveness, tachycardia, tachypnea, and agitation which prompted a repeat Brain MRI that was significant for leptomeningeal enhancement and infarction in the cerebral hemispheres bilaterally. A lumbar puncture revealed an elevated opening pressure and Cryptococcus neoformans within the CSF. After starting anti-fungal agents, the patient unfortunately decompensated further and was intubated in the ICU. A bronchoscopy later revealed Cryptococcus in the respiratory culture. Afterwards, he developed pupil asymmetry prompting a STAT Head CT, which revealed diffuse cerebellar edema, compression of the fourth ventricle, obstructive hydrocephalus, and tonsillar herniation. The patient was transitioned to comfort care and died shortly later.

Conclusion: Previous studies have shown that corticosteroids, even at low doses, predispose patients to opportunistic infections such as Cryptococcosis. Given how rapidly patients can decompensate, it is imperative to have a high level of suspicion and a low threshold to initiate empiric fungal agents in those who are hospitalized and on long-term immunosuppressive therapy.
Lung Transplant, a Definitive Intervention for Accelerated Silicosis

Introduction:
Accelerated Silicosis describes a subset of silicosis demarcated from chronic silicosis by its rapid onset and more intense exposure, with disease manifesting between 5-10-years of first exposure [1]. Epidemiology regarding silicosis is difficult to quantify although 3600-7300 cases were recognized annually from 1987 to 1996 [2]. Cases manifested in patients with heavy occupational silica exposure including sandblasting, mining, and glasswork, and is also associated with underlying autoimmune disorders [2].

Case:
A 39-year-old with a past medical history of persistent severe asthma, interstitial lung disease 2- 4 L NC at baseline, 20 year pack-history, Foundry work, who was initially admitted at OHS for dyspnea, hypoxemia with increasing 02 requirements on 01/04 and transferred to Butterworth facility on 01/07 for further pulmonary workup, and being consult for acute on chronic hypoxic respiratory failure. CTA obtained at an outside facility showed progression of his interstitial lung disease in RML/RLL/LLL with bilateral peribronchovascular nodularity and progression of his Subpleural cystic changes in lower lobes. Rheumatology work up within normal limits. Spirometry revealed severely reduced forced vital capacity and severely reduced ventilatory defect. Given advanced interstitial lung disease, the patient was evaluated by multidisciplinary cardiothoracic and lung transplant teams and deemed as an appropriate candidate for bilateral lung transplant. In subsequent admission, the patient received bilateral lung transplant.

Discussion:
Accelerated silicosis, a rare etiology of progressive massive fibrosis within 10 years of exposure to high levels of silica. As our case demonstrates, the first diagnostic step begins with minimally invasive spirometry and lung volumes, however it is equally vital to rule out autoimmune and rheumatological etiology. Although various therapeutic modalities exist for management of accelerated silicosis, lung transplant remains as the only curative option. A prompt multidisciplinary approach and evaluation at a lung transplant center for definitive management can have an incredible impact on a life.
Brain Tissue Found the Issue: Primary Angiitis of the CNS

CNS vasculitis requires a high index of suspicion given the protean nature of presentations.

Case-Presentation:
An otherwise healthy 21 year old woman presented with headache, confusion, twitching, nausea, and vomiting. Workup included an unremarkable head CT and LP consistent with aseptic meningitis. Viral meningoencephalitis was suspected, she improved and was discharged. Intermittent frontal headaches with associated dizziness, bilateral lower extremity numbness, and confusion recurred with fever (40.4 degree C), but repeat head CT and LP essentially unchanged with elevated protein, lymphocytic pleocytosis and elevated opening pressure. MRI brain and spine w/wo contrast notable for leptomeningeal enhancement and restricted diffusion in the splenium of the corpus callosum. Extensive infectious, autoimmune, rheumatologic, hypercoagulable, and paraneoplastic workup was unrevealing. Flow cytometry from CSF resulted with atypical lymphoid population. A CT chest, abdomen, pelvis, was negative for malignancy. Re-admitted with headache, lower extremity weakness and aphasia. Electroencephalogram revealed potential seizure focus and levetiracetam was started. Brain biopsy via right frontal craniotomy performed for definitive diagnosis. Final pathology revealed vasculitis, predominantly subarachnoid and superficial cortex. MRA brain showed no abnormal vessel wall enhancement. Following initiation and continuation of high-dose systemic steroids, she had near complete symptom resolution.

Discussion:
Over a 3 month time period, this patient presented to 4 different hospital locations, was admitted 5 times, and had an extensive workup including 4 LPs, over 10 imaging studies of brain/spine, 3 EEGs and 1 invasive brain biopsy. Her diagnosis of primary angiitis of the central nervous system (PACNS) is very rare and the cause is unknown. Her relatively expedited time to diagnosis, compared to the more common 6 months, is likely due to multidisciplinary approach and the red herring of the atypical CSF flow cytometry. This case highlights the importance of a broad differential, persistent curiosity, a multidisciplinary approach, and tissue biopsy while diagnosing rare illnesses.
Visceral artery pseudoaneurysms are rare but potentially life-threatening conditions that require early intervention to prevent rupture and hemorrhage. There are very few reported cases of left gastric artery (LGA) pseudoaneurysms. In some instances, LGA pseudoaneurysm has been associated with stomach ulcer formation and gastrointestinal hemorrhage. A 60-year-old male with alcohol use disorder presented to the emergency department with sudden onset massive hematemesis and hypotension. This improved following blood transfusions and volume resuscitation. Esophagastroduodenoscopy (EGD) demonstrated a 10 mm cratered non-bleeding ulcer at the gastric incisura of the lesser curvature, with no additional intervention at that time. The following day while in the Medical ICU, he had recurrence of large hematemesis, with profuse hematochezia and extreme hypotension, necessitating massive transfusion protocol. Emergent bedside EGD found active bleeding from the area of the previously seen ulcer, which was treated with epinephrine injection and hemostatic spray. Following this, angiography of the celiac trunk revealed a 10 mm pseudoaneurysm arising from a branch of the left gastric artery. The pseudoaneurysm was successfully embolized, and the patient had no further recurrence of bleeding. Review of reported LGA pseudoaneurysm cases have demonstrated similar presentations of melena and gastric ulcer in the lesser curvature. Risk factors include acute or chronic pancreatitis or surgical trauma. In this case, acute pancreatitis and trauma were ruled out. The patient did have a history of alcoholism, however, subsequent abdominal CT imaging did not reveal any sequelae of chronic pancreatitis. Thus, this appears to be a case of idiopathic LGA pseudoaneurysm which caused gastric ulcer formation and hemorrhagic shock. While exceptionally rare, LGA pseudoaneurysm should be considered in cases of GI bleed and ulceration in the lesser curvature of the stomach. When suspected, urgent angiography is necessary as embolization is highly effective for treating pseudoaneurysms and avoiding life-threatening complications.
Break My Heart Again: A Case of Recurrent Takotsubo Cardiomyopathy with Different Morphologies.

Takotsubo Cardiomyopathy (TTM) is characterized by transient LV wall motion abnormality without obstructive coronary disease. It usually affects postmenopausal women in mental or physical stress.

Our patient was a 65-year-old female with emphysema, Takayasu Arteritis, and hypertension who had an initial episode of TTM in 2010. She presented with hypoxic respiratory failure, was found to have an LV ejection fraction of 15% with hypokinesia in anterior, inferior, and apical segments. Basal segments were normal. She presented in June 2020 with shock and hypoxia. Echocardiogram revealed an EF of 22%, now with basal and mid-ventricular hypokinesis and her apical segments were hyperkinetic suggestive of a reverse TTM. Coronary angiography showed no evidence of coronary artery disease on both occasions. She was initiated on neurohormonal blockade. Follow-up echocardiograms noted improvement in LV function. The patient presented again on January 2021 with COPD exacerbation in the setting of medication non-adherence and continued smoking. Troponin was elevated without any ischemic changes on electrocardiogram. An Echocardiogram this time showed LVEF 25% with apical wall motion abnormality and basal segment hyperkinesis suggesting a reversal in morphology once again. She had symptomatic improvement after treating her COPD exacerbation. Initiation of neurohormonal blockade was challenging due to symptomatic hypotension, and she was discharged to continue management on an outpatient basis.

We present this case to stress the significant recurrence rate of TTM. It is also interesting to note the different ballooning patterns in each episode which questions the concept of the involvement of sympathetic nerve distribution in the myocardium as the actual pathophysiology. Coronary microvascular involvement is one theory that could explain the varying morphologies. Despite being optimized on beta-blocker therapy, the patient had recurrence which questions its effectiveness in preventing relapses. On the contrary, beta-blocker could have had some impact on COPD exacerbations, potentially contributing to recurrence.
Prediction, Causality, and Preventability of 30-Day Hospital Readmissions in Patients with Chronic Obstructive Pulmonary Disease

Background: Chronic Obstructive Pulmonary Disease (COPD) is a major global health concern. Twenty percent of COPD admissions get readmitted within 30-days, impacting the patient's quality of life and imposing a significant burden on the health care system. Prediction models may help tailor interventions to those who will benefit the most. We evaluated the ability of the Placement Resource Indicator for Systems Management (PRISM) score, LACE index, and a combination of both in predicting 30-day readmission and mortality. Additionally, we studied the difference in causality and preventability between early (within 7 days) versus late (8-30 days) readmissions.

Methods: Our retrospective study included patients admitted to St. Joseph Mercy Hospital, Ann Arbor, Michigan, with a principal diagnosis of COPD from January 2010 through December 2018. The predictive abilities of LACE index and PRISM score were assessed using receiver operating characteristic (ROC) curves and Classification and Regression Tree (CART) models. Determination of causality and preventability utilized a previously published framework.

Results: Admissions for COPD totaled 1,392. The LACE index, PRISM score, and combination of both had poor discriminative ability to predict 30-day readmission (c-statistic = 0.558, 0.548 and 0.569, respectively) and moderate ability to predict all-cause mortality (c-statistic = 0.679, 0.656 and 0.707, respectively). Secondary analysis of 423 patients demonstrated that management errors contributed to both causality (P = 0.012) and preventability (P = 0.014).

Conclusion: Generalized predictor models using risk stratification such as the LACE index and PRISM score are poor predictors of 30-day readmission in patients with an index admission for COPD. Actions taken by hospitals and health care providers during the index admission may prevent early readmissions.
Four Years of Intermittent Fevers: A Diagnostic Challenge

Fever of unknown origin (FUO) is one of the most complex diagnostic puzzles in medicine. FUO is defined as temperatures ≥ 101°F on separate occasions, with no identifiable etiology. Infections cause 20-40% of FUO cases, but are less likely the longer that FUO persists.

We present a 62-year-old male with chronic systolic heart failure with an implantable cardioverter-defibrillator (ICD), who was admitted with dyspnea and peripheral edema. He also had been experiencing intermittent fevers for the past four years, with no detectable cause despite extensive diagnostic evaluation. On admission, he was found to have acute renal failure, which was presumably due to cardiorenal syndrome. However, urinalysis showed hematuria and proteinuria, concerning for glomerulonephritis. Trans-esophageal echocardiogram (TEE) revealed a large vegetation on one of the ICD leads, prolapsing across the tricuspid valve. After six days, blood cultures grew Cardiobacterium hominis, one of the HACEK organisms. The patient responded well to Ceftriaxone. He underwent removal of the infected lead and vegetation, along with tricuspid valve repair.

We present a rare case of HACEK ICD lead endocarditis, with intermittent fevers for four years. Our literature review revealed that two years is currently the longest time period reported between ICD implantation and diagnosis of endocarditis. Additionally, ICD lead endocarditis has also been very rarely caused by HACEK organisms, the most common causes being Staphylococcus and Candida species.
Never Too Old; A Case of Chicken Pox in an Eighty-One-Year-Old Patient

Introduction:
Varicella-zoster virus (VZV) is a human herpes virus which can present in either an acute (varicella) or latent form (herpes zoster). Varicella is rarely encountered in immunocompetent adults. Since the introduction of the varicella vaccine in 1995 as a part of the standard childhood vaccination program, primary VZV infection has become exceedingly rare.

Case Presentation:
An 81-year-old female presented to the primary care office with one-week history of shortness of breath followed by the development of a pruritic rash. Supportive care was recommended for the diagnosis of a viral syndrome. Her shortness of breath progressed and a day later she was admitted to the hospital with fever of 101.4 F, malaise, severe dyspnea and worsening of her pruritic rash. Physical exam was notable for a vesicles and crusted papules on an erythematous base scattered on her bilateral upper and lower extremities, trunk and face, though sparing her palms and soles. There was no mucocutaneous involvement. Her chest x-ray did not demonstrate pulmonary infiltrates. Laboratory findings revealed mild transaminase elevation and thrombocytopenia. VZV PCR testing from an immature vesicle was positive. Punch biopsy of a vesicle showed keratinocytes with intranuclear inclusions. Valacyclovir was begun and the patient improved over the following three days.

Discussion:
The majority of patients with advanced age are seropositive for a prior VZV infection. Rarely, exposure of a seronegative elderly patient to VZV can occur, leading to an acute primary infection. In the adult population, diagnosis and treatment is important given the four times higher mortality risk compared with children.
A Rare Presentation of Acute Pancreatitis in Mild Coronavirus Disease 2019 Infection

Introduction: Viral-induced pancreatitis has previously been well-defined, however there is limited data regarding the association between Coronavirus Disease 2019 (COVID-19) and pancreatitis. A handful of cases have been described, but most commonly are in conjunction with severe COVID-19 infections as well as pancreatic enzyme elevation. It has been demonstrated that patients with acute pancreatitis and coexistent COVID-19 infections have a significantly higher morbidity and mortality than those without acute pancreatitis. We describe a case of mild COVID-19 infection resulting in acute pancreatitis in the absence of lipase elevation.

Case Description: A 39-year-old patient with no significant past medical history, including no history of chronic pancreatitis or alcohol use disorder, presented with epigastric pain, loss of taste and smell, fever and dry cough. Vital signs were within normal limits. Laboratory findings were significant for positive COVID-19 testing. Liver function panel, calcium, triglyceride, and lipase levels were all unremarkable. Computed tomography of the abdomen was consistent with acute pancreatitis without evidence of gallstones and without pancreatic, intra or extrahepatic biliary dilatation. The patient underwent conservative management with bowel rest and intravenous fluids and was discharged without complications.

Discussion: This case introduces a rare presentation of acute pancreatitis without lipase elevation in setting of mild COVID-19 infection. It has been unclear whether lipase elevation in COVID-19 is due to direct pancreatic injury or viral-induced systemic inflammatory response causing multiorgan dysfunction. Our case may indicate that there is a direct impact on the pancreas by the SARS-CoV-2 virus, which has been postulated given the presence of ACE-2 receptors in the pancreas. This case also highlights the importance of suspecting pancreatitis in patients with mild COVID-19 infections that present with atypical symptoms such as epigastric pain, even without lipase elevation. Early identification in these cases may help to reduce progression and prevent worse outcomes.
Low Bone Mineral Density and Recurrent Fractures During and After Pregnancy: Dilemma in an Overlooked Diagnosis

Background:
Decreased bone mineral density (BMD) during pregnancy and lactation are rarely described in literature. We present this case of multiple compression fractures during and after pregnancy, highlighting the diagnostic and therapeutic dilemma such an overlooked diagnosis.

Case Summary:
A 23-year-old female without significant past medical history, suffers from acute lower back pain in the third trimester of her first pregnancy. On initial evaluation, this back pain was thought to be musculoskeletal and was dismissed by her medical team without any imaging. On her second day postpartum, she heard a “pop” in her back and fell while holding her newborn. Investigative imaging revealed multiple vertebral compression fractures, in different stages of acuity. Due to persistently debilitating pain, she quit breastfeeding and would never hold her baby again. Before long she had to leave work as a secretary with debility. For decades, her compression fractures were managed with analgesics and vitamin D/calcium supplementation. At age 58, a CT spine demonstrated new compression fractures at T5-T12 & L1-L5, requiring kyphoplasty of T5, T7 and T8. Fortunately, a bone core biopsy showed no malignant pathology. At age 60, the patient was referred to the bone clinic at a tertiary health center. Initial labs showed an osteocalcin level of 3.9 ng/mL (NL 8.8-37.6 ng/mL). BMD scans showed T-scores of 0.6 and -2.6 at her lumbar spine and distal forearm respectively. Her osteoporosis is currently managed with teriparatide without active issues.

Conclusion:
This vignette highlights the rare development of low BMD in pregnant and breastfeeding women, without prior risk factors; jeopardizing future quality of life. There’s a deficiency in evidence behind the incidence and pathophysiology underlying these changes. There also remains a dearth of guidelines for defining and managing osteoporosis and low BMD in young peripartum women.
Pritikin Program Intensive Cardiac Rehabilitation Versus Traditional Cardiac Rehabilitation Outcomes Study (PICO)

Background: Conventional cardiac rehabilitation (CCR) is an exercise based reconditioning program whereas Pritikin Program intensive cardiac rehab (ICR) adopts a more holistic approach adding dietary education and stress management training to CCR. We compared the effectiveness of both programs to determine the incremental benefit of ICR.

Methods: Retrospective cohort study of 1018 patients enrolled in cardiac rehabilitation between 2016-2019, 233 were in the CCR and 785 in the ICR group. Primary outcomes were readmission rates, mortality rate, and DASI Mets. The secondary outcomes included Non-HDL and LDL Cholesterol, HbA1C level, SF36 Mental and Physical score, PHQ9, Dietary Fat Screener, Rate Your Plate score, and BMI. The outcomes were measured before and after the rehabilitation programs. Data was analyzed using propensity score matching and simple and adjusted Wilcoxon rank sum and chi square tests. P-values for secondary outcomes were not corrected for multiple comparisons.

Results: After propensity score weighting there was no significant difference in demographics and comorbidities. Readmission or mortality rates were not significantly different between groups, although there was a signal toward decreased 1-year mortality in the ICR group. DASI Mets increased by 1.7 points in the CCR group and 1.8 points in the ICR group (p=0.154). There was a significant difference between ICR versus CCR in the secondary outcomes of post rehab dietary fat screener score, 8 vs. 12 points, respectively (p<0.001), and rate your plate score, 63 vs. 59 points, respectively (p=0.003).

Conclusion: Our study demonstrates that when compared to CCR, ICR helps the patients significantly improve their dietary habits as measured by patient reported scores for dietary fat screener and rate your plate. There is a signal toward decreased mortality in the ICR group suggests a need for additional longer-term studies.
Identifying Barriers and Facilitators to Scalp Cooling Therapy Through a National Survey of Oncology Providers

Purpose: Scalp cooling therapy (SCT) is the most effective method to reduce chemotherapy-induced alopecia (CIA), a highly distressing and common side effect of cancer treatment. Despite a growing body of literature supporting its efficacy and safety, use of SCT for reducing CIA in the United States is not widespread. Oncologic providers’ knowledge, practice patterns, and attitudes regarding scalp cooling were examined to identify facilitators and barriers to SCT implementation.

Methods: In February 2020, our 33-question survey was distributed through the American Society of Clinical Oncology’s (ASCO) Research Survey Pool to a nationally representative, random sample of 600 oncology providers. Main outcome measures included providers’ knowledge of SCT, frequency of initiating conversations about SCT with patients, and degree of provider support for its use. The survey investigated barriers to support of SCT and institutional scalp cooling offerings. Chi-square and Fisher exact tests were used for analysis. Using Bonferroni multiple comparisons correction, significance was defined conservatively at $p < 0.001$.

Results: One hundred fifty-five providers responded (response rate 25.8%). Whereas 62% (94/152) were in favor of SCT “always/most of the time”, only 26.3% (40/152) reported initiating discussions about SCT “always/most of the time”. Providers who treat breast cancer ($p \leq .0001$), those who report being “very” familiar with scalp cooling ($p \leq .0001$), those who report having read literature in the past 2 years about SCT ($p \leq .0001$), and those who work at a facility with machine scalp cooling systems ($p \leq .0001$) were significantly more likely to initiate conversations with patients about SCT. Financial concerns for the patient was the primary reason for not recommending SCT use.

Conclusion: Our findings suggest that oncology provider experience with SCT increases the likelihood of offering SCT to patients. These findings suggest that provider education could increase patient exposure to and use of SCT.
Zollinger-Ellison Syndrome: A Fasting Serum Gastrin Level is not Enough

Introduction: Zollinger-Ellison syndrome (ZES) is a rare condition with only 1-3 cases a year per million people due to gastrinomas causing refractory peptic ulcer disease (PUD). Diagnosis presents a challenge to clinicians given widespread use of proton pump inhibitor (PPI) therapy that often controls symptoms and masks the underlying disease process.

Case Description: A 64-year-old woman with a history of PUD on chronic PPI therapy presented for melena. Upper endoscopy (UE) was significant for multiple duodenal ulcers with biopsies negative for helicobacter pylori. Fasting serum gastrin (FSG) level was 212 pg/mL (13-115 pg/mL) which was deemed appropriate given current PPI therapy and was discharged on high-dose PPI therapy. Shortly thereafter, patient presented with hematochezia and UE revealed extensive duodenal and jejunal ulcers. FSG was 251 pg/mL and gastric pH 3.0. Secretin test was not performed due to risks of holding PPI therapy. Positron emission tomography-computed tomography (PET-CT) with DOTATATE showed a 2 cm nonspecific focus of intense uptake in the second portion of the duodenum. Given low suspicion for gastrinoma per radiology report, patient declined surgery for further evaluation. Within the month, she presented with severe abdominal pain and pneumoperitoneum on CT scan. Exploratory laparotomy identified multiple perforations requiring resection of the jejunum and duodenum. Pathology revealed a well-differentiated neuroendocrine tumor consistent with gastrinoma. Patient remains hospitalized for management of post-operative complications.

Discussion: Diagnosing ZES with mildly elevated FSG in setting of chronic PPI use is a challenging task. Historically, gastrinomas were expected to cause FSG >1,000 pg/mL but recent evidence have shown that FSG < 10-fold increase are seen in roughly two-thirds of ZES and are non-diagnostic. This case illustrates that evaluation for ZES requires looking beyond FSG and utilizing a thorough history, imaging, and endoscopic examination for diagnosis.
Feet Matter! Increasing Diabetes Foot Exams in a Residency Primary Care Clinic

Background: Diabetes foot exams play a major role in preventing complications and costs from diabetic neuropathy. A quality improvement project was created to improve the frequency of foot exams in our internal medicine residency primary care clinic.

Methods: We gathered data on 115 patients with diabetes seen from September to October 2020. 70% of patients received a foot exam, with 60% completed by residents (the remainder from podiatry), lower than the national standard of 74.8%. A survey was performed assessing resident understanding of foot exams. Based on the gaps identified, an intervention was created. This involved a multidisciplinary approach with videos and a demonstration on documenting foot exams in the electronic medical record (EMR).

Results: The initial survey showed 64% of residents did not know the components of foot exams and 46% did not know how to document exams in the EMR. After intervention, the post-survey noted 100% of residents know how to complete and document a foot exam. Furthermore, an increase in the performance rate of foot exams (74.19% patients received an annual foot exam and 70% of foot exams were completed by a resident) was noted. 80% of patients had appropriate care documentation and 61% of patients had an accurate physical exam documented. There were substantially decreased referrals to podiatry.

Conclusion: Education of residents and implementation of an intervention geared towards utilizing a multidisciplinary approach to integrate performing and documenting diabetic foot exams into the workflow at resident primary care clinic can improve screening rates.
Syncope with Severe Aortic Valve Stenosis, But Something Else was Hidden

Aortic valve stenosis (AVS) incidence increases with age and affects up to 10% of the population. Severe AVS disease is associated with exertional chest pain, dyspnea, lightheadedness, or syncope. Conduction abnormalities including third-degree atrioventricular (AV) block count for 14% of causes of syncope and can be easily missed when coexists with severe AVS.

We are presenting a case of syncope, where severe aortic stenosis could have masked a life-threatening third-degree AV block.

A 55-year-old male with a history of ascending aortic aneurysm presented with exertional lightheadedness for one month and one syncopal episode without exertion lasted for 5 minutes.

Physical exam: A 3/6 systolic, crescendo-decrescendo murmur best heard at the aortic area, radiates to both carotid arteries with late peaking. Labs were unremarkable. ECG: normal sinus rhythm with a ventricular rate of 90 and left axis deviation. Chest CTA showed ascending aortic aneurysm measuring 5.3 cm without dissection. Echocardiogram showed an ejection fraction of 61%, severe aortic stenosis: valve area of 0.82 cm², peak velocity 4.1 m/sec, and mean gradient 40 mmHg.

The patient was scheduled for aortic valve replacement (AVR) for symptomatic aortic stenosis. During his hospital stay and prior to the surgery, the patient had a similar syncopal episode. Telemetry revealed a third-degree AV block with a 15-second pause. The patient eventually underwent AVR and permanent pacemaker (PPM) placement and was discharged in stable condition.

Both severe aortic stenosis and 3rd degree AV block present with syncope. Although the coexistence of both diseases is extremely rare, the diagnosis of one of these pathologies does not rule out the other one. Our patient did present with both diseases at the same time. Treating his AS alone would have not been the curative treatment to his syncope, hence he was successfully treated with AVR and PPM placement.


Acute Hypercalcemia Due to Advanced Chronic Liver Disease without Hepatic Malignancy: A Rare Entity

INTRODUCTION: Hypercalcemia due to chronic liver disease (CLD) without liver malignancy is extremely rare. Very few cases have been reported. We present a case of decompensated CLD with end stage renal disease (ESRD) due to hepatorenal syndrome (HRS), with acute hypercalcemia of CLD.

CASE PRESENTATION: A 60-year-old man with Child-Pugh Class C alcoholic and hepatitis C Cirrhosis, and ESRD due to HRS, presented for paracentesis. Physical exam showed spider nevi, tense ascites and anasarca. He developed hypercalcemia acutely, recognized on routine laboratory studies: Calcium: 14.3 mg/dl (normal: 9-10.5 mg/dl), albumin: 1.5 mg/dL (low), Parathyroid hormone (PTH): 10 pg/mL (normal range: 10-65 pg/mL). PTH was 1,322 pg/mL, consistent with uncontrolled secondary hyperparathyroidism associated with ESRD, 2 weeks prior to admission during outpatient evaluation. PTH related peptide (PTHrP) level was 28 pg/mL (range: 14-27 pg/ml), which was slightly elevated but consistent with levels seen in ESRD. Alpha fetoprotein: 0.5 ng/mL (range: 0-8.9 ng/mL). Vitamin D-25-hydroxy: 15.4 ng/mL (low), vitamin D 1,25 dihydroxy: 10 pg/mL (low). CT Scan: Small nodular liver without masses. Serum protein electrophoresis, bone biopsy (done during surgery for compression fracture of L1 6 months ago) and serum light chains were unremarkable ruling out other etiologies for hypercalcemia. Peritoneal fluid did not show malignant cells. Patient was not on hypercalcemia inducing medications and was on regular physical therapy during hospitalization.

DISCUSSION: Hypercalcemia of advanced liver disease is poorly understood, is a diagnosis of exclusion and may be transient. Proposed mechanism is bone resorption due to elevated tumor necrosis factor, osteoclast activating factor, interleukins, and prostaglandins, and occurs regardless of CLD etiology. It is treatable, with reported good response to bisphosphonates and diuresis. Our patient was given intravenous bisphosphonate and dialysis, but he expired shortly due to advanced cirrhosis with HRS.
Life-Threatening Euglycemic Ketoacidosis from Empagliflozin in a Patient with Type II Diabetes Mellitus - A Rare Case Report

Introduction
Sodium-glucose cotransporter-2 inhibitors (SGLT2i) are associated with a low risk of hypoglycemia but with an increased risk of a rare, life-threatening complication of Metabolic acidosis (MA). SGLT2i are associated with Euglycemic Diabetic ketoacidosis (EKA) and concomitant use of Metformin causes Lactic acidosis, and when combined contribute to high anion gap MA. We present a case of EKA who was treated with Empagliflozin.

Case presentation
46-years-old man with DM-2 presented with shortness of breath and nausea. He was diagnosed with DM-2 two years ago and was on Metformin. He was started on Empagliflozin 3 weeks prior to presentation and noticed significant polyuria and polydipsia since then. He had severe nausea, vomiting, and shortness of breath when he presented to the Emergency department (ED). He denied any alcohol intake. In the ED, arterial blood gas revealed a pH of 6.9, bicarbonate of 3.8 meq/L, and lactate of 2.6 mg/dL. Anion gap was 29 at admission. Serum glucose was 198 mg/dl. Beta-hydroxybutyrate was elevated. HbA1c was 13.3%. Urinalysis and chest x-ray were normal. Electrocardiogram revealed sinus tachycardia. Procalcitonin was 0.35. He was started on insulin and bicarbonate drip for severe acidosis. He received 3 L of normal saline boluses, was started on a 5% dextrose 0.9% saline drip at 200 ml/hr. He was bridged to a basal-bolus insulin regimen once the anion gap was closed and insulin drip was discontinued.

Conclusion
EKA is a rare condition with anion gap acidosis without marked hyperglycemia. EKA can occur after any duration of SGLT2i use. With expanding indications and benefits of SGLT2i in heart failure and chronic kidney disease, further studies are needed to evaluate the risk versus benefit of combination therapy with metformin, since SGLT2i alone or in combination with metformin can be associated with life-threatening high anion gap MA.
Global Warming Mean Ticks More Active in Winter: A Case of Bannwarth Syndrome

Bannwarth syndrome a.k.a. lymphocytic meningoradiculitis is a less common manifestation of early disseminated Lyme disease (EDLD). It is characterized by painful radiculoneuritis with lymphocytic pleocytosis in the CSF. EDLD must be considered in endemic regions of Ixodes Scapularis irrespective of the season to initial prompt treatment and minimize long-term debility.

43 y.o. F camper presented to the ED with low back pain, saddle and genital anesthesia, stool incontinence, and urinary retention x 2 weeks (RN background, was straight catheterizing herself initially). Patient denied any tick bites but endorsed painful, burning radiculopathy with numbness along S1-S3 distribution. MRI brain/spine was negative for any enhancement and CT myelogram did not show any cord compression. Serum Lyme IgM panel positive (23 and 41 kD bands), IgG negative. Lumbar puncture demonstrated lymphocytic pleocytosis (WBC 11, 99% lymphocytes) and CSF Lyme EIA was positive with LIV >1.21. Mild symptomatic improvement was noted after the initiation of high-dose IV ceftriaxone but the patient continued to experience overflow incontinence and burning radiculopathy.

Bannwarth syndrome is more prevalent in Europe, however likely underdiagnosed in the USA. It is notoriously difficult to diagnose due to its myriad of symptoms that mimic other diseases. Lyme disease is uncommon during the winter months due to decreased survival of disease-carrying ticks; however, global warming may be playing a role in challenging this pattern. With an increasingly temperate climate during winter, it is imperative for physicians to keep EDLD on their differential to avoid delayed or missed diagnosis.
Flecainide & Beta Blocker, A Lethal Duo?

Introduction: Flecainide is a class IC antiarrhythmic agent used to treat atrial fibrillation in patients with preserved left ventricular function. Major adverse effects include life-threatening ventricular arrhythmias. We present a case of flecainide toxicity which was masked by beta-blocker overdose.

Case: 87-yr gentleman with medical history significant for chronic kidney disease, atrial fibrillation (on beta-blocker and flecainide) presented with progressing altered mentation and unresponsiveness. Vital signs significant for heart rate 46 but otherwise remained hemodynamically stable. EKG revealed atrial fibrillation with slow ventricular rate with irregular narrow QRS complexes measuring 89 ms. Initial laboratory values were significant for acute kidney injury with creatinine 2.49 (baseline 1.2). Atropine was administered for symptomatic bradycardia immediately after which EKG revealed bizarrely widened QRS complexes measuring 294 ms with underlying atrial fibrillation without any change in patient’s mentation. Decision was made to administer sodium bicarbonate immediately after which the patient transitioned to normal sinus rhythm which was observed on the telemetry. Patient also received IV hydration with NaCl for acute kidney injury caused by prerenal azotemia. Subsequently, his mentation improved and he remained stable during the hospital stay. Beta-blocker and flecainide were discontinued upon discharge.

Discussion: It is a well-known fact that beta-blocker overdose can cause symptomatic bradycardia which can be treated with atropine. Flecainide causes use-dependent sodium channel blockade and hence does not manifest when heart rate remains low. In our case, flecainide and beta-blocker toxicity were likely secondary to prerenal azotemia resulting in elevated serum levels of the medications. Treatment of bradycardia with atropine potentiated the effect of flecainide which resulted in wide QRS complexes. Treatment includes symptomatic support and administration of sodium bicarbonate for reversal of sodium channel blockade.
Double Antineutrophil Cytoplasmic Antibody (ANCA) Positive Crescentic Pauci-Immune Glomerulonephritis Due to Hydralazine

Introduction: Hydralazine is a known cause of drug-induced Lupus. It can also lead to acute glomerulonephritis with or without pulmonary involvement due to Antineutrophil Cytoplasmic Antibody (ANCA) mediated vasculitis. The incidence of hydralazine-induced ANCA-mediated vasculitis is proportional to the dose and duration of use, especially in slow acetylators. Incidence rate can be up to 10.4% in patients taking a dose of 200 mg/day for greater than three years. We present hydralazine induced double ANCA positive pauci-immune crescentic glomerulonephritis.

Case Description: An 82-year-old woman with difficult to control hypertension, hypothyroidism, and dementia presented with weight loss and generalized weakness for 6 months. Physical examination: Blood pressure: 179/77 mmHg, otherwise unremarkable. She was found to have acute kidney injury (creatinine 6.73 mg/dl). There was anemia (hemoglobin 7.7 g/dL). Urinalysis revealed > 50 WBC, > 50 RBC and 1+ bacteria. Ultrasound: Left pelviectasis without obstructive uropathy. Subsequent etiological work-up showed high titer anti-nuclear antibody, and both myeloperoxidase and proteinase 3 ANCA. Anti-dsDNA antibodies were positive. Complement levels were normal. Renal biopsy: Crescentic glomerulonephritis and membranous glomerulopathy, pauci-immune by immunofluorescence consistent with ANCA-mediated glomerulonephritis. Considering the renal biopsy findings and patient’s prolonged history of uncontrolled hypertension on hydralazine, the most probable cause of the ANCA-mediated pauci-immune glomerulonephritis was hydralazine. Hydralazine was discontinued. Patient received high-dose steroids and Rituximab. There was an adequate response noted after six weeks with creatinine improving to 1.91 mg/dl.

Discussion: Hydralazine-induced ANCA mediated vasculitis is more common in women and in patients with thyroid disease. Eighty percent of patients have renal involvement on initial presentation. Pulmonary-renal syndrome can occur. Hydralazine use, multiple positive antibodies, double positive ANCA with pauci-immune glomerulonephritis should point towards diagnosis. Discontinuing hydralazine and treatment with steroids, rituximab, and plasmapheresis for severe disease can be performed for improving outcome.
21-Year-Old Man with Abdominal Pain? Your Differential Should Include Diverticular Disease!

INTRODUCTION:
The prevalence of diverticulosis in industrialized countries ranges from 5 to 45 percent. Risk factors include low fiber, high fat and high meat diets. Among patients with diverticulosis, the lifetime risk of developing acute diverticulitis (AD) is 4 – 5%. The median age for AD in the US is 66. Most reported cases are in patients over 30 years of age; however, the incidence of AD in 18-44 year-olds has risen from 0.151 to 0.251 per 1,000 population in recent years.

CASE DESCRIPTION:
A 21-y.o. gentleman with no significant past medical history presented to the ED with left lower quadrant abdominal pain. Vitals on presentation were within normal limits. The abdomen was flat, had no scars, and was tender to palpation in the left lower quadrant. CT demonstrated AD. On further questioning, the patient was on a keto-diet. He played video games for recreation and participated in no physical activity. For the past several years he worked at McDonalds and only ate meat patties, avoiding fruits and vegetables. He improved with oral antibiotics conservative treatment.

DISCUSSION:
In the US, hospitalization rates for AD in patients younger than 45-years-old are increasing, with cases being reported in patients in their early 20's. Reasons for this demographic shift includes poor diet, decreased physical activity, and obesity. When compared to "Western" United States, "Southern", and "Midwest" States have higher rates of AD. This correlates with the higher rates of obesity in those regions.
Not Lupus Nephritis BUT A Rare Case of Drug Induced Pauci-Immune Glomerulonephritis

Hydralazine induced Anti-neutrophil cytoplasmic antibody (ANCA) associated vasculitis or Pauci-immune glomerulonephritis (PIGN) is a rare cause of glomerulonephritis. It is classified as rapidly progressive and life-threatening. Drug induced PIGN is associated with dual ANCA positivity and sometimes associated with anti-nuclear (ANA) or anti-dsDNA antibodies. Most cases are asymptomatic and has been confused with lupus nephritis. PIGN shows no or minimal evidence of immunofluorescence on renal biopsy, as in ANCA-associated glomerulopathies. This case highlights the importance of recognition of this disease.

80-year-old woman with a history of hypertensions and diabetes mellitus type 2 presented with generalized weakness and weight loss. She was on hydralazine for blood pressure for months. She did not use tobacco, alcohol, or illicit drugs. Physical exam was remarkable for alopecia and non-pitting edema. Laboratory evaluation showed an elevated blood urea nitrogen, creatinine and bicytopenia. Hematuria with proteinuria was found on urinalysis. Due to the progression of non-oliguric acute kidney injury needing hemodialysis, further serologic workup was initiated. Work-up showed positivity for ANA, proteinase 3 ANCA, myeloperoxidase ANCA, low complements and anti-dsDNA. Hepatitis B surface antibody was consistent with immunity. The patient was diagnosed with drug induced ANCA vasculitis. This etiology was confirmed with PIGN seen on biopsy. This is not lupus nephritis, which was the initial thought by the team. The patient was treated with RAVE protocol with discontinuing hydralazine.

It can be easily missed or confused with other diseases like lupus. Also, in PIGNs with few or no immune deposits is not seen in lupus nephritis but drug induced vasculitis. It has a similar serologic profile to lupus. This can result in inappropriate treatment with continuation of the offending drug. Almost equally important as the immunosuppressive treatment, is to identify and discontinue the offending drug in a timely manner to prevent rapid organ failure.
Well Done Steak, Pasteurized Milk, Hard-Boiled Eggs, and Washed Produce to Reduce the Risk of Prosthetic Valve Endocarditis

Introduction:
Listeria monocytogenes (LM) is a gram-positive rod found in raw and unprocessed food. Listeriosis usually causes fever and diarrhea. Listeriosis is severe in pregnancy, increasing fetal mortality, and is invasive in the elderly and immunocompromised leading to sepsis, meningitis, osteomyelitis, prosthetic graft infection, etc. The incidence of listeria endocarditis is 1-6% of all prosthetic valve endocarditis in the USA. We describe a case of prosthetic aortic valve endocarditis caused by LM.

Case Presentation:
A 73-year-old male with a bioprosthetic aortic valve presented due to feeling sick. Physical examination: BP 96/45 mmHg, afebrile, heart rate 58/min, a loud systolic murmur in the aortic area that radiated to the neck, leg edema. Chest x-ray showed pneumonia. He was started on azithromycin, ceftriaxone, and vancomycin. Transesophageal echocardiogram revealed an ejection fraction of 25 to 30%, with severe central aortic bioprosthetic regurgitation and flail left coronary cusp with a mobile filamentous echogenic mass attached to the aortic aspect. Blood cultures were positive for LM. Antibiotics were changed to ampicillin and gentamycin, and he was transferred to a tertiary care center for valve replacement.

Discussion:
Listeria is the third common cause of death due to foodborne illness, with a case fatality rate of 20%. LM endocarditis is typically subacute, is more common in males, most often affects abnormal native or prosthetic valves, and has a high mortality. Ampicillin and gentamycin are the agents of choice for treatment; LM is not susceptible to any cephalosporin. Trimethoprim/sulfamethoxazole, meropenem, and linezolid are alternatives when there is allergy.
The Unresolved Forearm Cellulitis and Abscess Despite Drainage and Antibiotics

Introduction: Actinomycosis is caused by actinomyces species, anaerobic gram positive bacteria colonizing oral cavity, colon, and vagina. Classic presentations suggesting actinomycosis include mass-like features, chronicity, and development of mucosal tracts. Most actinomycotic infections are polymicrobial. The common presentations are cervicofacial, thoracic or abdominal actinomycoses. Pelvic actinomycosis may occur with long standing IUD. We report a case of Actinomycosis forearm abscess in an intravenous drug user.

Case Description: A 36-year-old man presented to the Emergency Department with right forearm pain and swelling. Two weeks prior to presentation, he underwent incision and drainage and took clindamycin. Then, he had a sharp excisional debridement and given doxycycline. The lesion kept worsening. The patient admitted to injecting brown heroin mixed with tap water. Patient was afebrile with normal vital signs. Examination: 2cm x 4cm draining wound on the forearm with surrounding cellulitis. Laboratory evaluation: C-reactive protein: 14.2 mg/L (elevated), anemia of chronic disease, mild thrombocytopenia without leukocytosis. Wound culture showed Actinomyces and Beta hemolytic Streptococcus. CT scan showed a 1cm retained needle fragment with cellulitis and abscess. Patient was started on intravenous vancomycin and ampicillin/sulbactam. After another incision and drainage, patient was discharged on oral amoxicillin/clavulanate for 2 weeks.

Conclusion: Actinomycosis is uncommon in the forearm. In this patient, the retained foreign body and licking of needle probably contributed to it. A case series of soft tissue abscess in injection drug users reports Actinomyces odontolyticus in 15% of the patients. Actinomyces usually have companion bacteria (in our case streptococcus) which are copathogens that enhance the invasiveness and earlier manifestations of actinomyces. When abscess does not resolve despite adequate surgical debridement and antibiotics, especially in injection drug users, polymicrobial infection with actinomyces should be considered. Penicillin is the treatment, and this patient’s treatment failure is probably due to empiric antibiotics targeting staphylococcus.
Obesity and its Effects on Morbidity and Mortality in COVID-19 Patients: A Retrospective Cohort Study of Patients in Michigan

Objective
To analyze if obesity is an independent risk factor for intubation and mortality in patients with CoVID-19.

Methods
1227 patients from a Michigan hospital network were admitted for CoVID-19 between March 13th and May 15th, 2020 for inpatient care. Demographic data and hospital course was recorded for analysis. BMI was calculated and separated into 5 categories of underweight, normal, overweight, obese, and severely obese. Basic correlative analysis and multiple logistic regressions were used to ascertain significance of obesity on mortality and requirement for intubation.

Results
Compared against CoVID-19 hospitalized patients with normal BMI, patients who were underweight (BMI < 18kg/m2) and overweight (BMI = 25-30 kg/m2) carried significantly increased risks of mortality, (OR 2.216, p = 0.032) and (OR 1.647, p = 0.025), respectively. Increased mortality amongst patients with severe obesity (BMI > 35kg/m2) is approaching significance (OR 1.630, p = 0.075). Obesity and severe obesity also conferred a significantly higher risk of requiring intubation as compared with patients with normal BMI ((OR 2.15, p = 0.008) and (OR 1.993, p = 0.019)). Mean age in each of the 5 BMI categories were inverse correlated (p = < 0.0001). Other factors that significantly contributed to mortality were increasing age (OR 1.062, p = <0.0001), diabetes (OR 1.572, p = 0.010), history of CVA or TIA (OR 1.698, p = 0.016), and intubation (OR 6.442, p = <0.0001).

Conclusion
Patients being hospitalized with CoViD-19 with higher levels of obesity are at increased risks for mortality and intubation, independent of age, gender, race, and other comorbidities. The famed “Obesity Paradox”, which is thought to confer some protection from mortality against critical illness appears to be absent amongst hospitalized patients with CoVID-19.
Crescentic IgA Nephropathy Precipitated by E. coli Pyelonephritis: An Unusual Presentation and If Missed, May Lead to Permanent Renal Loss

Introduction
IgA nephropathy (IgAN), an immune complex–mediated disease, is the most common glomerulonephritis throughout most developed countries with prevalence as high as 20%. Approximately 40 to 50% present with one or recurrent episodes of gross hematuria, precipitated by concurrent upper respiratory infections or bacterial tonsillitis. Other infections, such as E. coli pyelonephritis, are rare precipitating causes. Early diagnosis and prompt treatment is essential and may prevent permanent renal failure.

Case report
A 28 y.o. 7-week pregnant lady was admitted with E. coli pyelonephritis. She denied any respiratory symptoms. Vitals were: heart rate 112 bpm and blood pressure 113/51. Labs revealed: BUN 13mEq/dL, Cr 1.95mEq/dL, Hgb 11.6g/dL, WBC 25.1 x 10^9/L, platelets 291 x 10^9/L. Urinalysis revealed red, cloudy urine with 3+ blood, >50 RBCs, pyuria, 3+ leukocyte esterase, nitrite positive and 2+ bacteria. She developed acute hypoxic respiratory failure. Pulmonary embolism, ARDS, and pneumonia were excluded. Her creatinine rose to 4.4mg/dl. She underwent hemodialysis. Subsequently, she developed hemoptysis. A kidney biopsy revealed crescentic IgA nephropathy. She was started on steroids, cyclophosphamide, and plasma exchange. Her kidney function normalized at discharge.

Discussion
IgAN typically presents after respiratory or gastrointestinal infections with gross hematuria, limited proteinuria (>1g/day), azotemia, RBC casts and dysmorphic RBCs in the urine. Other precipitating infections are very rare. Clues to the diagnosis include hematuria and hemoptysis developing after infections. Most cases are mild and self-limited. Patients developing crescentic disease have a poor outcome but respond well to immunosuppression, so early detection and treatment is imperative.
Myocardial Bridging of Left Anterior Descending Artery Presenting with Recurrent Chest Pain

Background: A myocardial bridge (MB) is a congenital defect of the major coronary arteries. Coronary arteries course along the epicardium. MB is when the myocardium overlies an epicardial coronary artery segment.

Case: A 61-year-old man with no significant past medical history presented with complaints of exertional chest pain and shortness of breath for 2-3 weeks provoked by his customary jogging, and relieved after rest. The physical exam was normal. Electrocardiogram (EKG) showed sinus rhythm, without ST changes and troponin was negative. Chest X-ray was normal. Exercise Stress test with myocardial perfusion imaging showed ischemia in inferior and lateral leads with multiple PVCs. Perfusion images were unremarkable. Echocardiogram showed an ejection fraction of 64% and a normal diastolic function. The patient underwent cardiac catheterization and coronary angiogram with intracoronary injection of nitroglycerine. The left main coronary artery (CA) was patent and bifurcated into the left anterior descending (LAD) and left circumflex CA. LAD showed minimal disease in the proximal segment. The mid to distal segments showed an intramyocardial course with significant myocardial bridging with almost complete obliteration of the vessel during systole. There was minimal epicardial disease. The right CA had minimal disease. The patient was treated with aspirin, statin, and beta-blocker (BB) and advised to avoid vigorous exercises.

Conclusion: MB can present clinically like obstructive CAD such as silent ischemia, stable angina, and acute coronary syndrome. Most patients remain asymptomatic until vessels are complicated by accelerated atherosclerosis. Medical treatment is with a BB or calcium channel blocker. Nitrates can accentuate the systolic compression of the bridged segment and thus not recommended. CA stenting in MB can lead to stent fracture and artery perforation. CA bypass grafting and surgical myotomy are the surgical approaches for severe cases, or in athletes who want to continue vigorously sports.
Late Detection of a Rare RCA Anomaly Leading to Permanent Bradycardia

Anomalous right coronary artery (RCA) is a rare congenital coronary abnormality that has been reported in less than 0.1 % of the population. Most commonly RCA can originate from the left cusp, posterior cusp or ascending aorta. We are presenting a case of an anomalous RCA originating from the LM artery becoming a challenge during cardiac catheterization.

A 63-year-old male with a past medical history of stage III chronic kidney disease, hypertension, and alcohol dependence, presented to the emergency department with sudden onset chest pain and acute encephalopathy. Vitals heart rate 20 bpm with normal blood pressure. Physical exam, Patient was somnolent but following commands, no murmurs were appreciated. ECG: junctional bradycardia with nonspecific ST-T changes, Troponin:0.04 ng/ml.

The patient underwent emergent transvenous pacer placement followed by urgent left heart catheterization showed a patent LM artery arising from the left coronary cusp, giving rise to a diffusely diseased left anterior descending artery (LAD) and left circumflex artery (LCX). RCA could not be engaged despite multiple attempts. Subsequently, a CT Coronary Angiogram (CTA) was performed and interpreted as an anomalous RCA originating from the left cusp. Due to persistent chest pain patient was taken to the catheterization lab. RCA was successfully engaged and demonstrated an anomalous take off from proximal LM with 99% ostial stenosis, stent was placed, patient was started on guideline-directed medical therapy (GDMT).

Due to persistent junctional bradycardia, most likely caused by delayed intervention on the RCA, a permanent pacemaker was placed.

Anomalous coronary arteries are a rare condition that can be a challenge during cardiac catheterization. This may lead to delay in diagnosis and eventually treatment, causing long term consequences. Thus, anomalous origin should be considered when RCA cannot be engaged through the right cusp during coronary artery catheterization.
A Complication of a Complication! Providencia rettgeri Cellulitis and Bacteremia Associated with Infliximab Treatment for Pembro

Introduction
Providencia species are gram-negative bacilli belonging to the Enterobacteriaceae family and have been isolated from urine, throat, perineum, axilla, and stool cultures. Providentia rettgeri (P. rettgerri) is usually associated with urinary tract infections in hospitalized patients with underlying comorbidities like cancer and immunosuppression. Cellulitis and bacteremia are rare but are significant due to underlying antibiotic resistance and high mortality. We present P. rettgeri cellulitis and bacteremia in a patient who was started on infliximab to treat steroid-dependent immune-related enterocolitis due to pembrolizumab.

Case presentation
A 72-year-old woman with metastatic non-small cell lung cancer was admitted with a painful purplish rash on the left lower extremity for 2 days. It was sudden onset, extending from the foot to thigh associated with multiple painful weeping blisters. She was undergoing treatment with carboplatin, pemetrexed, and pembrolizumab for lung cancer. She had multiple episodes of severe diarrhea and was diagnosed with immune-related enterocolitis from pembrolizumab. She was given prednisone. Colitis relapsed every time with prednisone taper, making her steroid dependent. This rash started soon after infliximab treatment for steroid-dependent enterocolitis. Skin biopsy showed cellulitis. Blood and wound cultures grew P rettgeri. Transthoracic echocardiogram was negative for vegetations. She was treated with intravenous ertapenem for a week and was discharged on oral levofloxacin.

Conclusion
Providencia infections are rare and only a few case reports of bacteremia have been published so far. Many Providencia isolates produce extended-spectrum beta-lactamases and are intrinsically resistant to polymyxins and tigecycline. Rapid progression of sepsis especially in the setting of immunosuppression and limited antimicrobial options highlights the necessity for early detection and treatment of Providencia bacteremia. Infliximab is associated with faster resolution of diarrhea in immune-related enterocolitis due to programmed cell death inhibitors but may increase the risk of life-threatening infections due to rare pathogens.
Reactive Angioendotheliomatosis Associated with Alcoholic Hepatitis in a Young Woman

Introduction:
Reactive angioendotheliomatosis is a rare disorder, associated with intravascular proliferation of endothelial cells. It can be idiopathic or associated with systemic diseases, like lymphoproliferative, autoimmune, hepatic, and renal disorders. We present reactive angioendotheliomatosis associated with alcoholic hepatitis.

Case Presentation:
A 28-year-old woman with alcoholic hepatitis presented with pain, erythema, and swelling of bilateral inner thighs for 2 weeks. She completed prednisolone for alcoholic hepatitis and was being evaluated for liver transplant. The lesion started as a dark spot, progressively worsened, without improvement after 5 days of cephalaxin. Physical examination: Normal vital signs, pallor, significant jaundice, distended abdomen with palpable splenomegaly and bilateral lower extremity edema. There was erythema of inner thighs, 8x5 cm on the right and 12x7 cm on the left with central black eschar and multiple small vesicles. Laboratory evaluation: White blood cells: 28,000/mcL, hemoglobin: 10.8 g/dL, with aspartate and alanine aminotransferase elevation consistent with alcoholic hepatitis, and total bilirubin 22.5 mg/dL. Patient was empirically started on vancomycin and piperacillin/tazobactam for suspected necrotizing fasciitis. Blood and wound cultures remained negative. Hepatitis serology, HIV test and autoimmune workup were negative. Imaging studies ruled out abscesses. Biopsy revealed ulcer with epidermal and dermal necrosis with intraepidermal vesicular dermatitis, and expert pathology review revealed angioendotheliomatosis. Antibiotics were discontinued, she was started on oral prednisone 60 mg daily. After three days of treatment, symptoms improved. She was discharged on oral prednisone 60 mg for 1 week followed by a tapering dose.

Discussion:
Identification of reactive angioendotheliomatosis is critical to provide appropriate treatment and avoid unnecessary prolonged antibiotics. Reactive angioendotheliomatosis is hypothesized to occur due to tissue hypoxia and the growth factors that are released in response to the tissue hypoxia. It may resolve spontaneously or to treatment with steroids or laser, though some cases are refractory.
MINOCA

Myocardial infarction with non-obstructive coronary artery disease (MINOCA) is a clinical syndrome reported more often recently but yet no definitive treatment guidelines have been established. Treatment focuses on underlying etiology. MINOCA is usually caused by plaque disruption, coronary microvascular dysfunction, spontaneous coronary artery dissection, coronary vasospasm or thrombosis. We are presenting a case of MINOCA purely due to microvascular dysfunction.

A 49 year-old-female with past medical history of bipolar disorder and GERD presented with substernal chest pain, lightheadedness and palpitations.

Examination: Blood pressure: 120/80 mmHg, Pulse 90/min, normal cardiovascular examination. ECG: Normal. Laboratory evaluation: Troponin 10.62 ng/ml and peaked at 20.98ng/ml (normal: <0.03 nn/ml), normal lipid profile and ESR. Echocardiogram: Left ventricular ejection fraction 55% with normal diastolic function. CT angiogram was negative for pulmonary embolism and aortic dissection.

Patient was started on nitroglycerin and heparin drips. Patient then had coronary angiogram that showed minimal coronary artery disease, ventriculogram showed no evidence of Takotsubo cardiomyopathy.

MINOCA was diagnosed since patient did not have obstructive coronary disease and other causes for elevated troponin were excluded (Pulmonary embolism, Aortic dissection, Myocarditis).

Dual antiplatelet therapy (Aspirin, Clopidogrel), Atorvastatin and Metoprolol were given, chest pain resolved and patient was discharged home in stable condition.

Our case highlights MINOCA which was treated with DAPT, statin and B-Blocker, based on limited evidence. ACE inhibitor was not added due to hypotension.

MINOCA should be considered in younger females without dyslipidemia or other risk factors of coronary artery disease who present with an acute coronary syndrome. Hypercoagulable states, coronary artery dissection and coronary vasospasm should be considered as causes. Coronary microvascular disease is the etiology considered in our patient as we did not find any other mechanism.
Does the Use of Different Modes of Communication Affect Resident Education During Patient Emergencies?

Introduction: Expectations regarding preferred method of contact differ between residents and attending physicians and can lead to medical errors, especially in emergency situations. Rapid response team (RRT) residents voiced concerns that they had decreased contact with private attendings during RRT encounters, leading to worse patient outcomes. Is there a difference in patient acuity and communication patterns between private and faculty teams during an RRT encounter?

Methods: In a retrospective study, 320 RRT encounters were analyzed. We compared mean illness severity before and during the RRT encounter for private and faculty groups, using the National Early Warning Score (NEWS). Next, we compared reason for RRT and disposition at discharge for both groups. Finally, we conducted a survey of 19 resident physicians and 46 attending physicians on preferred method of communication and perceptions on communication during an RRT.

Results: We found no significant difference between the private and faculty teams in patient acuity at admission (p=0.515) and at time of RRT (p=0.703). There was no significant difference in reason for RRT between the two groups (p=0.452). Disposition at discharge was similar for both groups (p= 0.298). Survey results showed that attending physicians preferred to use answering service or pager, while residents preferred to use cell phone. Most residents believed it was important to speak to the attending during an RRT, however 32% stated they felt uncomfortable doing so.

Conclusion: There was no significant difference in RRT characteristics for the patients of private and faculty groups. Perceived difference in patient acuity may be related to familiarity and increased ease of communication with faculty over private attendings. Regardless of level of communication and support of resident physicians during an RRT, patient outcomes were comparable for both groups. A universal method of communication would be beneficial in reducing resident uncertainty.
INTRODUCTION
An estimated 27.8% of the US adult population over the age of 40 years use statins. The US is the largest grapefruit producing country in the world. US per capita grapefruit consumption has fallen from a peak of 24.6 pounds in 1978 to 5.51 pounds in 2013. One explanation is the reported risk of adverse effects associated with grapefruits and statin use. Is this drop warranted? What is the magnitude of grapefruit - statin interaction? Is this interaction clinically significant?

CASE
A 58-year-old female presented to the clinic for a periodic health evaluation. Her atorvastatin was held for 9 months earlier due to elevated liver enzymes. Recent labs revealed normalized AST and ALT but an LDL of 158. She was asked to resume her statin. She lamented that she would enjoy one last grapefruit before resuming her atorvastatin.

DISCUSSION
The interaction of grapefruit and statins was first described in 1989. Furanocoumarin, an inhibitor cytochrome CYP3A4, was identified as the putative agent. Follow up studies demonstrated consumption of one grapefruit (equivalent to 8 ounces of juice), taken concomitantly with 40mg of simvastatin, increases area under the curve (AUC) by 3.6-fold. Similarly, grapefruit consumption with 10mg of atorvastatin and 40mg of lovastatin elevated AUC by 1.8-fold and 1.9-fold, respectively. Other statins such as rosuvastatin, pravastatin, and fluvastatin are not metabolized via the CYP3A4 system and have no interactions with grapefruit ingestion. A review of medical literature revealed only one case of grapefruit induced rhabdomyolysis. A patient on 80mg simvastatin began consuming grapefruit daily. After 2 weeks she developed rhabdomyolysis.

CONCLUSION
Grapefruit consumption does increase plasma concentrations of certain statins. Clinical complications are very rare. If frequent consumption of grapefruit is anticipated, use of atorvastatin, pravastatin, or fluvastatin is recommended.
Occam’s Razor: A Case of Reversible Paralysis

Introduction:
Thyrotoxic Periodic Paralysis (TPP) is a preventable, curable and sporadic form of Periodic Paralysis seen in young Asian men, often as a first manifestation of hyperthyroidism. It is a triad of acute hypokalemia, muscle paralysis and thyrotoxicosis.

Case description:
A 37-year-old otherwise healthy Asian male presented to the Emergency Department with sudden onset painless bilateral lower extremity weakness. He woke up with tingling and numbness over bilateral lower extremities with inability to stand up, having to crawl out of bed in the morning for help. No personal or family history of prior similar episodes.

Examination: Normal vital signs, decreased strength both lower extremities, worse in proximal than distal. Laboratory evaluation: profound hypokalemia to 1.7 meq/L, which did not improve despite adequate intravenous potassium chloride and mild hypomagnesemia (1.4 mg/dL). Further investigation revealed TSH < 0.03 mIU/L (range: 0.2-4.5 mIU/L) with Free T3 16.41 pmol/L (range: 2.0-7.0 pmol/L) and creatinine kinase elevated at 2000 mg/dL. MRI of cervical/thoracic/lumbar spine were unremarkable. Due to profound thyrotoxicosis, propranolol and methimazole were started which led to significant improvement in clinical symptoms as well as serum potassium levels without further supplementation. He then had rebound hyperkalemia which is seen in approximately 40-59% of treated attacks of TPP.

Discussion:
TPP is usually seen in Asian men, more commonly after age 20. Affected patients are found to have KCNJ2 gene mutation which encodes for a potassium channel in muscles, regulated by thyroid hormone. Attacks are precipitated by epinephrine release after exercise, carbohydrate load or stress which causes augmented intracellular shift of potassium causing hypokalemia and muscle weakness. It is often associated with mild hypomagnesemia and hypophosphatemia which may help distinguish TPP from hypokalemic periodic paralysis. Preventive treatment includes propranolol as well as maintaining euthyroid state.
INTRODUCTION
According to Homeland Security, over one million foreign nationals were granted permanent residence in the US in 2019. Of this group, 352,593 arrived from Asia. Immigrants frequently seek primary care physicians and undergo age and sex appropriate cancer screening measures. Cancer rates vary around the world. Should recent immigrants undergo screening procedures based on current US guidelines, or should physicians modify screening recommendations based on the patient’s country of origin?

CASE
A 52-year-old female, who recently emigrated from China, is seen for a preventive visit. She has lived in the US for approximately 2 years. Her only complaint is persistent pyrosis. Her physical exam is unremarkable. A PAP smear is performed, and a mammogram and colonoscopy are ordered as part of age and sex appropriate cancer screening.

DISCUSSION
Preventive medicine attempts to limit the 5 D’s: disease, disability, discomfort, dissatisfaction and death. Three criteria are utilized to determine if preventive measures are warranted: the burden of suffering caused by the condition, the performance of the screening test and the effectiveness, safety and cost of the preventive intervention or treatment. The frequency of a condition plays a pivotal in determining the benefits of screening. Screening for rare conditions may benefit a limited population, but may generate excessive false positive results requiring additional testing and surging patient angst. The prevalence of cancer rates (reported as cases per 100,000) varies by country. The colon cancer prevalence is 17.52 in China and 36.8 in the US. Breast cancer is 21.6 in China and 125.1 in the US. Gastric cancer is 29.31 in China and only 6 in the US. Our patient was recommended to have an EGD for her heartburn symptoms and high risk for gastric cancer.

CONCLUSION
Physicians should account for patient’s country of origin when performing preventive cancer screening.
Is Endometrial Biopsy Enough for Diagnosis of Endometrial Carcinoma in Post-Menopausal Bleeding? A Case of Clear Cell Carcinoma

Introduction: Endometrial carcinoma is the most common gynecological malignancy in the United States. Clear Cell Carcinoma (CCC) of endometrium is an estrogen independent endometrial cancer which accounts for < 5% of endometrial cancers. About 45% of patients have extra-uterine metastases at diagnosis. Current guidelines recommend ultrasound for post-menopausal bleeding and a biopsy for those with endometrial thickness >5mm. However, biopsy failed to make the diagnosis in our patient.

Case Presentation: A 67-year-old female with a past medical history of goiter, uterine fibroid, factor XII deficiency, and hypertension presented to our clinic for periodic postmenopausal spotting for 5 to 6 years which has worsened recently. Prior transvaginal ultrasound (TVS) and biopsy was negative for malignancy and showed endometrial hyperplasia and multiple fibroids. She was closely followed with cervical papanicolaou smears. Repeat TVS showed increased endometrial stripe (2.4 cm to 2.6 cm). Biopsy was again negative for malignancy, but specimen was scant. Dilatation and curettage with hysteroscopy were performed which showed endometrioid adenocarcinoma with myometrial invasion. Vaginal hysterectomy with bilateral salpingo-oophorectomy and lymph node dissection was done which showed clear cell carcinoma of endometrium, with 44% myometrium involvement, cervical involvement and no lymph node involvement suggesting International Federation of Gynecology and Obstetrics (FIGO) stage II. She recovered from surgery and currently is undergoing brachytherapy.

Discussion: Patient’s spotting was attributed to fibroids and factor XII deficiency after negative biopsies twice. Worsening bleeding and scanty tissue in second biopsy lead us to do invasive biopsy. In addition, Factor XII deficiency does not cause clinical bleeding, though associated with prolonged partial thromboplastin time. Risk factors for CCC are unknown. Further studies are needed to identify risk factors for CCC, and to create a better screening tool or a scoring system for early detection to improve survival.
A Rare Case of Erlotinib-Induced Pneumatosis Intestinalis in a Patient with EGFR Positive NSCLC

Introduction
Pneumatosis intestinalis (PI) is a rare adverse effect reported for molecular-targeted agents such as Erlotinib, presenting as a significant gas accumulation in the small bowel. Clinical symptoms typically present as abdominal pain, diarrhea, nausea and vomiting. We present a case of PI manifesting in a patient taking long standing erlotinib for stage IV NSCLC.

Case Description
An 84-year-old female with stage IV, metastatic, EGFR positive NSCLC on erlotinib for several years was admitted for evaluation of left lower quadrant abdominal pain, nausea, vomiting and diarrhea. Patient has a history of prior hospitalizations most recent for small bowel obstruction s/p laparotomy four months prior. CT of the abdomen showed extensive pneumatosis involving the distal small bowel. CTA findings were noted to be progressive from prior hospitalization in March. Other workup for the patient’s imaging findings did not provide an etiology for the patient’s symptoms. Erlotinib was held and the pneumatosis markedly improved in the following days. Since other workup was non-contributory to finding an etiology and the patient showed marked improvement off Erlotinib, her symptoms could likely be associated to long-standing Erlotinib use.

Discussion
The mechanism of pneumatosis intestinalis associated with Erlotinib induced molecular therapy is yet unknown. Immediate management of patients presenting with PI associated with Erlotinib should typically include holding the medication and providing supportively management for clinical symptoms. Patients could be transitioned to another anti EGFR medications such as Gefitinib, which have been reported in literature, for long term management of patient’s NSCLC.
The Use of Exercise as an Adjunctive Therapy for Major Depressive Disorder: A Critical Appraisal

BACKGROUND: Major depressive disorder (MDD) affects over 350 million people worldwide, making it the most common diagnosis in primary care and cause of disability worldwide. The World Health Organization recommends antidepressant plus cognitive behavioral therapy as first-line treatment for MDD, though treatment failure and relapse rates remain high. In 2013, exercise emerged as recommended adjunctive therapy.

OBJECTIVES: Our project aims to answer the question: In adults with MDD, does medical therapy with adjunctive exercise versus medical therapy alone lead to a statistically significant improvement in ratings of depression?

METHODS: Authors performed a literature search within Pubmed for articles comparing medical therapy with exercise versus medical therapy alone. From this search, four articles were selected including a systematic review, a meta-analysis, and two randomized control trials.

RESULTS: Medical therapy with adjunctive exercise was shown to improve subjective ratings of depression in all studies evaluated, however the difference when compared to medical therapy alone was not significant in the two RCTs and trended towards significance in the systematic review and meta-analysis. In the meta-analysis, the effect size was moderate, but not significant with a g-value of -0.50 and p-value of 0.11. However, only four studies were included. Generally, the studies had a smaller sample size, but the trend towards significance is promising and requires follow up.

CONCLUSIONS: Overall, exercise as an adjunct to standard therapy improved symptoms of depression, though statistical significance varied study to study. Secondary outcomes showed that structured exercise led to lower required doses of antidepressants. Applicability becomes a concern as one of the core symptoms of depression is anhedonia. Furthermore, from a U.S. centric position, exercise can be expensive and difficult to sustain. Since exercise does show clear benefit, we would like to see it integrated into standard medical treatment more heavily.
**Spontaneous Pneumothorax in a Patient with COVID-19 Managed with Chest Tube**

**Introduction:**
Spontaneous pneumothorax (SP) is defined as the presence of air in the pleural space without an obvious precipitating event. In the general population, primary SP has an incidence of 7.4 to 18 cases per 100,000 males. It typically occurs in tall, thin males with cigarette smoking being an additional risk factor. Barotrauma was reported in 15% (89/601) of COVID-19 patients who required mechanical ventilation. However, SP in COVID-19 patients who are not mechanically ventilated has not been frequently reported and is estimated to be around 1%. We report a case of SP requiring chest tube placement in a COVID-19 patient who was not on mechanical ventilation.

**Case Presentation:**
A 68-year-old 6 foot tall, Caucasian male with obesity (BMI:30), hypertension, hyperlipidemia, former cigarette smoker (12.5 pack years) was admitted for acute hypoxemic respiratory failure secondary to COVID-19 pneumonia. Initial management included oxygen therapy through high flow nasal cannula (HFNC), 5 days of IV Remdesivir and 2 days of IV Methylprednisolone. Patient’s D-dimer up-trended from 227 ng/mL to 662 ng/mL. He became progressively hypoxic despite on HFNC. Therefore, a chest CT angiogram was ordered which was remarkable for a 3.1 cm right-sided pneumothorax. A chest tube was placed with subsequent x-ray showing improvement in the pneumothorax. His condition slowly improved and his chest tube was subsequently removed. Patient was later discharged home on supplemental oxygen.

**Discussion:**
SP in COVID-19 patients not on mechanical ventilation has not been frequently reported and only a few patients were found on our literature review. These cases were primarily treated with chest tube placement. We believe that COVID-19 infection might have contributed to the development of SP in our patient. SP should be considered as a differential diagnosis in a patient with COVID-19 with increasing oxygen requirements.
The Clot Thickens: Pylephlebitis as a Complication of Sigmoid Diverticulitis

Introduction:
Pylephlebitis is defined as suppurative thrombosis of the portal mesenteric venous system and exists as a rare and potentially fatal complication of intra-abdominal infections with a mortality rate of up to 25%. However, with early recognition and targeted treatment a favorable outcome can be achieved. Below, we outline a case of pylephlebitis that emerged as an unconventional complication of acute sigmoid diverticulitis.

Case Description:
A 59-year-old male with diverticulosis presented with fevers and generalized abdominal pain. CT abdomen on admission suggested sigmoid diverticulitis with surrounding mesenteric venous gas. Hospital course was complicated by recurrent fevers, rigors, worsening abdominal pain and acute respiratory failure requiring mechanical ventilation. Subsequent abdominal CT demonstrated worsening mesenteric ischemia and portal vein thrombosis, suggestive of pylephlebitis. Thereafter, anticoagulation with heparin was initiated and subsequent exploratory laparotomy with sigmoidectomy and descending colostomy was performed. Blood cultures grew Enterococcus faecalis and Candida glabrata and treatment involved ampicillin, ceftriaxone, metronidazole, and fluconazole. On discharge, the patient was transitioned to apixaban and antimicrobial therapy was changed to 6 weeks of ertapenem, vancomycin and fluconazole. The patient underwent colostomy reversal three months later with follow up CT showing interval improvement of the portal vein thrombosis.

Discussion:
Symptoms of pylephlebitis can be non-specific, thereby making its diagnosis elusive. This can lead to delayed recognition and a more complicated clinical course. While standard of care includes targeting the underlying infection through surgery and antibiotics, the role of anticoagulation remains unclear. We describe the use of oral anticoagulation with apixaban in conjunction with early source control in helping to achieve portal vein recanalization. In addition, we emphasize the importance of having a high index of suspicion for pylephlebitis, allowing for prompt intervention and targeted treatment to decrease downstream sequelae of the disease.
A Rare Case of Focal Segmental Glomerulo-Sclerosis as Renal Manifestation of SARS-COV-2 Infection

Introduction: Studies showed several extra-pulmonary manifestations of SARS-COV-2 infection, explained by systemic inflammatory response and interaction of virus with ACE-2 receptor widely distributed including lungs, kidney and ileum. Most common renal complications include electrolyte imbalance, acute kidney injury (AKI) and renal replacement therapy. We present case of Glomerulopathy in the setting of AKI and SARS-COV-2

Case Report: 47-year-old male with known PMH of DM-2, CKD-stage-2 came to ED with one-week history of fever, and malaise. Initial vitals showed tachypnea and tachycardia, SpO2 96% on room air. Investigations showed AKI, with BUN 28.5mg/dl and creatinine 1.92mg/dl, and no leukocytosis. Inflammatory markers including were elevated. Chest X-ray showed basal zone opacification bilaterally. Qualitative-PCR for SARS-COV-2 resulted positive. Treatment with IV-antibiotics, zinc tablet and Normal-Saline infusion. Although patient did not develop hypoxia, renal function continued to worsen during hospitalization. Urinalysis showed proteinuria 3+ and raised urine Protein:creatinine-ratio 11.48. Renal biopsy was performed which suggested collapsing-type Focal segmental Glomerulosclerosis(FSGS). Complement levels were slightly elevated, workup for FSGS including Hepatitis-B antigen was negative, and COVID-19 was suggested as likely etiology. Ongoing management involved hemodialysis corticosteroid therapy for nephrotic syndrome. Rapid recovery of renal function was observed. Patient continued with out-patient hemodialysis therapy for 3-weeks. Follow up over 6 months showed patient is maintaining creatinine at baseline level of 1.2mg/dl with GFR 70.

Discussion: AKI in SARS-COV-2 infected patient is commonly a combination of intrinsic disease that is non-ischemic acute tubular necrosis and hemodynamic factors. Nephrotic-level Proteinurinra is discovered as a common presentation especially in critically-ill patients. The rare finding of FSGS, can be explained by direct SARS-COV-2 invasion via ACE-2 receptors present on podocytes, like other viral-based glomerulosclerosis such as HIV and CMV especially in patients with APOL1-gene variant. Persistent decline in renal function, should suggest work up for other intrinsic renal pathologies including glomerulopathies.
A Rare Case of Malignant Carcino-Sarcoma and Pulmonary Hypertension in a Female with Neurofibromatosis Type 1

Introduction: Neurofibromatosis Type-1 (NF-1) is an autosomal-dominant ectodermal and mesodermal dysplasia syndrome with distinctive presentation of diffuse neurofibromas, café-au-lait spots and lisch nodules. Pulmonary manifestations reported in 10-20% patients commonly includes bullae/cysts in apical lobes and basal reticulations. We present a rare case of multiple, malignant solid pulmonary masses with progressive pulmonary hypertension linked with NF-1.

Case Report: 39 year old female with past medical history of NF-1 with neck mass status-post radiation, airway compromise and dysphagia secondary to neck mass status-post chronic tracheostomy and PEG-tube and multiple nerve-sheath neurofibromas admitted from nursing facility for evaluation of dislodged PEG-tube. On examination, she was afebrile, had chronic sinus tachycardia, and was requiring 8L Oxygen supplementation via trach-collar. She had strong family history of NF-1 and classic lisch nodules, café-au-lait spots on exam and was non-verbal at baseline. Lungs were clear to auscultation bilaterally. Labs revealed leukocytosis and elevated D-dimer but CTA-chest was unremarkable for pulmonary embolism while duplex-ultrasound of right upper extremity showed acute occlusive-DVT and patient was started on a heparin drip. CTA-chest also showed multiple solid circumscribed lesions throughout the lung. Transbronchial biopsy confirmed malignant sarcomatoid neoplasm consistent with malignant nerve sheath tumor. Echocardiogram also showed new findings of moderate pulmonary hypertension with RVSP of 48mmhg. She was scheduled for radiation to neck and chest at her primary hospital.

Discussion: NF-1 lesions can present on the neuraxis as well as oropharynx, larynx and gastrointestinal-tract. Solid tumor presentation in lungs is rare and usually benign. Findings of malignant sarcomatoid neoplasm with poor clinical outcome suggests that solid lesions in a patient with NF-1 require high suspicion, screening, and strict follow-up. Pulmonary hypertension is rare sequelae with female predominance, and pathophysiology involves NF-1 gene mutation along with unidentified factors of pulmonary artery remodeling. Diagnosed cases require early transplantation among eligible patients.

BACKGROUND: Throughout US history, women have been underrepresented in medicine, especially in procedural fields. In 1980, women accounted for 29.5% of medical students. In 2005, females surpassed 50% of medical student enrollment and have remained between 46-51%. We aimed to review the closing of the gender gap in medical specialties with specific interest in procedural Internal Medicine subspecialties and surgical specialties.

METHODS: We obtained data from the National Resident Matching Program (NRMP) and Association of American Medical Colleges (AAMC) websites from 2007-2017. Physician specialty data is published by AAMC every 2-3 years, providing the percentage of women in each specialty as active physicians and in training. The NRMP publishes data on resident and fellow matches, publicly available from 2016-2020. Using simple descriptive analysis, we generated trends of women in various specialties. AAMC and NRMP were unable to provide further historic data.

RESULTS: Women have been closing the gap in procedural medicine since 2007. The foremost example is general surgery with 40% female residents in 2017 and a rise of an average of 0.9% a year of female trainees over 10 years. Internal Medicine procedural fields are lagging in the rate of growth, although continue to have an uptrend of 0.7% a year average in critical care medicine, 0.4% a year growth in cardiology, and 0.3% a year growth in gastroenterology over 10 years. In 2017, cardiology fellowships were 23% women, critical care 33%, and gastroenterology 34%.

CONCLUSION: There has been a steady increase in female representation in procedural subspecialties since 2007. General surgery has had the most steady and significant increase compared to Internal Medicine subspecialties. More research is warranted in evaluating the growth of female enrollment in general surgery and evaluating how Internal Medicine procedural fields can aim to recruit more women in their fellowships and close the gap.
Bleeding Mystery in a Thrombogenic Disease

Introduction: Myeloproliferative neoplasms are associated with an acquired hypercoagulable state, especially for patients with the JAK2 V617F mutation. However, we present an atypical case of a patient with myelofibrosis who presented with a spontaneous iliopsoas hematoma relating to an acquired bleeding diathesis.

Case: A 44-year-old female with JAK2 V617F positive post-polycythemia vera myelofibrosis and beta-thalassemia minor presented with left lower extremity pain. Exam was notable for femoral neuropathy. Imaging showed a large left iliopsoas hematoma. Her coagulation studies were notable for a prolonged partial thromboplastin time (PTT) but normal prothrombin time. She had normal factor VIII, IX, and XI levels.

In follow-up, labs showed a low factor XII level as the cause of her prolonged PTT. However, factor XII deficiency is not associated with bleeding. Additional testing was pursued that showed evidence of acquired von Willebrand (VWD) syndrome. Noting that a spontaneous intramuscular bleed was still unusual for VWD syndrome, platelet function studies were performed. These showed decreased platelet aggregation and secretion. Platelet electron microscopy confirmed a concomitant combined dense and alpha granule deficiency, suggestive of an acquired platelet storage pool disorder.

Given her symptomatic splenomegaly despite ruxolitinib, she underwent splenectomy with von Willebrand factor (VWF) concentrate and platelet transfusion support. She then was treated with a bone marrow transplant. Repeat testing post-transplant did not demonstrate evidence of acquired VWD, alpha or dense granule deficiency, or beta-thalassemia minor. Her factor 12 deficiency persisted.

Discussion: While one of the feared complications of myeloproliferative disorders is thrombosis, this case highlights that acquired bleeding disorders are also possible. Furthermore, while spontaneous intramuscular bleeding is most often associated with a clotting factor deficiency, it is important to consider the potential of multiple concomitant hemostatic abnormalities when evaluating such abnormal bleeding.
An Unusual Migraine: Acute Disseminated Encephalomyelitis in an Adolescent

Case Presentation:
A 16-year-old female immigrant from Syria with a history of aseptic meningitis at 4-months of age presented to her Pediatrician with 4 days of a frontotemporal headache with photophobia, phonophobia, and fatigue. Neurologic exam was non-focal and she was prescribed sumatriptan for presumed migraine. The following day she developed nausea and vomiting, and was evaluated in the ER where brain MRI revealed vasogenic edema and multiple areas of hyperdensity in both cerebral hemispheres. Lumbar puncture (LP) was positive for Varicella Zoster Virus (VZV) by PCR. She received 10-days of IV Acyclovir with symptomatic improvement and discharged home. Two months later, she presented to the ER again with headache, photophobia, and phonophobia. Brain MRI demonstrated increased regions of edema and enhancement, with CSF negative for VZV by PCR although oligoclonal bands were elevated. She received a 5-day steroid burst followed by 2 days of immunoglobulins (IVIG) for suspected post-viral Acute Disseminated Encephalomyelitis (ADEM) with subsequent imaging confirming regression of edema and lesions.

Discussion:
This represents an interesting case of VZV encephalomyelitis followed by ADEM in an adolescent presenting with migrainous symptoms. The case was reviewed by tumor board and neuroradiology, with low concern for CNS lymphoma or multiple sclerosis. ADEM is a diagnosis of exclusion, made based upon clinical and radiologic findings following a negative work-up for infectious, vasculitic, demyelinating, and other CNS inflammatory syndromes. The majority of ADEM cases are post-infectious. Treatment includes high-dose IV corticosteroids, IVIG, and plasma exchange.

Conclusion:
A non-focal exam does not exclude encephalomyelitis or vasogenic edema, and such diagnoses should still be considered in a patient presenting with severe migrainous symptoms without response to conventional treatment. Additionally, suspect ADEM in a child or adolescent with multifocal findings on MRI in a post-infectious period after completing a comprehensive work-up to exclude other causes.
A 34-year-old female with ulcerative pancolitis on infliximab and azathioprine presented to her gastroenterologist with several weeks of fevers, abdominal pain, nausea, and diarrhea. Initial labs were remarkable for a C-reactive protein of 4.2 mg/dL. CT enterography demonstrated interval development of proximal ileitis and a short segment stricture without obstruction. This new finding raised suspicion for Crohn’s disease; however, further workup with retrograde double balloon enteroscopy (DBE) was unrevealing. Her symptoms progressed, and empiric steroids were trialed with partial improvement. Given her persistent symptoms and unusual pattern of inflammation, anterograde DBE was performed and revealed moderate inflammation and ulceration in the proximal ileum. Histology demonstrated inflammation with numerous intracellular fungal organisms consistent with Histoplasma capsulatum on silver stain. She was subsequently admitted for 14 days of treatment with amphotericin B and discharged on a prolonged course of oral anti-fungal therapy. Infliximab was transitioned to vedolizumab due to the better safety profile with a gut-selective mechanism of action. The patient responded to treatment with near resolution of symptoms and improvement in serum histoplasma antigen levels.

Histoplasma capsulatum is a dimorphic fungal organism endemic in much of the Midwest. While histoplasmosis classically involves the lungs, involvement of the gastrointestinal tract, skin, central nervous system, adrenal glands, and heart have also been reported, often making it difficult to distinguish from other diseases processes. Additionally, patients on anti-tumor necrosis factor agents such as infliximab are at increased risk of H. capsulatum infection and disseminated disease. Our patient was not only more susceptible to histoplasmosis, but the infection was able to masquerade itself as the very disease infliximab was meant to treat. This case illustrates the importance of pursuing a comprehensive work-up in inflammatory bowel disease patients where the signs, symptoms, and radiological finds do not fit the typical established disease pattern.
Knee Pain - It's Not Always Patellofemoral Pain (Syndrome)

Avascular necrosis (AVN) is a condition caused by disruption of the blood supply to a bone ultimately resulting in bone death. It can occur in any bone, but if involving a joint, cartilage damage and subsequent joint collapse can occur.

A 22-year-old female presented to her primary care provider for four months of intermittent posterior right knee pain, starting after kickboxing without discrete injury. Exam was noted to be unremarkable and imaging was not obtained. Conservative treatment was recommended. Pain continued with impact activities such as running, kickboxing, and lunging motions with squats/weightlifting. She subsequently presented to urgent care for worsening pain where radiographs were done and reportedly normal. She was referred to Sports Medicine, now 5 months after onset. In addition to continued symptoms, she complained of mechanical symptoms and difficulty with ADLs. Review of history revealed prednisone use for 10 years for pemphigus vulgaris. Exam showed tenderness of the lateral joint line, normal range of motion, no effusion, and negative special tests. Knee radiographs including notch view, demonstrated a subchondral linear lucency in the lateral femoral epicondyle concerning for AVN. Follow up MRI confirmed the diagnosis. She was referred to orthopedics. Given age, job, and significant involvement of AVN, non-operative management with aquatic physical therapy, lateral unloader brace, and modified activity was pursued and symptoms improved.

The cause of AVN may be unclear, though there are many risk factors, including chronic/frequent steroid use. This case illustrates the importance of reviewing medications and history when evaluating a patient for musculoskeletal (MSK) complaints. Additionally, X-ray imaging with proper views should be obtained in MSK cases with prolonged symptoms, risk factors for bone/joint injury, or unclear history. Particularly with AVN, recognition is imperative as worsening occurs over time, complicating potential treatments.
Growing Pains: Gitelman Syndrome and Pseudogout

Gitelman syndrome (GS) is a rare, autosomal recessive tubulopathy resulting in hypokalemia, hypomagnesemia, hypocaliuria and metabolic alkalosis. Chondrocalcinosis is one of the complications of GS, though the two conditions off hand seem to be unrelated. Hypomagnesemic states, like that seen in GS, decrease alkaline phosphatase function resulting in increased extracellular inorganic pyrophosphate and insoluble calcium levels. These substances then deposit into joint spaces as calcium pyrophosphate crystals resulting in chondrocalcinosis and pseudogout.

We present a case of a 44 year old female with GS who presented with complaints of episodic joint pain, primarily of the knees for the past 2 years. The pain is severe, comes on suddenly, lasts for a few days before remitting and is associated with joint swelling and warmth, though range of motion remains intact. Imaging revealed symmetric joint space narrowing with crystalline deposition in the cartilage around her knees bilaterally suggestive of chondrocalcinosis. Lab workup revealed a serum magnesium level of 1.1. A diagnosis of pseudogout was made in the setting of GS and she was started on aggressive magnesium supplementation in addition to daily colchicine and as needed prednisone, with subsequent improvement in her symptoms.

Chondrocalcinosis, overtime, can result in joint damage due to local inflammation, which has a significant impact on patient morbidity and quality of life. Awareness of the link between these two seemingly unrelated conditions, can allow physicians to take measures to prevent joint disease development. Studies have shown that aggressive magnesium repletion in GS patients can prevent further chondrocalcinosis and may even prevent it altogether. Additionally, cases of Gitelman syndrome have been reported as initially presenting with pseudogout, further highlighting the importance of recognizing the link between these two conditions in order to keep metabolic causes of joint pain on ones differential.
Midostaurin-Induced Acute Febrile Neutrophilic Dermatosis in Neutropenic Patient

Sweet syndrome, also referred as acute febrile neutrophilic dermatosis, is an inflammatory process characterized by the abrupt appearance of erythematous papules, plaques, or nodules on the skin with predominant neutrophilic infiltration in the dermis. It presents in three clinical subtypes: classical, malignancy-associated, and drug-induced. Midostaurin, a recently FDA-approved medication for FLT3-ITD (FMS-like tyrosine kinase 3-internal tandem duplication) positive Acute Myeloblastic Leukemia, was reported once in the literature as a cause for Sweet syndrome. Our 70-year-old with FLT3-ITD positive AML who completed induction chemotherapy, was admitted two days after initiation of Midostaurin for neutropenic fever and was started on Cefepime. On the second day of admission, he developed painless, non-itchy rash on the legs and back. No other medications were added inpatient beside the broad-spectrum antibiotic and infectious workup was negative. Cefepime was switched to Aztreonam and Vancomycin in consideration of possible drug eruption; however, the rash progressed. Punch biopsy of the skin lesion revealed neutrophilic dermatoses consistent with Sweet syndrome. Patient received two doses of methylprednisolone with significant improvement. Drug-induced Sweet syndrome is a common cause of acute febrile neutrophilic dermatosis accounting for up to 26% of the cases. It usually develops within two weeks after initiation of therapy. Although neutrophilia is a common laboratory finding, FLT3-ITD inhibitor-induced Sweet syndrome has been reported in neutropenic patients, which could be part of differentiation syndrome as this class of medication is associated with dramatic differentiation of the leukemic cells. The mainstay of treatment consists of withdrawal of the offending medication or the initiation of systemic or topical corticosteroids. This case serves to alert the clinician of this new possible drug association and highlights the importance of considering Midostaurin as a novel etiology of Sweet syndrome. How to weigh the risk and benefit of continuing FLT-ITD inhibitors is a challenge.
Gradenigo’s Syndrome: An Uncommon Complication of Otitis Media

Triad of ipsilateral abducens nerve palsy, facial pain and suppurative otitis media, is referred to as Gradenigo’s syndrome. Since the advent of antibiotics, the incidence of this condition has diminished. We present an unusual case of Gradenigo’s syndrome presenting as acute left eye diplopia.

74 year old male with PMH of chronic sinusitis, peripheral arterial disease and hypertension who was admitted for arterial arteriogram for evaluation of left lower extremity claudication. After the procedure, he endorsed diplopia, which was noted to be monocular and left sided on examination with inability to fully abduct the left eye.

Primary diagnosis was a subcortical CVA accounting for this presentation. CT head was negative for hemorrhage and infarct. MRI revealed a small focus of acute ischemia with complete opacification of the left mastoid air cells.

This neurological deficit was not explained by the cerebellar infarct with the incidental finding of left mastoid opacification, both Neurology and ENT were consulted. CT of bilateral auditory canals revealed near complete opacification of the paranasal sinuses and left otomastoiditis and petrous apicitis.

Review of the EMR revealed several treatments for otitis media with an audiogram resulted in mixed hearing loss. After collaboration with ENT and Neurology it was felt that this presentation was consistent with Gradenigo’s syndrome and the left cerebellar infarct was deemed an incidental finding.

Treatment entitled placement of tympanostomy tube with IV vancomycin and Zosyn. He experienced improvement in his symptoms and was discharged to complete a six weeks course of IV antibiotics.

The patient’s undertreated chronic otitis media lead to otomastoiditis and petrous apicitis ultimately presenting as new sixth nerve palsy. This highlights the complication of undertreatment of otitis media which could result in Gradenigo’s syndrome. Early diagnosis and treatment of chronic otitis media is essential and can prevent the intracranial complication which manifested in our patient.
Atypical Hemolytic Uremic Syndrome Precipitated by Pancreatitis

Objective: To describe a case of a recurrent complement-mediated atypical hemolytic uremic syndrome (aHUS) likely precipitated by alcohol-induced pancreatitis.

Methods: Suspect atypical HUS in coombs negative hemolytic anemia and renal failure. Treat complement-mediated aHUS with eculizumab, an anti-C5 monoclonal antibody.

Results: A 35-year-old man with PMHx of alcohol use disorder, recurrent prior presentations of renal failure, anemia, thrombocytopenia, and pancreatitis presented with acute alcoholic pancreatitis. He reported abdominal pain, emesis but no bloody diarrhea. Physical exam revealed diffuse abdominal pain. Platelets on D0 were 232K/mm3. On D2-D3, he reported hematuria with platelets dropping from 44 to 10 K/mm3 and hemoglobin from 14.6gm/dL to 9.7gm/dL. Peripheral blood smear revealed >5 schistocytes/HPF. LDH was elevated to 754U/L, haptoglobin <30mg/dL, reticulocyte count at 121,900/mm3, and direct bilirubin 5.63mg/dL. Coombs test (DAT) was negative. Emergent plasma exchange (PLEX) was initiated for suspected TTP, but further PLEX was stopped when ADAMTS13 resulted normal [102%]. Renal function worsened [BUN 90mg/dL, creatinine 7.26mg/dL] requiring hemodialysis. Complement C4 was low [17mg/dL], C3 was normal, with other complement levels pending. Eculizumab was given on D5 of hospitalization, with improvement noted in hemolytic labs, platelets, hemoglobin, and kidney function by discharge. Alcohol abstinence counseling and referral for germline testing of activating complement mutations were given.

Discussion: Atypical HUS presents with DAT negative microangiopathic hemolytic anemia, acute kidney injury, and thrombocytopenia in the absence of diarrhea. Atypical HUS may be due to drugs, complement dysregulation, infections, systemic lupus erythematosus, or rarely pancreatitis as in this patient. Further, recurrent HUS should raise suspicion for complement-mediated aHUS. Eculizumab can inhibit complement-mediated thrombotic microangiopathy and improve renal function in aHUS patients.

Conclusion: We report an interesting case of atypical HUS likely secondary to pancreatitis. Providers treating patients with a constellation of renal failure, thrombocytopenia, and DAT negative hemolytic anemia need to consider atypical HUS.
Invasive Ductal Carcinoma Discovered by Leser-Trelat Sign

Leser-Trelat Sign is characterized by the sudden occurrence of pigmented skin lesions known as seborrheic keratoses that is indicative of paraneoplastic syndrome thought to be mediated by various humoral factors secondary to a malignancy, most often associated with gastrointestinal adenocarcinomas and lymphoproliferative malignancies.

An 81-year-old female with no history of malignancy, up to date on her age-appropriate screening, presented to her primary care physician after noticing an increased number of moles under her right breast over the previous few weeks. They were causing her stinging pain and irritation, but without any bleeding, discharge, or erythema. Physical exam confirmed a cluster of elliptical, uniformly pigmented nevi with regular symmetrical borders present under both breasts, and discovered a rigid 6cmx5cm mass in the right breast inferior to the areola, as well as lymphadenopathy on deep palpation of the right axilla. Mammography confirmed the presence of a hypoechoic mass measuring ~2cmx2cmx2cm, as well as multiple abnormal lymph nodes in the axilla with the largest measuring ~2cmx2cmx3cm. Further investigations with biopsy and imaging confirmed a triple-negative grade 2 invasive ductal carcinoma - Stage T2N2M0 (clinical stage IIIA). Case was discussed at tumor board and determined to be best treated with neoadjuvant Paclitaxel pending surgical management with breast-conserving lumpectomy and lymphadenectomy.

Seborrheic keratosis is a common type of skin lesion that is prevalent in elderly individuals. We would like to draw attention to the fact that Leser-Trelat’s sign can be associated with breast malignancy as one of the earliest presenting symptoms. Rapid dermatologic presentations can be a potential indicator of a possible malignancy, and reinforces the importance of a broad differential in malignancy workup to include common and uncommon malignancies as potential sources of this paraneoplastic process.
Mycoplasma Hominis Abscesses After Caesarean Section: A Rare Complication of a Common Procedure

Pelvic abscesses after endometritis occurs in less than 1% of postpartum women, with the most common areas being the broad ligament, posterior cul-de-sac, and anterior to the uterine wall. Our case is even more uncommon as multiple Mycoplasma Hominis abscesses developed after Cesarean section and were not all contained in the pelvis.

A 24-year-old woman who delivered via C-Section eight days prior to admission presented with abdominal pain and signs of pulmonary embolism confirmed on CT-angiography. Of note, she had been treated for chorioamnionitis. The development of leukocytosis and fever, with recent abdominal surgery prompted imaging. CT Abdomen/Pelvis showed a multilocular fluid collection anterior to the uterus, and abdominopelvic ascites. Piperacillin-Tazobactam and Linezolid were initiated but she did not clinically improve; repeat imaging showed a posterior cul-de-sac fluid collection developing into an abscess. The patient underwent drainage of both anterior and posterior collections after which, initially, leukocytosis improved and the patient defervesced. However, due to relapsing fever and worsening leukocytosis, antibiotics were escalated to Meropenem. Anterior abscess cultures grew Mycoplasma hominis, which had not been covered; the posterior fluid grew E. coli and E. faecalis. Moxifloxacin was added to cover M. hominis. Later on, repeat imaging showed new peri-aortic and peri-splenic abscesses, which, after drainage, resulted in significant clinical improvement. Only the peri-aortic abscess showed culture growth, which was M. hominis. She was prescribed a six-week course of Piperacillin-Tazobactam for E. coli and E. faecalis coverage, and Moxifloxacin for M. hominis coverage, along with follow up and repeat imaging.

M. hominis infections after C-sections have been reported in a few cases. Due to its resistance to common antimicrobial drugs, it is important to bear Mycoplasma in mind in the lack of clinical improvement. This can prevent the potential spread and abscess formation in other organs.
Statins are used in the primary and secondary prevention of cardiovascular disease. Muscle toxicity, a known side effect with an incidence of 33%, ranges from myalgia, to myositis, rhabdomyolysis, severe myonecrosis or life-threatening rhabdomyolysis leading to acute compartment syndrome. Here we present a case of statin-induced myositis progressing to acute compartment syndrome of the left lower extremity.

A 39-year-old man with a history of essential hypertension and hypercholesterolemia presented to the emergency department with four days of progressively worsening left lower leg pain unrelieved by regular analgesia. He denied trauma or vigorous exercise in the days prior to his presentation. His medications included hydrochlorothiazide-losartan 100-25mg and atorvastatin 40 mg at nighttime, which he started three months prior. He reported no known drug allergies, was a non-smoker and consumed alcohol occasionally. On physical examination, there was pain with dorsiflexion and plantar flexion of the left leg and the overlying skin looked firm. Over the course of several hours, the pain with passive and active motion increased together with paresthesia. Dorsalis pedis pulses in the left leg were present but weaker compared to presentation. Compartment pressures were measured with a Stryker needle and were elevated in the superficial posterior and lateral compartments of the left lower leg. Emergent four compartment fasciotomy was done with wound vac placement. Patient was discharged home with follow up in the Surgical clinic.

Statins are recommended for those with an LDL > 100 mg/dL and ASCVD > 10%. Those with ASCVD between 5-10% could benefit from therapy. Younger patients, like ours, could have benefitted from a discussion about potential adverse effects of statin therapy. Severe rhabdomyolysis from statin use could result in compartment syndrome, a surgical emergency. PCSK9 inhibitors have been used in the treatment of high LDL-cholesterol levels, and may be a good option in patients formerly receiving statins.
Sciatic Neuropathy: An Uncommon Presentation of Anticoagulant Induced Iliopsoas Hematoma

Spontaneous iliopsoas hematomas are rare but well documented in association with femoral nerve paralysis. We describe an unusual case of spontaneous iliopsoas hematoma presenting as sciatic neuropathy. An 80-year-old man with bladder cancer admitted for hypercalcemic encephalopathy was noted to have a gradual onset of progressive pain around the right hip joint associated with paresthesia in his right leg and foot. Patient denied recent trauma or fall. Home medications included Apixaban for PE diagnosed three weeks ago. On exam, passive extension of the right hip joint was painful and restricted with a motor strength of 2/5 of entire right lower extremity muscles with decreased sensation in the L3-L5 and S1 dermatomes. The right patellar and Achilles reflex were reduced. With concern for metastatic spinal involvement, an MRI was done demonstrating a partially calcified large multiloculated iliopsoas hematoma draping the exiting right sciatic nerve and right iliac vessels. The patient’s hemoglobin had declined from 13 g/dl to 9 g/dl over three weeks. Anticoagulation was discontinued and a JP drain was placed by IR but with unsuccessful drainage due to calcified nature of hematoma. Hence, patient’s symptoms did not improve and follow up CT did not demonstrate a change in the size of the hematoma. Patient’s condition declined due to sepsis preventing further interventions. Spontaneous muscle hematomas can be frequently overlooked. Our case demonstrates that iliopsoas hematoma should be included in the differential when assessing unilateral lower extremity neuropathy. Early recognition can lead to successful IR guided decompression and can potentially avoid life-threatening complications.
Resident Error Reporting: A QI Initiative

It's important to recognize, report and correct any errors made in the medical fields. Simple errors lead to lethal consequences. Residents are frontline witnesses to patient care errors that are made on a day-to-day-settings. It is imperative residents report events and are educated regarding its significance. There are many barriers to safety event reporting including not understanding what constitutes an error, fear of repercussions following an event report, cumbersome and time-consuming process of reporting an error. In 2014, our hospital was evaluated by Clinical Learning Environment Review and lack of error reporting by residents was recognized as an area of concern. This project’s aim is to improve error reporting by resident physicians.

We are using the IHI model to guide this study. The Plan Do Study Act cycle was used to study the changes.

Three PDSA cycles involved re-educating residents each year regarding the error reporting system. Each cycle did show improvement in resident error reporting. Survey done in 2019-2020 year. >90% of the residents are aware of the process of error reporting, have been encouraged to report an error, and are comfortable reporting errors. Between 07/2019-06/2020, there were 63 error reports; 84% were severe harm, 6.3% were mild harm, 60.3% were actual events and 39.6% were near misses. Promising results were achieved with education. Continued improvement in error reporting was seen following implementation of workflow process and incorporation of multidisciplinary teams. This resulted in improved engagement of more residents in error reporting. PDSA 4 is currently being conducted. Further improvement in safety event reporting by residents will be continually encouraged and re-evaluated.

Currently incorporating weekly dedicated times for reporting errors as well as more faculty encouragement. The residents have been re-educated on this topic this year. Medical errors represent opportunities for improvement. Reporting and resolution of errors ultimately results in improved patient safety.
A Rare Case of Combined Small-Cell and Large-Cell Neuroendocrine Lung Carcinoma

Lung cancer is the number one cause of cancer worldwide and is still the most common cause of cancer deaths. Of these, small cell lung carcinoma (SCLC) comprises about 15% of all bronchogenic carcinomas. A rare subset, labeled as combined small-cell lung carcinoma (C-SCLC), is defined as SCLC with histological components of non-small-cell lung carcinoma (NSCLC) and account for only 5% of all SCLC. Here, we present a case of primary pulmonary neoplasm with two distinct underlying histopathologic components.

A 76-year-old male with an estimated 30-pack-year smoking history presents with four weeks of worsening dyspnea, productive cough, 13-pound weight loss and hemoptysis. Chest imaging reveals a large mass within the right hilum extending into the mediastinum, causing compression of the right mainstem bronchus. A large right pleural effusion and multiple lytic lesions throughout the vertebral bodies with hepatic and left renal lesions, concerning for metastatic disease, are also noted. Pleural fluid analysis demonstrated immunohistochemical staining patterns consistent with a high-grade neuroendocrine carcinoma with mixed large cell and small cell features, expressing thyroid transcription factor-1 and synaptophysin while negative for cytokeratin 7, cytokeratin 20 and chromogranin. A subsequent left hepatic lobe mass biopsy confirmed metastatic neuroendocrine carcinoma consistent with lung primary.

The patient was discharged after treatment of post-obstructive pneumonia and initiation chemotherapy with Carboplatin/Etoposide.

The diagnosis of a primary lung cancer is clinicopathologic. According to the World Health Organization, C-SCLC includes SCLC with components of adenocarcinoma (ADC), squamous-cell carcinoma (SCC), large-cell carcinoma (LCC), large-cell neuroendocrine carcinoma (LCNEC), giant cell or spindle-cell carcinoma. The optimum treatment for C-SCLC is still unknown and most patients are treated based on SCLC guidelines, with a combination surgery, radiotherapy and chemotherapy. Developing awareness of these rare subsets and subtle differences between them will help guide future diagnostic and therapeutic strategies in this vulnerable patient population.
Double Trouble! Diabetic Ketoacidosis Associated Ischemic Stroke

Introduction: DKA is a state of severe insulin deficiency, either absolute or relative, resulting in hyperglycemia, ketonemia, acidemia, and systemic inflammation. The most common complication of DKA is cerebral edema, which resulted in the death of 21 to 25% of affected patients. Less common, is the risk of acute ischemic stroke during the acute DKA episode. Although diabetic ketoacidosis associated stroke is observed more in the pediatric population, our case was a young male with type 1 diabetes who presented with DKA and subsequently developed an ischemic stroke.

Case Description: 46-year-old male with past medical history of Uncontrolled Type 1 Diabetes Mellitus with Hyperglycemia presented to the hospital with altered mental status. Patient had multiple admissions in the past for DKA in setting of insulin non-compliance. Upon arrival to Emergency room, patient was lethargic but was following verbal commands. CT head on admission revealed no signs of cerebral edema and no acute hemorrhage or mass was present. Patient was managed per DKA protocol. Day 2 patient was obtunded but responded to noxious stimuli. On Day 3, patient was noted to not having a pupil or gag reflex. To our surprise, repeat CT Head showed Extensive hypoxic ischemic brain injury involving the entire territories of the right and left internal carotid arteries. CTA head revealed occlusion of the left and right internal carotid artery.

Clinical discussion: DKA is associated with systemic inflammatory response characterized by vascular endothelial injury and coagulopathy. Stroke in DKA is very rare but life-threatening. Since the presentation of cerebral edema and primary stroke in DKA can overlap, clinicians should have a high index of suspicion for stroke, hence early imaging for any patient with neurological deterioration is often required. Magnetic resonance imaging with perfusion has a sensitivity nearing 100% for identification of ischemia associated with stroke.
Rapidly Progressive Epithelioid Mesothelioma

Malignant pleural mesothelioma (MPM) is an aggressive cancer that arises from mesothelial pleural cells exposure to mineral fiber, specifically with prolonged exposure to asbestos. We are presenting a case of rapidly progressive MPM with several unique features. The patient is a 72 year-old male who presented with a chief complaint of dyspnea. He was previously admitted for dyspnea where CT chest was significant for extensive ground glass and alveolar opacities in his right lower lung with a small multiloculated right pleural effusion. His last thoracentesis was significant for exudative fluid with lymphocyte predominance, flow cytometry and cytology were negative for lymphoproliferative disorder and malignancy respectively. During this admission, he complained of worsening dyspnea and an unintentional 18-pound weight loss within the past month. He underwent VATS for pleural biopsy, intraoperatively, his right pleural cavity was covered by innumerable masses. Post-procedure he became hypotensive requiring vasopressor support along with worsening hypoxia requiring re-intubation. His CXR showed complete opacification of the right hemithorax. The CTA chest showed extensive consolidative changes involving the right chest with poor aeration in right lower lobe with air bronchograms. Unfortunately, the patient had a cardiac arrest due to hypoxia. The final pathology discovered infiltrating epithelioid mesothelioma. Multilocular effusions are associated with infection, and not MPM, our patient’s CT chest was significant for multilocular effusion, with no underlying infection, and a final fluid pathology was significant for epithelioid mesothelioma. It is known that epithelioid subtypes are less aggressive compared to their counterparts with a medium survival between 12 to 18 months. However, this case presents a rapid progression of epithelioid mesothelioma and abnormal development of complete right opacification of the right lung within hours after VATS. It is vital to be aware of MPM in refractory pleural effusions and rapid progression.
Is There a Safe Way to Reverse Anticoagulation in Patients with Hypercoagulable State?

Acute hip fracture is deemed an emergent orthopedic intervention, such interventions can be complicated if the patient is on anticoagulation for chronic thromboembolic disease. We are presenting a case of an elderly patient who presented with an acute hip fracture with an elevated INR that required rapid reversal, which unfortunately led to a fatal thromboembolic event.

The patient 80 year-old female with PMH of DVT, PE, and Factor V Leiden on warfarin was admitted for management of an acute hip fracture. On presentation, her INR was 4, and was given vitamin K for INR reversal. Intraoperatively, there were no acute events. The following morning, the patient was found to be less responsive. The laboratory studies revealed high anion gap metabolic acidosis secondary to elevated lactate indicative of bowel ischemia. The patient was intubated due to worsening mentation. Her CTA of abdomen and chest was significant for moderate to severe stenosis of celiac axis, severe narrowing of splenic, common hepatic artery and gastric artery. Unfortunately, she eventually was placed in comfort care. Studies have shown that the patients on long term anticoagulation have 1-3% overall risk of bleeding. An extreme caution must be taken while reversing anticoagulation. Our patient underwent an emergent hip replacement surgery after reversal of warfarin, however due to her underlying hypercoagulable state and stress of surgery, she suffered from multiple thrombotic events that eventually led to her demise. There are no guidelines that provide the most appropriate way to reverse anticoagulation in patients with underlying hypercoagulable state. 1 out of 500 people suffer from an underlying disease that puts them at risk of thromboembolic events. It is imperative to develop guidelines for reversal of anticoagulation in patients with diseases that places them at risk of hypercoagulable state to ensure safety while undergoing surgical procedures.
Acute Symptomatic Occlusion of Internal Carotid Artery Following Carotid Endarterectomy: A Rare Complication

Introduction:
Carotid endarterectomy (CEA) is the gold standard for treatment of internal carotid artery (ICA) stenosis. However, neurovascular complications like stroke can develop in the perioperative period. We report a patient status post CEA who developed stroke, an uncommon complication that the procedure was supposed to prevent.

Case:
60 years old female underwent elective right CEA due to severe stenosis of ICA. After the procedure she developed vomiting and headache. 90 minutes after the procedure, she was noticed to have left sided facial droop along with left sided weakness. Emergent carotid duplex ultrasound was inconclusive. CT angiography of the head and neck showed complete occlusion of proximal right ICA. The patient underwent emergent surgical re-exploration and a fresh thromboemboli at proximal portion of ICA was retrieved. Repeat angiography showed complete patency of common, external and internal carotid artery all the way up to the brain without any evidence of additional thromboembolus. Patient was then transferred to ICU for closer monitoring. She remained asymptomatic with remarkable improvement in neurological examination.

Discussion:
Perioperative stroke following CEA leads to residual neurological defects or even death in about 10% of perioperative stroke patients. We observed this rare complication, however the patient improved remarkably following surgical re-exploration and thrombus retrieval with only mild residual neurological deficits. Even if the immediate postoperative course seems normal, physicians should be vigilant as internal carotid artery occlusion can occur after several days of CEA.
My Watch Saved Me! --Smartwatch Detection of Ventricular Tachycardia

Introduction
Smartwatches have become commonplace, but their features are well evolved. New sensors have been developed, including those designed to detect heart rate and provide electrocardiogram (ECG) reads. Studies have demonstrated the watch’s ability to detect atrial arrhythmias such as atrial fibrillation. In this case, we describe a patient whose smartwatch detected an arrhythmia, subsequently leading to the diagnosis of ventricular tachycardia and thankfully appropriate hospital management.

Case Presentation
A 49-year-old woman with a past medical history of Hypertrophic Obstructive Cardiomyopathy, Heart Failure with Reduced Ejection Fraction (HFrEF), and Chronic Kidney Disease Stage III presented to the hospital with chief complaint of palpitations. According to the patient, she was sitting at home and had acute onset of palpitations associated with lightheadedness and mild dizziness. She checked her Apple Watch and found her heart rate was 160 bpm. She did an ECG using her watch and sent it to her Cardiologist. She was advised to go to the emergency department (ED) immediately. Her blood pressure was found to be 70/40 mmHg. ECG showed ventricular tachycardia and urgent cardioversion was performed. Patient’s rhythm returned to sinus afterwards. She was hospitalized for three days for further cardiac management and was discharged home in stable condition.

Discussion
One of the main causes of death in patients with HFrEF is cardiac arrhythmias, including ventricular tachycardia and ventricular fibrillation. New smartwatches are being studied for monitoring of atrial arrhythmic events but not ventricular arrhythmias. There are some case reports of ventricular arrhythmic events recorded, although there is no definitive literature in this regard. The ease of use makes smartwatches a promising technology for detecting ventricular arrhythmias and likelihood of improving patient outcomes.
Combined Serotonin Syndrome and Neuroleptic Malignant Syndrome Occurring in Escitalopram Overdose

Introduction: While escitalopram can cause serotonin syndrome (SS), rare reports exist of associated neuroleptic malignant syndrome (NMS). Here, we are presenting a case of an adolescent on escitalopram for major depressive disorder (MDD), who presented after ingestion of 26 count 40mg tablets and developed SS and NMS.

Case: 17 year-old male teen with MDD, presented to ER for overdose of escitalopram. Apart from an elevated heart rate of 120-130, his vitals were stable. He had an episode of tonic-clonic seizure followed by asystolic cardiac arrest. After ACLS, he achieved return of spontaneous circulation (ROSC), was intubated and transferred to intensive care unit. His QTc was prolonged to 538 msec and sodium bicarbonate was administered. He developed rigidity and was treated for SS with cyproheptadine. His systolic blood pressures varied from 88 to 154 mm hg. Due to elevated CPK, autonomic lability, continued rigidity post 24 hours, he was eventually diagnosed with NMS, and received treatment for it. He was successfully extubated on day-6, and transferred to inpatient psychiatry for further care.

Discussion: Selective serotonin reuptake inhibitors (SSRI) and serotonin norepinephrine reuptake inhibitors (SNRI) are associated with suicide risk in adolescents. While QTc prolongation is a known side-effect of escitalopram, seizures are rare. SS develops acutely on addition of serotonergic medication to an already established treatment or dose on adjustment. It’s clinically diagnosed with presence of rigidity, hyperthermia, agitation, tremors etc. While there is overlap between SS and NMS; autonomic lability, prolonged rigidity and elevated CPK are more indicative of NMS. Benzodiazepines are used for sedation and cyproheptadine is the antidote for SS. Dantrolene is used for NMS rigidity and bromocriptine to restore dopaminergic tone.

Conclusion: Anti-depressants are common overdose medications in adolescents suffering from MDD. Due to high mortality associated with NMS, it's timely diagnosis and treatment is imperative.
Introduction: Vasculitides are difficult to diagnose and often are not diagnosed until the disease process is advanced. Testing for antineutrophil cytoplasmic antibodies (ANCA) reactivity is a common practice, though only 30 to 60 percent of patients with eosinophilic granulomatosis with polyangiitis (EGPA) are ANCA positive which can lead to a diagnostic delay. We report a case of EGPA with multi-system involvement in a patient with an initially negative ANCA.

Case Description: A 56-year-old Caucasian female with a past medical history of hypertension, epilepsy, asthma vs. chronic obstructive pulmonary disease, chronic hepatitis C, nasal polyps, and generalized anxiety disorder initially presented to the emergency department (ED) for a presumed urinary tract infection with acute kidney injury and sepsis with shortness of breath. Urine cultures and ANCA were negative, she was treated for presumed pneumonia. She was discharged with nephrology follow-up who recommended renal biopsy due to increasing hematuria and proteinuria. She returned to the ED one month later with a 15-pound weight loss, arthralgias, weakness, diarrhea, hemoptysis, elevated troponins, and decreased urine output. She was discharged with high-dose steroids and her hydralazine was discontinued due to concern for drug-induced lupus. Her repeat ANCA came back positive after discharge, she was called to return to the hospital. She was now anemic requiring transfusion, in hypertensive urgency, had a severe acute kidney injury, had new oxygen requirements, and had likely diffuse alveolar hemorrhage on imaging. Renal biopsy supported a diagnosis of EGPA.

Discussion: ANCA positivity is a hallmark of EGPA. Unfortunately, some cases of EGPA present with a negative ANCA which can be falsely reassuring of disease absence. Awareness of this irregularity allows the clinician to keep EGPA on the differential despite a negative ANCA. To overcome this, early biopsy allows for prompt therapy and potential prevention of morbidity and mortality.
Introduction: As much as 25% of patients with rheumatic disease cannot be definitively diagnosed or present with overlap syndromes. These patients often display one or more disease patterns along with nonspecific serologic abnormalities. Such patients have features of widespread inflammation and are considered to have “undifferentiated” rheumatic disease or have features that overlap two or more specific diseases.

Case: 28-year-old female with a past medical history of type 1 diabetes mellitus, ESRD on HD, and Hashimoto’s thyroiditis presented with a left sided empyema. A left chest tube was placed, and cultures grew Streptococcus constellatus. Despite antibiotic therapy, she had a prolonged hospitalization due to lack of improvement of the empyema. On further history, patient had arthralgias for the past several months and had a history of 4 miscarriages. Echocardiogram showed a moderate pericardial effusion. Autoimmune panel showed: ANA-1:320 with speckled pattern, C3- 35, C4- 2, +anti-RNP antibody- 1.4, +direct anti-globulin, +RF, +CCP, -dsDNA. Patient was started on oral prednisone with consultation of rheumatology secondary to concern for autoimmune flare. After the initiation of prednisone and intrapleural tPA, the patient improved and was discharged home.

Discussion: Overlap syndromes satisfy the classification criteria of at least two CTD’s occurring simultaneously or at different times. “Rhupus” is one entity that has overlapping features of both systemic lupus erythematosus (SLE) and rheumatoid arthritis (RA). Streptococcus constellatus empyema is a clinical entity that is rarely described in the literature; however, in this immunocompromised patient, it was the initial presentation of her overlap syndrome. The clinical manifestations and management of Rhupus varies per patient and treatment is directed towards specific clinical symptoms. The prognosis of Rhupus syndrome is different from that of patients with SLE or RA. Consequently, the proper recognition and early diagnosis of Rhupus syndrome is important for choosing treatment regimens.
Bilateral Facial Palsy and Acute HIV in Pregnancy

Introduction
Bilateral facial nerve palsy (BFNP) is a rare disorder that is responsible for 0.3 - 2.0% of facial nerve palsies. In a ten-year study, numerous causes of BFNP were reported including Lyme disease, Guillain-Barre syndrome, sarcoidosis, and AIDS. The risk of facial nerve palsy for pregnant women is 3.3 times that for nonpregnant women.

We present a case of BFNP in pregnancy with acute HIV infection.

Case
A 42-year old G7P5015 female at 37 weeks gestation presented with BFNP. Three months earlier, she had negative HIV screen. She also had a diffuse rash 1 month prior to presentation that resolved with diphenhydramine treatment. Five days prior to admission, she noticed numbness and weakness extending from her forehead to upper lip bilaterally. Examination on presentation was consistent with term pregnancy and bilateral CN VII deficits. Workup including MRI of the brain, lumbar puncture, myasthenia gravis panel, Lyme antibodies, syphilis screen, serum ACE levels, routine hematology and chemistry labs were all normal. HIV screening returned positive with a confirmatory 129,455 copies/ml. One week after discharge, labor was induced resulting in the birth of a healthy newborn. Patient was started on emtricitabine, tenofovir and raltegravir, resulting in complete recovery of BFNP in 1 month.

Discussion
BFNP is a rare disorder but it should be considered in pregnant women with facial weakness. Acute HIV infection in pregnancy can also increase the risk of it. Delivery of the fetus and timely antiretroviral therapy can lead to complete neurological recovery.
A Case of Eustachian Valve Endocarditis, and Importance of Synergistic Antibiotic Therapy

Eustachian valve (also known as the valve of the inferior vena cava) is an embryological remnant that diverts oxygenated blood from inferior vena cava towards the foramen ovale. This remnant usually regresses after birth, but if remains, it does not cause any abnormality. We report a rare case of eustachian valve endocarditis in a patient who had no other signs of right sided endocarditis.

A 46 year old woman with medical history of type 1 diabetes mellitus, end stage renal disease on chronic hemodialysis presented with one week of fever, chills, altered mental status and hand pain. She was afebrile on presentation with pulse rate of 102 bpm, blood pressure was 138/85 mmHg. On exam, she was ill appearing and had dialysis catheter in right neck. Hand exam demonstrated swollen, erythematous and tender wrist. Cardiovascular exam demonstrated no murmurs. Blood cultures were obtained on admission, she was started on empiric vancomycin and cefepime. Computed tomography hand showed abscesses involving right forearm. She underwent incision and drainage with extensive debridement with cultures growing MRSA. Her blood cultures also confirmed MRSA bacteremia. Transesophageal echocardiography showed 1.0 x 1.0 cm mobile vegetation involving eustachian valve with no other valvular involvement. She remained bacteremic for 18 days even being on vancomycin with appropriate blood levels. Imaging of the back and hand showed no peripheral sources of persistent bacteremia. Her antibiotics were switched to daptomycin and ceftaroline which cleared her cultures and repeat TEE showed improved vegetation size (0.28 cm).

Right sided valvular infective endocarditis has been well-described, but lesions affecting Eustachian valve are distinctly rare. Right sided endocarditis mainly affects intravenous drug users, but in our it was linked to dermatological penetration or dialysis catheter infection. Literature on the management is scarce; our case demonstrates important synergistic effect of ceftaroline and daptomycin for persistent bacteremia.
A Case of Burkholderia Cepacia Prosthetic Valve Endocarditis Leading to Mitral Valve Stenosis

Prosthetic valve endocarditis (PVE) is predominantly caused by Staphylococcus aureus and Streptococcus viridians. Infective endocarditis (IE) due to Burkholderia cepacia is rare. We present a case of a 47-year-old man with history of injection drug use, IE of aortic and mitral valve status post replacement with bioprosthetic valves 6 months ago, who presented with fever, chills, dyspnea and lightheadedness for three days. He was afebrile, hypotensive (97/51 mmHg) and tachycardic (147 bpm) on presentation. Cardiovascular exam revealed a grade 3/6 systolic murmur at the left sternal border. Vancomycin and cefepime were started empirically after obtaining blood cultures. TTE demonstrated reduced ejection fraction (EF 25-30%). TEE confirmed 1.8 x 1.2 cm vegetation on posterior leaflet of mitral valve causing functional mitral stenosis. Gentamicin and rifampin were added to treat PVE. Blood cultures were positive at 21.39 hours for gram negative rods. On day 5, Burkholderia cepacia sensitive to levofloxacin, meropenem, and trimethoprim/sulfamethaxozole and resistant to ceftazidime was identified. Antibiotics were changed to meropenem and levofloxacin. Blood cultures became negative two days after the antibiotics change and remained negative on blood culture drawn 10 days post discharge. Cardiothoracic surgery was consulted who deemed him a nonsurgical candidate because of his acute biventricular failure. He was discharged on meropenem for 8 weeks and life-long suppressive levofloxacin.

Burkholderia cepacia is an opportunistic pathogen that causes respiratory illness and bacteremia particularly in patients with cystic fibrosis or chronic granulomatous disease. The treatment of Burkholderia cepacia endocarditis consists of administration of antibacterial agents, and/or surgery. Our decision to start levofloxacin and meropenem was based on IDSA guidelines which encourages use of double gram-negative coverage for endocarditis due to non-HACEK gram-negative bacilli. Vegetations leading to mitral valve stenosis is a rare complication, and is an indication of the surgery, but was not pursued due to patient’s condition.
Thyrotoxicosis Secondary to Supplement Use Leading to Cardiac Arrest

Introduction:
Thyrotoxicosis secondary to exogenous thyroid hormone use is commonly seen in body-builders and athletes, who abuse such supplements in order to lose weight and enhance performance. We report a case of a man who presented with decompensated heart failure and subsequently died due to the complications of supplement use.

Case:
A 40-year-old man with a history of supplement use and anxiety presented with chest tightness, shortness of breath, and palpitations. His chest tightness was non-exertional in nature but the dyspnea was progressive in nature. His symptoms started 3 days prior and what brought him to the hospital was his palpitations which were non-resolving. On presentation, his vitals were heart rate 202 beats/min, breathing 40/min, blood pressure 140/90mmHg, SpO2 92% on ambient air, and afebrile. His exam revealed a normal neck exam without any goiter, tachycardia, irregularly irregular heart rate of 170bpm, without any murmurs appreciable. EKG revealed atrial fibrillation with a rapid ventricular rate of 170bpm. He was diaphoretic and in respiratory distress with the use of accessory muscles. Shortly after intubation, he became hypotensive to the point of being non-responsive to maximum doses of vasopressors. A bedside echo revealed global hypokinesis with EF of 10-15%. His labs were T3 346ng/dL, TSH <0.01u with a negative antibody panel. The patient underwent asystole cardiac arrest with multiple attempts at resuscitation but in vain. The post-mortem revealed that he had atrophic thyroid gland and cardiomegaly.

Discussion:
Exogenous thyroid hormone use can lead to varying cardiac complications ranging from sinus tachycardia to ventricular fibrillation to death. This is commonly seen in pediatric populations because of accidental overdose. The treatment comprises beta-blockers, steroids, and indefinite discontinuation of the offending drug.

Conclusion:
Thyrotoxicosis secondary to supplement use is a common entity and should be considered in such situations as prompt recognition and treatment can be life-saving.