American College of Physicians- Minnesota Chapter Annual Abstract Competition
Poster Session
October 28, 2022
Abstracts Submitted for Competition

Medical Students	
Research - Medical Students	
Austin Hoeg Dr. Alexander Khoruts	*Risk Factor Analysis of Encapsulated Fecal Microbiota Transplant in Treatment of Recurrent Clostridioides Difficile* Background: MTP-101C, a standardized freeze-dried, encapsulated fecal microbiota preparation, is being used at the University of Minnesota and several other tertiary medical centers to treat multiple-recurrent Clostridioides difficile infection (rCDI) after the failure of standard antibiotic therapies. A single dose of MTP-101C is ~ 85% effective in curing rCDI. Our long-term goal is to optimize the microbiota transplant treatment protocols to improve outcomes further. Here, we aim to identify clinical risk factors associated with the failure of microbiota transplant therapy. Methods: The University of Minnesota MicrobiotaTherapeutics Program maintains the database of all microbiota transplant treatments. Most of these patients have collected stool specimens for donor microbiota engraftment analysis. We hypothesized that advanced age (≥ 65 years old), moderate to severe comorbid burden (Charlson Comorbidity Index ≥ 3), and polypharmacy (≥ 5 medications) would be risk factors for MTP-101C clinical failure. We reviewed medical records from 100 consecutive patients without underlying inflammatory bowel disease who underwent treatment with MTP1-10C for rCDI and performed one-way ANOVA to identify associations between our hypothesized risk factors and microbiota transplant failure. Results: In the analyzed cohort, 56% of patients were of advanced age, 73% of patients had a moderate or severe comorbid burden, and 90% of patients were of polypharmacy status. 16% of patients experienced a relapse of CDI within two months of post-treatment follow-up. Advanced age (> 65 years) correlated with relapse (p=0.026). Moderate to severe comorbid burden and polypharmacy status were more common in patients who experienced clinical failure of MTP-101C, but the differences did not reach statistical significance. Conclusion: Older age (≥ 65) is a risk factor for failure of microbiota transplant treatment for rCDI. Older patients have a higher burden of medical comorbidities and polypharmacy status. In the next phase of this project, we will test whether older age is associated with decreased engraftment of donor microbiota.

Laura Maciejko Dr. Liset Falcon Rodriguez	*Improving Health Care Engagement in the Hispanic Community of Southeastern Minnesota* Background: Despite being the second largest group in the U.S. after Non-Hispanic Whites, the Hispanic population faces numerous barriers to accessing health care. These barriers occur on multiple levels: patient level (e.g. health beliefs and personal health practices), the provider level (e.g. provider skills and attitudes) and the system level (e.g. how the healthcare system is organized). Little data has been collected in These barriers occur on multiple levels: patient level (e.g. health beliefs and personal health practices), the provider level (e.g. provider skills and attitudes) and the system level (e.g. how the healthcare system is organized). Little data has been collected in Southeastern Minnesota (SEMN). Healthy Minds Healthy Bodies (HMHB) is an adult educational program whose goal is to improve health care engagement in the Hispanic community in Southeastern Minnesota. The goal of this project was two-fold, to evaluate barriers faced by the Hispanic population in SEMN and to evaluate the efficacy of HMHB in promoting health care engagement. Methods: HMHB 2021 was run virtually over five sessions. Participants completed a pre- and post-program survey, which included questions about general access to health care, self-efficacy, outcome expectations, and social support. Pre- and post-program survey results were compared using paired t-tests. Results: Seventeen participants completed both the pre- and post-program surveys. Overall, 2 (11%) respondents report having poor health, 6 (33%) report having fair health, 8 (45%) report having good health, and 2 (11%) report having very good health. Thirteen (72%) participants have been to the doctor within the past 12 months while 3 (17%) report never getting a wellness checkup. Fifteen (83%) of the respondents state that they have a place to go if they need healthcare. Six (33%) of the participants state that they have delayed getting healthcare during the past 12 months due to the cost. When asked about barriers to healthcare access, 5 out of 12 respondents indicated that cost was the most significant barrier to receiving healthcare. There was no statistically significant change in the self-efficacy and outcome expectations portions. Meanwhile, there was a statistically significant increase in social support after program completion (median 19, 11-24) compared to pre-program (median 14, 0-24) ($p = 0.0128$). Conclusions/discussion: HMHB highlighted barriers to accessing healthcare in SEMN, which can help create more targeted interventions aimed to reduce healthcare barriers in this population. While self-efficacy is the biggest predictor of behavior, our study showed no statistically significant increase in participant self-efficacy after attending HMHB 2021. This might be due in part to the small sample size, which is a limitation of the study. Our findings highlight the importance of social support in this community, which should be taken into consideration when organizing future HMHB programs.
Shannon Thomas Dr. Terin Sytsma Dr. Laura Greenlund	*Cumulative Dose of Repeated Joint/Bursa Corticosteroid Injections and ED Visits/Hospitalizations* Background: Joint and bursa corticosteroid injections have been used to manage musculoskeletal pain and inflammation for many decades. Despite the decades of use, there is very little literature regarding the systemic effects of cumulative doses of intraarticular/bursa corticosteroid injections. Systemically administered corticosteroids can potentially cause

	immunosuppression, adrenal suppression, and significant hyperglycemia, all of which can result in emergency department (ED) visits and potentially hospitalization. We evaluated whether increased cumulative dose of intra-articular/bursa corticosteroid was associated with an increased number of ED visits/hospitalizations. Methods: Our group has followed a large cohort of 8816 local patients receiving joint/bursa corticosteroid injections from May 2018 to July 2022. An institutional database was used to collect all clinical data. Retrospective injection data was collected for the years 2018-April 2021, and prospective data was collected from May 2021 onward. Based on the cumulative dose received, the injection group was divided into "High"(> 600 mg) , "Medium"(160-599 mg), and "Low"(<160 mg) dose. Descriptive statistics and linear regression were used to analyze data. Results: The High dose group was older (median age 72 y) than the Medium (median age 70 y) and Low dose (median age 66 y) groups, P<0.01. A larger percent of High dose patients were female (75%) than Medium (67%) or Low dose (59%). Patients in the High dose group received an average of 847 mg (range 2140 mg-600 mg) methylprednisolone equivalents, the Medium group 276 mg (range 599 mg-160 mg) and the Low group 70 mg (range 159-2.7 mg). The total number of ED visits/hospitalizations was 19,124 over the 4-year study period. ED visits accounted for 12,777 of these, and the most common complaint was chest pain (n=1310) followed by abdominal symptoms (n=953). The median number of ED/hospital visits for all three groups was 1 (High range 0-53, Medium range 0-94, and Low range 0-88). In each group there were a few extreme outliers with large numbers of visits. A scatter plot with linear regression showed no correlation between cumulative corticosteroid dose and ED/hospital visits (r2 <0.01). Conclusion: There was no correlation between cumulative joint/bursa corticosteroid dose and ED/hospital visits, suggesting that joint/bursa corticosteroid injections do not increase the risk for urgent/emergent adverse health outcomes.
Clinical Vignette - Medical Students	
Andrea Anampa-Guzman Dr. Ba Aqeel Sheeba Dr. Torka Pallawi	*Hidden in the Castle* Introduction: Castleman disease (CD) is an uncommon group of heterogeneous lymphoproliferative disorders which cause nonmalignant lymphadenopathy related to increased release of cytokines, particularly interleukin 6 (IL-6). Multicentric CD (MCD) is a systemic, often symptomatic condition involving multiple lymph node stations. MCD has a more severe clinical course and is associated with worse outcomes as well as malignancy. Subtypes of MCD include idiopathic MCD and human herpesvirus-8-associated MCD. Case Presentation: We present a case of a 33-year-old man who presented with a persistent dry cough, loss of appetite, and unintentional weight loss for seven months. Computed tomography revealed a 9.6 cm mass-like consolidation in the right hilar region, extensive mediastinal and bilateral hilar lymphadenopathy, and a small pericardial effusion. The biopsy of the mediastinal lymph nodes showed extensive plasma cell infiltration in the lymph nodes. A positron emission tomography (PET) scan revealed a right-sided perihilar pulmonary lesion, right-sided para-mediastinal pulmonary nodules, and multiple lymph nodes throughout the chest. He was started on a seven-day prednisone regimen. Bone marrow biopsy was negative, and Human Herpesvirus-8 was not detected. Inflammatory markers, including

	ESR, CRP, and IL-6 levels were high. This together with a constellation of clinical findings and imaging, a diagnosis of multicentric HHV-8 negative Castleman's disease was made and Siltuximab every three weeks was initiated. His symptoms resolved after the first cycle of Siltuximab but recurred after the second cycle when the course of prednisone was completed. The PET scan after 2 cycles showed a mixed response with increased uptake in supraclavicular lymph nodes and decreased uptake in the chest lymph nodes. There was a slight increase in left pleural and pericardial effusions. Inflammatory markers remained stable with slight improvement. He underwent thoracentesis of exudative effusion with no evidence of lymphoma. He also underwent pericardiocentesis of inflammatory effusion. These interventions led to significant improvement in his symptoms, and the treatment was changed to weekly Siltuximab to which he was responding well. However, four weeks later, his symptoms progressed again with chest pain, and shortness of breath and he noticed a new suddenly enlarged right supraclavicular lymph node. Repeat pericardiocentesis for recurrent pericardial effusion showed negative cytology. The biopsy of the new lymph node biopsy revealed large cell lymphoma with extensive necrosis with the expression of PAX-5, CD15, CD30, MUM-1, and negative for CD20, CD19, CD79a, and CD45 favoring the diagnosis of gray zone lymphoma. Conclusion: We presented the case of an idiopathic MCD who developed gray zone lymphoma despite treatment. We conclude that a rebiopsy should be performed when in doubt about the diagnosis.
Michelle Berning Ben Byun Dr. Kevin Chang Dr. Jessica Hane	*Dermatomyositis as the Initial Manifestation of Lung Cancer* Introduction: Dermatomyositis is an inflammatory myopathy characterized by symmetric, proximal muscle weakness, evidence of muscle inflammation, and characteristic cutaneous findings (e.g., shawl sign, Gottron's papules, heliotrope eruption). We present an atypical case of dermatomyositis that led to diagnosis of metastatic small cell lung cancer. Case Report: A 59-year-old man with a history of 30 pack-years smoking and asthma presented with a 2-month history of facial and tongue edema, as well as diffuse erythematous maculopapular rash that began several weeks after completing a course of cephalexin. Physical exam revealed a temperature of 99.2 F, periorbital and tongue edema, erythematous maculopapular rash of face, chest, upper extremities, and upper back, purpuric tender papules at distal fingertips, and lungs with diffuse crackles bilaterally. Initial lab workup revealed leukocytosis to 16,200 WBCs/μL with neutrophilia and normal eosinophils, AST 379 IU/L, ALT 144 IU/L, and elevated CRP to 44.0 mg/L. CT Chest demonstrated significant mediastinal, supraclavicular, and left axillary adenopathy. Punch biopsy revealed interface dermatitis, consistent with drug eruption versus Drug Reaction with Eosinophilia and Systemic Symptoms Syndrome (DRESS). The patient received oral prednisone for presumed DRESS with minimal improvement of his rash and facial edema. Upon further interview, the patient described over a year of generalized weakness and several weeks of dysphagia. Physical exam revealed proximal muscle weakness of bilateral lower extremities. Subsequent labs revealed elevated CK to 2964 units/L, elevated aldolase to 28.1 units/L, positive ANA, and negative Jo 1 Ab. MRI of the lower extremities showed asymmetric intramuscular signal in both thighs consistent with polymyositis. The patient was treated with high-dose oral prednisone followed by a taper with improvement in his rash and edema. Lymph node biopsy revealed metastatic

	small cell carcinoma. Oncology evaluated the patient and initiated chemotherapy. Discussion: This case highlights both typical and atypical features of dermatomyositis, as well as the importance of screening for cancer in patients with dermatomyositis. While proximal muscle weakness with characteristic rash are typical manifestations of dermatomyositis, dysphagia has also been reported in 18-20% of patients with dermatomyositis. Subcutaneous edema is a rarer manifestation; as of 2019, there were <30 cases of paraneoplastic edematous dermatomyositis reported in literature. As illuminated in our case, approximately 20-30% of patients with dermatomyositis develop malignancy within 5 years of symptom onset. Clinicians should have a high index of suspicion for malignancy in patients with dermatomyositis, and age-appropriate cancer screening is recommended.
Mauricio Jin Dr. Gerardo Calderon	*Recurrent Intussusceptions after Roux en Y Gastric Bypass* A 44-year-old woman presented to the Emergency Department with three days of abdominal pain, nausea, vomiting, and diarrhea. Her past medical history is significant for Roux-en-Y gastric bypass (RYGB) complicated by gastrojejunal ulcer perforation status-post anastomosis revision and multiple intussusception episodes requiring Petersen's defect repair. She was hospitalized twice within the past three months for recurrent episodes of small bowel intussusception, which resolved spontaneously. Bariatric surgery team planned for gastric bypass reversal following smoking cessation. On examination, the patient was afebrile with stable vital signs. Physical exam demonstrated abdominal pain in the left quadrants with voluntary guarding, no peritoneal signs, and pain similar to prior intussusception episodes. Abdominal CT demonstrated small bowel-small bowel intussusception at the level of the jejunojejunostomy without obstruction. The patient was diagnosed with recurrent nonobstructive intussusception and managed conservatively. Intussusception is a potentially life-threatening complication following RYGB. While incidence in the general population is as low as 2-3/1,000,000, incidence of post-RYGB intussusception is reported up to 6/1,000. Furthermore, while non-post-RYGB intussusception is associated with a lead point and anterograde telescoping, post-RYGB intussusception is associated with no lead point, jejunojejunostomy involvement, and retrograde telescoping. Complications of intussusception include bowel obstruction, intestinal ischemia, necrosis, perforation, and peritonitis. Common physical exam findings include acute abdominal pain, nausea, and vomiting. Symptom intensity is influenced by intussusception extent. Fever is typically not seen until necrosis or bowel perforation occurs. Diagnosis is frequently made by abdominal CT, demonstrating an outer intussuscipiens, inner intussusceptum, and a fat density within the intussusception representing invaginated mesenteric fat, corresponding to the classic “target sign”. The etiology of post-RYGB intussusception is poorly understood, postulated to be iatrogenic motility disturbance. Roux limb construction involves transection of jejunum from duodenal pacemaker cells, where small bowel motility begins. One theory proposes that ectopic pacemaker cells may subsequently arise in the roux limb, inciting anterograde and retrograde peristalsis. The jejunojejunostomy then becomes a rendezvous between

	peristaltic waves from duodenal and Roux limb ectopic pacemaker cells, resulting in intussusception. Another theory proposes that weight loss and subsequent mesenteric thinning leads to increased mobility around the jejunojejunostomy, contributing to intussusception. Non-complicated intussusception can be managed supportively. Complicated post-RYGB intussusception is surgically managed with reduction, reduction and plication, bowel resection, jejunojejunostomy revision, or RYGB reversal. The current data, while sparse, reports recurrence rates up to 33% following reduction with or without plication and up to 12.5% following resection. Intussusception should be considered in the differential of acute intermittent abdominal pain in patients post-RYGB. Noncomplicated intussusception may be managed supportively, while complicated intussusception requires prompt surgical intervention. Despite surgical treatment, intussusception recurrence rates remain high and remains an important process to consider post-RYGB abdominal pain.
Cyrus Nouraee Dr. Matthew Colin Turner	*Severe Exertional Rhabdomyolysis in a Young Ultra-Athlete: Management and Practical Recommendations for Return to Activity* Introduction: Exertional rhabdomyolysis (ER) is a phenomenon characterized by muscle breakdown associated with strenuous exercise and has been well documented amongst athletes. Case series have documented its incidence in runners, swimmers, bodybuilders, and American football players. While it is relatively uncommon (for example, 29.9 cases per 100,000 patients per year among military personnel), it requires prompt medical attention. Patients typically present with severe myalgias, transient elevation of serum creatinine kinase (CK), and possible myoglobinuria and/or acute kidney injury. Risk factors for ER include drug and alcohol abuse, stimulant use such as phentermine, sickle cell trait, hyper- and hypothermia, and viral infections. Case Presentation: We present the case of a 30-year-old ultra-athlete male with no significant past medical history who presented after running continuously in a race for 18 hours. The patient urinated dark urine at 6 hours which later progressed to “Dr. Pepper” urine and severe myalgias prompting medical evaluation. In the emergency department, labs were significant for: CK >80,000, creatinine of 1.31 (baseline 0.94), AST 3247/ALT 669. Urinalysis showed brown urine with a large amount of blood but 12 red blood cells; urine myoglobin was 261. The patient received 2L normal saline in the emergency department and was started on combination hydration therapy with normal saline as well as sodium bicarbonate in D5W to alkalinize the urine and reduce renal toxicity. Creatinine worsened, before peaking, and coming down to 1.20 at discharge. For this patient, return to very light activity was recommended at discharge and follow-up with a sports medicine physician shortly thereafter. Guidelines on return to activity in those with ER are widely inconsistent and anecdotal. The most commonly utilized model is the three-phase approach: Phase I (oral hydration, good sleep, staying cool, and waiting until CK levels are <5x the upper limit of normal and urinalysis is normal before progressing to Phase II), Phase II (light activities at one’s own pace and if asymptomatic for two weeks progression to Phase III), Phase III (gradual return to regular activity). Conclusion: Risk stratification must be considered since those at high risk of recurrence should see a sports medicine physician before returning to normal

	activity. Although relatively rare, internists should carry a high index of suspicion for ER in athletes who present with severe myalgias. Risk factors, treatment, and guidelines for return to activity are important to understand in order to minimize morbidity.
Meagan Nowariak Dr. Andrea Elliott	*Mending Broken COVID-19 Hearts: A Case of Multisystem Inflammatory Syndrome in an Adult* Introduction: Multisystem inflammatory syndrome (MIS) is an immune-mediated syndrome associated with COVID-19 infection first described in 2020. While rare, effecting ≤1% of patients with COVID-19, this condition can be severe and present with a wide array of clinical symptoms including gastrointestinal symptoms, cardiogenic shock, rash, fever, and neurocognitive symptoms. We present a case of MIS in a previously healthy 21-year-old female who initially presented with gastrointestinal symptoms, then later developed a pulseless electrical activity (PEA) arrest requiring extracorporeal membrane oxygenation (ECMO). Case Description: A 21-year-old female with no past medical history presented to a rural emergency department with a two-day history of abdominal pain, chest pain, shortness of breath, nausea, vomiting, and diarrhea. She also reported a three-week history of cold symptoms and was found to be COVID-19 positive. An abdominal ultrasound showed gallbladder thickening, and she was transferred to obtain a higher level of care for possible cholecystitis and sepsis. During transit, she became hypotensive with a blood pressure of 69/58. Emergent echocardiogram revealed an ejection fraction of 15%, myocardial wall edema, and a small pericardial effusion. A right heart catheterization demonstrated severe biventricular failure. Laboratory results showed white blood cell count of 18,600, rising lactate at 9.44 mmol/L, and rising troponins at 5.0 ng/ml. She was subsequently transferred to a higher level of care for consideration for ECMO support. Shortly after arrival, the patient had PEA arrest and was taken emergently to the catheterization lab and placed on venous-arterial ECMO. Her hospitalization was complicated by abdominal compartment syndrome secondary to a large retroperitoneal hematoma requiring surgical decompression, acute kidney injury requiring continuous renal replacement therapy, shock liver, acute respiratory distress syndrome, and multiple ischemic cerebellar strokes. Further laboratory workup revealed c-reactive protein 140mg/L, erythrocyte sedimentation rate 101 mm/hr, interleukin-6 733 pg/mL, and ferritin 2,598 ng/mL. Throughout her admission, she was treated with high dose steroids, anakinra, and intravenous immunoglobulin for suspected MIS. She was successfully decannulated from ECMO after 13 days, discharged to an acute rehabilitation unit 57 days after admission, and is now home with full cardiac, hepatic, neurologic (Cerebral Performance Category scale of 1) and renal recovery. Discussion: Since its recognition in 2020, MIS has been reported to cause a wide array of clinical manifestations. Our patient initially presented with abdominal pain but developed cardiogenic shock shortly after presenting to the emergency department. Her condition progressed to meet the Centers for Disease Control criteria for MIS in an adult. While first described and best known in children, MIS is increasingly being identified in adults, typically with gastrointestinal and cardiovascular manifestations. While society is quickly becoming complacent to COVID-19, this case report highlights the continued devastating effect of COVID-19 and importance of early recognition for providers.

Afsar Sandozi	*A Weighty Matter: a Clinical Presentation of Differentiation Syndrome in Acute Promyelocytic Leukemia* Introduction: Acute Promyelocytic Leukemia (APL) is a subtype of acute myeloid leukemia characterized by a 15:17 chromosomal translocation, resulting in an abnormal fusion gene called PML/RARa. While aggressive, this malignancy is highly curable with administration of all-trans retinoic acid (ATRA) and arsenic trioxide (ATO). Differentiation syndrome (DS) is a potentially fatal complication of this treatment regimen that occurs in 25% of patients, and has a varying presentation including fever, hypotension, hypoxia with pulmonary infiltrates, peripheral edema, and end-organ damage. Prompt recognition and treatment of DS is imperative to the well-being and survival of patients. Case Presentation: A 45 year-old male with no prior medical history presented to the emergency department with a one-week history of fatigue, bruising, epistaxis and gingival bleeding. Laboratory studies revealed hemoglobin of 9.2 g/dL, white blood cell count of 0.96 k/cmm, platelet count of 16 k/cmm, INR of 1.2, and creatinine of 0.98 mg/dL. A peripheral smear showed numerous malignant-appearing promyelocytes, highly suspicious for APL. He was initiated on ATRA and ATO on hospital day 1, with confirmatory FISH testing showing t(15;17) chromosomal translocation on day 2. During treatment induction, he was closely monitored for complications including disseminated intravascular coagulation with associated hyperfibrinolysis, infection due to immunosuppression, bleeding diathesis due to thrombocytopenia, and tumor lysis syndrome. On hospital day 10, the patient's WBC rose to over 10 k/cmm and his LFTs were mildly elevated. Apart from a 5 kg weight gain, he did not have any other initial presenting symptoms or exam findings. DS was immediately suspected and a three-day course of glucocorticoids was administered. His WBC subsequently rose to over 60 k/cmm, and he experienced increased weight gain, pedal edema, and facial puffiness over this time. On hospital day 12, hydroxyurea was added to mitigate hyperleukocytosis, and on day 18, ATO was held. The patient's condition and labs improved with the modified regimen over the next few days, and on day 21 full-dose ATO was resumed. On day 29, the patient completed induction therapy. Bone marrow biopsy on day 31 confirmed complete remission with incomplete hematologic recovery, and he was started on consolidation therapy. Conclusion: Differentiation syndrome is a potentially fatal complication of APL and can be caused by either ATRA or ATO. Varied presentation makes diagnosis challenging. Prompt recognition is critical to avoid end-organ damage and mortality. This case showcases timely recognition of APL and DS. Such vigilance is critical to the administration of appropriate therapy to prevent mortality of patients.
Sarah Schrup Dr. Daniel Khan Dr. Jesse Vance Dr. William Ward	*A Case of Rapidly Advancing Dysphagia Presenting with Pulmonary Embolism* Introduction: An estimated 20% of episodes of new venous thromboembolism (VTE) are associated with malignancy and VTE is a leading cause of mortality in patients with malignancy. The primary site of cancer and the presence of metastasis are strong predictive factors for the risk of VTE in patients with malignancy. Medical comorbidities also affect the individual risk of VTE.

	Case Presentation: Here we present the case of a 77-year-old man with a pertinent medical history including atrial fibrillation, ischemic congestive heart failure, history of coronary artery bypass grafting, previous deep vein thrombosis (DVT), type II diabetes mellitus, stage III chronic kidney disease, gastroesophageal reflux disease and microcytic anemia presenting with generalized weakness and dizziness in the setting of a 3-month worsening history of dysphagia and dizziness. The patient had been undergoing outpatient workup for dysphagia progressing to inability to swallow fluids or solids at the time of admission. He also reported a 35-pound weight loss over 3 months. Endoscopy two months prior revealed erythematous esophagus and abnormal mucosa but no obvious signs of malignancy. Repeat endoscopy 4 days prior to admission was aborted due to a large amount of food in the stomach despite the very limited intake of the patient. CT angiogram of the chest abdomen and pelvis on admission revealed several bilateral segmental and subsegmental pulmonary emboli as well as marked thickening of the gastroesophageal junction with enlarged lymph nodes in the gastric hepatic ligament concerning for distal esophageal neoplasm. Multiple new hypoenhancing masses throughout both hepatic lobes had developed since previous imaging were concerning for metastases. A masslike area near the hilum of the liver with intrahepatic biliary ductal dilatation was noted, possibly representing an obstructing metastasis. Ultrasound revealed bilateral leg lower leg DVTs. Anticoagulation with high intensity heparin was initiated. Endoscopic ultrasound was performed during hospitalization. On endoscopy, an ulcerating mass was identified in the lower-third of the esophagus. Biopsies were retrieved and indicated adenocarcinoma. Stomach and examined duodenum appeared normal. On endosonography, evidence of invasion into the adventitia and regional lymph nodes, as well as multiple lesions in both hepatic lobes were identified. Discussion: Compared to other primary malignancy sites such as brain, pancreatic, and gastric, the rate of VTE in esophageal cancer has been found to be moderate. The presence of likely metastatic disease, as in this patient, increases the risk for VTE. Finally, comorbidities play a role in VTE risk. Applied to this case, congestive heart failure, renal disease and anemia increased this patient's risk for VTE. Patients with a malignancy-related VTE have been shown to be at higher risk of mortality. This case highlights the importance of identifying individual risk factors for VTE for patients with malignancy.
David Wu Dr. Keith Skubitz	*Lack of Response to Targeted Therapy of a Desmoplastic Small Round Cell Tumor Bearing a Novel Sonic Hedgehog Pathway Mutation* Introduction: The dramatic response of gastrointestinal stromal tumors, a tumor for which no effective therapy was previously available, to the tyrosine kinase inhibitor imatinib heralded a new era of targeted therapy in solid tumors. For some cancers, targeted therapy now shows a higher response rate and longer survival as compared to conventional chemotherapy, and currently targeted therapy is the preferred approach for some tumors. Next generation sequencing (NGS) is often used to identify potential targets. Case Presentation: In this clinical vignette we present a case of desmoplastic small round cell tumor, a member of the Ewing family of tumors, with a characteristic CIC-DUX fusion, who relapsed after standard chemotherapy.

	The patient was a 39-year-old woman who presented with an arm nodule that was removed showing a CIC-DUX sarcoma. The CIC-DUX fusion results in a transcription factor with oncogenic properties, although the relevant downstream signaling pathways are unknown. CIC-DUX4 sarcomas have been shown to have a wide spectrum of morphology with generally a more aggressive course and inferior overall survival as compared to Ewing sarcoma. There are no therapies targeting CIC-DUX4 rearrangements available at present. The patient relapsed 2 years after standard chemotherapy, and following a transient response to pegylated-liposomal doxorubicin, the decision was made to explore an experimental therapy. NGS in our patient revealed a mutation in patched homolog 1 (PTCH1). PTCH1 is part of the sonic hedgehog (Shh) signaling pathway; mutations in this pathway have not been reported in Ewing family tumors and have traditionally been implicated in cancers such as nevoid basal cell carcinoma syndrome, or Gorlin-Gotz syndrome. Vismodegib is a small molecule inhibitor of smoothened (SMO), a key part of the Shh pathway. Vismodegib inhibits Shh signaling and is approved by the FDA for treatment of locally advanced and metastatic basal cell cancer (BCC). BCCs commonly have PTCH1 mutations and those with PTCH1 mutations are often responsive to Shh inhibition via vismodegib. We therefore treated this patient with vismodegib at 150 mg/d orally. After one month, imaging showed tumor progression. Conclusion: This case demonstrates that identification of a mutation that is a target for a specific therapy does not guarantee that the tumor will respond to the targeted therapy. The utility of targeting a potential mutation likely depends upon the background biochemistry of the malignant cell and the degree to which cell survival depends on the targeted protein. In our case, it appears that the CIC-DUX4 rearrangement resulted in sarcoma cell growth that was not dependent on Shh signaling.
Agnes Zhu Dr. Cecilia Mitchell Dr. Jonathan Lang Dr. Alexander Theofiles	*Disseminated Gonococcal Infection: A Case of Septic Polyarthritis without Dermatitis* Introduction: Neisseria gonorrhoeae infection is the second most common sexually transmitted bacterial infection worldwide, with over 600,000 cases reported annually in the United States. Between 0.5 and 3% of patients with gonorrhea develop disseminated gonococcal infection. Patients may present with localized purulent arthritis or “arthritis-dermatitis syndrome,” which comprises tenosynovitis, dermatitis and polyarthralgia. Therapy typically consists of intravenous ceftriaxone. Patients should be tested for Chlamydia trachomatis co-infection. Case Description: A 57-year-old man with type 2 diabetes mellitus and hypothyroidism was admitted to the hospital with a 5-day history of swelling, redness, and pain in his right elbow, bilateral ankles, and upper neck. He endorsed several days of dark brown urine, but otherwise denied hematuria, penile discharge, other genitourinary symptoms, or rash. He had no recent tick or mosquito bites and no personal or family history of crystalline arthropathy or autoimmune disease. He denied headache or changes in mental status. Sexual history did not reveal risk factors for sexually transmitted infection. At presentation, he was afebrile and tachycardic. Laboratory studies were notable for WBC 23 × 109/L, sodium 126 mmol/L, creatinine 2.20 mg/dL, sedimentation rate 118 mm/h, and c-reactive protein >400 mg/L. Ankle x-ray was negative for fractures or dislocations. On physical exam, right elbow and

	bilateral ankles were warm, swollen, erythematous, and had decreased passive range of motion. There was paraspinal tenderness in the upper cervical spine. A cervical spine MRI showed findings suspicious for early inflammatory versus septic arthritis involving the atlantoaxial and atlantooccipital joints. Rheumatologic workup revealed mildly positive rheumatoid factor and negative ANA, CCP, ANCAs. Infectious workup revealed negative blood testing for Lyme, HIV, Hepatitis B and C, and syphilis. Urine gonorrhea and chlamydia PCR were negative. Joint aspiration was performed, and fluid analysis showed 135,000 cells/mcL from the right ankle and 6,000 cells/mcL from the right elbow. Gram stains were negative, and no crystals were seen on polarized microscopy. He was started on vancomycin and ceftriaxone for suspected septic arthritis. The patient subsequently underwent irrigation and debridement of the right ankle. Synovial fluid cultures and 16S ribosomal RNA broad range PCR resulted positive for Neisseria gonorrhoeae. The patient's symptoms improved IV ceftriaxone and he was dismissed to continue a four-week course. Discussion: This case demonstrates the importance of keeping a broad differential when patients present with polyarticular pain and swelling. In this case, initial suspicion for septic or gonococcal arthritis was lower given the patient's history, nontoxic appearance, negative blood cultures and gonococcus testing, and lack of genitourinary symptoms. Synovial fluid analysis was imperative for diagnosis and to guide both medical and surgical management.
Quality Improvement - Medical Students	
Sarah Alabsi Dr. Brian Hilliard Dr. James Grace	*Evaluation of Cardiac Telemetry Ordering Practices: A Multi-Site Retrospective Chart Review* Introduction: Many hospital patients with cardiac-related reasons for admission are placed on telemetry monitoring. Telemetry has expanded from use predominantly in the intensive care unit (ICU) to more frequent usage in lower-risk units. Even though many well-respected organizations have specific guidelines for telemetry use, patients are often still placed on telemetry for non-indicated reasons. Unsurprisingly, this leads to unnecessary financial costs, with estimates between $53 and $1400 per day dependent on the hospital system. Additionally, inappropriate telemetry orders may reduce availability of this technology for those who have an indicated need. In this study, we reviewed the telemetry orders of physicians at various M Health Fairview hospital sites in order to understand telemetry usage rationale including reason for and duration of the orders. Case Presentation: We retrospectively examined 2,119 records of every adult inpatient who was on cardiac telemetry within the health system during the month of June 2021. Patients admitted under observation or admitted to the ICU were excluded. Orders were then compared to the American Health Association's (AHA) 2017 practice standards for the appropriate use of continuous cardiac monitoring. Orders were said to meet practice standards if the indication was appropriate and the monitoring ended within the recommended time frame (either by order discontinuation or patient discharge). Excess hours of telemetry were calculated by subtracting

	maximum indicated time from the total time on telemetry. For indications that were not included in the practice standards, maximum time indicated was zero hours. 76 ICU charts were removed from the total count. 31.5% of reviewed telemetry orders were deemed appropriate while 57.2% were inappropriate orders due to their indication and/or duration. The remaining 11.3% of orders were made without indication and therefore could not be evaluated. The three most commonly used order indications not included in the 2017 AHA guidelines were alcohol withdrawal, sepsis, and evaluation for arrhythmia. Inappropriate orders resulted in 97,953 hours of excess telemetry use. Using a conservative value of $53.44 per day for the cost of inpatient cardiac monitoring, monetary waste in June 2021 was calculated to be $218,108.68. Conclusion: Decreasing inappropriate telemetry usage by identifying a standard with which to follow presents an opportunity for major time and monetary savings within this health system. Changing EHR order sets and adapting EHR best practice alerts that abide by AHA guidelines may be a way to reduce excess telemetry usage.
Medical Transitional Graduates	
Clinical Vignette – Medical Transitional Graduates	
Misha Gautam Dr. Pallawi Torka Dr. Karan Jatwani	*Think About Thiamine: A Rare Case of Wernicke's Encephalopathy in a Patient with Lymphoma* Introduction: High-grade hematological malignancies are a common cause of thiamine deficiency, but it is generally asymptomatic and rarely severe. Common causes of encephalopathy in malignancy patients include CNS spread, infections, electrolyte abnormalities, and drug induced. We report a rare case of diffuse large B-cell lymphoma (DLBCL) patient with Wernicke's encephalopathy (WE). Lack of awareness among clinicians and undue association with alcohol misuse often causes a delay in diagnosis which can lead to death from this otherwise treatable entity. Case Report: A 58-year-old woman presented with anemia and thrombocytopenia. CT chest demonstrated extensive axillary and supraclavicular lymphadenopathy. Lymph node and bone marrow biopsies revealed chronic lymphocytic leukemia (CLL) with Richter's transformation to DLBCL. She was lethargic but had no B symptoms. A PET/CT scan revealed metabolically active lymphoma involving lymph node chains above and below the diaphragm, and hepatic infiltration- stage IV disease. On day one of therapy with rituximab and dose-adjusted etoposide, doxorubicin, vincristine, cyclophosphamide, and prednisone (R-DA-EPOCH), she developed progressive confusion. CT brain and CSF examination showed no evidence of malignant cells by cytological exam or flow cytometry. No electrolyte abnormalities or infectious causes were discovered. Her medications were checked for delirium-causing drugs and polypharmacy and no potential culprits were found. She continued to demonstrate acute fluctuating altered consciousness and cognition. Subsequent MRI brain showed T2 hyperintensity involving dorsomedial thalami, periaqueductal gray matter, and mammillary bodies suggesting WE. Her whole blood thiamine level was 36 nmol/L (normal = 74-222 nmol/L). High-dose IV thiamine- 500 mg every 8 hours, folic acid 1 mg daily, and TPN were started.

	Rapid improvement to baseline and return to a normal diet was seen. After 3 days, the dose was reduced to100 mg every 6 hours for 2 days, then 250 mg once daily for 5 days, and 100 mg oral thiamine thereafter. She completed 2 more cycles of chemotherapy with prophylactic intrathecal methotrexate achieving a complete metabolic response. She was discharged after 54 days on thiamine supplementation, neutropenia prophylaxis, intensive rehabilitation, and close clinical surveillance. Discussion: Cancer patients have several predispositions to thiamine deficiency- decreased availability [e.g., anorexia, vomiting, malabsorption, malnutrition], accelerated use by rapidly growing cancer, or hypermetabolic states [e.g., infections, critical illness, steroid use], iatrogenic causes [e.g., hemodialysis, glucose loading, parenteral nutrition], impaired function of thiamine-dependent enzymes [e.g., cofactor deficiency; hypomagnesemia, chemotherapy-induced], increased loss, or other yet to be explored mechanisms. Rarely, the deficiency may be severe enough to cause WE. Hence, otherwise unexplained neurological symptoms in cancer patients should prompt oncologists to consider WE and initiate timely treatment and clinical suspicion must not be limited to the presence of the classic triad of altered mentation, ataxia, and ophthalmoplegia. Our case highlights that once identified, it is relatively easy to treat, and timely therapy can lead to complete resolution of symptoms. Raising awareness about this entity will help prevent serious and permanent neurologic sequelae in patients.
Beatriz Sordi Chara Dr. Daniel Penrice Dr. Kamalpreet Sing Hara Dr. Douglas Simonetto	*NASH Cirrhosis in a Young Patient Diagnosed with Telomere Biology Disorder: A Case Report* Introduction: Telomeres are repeat DNA-protein sequences that cap the ends of chromosomes, preventing chromosomal tangling, interruption of cell proliferation and apoptosis. The telomere structure shortens with each cellular division, until it reaches a critical point where it activates the natural senescence phenomenon. When telomeres suffer from premature length reduction, due to a genetically inherited disorder, several organ systems can be affected, a condition termed Telomere Biology Disorder (TBD). Bone Marrow Failure, Pulmonary Fibrosis and Liver Disease are some of the conditions known to be associated with TBD. We present a case of a patient with hepatic steatosis and advanced liver fibrosis, who was diagnosed with TBD. Case Presentation: A 45-year-old male patient presents to the Hepatobiliary Clinic for evaluation of abnormal transaminases and presumed advanced liver fibrosis. Previous laboratorial and imaging exams showed elevated Alanine Aminotransferase (55 IU/L), and Aspartate Aminotransferase (42 IU/L), hepatic fibrosis stage 3-4 on Transient Elastography and splenomegaly, of 14.7 cm, on abdominal computed tomography (CT) scan. Initial workup in our clinic included a Magnetic Resonance Elastography, demonstrating 20% of hepatic fat content and a mean liver stiffness of 4.98 kPa, consistent with advanced liver fibrosis. Chest CT scan excluded interstitial lung disease, and esophagogastroduodenoscopy ruled out esophageal varices. The patient did not suffer from any skin condition, and there was no clinical or laboratory evidence of hematologic disorders. Given his young age and lack of risk factors, telomere testing was ordered, revealing lymphocyte telomere length of 10% and granulocyte telomere length around 1%, suggesting TBD. One year follow-up Transient Elastography revealed hepatic steatosis grade S3 with a 16.3 kPa stiffness. The patient was scheduled for Gastroenterology visits every 6-months for

	laboratory exams and Hepatocellular Carcinoma screening. Conclusion: Telomere Biology Disorders are a group of diseases related to germline mutations that affect telomere maintenance. The telomere length can be evaluated in peripheral blood leukocytes with results below the age-adjusted 10th percentile considered suggestive of TBD. Genetically inherited telomere disorders have been associated with the occurrence of cryptogenic cirrhosis, likely secondary to reduced regenerative capacity of hepatocytes. It was also demonstrated that TBD may accelerate the development of advanced fibrosis in patients with liver diseases of different etiologies. Recent publications, correlating cell senescence and Nonalcoholic Fatty Liver Disease, reported a higher incidence of telomere shortening in hepatocytes of patients with hepatic steatosis, when compared with controls. TBD must be considered in young patients who develop hepatic fibrosis, especially if when risk factors for chronic liver disease are absent or there is no personal or family history of pulmonary disease, premature greying of the hair, or bone marrow failure. Awareness of the connection between premature telomere shortening and early onset liver disease can benefit the clinician in identifying the etiology in challenging cases and to guide screening and surveillance for other manifestations of TBD.
Residents	
Quality Improvement - Residents	
Thomas Schmidt Dr. Bickey Chang Nellie Adams Elsie Gertner	*Tocilizumab Use in COVID-19: A Retrospective Review of Secondary Bacterial Infections and Side Effects* Background: The clinical course of COVID-19 is defined by its ability to cause a hyperinflammatory state in its hosts, leading to significant morbidity and mortality worldwide. In the rush to develop therapeutics to prevent and or diminish the effect of COVDI19, tocilizumab, a potent-anti-inflammatory drug, was found to reduce the mortality of patients who were hospitalized with COVID19. In rheumatologic disease, tocilizumab is utilized, though patients have experienced side effects such as bacterial infections, fungal infections, unmasking inflammatory bowel disease or gastrointestinal perforation. Methods: In a retrospective single arm quality improvement project, we assessed the frequency of secondary infections or GI complications in patients hospitalized with COVID19 and were treated with tocilizumab. Of the 104 patients analyzed, 41% of patients developed and were treated for a secondary bacterial infection, 1.9% for fungal infections, which is similar to previous studies of secondary bacterial infection in intubated ICU patients. Conclusions: No patients developed GI side effects, where it is seen in about 1.9 per 1000. Tocilizumab use did not lead to significant increase in secondary infections nor did it cause GI side effects.
Zafar Siddiqui Dr. Brett Austin Dr. Joshua Daum Dr. Jack Korleski	*Breaking Down Barriers: A Qualitative Analysis of Colorectal Cancer Screening in Somali-speaking Populations in Primary Care* Introduction: Colorectal cancer (CRC) is the second most common cause of

Dr. Jack McHugh Dr. John Matulis	cancer-related death in the United States. While rates of CRC screening in the internal medicine resident continuity clinic at Mayo Clinic Rochester are 75%, CRC screening rates are only 46% for the Somali-speaking population in this clinic. Our project focused on understanding the barriers to successful multitarget stool DNA testing (Cologuard®) completion in the Somali-speaking population in our primary care clinic. Methods: An audit of CRC-eligible Somali-speaking patients attending the resident continuity clinic between July-September 2022 was conducted. While eight patients had a Cologuard ® ordered, only one patient subsequently completed screening. A survey with open-ended, standardized questions was utilized to identify barriers patients faced when attempting to complete the Cologuard® kit and to evaluate the potential efficacy of interventions aimed at minimizing these barriers. Results: Five out of eight (63%) surveys were completed via phone call. Various barriers were identified including: confusion regarding how to complete and submit a test, with patients relying on English-speaking family members who were not always available to assist with Cologuard® instructions, and a lack of patient awareness when an inadequate sample was submitted, which occurred in 2 of 8 cases. It was also noted during the course of the study that in cases where the sample was found to be inadequate, the ordering physician was not directly notified. Three out of the five patients who completed the survey indicated that in-person nursing education visits with a Cologuard ® demonstration kit, video instructions in Somali, and written instructions in Somali would all be helpful in completing the screening. Conclusion: This data elucidated several barriers to the successful completion of CRC screening with Cologuard® in the Somali-speaking population. Additionally, this study highlights the lack of Somali instructions and resources available to aid in completing this kit. Limitations to this project include small sample size and a low success rate in reaching patients. In the future, this study will continue to grow in sample size and will include data regarding the impact of interventions identified as potentially efficacious in this study. These findings may help providers understand how to help extend equitable care to Somali communities and other minority populations with significant language barriers.
Benjamin Swart Dr. Aileen Ahiskeli Jack Wolf	*Implementation of an Empiric Ivermectin Treatment Protocol for Patients Receiving High-Dose Corticosteroids for Severe COVID-19* Introduction: Strongyloides stercoralis is a parasitic roundworm that is present worldwide and can cause lifelong and largely asymptomatic infection. Immunosuppression, particularly use of corticosteroids, is a risk factor for disseminated strongyloidiasis, a severe disease state that can lead to septic shock and death. Our institution implemented an empiric strongyloidiasis screening and treatment protocol using ivermectin for inpatients being treated with high-dose corticosteroids for severe COVID-19. Methods: The electronic medical record was queried for all severe COVID admissions from June 10, 2020 until May 31, 2021. Patients meeting criteria for severe COVID received dexamethasone 6-20mg daily for 10 days depending on the amount of ventilatory support required. If patients were from a tropical or semi-tropical area, defined as anywhere the ground does

	not freeze, they were also recommended to empirically receive ivermectin 200mcg/kg daily for two consecutive days and have Strongyloides serology drawn. If serology returned positive, they were also recommended to receive an additional round of ivermectin 200mcg/kg daily for two consecutive days two weeks after their initial ivermectin administration. Patient-reported race, ethnicity, and language was used to place patients into demographic categories. These demographic categories were used as surrogates for Strongyloides exposure. High-risk groups for Strongyloides exposure at our institution were primarily comprised of North American – Hispanic, Sub-Saharan African, and Southeast Asian demographics. A chart review was performed for every individual with positive Strongyloides serology or who received additional doses of ivermectin outside of the empiric treatment protocol to determine if there was suspicion or evidence of disseminated strongyloidiasis. Suspicion for disseminated strongyloidiasis included documentation in primary team or Infectious Diseases consult service progress notes, unexplained Gram-negative bacteremia, or unexplained septic shock. Results: From June 10, 2020 until May 31, 2021 there were 899 admissions for primary COVID representing 839 individual patients. 58% of COVID admissions received steroid treatment. Of those receiving steroids, 31% had Strongyloides serology drawn. Of those who received steroids and had Strongyloides serology drawn, 136/151 (90%) also received ivermectin. Approximately 65% of those with demographics consistent with Strongyloides exposure were screened for Strongyloides and 63% were empirically treated with ivermectin. There were three cases of concern for possible disseminated strongyloidiasis, though none were confirmed. All three cases were among the North American – Hispanic demographic and had positive Strongyloides serology, and all survived. Conclusion: Our empiric ivermectin protocol appeared to be successful, as most patients included in high-risk demographics received Strongyloides screening and empiric treatment with ivermectin. It is unclear from the results of our study whether this had a measurable effect on risk of disseminated strongyloidiasis or mortality rate among our sample population.
Research - Residents	
Bethlehem Atoma Angela Fabbrini Barbara Clothier Megan Campbell Dr. Anne Melzer	*Variation in Reporting of Incidental Findings on Lung Cancer Screening and Association with Subsequent Assessment* Rationale: The presence of incidental findings (IFs) on low-dose CT (LDCT) for lung cancer screening (LCS) generates workload. The association between how IFs are reported and subsequent evaluation is not well understood. We quantified the distribution, frequency, and clinical significance of IFs on LDCT and the association of report characteristics with subsequent assessment. Methods: Retrospective chart review of patients undergoing initial LCS at the Minneapolis VA Health Care System (2015-2018). Electronic health records were reviewed to extract demographics, LungRADS coding, IFs, and subsequent documentation, testing and clinical outcomes. IFs were any non-nodule findings in the LDCT report. IFs were considered potentially significant (SIFs) if they were expected to require follow-up intervention

	which could include testing, referrals, counseling, or tracking. Category "S" is the Lung-RADS code applied when a SIF is present. Primary outcome was follow-up, defined as any assessment or intervention ordered and/or completed in relation to the finding or documented as unnecessary. High-risk SIFs were defined as potentially malignant. Outcomes were analyzed using a mixed effects model with individual patient as a random effect. Results: Patients (n=901) were primarily male (94.1%) current smokers (62.1%) with a mean age of 65.2 years. IFs were extremely common (93.9%) with an average of 2.6 IFs per scan (n=2296). Pulmonary findings (48.6%) were most common, followed by atherosclerosis (25%). 786 IFs (34.2%) were deemed likely significant. 58/786 (7.4%) were high-risk and 222/786 (28.2%) had workups ordered, completed, or documented to be unnecessary, of which 104/786 (13.2%) completed additional testing or evaluation. A minority of SIFs (293/786, 37.2%) had "S" applied to the LDCT. Reporting of IFs varied greatly by Radiologists (see Table). Despite not applying "S" category, radiologists frequently recommended testing, including for benign findings (e.g. simple cysts). Presence of a radiologist recommendation (OR 3.3, 95% CI 1.9-5.7), high-risk finding (OR 2.9, 95%CI 1.5-5.6), and reporting in the impression (OR 2.1 95%CI 1.2-3.8) were associated with increased odds of workup or documentation, while "S" code, number of IFs, presence of a suspicious pulmonary nodule, reading radiologist, and patient age were not associated with odds of workup or documentation. Conclusions: IFs are extremely common on LDCT and may be clinically significant but are not reported systematically. Reporting characteristics have a large impact on subsequent evaluation. Guidance and training to support structured reporting of SIFs may improve this process, with the goal of generating appropriate testing when needed and preventing low-value care.
Thanat Chaikijurajai Timothy Engelman Dr. Emanuel Finet Dr. Wilson Tang	*Prognostic Value of the Metabolic Gain Index Compared to Other Cardiopulmonary Exercise Testing Parameters in Patients with Chro* Background: Cardiopulmonary exercise stress test (CPET) is an essential prognostic tool in patients with heart failure with reduced ejection fraction (HFrEF) for advanced therapy evaluation. We developed the metabolic gain index (MGI) from the relative gain of the CPET-estimated stroke volume by the relative change in the product of VO2 and heart rate (HR) from resting to peak exercise, which reflects cardiac reserve function, and, potentially, prognosis in patients with HFrEF. Objective: To demonstrate the prognostic significance of the MGI compared to other standard CPET parameters in patients with HFrEF Methods: We reviewed medical records of 1,067 HFrEF patients (EF $\leq$ 40%) undergoing CPET from 12/2012 to 9/2020. The MGI was calculated from [(Peak VO2 x Peak HR)-(Resting VO2 x Resting HR)]/(Resting VO2 x Resting HR). Patients with hypotensive or bradycardia response during exercise were excluded. Primary outcome was the composite of death, LVAD implantation, and heart transplantation. We used multivariable Cox regression with subgroup analysis. ROC curves with AUCs were used to compare the MGI and other CPET parameters. Results: Among 843 HFrEF patients included (mean age 56.2±12.2 years and LVEF 24.8±8.0%), 279 (33.1%) patients had adverse events (median follow up time 897 days). After adjustment for age, sex, comorbidities, beta-blocker use, EF, heart rate reserve and peak VO2, higher MGI was associated with

	better outcome (hazard ratio per unit increase 0.92, 95% CI 0.86-0.98, p=0.016), especially in patients with age ≥ median (58 years), BMI < 35 kg/m2, ICM, and without AF (all pinteraction > 0.05). The MGI also had the highest AUC (0.73, 95% CI 0.69-0.76) among the CPET parameters including peak VO2 (0.71, 95% CI 0.67-0.74), VE/VCO2 slope (0.64, 95% CI 0.60-0.68), peak end-tidal pressure of CO2 (0.62, 95% CI 0.58-0.66) and peak work rate (0.61, 95% CI 0.57-0.65). Conclusion: Higher MGI is associated with improved LVAD/heart transplant-free survival, and the MGI may provide incremental prognostic value to standard CPET parameters in HFrEF.
Spencer Goble Dr. Philippe Nyembo Dr. George Konstantinides Dr. Brian Goodroad Dr. Amanda Noska	*Statin Prescribing Patterns Based on FIB-4 as a Non-invasive Assessment of Liver Function* Introduction: Prevention of cardiovascular disease in patients with HCV is frequently complicated by concerns for increased side effects with statins in patients with chronic liver disease. While recent literature has suggested that statins are safe in stable chronic liver disease, they remain underutilized in clinical practice and it is unclear what degree of liver dysfunction the medical community at large feels is a contraindication. The FIB-4 score provides a reasonable non-invasive diagnostic evaluation for fibrosis and cirrhosis in HCV-infected patients. In order to better understand the effects of liver disease on statin prescribing patterns, we examined the relationship between FIB-4 score and statin utilization in HCV-infected patients. Methods: We performed a retrospective study of HCV-infected individuals aged 40-75 years of age. Inclusion criteria included available information to complete the Pooled Cohort Equation and to calculate the FIB-4 score. Patients coinfected with hepatitis B or HIV were excluded. Subjects were evaluated for statin utilization based on FIB-4 scores of < 1.45, 1.45-3.24, and ≥ 3.25, corresponding respectively with high negative predictive value for severe fibrosis, intermediate risk, and high positive predictive value for severe fibrosis. Results: A total of 1,018 subjects were identified for analysis between 2019-2021. For those with FIB-4 values by group < 1.45, 1.45-3.24, and ≥ 3.25, statins were prescribed in 162/516 (31.4%), 100/363 (27.5%) and 30/139 (21.6%) of individuals respectively. Proportions for use in those who met 2019 American College of Cardiology/American Heart Association criteria for treatment by FIB-4 group were 144/347 (41.5%), 91/272 (33.5%), and 27/95 (28.4%) respectively. Discussion: Our results support that statins are underutilized in HCV-infected patients. While there was a clear trend towards greater utilization in patients with lower FIB-4 scores < 1.45, reassuring against significant liver disease, this finding was not significant at p < 0.05. Further large-scale assessment is needed to better understand statin prescribing patterns as these relate to non-invasive markers of liver dysfunction. A better understanding of the correlation between FIB-4, statin utilization, and safety and efficacy of statins in HCV-associated liver disease might help clinicians to make more individualized, informed decisions on the appropriateness of statin therapy in this high-risk population.
Andres Gonzalez Coba	*Outcomes and clinical characteristics of COVID-19 in patients with COPD*

	Introduction: COVID-19 illness severity and mortality are associated with certain risk factors, including older age, cancer, cardiovascular disease, diabetes, and chronic lung diseases. Chronic obstructive pulmonary disease (COPD) severity and outcomes also correlate with similar comorbidities. Reports on COVID-19 infection in the setting of COPD have conflicting outcomes related to disease severity and/or mortality. Our study evaluated clinical characteristics, illness severity, and mortality of COVID-19 infection in patients with and without COPD. Methods: Retrospective cohort, conducted with the Minneapolis VA COVID-19 registry included all patients admitted to the hospital with a positive COVID PCR between March 1, 2020 and January 10, 2021. Outcomes included mortality, severe COVID disease defined by 1) new or increase over baseline supplemental oxygen requirements and/or hypoxemia with SpO2 ≤94% on room air; 2) high flow nasal canula use; and/or 3) non-invasive or invasive mechanical ventilation); length of stay, venous thrombosis events (VTE). Results were stratified by inhaled corticoid steroids (ICS) or/and chronic steroid therapy. Results: The cohort consisted of 50 patients (98% male) with COPD and 159 patients (97.5% male) without COPD. Mortality was 26% vs 22% (OR: 1.24; CI 0.59-2.59, p:0.55) in the COPD vs non-COPD patients, respectively. Patients requiring invasive mechanical ventilation was 22% vs 13.2% (OR: 1.85; CI 0.82-4.17, p:0.13). Mean length of stay was 16.25 days (SD± 14.3) vs 16.26 days (SD± 14.27). Severe COVID Pneumonia was identified in 78% vs 69.2% (OR: 2.54; CI 0.94-6.9, p:0.06). Use of ICS and/or chronic steroid therapy in COPD patients in association to mortality was 29.6% vs 21.7% (OR: 1.52; CI 0.42-5.5, P:0.53). VTE events occurred in 2% vs 6.9% (OR: 0.27; 0.03-2.18, p: 0.22) in COPD vs non-COPD patients, while DVT prophylaxis was present in 98 vs 98.1%, respectively. Among comorbidities, CHF (36 vs 15%, p <0.05), CAD (58 vs 47.1%, p: 0.181), DM (34 vs 44%, p: 0.276) differed the most between COPD and non-COPD patients. Active smoking was present in 14% vs 6.2% (p: 0.08) with pack per year index of 44 vs 27 in active and prior smokers. Discussion: In this retrospective cohort of COVID-19 with and without COPD we found an increased risk of severe COVID pneumonia, increased use of invasive mechanical ventilation and increased risk of death in COPD patients, however, these did not reach statistical significance. These trends were irrespective of ICS or chronic steroid use. There was no difference in length of stay between groups. However, VTE events were associated with a decreased trend in COPD patients with no difference in rate of VTE prophylaxis. Limitations of this study include relatively small sample size, single site, and predominantly male population.
Zachary Hartnady	*Outcomes and Clinical Characteristics of COVID-19 in Patients with Tuberculosis: A Retrospective Matched Cohort Study* Objectives: Outcomes of COVID-19 in patients with tuberculosis (TB) are largely undescribed. This study evaluated demographic and clinical characteristics, illness severity, complications, and mortality of COVID-19 infection in patients with TB compared to a matched non-TB cohort. Methods: Retrospective cohort study (3/1/20-1/14/21) of in- and outpatients within a large United States health system with a positive COVID PCR and

	no TB compared to age/sex-matched (3:1) patients with a positive COVID PCR and new or previous diagnosis of TB. Results: A total 93 and 31 COVID-19 patients were included in the non-TB and TB matched cohorts. For TB patients, 32% of patients had active infection vs. 65% with latent infection. Most TB was pulmonary (55%), and most patients with TB had a history of past treatment (68%). For patients with TB vs. those without, hospitalization was required in 45% vs. 36% (p=0.34), ICU care in 16% vs. 8% (p=0.16), and mechanical ventilation in 13% vs. 3% (p=0.06), respectively. However, length-of-stay (5.0 vs. 6.1 days, p=0.97), in-hospital mortality (3.2% vs. 3.2%, p=1.00), and 30-day mortality (6.5% vs 4.3%, p=0.63) did not differ significantly between TB and non-TB groups. Conclusion: Despite patients with COVID-19 and TB being hospitalized, receiving ICU care, and requiring mechanical ventilation at higher rates, this did not correlate with higher mortality nor increased LOS. This finding is counter to the clinical presumption that TB infers worse outcomes in patients with COVID-19 and may help inform treatment and resource-limited discussions for this patient population.
Hussein Magale Derek Kamal Nellie Adams Dr. Jessica Park Dr. Elie Gertner Dr. Michael Schnaus	*A Retrospective Review: CT Pulmonary Angiogram Use in COVID-19* Pulmonary Embolism (PE) is recognized as a major thrombotic finding in COVID-19 patients. Diagnosing PE in the setting of COVID-19 infection can be challenging as the symptoms often overlap and no risk stratification tools has been validated in patients with COVID-19. While CT Pulmonary Angiogram (CTPA) remains the gold standard imaging test to diagnose PE, internal evaluation cautions CTPA overuse at our facilities to rule out PE in COVID-19 patients. Our single-center retrospective study aims to evaluate clinical predictors of PE in patients admitted with COVID-19 to guide clinicians on when and which patients to obtain CTPA. Of the 75 charts reviewed so far, we found that 9.3% of COVID-19 patients with CTPA were positive for PE which is consistent with prior estimates in the literature. We also noticed a statistically significant trend toward higher D-Dimer levels in CTPA-positive patients and more charts need to be abstracted for higher confidence. With ongoing final data abstraction and analysis, our findings will guide clinicians with clinical criteria to limit the overuse of CTPA as a screening test in COVID-19 patients.
Alexandria Roy Jill Killian Phillip Schulte Veronique Roger Dr. Shannon Dunlay	*Activities of Daily Living and Outcomes in Patients with Advanced Heart Failure* Background: Functional debility is associated with worse outcomes in the general heart failure population, but the prevalence of difficulty with activities of daily living and clinical significance once patients develop advanced heart failure requires further examination. Methods: This was a population-based, retrospective cohort study of Olmsted County, Minnesota adults with advanced heart failure from 2007-2018. Difficulty with 9 activities of daily living were assessed by questionnaire. Predictors of difficulty were assessed by a proportional odds model. Associations with risks of mortality and hospitalization were examined using Cox and Andersen-Gill models. Results: Among 765 patients with advanced heart failure, 565 (73.9%)

	reported difficulty with activities of daily living at diagnosis. Of those, 257 (45%) had moderate difficulty and 148 (26%) had severe difficulty. Independent predictors of difficulty included female sex (OR 1.72, 95% CI 1.27-2.38, p=0.001), older age (OR per 10-year increase 1.17, 95% CI 1.05-1.31, p=0.005), dementia (OR 1.85, 1.06-3.24, p=0.031), depression (OR 1.75, 1.28-2.40, p=0.001), and morbid obesity (OR 1.49, 1.04-2.13, p=0.031). Estimated 2-year mortality was 61.5%, 64.2%, and 67.6% in patients with no/minimal, moderate, and severe difficulty. The adjusted HR (95% CI) for death were 1.08 (0.90-1.28) and 1.17 (0.95-1.43) for moderate and severe difficulty versus no/minimal difficulty (p=0.33). There were no statistically significant associations of difficulty with activities of daily living and hospitalization risks. Conclusions: Most patients with advanced heart failure have difficulty completing activities of daily living and are at high risk of mortality regardless of impairment. Independent predictors of difficulty included female sex, older age, dementia, depression, and morbid obesity. Patients with advanced heart failure may benefit from holistic care incorporating support with activities of daily living.
Gurmandeep Singh Sandhu Dr. Jerry Ash Dr. Marie-Annick Clavel Dr. Philippe Pibarot Dr. Sue Duval Dr. Prabhjot Nijjar	*Performance of CT-based Aortic Valve Area for Assessment of Aortic Stenosis* Introduction: Up to 40% of patients with severe aortic stenosis (AS) have discordant or low-gradient AS raising uncertainty about the actual AS severity. Computed Tomography (CT) is guideline endorsed to aid in such cases, with aortic valve calcification (AVC) considered a robust arbiter of AS severity. The performance of different CT derived aortic valve areas (AVA) is less well studied. Methods: Consecutive adult patients with presumed moderate and severe AS based on echocardiography (AVAEcho <1.5 cm2) that underwent a cardiac CT were identified retrospectively from the TAVR CT Registry. AVA by direct CT planimetry (AVACT) and by a hybrid approach (AVAHybrid) in which annular area is traced by CT and velocities by echocardiography, were measured. Flow status was assessed based on stroke volume. Sex-specific AVC thresholds (≥1200 AU in women and ≥2000 AU in men) were applied to arbitrate severe or non-severe AS. Measures of diagnostic performance for thresholds of <1.0 cm2 and <1.2 cm2 for AVACT and AVAHybrid were calculated. Results: 215 patients (38.0% female, mean age 78 ± 8 years) were included, normal flow: 59.5% and low flow: 40.5%. Based on AVC, 78.6% had severe AS. Mean AVACT was 0.99 ± 0.27 cm2 and mean AVAHybrid was 1.17 ± 0.26 cm2. Diagnostic performance is listed in Table 1. AVACT cut-points of 1.52 cm2 for normal flow, and 1.56 cm2 for low flow, provided 95% specificity for excluding severe AS. Conclusion: In a cohort with mostly severe AS based on AVC thresholds, CT derived AVAs have poor discrimination for severe AS. AVACT is smaller than AVAHybrid. AVACT <1.2 cm2 threshold has better diagnostic performance for severe AS than AVACT <1.0 cm2 or AVAHybrid <1.2 cm2. Just like AVAEcho <1.0 cm2, AVACT <1.2 cm2 is a sensitive but a non-specific marker for severe AS. Higher AVACT thresholds improve specificity.

Fadumo Warsame Nadia M. Chu Jingyao Hong Deidra C. Crews Aarti Mathur George Bayliss Dorry L. Segev Dr. Mara A. McAdams-DeMarco	*Sleep Duration and Cognitive Dysfunction Among Older Adults with Chronic Kidney Disease* Background: Short and long sleep durations are associated with cognitive dysfunction. Given the increased prevalence of sleep abnormalities in the CKD population, we tested whether the association between sleep duration and cognitive function differed between older adults with and without CKD. Methods: This was a study of 3,215 older adults (aged≥60years) enrolled in the National Health and Nutrition Examination Survey (2011-2014) evaluating sleep duration, cognitive function (immediate recall, delayed recall, verbal fluency, executive function and processing speed, and global cognition), and kidney function. We quantified the association between sleep duration and cognitive function using linear regression and tested whether the associations differed among those with CKD and without using a Wald test for interaction. Results: Among 3,215 participants, 13.3% reported 2-5 hours of sleep per day, 75.2% reported 6-8 hours, and 11.5% reported ≥9 hours. Persons with CKD were more likely to sleep ≥9 hours (OR=1.73, 95% CI: 1.22-2.46). Among participants with CKD, those with sleep duration ≥9 hours demonstrated worse global cognitive function (p for interaction=0.01), immediate recall (p for interaction=0.01), and verbal fluency (p for interaction=0.004) than those with 6-8 hours sleep duration; no differences were observed for participants with CKD who slept 2-5 hours. Among participants without CKD, sleep was not associated with any measures of cognitive function. Conclusions: Longer sleep duration is associated with worse cognitive function only among persons with CKD and global cognition, delayed recall, and verbal fluency are particularly affected. Studies should identify interventions to improve sleep patterns and quality in this population. -- KEY LEARNING POINTS What is already known about this subject? • Sleep duration is an extensively studied component of sleep quality and has been found to have a U-shaped association with cognitive decline. Given that shorter and longer sleep durations are associated with cognitive dysfunction, we sought to explore whether this association differs among persons with CKD. What does this study add? • Long sleep duration is more common among persons with CKD. There were no significant associations between cognitive function and sleep duration among those without CKD. Among participants with CKD, those with long sleep duration demonstrated worse global cognitive function, immediate recall, and verbal fluency compared to those with shorter sleep duration. What impact may this study have on practice or policy? • Sleep duration may be a salient factor in the relationship between kidney function and cognitive decline. Primary care physicians and nephrologists should consider counseling patients with CKD about excessive sleep, and further studies should identify any underlying sleep disturbances in this patient population that may mediate cognitive decline.

Pitchaya Worapongsatitaya Dr. Thanat Chaikijurajai Dr. Jakrin Kewcharoen Dr. Ben Ponvilawan Dr. Nipith Charoengnam Dr. Patompong Ungprasert	*Hypochloremia and Mortality Risk in Patients with Acute Decompensated Heart Failure: A Systematic Review and Meta-Analysis* Background: Accumulating evidence suggests that hypochloremia is a predictor of mortality in patients with acute decompensated heart failure (ADHF), independently of traditional prognosticators such as serum sodium and natriuretic peptides. However, the definition of hypochloremia and statistical analyses were inconsistent across studies. Objective: The current systematic review and meta-analysis was conducted with the aim to summarize all the available data on hypochloremia and the risk of mortality in patients with ADHF. Methods: A systematic review was conducted using MEDLINE and EMBASE database from inception to January 2022 to identify all studies demonstrating the association between hypochloremia and mortality in patients with ADHF. Eligible studies must be cohort or cross-sectional studies that compared two groups of adult participants, one with hypochloremia (serum chloride ≤ 96 to 104 mmol/L or mEq/L) and one with normal level of serum chloride (serum chloride > 96 to 104 mmol/L or mEq/L) for mortality using a time-to-event analysis. We also included studies that analyzed serum chloride as a continuous value and reported hazard ratios (HRs) per unit or interval change of serum chloride for mortality. HRs with corresponding 95% confidence interval (CI) per unit increase of serum chloride were converted to HRs per unit decrease of serum chloride by inversion, whereas HRs per interval change were converted to HRs per unit change using the formula, HR per unit change = e^[(ln HR per interval change)/interval change]. Results: A total of 14 studies were eligible for the meta-analysis. There were 9 studies reporting HR per unit of interval change of serum chloride, 8 studies reporting HR comparing hypochloremia and normochloremia, and 3 studies reporting both. One study (Nakamura et al.) reported HRs separately for HF with reduced (LVEF < 40%), mildly-reduced (LVEF 40-59%), and preserved ejection fraction (LVEF ³ 50%). For each unit decrease of serum chloride was associated with a 7% increase in mortality risk (pooled HR 1.07, 95% CI 1.04 to 1.09, p = 0.007), and the presence of hypochloremia was associated with a 71% increase in mortality risk (pooled HR 1.71, 95% CI 1.41 to 2.09, p = 0.020). The statistical heterogeneity of these meta-analyses was high with the I2 of 62% and 54.4%, respectively. Conclusion: Hypochloremia on admission is significantly associated with an increased risk of mortality in patients with ADHF.
Clinical Vignette- Residents	
Mohamad Alabdaljabar Dr. Meltiady Issa	*The Heart is Innocent, Until it is Not!* Introduction: Infectious endocarditis (IE) is a serious condition that could result in dramatical consequences if misdiagnosed. Although it commonly affects patients with risk factors, it should be suspected and thoroughly investigated even in their absence if the clinical context is suggestive. Case Presentation: A 20-year-old healthy man presented to the ED with right arm pain and fever for 1 week. He also had pain in his right calf and left thigh, in addition to lower urinary tract symptoms. No recent travels, or illicit

	drug use. He is sexually active with one female partner. Few days earlier, he was seen in a local clinic and was given ceftriaxone and cephalexin course for possible right arm cellulitis and UTI. His symptoms progressed, so he presented to the ED for further evaluation. He had tachycardia with temperature of 39.9°C. Physical examination revealed erythema, swelling, and tenderness over the right upper arm with similar changes over the right anterior thigh and left posterior calf. Cardiac auscultation showed grade 3/6 systolic murmur. The rest of the physical exam was unremarkable. Labs showed leukocytosis, with high ESR, CRP, and CK. Urinalysis was positive for leukocyte esterase and WBC. Right upper extremity ultrasound showed signs suggestive of cellulitis and intramuscular micro-abscesses. Blood cultures were obtained, and he was admitted to the hospital. HIV was negative, with normal immunoglobulin levels. Further imaging showed abscesses in all three affected extremities and in the prostate. Transthoracic echo (TTE) was negative for vegetations. Ultrasound guided aspiration of his abscesses revealed pus that grew methicillin resistant staphylococcus aureus (MRSA). Blood and urine cultures continued to be negative, even after 5 days of incubation. Trans-esophageal echo (TEE) showed 4x2 mm soft echo density on the mitral valve with multiple strands and mild-moderate mitral regurgitation The patient was treated with IV vancomycin and improved significantly. Conclusion: Positive blood cultures is one of the major Duke criteria for IE diagnosis; however, its absence does not rule out the diagnosis. Our patient is young with no risk factors suggesting IE. His negative TTE and blood cultures only after cephalosporin administration further decreased the possibility of IE, especially after isolating MRSA from his abscesses. On the other hand, the multi-focal abscesses and heart auscultation were pointing towards one organ- the heart. In such cases, clinicians should have a low threshold in pursuing the more specific imaging modality, TEE, even in the absence of IE's risk factors. Diagnostic reasoning and thorough physical exam are essential in the early diagnosis and management of IE, which can improve outcomes and prevent life-threatening complications.
Nahdiya (Safia) Ali	*Spontaneous Splenic Rupture as the First Manifestation of Mantle Cell Lymphoma* Introduction: Mantle cell lymphoma (MCL) is a rare subtype of B-cell non-Hodgkin lymphomas (NHLs) usually identified by a translocation of the CCND1 gene and is characterized by reciprocal chromosomal translocation t(11;14)(q13:q32). MCL comprises around 5-10% of all non-Hodgkins lymphomas. Case Presentation: A 67-year-old male with no significant PMHx presents to the ED with SOB and hypoxia. In the ED, he was found to be tachypneic in the 40-50s, hypoxic, tachycardic and was subsequently emergently intubated for impending respiratory failure. On physical examination he was found to have protuberant abdomen with tenderness in the LUQ. eFAST exam was positive for free fluid in the abdomen. CT CAP showed shattered spleen equivalent of a grade IV laceration and diffuse abdominal lymphadenopathy. Prior to intubation the patient denied any recent trauma. Secondary to concern for active extravasation with intraperitoneal blood, trauma surgery was consulted, and the patient was taken to the OR for exploratory laparotomy and splenectomy. Given the CT findings of lymphadenopathy

	and patient denying recent trauma there was an overall suspicion for a possible hematologic malignancy. Post- operatively Hematology- oncology was consulted for further workup. Initial flow cytometry of the spleen and lymph-nodes were consistent with CD5 positive B-cell lymphoma of blastoid variant involving the spleen and perihilar lymph nodes. Fluorescent in-situ hybridization of the pathological specimen showed CCND1/IGH rearrangement which is a common finding in mantle cell lymphoma and del(17p) which is associated with adverse prognosis in mantle cell lymphoma patients. Flow cytometry of the bone marrow aspirate showed CD5-positive kappa-monotypic B-cell population, hypercellular bone marrow with 80-85% cellularity, and 50% replacement by mantle cell lymphoma. Diagnostic LP showed no CNS involvement with negative FISH for t(11;14). The patient was discharged with a plan for outpatient chemotherapy management where he completed treatment with rutiximab, dexamathasone, bendamustine as well as salvage chemotherapy with R-HDAP. Conclusion: Although splenic enlargement occurs in nearly 40 percent of patients with MCL, spontaneous splenic rupture is extremely rare. This case presents a rare phenomena of splenic rupture as the first manifestation in mantle cell lymphoma and illustrates that clinicians should be aware of the incidence and probability of spontaneous splenic rupture as the first manifestation of mantle cell lymphoma. Reference: Tan, Christopher, et al. Pathologic Rupture of the Spleen in Mantle-Cell-Type Non-Hodgkin's Lymphoma. NIH, Apr. 2012, https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3347250/.
Alexandra Allman Dr. Naima Hashi Dr. Neel Shah	*Epidural Abscesses are a Pain in the Neck* Introduction: Neck pain is a common complaint; the differential includes five main categories: mechanical, degenerative, malignancy, inflammatory, and infectious. Up to 75% of patients who initially present with spinal epidural abscesses are misdiagnosed. While uncommon, epidural abscesses must remain high on the differential due to serious complications from treatment delays. Case Description: 69 year old woman with past medical history of severe osteoarthritis and ESRD on HD, presented to the hospital with 1 week of malaise, and neck pain. On admission, labs were notable for WBC 15 K/uL, ESR 81 mm/h, and CRP 427 mg/L. On physical exam patient was unable to move her neck and her tunneled IJ was erythematous, warm, and tender. Patients' blood and IJ catheter cultures were positive for methicillin-susceptible Staph aureus (MSSA). The catheter line was removed, and oxacillin was started. CT of her cervical spine showed diffuse degenerative changes but was otherwise unremarkable. The patient continued to endorse severe neck pain with limited neck rotation, and intermittent low back pain. Her pain was thought to be musculoskeletal in nature, however she continued to require IV opioids. Despite appropriate antibiotic treatment, her MSSA bacteremia persisted, and her leukocytosis worsened. TEE for endocarditis was negative, CT Chest/Abdomen/Pelvis were negative for infection. MRI spine was obtained which demonstrated extensive infection including multiple epidural abscesses in the cervical, thoracic, and lumbar spine. After 10 days of oxacillin and drainage of the largest epidural abscess, her MSSA bacteremia cleared, and her neck pain and range of motion improved.

	Discussion: This case demonstrates the importance of keeping a broad differential for back and neck pain, especially in a patient who has a persistent infection. The pain was thought to be musculoskeletal, especially in a patient who is obese and minimally mobile with a known history of osteoarthritis. She continued to require multiple doses of IV and oral opioids to manage her pain which could have prompted earlier imaging since this is atypical of osteoarthritis. Unfortunately, the classic triad for epidural abscess only presents in as few as 8% of patients. This patient was immunocompromised (ESRD on dialysis) so she would be less likely to mount a fever. The patient was generally weak and minimally able to give full effort for neuro exam, however neurologic signs due to direct compression from the abscess and are often a late finding. Clinicians should be vigilant in imaging for epidural abscess even without the full triad of symptoms. CTs even with contrast have lower sensitivity than MRI for epidural abscess, the test of choice. In this patient with persistently positive blood cultures and leukocytosis, MRI spine could have been considered earlier, and epidural abscess should not be ruled out with a negative CT.
Talal Almasri	*Aortic Mural Thrombus Embolization as a Manifestation of Essential Thrombocythemia* Introduction: Essential thrombocythemia (ET) is a chronic myeloproliferative neoplasm that results from clonal proliferation of megakaryocytes within the bone marrow. This leads to elevated platelets count that can result in both hemorrhagic and thrombotic complications. While thrombosis is associated with ET, it usually occurs in veins and small to medium -sized arteries. However, large-vessel thrombosis is extremely rare. Herein, we present a case of aortic mural thrombus as a manifestation of ET. Case Presentation: 75-year-old male with insignificant prior medical history who presented with 3 day- history of left-sided abdominal pain and vomiting. Upon evaluation, patient was hypertensive (163/77), but otherwise vitally stable. Exam was notable for tenderness in the LUQ but abdomen was soft with no guarding. Initial labs showed Hg of 10.3, platelets of 650 and Cr 1.74. On imaging, patient was found to have a mural aortic thrombus. Also, multiple splenic infarcts and right kidney infarct were noted on imaging. Vascular surgery was consulted but recommend against surgery given benign abdominal exam and intact distal pulses. Medical management was pursued and patient was started on IV heparin. TTE showed EF of 70% and no intracardiac thrombus was noted. Given the unexplained thrombocytosis in the setting of splenic/renal infarcts, hematology team was consulted. Initial impression was that thrombocytosis is likely reactive in the settings of iron deficiency anemia and splenic infarcts, however, work up for hypercoagulable disorder was sent. Two days later, CTA was repeated and showed complete resolution of the aortic thrombus. Therefore, patient was discharged on DOAC as a bridge to warfarin with plan to follow up as an outpatient. Hypercoagulability workup was pending on discharge. Unfortunately, patient was re-admitted 3 days later with SMA embolism complicated by ischemic colitis. He underwent ileocecectomy with no complications. Subsequently, hypercoagulability work-up showed CALR+ and JAK-2 negative concerning for ET Diagnosis was confirmed with bone

	marrow biopsy which showed hyper-cellular marrow with megakaryocytic hyperplasia and minimal reticulin fibrosis. Patient was discharged on aspirin and hydroxyurea with follow up with an outpatient hematology follow up. Discussion: Aortic mural thrombi in a normal (non-aneurysmal or minimally atherosclerotic) vessels are extremely rare and could be associated with hyper-coagulable disorders, malignancies and myeloproliferative disorders. While small to medium-vessel thrombosis can be associated with ET, aortic thrombus is only described in few case reports. Therefore, there is no clear consensus whether medical management versus surgery is best in these cases. Since platelet aggregation is thought to be the main mechanism of thrombosis, medical management usually includes aspirin in addition to a cytoreductive agent. Early differentiating between ET and reactive thrombosis is crucial for providing appropriate management and preventing complications.
Momen Alsayed	*I Broke my Heart in Connecticut; A Case of Lyme Carditis with High Degree AV Block* Introduction: Lyme disease is the most common vector-borne disease in the US. It is caused by the bacterium Borrelia burgdorferi and rarely, Borrelia Mayonii which is transmitted to humans through black-legged ticks. In 2019 alone, there were 23,453 confirmed cases in the US. Lyme carditis remains an uncommon complication in the early disseminated stage of the disease. In this report, we hope to shine a light on the importance of cardiac work-up and possible intervention in this population. Case Presentation: A 41-year-old female with a past medical history of HTN, PCOS, saphenous vein thrombosis, and splenectomy post-MVA, presented to the clinic feeling fatigued for 1 month after a cabin trip to Connecticut. A week before the visit, she had low-grade fevers, a disseminated rash on her abdomen, and low blood pressure and heart rate readings. She denied palpitations, chest pain, shortness of breath, lightheadedness, or syncope. Her heart rate was 53 bpm, BP was normal. Her respiratory rate and oxygen saturations were normal. Erythema migrans was noted on the upper abdomen, otherwise, exam was largely unrevealing. EKG showed evidence of a high-degree AV block. Initial work-up was negative for COVID-19, electrolyte and hepatic derangements, and thyroid dysfunction. Her WBC count was 11.36 and neutrophils 7.99. During admission, serial EKGs revealed progression in the degree of her AV block to persistent high-degree AV block and evidence of intermittent ventricular escape rhythm. TTE showed borderline increased LV wall thickness and asynchronous septal-apical motion but was negative for structural, valvular, or wall motion abnormalities. Temporary cardiac pacing was indicated, and she was started on 3g IV ceftriaxone after a presumptive diagnosis of Lyme carditis was made. On day 3, Lyme IgG & IgM western blots were positive and the patient was continued on IV ceftriaxone. After 7 days of antibiotics, the pacemaker was removed after sustained improvement and resolution of high-degree AV block on repeat EKGs. She was switched to 100mg oral doxycycline BID on discharge for a total of 21 days of antibiotics. She was then seen by cardiology 1 day after completing her antibiotic course, was asymptomatic and her EKG was normal with complete resolution of AV block. Conclusion: This case highlights the importance of early recognition and

	management of Lyme disease to prevent cardiac complications such as acute AV nodal disorders which can rapidly evolve to a high degree AV block and lead to cardiac morbidity and mortality. Having a higher clinical suspicion for Lyme carditis, especially in patients with a travel history to endemic areas who present with bradycardia or AV block with or without cardiac symptoms is imperative. Early recognition allows for timely management which includes IV antibiotics and, in some cases, temporary cardiac pacing to prevent possible negative outcomes.
Zachary Anderson	*An Uncommon Connection: Retroperitoneal Pancreatic Pseudocyst with Fistulization to the Portal and Superior Mesenteric Veins* Introduction: Chronic alcohol use disorder is linked to pancreatitis and pseudocyst formation. Here we present an adult man with chronic pancreatitis, weight loss, and concerning features on abdominal imaging with an alternate, rare explanation for these findings. Case Description: A 50 year old man presented to the Emergency Department for failure to thrive and weight loss in the setting of alcohol use disorder, general anxiety disorder, and major depressive disorder. The patient also had complaints of epigastric discomfort, fatigue, and decreased appetite. Physical examination was notable for cachexia and mild abdominal tenderness in the epigastrium and right upper quadrant. Laboratory studies were remarkable for a hemoglobin 9.2 g/dL with an MCV of 82.1 fL and a lipase of 284 IU/L. Concerned for malignancy, CT of the abdomen and pelvis was obtained. There was diffuse portal venous thrombosis with extension into the superior mesenteric, inferior mesenteric, and proximal splenic veins with pronounced collateral circulation. Also seen was enhancement of the portal venous walls and an infiltrative appearance of mesenteric walls. We then obtained a magnetic resonance abdomen and pelvis study which revealed a necrotic fluid collection at the pancreatic head communicating with a retroperitoneal pseudocyst which itself was communicating to both the portal vein and superior mesenteric vein. Biopsy of the tissue was taken and consisted of benign pancreatic tissue, although carbohydrate 19-9 antigen was elevated at 43 U/mL. EUS and ERCP confirmed fistulization and thus sphincterotomy was performed with ventral pancreatic ductal stent placement to offload pressures and encourage drainage. The clinical course was complicated by development of ascites with pancreatic enzymes in the fluid. Later, treated for bacteremia secondary to bacterial peritonitis presumed from the ERCP. Further in the clinical course, he developed a right lower intraabdominal abscess requiring drain placement. On subsequent EUS procedures, there was improvement in pancreatic inflammation and fluid content, allowing for removal of the stent. The patient has since developed chronic abdominal pain and continues to be malnourished due to poor appetite. Discussion: Here is presented a patient with chronic pancreatitis in the setting of alcohol use disorder which led to pancreatic pseudocyst formation and subsequent structural changes. Pancreatic pseudocysts have been associated with appetite intolerance but rarely the phenomenon of both portal and superior mesenteric veins. No definitive treatment guidelines for this exist, and in our case, pancreatic stenting improved the inflammation and structural changes with resolution of elevated lipase. However, there was no

	improvement in abdominal pain or appetite and clinical course was complicated by ascites, bacterial peritonitis, bacteremia, and right lower abdominal abscess requiring drainage.
Claire Arnold Dr. Christopher Stephenson	*A Case for PCSK9 Inhibitors in a Patient with Familial Hypercholesterolemia* Case Description: A 34-year-old male presented to an outpatient clinic for lipid management. He had a history of CAD with a prior NSTEMI requiring stenting, former tobacco use and obesity. Family history was notable for hypercholesterolemia in his brother and father, but no premature history of ASCVD. His physical exam revealed arcus senilis and tendon xanthomata. Before his NSTEMI two years prior, his medical history was notable for hypercholesterolemia with an LDL of 397. After his NSTEMI and atorvastatin initiation, his LDL decreased to only 207. Given his extremely elevated LDL, early history of CAD, family history, and physical exam findings, he elected to undergo genetic testing of FH. A heterozygous pathogenic variant was identified in the LDLR gene consistent with FH. He was started on triple therapy with evolocumab, ezetimibe and atorvastatin. After starting triple therapy, his lipids improved with an LDL of 39. However, due to cost concerns, the patient discontinued evolocumab and his LDL subsequently increased to 147 on dual therapy ezetimibe and atorvastatin. Discussion: Familial hypercholesterolemia (FH) is the most prevalent monogenic, autosomal dominant genetic disease in humans. Previous studies have suggested that 1:217 people in the general population have mutations that cause FH. The most common genes involved include low-density lipoprotein receptor (LDLR) gene, proprotein convertase subtilisin kexin 9 (PCSK9) gene and apolipoprotein B gene, all which are crucial for low-density lipoprotein cholesterol (LDL-C) catabolism and result in higher LDL-C in the blood if mutated. The diagnosis of FH is established through genetic testing or clinical picture, which typically includes high LDL-C. Patients with an elevated LDL-C have the propensity to develop early ASCVD and intense LDL-C lowering in patients with FH leads to marked improvement in prognosis. Treatment typically begins with maximally tolerated statin therapy followed by ezetimibe and a PCSK9 inhibitor if LDL-C is not at goal. PCSK9 is a protease that prevents recycling of LDLR to the cell surface resulting in higher LDL-C in the blood, thus, inhibiting it leads to lower LDL-C. There is no consensus on LDL-C target in FH heterozygotes. However, a more aggressive target of an LDL-C <70, is typical in very high-risk FH individuals, especially those with prior ACS. Studies such as the FOURIER trial are thought to support the use of PCSK9 inhibitors in people with FH since they have a higher risk of cardiovascular disease events. However, there is a financial barrier with these medications. PCSK9 inhibitors might not be covered by insurance companies even in areas where they have received approval for use, or they might have high monthly copayments. This case highlights the challenges of pharmaceutical costs in the management of FH as well as how social determinants of health can impact an individual's care.
Danielle Bullard	*An Elusive Cause of Pericardial Effusion* Introduction: Pericardial effusion is common and has an extensive differential diagnosis. We present a case with an unusual and elusive cause.

	Case Description: A 74-year-old male presented with a two-week history of low-grade fevers and poor appetite with a 7-pound weight loss. He was tachycardic with mild anemia (hgb 12) and mild transaminitis. These findings were attributed to viral illness and treated with supportive care. Over the subsequent two weeks, his fatigue worsened, a cough developed and both his anemia (Hgb 10) and LFTs worsened (~200). Labs were notable for d-dimer 1.6, CRP 15.1, ferritin 1,852. There was no evidence for hemolysis or acute hepatitis, and ANA screen was negative. CT imaging confirmed no evidence for pulmonary embolism but demonstrated a diffusely thickened pericardium with trace pericardial effusion. The patient was admitted for further evaluation. Cardiac MRI confirmed pericardial thickening (7 mm) and a small pericardial effusion. Extensive infectious disease work up was negative. Rheumatologic labs including RF, anti-CCP, and ANCA returned without evidence of abnormality. He was discharged on colchicine. Two weeks later, he had lost an additional 5 pounds. Repeat labs showed CRP of 23, WBC 11, thrombocytosis to 553, hemoglobin decreased to 9. The etiology was theorized to be an autoimmune process with serositis. C3, C4, dsDNA, SM RNP, and IgG4 were unrevealing. Prednisone and hydroxychloroquine were initiated. Several weeks later, the patient developed atrial flutter with RVR. Repeat echocardiogram showed a large circumferential pericardial effusion with tamponade. He underwent pericardiocentesis with removal of 300 cc of hemorrhagic fluid. Ferritin returned further elevated at 4,159. Rheumatology was consulted and expressed suspicion for Adult-Onset Still's disease vs. Giant Cell Arteritis. He was discharged home with plans for temporal artery biopsy which later resulted negative. One month later, he was re-admitted with worsening dyspnea and new bilateral pitting edema. Repeat echo revealed development of constrictive pericarditis and resultant acute diastolic heart failure. Both ventricles were of small volume with ventricular interdependence and tethering of both the LV and RV lateral walls. Pericardiectomy was recommended. Pathology revealed sarcomatoid type malignant mesothelioma of the pericardium. Discussion: Primary pericardial mesothelioma is an extremely rare entity; there have been only 200 reported cases in the literature with only 25% of those diagnosed antemortem. Risk factors remain poorly understood but include tobacco use and history of chest radiation; no definitive link to asbestos exposure has been established. Unfortunately, overall prognosis is poor. This case illustrates the importance of maintaining a broad differential diagnosis in determining the etiology of a pericardial effusion. Analysis of the pericardial fluid and the pericardium itself should not be delayed in the setting of severe disease with an elusive etiology.
Jared Buschette Dr. Breanna Zarmbinski	*An Uncommon Cause of Ventricular Tachycardia* Introduction: Approximately one percent of all ambulatory visits and five percent of emergency department visits are for chest pain. Musculoskeletal and gastrointestinal etiologies are most common. However, it is important to rule out the life threatening diseases because of the mortality associated with acute coronary syndrome. Case Presentation: A 40-year-old female with a history of anxiety and tobacco use presented to urgent care with two weeks of tight substernal chest

	pain with activity and at rest without associated symptoms. Notably, family history was significant for early coronary artery disease in her mother and father. An ECG and troponin I were normal and a CT scan was negative for pulmonary embolism. Her primary care provider ordered a cardiac stress test at follow up. During her treadmill stress echocardiogram, the patient complained of "mild" sternal chest pain and palpitations and developed decreased blood pressure and several beats of nonsustained ventricular tachycardia. Echocardiogram revealed wall motion abnormalities in the inferolateral distribution. Exercise was stopped immediately, and the patient was transported to the emergency department for expedited work-up and management. A heparin drip was started as well as low dose aspirin and beta-blocker. Coronary angiogram demonstrated no significant coronary artery disease or evidence of spontaneous coronary artery dissection, and coronary anatomy was normal. A cardiac MRI was normal. A repeat exercise stress test was then stopped due to decreased blood pressure, shortness of breath, and EKG changes including ST elevation in leads V5 and V6 with ST depression in V1 through V3. With new concern for vasospastic angina, the beta-blocker was stopped, and a calcium channel blocker (CCB) was started. She then had a coronary angiogram with acetylcholine provocative challenge test. High dose intracoronary acetylcholine into the right coronary artery (RCA) resulted in significant coronary spasm with decrease coronary flow rate and associated ST-depression on ECG, confirming the diagnosis of vasospastic angina. The CCB was continued. Repeat exercise stress test several days later was normal. Conclusion: Vasospastic angina is defined by an international group of cardiologists (Coronary Vasomotor Disorders International Study Group) according to criteria including nitrate responsive angina, transient ischemic ECG changes, and evidence of coronary artery spasm in response to a provocative stimulus. Coronary vasospasm can cause complete or near-complete occlusion of a vessel which can present from stable angina to acute coronary syndrome. A non-insignificant proportion of patients with suspected coronary artery disease will undergo coronary angiography and have normal or nonobstructive coronary disease; approximately half of these cases will have coronary microvascular disease or coronary vasospasm. After diagnosis is made, the mainstay of treatment includes CCBs and sublingual nitroglycerin for acute symptoms. The overall prognosis of patients without underlying coronary artery disease is good with up to 90% or greater survival at five years.
Bradley Busebee	*71-Year-Old Man with Dyspnea on Exertion* Case Presentation: A 71-year-old male with history of liver transplantation in 2013 secondary to non-alcoholic fatty liver disease related cirrhosis with comorbidities of CKD stage 3, COPD maintained on tacrolimus presented to a community based internal medicine resident clinic with complaints of back discomfort and dyspnea on exertion. He reported insidious onset of above symptoms over the last 1 month. Pain was sharp and mid-scapular. He denied any fevers, cough, chest pain, or edema. Patient was observed to have heart rate of 110 ambulating in the clinic hallway though he maintained oxygen saturation greater than 94%. He reported dyspnea during this activity. He was recommended to present to the ED. In the emergency department troponins were within normal range and static, ECG demonstrated sinus tachycardia with low-voltage QRS. A chest CT demonstrated a large pericardial effusion without any evidence of

	tamponade. He was admitted to the hospital for further medical management. Upon admission he underwent a TTE guided pericardiocentesis with 700 ml of bloody drainage which was sent for cytology. This was negative for malignancy however was consistent with an acute inflammatory process based on nucleated cell count of 3879 and neutrophil predominance of 69%. A pigtail catheter was left in place and he received lidocaine infusions for pain management. Patient's pain and dyspnea resolved almost immediately. ESR was elevated to 130 and CRP was elevated to 46 and he was started on colchicine 0.3 mg twice daily. Nonsteroidal anti-inflammatory drug therapy was withheld due to chronic renal insufficiency. Based on absence of reaccumulation of pericardial fluid on repeat TTE, the pericardial drain was removed on day 3. Sedimentation rate had fallen to 28 and C-reactive protein to 43.1 within 1 week of treatment. The patient was evaluated by rheumatology. Workup was notable for rheumatoid factor of 78 but without any correlating synovial changes and degenerative disease on x-ray. Hepatitis serologies were negative. Repeat cardiac MRI shortly after discharge showed minimal pericardial effusion suggesting absence of significant ongoing inflammatory process. Patient did not have recurrence of symptoms and was able to return to prior activity level. Conclusion: Ultimately, the patient was diagnosed with idiopathic pericarditis. This can often be attributed to respiratory viruses among other etiologies. While there are case reports of pericarditis from tacrolimus, this usually occurs early on into therapy with that agent, such that it would be doubtful tacrolimus would be relevant to pericarditis 10 years into treatment. At most recent follow-up, this patient reported that he was feeling well. He is on colchicine still and therapy plan will be revisited after subsequent 4 month cardiac imaging.
Ahsan Butt Dr. Garrett Welle Dr. Madeline Mahowald Dr. Robert Frye	*Sinus Node Dysfunction secondary to Ophthalmic Timolol* Introduction: Sinus node dysfunction (SND) is a clinical syndrome with electrocardiogram findings of chronic sinoatrial node irregularity as well as associated clinical signs and symptoms. SND also manifests as chronotropic incompetence, resulting in inappropriate heart rate responses to physiologic demands during activity. Many clinicians diagnose chronotropic rate incompetence as the inability to achieve >80% of the maximum predicted heart rate with exercise. In patients with suspectded SND, a careful review for potentially reversible etiologies is warranted. If there is a concern for drug-induced bradycardia, the patient should remain on ECG monitoring while medications are withdrawn. If symptoms and ECG abnormalities persist despite withdrawal of medications, then SND can be diagnosed. This would be a Class 1 indication for permanent pacemaker implantation (PPM). This report presents a case of a patient with dyspnea on exertion and chronotropic incompetence who was found to have an often forgotten cause of AV node blockade, which highlights the need for clinicians to take a cautious approach to ruling out secondary causes of SND to prevent harm through the unneccessary PPM. Case Presentation: A 75-year-old male with history of open-angle glaucoma, on ophthalmic timolol, presented to clinic for evaluation of several years of dyspnea on exertion, with bradycardia and hypotension. His vital signs were notable for resting heart rates in the 40-60 range. He underwent cardiopulmonary exercise stress testing which showed exercise time of 6

	minutes by Mayo treadmill protocol, 4.8 METS (79% predicted), peak heart rate of 84 BPM (58% predicted), and peak systolic BP 134 mmHg. This was consistent with chronotropic incompetence with blunted blood pressure response and limited exercise capacity. There was suspicion timolol was causitive. He underwent laser trabeculoplasty with Ophthalmology and was weaned off timolol. On follow-up, repeat exercise stress testing showed 6.5 minutes by Mayo treadmill protocol peak heart rate 107 BPM (16% improvement). He noted complete resolution of cardiopulmonary symptoms following discontinuation of timolol. Follow-up one year later showed continued resolution of dyspnea. Discussion: In general, topical medications are often overlooked when evaluating for drug-induced systemic side-effects. As seen here ophthalmic timolol was the culprit drug. Interestingly, timolol drops notably avoid first pass hepatic metabolism through absorption via the lacrimal ducts into the venous circulation, which leads to increased systemic drug levels. This nuanced concept illustrates the importance of understanding the pharmacokinetics and side-effect profiles of medications. Furthermore, this case highlights the importance of a judicious approach in evaluation of a patient's pharmacologic history. In this case, appropriate recognition of drug side effects led to discontinuation of the offending medication and avoided unneccessary permanent pacemaker placement. .
Matteo Castrichini Dr. Matthew Samek Sarah Schrup Dr. Floranne Ernste	*Nonbacterial Thrombotic Endocarditis in anti TNF-alpha Induced Lupus with Antiphospholipid Antibody Syndrome. A Case Report* Background: drug induced lupus (DIL) encompasses a broad spectrum of manifestations, most frequently of the skin, but also systemic complex conditions, especially when induced antiphospholipid antibody syndrome is also present. A stepwise diagnostic strategy with a correct medical history and a thoughtful imaging evaluation is the cornerstone for the appropriate approach. Case Presentation: we report the case of a 45-year-old woman with past medical history notable for rheumatoid arthritis in infliximab, presenting at the emergency department for thalamic stroke with the evidence of a patent foramen ovale (PFO) at the transthoracic echo. At the transesophageal echocardiogram the absence of shunt at the PFO, and the evidence of mobile small vegetations of the aortic valve. The presence of positive antiphospholipid antibodies, a new skin rash together with the imaging findings, suggested a new diagnosis of DIL most likely due to infliximab, with antiphospholipid antibodies syndrome and nonbacterial thrombotic endocarditis, emphasizing the critical role of imaging and medical history in the differential diagnosis of source of embolism. Discussion: in the complex field of source of embolism a proper differential diagnosis is extremely important in order to start the appropriate treatment, however sometimes it could be extremely challenging especially in the contest of an underlying autoimmune disorders.
Maroun Chedid Dr. Sam Ives	*Retinal Artery Cholesterol Plaque of Unclear Origin Causing Recurrent Amaurosis Fugax* Introduction: Amaurosis fugax is a sudden painless loss of vision loss followed by spontaneous recovery. It is most commonly due to retinal

	ischemia following transient occlusion of a retinal artery by micro emboli, usually from an atherosclerotic plaque in the carotid artery. It is considered a transient ischemic attack. Case Presentation: A 69-year-old male with a mechanical aortic valve on warfarin, dyslipidaemia, and poorly controlled hypertension presented to the emergency department for two successive episodes of abrupt complete but reversible vision loss in his left eye. His vision loss was also associated with sensation of flashing lights in the same eye. Physical examination was remarkable for decreased acuity in his left eye, but no other deficits were seen. A presumable diagnosis of amaurosis fugax was made and the patient was admitted to the hospital for further workup to determine the origin of the amaurosis fugax. Brain MRI was negative for acute ischemia; however chronic small ischemic changes of the cerebral white matter were seen. There was no evidence of optic nerve inflammation or damage. CT angiography of the neck showed patent carotid arteries bilaterally with less than 10% obstruction in each artery. Head CT angiogram demonstrated no intracranial aneurysms or significant stenosis of the major intracranial arteries. Cardiac ultrasound was negative for ventricular thrombus and showed good functioning of the prosthetic valve without any marked valvular abnormalities. Ophthalmology was consulted, fundus imaging showed evidence of Hollenhorst plaque at a nasal bifurcation of the retinal artery with cotton wool spots on the retina suggestive of ischemia. The patient was started on high intensity statin therapy as well as aspirin and was recommended to follow up with his primary care physician for adequate blood pressure control, and with neurology and ophthalmology after discharge. Conclusion: Amaurosis Fugax is a type of transient ischemic attack that is equivocal to ischemic stroke. Although atherosclerotic disease of the carotid arteries is the most common aetiology, some patients have no clear source of embolism. A comprehensive and multidisciplinary approach is required for adequate diagnosis and prognostication. Even in the absence of a specific aetiology, amaurosis fugax should be treated with high urgency to prevent occurrence of permanent vision loss.
Deandra Chetram Dr. Kyla Lara-Breitinger Dr. Courtney Bennett	*Too Young to Have a Myocardial Infarction? Check a Lipoprotein (a) Level* Background: Lipoprotein(a) [Lp(a)] is a low-density lipoprotein (LDL) particle that includes apolipoprotein (a) attached to apolipoprotein B-100. Lp(a) is pro-atherogenic, pro-thrombotic, pro-inflammatory and an independent risk factor for coronary artery disease (CAD), ischemic stroke, and aortic valve stenosis. Lp(a) is present in approximately 20% of the population and is genetic. Case Presentation: A 42-year-old male with no cardiovascular risk factors presented to an Emergency Department due to crushing chest pain with exertion. Associated symptoms included diaphoresis, dyspnea, palpitations, and arm paresthesia. The week prior, he experienced similar chest pain when walking longer distances. He had a less than 2 pack year smoking history, smoked cigars occasionally, consumed alcohol socially and denied illicit drug use. His family history was notable for a brother with a possible myocardial infarction (MI) at the age of 42. His workup included an electrocardiogram that demonstrated an anterolateral ST-segment elevation (STEMI) in leads I, aVL, V2, and V3 and an initial

	troponin of 19. He was loaded with aspirin and clopidogrel, and was initiated on a heparin drip while being transferred to a percutaneous coronary intervention-capable facility. Emergent coronary angiogram was significant for 99% occlusion with intracoronary thrombus of the proximal left anterior descending artery for which he received a drug eluting stent. Multivessel disease was also present ranging from 10%-80% stenosis. Transthoracic echocardiogram demonstrated regional wall motion abnormalities with an ejection fraction of 60%. Further work-up revealed a normal hemoglobin A1c, normal thyroid stimulating hormone, and negative urine drug screen. A lipid panel revealed a total cholesterol of 163, triglyceride level of 117, LDL of 112, and a high-density lipoprotein (HDL) of 30 mg/dL. His Lp(a) level was elevated at 94 (reference range <75 nmol/L). In addition to dual antiplatelet therapy, he was initiated on lisinopril, metoprolol, and atorvastatin. Discussion: The patient's only risk factors for MI was his short smoking history, undiagnosed hypertension, and family history. The elevated lipoprotein (a) level is likely the most important independent risk factor for his premature CAD presenting as a STEMI. The National Lipid Association recommends (Class IIa) that it is reasonable to obtain an Lp(a) level in patients with a history of early ASVCD in a first-degree relative and premature ASCVD (particularly without traditional risk factors). This case highlights the need to test certain individuals for Lp(a) to guide treatment. The general goal in management of patients with an elevated Lp(a) level is strict LDL control, but statin agents typically have neutral to limited effect on lowering Lp(a). PCSK-9 inhibitors have shown significant reduction in the Lp(a) levels. Other agents are on the horizon, which have demonstrated promising results for reducing the risk of major cardiovascular events.
Megan Conlon	*Miliary Opacities: It's not always TB* Introduction: Miliary opacities on chest imaging refer to the presence of innumerable 1-4mm nodules scattered throughout the lungs. While tuberculosis should always be considered, the differential for miliary opacities is broad and includes other infectious etiologies (e.g., fungal infections such as histoplasmosis, cryptococcosis, coccidioidomycosis or paracoccidioidomycosis), as well as non-infectious causes including hypersensitivity pneumonitis, sarcoidosis, pneumoconiosis, or lymphangitic carcinomatosis. Case Description: A 78-year-old man from Argentina with a history of hypertension presented to the emergency room with six months of progressive weakness, malaise, and shortness of breath. On physical examination, he was afebrile with a pulse of 105bpm, BP of 122/75, and SpO2 of 88% on room air. Lab workup revealed a hemoglobin of 13.9 g/dL, platelets of 466 k/cmm, WBC of 14 k/cmm with neutrophilic predominance and eosinophilia to 0.84 k/cmm, and COVID-19 negative. CT chest pulmonary angiography demonstrated innumerable 2mm bilateral pulmonary nodules highly concerning for miliary tuberculosis. Upon admission, the patient had four negative AFB smears (and subsequently negative AFB cultures) followed by extensive rheumatology and infectious workups. Tissue obtained by FNA of the right hilar lymph node via bronchoscopic ultrasound was positive for CK7 and TTF-1 while negative for p40 supporting a diagnosis of primary adenocarcinoma of the lung. This case represents a rare presentation of primary pulmonary non-small cell

	adenocarcinoma. The patient's 21-day hospitalization was complicated by increasing oxygen requirements and fever. He ultimately passed away on comfort cares with rapidly progressive malignancy and superimposed pneumonia. Discussion: The finding of miliary opacities on chest imaging necessitates collection of a robust history including B symptoms, presence of chest pain or hemoptysis, country of origin, recent travel, history of incarceration, work exposures, hiking or cave exploring, pet exposures, and tobacco or other inhalant exposures. This case exhibits a rare presentation of primary pulmonary non-small cell adenocarcinoma which was initially highly concerning for tuberculosis. The patient's CT findings and his country of origin, Argentina, resulted in multiple teams anchoring on the diagnosis of tuberculosis to the extent that the patient received empiric TB treatment despite multiple negative AFBs and the presence of peripheral eosinophilia which is exceedingly rare in the setting of TB but well described in the setting of lung cancer. In advanced unresectable adenocarcinoma of the lung, tissue should be sent for PD-L1 testing which can predict favorable response to immunotherapies with tyrosine kinase inhibitors. Though patients with miliary non-small cell lung adenocarcinoma have an overall poor prognosis, treatment has been shown to prolong overall survival when initiated early. This case exhibits the importance of recognizing anchor bias which may cause a delay in diagnosis when early identification is key for treatment.
Sara Cuadra Aruguete	*A Non-healing Wound: Calciphylaxis in a Non-ESRD Patient* Introduction: Calciphylaxis is a cutaneous vascular disease defined by medial wall calcification and intimal hyperplasia.1 It is also known as calcific uremic arteriolopathy because it is largely seen in those with end stage renal disease (ESRD) undergoing dialysis. However, it has also been seen in those with normal kidney function with primary hyperparathyroidism, malignancy, alcoholic liver disease, hypercoagulable states, and warfarin and corticosteroid use.2 It can present with painful, violaceous cutaneous or subcutaneous lesions that can ulcerate and become necrotic with overlying eschar. The diagnosis can be confirmed with punch biopsy.3 Management consists of debridement, mitigating risk factors, and potentially sodium thiosulfate.4 Case Presentation: A 77-year-old male with past medical history significant for chronic left lower extremity wound, type II diabetes mellitus, hypertension, chronic kidney disease stage III, osteoporosis, deep venous thrombosis and pulmonary embolism presented with left lower extremity pain and was admitted with concern for cellulitis. The patient had an ulcerated area over his left lower extremity for the past three months that started increasing in size. Initial biopsy at that time was concerning for non-melanoma skin cancer and he was prescribed topical fluorouracil. He was later seen in dermatology clinic for worsening erythema and advised to present to the emergency department. He was prescribed oral dicloxacillin and directly discharged. The only other medication changes included recent warfarin discontinuation. After no improvement with antibiotics, he presented again to the emergency department. Vitals were within normal limits and physical exam was significant for distal left lower extremity ulcer with black eschar and

	erythema. Patient's labs were significant for WBC 13.04, CRP 104, and procalcitonin 0.11. Wound cultures grew Pasteurella, Pseudomonas, and Enterococcus. He was started on ceftriaxone without significant improvement. Infectious disease recommended considering alternative diagnoses to cellulitis. Dermatology was consulted for repeat biopsy and suspected an underlying vasculitis or calciphylaxis. An autoimmune and hypercoagulable work-up was conducted with unremarkable hepatitis B, hepatitis C, rheumatoid factor, cryoglobulins, SPEP, ANCA, prothrombin, protein C and S, antithrombin III, and lupus anticoagulant. Parathyroid hormone was slightly elevated and calcium was normal. Biopsy results were indicative of calciphylaxis. A malignancy work-up was initiated with CT of the chest, abdomen, and pelvis that demonstrated indeterminate renal lesions. Patient was administered one dose of sodium thiosulfate and was recommended to follow-up with dermatology and obtain additional imaging of renal lesions. Conclusion: This case demonstrates the importance of biopsy in non-healing wound. The diagnosis of calciphylaxis had significant therapeutic ramifications including avoiding hypercalcemia and hyperphosphatemia, normalizing parathyroid hormone, avoiding high risk medications such as warfarin, and trialing sodium thiosulfate.5 The diagnosis was also important for prognostication. Patient should be consulted on infection risk since calciphylaxis has an estimated mortality rate of 52-80% largely secondary to sepsis.2
Alex Danielson Dr. David Raslau	*A Case of Burkitt's Lymphoma Presenting as Cavernous Sinus Thrombosis* Introduction: A 75-year-old male presents with weakness and malaise after being recently diagnosed with a cavernous sinus thrombosis. Case presentation: A 75-year-old male presented to the emergency room with worsening generalized weakness, headache, and nausea. He had been diagnosed 2 weeks prior with a cavernous sinus thrombosis and was undergoing treatment with apixaban. On presentation, he was found to have right ptosis in addition to right cranial nerve 3 and 6 palsy. He was also noted to have an ulcerated mass on the inside of his mouth along the left lower gumline that was new on exam. Admission labs were notable for a 4 g hemoglobin drop over the span of 1 week. Admission CT head venogram showed redemonstration of the cavernous sinus thrombosis. Patient was started on a heparin drip and underwent an esophagogastroduodenoscopy to investigate his anemia in the context of recent anticoagulation initiation. His EGD showed several oozing ulcers that were biopsied. He also underwent a mandibular biopsy to investigate his new ulcerated mandibular mass. While waiting on the pathology reports, the patient started developing new cranial nerve impairment and ptosis of the left eye despite anticoagulation treatment. Magnetic resonance brain venogram was done given his worsening neurological symptoms. This revealed soft tissue masses around the cavernous sinus and no thrombus, which prompted discontinuation of his anticoagulation. Both the stomach ulcer and mandibular lesion biopsy reports later showed Burkitt's lymphoma, Epstein–Barr virus positive. Patient underwent a PET-CT which showed extensive bony and extramedullary disease. A lumbar puncture showed malignant cells in the CSF and bone marrow biopsy showed 90% marrow involvement. Patient was admitted to the hematology service to start chemotherapy. He underwent three cycles of dose-adjusted chemotherapy. At the end of cycle 3,

	his PET-CT showed treatment response and his CSF was negative for malignancy. He was then discharged with the plan to undergo outpatient chemotherapy. Discussion: Burkitt's lymphoma usually presents in the U.S. as the non-endemic sporadic clinical variant. This variant presents most often in adults as GI bleeding in addition to involvement of the jaw or facial bones, which correlates with this patient's clinical presentation. The patient's work up was complicated by his previous diagnosis of cavernous sinus thrombosis being treated with anticoagulation, which initially explained his neurological symptoms and anemia. His physical exam findings were important in leading to the correct diagnosis. The admission physical revealed his ulcerated mandibular mass, which prompted the initial concern for malignancy. His worsening neurological symptoms despite anticoagulation led to repeat imaging that showed metastatic disease instead of thrombosis. In the end, the patient's systemic symptoms, cranial nerve impairments, mandibular mass, and anemia were all attributed to a singular etiology of Burkitt's lymphoma.
Jose Francisco De Melo Junior Dr. Alicia Hinze	*An Ominous Diagnosis* Case Presentation: A 48 y.o. woman presented to the ED at an outside hospital in early August due fever, abdominal pain, diarrhea, facial rash, and arthralgias. Symptoms started approximately 7 days prior to presentation during a cruise ship to the Galapagos Islands. She had a history of triple positive antiphospholipid antibody syndrome (APLS) and a recent diagnosis of bilateral DVT of the lower extremities, on warfarin, and rosacea, on minocycline. She had transitioned to enoxaparin injections twice daily in preparation for her international travel. In the ED, she was febrile with T 100.6o, but hemodynamically stable. Laboratory was significant for hemoglobin 12.4g/dL, platelet count 47,000/uL, white blood cell count 12.8g/dL, AST 142U/L, ALT 166U/L, total bilirubin 1.0mg/dL, alkaline phosphatase 132U/L, creatinine 0.73 mg/dL, CK 75U/L, CRP 270mg/L, ESR 129mm/h, and urinalysis with 182 RBC/HPF and albumin of 300mg/dL. Peripheral smear showed no schistocytes and haptoglobin was elevated at 240mg/dL. Chest x-ray revealed bilateral opacities. An extensive infectious disease workup including but not limited to mononucleosis, viral hepatitis, HIV, Erhlichia, Babesia, Malaria, and Dengue fever were negative. Rheumatologic studies were consistent with negative ANA, normal C3 and C4, c-ANCA 1:160 with negative MPO and PR3 antibodies. Platelets trended down to a nadir of 19,000/uL that was unresponsive to IVIG. She was started on 60mg of prednisone for possible ANCA-associated glomerulonephritis prior to being transferred to Mayo Clinic. On presentation to this hospital, she had a malar and periorbital raised, erythematous to violaceous plaque and exquisite tenderness over the right sacro-iliac joint. MRI showed atypical signal foci in the iliac bones bilaterally adjacent to the SI joints compatible with developing infarcts. Lower extremity ultrasound showed extension of previously diagnosed DVT. Differential diagnosis included catastrophic APLS, acute cutaneous lupus, dermatomyositis, TTP and, less likely, ANCA vasculitis. She was started on high intensity intravenous heparin, methylprednisolone 1,000mg for 3 days, and plasma exchange therapy, followed by a slow prednisone taper and obinutuzumab, an anti CD-20 monoclonal antibody. Appropriate vaccination, PJP prophylaxis with pentamidine, GI prophylaxis with proton pump inhibitor, calcium and vitamin D supplementation were prescribed. She was transitioned to therapeutic warfarin with INR goal 2.5-3.5 prior to discharge

	in improved condition. Discussion: CAPS is an extremely rare condition and a life-threatening manifestation of APLS. It is the initial manifestation of half the diagnosis of APLS. It is characterized by widespread thrombosis that may lead to multi-organ dysfunction. The diagnostic criterion for CAPS includes the simultaneous development of three or more organ dysfunction in less than a week. Histopathologic confirmation of small vessel occlusion is necessary but often not feasible. Infections are the most common triggers, but withdrawal of anticoagulation or sub-therapeutic anticoagulation, as occurred to our patient, are also common precipitates. Treatment for APLS is not well defined but usually involves steroids, intravenous immunoglobulins or plasma exchange therapy, and a steroid-sparing agent, such as eculizumab or rituximab.
Elizabeth Doll Dr. Matthew Ho	*Recurrent Pneumaturia in a 58-Year-Old Man* Case Presentation: A 58-year-old man with a history of benign prostatic hypertrophy (BPH), erectile dysfunction, and previous Staph epidermidis UTIs, presented to clinic with recurrent urinary tract infections (UTIs). He reported four symptomatic UTIs in the past six months, with severe dysuria, frequency, and sensation of incomplete voiding. All were nitrite-positive with large leukocyte esterase, WBCs, and bacteria on urinalysis with microscopy, and Klebsiella pneumoniae with >100,000 CFU in urine cultures. Antibiotic courses included: Levofloxacin x7 days, Cefdinir BID x7 days, extended to 14 days due to persistent symptoms, and a second course of Cefdinir x7 days, all based on culture sensitivities. Severe dysuria and frequency would return within days to weeks after treatment completion. He also reported intermittent pneumaturia, not always associated with UTI episodes, and experienced gradual worsening of his obstructive symptoms despite increased tamsulosin. Eventually, this patient developed decreased semen output with ejaculation, mild perineal pain, and was found to have prostatic tenderness on examination, which finally led to the diagnosis of chronic bacterial prostatitis (CBP). An empiric 6-week course of Ciprofloxacin BID was initiated. However, he developed severe diarrhea (C. difficile negative) and full-body hives, requiring cessation of treatment. A post-prostatic massage urine culture obtained to inform subsequent antibiotic selection grew Staph epidermidis, which was suspected to be an additional contributor to his CBP based on its resistance profile. Trimethoprim-sulfamethoxazole BID was initiated to treat the Klebsiella, along with Daptomycin once daily to treat the Staph epidermidis, per recommendations from Infectious Disease. Urology saw the patient for management of his BPH, prostatitis, and assessment of pneumaturia during this time. CT urogram showed enlarged prostate with peripheral calcifications. There was a small air bubble at the bladder dome, but no air in the bladder wall and no enterovesical, colovesical, or rectovesical fistulas. Cystoscopy was normal aside from trilobar prostatic hyperplasia. Uroflow showed small postvoid residual volume with low Qmax. The pneumaturia was suspected to be secondary to gas production from Klebsiella, while his prostatic calcifications were thought to be a nidus for infection. Transurethral enucleation of the prostate is planned. Conclusion: This case illustrates the importance of considering CBP in patients with repeated UTIs from the same organism. It also highlights several risk factors for bacterial prostatitis--specifically BPH and prostatic

	calcifications--management of which may make collaboration with Urology an important part of treatment and source control. Further, this case is an interesting example of a commonly causative organism of CBP, Klebsiella, causing an uncommon symptom, pneumaturia. First ruling out fistulous connection to the GI tract, and emphysematous cystitis or prostatitis is essential. CBP requires 4-6 weeks of antibiotics with prostatic penetration, such as fluoroquinolones, trimethoprim-sulfamethoxazole, tetracyclines, or macrolides, tailored based on organism susceptibilities and patient allergies.
Michael Downey Dr. Kellen Albrecht Dr. Andrew Olson	*Heyde-ing in Plain Sight: Acquired von Willebrand Deficiency due to Aortic Stenosis* Case Presentation: A 75-year-old female was admitted after a visit with her primary care provider for melena, where she was found to be in early hemorrhagic shock while at her primary care provider's office. Her past medical history was significant for moderate aortic stenosis. Investigation during her hospitalization revealed angiodysplastic vessels in her duodenum (responsive to endoscopic intervention) as well as progression of the gradient across her aortic valve without other identified underlying risk factors for hemorrhage nor vascular malformations. Following endoscopic control of her hemorrhagic lesions, the patient had a robust improvement in clinical status, and was able to be discharged with gastrointestinal and structural cardiology follow-up. This case raises the question of Heyde Sydrome (HS) – a previously described entity composed of aortic stenosis, gastrointestinal vascular malformations, and an acquired (consumptive) von Willebrand factor deficiency. Initially described in 1958 as letter from Dr. Heyde reporting their clinical observation between aortic stenosis and the occurrence of massive gastrointestinal hemorrhage (GIH) in 10 patients, HS gained recognition as having a pathophysiological underpinning once surgical aortic valve replacement was found to reduce the recurrence of GIH in these same patients (despite these patients having been started on anticoagulation after valve replacement surgery). Contemporary mechanistic investigations into HS suggest that excessive shear forces across the aortic valve cleave high molecular weight multimers of von Willebrand Factor (vWF) with a resultant acquired vWF deficiency. In addition to the increased risk of bleeding from mucosal surfaces seen in most vWF deficient and defective states, the selective loss of the high molecular weight vWF in HS appears to promote dysregulated angiogenesis –most notable in the gastrointestinal tract. Conclusion: Besides pathophysiologically bridging three distinct organ systems (cardiology, gastroenterology, and hematology), and thereby establishing itself firmly within the internists' purview, HS is not isolated to aortic stenosis – and can occur in any state wherein the circulating blood volume encounters high shear stress: e.g. hypertrophic cardiomyopathy, left ventricular assist devices (LVADs), extracorporeal membrane oxygenation, and others. As LVADs become increasing more prevalent in the population at large, extra vigilance will be warranted for HS on every practitioner's differential of GIH.
Mitchell Dumais Dr. Carmen Montagnon Dr. Emma Johnson	*The Tell-Tale Blisters: A Case of Bullous Pemphigoid* Introduction: Bullous Pemphigoid is a rare autoimmune mucocutaneous blistering disease classically characterized by tense, fluid-filled bullae, erythema, and pruritus. Bullae are typically present in the axillae, trunk, or

	flexor surfaces of the arms. Diagnosis of bullous pemphigoid requires a compelling clinical scenario and is definitively made by diagnostic histology of a representative lesion. Skin biopsy sent for routine processing should reveal a subepidermal split with an eosinophil rich inflammatory infiltrate, with biopsy sent for direct immunofluorescence revealing linear C3 and IgG deposition along the epidermal basement membrane zone. In this case, we will describe a classic case of bullous pemphigoid, where a small cluster of lesions later progressed to bilateral extremity blistering. Case Presentation: An 84-year-old woman presented to the clinic with two days of worsening bilateral upper and lower extremity rashes, characterized by intense pruritis and blisters. Two months earlier, the patient was seen for a cluster of small fluid-filled blisters near her umbilicus, which resolved after topical application of hydrocortisone 2.5% cream. She denied any other systemic symptoms such as fevers or fatigue, mucosal pain or bleeding, or ocular symptoms. On exam, the patient had tense fluid-filled blisters on an erythematous base involving the bilateral forearms and anterior thighs. There was no visible mucosal involvement in the oral cavity. She was prescribed topical clobetasol 0.5% ointment which provided minimal relief and was seen in Dermatology Clinic. A shave biopsy was obtained from perilesional skin. Dermatopathology revealed eosinophilic spongiosis with subepidermal separation and abundant eosinophilic inflammation. Direct immunofluorescence showed continuous strong linear deposition of IgG, IgG4, and C3 along the basement membrane zone, consistent with subepidermal autoimmune mucocutaneous blistering disorders. Primate Split Skin IgG antibodies were positive in an epidermal pattern, with a positive anti-BP 180 but negative anti-BP 230 with ELISA. The patient was advised to continue topical clobetasol, with the addition of prednisone 30 mg daily, doxycycline 100 mg twice daily, and niacinamide 500 mg twice daily for 4 weeks. After 2 weeks, the patient reported healing of her blistering lesions and no new blisters forming. Discussion: Identifying bullous pemphigoid requires both a pertinent clinical history and objective findings via direct immunofluorescence to confirm the diagnosis. Additional laboratory evaluation for serum autoantibodies anti-BP180 and anti-BP230 can be used to help diagnose and stratify disease activity of bullous pemphigoid but are not required. Though bullae are classic on presentation, many patients present without bullae due to the waxing and waning nature of the disease, which can delay diagnosis. Due to this phenomenon, our patient's diagnosis was delayed 8 months. For patients with an evolving rash and any history of blistering, bullous pemphigoid or other mucocutaneous blistering diseases should be on the differential and biopsy should be considered.
Mohamed Elhadi Dr. John Bundrick	*Clinicians and Cognitive Bias: A Case of COVID-19 related Transverse Myelitis Misdiagnosed as Functional Disorder* Introduction: Physicians are subject to cognitive biases that can render diagnosis and treatment decisions vulnerable to error. Anchoring bias is locking on to a diagnosis too early and failing to adjust to new information. Diagnostic momentum is accepting a previous diagnosis without sufficient skepticism. Here in we present a case of COVID related transverse myelitis where, the aforementioned biases have led to delay in diagnosis and treatment. Case presentation: A 19 year-old man presents to the ER with a 2 day history

	of lower abdominal pain and bilateral lower extremity weakness, pain and numbness. Initial neurological exam revealed intact strength but did reveal reduced perianal sensation with normal anal tone. The patient was COVID-19 positive without respiratory symptoms and a CT of the abdomen ruled out intra-abdominal pathology. The patient was discharged and subsequently returned to the ER with the same symptoms as well as acute urinary retention. Neurology was consulted and on repeat examination the patient was found to have 4/5 strength in the lower extremities. He was found to have significant social stressors with the recent death of his father and cousin. He received a diagnosis of functional disorder and was recommended to be discharged with outpatient follow up. His lower extremity weakness continued to worsen, and the patient was reassessed by a different neurologist who recommended an MRI of the lumbar spine which was normal. The second neurologist agreed with the diagnosis of functional disorder. The patient was then admitted to the medicine team due to inability to walk and persistent urinary retention. On day 2 of admission and on reassessment by a third neurologist, the patient was found to have a sensory level at T10 and a pattern of weakness (even if subjective, as there was substantial functional overlay) suggesting myelopathy (flexors more impaired than extensors). An MRI of the cervical and thoracic spine revealed signal abnormalities involving T9 to T12 segments of the cord indicating acute transverse myelitis. The patient was commenced on high dose IV Methylprednisone for 5 days. The patient improved symptomatically and was discharged to rehab where he continued to improve. Discussion: This case highlights how anchoring and diagnostic momentum bias can lead to a delay in reaching the correct diagnosis. The presence of social stressors in the history and the false reassurance provided by the neurology consult and the MRI lumbar spine in the ER lead to the diagnostic label of functional disorder without complete rule out of other potential causes. These biases can make it difficult for the internist to remove the diagnostic label and interpret symptoms and signs with fresh eyes. Physicians should be aware of cognitive biases and their potential impact on the care of their patients.
Megan Ellis Dr. Nayla Ahmed	*Drug Induced Pneumonitis and Acalabrutinib, An Uncommon Adverse Reaction* Introduction: Drug induced pneumonitis (DIP) is an adverse reaction (AR) with a variable course, ranging from remission to respiratory failure requiring mechanical ventilation[3]. DIP is associated with medications like Amiodarone and Nitrofurantoin, but is a rare AR of certain anti-neoplastic drugs like Ibrutinib. Unlike the first approved BTK inhibitor (BTKi), Ibrutinib, Acalabrutinib is a next-generation highly selective BTKi studied to have increased tolerability and decreased ARs[1, 2]. Our case describes a rare incidence of DIP with Acalabrutinib. Case Presentation: A 73-year-old male from Northern Minnesota with recent diagnosis of chronic lymphocytic leukemia (CLL) presented to a tertiary care hospital with 2 months of progressive dyspnea and chronic cough. He noted progressive cough productive of gray/white sputum when in the lateral recumbent position, with associated dyspnea limiting activities, fever to 101F, anorexia, and 30lb weight loss. He reported travel to Alaska but no sick contacts. He had received 1 dose Rituximab 3 weeks prior to onset of cough and started Acalabrutinib two weeks prior to cough.

	Upon examination the patient was afebrile, tachypneic with mild exertion requiring 2L nasal cannula (baseline room air) and speaking in short sentences due to coughing bouts. Physical exam pertinent for fine crackles bilateral lower lung fields without associated wheezing or crackles. Laboratory evaluation pertinent for WBC 20.6 x 109/L (baseline 35 x 109/L, with 3.6 neutrophilic predominance), normocytic anemia Hgb 8.8 (baseline 11g/dL), and hyponatremia 133mEq/L (baseline 135mEq/L). Chest x-ray showed increased perihilar interstitial opacities bilaterally most notable in the lower lungs. A broad infectious workup with sputum testing and bronchoscopy with bronchoalveolar lavage was unrevealing. Bronchoscopy only pertinent for vocal cord edema. Given negative infectious workup, Acalabrutinib was discontinued and oral corticosteroids were started due to concern for grade 3 DIP. Over the next few days his symptoms and oxygenation improved. Discussion: DIP can be challenging to diagnose due to vague presentations. The complexity increases in immunosuppressed patients on antineoplastic agents as their nonspecific symptoms and imaging studies could be multifactorial, often delaying diagnosis[4]. Our patient developed a progressive cough shortly after initiation of Acalabrutinib which appeared to be grade 3 pneumonitis. This appears to be a very rare AR of this medication, as review of a pooled assessment of safety for Acalabrutinib, based on 9 randomized clinical trials, indicated the incidence of grade 3 or higher pneumonitis was 0.3% and the incidence of pneumonitis of any grade was 1%[2]. Patients with B-cell malignancies are often candidates for therapy with BTKi such as Acalabrutinib. However, it is important to have a high index of suspicion for ARs that require change in therapy. Factors holding patients back from receiving effective doses and treatment maintenance include drug toxicity and tolerability, as was seen in this case[1].
Tesfatsiyon Ergando Dr. Bellony Nzemenoh Dr. Abdisamad Ibrahim	*A Rare Coincidence, Causation or Correlation? Pseudoaneurysm & Pulmonary Embolism* Case Presentation: A 75-yo, M, hx AF and provoked b/l PE on apixaban presented to the ER following 3 pre-syncopal episodes and acute sharp, central abdominal pain. VS; HR- 110, BP- 74/49 , hemoglobin of 11 g/dl. He was given 3 liters of crystalloid and one unit of PRBC. CT of the abdomen showed a retroperitoneal hemorrhage and hemoperitoneum. General surgery and IR were consulted and deferred surgical intervention given no overt site of extravasation. The following day, Hb dropped from 11 to 6 and the Pt unstable with HR in 120s-140s, and BP 70/50, pt was transferred to ICU. Days following this, he developed dyspnea (needing 2 L supplemental oxygen), tachycardia (150s), and tachypnea (26) with asymmetric LE edema. U/S confirmed a DVT, extending from the L femoral vein to the level of the ankle. CT showed a sub massive PE and signs of right heart strain. Ultimately, a CTA showed a 1.4cm x 0.9 cm gastroduodenal artery pseudoaneurysm (GDA), which was identified as the source of the bleeding. IR placed two coils in the GDS and IVC filter. He was started a heparin drip after the coiling due to concern for rebleeding from complications surrounding the aneurysm. After stabilization, anticoagulation was changed

	to apixaban 10 mg orally twice a day for 7 days, then to 5 mg orally twice a day.
Emily Ewan Dr. Demetrios Andrisani Dr. Chrisanne TimpeDupuis	*Heart Failure at Home: Safe and Effective Management of an Acute Heart Failure Exacerbation in a Home-Based Acute Care Setting* Introduction: Heart failure is the leading cause of hospitalization in adults over the age of 65, and hospitalizations account for more than 70% of the annual cost of heart failure care. Particularly in the first few months after diagnosis, patients and their families struggle to understand and cope with the complexities of the disease, leading to dietary indiscretions, medication non-adherence, and confusion that contributes to high readmission rates. A recent trend to diurese patients to dry weight in the hospital has contributed to prolonged lengths of stay, further exacerbating hospital capacity issues and frustrating patients. The HealthPartners Hospital@Home (H@H) program aims to identify these patients early in their treatment course and complete their acute care in the home, where social determinants can be incorporated into the plan of care. Care Presentation: Here we present the case of a 63-year-old man with a past medical history of coronary artery disease, aortic insufficiency, chronic kidney disease, and recently diagnosed ischemic cardiomyopathy with an ejection fraction of 45% who was admitted to the hospital with acute kidney injury, dizziness, and falls in setting of medication titration. This was his third hospitalization in a span of 4 months since his initial diagnosis of heart failure. On admission, he was noted to be orthostatic and bradycardic, was seen by cardiology who felt chronotropic incompetence could be contributing, and his beta blocker dose was reduced with improvement in symptoms. As he was 20 pounds over his dry weight with increasing peripheral edema, he was started on IV diuresis for an acute heart failure exacerbation. His creatinine and electrolytes remained stable and he was transitioned to home with the H@H program to continue diuresis to dry weight. Through H@H, he had twice daily visits from a community paramedic (CP) who collected vitals (including intake/output) and blood work to monitor kidney function and electrolytes, and administered IV furosemide. The patient additionally had daily virtual visits with a hospitalist who added metolazone to augment diuresis as he remained >10 pounds above his dry weight on H@H day 3. Throughout the H@H course, patient and his wife received regular education and assessment of adherence to dietary restrictions by CPs. His weight did fluctuate, but with close monitoring and reinforcement, the patient was able to appreciate how changes in fluid and salt intake affected his weight and activity tolerance. On H@H day 8, lab monitoring revealed increasing creatinine, and diuretics were held over the subsequent days with improvement in kidney function. Oral diuretics were safely restarted on H@H day 15, and the patient was ultimately discharged from H@H on day 17 a few pounds above his dry weight, saving him over 2 weeks of time in the hospital. References: [1]Nieminen MS, et al. Am J Cardiol. 2005;96(6A):5G-10G. [2]Go AS, et al. Circulation. 2013;127(1):e6-e245.
Matthew Ewy Dr. Resham Ramkissoon Dr. Sunanda Kane	*Small Bowel Carcinoid Preceding Development of Ulcerative Colitis: A Rare Sequence of Events* Introduction: Inflammatory bowel disease (IBD) is associated with an

	increased incidence of bowel carcinoma and rare types of cancer, such as carcinoid tumors, are also more prevalent in patients with IBD. The relationship between carcinoid tumors and IBD is not well understood but is hypothesized to be related to chronic mucosal inflammation. However, not all cases of carcinoid tumors are located at the anatomical sites of active inflammation. It is unclear whether such an association is coincidental due to increased incidental findings during endoscopy and surgery or if common pathogenesis exists. Carcinoid tumors of the small intestine preceding the development of Ulcerative Colitis (UC) are particularly rare. Case Presentation: A 58-year-old female with a history of tubular adenomas underwent a surveillance colonoscopy to discover a three-millimeter semi-pedunculated polyp located ten centimeters from the ileocecal valve in the terminal ileum. After polypectomy, histology revealed evidence of a carcinoid tumor. A follow-up CT scan one month later revealed a 2.2 cm enhancing polypoid mass in the distal ileum and enlarged mesenteric lymph nodes. An ileocolectomy and elective cholecystectomy were performed, with pathology revealing a 2.2 cm, well-differentiated neuroendocrine tumor with five positive mesenteric lymph nodes. PET-CT scan did not show evidence of metastatic disease. The final oncologic stage was Stage III (pT3pN1cM0). Following her surgical resection, the patient developed chronic diarrhea for one year. She was treated with cholestyramine for suspicion of bile acid diarrhea due to terminal ileum resection and previous cholecystectomy. After worsening abdominal pain, hematochezia, and fatigue, a CT scan showed pancolitis with mesenteric lymph node enlargement. Subsequent sigmoidoscopy revealed diffuse colonic inflammation with histology demonstrating moderate chronic colitis without granulomas or dysplasia. Infectious causes of colitis were excluded. The patient was diagnosed with UC and started on mesalamine. She was eventually placed on budesonide for persistent symptoms but could not achieve remission. After carefully considering her carcinoid history and staging, vedolizumab was initiated for its gut specificity, and the patient had resolution of her inflammation. Conclusion: Compared to the general population, there is an estimated 7-fold increase in carcinoid tumors in patients with IBD, with most cases discovered incidentally after surgical resection. However, tumor location does not always correlate with local inflammatory effects, as reported cases document carcinoid tumors outside areas with active inflammation. These cases suggest that the systemic effects of pro-inflammatory cytokines may be involved in the pathogenesis of a carcinoid tumor development. A systemic pro-inflammatory state may well have existed, and the stress of surgery triggered an inflammatory reaction promoting mucosal inflammation and UC development in this patient. While de novo IBD development cannot be excluded, this case represents an unusual presentation of a small intestinal carcinoid tumor before the development of UC.
Katarina Fabre Dr. Alan Hu Dr. Anna Svatikova	*Endocarditis and Septic Emboli, The Feared Risk of Anticoagulation* Introduction: Infective Endocarditis is a disease with high morbidity and mortality, which can often present with nonspecific symptoms. Delay in diagnosis can have profound long-term effects, including valvular damage, embolic phenomena, and heart failure, particularly in patients with prosthetic valves and intracardiac devices. [1] Case Presentation: A 70-year-old male presented with fevers, chills, nausea,

	vomiting and dyspnea 1 day after cardioversion for atrial fibrillation. Medical comorbidities included atrial fibrillation (on warfarin), heart failure with mid-range ejection fraction of 45%, hypertension, severe mitral regurgitation, moderate-severe tricuspid regurgitation, coronary artery disease with history of STEMI (ST Elevation Myocardial Infarction) status post drug eluding stent to the left main coronary artery. His surgical history included coronary artery bypass grafting to the right coronary artery, prior aortic valve replacement with On-X mechanical valve and composite root with ascending aortic graft. 24 hours post cardioversion, the patient appeared febrile, diaphoretic with nausea and vomiting. His exam was unremarkable outside of a baseline murmur and normal neurological exam. Laboratory evaluation yielded: WBC 17.6 x10 9 /L (normal 3.4 to 9.6 ×109/L), lactic acidosis 12.9 mm/L (normal 0.5-2.2 mmol/L). Blood cultures were positive for methicillin sensitive staphylococcal aureus, and he underwent transesophageal echocardiogram which revealed mechanical aortic valve and tricuspid valve vegetations, suggestive of endocarditis. He was initiated on oxacillin. Despite his diagnosis of endocarditis, anticoagulation was carefully continued given recent cardioversion and normal neurological exam. An MRI (Magnetic Resonance Imaging) brain was obtained upon transfer to assess for evidence of septic emboli which noted multiple areas of septic emboli and the patient's anticoagulation was discontinued. Discussion: This patient's indication for anticoagulation was recent cardioversion and mechanical valve. In the acute period after cardioversion, there is an increased risk of thromboembolic events (up to 7%). This increased risk has been attributed to atrial stunning, which is transient dysfunction of the left atrium and left atrial appendage [5]. In a patient with newly diagnosed endocarditis and a clear indication for anticoagulation, this becomes a complex risk-benefit medical decision. In one large multicenter retrospective analysis, anticoagulant therapy at infective endocarditis diagnosis had a hazard ratio of 1.31 for development of neurological complications, and hazard ratio of 2.71 for cerebral hemorrhage. [4]. In this case, given the presence of multiple septic emboli, anticoagulation was discontinued. There is now some evidence that discontinuation of anticoagulation after 10-14 days could be possible and safe, but it is an area needing further investigation [2]. This case illustrates a difficult scenario of weighing the risk and benefit of anticoagulation with clear indications post cardioversion, mechanical aortic valve, atrial fibrillation against newly diagnosed endocarditis. Anticoagulation in this setting is controversial, particularly in patients who with pre-existing indication for it.
Kyle Farrell Dr. Savannah Liddell Dr. Bradley Salonen	*Transient Global Amnesia: Have I told you this before?* Introduction: Transient Global Amnesia (TGA) is a clinical syndrome characterized by the acute onset of self limited anterograde amnesia that resolves within 24 hours.1 During the episode, cognitive function is otherwise intact. Currently, there is no consensus for a common underlying pathophysiology of TGA. Several mechanisms have been proposed and supported through epidemiologic studies including vascular, epileptic, migrainous, and psychogenic.1 Below is a case of patient with TGA due to arterial ischemia. Case Presentation: A 61-year-old male with a past medical history of

	hypertension and hyperlipidemia presented to the Emergency Department (ED) with the chief complaint of altered mental status ongoing for the past six hours. Memory deficits included inability to recall the events of the past 48 hours. No systemic symptoms or history of head trauma was present. His medications included Lisinopril 20 mg daily and Atorvastatin 40 mg daily. On physical exam, the patient was hypertensive to 156/79, but otherwise, vital signs were within normal limits. Mental status exam showed the patient was alert and oriented to person and place but not to time or events in the last 48 hours only. Language, comprehension, and speech were normal as well as his knowledge of personal identity. His neurologic exam was non-focal. Workup in the ED was unremarkable including CBC, BMP, PT/INR, Troponin, TSH, VBG, Acetaminophen & Salicylate levels, ethanol level, ECG, CT head, chest x-ray. The patient was admitted to the hospital for further observation and management. EEG did not show any epileptiform discharges. MRI brain with and without contrast demonstrated a 3 mm focus of restricted diffusion within the body of the left hippocampal formation consistent with small acute infarction. He was started on Aspirin 81 mg and Clopidogrel 75 mg daily for twenty-one days followed by Aspirin 325 mg daily thereafter. He subsequently underwent CTA and head neck which was unremarkable and TEE which demonstrated a left ventricular ejection fraction of 60%, atrial septal aneurysm without evidence of a PFO, and trileaflet aortic valve. Over the course of twenty-four hours his symptoms gradually resolved and his antegrade memory function was intact. He continued to have retrograde amnesia to the events of the morning of his initial presentation. He was discharged in stable condition with a 30-day Holter monitor and outpatient follow-up. Conclusion: This is an interesting case of a patient presenting with altered mental status found to have TGA due to a hippocampal infarct. There is no specific treatment for TGA; however, due to this patient suffering from ischemic stroke, he was started on appropriate anti-platelet treatment. This case demonstrates the importance of neuroimaging to assist with delineating the etiology of TGA. For this patient, it was particularly important to optimize his health with appropriate treatment and mitigate the risk of recurrent strokes. 1. Arena JE, Rabinstein AA. Transient global amnesia. Mayo Clin Proc. 2015 Feb;90(2):264-72. doi: 10.1016/j.mayocp.2014.12.001. PMID: 25659242.
Bradley Fredrickson	*IgG4 Disease, Not Just Autoimmune Pancreatitis Anymore* Introduction: IgG4-related disease (IgG4-RD) is now understood to encompass 14 previously named diseases, including the most common form of autoimmune pancreatitis (1). It often presents as a tumefactive or fibrotic process and can involve almost any organ system, most commonly the pancreas, salivary glands, lacrimal glands, and retroperitoneum (1, 2). IgG4-RD often mimics malignant, infectious, or other autoimmune diseases, and diagnosis typically depends on biopsy, as serum IgG4 testing has a poor positive predictive value (34%) (2,3). Case Presentation: A 28-year-old man with no relevant past medical history presented to the emergency department because of 8 months of left eye symptoms. He endorsed a sensation of fullness in that eye, reduced visual acuity in that eye, and left-sided maxillary dental pain. He had previously

	presented several times to emergency rooms, a dentist, and an ophthalmologist that collectively resulted in several failed treatments including antibiotic ointments, oral antibiotics, and tooth extraction. On this presentation, exam revealed unilateral left eye proptosis. The remainder of the physical exam and basic laboratory workup was unremarkable. MRI revealed a diffusely infiltrating mass involving the left orbit. Ophthalmology and ENT were consulted and felt the mass was most concerning for malignancy or infection and brought the patient for nasopharyngoscopy and needle biopsy. The sample was non-diagnostic so the decision was made to perform surgical biopsy via lateral orbitotomy which revealed dense fibrous tissue with a high percentage of IgG4+ plasma cells, suggestive of IgG4-RD. Serum IgG subclass analysis was then performed which revealed a lone elevation of serum IgG4. Because of known possible involvement of other organs with IgG4-RD this patient underwent CT CAP with contrast which identified asymptomatic aortitis. The patient was initiated on a prednisone taper and had near complete resolution of orbital symptoms. Conclusion: This case illustrates the variable presentation of IgG4-RD and the importance of its early inclusion on the differential diagnosis for any new mass or fibrosis. Its ability to mimic other conditions amplifies the importance of early recognition and diagnostic persistence. This patient underwent several inappropriate treatments during the 8 months prior to diagnosis and multiple biopsies were required to confirm the diagnosis due to the initial needle biopsy being insufficient. Sampling error is a known flaw of needle biopsy and is particularly likely in IgG4-RD given its typical patchy storiform fibrosis that can be missed by a needle (1, 2). Additionally, as seen in this patient and as has been demonstrated in the literature, IgG4-RD is a systemic disease not isolated to one organ at a time (4). If IgG4-RD is identified or suspected, it may be reasonable to screen for involvement in other parts of the body.
Luis Gasca Dr. Jack McHugh	*When Good Arteries Go SCAD* Background: Spontaneous coronary artery dissection (SCAD) is an established, but uncommon cause of acute myocardial infarction with non-obstructive coronary arteries. SCAD has historically been underdiagnosed and there is an incomplete understanding of this disease. Here we present a case of a patient with typical symptoms and angiographic findings of SCAD and briefly discuss the causes and treatment of this disease. Case Presentation: A 48-year-old male with comorbid hypertension, obstructive sleep apnea, and obesity presented to an outside hospital for chest pain. In the hour prior to symptom onset, the patient completed a vigorous workout at his gym. After returning home, he developed sudden-onset, central, burning chest pain. The pain intensity or character were unchanged by milk intake or nitroglycerin, worse with activity and anxiety, and improved with a shower and lorazepam provided in the emergency department. Initial ECG in the emergency department demonstrated ST elevation in leads III and aVF and poor R wave progression, but subsequent ECGs had no significant ST findings. Posterior lead ECG was without ST changes. hs-Troponin I at the outside hospital trended from 350 ng/L to 9500 ng/L [reference 0.0-17.4 ng/L]. Given observed ST-elevation with dynamic change in troponin, he was taken to the cardiac catheterization laboratory for evaluation of his coronary arteries. Angiography demonstrated mild coronary

	artery atherosclerosis. Images were reviewed by a second interventionalist, who noted moderate diffuse narrowing and pruning appearance to the distal apical left anterior descending and distal right posterior descending coronary arteries, suggestive of SCAD. Given the initially indeterminate finding, cardiac MRI was obtained, which showed changes consistent with acute infarction of the apical septum and left ventricular dysfunction with LVEF 40-45%. The patient was monitored with telemetry for five days without recurrence of chest pain or development of ventricular arrhythmias. He was discharged on a beta-blocker alone with plans for cardiac rehabilitation and follow up in the SCAD clinic. Discussion: This patient had classic risk factors for coronary artery disease and presented with features highly suggestive of an occlusive myocardial infarction (OMI). However, angiography revealed SCAD. SCAD involves progressive separation of the coronary arterial wall and development of intramural hematoma, resulting in ischemia and infarction. Associated precipitants include emotional stress and intense physical exertion. This condition is more common in younger women (only 10% in men) and in those with fibromuscular dysplasia, connective tissue disorders, and systemic inflammatory diseases. This case emphasizes the importance of the newer classification schemes for acute coronary syndrome (OMI/NOMI vs. STEMI/NSTEMI/UA) and the need for increased clinical research for SCAD. It also provides an opportunity to review modified-lead placement ECGs, limitations of telemetry, risk factors and associated precipitants of SCAD, and outpatient counseling and follow-up for patients with history of this diagnosis.
Brent Gawey Dr. Trevor Simard Dr. Jissy Cyriac	*Not All Hearts Break Even: A Unique Case of Stress Induced Cardiomyopathy* Case Presentation: A 70-year-old female with hypertension and hyperlipidemia presented to the emergency department with chest discomfort, dyspnea, and diaphoresis. Prior to symptom onset, she was asked to heavily strain while being evaluated for rectovaginal prolapse. Intake exam was notable only for hypertension. Baseline troponin T was 195 ng/L and rose to 999 ng/L, and 1,318 ng/L at two and six hours, respectively. Electrocardiogram (EKG) showed normal sinus rhythm with T wave inversions throughout the inferior and precordial leads. Computed tomography chest with angiography was negative for pulmonary emboli and demonstrated patent coronary arteries. Given the chest pain and dynamic troponin elevation, invasive coronary angiography was performed to exclude obstructive or embolic lesions. Angiography demonstrated mild coronary artery disease (CAD) without evidence of plaque rupture or spontaneous coronary artery dissection (SCAD). Transthoracic echocardiography (TTE) demonstrated a left-ventricular ejection fraction (LVEF) of 33% and circumferential akinesis of the left ventricle. Hence, as a diagnosis of exclusion, the patient's presentation was deemed most consistent with stress induced (Takotsubo) cardiomyopathy (SCM), prompting treatment with a beta blocker, angiotensin converting enzyme (ACE) inhibitor, and aspirin. The patient remained asymptomatic to discharge and was scheduled for a repeat TTE in 3 months to assess LVEF recovery. Discussion: SCM is a clinical syndrome where patients present with acute angina, dyspnea, EKG changes, and troponin elevation similar to acute

	coronary syndrome, but without underlying obstructive lesions in the coronary vasculature. Most cases occur in postmenopausal women, and presentation is typically preceded by physical and/or emotional stressors. Notably, no cases following straining for rectovaginal prolapse assessment have been previously described. TTE reveals a reduced LVEF and most commonly shows apical dyskinesia of the left ventricle, although multiple variants exist, including mid-ventricular, basal, and focal patterns. A key finding on TTE are regional wall motion abnormalities not confined to a single coronary territory. Cardiac catheterization should be performed to rule out obstructive coronary pathology, including spontaneous coronary artery dissection and embolic disease, both of which can present similarly. While the pathophysiology is uncertain, SCM is thought to result from excessive sympathetic nervous system activation, causing a catecholamine surge. This disrupts coronary microvasculature and causes myocardial stunning. Once diagnosed with SCM, patients should be monitored for 48 to 72 hours for clinical improvement. Important to the general internist, clinical complications include QT prolongation, congestive heart failure, cardiogenic shock, left ventricular outflow obstruction, and ventricular arrhythmias. Treatment consists of aspirin, beta-blockers, and ACE inhibitors or angiotensin receptor blockers (ARBs) until LVEF recovery. Notably, only ACE inhibitors and ARBs have been associated with improved mortality. Most patients with SCM make a complete recovery in weeks to months. Repeat TTE three months after diagnosis is recommended to assess ventricular recovery.
Christine Grech	*Blast Crisis: Acutely Ill Presentation of B-Cell Acute Lymphoblastic Leukemia in a 46-Year-Old Man* Introduction: Symptomatic hyperleukocytosis is one of the most concerning complications in leukemia due to its high mortality with 20-40% dying within first week of presentation.[1] This clinical vignette in the acutely ill setting depicts a patient with respiratory failure later determined to be in a blast crisis. Uniquely, the patient was found to be in B-cell ALL as a 46-year-old adult; a leukemia with 75% of its incidence in children less than 6 years of age.[2] Case Presentation: A 46-year-old male who originally presented to an urgent care with a sore throat. There he received penicillin and ibuprofen. Shortly after he developed worsening shortness of breath. He returned to the urgent care and received epinephrine for presumed anaphylaxis. In the ED, he continued to be tachypneic, tachycardic, and febrile. He received steroids and epinephrine without good effect. Patient was intubated for airway edema. There was a small amount of bleeding with intubation. He was given broad spectrum antibiotics and fluids. To this point, the patient was being treated for distributive shock, presumed to be secondary to anaphylaxis. His clinical course changed when initial labs came back with severe pancytopenia and coagulopathy. His peripheral blood smear showed a heavy burden of blasts. CT of the chest and neck with significant burden of debris throughout the respiratory tract with concern for bleeding. The patient was transferred to the ICU. He was hypotensive and tachycardic. His extremities were warm, and his pulses were hyperdynamic. He was febrile and requiring oxygen support on vent. He was started on Levophed

	and heme-onc was consulted. He was severely neutropenic with an ANC of 0, platelets 19,000, WBC 1.46, and fibrinogen 746. At which point he was given blood products for the bleeding and started on broad spectrum antibiotics with prophylaxis for opportunistic infections. Initial picture concerning for DIC secondary to APL, but flow cytometry was positive for B cell ALL. He was on maintenance fluids already with concern for TLS as patient was in a blast crisis and elevated uric acid. Heme-onc discussed a possible clinical trial that may help the patient at the University of Minnesota, as patient was relatively stable on Levophed, it was determined that he would transfer. Discussion: This case illustrates the medical urgency of a hyperleukotic state given the clinical severity of the patient. It highlights the necessity for protocol in stabilization of critically ill patients to avoid premature closure, as he was initially presumed to be in anaphylaxis. Furthermore, while epidemiology can be a useful tool for diagnostic patterns, atypical presentations must be considered to avoid delays in care. While this patient age was atypical for ALL, his flow cytometry resulted quickly, and we were able get him to appropriate treatment.[2]
Naima Hashi Dr. Jack McHugh	*Inflammatory Bowel Disease in the Elderly Population* Introduction: The differential for chronic diarrhea is broad. It is important to keep inflammatory bowel disease (IBD) in the differential even in elderly patients. The occurrence of IBD in elderly individuals appears to be rising by 5.2% annually and up to 15% of IBD in North America and Asia is diagnosed after the age of 60 years (Taleban, 2015). Case Description: An 84-year-old female with a past medical history of hypertension and type 2 diabetes presented with a two-month history of non-bloody diarrhea, up to 10 episodes a day. She had associated nausea, decreased appetite, abdominal cramping, and an unintentional 20 lb weight loss. She was hospitalized one month prior for similar symptoms; at which time she was found to have Clostridioides difficile infection. She completed a course of oral vancomycin and continued to have loose stools. Several PCR tests for C. difficile were negative. On presentation, patient was afebrile and hemodynamically stable. She had mild diffuse tenderness of her abdomen; otherwise, the physical examination was unremarkable. The patient's labs were pertinent for C-reactive protein (CRP) 112mg/L, white cell count 10.0x10(9)/L, stool calprotectin 125mcg/g (from 2700mcg/g three weeks prior) and repeat C. difficile PCR was negative. Due to the elevated inflammatory markers, she underwent a colonoscopy which revealed mucosal changes consistent with active ulcerative colitis. She was diagnosed with ulcerative colitis, likely triggered by her prior C. difficile infection. The patient was initiated on intravenous methylprednisolone for 3 days. Symptoms improved and her C-reactive protein downtrended. She was transitioned to oral prednisone 40 mg and discharged with close follow-up in the department of Gastroenterology and with plans to start biologic therapy in the outpatient setting. Discussion: It is important to have a broad differential for chronic diarrhea. Chronic diarrhea can be broken down into three categories: inflammatory (infection and autoimmune), fatty (malabsorption), and watery (secretory and

	osmotic). Stool calprotectin can be obtained if there is suspicion for IBD, if it is elevated colonoscopy can be ordered for further evaluation. It is important to know that IBD diagnosis in the elderly population is on the rise. During an IBD flare, it is imperative to exclude infection (C. difficile, CMV). Patients with IBD are at a high risk of developing C. difficile and having severe complications. A flare is treated with three days of intravenous methylprednisolone with close monitoring of CRP and symptoms. Patients can then be discharged with prednisone taper and close gastroenterology follow-up.
Christopher Heinrich	*Not So Essential Hypertension: Avoiding Premature Closure* Introduction: Hypertension is exceedingly common, with prevalence estimated at 47% of the US population based on ACC/AHA definitions. (1) The majority of patients with hypertension do not have an identifiable secondary cause and are classified as primary hypertension. However, a variety of disease processes can also cause hypertension, or worsen hypertension, and should remain on the differential diagnosis. Case Presentation: A 57-year-old male was referred to the Nephrology Clinic for resistant hypertension. His other medical history was notable for obstructive sleep apnea and active tobacco use. He was initially started on lisinopril and amlodipine for hypertension, but over the year prior to presentation, his regimen had increased to 6 agents. In the clinic, he was noted to be bradycardic to the 40s and his home measurements corroborated this finding. He did not have any symptoms of lightheadedness or syncope. He had previously undergone work up for secondary hypertension which was notable for hypokalemia with preserved renal function. Additionally, he was noted to have an elevated aldosterone level at 9.6 with a suppressed renin level, even on an ACE-inhibitor. Serum metanephrines were normal and a 24-hour urine aldosterone after salt-loading was not consistent with primary hyperaldosteronism. A renal ultrasound with Doppler did not demonstrate evidence of chronic renal disease. Due to the bradycardia, a 24-hour Holter monitor was obtained which demonstrated periodic complete heart block. A cardiac MRI demonstrated regional hypertrophy involving the basal to mid inferoseptum with associated low intensity mesocardial delayed enhancement without wall motion abnormality. Subsequently, PET CT demonstrated FDG avid lymph nodes in the neck, chest, and upper abdomen, but there was no evidence of an adrenal adenoma on any imaging. He then underwent bronchoscopy with ultrasound guided biopsy which demonstrated granulomatous inflammation consistent with cardiac sarcoidosis. He underwent pacemaker placement for complete heart block and had significant improvement in his blood pressure and was weaned to a simplified, four-drug regimen. Given the high aldosterone and suppressed renin, he was placed on spironolactone to good effect. Discussion: It appears that this patient's resistant hypertension manifested as a compensatory response to significant bradycardia caused by sarcoidosis-induced conduction disturbance. Although sarcoidosis is not at the top of the differential diagnosis for causes of hypertension, and rightly so, this case demonstrates the importance of astute investigation into a seemingly routine diagnosis. For perspective, it is estimated that clinically significant cardiac sarcoidosis occurs in about 5% of patients with pulmonary sarcoidosis. (2) Additionally, AV block is the most common presentation of cardiac sarcoidosis, and this should be higher on the differential in younger patentis who develop complete heart block. (3,4) Once this was diagnosed and

	treated, his hypertension came under better control. This highlights the need to at least consider uncommon causes of hypertension.
Mason Hinke	*Anaplasmosis Presenting as Atrial Fibrillation with Rapid Ventricular Response* Background: Anaplasmosis is a tick-borne illness most commonly seen in the northeastern United States and Wisconsin. There were over 5,000 cases in 2017 but this is likely under reported. Patients most commonly present with nonspecific symptoms such as fever, malaise, myalgia, headache and arthralgia. Methods: The patient in this case is a 61-year-old male with a history of type 2 diabetes mellitus and hypertension who went hiking in Western Wisconsin approximately three weeks prior to hospitalization. One week following the hike, he discovered a tick on his abdomen and had an onset of fevers, up to 104F, one to two days later. He presented to the ED given worsening fevers and was found to be atrial fibrillation with rates into the 180's and hypotensive with maps in the 60s. Physical exam showed an erythematous rash over the lower abdomen with central necrosis. Labs were notable for thrombocytopenia to 36 k/cmm, leukopenia at 3.31 k/cmm, elevated transaminases (AST 297 IU/L, ALT 170 IU/L). It was unclear the duration of which he was in atrial fibrillation and was thought to be secondary to hypovolemia and acute infection. He had a CHA2DS2-VASc score of 2 (history of hypertension and type 2 DM) and given the acute nature of his atrial fibrillation and overall low risk of stroke given his CHA2DS2-VASc and potential risk of bleeding given his thrombocytopenia, it was decided to proceed with an electrical cardioversion which converted him to normal sinus rhythm. He received a total of 3 L IV fluids and 2 g of magnesium and admitted for further evaluation. Infectious disease was consulted given concern for tick borne illness such as anaplasmosis, ehrlichiosis, RMSF, or Lyme. The patient provided a photo of the tick which appeared to be a deer tick. He was empirically started on doxycycline 100 mg BID for fourteen days, which would empirically cover both anaplasmosis and Lyme disease, while awaiting PCR results. Other causes of pancytopenia, such as HIV or Hepatitis C were negative. The peripheral smear showed normocytic/normochromic anemia. His platelets, white blood cell count and liver enzymes remained stable and continued to improve at the time of discharge. Conclusion: This case highlights how atrial fibrillation with rapid ventricular response can be an abnormal presentation and complication of anaplasmosis. Anaplasmosis can be challenging to diagnose but should be considered in individuals with febrile illness in endemic areas. It is crucial to begin antibiotics immediately when tick borne illness is suspected, as delayed initiation of therapy is associated with increased rates of transfer to intensive care units.
Sara Inglis	*A Case of Unusual Bleeding in AA Amyloidosis* Introduction: AA amyloidosis may complicate any chronic inflammatory condition and is characterized by extracellular tissue deposition of fibrils composed of serum amyloid A protein. Bleeding may occur with this disorder and is typically due to either spontaneous cerebral or gastrointestinal hemorrhages or peri-procedural hemorrhage.

	Case presentation: A 43-year-old woman with AA-type renal amyloidosis and stage 3 CKD presented with left flank pain. The diagnosis of AA amyloidosis was made 12 years previously by renal biopsy and mass spectrometry and thought to be secondary to a periodic fever syndrome. She had a significant family history with unremarkable genetic testing. She had been on treatment with canakinumab but planned to transition to Anakinra secondary to concerns over treatment resistance. The patient was hesitant to start immunosuppression amid the Covid-19 pandemic and did not elect to start therapy. On this occasion, she presented with acute left flank pain and was found to have a spontaneous left subcapsular and renal hematoma with mass effect on the kidney. She underwent embolization of a branch of the left renal artery. This was unfortunately complicated by progressive renal failure, and she was initiated on hemodialysis via a tunneled line. Her hospitalization was further complicated by worsening anemia and thrombocytopenia requiring multiple blood products. Repeat imaging was reassuring for a stable hematoma. She had bleeding from her catheter insertion site and IV puncture sites but there was no clear evidence of DIC. Inflammatory markers were elevated with a CRP of greater than 320 mg/L and a leukocytosis peaking at 38 x10^9/L. Extensive workup was negative for underlying infection or malignancy. Ultimately, she was initiated on anakinra at 100 mg on alternate days for treatment of AA amyloidosis and discharged in stable condition. The patient continued to have recurrent bleeding complications after hospital discharge. She developed pseudoaneurysms of her right groin and antecubital area, hematomas following AV fistula placement, and later bleeding post-needling for dialysis. Approximately one month after hospitalization, she was diagnosed with a new large right subcapsular hematoma and retroperitoneal hemorrhage. She underwent bilateral nephrectomies and had further bleeding complications postoperatively. Discussion: The mechanisms that predispose patients to bleeding in amyloidosis depend on amyloid type and pattern/extent of disease. Amyloid deposition in blood vessels and perivascular tissue leads to amyloid angiopathy with increased fragility of vessels and impaired vasoconstriction. Patients with renal amyloidosis may suffer from further hemostatic complications relating to renal dysfunction, which was likely observed to some extent in this case. Perirenal hematoma is a rare bleeding complication of amyloidosis, and the current literature is limited. Presented here is a patient with bilateral spontaneous perirenal hematomas and multiple bleeding complications in the setting of an inflammatory state, concerning for amyloid vasculopathy secondary to AA amyloidosis.
Rachel Jenkins Dr. Reema Tawfiq	*Immune Checkpoint Inhibitors: A Double-Edged Sword* Introduction: Immune-checkpoint inhibitors (ICIs) are a group of novel oncologic therapies that modulate immune system response. Utilization of immune check point inhibitors is becoming increasingly common. As such, immune-related adverse events (irAEs) are becoming increasingly recognized. Immune-related adverse events have been recognized in nearly every organ system and each carry their own associated incidence, mortality, and morbidity [1]. Case Description: A 73-year-old male with mucinous adenocarcinoma of the

	lung undergoing treatment with pembrolizumab, carboplatin, and pemetrexed presented for his fourth cycle of chemotherapy. Pre-labs showed elevated creatinine kinase (1325 U/L), aspartate aminotransferase (68 U/L), alanine aminotransferase (168 U/L), and troponin (292 ng/L). Chemotherapy was not given, and he was sent to the emergency department for further evaluation. He denied chest pain, palpitations, increased shortness of breath, abdominal pain, nausea, or vomiting. Electrocardiogram showed normal sinus rhythm with right bundle branch block (present previously) and new premature ventricular complexes. He was admitted to cardiology due to concern for ICI cardiotoxicity. He was given a one-time dose of prednisone 80 mg. Oncology was consulted and recommended continuation of daily prednisone, cardiac MRI, and transthoracic echocardiogram for evaluation. Cardiac MRI showed patchy late gadolinium enhancement, subtle edema at the inferior left ventricle, and mild hypokinesis of the inferior and inferoseptal walls consistent with active myocarditis. Echocardiogram demonstrated an ejection fraction of 60% without regional wall motion abnormalities. Given evidence of myocarditis on cardiac MRI and continually increasing troponins, oncology recommended initiating IV methylprednisolone 500 mg for a three-day course. Troponins peaked at 589 on day 4 of admission. After completing the three-day course of IV high dose steroids, troponins, creatinine kinase, and liver enzymes were down trending. At this time, he transitioned to high-dose oral prednisone. The patient followed up with oncology for continued monitoring and management of prednisone taper. Chemotherapy was restarted with pemetrexed and carboplatin only. A re-challenge of the ICI, pembrolizumab, may be trialed in the future. Discussion: As use the of ICIs for the treatment of malignancy becomes more common, irAEs are increasingly encountered in clinical practice. Although ICI-associated myocarditis has a relatively low incidence, it has a reported mortality rate of 25-50% [2]. Disease presentation and progression can range from asymptomatic elevations in cardiac markers to life-threatening cardiac dysfunction. Therefore, prompt recognition and management are necessary to prevent fulminant progression. References: 1. Ho, A. K., et al. “Immune-Related Adverse Events Associated with Immune Checkpoint Inhibitor Therapy.” Anesthesia & Analgesia. 132: 2 (2021) 2. Palaskas, N., et al. “Immune Checkpoint Inhibitor Myocarditis: Pathophysiological Characteristics, Diagnosis, and Treatment.” JAHA. 9:2 (2020)
Whitney Johnson Jacob Kohlenberg	*Seizing the Diagnosis and Management of Pseudohypoparathyroidism* Introduction: Pseudohypoparathyroidism (PHP) encompasses a group of heterogeneous disorders characterized by target organ resistance to parathyroid hormone (PTH) mediated by various defects within the PTH/PTH-related peptide signaling pathway. Each disorder within the PHP spectrum is unique as the clinical presentation varies based on the location of the defect. For example, PHP type 1B is characterized by progressive development of PTH resistance within the proximal renal tubule; defined by elevated PTH, hypocalcemia, and hyperphosphatemia in the setting of normal

	vitamin D, magnesium, and renal function. Patients with PHP type 1A also have Albright's hereditary osteodystrophy (AHO) with features of round facies, short stature, and brachydactyly. We present a case of diagnosis of PHP type 1B after presentation with a seizure. Case Description: A 24-year-old man presented after a generalized tonic-clonic seizure. His medical history was notable for one remote seizure and chronic lower extremity paresthesias and cramping. In childhood, he underwent multiple tooth extractions and puberty was delayed compared to peers. He did not have cognitive deficits, fractures, cataracts, or obesity. Family history was notable for similar symptoms in his mother. On admission, calcium was 5.1 mg/dL with normal albumin, phosphorus was 5.6 mg/dL, and PTH was elevated at 97 pg/mL. TSH was also elevated at 6.9 mU/L with a normal free T4. Magnesium, vitamin D, and creatinine were normal. CT head demonstrated dense calcifications of the basal ganglia and subcortical white matter. Physical exam was significant for teeth crowding, positive Chvostek sign, and absence of typical AHO features. Based on his biochemical findings and absence of AHO features, he was diagnosed with PHP type 1B. Calcitriol, calcium supplementation, and a low phosphorus diet were started. Serum calcium levels one day and one week after discharge were normal. There was no need for thyroid hormone supplementation in the setting of mild TSH resistance with a normal free T4. Discussion: Major criteria for diagnosing PHP are PTH resistance with or without subcutaneous ossifications, early onset obesity, TSH resistance, AHO features, and family history. Diagnosis is mainly clinical, but genetic testing can be pursued on an individual basis to characterize the disease subtype if there are atypical features. Seizure is a more common presentation in PHP type 1B than type 1A because the diagnosis can be delayed due to lack of characteristic physical exam findings. Importantly, treatment of PHP type 1B differs from hypoparathyroidism. In hypoparathyroidism, the goal serum calcium is typically slightly below the lower limit of normal because fully normalizing serum calcium will cause hypercalciuria with an increased risk for nephrolithiasis and kidney disease. In contrast, the effect of PTH on the distal renal tubule is preserved in PHP type 1B so calcium can be repleted with less concern for hypercalciuria.
Simranjit Singh Kahlon Dr. Abdilahi Mohamoud	*Recurrent Tricuspid Valve Replacement Due to Continuos IVDU-Related Endocarditis* Introduction: Endocarditis is a life-threatening inflammation of the endocardium which is the inner lining of the heart's chambers and valves usually caused by bacteria. Infective endocarditis is a common complication in Intravenous Drug Users with or without damaged heart valves, but the second episode of IE was seen in only 32% of IVDUs and the third episode in only 12% of these patients.[1]. In an IVDU, treating IE is considerably more challenging due to continued IV drug use and high recurrence rates that result from non-compliance with medical care. Case Presentation: A 28 y.o. male who is an IVDU, dependent on opioids, had 3 prior episodes of endocarditis, status post tricuspid valvectomy x2(native and prosthetic), tricuspid valve replacements, and right heart failure presented to the emergency for heroin overdose. He was found to be severely edematous with evidence of pulmonary edema. An echocardiogram showed evidence of tricuspid endocarditis with torrential tricuspid stenosis. A repeat

	echo revealed right to left shunt on bubble study. Given his high oxygen requirement(8-15L), right to left shunt physiology, recurrent large vegetations, and severe tricuspid stenosis, surgical intervention with tricuspid valvectomy was performed. PFO was reported to be closed. A completed prolonged course of IV antibiotics with Amikacin, Imipenem, and Tigecycline for Enterococcus Faecalis, Methicillin-Resistant Staph Epidermis. ESBL Klebsiella Oxytoca, Candida Dublinensis, and Mycobacterium Abscessus. The patient has been living on supportive care without a tricuspid valve awaiting tricuspid valve replacement in light of pending sobriety for 6 months. The patient is receiving Methadone with guidance from addiction medicine and working titration of diuretics for volume overload. Conclusion: Infective endocarditis is a severe condition with high morbidity and mortality. Many IV drug users who do not comprehend the severity of the circumstances can continue to use IV drugs even after a valve replacement resulting in prosthetic valve endocarditis. The possibility of a third valve replacement becomes questionable, regardless of how rare this event might be. Surgical intervention is delayed owing to their irregular behavior, which creates reservations regarding surgical intervention. A comprehensive and multidisciplinary approach with a special focus on Addiction Medicine is required to manage the morbidity and mortality related to Injection drug use-related endocarditis. [1]Rodger L, Shah M, Shojaei E, Hosseini S, Koivu S, Silverman M. Recurrent Endocarditis in Persons Who Inject Drugs. Open Forum Infect Dis. 2019 Sep 9;6(10):ofz396. doi: 10.1093/ofid/ofz396. PMID: 31660358; PMCID: PMC6796994.
Michael Kalinoski	*Bacillus Thuringiensis Bacteremia in an IV Drug User* Case Presentation: A 38-year-old female with history of IV drug use, tobacco abuse, and morbid obesity presented with multiple systemic symptoms that started soon after injecting heroin the day before admission. Our patient noted to have filtered her heroin through a cotton swab. She noted subjective fevers, chills, headaches, dizziness, nausea, vomiting, diarrhea, and body aches. She presented to the Emergency Department with sepsis physiology and was started on empiric broad-spectrum antibiotics. There were various abnormalities present on the initial workup, however, no definitive source of infection was identified. Our patient's condition continued to deteriorate necessitating ICU transfer for vasopressors and airway support. Blood cultures grew Bacillus thuringiensis and Serratia marcescens after <24hrs of inoculation. Despite maximal medical support, our patient developed refractory septic shock with multi-organ failure and expired on comfort measures. Conclusion: Bacillus thuringiensis is an uncommon cause of Bacillus bacteremia, only previously reported in the literature as a nosocomial infection. This report describes a case of Bacillus thuringiensis in an IV drug user who was using cotton to filter her heroin.
Victoria Kalinoski-Dubose Dr. Jordan Voss Dr. Connor Loftus	*Black Esophagus* Introduction: A 60-year-old male presented with one-day history of bloody emesis, dysphagia, and odynophagia. His past medical history was pertinent for alcohol use of 6-8 vodka drinks per day. Exam revealed tachycardia, mild

	epigastric tenderness and he was spitting oral secretions into an emesis bag due to odynophagia. Diagnostic Evaluation: Pertinent labs included a white blood cell count of 13.4 per µL, bicarbonate of 15 mmol/L, anion gap of 40, beta hydroxybutyrate of 4.4 mmol/L (ref <0.4), urine ketones markedly positive at >160 mg/dL, and lactate of 5.3 mmol/L (ref 0.5-2.2). Esophagogastroduodenoscopy was performed and biopsies were taken confirming the diagnosis of acute esophageal necrosis. Management: The patient was managed with intravenous fluid resuscitation, electrolyte repletion and a proton pump inhibitor. His symptoms gradually improved and his oral diet was advanced over the course of three days. Discussion: Black esophagus, formally known as acute esophageal necrosis is thought to be caused by combined ischemic injury and direct mucosal injury resulting from gastroesophageal reflux or other insults. This patient likely suffered hypoperfusion secondary to volume depletion from alcohol consumption and vomiting and direct mucosal injury from gastric contents and severe alcoholic ketoacidosis. The distal esophagus is often more affected, possibly due to less robust blood supply and increased gastric acid exposure. Diabetic ketoacidosis has been previously identified as a risk factor suggesting the metabolic state of ketoacidosis might play a key role in the pathogenesis. Treatment is supportive with fluid resuscitation, proton pump inhibitors, and management of underlying conditions. Complications include esophageal perforation and stricture. Mortality rates may exceed 30%.
Alexis Keefe Dr. Susan Lou	*Toeing the Line of Liver Failure on Terbinafine* Introduction: Terbinafine is a commonly prescribed fungicidal agent frequently prescribed topically for cutaneous tinea infections and orally for onychomycosis and tinea capitis. Systemic terbinafine has rarely been associated with serious and potentially fatal drug-induced liver injury as well as hematologic toxicity including aplastic anemia. Case Description: A 62-year-old female with a past medical history of Type 2 Diabetes Mellitus and hypothyroidism was transferred to the Minneapolis VA from an outside hospital where she presented with three weeks of fatigue, pruritis, jaundice, nausea and unintentional weight loss. Initial workup showed a primarily cholestatic liver injury (ALT 230, AST 139, Alk Phos 1542, Tbil 44.3 (direct 30.8), INR 2.0, and pancytopenia requiring transfusion. Physical exam revealed jaundice and mild right upper quadrant pain without asterixis, hepatic encephalopathy or stigmata of chronic liver disease. A comprehensive imaging evaluation revealed no biliary obstruction or other etiology acute injury including vascular causes. Further, a comprehensive infectious evaluation did not reveal a cause for liver injury from common or uncommon etiologies, there was no serological evidence of autoimmune disease, and no history of toxic ingestion. Subsequent liver biopsy showed predominantly zone 3 hepatocanalicular cholestasis without steatosis or fibrosis, no histologic features of autoimmune hepatitis, obstruction, or chronic biliary disease. Overall, this raised the possibility of drug/toxin induced liver injury with the possible offending agent terbinafine which was initiated six weeks prior for onychomycosis and held on admission after consulting LiverTox®. Bone marrow biopsy showed marked pancytopenia,

	without evidence of hematolymphoid or metastatic neoplasm, and negative fungal and mycobacterial stains, overall consistent with acute bone marrow injury. Although the patient did not yet meet criteria for acute liver failure, bilirubin and alkaline phosphatase continued to rise, prompting transfer to Madison, WI for liver transplant evaluation. Discussion: This case demonstrates how the diagnosis of drug-induced liver injury is one of exclusion. It also highlights the utility of LiverTox® as a resource for clinicians to easily access information about liver injury risk associated with prescription and nonprescription medications. This, in combination with thorough medication reconciliation and history taking, can help recognize potential side effects of medications and facilitate rapid cessation of possible offending drugs.
Mohammed Khalid	*Pain in Z-Pak: A Case of Diffuse Pan-Bronchiolitis (DPB) in a Kidney Transplant Patient* Introduction: DPB is a chronic progressive pulmonary disease with unknown pathogenesis and characterized by chronic inflammation in respiratory bronchioles and sino-bronchial infection (1). If left untreated, it can result in bronchiectasis, respiratory failure, and death (3). It is a rare genetic disease affecting East Asians and is strongly associated with the HLA B24 in Japanese and A11 in Koreans (2). In addition to the limited number of DPB cases reported from outside East Asia, about half of the limited number of DPB cases reported from Western countries are Asian immigrants (3). DBP in kidney transplant patients has not been reported. This study describes our experience with DBP in a kidney transplant patient. Description: A 54 y.o. Asian female with PMH of ESRD due to anti-GBM GN, kidney transplant in 2004 on prednisone and mycophenolate. Initially presented with daily productive cough for 1 year with no SOB, wheezing, or chest pain. Recalls of getting antibiotics in the past helped a little but did not resolve symptoms. Lived in the countryside of Laos but does not recall respiratory symptoms. O2 Sat > 90 on room air. PFT showed mild airflow obstruction with FEV1/FVC < 70%. CT chest shows some tree-in-bud opacities and bronchiectasis. MAI, TB, and Histoplasmosis were negative. Bronchoscopy showed airways filled with thick mucoid/mucopurulent secretions that were white to yellow but otherwise normal. BAL grew Moraxella and was given a 14-day course of Azithromycin, which led to complete resolution of her cough, A repeat CT chest showed excellent resolution of diffuse bilateral nodular opacities. Diagnosis of DPB was made. 3 months later, the patient came to the clinic complaining of a recurrence of cough for 1 month with repeat CT chest showing recurrence of extensive bilateral opacities. She was restarted on azithromycin and seen again in a month with a resolution of symptoms and improved imaging. Discussion: DBP is characterized clinically by chronic cough, sputum, exertional dyspnea, chronic sinusitis, radiologically by small diffuse nodules, and histologically by chronic inflammatory lesions around respiratory bronchioles. Diagnostic criteria (i) persistent cough, sputum, exertional dyspnea; (ii) history/current chronic sinusitis; (iii) bilateral diffuse small nodular shadows on CXR or centrilobular micronodules on chest CT. (iv) coarse crackles; (v) FEV1/FVC <70%, PaO2 < 80 mmHg; and (vi) cold haemagglutinin titer > 64. Diagnosis should fulfill criteria 1, 2, 3, and at least two of criteria 4, 5, and 6 (4). Currently, treatment is with macrolides (5). Erythromycin has been the first choice according to guidelines in Japan (6)

	Azithromycin has fewer side effects (7), is well tolerated, and achieves a good response in treatment (8). The optimal duration of therapy is unknown, but most patients are treated for a minimum of six months (3).
Priscilla Koirala Dr. Savannah Liddell Dr. Jennifer Kleinman Sween	*A Tick-ling Case of Hemolytic Anemia* Introduction: Human babesiosis is a parasitic infection caused by Babesia species. B. microti is the predominant species that infects humans in the United States, particularly in endemic areas such as the Northeast and Midwest. The protozoan infects erythrocytes, causing non-immune mediated lysis resulting in anemia. Very rarely, babesiosis causes a warm autoimmune hemolytic anemia, characterized by antibody formation after intraerythrocytic parasitemia. We present a case that highlights the challenges in diagnosing and treating this rare complication. Case description: A 53-year-old gentleman with history of splenectomy presented with fever, chills, headache, and malaise. A diagnosis of babesiosis was made via PCR and the patient was prescribed a 10-day course of atovaquone and azithromycin. His symptoms persisted, leading to an additional course of clindamycin and quinine. He continued to have worsening malaise, fevers, weakness, and jaundice, which prompted further evaluation. CBC was remarkable for a hemoglobin of 6.7 g/dL down from 13.3 g/dL at baseline. Work up revealed LDH elevated to 1675 U/L, undetectable haptoglobin, absolute reticulocyte counts elevated to 334 x109/L, and indirect hyperbilirubinemia consistent with hemolysis. Severe refractory babesiosis was initially suspected due to the ongoing symptoms and severe hemolytic anemia, however peripheral smear demonstrated only 0.1% babesia burden. The patient was restarted on atovaquone and azithromycin. Blood transfusions were given; however, multiple antibodies identified on crossmatch required transfusion of uncrossmatched blood. Despite multiple transfusions, hemoglobin levels did not stabilize, and labs suggesting ongoing hemolysis. Coombs test was positive for polyspecific IgG and C3 antibodies, confirming the diagnosis of post-babesiosis warm autoimmune hemolytic anemia. He was initially treated with high dose prednisone 60mg daily, however due to lack of improvement this was increased to120mg daily and weekly rituximab infusions were added. Following his transfusions, the patient's urine was noted to turn dark brown in color. Urinalysis showed 1-3 RBC/hpf with 'large' hemoglobin, consistent with hemoglobinuria and pigment nephropathy. Transfusions were stopped to prevent worsening kidney injury, as the patient was hemodynamically stable and asymptomatic despite low hemoglobin levels. Intravenous fluids were initiated and renal function normalized. Following stabilization, the patient was discharged home on prednisone, rituximab, atovaquone and azithromycin. Discussion: Post-babesiosis warm autoimmune hemolytic anemia is a rare immune-mediated hematologic complication of babesiosis. Asplenic patients are at particularly high risk, and this condition typically presents 2-4 weeks after antiparasitic treatment even in the absence of parasitemia. The treatment is via immunosuppressive medications rather than antiparasitics. This case illustrates the importance of awareness of this condition in babesiosis patients with ongoing hemolysis despite low level or absent parasitemia. A particularly high index of suspicion is warranted in patients with asplenia, as they are at highest risk for this complication.
Grace Kollannoor	*A Rare Case of Ischemic Gastritis Secondary to Gastric Distention*

Samuel Dr. Mehria Sayad-Shah	Introduction: Ischemic gastritis is a rare disease with high mortality, often requiring urgent medical and surgical interventions. Patients presenting with this condition often have either compromised gastric perfusion or extensive gastric distension. Unlike other parts of the gastrointestinal tract, ischemia of the stomach is uncommon due to its rich blood supply. Case presentation: A 75-year-old male with a history of gastric and bowel dysmotility secondary to cerebral palsy, partial colectomy with a colostomy, and chronic back pain on NSAIDs came to the emergency department with abdominal distension, pain, vomiting, dysuria, and loose stools. At presentation, the patient was afebrile, hypotensive, and tachycardic with metabolic acidosis, mildly elevated lactic acid, leukocytosis, normal liver function, and normal lipase. CT imaging with contrast showed significant gastric gas and distension with portal vein gas, and patent celiac artery and branches. General surgery and gastroenterology were consulted early. Efforts to decompress the distended stomach were initiated immediately with the aid of a nasogastric tube and low intermittent suction. In addition, high-dose intravenous pantoprazole and antibiotics (ceftriaxone and metronidazole) were started. Abdominal distension and pain were significantly improved immediately following the initiation of decompression. Upper endoscopy findings were consistent with ischemic necrosis of the greater fundus and body of the stomach with areas of hemorrhage. Pathology results were consistent with gastritis and negative H pylori. By the third day of admission, patient was able to tolerate small quantities of clear liquid; however, diet could not be progressed beyond clear liquids due to recurrent abdominal distension and discomfort. To promote bowel rest and to meet calorie needs, a nasojejunal tube feeding was started while encouraging oral intake. By the ninth day of hospitalization, patient met daily calorie requirements without tube feeds. He was discharged with instructions for outpatient follow-up, high-dose oral pantoprazole, oral ciprofloxacin and metronidazole (to complete a 14-day course of antibiotics), and a follow-up upper endoscopy in 11 weeks to assess the areas of ischemia. In addition, the patient was asked to avoid any NSAIDs in the future. Discussion: This rare case of ischemic gastritis identifies chronic gastric and gut dysmotility as potential risk factors for ischemic gastritis. It is possible that extensive gastric distension might have compromised blood supply and led to tissue necrosis and pneumatosis. It is unclear if an unidentified source of gastric infection played a role in increasing gastric pneumatosis. Lessons learned: If detected early, gastric distention-related ischemic gastritis can be managed medically. Treatment should be aimed at decompression, reducing gastric irritation, and treating possible infections. We also learned that CT abdomen with contrast can provide important clues for diagnosis including visualization of gastric distension, portal vein gas, and often the patency of gastric blood vessels.
Marinos Kosmopoulos	*An Unusual Effusion: Focal Mesothelial Hyperplasia in a Patient with Cardiac Tamponade* Introduction: Clinically significant primary pericardial effusions pose a fascinating diagnostic and therapeutic challenge, with a diverse underlying etiology and an uncertain prognosis. Case Presentation: A 75-year-old male with a past medical history significant

	for hypertension, gout, Raynaud's phenomenon, and post-traumatic stress disorder presented to the emergency department with dyspnea and cough. Six years prior to this presentation, he had a chest CT for lung nodule surveillance showing a trace pericardial effusion, although he was asymptomatic at the time. Chest x-ray revealed an enlarged cardiac silhouette. CT scan and an echocardiogram demonstrated a moderate pericardial effusion. The patient was started on colchicine and indomethacin for effusive pericarditis and was discharged. His dyspnea progressed, and a follow-up echocardiogram three months after the initial presentation demonstrated enlargement of the pericardial effusion with diastolic compression of the right ventricle and echocardiographic features of cardiac tamponade. The patient was emergently transferred to our hospital and underwent pericardiocentesis. A total of 470 ml of yellow fluid was removed with elevated protein and normal white blood cell differential. Labs were notable for mild neutrophilic leukocytosis with normal inflammatory markers. Pericardial fluid cultures were negative and auto-immune work-up was unremarkable; however, cytology was notable for mesothelial hypercellularity, generating concern for malignant pericardial effusion. The patient had a repeat CT of the chest that demonstrated no evidence of pleural disease. To further evaluate the etiology of his effusion, he underwent pericardial biopsy with a pericardial window. The pericardial biopsy showed areas of chronic inflammation and focal mesothelial hyperplasia with papilla formation without evidence of malignancy. He was seen in the clinic 3 weeks after hospitalization and was in good health reporting resolution of chest pain and no recurrence of dyspnea. The patient had served as a veteran in Vietnam near the Mekong delta, had a 50-year history of smoking, and did not have any family history of mesothelial malignancies. He is unaware of whether he had agent orange exposure. Conclusion: This case describes a highly unusual cause of cardiac tamponade due to a slowly enlarging pericardial effusion secondary to focal mesothelial hyperplasia, that, to the best of our knowledge, has not been reported in the literature before. This case underlines the importance of pericardial biopsy in the diagnosis and prognostication of pericardial neoplasms, as cytologic evidence of mesothelioma may be misleading in the absence of concomitant pleural disease. Moreover, it highlights the absence of evidence regarding the optimal follow-up and management of incidental pericardial effusions, pointing to the need for a comprehensive re-evaluation of current practice and establishment of clinical guidelines.
Ameya Kumar	*Disseminated Gonococcal Infection as a Cause of Polyarthritis in a 61-Year-Old Male* Introduction: Disseminated gonococcal infection is a common cause of polyarthritis. Approximately 0.5% to 3% of patients who are infected with N. gonorrhoeae develop disseminated gonococcal infection (DGI). Historical data in the 1980s show that N. gonorrhoeae were associated with up to 14% of patients who have arthritis. Case description: A 61 year old with history of ground level mechanical fall was admitted to the hospital with complains of polyarthralgia , weakness and subjective fever and chills. Initial labs were notable for an elevated WBC cpunt of 31,000. He was thought to have sepsis from an unclear source. Rheumatology was consulted for polyarthralgia. Most of the joints were sterile with nucleated cells <10,000, however aspiration of left knee showed Gonococcal infection on culture and PCR. He was started on therapy with

	Ceftriaxone, and had significant improvement. However, he still had joint pain which was attributed to reactive arthirits secondary to disseminated gonococcal infection. He was started on Solumedrol therapy, and eventually tapered with Prednisone and reported significant improvement in his pain. Discussion: Polyarthritis is a common presentation that can have multiple etiologies. The diagnostic approach to patients presenting with polyarthritis often takes into account demographic factors like age and sex. For people belonging to an older demographic the diagnostic approach is often biased towards rheumatoid arthritis, crystal arthropathy, vasculitis and degenerative joint disease. However, it should be stressed that sexually transmitted infections like gonorrhea, chlamydia and syphilis should be considered as potential etiology of polyarthritis.
Andrew Lesser Dr. Breanna Zarmbinski	*An Atypical, Fatal Infection Associated with a Typical, Life-Prolonging Medication* Introduction: Disseminated aspergillosis is a rare but potentially fatal fungal infection that primarily affects immunocompromised patients. The fatality rate for this illness has been estimated from 40% - 90% of cases1. Thankfully, it can be treated if caught early enough in the disease process. As targeted therapies with immunosuppressive side effects become more commonplace treatments for many cancers and autoimmune diseases, it has become increasingly important to recognize rare but serious infections such as disseminated aspergillosis that have occurred after initiation of such medications. Case Presentation: An 80 year-old female was admitted for further workup of a three day history of confusion and lethargy. She had a past medical history of ITP from CLL/SLL with recurrent severe GI bleeds secondary to AVMs managed with transfusions, Rituximab, high doses of oral prednisone, and IVIG, which she last required approximately six weeks prior to presentation. When her platelets stabilized approximately one month prior to presentation, she was started on ibrutinib for her CLL/SLL. On admission, the patient was afebrile and vitally stable. She was alert but only oriented to person and situation, not year or place. She otherwise had no notable focal neurologic deficits. WBC was 18k (her baseline) and platelets 95k. Other labwork including BMP, LFTs were unremarkable. Chest XR showed an irregular soft tissue opacity within the RUL. Head CT showed a 1.3 x 1.1 cm rim-enhancing lesion, concerning for cystic neoplasm vs abscess. Brain MRI showed several small-moderate lesions with thin peripheral enhancement within her cerebral hemispheres. The differential for her combination of lung and brain lesions included sequelae of atypical including fungal infection or metastatic disease. Given these concerns, ibrutinib was held and she was empirically placed on imipenem-cilastin and valacyclovir. A lumbar puncture was performed that night, which showed no evidence for viral or bacterial meningitis and no initial growth on fungal cultures. A lung biopsy was pursued the next day and showed evidence of aspergillosis. She was promptly started on voriconazole and caspofungin. Conclusion: Early diagnosis of disseminated aspergillosis is important. If disseminated fungal infection is considered, biopsies should be pursued as soon as possible and immunomodulatory medications such as ibrutinib should be held, as was done in our case. While our patient was immunocompromised for several reasons, it is notable that she started

	ibrutinib four weeks prior to admission. Several case reports have demonstrated a link between ibrutinib and the development of disseminated aspergillosis, in particular2,3. This case adds to the emerging examples of patients starting this medication and subsequently developed this highly fatal infection. More broadly, it also demonstrates the importance of keeping atypical infections on the differential for the growing number of patients on immunosuppressive medications.
Alex Liu Dr. Rachel Suen	*Where are the Platelets? A Case of TTP in a Liver Transplant Recipient* Introduction: Thrombocytopenic Purpura (TTP) is a condition with significant morbidity and mortality. Early recognition is critical to successful treatment. Here, we present a case of TTP preceded by immune thrombocytopenic purpura (ITP). Case Presentation: The patient is a 46-year-old male with a past medical history of ulcerative colitis/primary sclerosing cholangitis status post liver transplant with graft failure, post-transplant lymphoproliferative disease status post right hemicolectomy and rituximab-based chemotherapy and ITP who presented with acute on chronic thrombocytopenia. The patient has a history of chronic thrombocytopenia, with a baseline platelet count of 100k. He initially presented to the hospital with a platelet count of 3k, unresponsive to transfusion. Peripheral smear did not show evidence of schistocytes. The patient was diagnosed with ITP and started on steroids and eltrombopag. His platelets improved to 100k. However, eltrombopag was discontinued due to hyperbilirubinemia. Subsequently, the patient's platelets declined to 20K. Several days later, the patient presented with fatigue, lethargy, and weakness. At an outpatient blood draw, the patient's platelet count was 6k. The patient was then directly admitted to the hospital. Exam was significant for hepatosplenomegaly. The patient also had a hemoglobin of 9.1 and a normal white count. Basic metabolic panel was significant for a Na of 126 and creatinine of 1.84 (baseline 1.1). Platelet smear showed mild schistocytes. PLASMIC score was 4. The patient was continued on prednisone for presumed ITP treatment failure. However, the patient's platelets did not improve, and his kidney function continued to worsen. Further testing showed a PT of 16.3, PTT of 39, d-dimer of 1797, fibrinogen of 277, and coagulation factor activity levels were reduced. ADAMTS13 was undetectable, and inhibitor screen was positive. We diagnosed the patient with immune mediated TTP and started the patient on plasma exchange, increased steroid dosing, and initiated caplacizumab. The patient's creatinine improved but his platelets did not. We planned on initiating rituximab, but the patient died after a GI hemorrhage. Discussion: We present a case of TTP following ITP, despite a PLASMIC score showing a low probability of TTP. TTP classically presents with a pentad of microangiopathic hemolytic anemia, thrombocytopenia, fever, kidney dysfunction, and neurological findings. However, the full pentad is rare and only occurs in less than 5% of cases. The PLASMIC score is helpful in ruling out TTP, but a high index of suspicion is required due to its morbid nature. Immune mediated TTP is due to decreased ADAMTS13 activity, usually in the setting of an antibody inhibitor to ADAMTS13. The mainstay of treatment is with plasma exchange and corticosteroids. Refractory cases can be treated with rituximab. If the disease is severe, caplacizumab can be

	initiated.
Tyler Loomer Dr. Amy Holbrook	*I've got Nocardia on the Brain* Introduction: Nocardiosis is typically regarded as an opportunistic infection, but approximately one-third of infected patients are immunocompetent. Two important characteristics of nocardiosis are its ability to disseminate to virtually any organ, particularly the central nervous system, and its tendency to relapse or progress despite appropriate therapy. Most classically, it effects the pulmonary system, followed by central nervous system and cutaneous manifestations. CNS norcardiosis can present with symptoms suggesting a mass lesion without any symptoms typically associated with infection. In such patients, nocardial brain abscess may be erroneously diagnosed as a primary or metastatic neoplasm prior to biopsy, as illustrated by this case. Case Presentation: 75-year-old male admitted in November 2021 with new onset left upper extremity weakness. Past medical history was significant for adenocarcinoma of the lung diagnosed in June 2021 via lymph node biopsy. He was status-post stereotactic brain radiation for known secondary brain metastases (right frontal lobe and right cerebellum) which were felt to be causing left lower extremity paralysis. He was receiving palliative chemoimmunotherapy. MRI on admission showed increase in size of previously demonstrated metastatic lesion in the right frontal lobe, with moderate increase in surrounding vasogenic edema. Interestingly, the previously known cerebellar lesion was not visualized on this repeat MRI; which was felt to be secondary to motion artifact. Labs largely unremarkable. No infectious or B-symptoms upon questioning. Oncology felt that MRI findings were likely due to post-radiation changes and not true progression of disease. Dexamethasone was increased, however he had minimal improvement in symptoms, thus he was started on bevacizumab prior to discharge for the possibility that the changes did represent worsening metastatic disease. Repeat MRI in December 2021 demonstrated significant increase in size of the right frontal lobe mass and recurrent lobulated enhancing lesion within the right central cerebellar hemisphere. He underwent second round of stereotactic radiosurgery directed to the right cerebellar lesion on 12/23/2021. Subsequent MRI demonstrated a cystic portion of the right frontal lobe lesion, in which neurosurgery recommended placement of Ommaya catheter which would allow for subsequent aspiration if needed. Unfortunately, the patient had a large generalized seizure on 1/25/2022, thus surgery was expedited. Patient was admitted on 1/26/2022 and was supposed to undergo cyst decompression and placement of a right frontal Ommaya reservoir on 1/28/2022. However, intra-operatively he was discovered to have thick purulent "yellow pudding like" material with aspiration, thus the Ommaya device was not placed. Gram stain demonstrated 4+ branching gram-positive bacilli, most consistent with Nocardia. Repeat procedures completed on both the frontal and cerebellar "metastatic mass" lesions demonstrated multiple abscesses and no true solid metastatic lesions. Nocardiosis was confirmed with culture data. Chemotherapy was held and antibiotics were started and continued for extended duration.
Ian Lorang	*Keeping Cards Close to the Chest: Cardiac Amyloid's Poker Face* Introduction: Cardiac amyloid (CA) pathogenesis involves infiltration of misfolded fibers into the myocardial extracellular space (1). There are two main types of amyloid fiber involved in CA (1). Light Chain amyloidosis (AL) is the more common of the two subtypes (1). Transthyretin amyloidosis

	(aTTR), either wild type (wtATTR) or hereditary (haTTR), is due to a misfolded liver protein called transthyretin. A Mayo Clinic study in 1992 found AL amyloid to be present in 2-3/1 million patients (2). That was 30 years ago. Upon my literature review, the true prevalence of CA remains widely unknown. Case Presentation: Our patient is a 76 y/o female with past medical history of hypothyroidism, chronic kidney disease, insulin dependent diabetes, and breast cancer status-post chemo/radiation who developed systolic heart failure 6 years prior to admission. Etiology at the time was unclear but there was suspicion for chemotherapy induced cardiomyopathy. An incidental finding on chest CT suggested possible pulmonary amyloid; therefore, diagnostic testing for amyloid was pursued including serum free light chains, fat pad biopsy, and hematology consultation, all of which were negative or reassuring. The following year, the patient presented in acute HF and was found to have new atrial fibrillation as well as an ejection fraction (EF) of 40% with abnormal left ventricular longitudinal strain. She was started on directed therapies with losartan, metoprolol, furosemide, and apixaban and her EF recovered to >60%. The next 6 years she did not experience a HF exacerbation and had stable echo findings but recently was admitted with acute decompensated HF symptoms. Echo revealed EF of 28%, concentric left ventricle hypertrophy, and longitudinal strain with apical sparing. A right heart catheterization with endomyocardial biopsy confirmed amyloid deposits. Bone marrow biopsy was negative, confirming aTTR. She was diuresed aggressively, discharged, and is now awaiting CA and hepatology specialty consultation to discuss treatment options. Conclusion: This case highlights several important points. Clinicians must consider amyloidosis as a potential cause of heart failure, be cognizant of classic imaging clues (e.g. "longitudinal strain with apical sparing", which is 93% sensitive for CA) (3), and look for associated systemic organ involvement. Once the diagnosis is confirmed, the subtype of CA must next be determined to consider targeted treatment options and appropriately counsel patients on prognosis. And most importantly, this case outlines how elusive the diagnosis can be. Appropriate diagnostic steps were taken early in this patient's workup when CA clues were identified, however both negative test results and a recovered EF in response to targeted HF therapies, atypical in CA, likely contributed to delayed diagnosis. Future studies to determine current CA prevalence, screening recommendations for targeted populations, and appropriate diagnostic strategies are needed to help clinicians diagnose CA as early in the course as possible.
Mackenzie Maberry Dr. Dhurv Sarma Dr. Jack McHugh	*A Case of Yellow Nail Syndrome* Introduction: First described in 19271, yellow nail syndrome (YNS) is a rare idiopathic disorder that is characterized by a triad of hard, yellow, dystrophic nails, lymphedema, and respiratory or intrathoracic disease. Early recognition that this diverse constellation of findings may represent a single clinical syndrome is important to ensure avoidance of inappropriate treatments. Case Presentation: A 72-year-old male with a past medical history significant for hyperlipidemia presented for a second opinion regarding management of bilateral pleural effusions. Four years prior to presentation, the patient developed dyspnea on exertion that was associated with a dry cough. Three years prior to presentation he

	developed recurrent right-sided transudative pleural effusions which were managed with Prednisone 10 mg daily and monthly thoracenteses for alleviation of dyspnea. Two years prior to presentation he developed lymphedema in the lower extremities and thickened yellow fingernails. Unfortunately, his pulmonary disease continued to progress, and he developed a left-sided pneumothorax and right hydropneumothorax, prompting presentation to our institution. Examination at time of admission was notable for bilateral non-pitting edema of the lower extremities with positive Stemmer's sign. The toenails exhibited xanthonychia with loss of the lunulae and cuticles. For management of recurrent pleural effusions, the patient underwent thoracotomy, decortication, and mechanical pleurodesis. Lymphedema was managed with lower extremity exercises and application of compression stockings. At time of discharge, it was recommended that the patient taper off prednisone. Discussion: Yellow nail syndrome is a very rare acquired condition of unknown etiology characterized by the presence of two of the following: (1) hard, yellow, dystrophic nails, (2) lymphedema, and (3) pulmonary or intrathoracic disease. It almost always occurs after 50 years of age, and has been observed in association with diseases implicating the lymphatic system, autoimmune diseases and cancers. In addition to xanthonychia, the nails typically become thickened with a reduction in longitudinal growth. These findings are often mistaken for onychomycosis; ironically, fluconazole use has been associated with improvement in nail appearance, and it is hypothesized that azole therapy may stimulate linear nail growth. Involvement of the respiratory tract occurs in >50% of patients and can lead to significant morbidity. Chronic cough and sinusitis may progress to recurrent pleural effusions as in this case. Other lung findings include bronchiectasis, recurrent pneumonia, and pulmonary fibrosis. Treatment is symptomatic as in this case; there is no role for corticosteroids. Lymphedema is present in 30-80% of YNS patients and is the primary presenting symptom of YNS in one third of cases. Lymphedema is managed with low-stretch bandages and exercises followed by application of high-pressure elastic garments. Further research is required to better understand and treat this very rare syndrome. However, early recognition of the constellation of symptoms is important to reduce exposure to unnecessary and potentially harmful treatments.
Sanjoyita Mallick	*HFrEF Requiring Intravenous Inotropic Therapy via PICC in the Setting of MRSA Bacteremia* Introduction: MRSA bacteremia and HFrEF requiring continuous Milrinone infusion are associated with high morbidity. This case describes a patient developing MRSA bacteremia where the source of the infection was the PICC line. Per guidelines, the patient requires a line holiday in order to appropriately clear the infection. This management, however, is complicated due to the fact that the patient requires continuous infusion of Milrinone via the infected PICC line. Case Description: A 27-year-old woman with a past medical history significant for biventricular HFrEF (~ 10%) on milrinone infusion, LV thrombus on Warfarin, and sacral decubitus ulcers was admitted for the

	management of chest pain. The course was complicated with RUE DVT from the PICC line, that was likely present on admission, and PICC line associated MRSA bacteremia. Infectious Disease was consulted. Patient was started on Vancomycin and Cefazolin, later switched to Daptomycin. PICC line was not initially removed due to the Milrinone Infusion. Repeat blood cultures after 48 hours had not cleared the infection. After discussion with Infectious Disease, a plan was made to attempt a line holiday. We established two peripheral IV access and infused the Milrinone through one of them with fluids (25cc/hr). The second IV was left in place as a backup. The milrinone infusion via the PICC line was stopped. We checked a lactate every four hours for the first 24 hours. The following day, after ensuring there was no rise in lactate, the PICC line was removed. A PICC line was reinserted on the left upper extremity six days later, after 48 hours of negative blood cultures. The patient was later discharged with plans to continue Daptomycin infusion for a total of 4 weeks. Discussion: A prospective study involving 324 patients with catheter related S. Aureus bacteremia, failure to remove the line was associated with an increased risk of hematogenous spread and higher relapse rate in patients who did not have their line removed/exchanged within the first three days. 12.7% of the patients who did not have a line holiday were found to relapse versus 4.7% of the patients who did have a line holiday. This case illustrates the significance a line holiday plays in clearing a line associated bacteremia. It also demonstrates a unique alternative to infusing Milrinone for a patient with ambulatory cardiogenic shock.
Jasraj Marjara Dr. Elyse Conley Dr. Mitchell Padkins	*Post-Myocardial Infarction Ventricular Septal Defect After Re-perfusion Therapy* Introduction: Post-infarct ventricular septal defects (PI-VSD) are a rare but deadly mechanical complication of acute myocardial infarction (AMI) since the advent of percutaneous coronary intervention (PCI).1,2 Despite occurring in only 0.15-0.3% of AMI cases, the in-hospital mortality rate of patients with PI-VSD is disproportionately higher than other patients with cardiogenic shock, reaching as high as 60% with treatment and 80% in untreated patients. Caused primarily by postinfarction cardiomyocyte inflammatory changes and tissue friability often in the setting of prolonged ischemia or delayed reperfusion, patients with PI-VSDs often present with recurrence of their initial anginal symptoms, arrhythmias or hemodynamic instability most often between 1-5 days after AMI. Medical stabilization followed by surgical repair is preferred to maximize surgical success, though exact timing of surgical intervention depends upon a multitude of factors best assessed by a multidisciplinary heart team. Case Presentation: In this report, we describe the treatment of a patient with a large PI-VSD of the basal inferior septum who presented with inferior STEMI 2 weeks after initial development of ischemic symptoms. After undergoing emergent coronary angiography demonstrating multivessel disease with chronic total occlusion of the mid right coronary artery and receiving drug-eluting stents to the left circumflex and left anterior descending arteries, patient was transferred to our institution where the presence of a large irregular basal-inferior ventricular septal defect measuring 2.9 cm2 in cross-sectional area was confirmed by echocardiography. Marked fifth generation high sensitivity cardiac troponin T levels of 1377 ng/L on arrival, and 1322 ng/L at 2 and 6 hours were noted and serial ECGs demonstrated inferior Q waves which were unchanged. Lactate levels were

	within normal limits. Initial cardiac index by echocardiography was low at 2.2, however, prompting initiation of dobutamine and placement of an IABP with subsequent improvement. Cardiovascular surgery was consulted and recommended delayed surgical intervention given his stability with medical support. Inotrope-induced atrial fibrillation subsequently developed with successful reversion to sinus rhythm using amiodarone and digoxin, and the patient underwent successful surgical repair of his VSD and patch repair of his LV apical aneurysm. Post-bypass echocardiogram demonstrated an LVEF of 50% with a residual VSD which would likely resolve spontaneously. He was discharged from the hospital on day 17 and continued to do well, participating vigorously in cardiac rehabilitation without interval readmission or procedures 30 days after discharge. Conclusion: This case demonstrates how the use of IABP as a first-line mechanical circulatory support device can augment the medical stabilization of patients with PI-VSD and act as a bridge to surgery even with an exceptionally late presentation to care. A nuanced multidisciplinary approach is needed to best manage this rare complication of AMI, and prompt recognition and referral is essential to maximizing surgical success.
Dillon Medlock Dr. Harikrishna Halaharvi Dr. David Thomas Dr. Kelsey Angell	*An Expedited Diagnosis of Stiff Person Syndrome: A Rare Autoimmune CNS Disorder* Introduction: Stiff person syndrome (SPS) is a progressive CNS disorder due to acquired autoimmunity against glutamic acid decarboxylase (GAD): an enzyme involved in synthesis of gamma-aminobutyric acid (GABA), an inhibitory neurotransmitter. In classic SPS, loss of GABA function leads to myalgias, muscle rigidity, and muscle spasms. Due to its rarity, episodic symptomology and unremarkable initial workup, diagnosis is significantly delayed from symptom onset and relies on a broad work-up including anti-GAD antibodies, which are positive in 70-80% of SPS cases. Benzodiazepines are the first line therapy, but baclofen, intravenous immune globulin therapy (IVIG) and immunomodulators may be used in individuals without a response to benzodiazepines. Unfortunately, SPS patients experience progressive functional decline, but progression can be slowed with early diagnosis and treatment. Case Description: A 43 year-old woman with a history of chronic anemia and hypothyroidism following ablation for Graves Disease presented with several months of fluctuating lower extremity weakness, dizziness, blurred vision and diffuse muscle pain. Physical exam revealed exaggerated lumbar lordosis, shuffling gait with short steppage, delays in transfer from seated to standing position and diffuse hyperreflexia. No acute or chronic pathology was found on CTA of the head and neck, thoracic spine MRI or lumbar spine MRI. Urine drug screen revealed prescribed oxycodone. Infectious work-up including urinalysis, lumbar puncture and RPR were unremarkable. Labs were notable for an elevated erythrocyte sedimentation rate, folate deficiency and a positive ANA with a 1:320 titer and speckled pattern. On hospital day 7, a GAD-65 antibody also returned positive, suggesting SPS. The patient was started on diazepam and IVIG with significant improvement and eventually discharged home with oral diazepam. Three months later, she returned with worsening symptoms after self-discontinuation of diazepam due to intolerable sedation. During this admission, she presented with new tactile and auditory hallucinations, photosensitivity, phonosensitivity and gait instability. Exam revealed hyperreflexia, upper extremity myoclonus, opsoclonus, and truncal and lower extremity rigidity. She immediately

	improved following plasmapheresis, IV steroids and diazepam and ultimately discharged home with a steroid taper and diazepam. She is currently undergoing Rituximab therapy and long term steroid taper through outpatient Neurology. Discussion/Learning Points: Stiff person syndrome is an insidious, rare disorder with an often several year delay to diagnosis. It is frequently misdiagnosed as a psychiatric or a functional neurological disorder. Recognizing the progressive neurologic symptoms of SPS and a broad autoimmune work-up including GAD65 antibody were key to diagnosis within the first year of symptom onset. Benzodiazepines are crucial first line therapy in SPS management, but maintenance therapy is critical: a fact underscored by the patient's return following self-discontinuation of diazepam. Additional education regarding maintenance therapy and side effects may have prevented this relapse and must be emphasized in the management of SPS.
Abdilahi Ali Mohamoud Dr. Simranjit Singh Kahlon	*Syncope: A Sole Presenting Symptom of NSTEMI in an Elderly Patient* Introduction: Syncope is a transient loss of consciousness and postural tone caused by decreased cerebral blood flow. It represents 1-3.5% of all ED visits and 6% of all hospital admissions in the US. The prognosis of syncope varies according to etiology, with 1-year mortality estimates ranging from 0% among patients with vasovagal syncope to 30% in patients with cardiac syncope. Cardiac syncope is due to a defect in either structural or electrical, which prevents the generation of enough cardiac output to perfuse the brain adequately. Case Presentation: A 79-year-old male with no known cardiac history presented to the ED following a motor vehicle accident due to a syncopal episode while driving. He reported previously feeling lightheaded but denied any history of prior syncope, chest pain, dyspnea, and palpitations. He had troponin of 141 ng/L (Reference: < 34 ng/L) and EKG showed sinus bradycardia and first-degree atrioventricular block. Given possible cardiac etiology for syncope, he was admitted for further workup. On day 1 of hospitalization, his troponin progressively trended up to 10,000s and subsequent EKGs showed non-specific ST-elevation in the septal precordial leads that could be suggestive of possible ischemia. Telemetry also showed ectopy and a 3-second sinus pause. TTE demonstrated regional wall-motion abnormalities in the septum and inferolateral with an ejection fraction of 57%. Cardiac angiography showed severe 100% occlusion of the proximal RCA with grade 1 R-R and L-R collaterals and 70-80% OM2 occlusion. PCI was not done at the time as the lesions appeared to be chronic with apparent collateral vasculature. Cardiac MRI was done to further evaluate the structure of the RCA territory. It showed normal LV function with subtle wall-motion abnormality and small foci of near transmural scar in the inferior septum. The majority of the myocardium, including RCA territory, was viable. The troponin continued to rise with a peak of 18,600 on hospitalization day 2. Given these findings, he was started on DAPT and heparin. A repeat angiogram revealed recanalization of proximal RCA with 95% stenosis and ongoing OM occlusion. PCI was done at this time with stents to proximal RCA and OM2. A final diagnosis of NSTEMI was made. On telemetry, patient had few sinus pauses that might be related to his RCA occlusion or underlying conduction disease. Electrophysiology was consulted and the decision was made for long-term rhythm monitoring with a loop recorder which was implanted before discharge.

	Conclusion: Older patients more commonly have an atypical presentation of acute coronary syndrome including syncope being the sole presenting symptom. This case illustrates the difficulty in differentiating syncope due to NSTEMI in elderly patients from cardiac arrhythmia etiology. A comprehensive and multidisciplinary approach is required for adequate diagnosis and prognostication of syncope in the elderly.
Shirin Nour Dr. Jack McHugh	*Our Patient Still has a Fever!* Introduction: Fever of unknown origin represents a diagnostic challenge for the internist. The differential is broad and includes infectious, malignant, and inflammatory conditions. We review the presentation of Adult-Onset Still's Disease (AOSD), a disease that can cause a constellation of symptoms including daily fevers. Case description A 34-year-old woman was admitted to the hospital for evaluation of fevers, pharyngitis, and myalgias. She reported a one-week history of fevers and sore throat. She had been experiencing additional symptoms for the last 4 months including non-pruritic rash, diffuse myalgias, and migratory polyarthralgias. Her medical history was notable for obesity with a BMI of 39kg/m^2, depression, and anxiety. Patient denied any sick contacts, recent outdoor activities, or travel. On admission, she was febrile with temperature of 38.4°C. Physical examination was notable for cervical lymphadenopathy, a pink, maculopapular rash on the trunk and upper extremities, and evidence of synovitis involving the bilateral knee, hip, and shoulder joints. Laboratory studies revealed white cell count (WCC) 22.4x10/9(L) with 80% neutrophils, erythrocyte sedimentation rate 62mm/hr, C-reactive protein (CRP) 196 mg/L, ferritin 241mcg/L, and normal liver enzymes. Infectious workup was negative and included blood cultures and testing for Lyme disease, syphilis, chlamydia, gonorrhea, tuberculosis, HIV, CMV, EBV, parvovirus B19, influenza, and SARS-CoV-2. Arthrocentesis of the right knee demonstrated 25,775 total nucleated cells, with 66% neutrophils. Rheumatologic testing was negative and included anti-nuclear antibody (ANA) with HEp2 substrate, human leukocyte antigen B27, and anti-cyclic citrullinated peptide antibodies. Transthoracic echocardiogram did not show valvular lesions. Computed tomography of the abdomen revealed borderline splenomegaly with spleen measuring 13.5 cm. A biopsy of the skin rash showed dermal perivascular and interstitial mixed dermal inflammation with numerous neutrophils. During the one-week hospitalization, the patient had daily fevers with temperatures ranging from 39°C to 39.8°C. She was started on scheduled non-steroidal anti-inflammatory medication and 30 mg of prednisone for treatment of presumed AOSD, with rapid improvement in her arthralgias/myalgias and no further fevers. During prednisone taper, the patient's symptoms returned. She was initiated on anakinra 100mg injection daily. Her symptoms resolved and CRP/ESR normalized. Discussion: AOSD should be in the differential for fever of unknown origin. No definitive diagnostic test is available, however there are clinical criteria that can aid in the diagnosis. Our patient didn't have an elevated ferritin or liver enzymes, but she met the Yamaguchi criteria which requires the presence of five features, two of which must be major criteria. Major criteria include fever of at least 39°C lasting one week, arthralgias lasting two weeks

	or longer, nonpruritic macular or maculopapular rash, leukocytosis of 10,000x10(9)/L or greater, with at least 80 percent granulocytes. Minor criteria include sore throat, lymphadenopathy, hepatomegaly, splenomegaly, abnormal liver function studies, and negative tests for ANA and rheumatoid factor.
Jordan Nunnelee Dr. John Bretzman	*Cavitary Lung Lesions: A Broad Differential* Introduction: Pulmonary cavitary lesions can be caused by a wide variety of pathology. We present a case of pulmonary cavitary disease that led to broad workup and ultimately found a disease infrequently encountered in the United States. Case Description: A 53 year-old gentleman with no known medical history presented with a 2.5 month history of unintentional weight loss, night sweats, chills, and productive cough. His symptoms began shortly after returning from a three-month trip to Senegal. He denied hemoptysis, gastrointestinal symptoms, musculoskeletal pain, neurologic symptoms, chest pain, dyspnea, or rashes. He smoked 1.5 packs of cigarettes per day for decades. He was in a monogamous heterosexual relationship, did not use IV drugs, and had no other relevant exposure history. He was admitted to the hospital, and was febrile to 39.1 C, tachycardic, and tachypneic with normal blood pressures. His exam was significant for cachexia, coarse rales in left lobes more than right, with stridor in anterior lung fields on inspiration. CT angiogram of the chest was completed and negative for pulmonary embolism. However, he did have extensive upper-lung-predominant cavitary lesions and tree-in-bud micronodularity in the left greater than right lobes. He was started on broad spectrum antibiotics and placed in a negative pressure room. Our differential for his cavitary lesions included infectious, auto-inflammatory, and malignant diseases. Considering his recent travel history, evaluation for tuberculosis with three separate sputum samples including acid fast staining, mycobacterial culture, and tuberculosis PCR were obtained. Blood was sent for mycobacterial and routine bacterial/fungal culture. We investigated histoplasma, blastomyces, and cryptococcus with urine and serum antigen/antibody testing. We considered MRSA necrotizing pneumonia with a MRSA swab. Due to lack of IV drug use history and absence of murmur on exam, we deemed endocarditis causing septic emboli and necrosis less likely. We considered malignancy due to his smoking history. ANCA associated vasculitis was also on the differential, for which MPO and PR3 antibody testing were obtained Ultimately, his sputum stain was positive for acid-fast bacilli, and tuberculosis PCR was positive without rifampin resistant mutations. He was initiated on rifampin, isoniazid, pyrazinamide, and ethambutol (RIPE) therapy for at least 6 months. Broad-spectrum antibiotics were discontinued. Discussion: This case illustrates the diagnosis of a disease with low incidence/prevalence in our patient population, and the importance of keeping a broad differential. While tuberculosis is a common cause of cavitary lung lesions world-wide, there are many other diseases that present with cavitary lung lesions, especially in the Midwest. These include blastomycosis, histoplasmosis, non-tuberculous mycobacteria, MRSA, actinomyces, coccidiomycosis, cryptococcus, pulmonary primary and

	secondary malignancies, lymphoma, and ANCA associated vasculitis Patients with nonspecific symptoms should have a broad workup so as not to delay treatment and to provide the best patient care.
Anshula Prasad Dr. Dillon Medlock	*Spontaneous Hemorrhagic Pericardial and Pleural Effusions - A Kale Story* Introduction: In nonvalvular atrial fibrillation, options for anticoagulation include direct-acting oral anticoagulants aka DOACs and warfarin. The latter includes frequent lab draws and issues with drug interactions. Leafy, green vegetables change the activity of warfarin due to the presence of vitamin K. A stable diet is important to allow for stable warfarin dosing. Here we present a patient who was found to have a hemorrhagic pericardial and pleural effusion in the context of labile dietary habits on warfarin. Case Description: A 74 year-old male with a history of atrial fibrillation presented for three weeks of progressive fatigue. Symptoms included pleurisy, dyspnea, anorexia, weakness, and melena which led him to stop taking warfarin. He reported dietary changes in the recent past such as a ketogenic diet and incorporating more vegetables. Due to significant kale intake, he was on a high warfarin dose. There was a history of dietary variability in anticoagulation notes and he told ED staff he had cut kale out of his diet prior to symptom onset. Physical exam found elevated JVP, atrial fibrillation, bilateral pedal edema, and diminished breath sounds at the left lung base. His initial lab work was notable for leukocytosis, acute anemia, INR 2.7, and elevated BNP. A CTA chest showed a large pericardial effusion, moderate left pleural effusion, cardiomegaly, and intrahepatic reflux of contrast. The effusions were presumed hemorrhagic given a therapeutic INR despite being off warfarin for 4-5 days, so he received vitamin K. A pericardiocentesis and thoracentesis confirmed hemorrhagic effusions. Inflammatory markers were high, so colchicine was started for potential effusive constrictive pericarditis. Due to dietary lability, the decision was made to transition to apixaban. He was discharged after a few days without melena or reaccumulation of the effusions. Discussion/Learning Points: Pericardial effusions have a range of manifestations from incidental to cardiac tamponade. Hemorrhagic pericardial and pleural effusions are rare, but potentially life-threatening complications of warfarin therapy. Risk of bleeding should be discussed regarding anticoagulation with consideration for falls, past bleeding history, coagulopathy, and financial accessibility. While direct oral anticoagulants are often preferred for nonvalvular atrial fibrillation due to convenience and safety profile, they remain inaccessible for some due to kidney dysfunction and finances. Warfarin can increase the risk of bleeding into any site; a large study cited a 3.9% total risk of bleeding on warfarin, with GI tract being the most common site and rare mention of hemopericardium. However, there are other case reports documenting hemopericardium and moving forward it may be critical for providers to consider dietary variability including ketogenic diet as an independent risk factor given for DOAC consideration.
Pavithra Ramakrishnan Dr. Nuttavut Sumransub Dr. Gregory Vercellotti	*New Report of Cutaneous Small Vessel Vasculitis to Ferumoxytol Infusion in Chronic Granulomatous Disease Patient* Introduction: Chronic granulomatous disease (CGD) is a condition characterized by phagocytic oxidative burst defect leading to recurrent infection and granuloma formation. Most commonly reported autoimmune

	disorders associated with CGD include systemic lupus erythematosus (SLE) and juvenile idiopathic arthritis (JIA). In this report, we are the first group to describe a patient who developed cutaneous small vessel vasculitis (CSVV) after ferumoxytol infusion highlighting the intricacy of inflammatory response in the patient with CGD. Case summary: 36 year old female with past medical history of CGD and bronchiectasis secondary to recurrent pulmonary infection with multidrug resistant Aspergillus niger. This patient presented with upper and lower extremity rash concentrated around bilateral elbow 5 days after IV infusion of Feraheme (ferumoxytol) for iron deficiency anemia at bilateral antecubital fossa. Patient reported acute worsening of chronic joint pain in conjunction with swelling of bilateral elbow 3 days after infusion. This was resolved in < 24 hours with aspirin and diphenhydramine. The following day the patient noted pinpoint red spots in bilateral upper extremities which increased in number, coalesced and eventually developed similar rashes in lower extremities. Patient denies any other systemic symptoms such as fever or viral prodrome. No documented allergy to IV or oral iron supplements. No recent change in the medications except for IV iron infusion as described above. Broad autoimmune and viral/bacterial/fungal workup were non-reactive. Skin punch biopsy showed leukocytoclastic vasculitis (LCV) with subtle IgA, C3 and fibrinogen granular deposition within the superficial blood vessel wall. Patient was discharged on topical treatments with triamcinolone and a 5 day course of systemic steroids with significant improvement in rashes and symptoms of joint pain at 1 week follow-up. Discussion: Patient's clinical picture as well as pathological findings are consistent with IgA mediated leukocytoclastic vasculitis which is characterized by IgA deposition accompanied with neutrophil recruitment and inflammation. The condition is a reactive process and occurs 5-10 days after exposure to the offending culprit. Typically, both proteolytic enzymes and reactive oxygen species(ROS) can be responsible for local and systemic damage, though in this patient ROS would be unlikely given her history of CGD. This is likely the reason behind the cutaneous limited phenotype of presentation. This is also the first case report of CSVV as a reaction to ferumoxytol (Iron oxide coated polyglucose sorbitol carboxymethyl ether) infusion. This carbohydrate compound is different from previously reported CSVV related to iron dextran which is a liquid complex of ferric hydroxide and dextran.
Lucas Ramsey Dr. Amy Holbrook	*An Ovarian Source of Cor Pulmonale* Introduction: Patients with BRCA mutation are at increased risk for ovarian and breast cancers among others. Prophylactic bilateral salpingo-oophorectomy (BSO) is an option for patients to reduce the risk of ovarian cancer by up to 96%. We report an unusual presentation of metastatic ovarian cancer in a BRCA+ patient who had undergone prophylactic BSO. Case Presentation: A 42 year old woman presented to her outpatient provider with 6 weeks of progressive dyspnea on exertion. Her history was pertinent for BRCA1 mutation s/p prophylactic bilateral salgpingo-oophorectomy. Echo revealed severely reduced RV function and severely increased pulmonary artery pressure, and she was admitted to the hospital for concerns of pulmonary hypertension and cor pulmonale. On admission, she was requiring 4 L supplemental O2 to maintain saturations. Otherwise vitals unremarkable. Labs were notable positive troponin and new

	thrombocytopenia, CT PE and abdomen pelvis revealed no evidence of pulmonary embolus but was significant for mediastinal, axillary, retroperitoneal, and mesenteric lymphadenopathy. A few small nodules were noted in the right middle lobe, thought to be benign. She was up to date on all screening mammograms and MRIs. Recent colonoscopy was unremarkable. CA-125 later returned at 2000. Oncology, cardiology, and pulmonology were consulted. Right heart catheterization revealed severe pre-capillary pulmonary hypertension and negative vasodilator challenge; V/Q scan to evaluate for chronic thromboembolic pulmonary hypertension was pursued and resulted as low probability. She then underwent biopsy of a para aortic lymph node. She became progressively more short of breath while awaiting biopsy results and was placed on digoxin and sildenafil without improvement in oxygenation. Pathology returned suggestive of a high grade metastatic ovarian cancer. Gynecologic oncology was consulted and suspected her course was consistent with lymphangitic spread. The patient elected to start neoadjuvant chemotherapy with carboplatin and paclitaxel. Given her tenuous cardiac function, she elected to change code status to DNR prior to chemotherapy. She became rapidly hypoxic after paclitaxel and did not recover despite BiPAP, epoprostenol, and steroids. She passed away with family at bedside, 7 days after admission. Conclusion: The risk of ovarian and breast cancer is reduced for BRCA+ patients who undergo BSO. Despite its efficacy, providers should still consider these cancers in patients who have undergone the procedure. Patients should also be made aware that BSO does not completely mitigate the risk for ovarian cancer, even if pathology is reassuring at the time of the procedure.
Carolina Rojas-Salvador Dr. Bellony Nzemenoh Dr. Moaiad Salous Dr. Meghan Rothenberger	*Atypical Disseminated Cutaneous Herpes Zoster in a Young Immunocompetent Man after COVID-19 Infection* Introduction: Disseminated cutaneous herpes zoster (DCHZ) is the presence of more than 20 maculovesicular lesions outside the primary and adjacent dermatomes that results from the reactivation of varicella-zoster virus (VZV) in immunosuppressed patients. Here we present a case of DCHZ in a young immunocompetent man who developed hepatitis and aseptic meningitis after COVID-19 infection. Case Presentation: A 27-year-old man with a history of recently treated genital Chlamydia developed painful vesicles initially involving his right anterior hip, which spread to his entire trunk, extremities, head, scalp, and palms. Soles and genitals were not involved. Fever, headaches, and fatigue were also present. Aside from a childhood history of chickenpox at seven years old, an uncompleted chickenpox vaccination, and mild COVID-19 a month before admission, he had no history of immunosuppression, chronic illness, or malignancy. On physical examination, disseminated fluid-filled vesicles (>30) were found all over his body; ulcers were present on the gingiva and hard palate along with periorbital edema. His laboratory workup was notable for elevated aspartate and alanine aminotransferase (220 U/L and 113 U/L, respectively), C-reactive protein (120 mg/L), creatinine (1.49 mg/dL), and thrombocytopenia (110 uL). Complete blood count, biochemistry, urinalysis, blood cultures, and chest x-ray were normal. PCR performed on vesicular fluid and skin biopsy was positive for VZV DNA. CSF sampling was consistent with aseptic meningitis, with a positive PCR for VZV. Further workup including testing for HIV, HSV, HTLV 1-2 antibody, syphilis, hepatitis B and C, Chlamydia, Gonorrhea and

	immunoglobulins were unremarkable. However, flow cytometry revealed a low CD4/CD8 ratio of 0.27 (<1), a normal CD4 (788 cell/uL) and elevated CD8 counts (2096 cell/uL). Intravenous (IV) acyclovir was immediately started. The eruption of new vesicles ceased and symptoms improved after two days of IV therapy. He was discharged with oral valacyclovir to complete a 10-day course. Upon regular follow-up with Dermatology, he reported improved crusting of all skin lesions. Unfortunately, he did not attend an Immunology follow up. Discussion: Our patient presented with painful non-dermatomal disseminated vesicular lesions, a low CD4/CD8 ratio and overexpression of CD8 counts. The possibility of primary varicella was ruled out because the patient had a history of chickenpox as a child. This atypical presentation occurs almost exclusively with underlying immunodeficiency. Moreover, DCHZ-related complications such as hepatitis and meningitis in apparently immunocompetent, non-elderly patients are extremely rare. A predisposing condition such as a recent COVID-19 infection may have triggered an immune dysregulation in T-cell function, necessary for VZV reactivation. While studies have demonstrated associated COVID-19 overexpression of CD8, there is only few case reports of DCHZ within weeks of COVID-19. Conclusion: We present a case of DCHZ in a young man without apparent immunosuppression. COVID-19 may have played a role and triggered VZV cell-mediated immunity.
Lizbeth Rondon Rueda Dr. Roberto Fu	*Immunotherapies Adverse Effects: A Multidisciplinary Dilemma* Introduction: The use of immunotherapy in the form of checkpoint inhibitors is nothing short of a paradigm shift in the field of oncology. Encouraged by results from the treatment of melanoma, checkpoint inhibitors are now being applied to a wide range of malignancies with a vast network of clinical trials currently underway. It is necessary that clinicians become familiarized with these relatively new medications to identify and promptly manage associated toxicities. The clinical spectrum of toxicities is compiled under the umbrella term of immune related adverse events (irAEs) which can be intuitively correlated with these medications' mechanisms of action. We explore the heterogeneity with which irAEs manifest by examining an individual case in which multiple organ systems were affected simultaneously, translating into endocrine and rheumatological disease on the backdrop of treating a breast malignancy. Case presentation: A 49 yo female is diagnosed with triple negative invasive ductal carcinoma of the right breast, stage IIB, treated with neoadjuvant chemotherapy and immunotherapy with pembrolizumab. Her course is complicated by hypotension and malaise. She is found to have a low cortisol and a suppressed ACTH raising concern for hypophysitis presenting itself as adrenal insufficiency. She was started on hydrocortisone and Pembrolizumab was discontinued. 4-5 months later nodular liver lesions were noted on CT abdomen-pelvis. Biopsy of the lesions were described as non-caseating granulomas, which alongside axillary/inguinal lymphadenopathy, skin rash and subpleural nodules confirmed a diagnosis of Sarcoidosis. Both complications are thought to be irAEs and as such, a consequence of exposure to pembrolizumab. Later through her course, the cancer unfortunately progressed to stage IV. To this date, the patient continues to follow up with endocrinology and rheumatology, requiring treatment with steroids and DMARD for management of her irAEs along with difficult

	decisions about palliative chemotherapy which itself includes checkpoint inhibitors as options. Discussion: The use of immune checkpoint inhibitors will continue to increase, and in combination with other agents the patterns of irAEs are expected to become more complex. Conscientious assessment of medication exposures and recognition of associated toxicities is crucial to provide prompt treatment and reduce morbidity and mortality associated with these therapies. Identification and management of irAEs may require a complex multidisciplinary approach along with close monitoring and use of significant clinical resources as exemplified above. Prognosis and course of illness will be defined by the organ system that is affected. Toxicity is graded in severity per recent guidelines and discontinuation of these medications requires weighing the potential benefits and risks in each treatment scenario paired with a shared decision-making model. When there is exposure to these medications a high level of suspicion must be maintained that any new symptoms can be treatment related.
Bibek Saha Dr. Joshua Daum Dr. Thomas Beckman	*Malaria and Chikungunya Co-Infection in a Traveler Returning from Sudan* Introduction: Globalization and increased international travel have made the diagnosis of infectious diseases outside of their known endemic regions not uncommon. Vector-borne illnesses including chikungunya, dengue, and malaria, with common species such as Plasmodium (P.) falciparum, P.vivax, and P.ovale, are known to have shared endemic profiles predominantly in tropical regions such as Asia, Africa, and Central and South America. Similarly, all three diseases present as an acute febrile illness and may have other overlapping symptoms including fatigue, headache, nausea, and myalgias. Here we present a rare case of a returning traveler diagnosed with P.falciparum and chikungunya, with a possible third co-infection with P.vivax/ovale. Case Presentation: A 52-year-old male presented with a 2-day history of fever, chills, fatigue, diaphoresis, headache, chest pain, decreased appetite, abdominal pain, diffuse myalgias of the back, neck, and extremities, and brown urine. He denied arthralgias. He recently traveled to Sudan, without taking antimalarial prophylaxis. While in Sudan, he was bitten by mosquitos, and 3 weeks prior to presentation (PTP), he developed this same symptomology and was diagnosed with Malaria. He was treated with quinine and dexamethasone with symptom resolution 2 weeks PTP. On exam, the patient was initially afebrile (37.7°C) but subsequently developed a fever (38.9°C), tachycardic (110bpm), normotensive (112/69mmHg), tachypneic (21breaths/min) with an oxygen saturation of 99%. He had abdominal tenderness and diffuse back tenderness from the neck to the lumbar region. There was no scleral icterus or jaundice. Initial labs showed decreased hemoglobin (12.7) and platelets (81), but increased LDH (277) and total bilirubin (3.1, 2.7 being indirect bilirubin). Peripheral smear did not show schistocytes. Giemsa-stained blood smears was positive for P.falciparum infection (0.59% parasitemia). The patient was diagnosed with uncomplicated mild malaria, ID was consulted, and he was started on a 3-day course of Artemether-Lumefantrine. During the 3-day hospital course, the patient was mostly afebrile, with the parasitemia percentage down-trending (day2:0.46%; day3:0.33%). On day 3

	(patient had received 2/3 doses of Artemether-Lumefantrine), he felt well and was adamant about discharge. Just prior to discharge, the smear suggested a possible co-infection with P.vivax/ovale. Post-discharge, serologies of other vector-borne diseases was positive for chikungunya (IgM+, IgG+), and suggested a prior infection with dengue (IgG+, IgM-). Unfortunately, the patient did not follow-up with his PCP. Conclusion: Here we reported, to our knowledge, the 2nd case of P.falciparum and chikungunya co-infection diagnosed in the US, and possibly the first case of a triple infection with P.vivax/ovale. This case highlights that in a febrile patient with a recent travel history to endemic regions, a high clinical suspicion of vector-borne infectious diseases is warranted. Additionally, our case emphasizes the ever-expanding knowledge base that Physicians need to possess of infectious agents on a global scale.
Rachel Salz	*The Eyes Have It, At Any Stage: Considering Ocular Syphilis* Introduction: Known as the "great imitator," syphilis infrequently presents with ocular symptoms including anterior/posterior/panuveitis, interstitial keratitis, retinal vasculitis, and optic neuropathy. 1 This case aims to highlight consideration of syphilis, at any stage, as an etiology of visual acuity changes, especially in at-risk populations. Case Description: A 44-year-old male with history of HIV/AIDS (on anti-retroviral therapy) presented to an eye clinic with complaints of left visual field blurriness and photosensitivity. He had "hand motion" visual acuity of the left eye. On slit lamp examination, anterior chamber, iris, lens and anterior vitreous abnormalities were noted. He was diagnosed with posterior uveitis and prescribed antibiotic and steroid eye drops. He was re-evaluated 2 weeks later, at which time visual acuity and a repeat slit lamp exam were unchanged. Treponemal and non-treponemal testing were obtained and found to be positive. He was admitted for initiation of IV Penicillin G with plan for a 14-day course. Subsequent slit lamp examination on Day 11 of treatment demonstrated resolution of anterior chamber cells, anterior vitreous cells, and subsequent visual acuity examination demonstrated improvement to "counting fingers." Discussion: This case illustrates the importance of consideration of syphilis as a cause of vision loss in the outpatient setting. Prompt diagnosis is essential, as diagnosis beyond 4 weeks of ocular symptoms portends poorer outcomes and increased risk of asymptomatic recurrence.2 Given the accessibility and affordability of testing and the importance of early recognition and treatment, special consideration should be given in cases of vision changes in patients with risk factors for syphilis including high-risk sexual practices and IV drug use. References: 1 Parthopratim Dutta Majumder, Elizabeth J. Chen, Janika Shah, Dawn Ching Wen Ho, Jyotirmay Biswas, Leo See Yin, Vishali Gupta, Carlos Pavesio & Rupesh Agrawal (2019) Ocular Syphilis: An Update, Ocular Immunology and Inflammation, 27:1, 117-125, DOI: 10.1080/09273948.2017.1371765 2 Emmett T. Cunningham Jr, Chiara M. Eandi & Francesco Pichi (2014) Syphilitic Uveitis, Ocular Immunology and Inflammation, 22:1, 2-3, DOI: 10.3109/09273948.2014.883236

Joshua Samec Dr. Molly Wyman	*A Medication Reconciliation Lapse: A Case Report of Minocycline-Induced Acute Eosinophilic Pneumonitis* Introduction: Acute eosinophilic pneumonitis (AEP) is an uncommon diagnosis that was first described as a cause of acute respiratory failure in 1989. The etiology of this pathophysiology is unknown with most diagnoses of AEP either related to recent onset of smoking, inhaled antigens, or drug induced.1 Minocycline is a medication used to treat bacterial infections, acne vulgaris, and moderate to severe rosacea and has documented case reports of inducing AEP. This case report demonstrates a prolonged diagnosis of AEP after missing minocycline on a medication reconciliation upon admission for an atypical pneumonia presentation. Clinical Case: A 65-year-old female presented with a past medical history of extensive smoking history, atrial fibrillation, and recent diagnosis and completed treatment of community-acquired pneumonia with persistent symptoms of shortness of breath and dry cough. A computed tomography of the chest revealed multifocal ground glass opacities suspicious for atypical infectious etiology or inhalational lung diseases. Medications from the previous admission one week prior within the electronic medical record were reconciled but missed a new minocycline prescription from a private dermatology practice that was started one month prior. The patient was then treated with a broader spectrum of antibiotics for suspected atypical pneumonia and despite this infectious treatment, the symptoms of shortness of breath and dry cough continued to worsen. Fungal, bacterial, and viral testing continued to result negative prompting completion of a bronchial-alveolar lavage (BAL). The cell count and differential from the BAL showed 80% predominance of eosinophils. After the procedure, a bottle of minocycline was found at the patient's bedside. The patient was asked about this medication and stated they brought it in from home and have been taking the medication for over a month. The BAL results in accordance with the discovery of minocycline being used to treat rosacea which was not initially captured on the admission medication reconciliation, a diagnosis of AEP was made. This patient was started on an oral prednisone taper while stopping minocycline, and the symptoms of shortness of breath and dry cough improved quickly thereafter. The patient was discharged with repeat computed tomography of the chest one month later showing complete resolution of multifocal ground glass opacities. Conclusion: Minocycline is a medication used to treat bacterial infections, acne vulgaris, and moderate to severe rosacea that was the causing agent of AEP in our patient. AEP is an uncommon diagnosis that without prompt identification, diagnosis, and treatment, can be a perplexing patient presentation of pneumonia that is easily treated with oral steroids and without long-term sequalae. Works Cited: 1. King, T.E. (2022). Idiopathic acute eosinophilic pneumonia. In K.R. Flaherty & P. Dieffenbach (Eds.), UptoDate. Available from https://www.uptodate.com/contents/idiopathic-acute-eosinophilic-pneumonia?search=eosinophilic%20pneumonitis&source=search_result&selectedTitle=1~150&usage_type=default&display_rank=1
Philip Sang	*A Strong Suspicion*

Dr. Daniel Witt Dr. Jack McHugh	Introduction: Secondary eosinophilia can be caused a broad range of pathologies that include neoplastic, infectious, allergic, and inflammatory clinical entities. Here, we describe a case that illuminates the varied causes of eosinophilia, and the need for correlation to patient's presentation, travel history, and constellation of symptoms. Case Report: A 40-year-old male Buddhist monk, who immigrated to the United States from Cambodia in 2018, presented to our continuity clinic with a complaint of new pruritic rashes at site of electrocardiogram (ECG) lead placement three weeks prior. He had also been experiencing daily episodes of tachycardia, palpitations, chest tightness, dyspnea with wheezing, and flushing over his face and upper chest. The patient's medical history was significant for biopsy proven mastocytoma of the left arm and chronic hepatitis B. On physical examination his rashes were erythematous with sparce vesicular lesions painful to the touch, present in square shapes beneath the ECG pads. Laboratory testing showed a white cell count of 8.4x10(9)/L, 10.7% eosinophils with an absolute eosinophil count = 0.90x10(9)/L, ALT = 71U/L, and recent measurement of HBV DNA = 113 IU/mL. Recent imaging showed mild hepatomegaly with mild diffuse steatosis and no splenomegaly on abdominal computed tomography scan, ECG showed normal sinus rhythm with mild first-degree A-V block. A recent bone marrow biopsy, obtained to rule out systemic mastocytosis, was normal. A work-up for secondary eosinophilia was initiated. Follow-up investigation showed positive Strongyloides IgG, equivocal Echinococcus IgG, and negative Schistosoma and Toxocara IgG antibodies. Serum IgE levels were significantly elevated at 788 kU/L and unremarkable stool assessment for Ova & Parasites. The patient was initiated on two days of oral Ivermectin for uncomplicated Strongyloidiasis, and topical Triamcinolone for a suspected dermatitis flare. One month following therapy patient reported resolution of symptoms. Discussion: It is estimated that there are 100 million persons infected with Strongyloides globally. Immigrants from endemic areas can become infected and remain asymptomatic, or only experience mild symptoms that go untreated. These patients may also only present with mild eosinophilia on laboratory evaluation. Stool evaluation for Ova & Parasites is a common initial test, but it is important to recognize that given the intermittent nature of larvae excretion into the stool the sensitivity of this test can be less than 50%. Thus, when evaluating a peripheral eosinophilia of unknown etiology, it falls upon the internist to assess the patient's risk factors and obtaining accurate residential and travel history is critical in guiding appropriate diagnostic work-up. This is particularly important since certain disseminated parasitic infections can present with allergic-appearing complaints, and starting systemic steroids can have severe adverse effects. In patients with strongyloidiasis placed on long-term steroid regimens, hyperinfection can occur which has a reported mortality rate of up to 87%.
Abdulsabur Sanni	*A Waxing and Waning Diagnostic Dilemma; An Atypical Presentation of Post-Transplant Lymphoproliferative Disorder.*

Introduction: Post-transplant lymphoproliferative disorders (PTLD) are heterogeneous lymphoid disorders ranging from indolent polyclonal proliferations to aggressive lymphomas that complicate solid organ or hematopoietic transplantation (1). Multiple risk factors have been associated with the onset of PTLD such as age, reduced intensity conditioning, EBV serology mismatch and cytomegalovirus (CMV) reactivation (2).

Case Presentation: Our patient is a 68-year-old female with past medical history of ESRD secondary to SLE s/p DDKT x 2 (1991 and 2011), hypertension, and bilateral nephrectomies of native kidneys (2021) that presented with altered mental status and fever of 38.4C. Patient was started on vancomycin and meropenem with improvement in WBC and fever, patient underwent an extensive work-up for fever of unknown origin that was negative for Kikuchi's disease, Malaria, Babesiosis, Blastomycosis, Histoplasmosis, Coccidiomycosis, Cryptococcus, Coxiella, Toxoplasmosis, Tuberculosis, Brucellosis, Bartonella, amongst many others. She was discharged and presented three more times within two months for fevers and weakness in a cyclical manner every 3 weeks, with improvement after antibiotics in the hospital. Imaging showed multifocal lymphadenopathy especially in the cervical, mediastinal, and retroperitoneal lymph nodes. This led to a suspicion for a metastatic process, and she underwent EBUS of the mediastinal lymph nodes which came back negative for malignancy on flow cytometry and cytology. Our patient continued to have cyclical fevers and painless lymphadenopathy, which led to biopsy of retroperitoneal lymph nodes that came back positive for Polymorphic PTLD with destructive lymphoid cells seen on pathology.

The patient underwent multidisciplinary consultation and started guideline directed therapy for Polymorphic PTLD. Her immunosuppression regimen gradually decreased coupled with 4 weeks of Rituxan therapy with improvement clinically. Response to Rituxan will determine the need for chemotherapy in the future.

Discussion: This case emphasized the elusive nature of this disease, thanks to the persistent determination of the excellent providers that cared for this patient, the diagnosis was able to be achieved through multidisciplinary teamwork amongst multiple specialties. Many things struck this case as interesting and unique, for example the patient had chronic cyclical EBV levels since 2020 which are an independent risk factor for PTLD.

Of note, she had undergone bilateral nephrectomies of her native kidneys in 2021 due to concern for renal cell carcinoma after abdominal imaging showed multiple contrast enhanced foci during routine imaging for gastroenteritis.

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Deepon Sarkar	*Atropine-Induced Complete Heart Block and Ventricular Asystole: A Paradoxical Response During Dobutamine Stress Testing* Introduction: Dobutamine stress echocardiography (DSE) is an increasingly used modality to diagnose coronary artery disease, evaluate valvular heart disease, assess myocardial viability, and risk-stratify patients prior to non-cardiac surgery. Atropine administration with DSE has been implemented into standard protocols to allow for a reduction in test duration without an increase in the rate of complications. Ventricular asystole is a rare complication during DSE, occurring in less than 0.01% of cases. Here we present a case of transient third-degree AV heart block resulting in ventricular asystole following atropine administration during DSE. Case Description: A 52-year-old male with a history of ESRD on hemodialysis, type 2 diabetes mellitus, and essential hypertension on carvedilol presented to the echo lab to undergo DSE for cardiac risk stratification for renal transplant evaluation. At rest, echocardiography demonstrated an ejection fraction of 65% with no wall motion abnormalities, and the initial EKG showed no evidence of conduction system abnormalities. Following administration of two doses of 0.2 mg of atropine, the patient had a syncopal episode with witnessed loss of consciousness for approximately ten seconds. At that time, the EKG showed development of a complete heart block with a fifteen second episode of an asystolic pause due to non-conductive p-waves without ventricular escape or a compensatory increase in atrial rate. In addition, the EKG did not show ischemic changes during stress. The patient denied having any symptoms after waking up. The patient was admitted, and two hours after the initial syncopal episode, he had an episode of lightheadedness without loss of consciousness. At that time, telemetry showed a 13.5-second period of ventricular asystole similar to the previous episode. Repeat EKG showed 1st degree AV block. Following the second episode of ventricular asystole, the patient's carvedilol was held. Electrolytes were within normal limits. Urgent coronary angiography did not identify any obstructive lesions. An electrophysiology study did not demonstrate evidence of infra-His conduction block or HV interval change with atropine administration. The patient had no further episodes of heart block and was discharged on his home carvedilol with the presumed diagnosis of atropine-induced heart block. Discussion: The mechanism of third-degree AV block in this patient was most likely related to atropine-mediated hypervagotonia, a paradoxical response suggested by the close temporal association between the arrhythmia and atropine administration, the second episode occurring within the half-life of atropine but not dobutamine, and the lack of a compensatory increase in SA nodal rate. Though atropine is a first-line therapy for symptomatic bradycardia, this case demonstrates that atropine can cause a vagally-mediated reflex that slows SA nodal conduction, potentiates AV nodal conduction, and results in complete heart block - a rare complication that should be recognized prior to DSE and atropine administration.
Dhruv Sarma Dr. Mackenzie Maberry Dr. Samuel Garcia Dr. Timothy Aksamit	*I'm Confused: Urea-lly think it's the Ammonia?* Introduction: Hyperammonemia is a frequent cause of encephalopathy in critically ill patients but is mainly limited to those with hepatic dysfunction; non-hepatic hyperammonemia (NHHA) represents a minority of cases. If uncontrolled, hyperammonemia can lead to seizures, cerebral edema, coma, permanent neurologic damage or death. We describe a case of

	hyperammonemia with encephalopathy presenting for the first time in an adult patient heterozygous for carbamoyl phosphate synthetase 1 (CPS-1) deficiency. Case presentation: A 38-year-old female was admitted with persistent nausea and vomiting, intermittent abdominal pain and confusion. Her past medical history included obesity and daily alcohol use (2-3 beers). However, she had been abstinent from alcohol for the past 3 months. Initial evaluation revealed no physical examination findings for portal hypertension or ascites, normal liver enzymes but an elevated ammonia level to 170 µmol/L. She was commenced on lactulose and rifaximin but had rising ammonia (up to 250 µmol/L) with worsening encephalopathy necessitating transfer to the ICU and intubation for airway protection. Serial Head CTs revealed cerebral edema managed with mannitol and hypertonic saline. She required continuous renal replacement therapy (CRRT) and intravenous sodium benzoate to facilitate ammonia clearance. Liver biopsy showed mildly active steatohepatitis and evolving cirrhosis. Given the lack of any other clinical features consistent with decompensated cirrhosis, her hyperammonemia and encephalopathy were felt to be out-of-proportion to the degree of liver disease. As such, a work-up for NHHA was undertaken. On whole genome sequencing, the patient was found to be heterozygous for a likely pathogenic variant of the CPS1 gene, encoding a urea cycle enzyme. Additionally, she was found to have a portosystemic shunt between the inferior mesenteric vein and IVC which was percutaneously embolized. She was initiated on a low-protein metabolic diet with citrulline and glycerol phenylbutyrate supplementation and transitioned off CRRT. She showed gradual neurologic improvement, down-trending serum ammonia levels and was discharged from the ICU after tracheostomy placement. Discussion: We present a case of hyperammonemia causing severe encephalopathy in a patient with possible adult-onset CPS-1 deficiency, with improvement following initiation of protein restriction. Interestingly, clinical manifestations are highly unusual in heterozygous carriers. Current consensus is that the patient's second CPS-1 allele may be an unidentified pathogenic variant causing late-onset disease. The etiology of her liver disease is uncertain but may be secondary to her urea cycle disorder and/or alcohol use. This case highlights principles of the management of undifferentiated hyperammonemia in adults. Ruling out hepatic and medication-related causes remains paramount, but investigations for NHHA are important if hyperammonemia is severe and refractory to traditional management strategies for hepatic encephalopathy. These include screening for inborn errors of metabolism, porto-systemic shunts and infection caused by urease-producing organisms. Prompt recognition is essential to prevent serious neurologic complications.
Katlin Schmitz Dr. Grayson Ashby	*A Case of Recurrent BRASH Syndrome* Introduction: BRASH syndrome is a newly described clinical pentad involving bradycardia, renal failure, AV nodal blockade, shock, and hyperkalemia. This disease is caused by the synergistic combination of ACE/ARB and AV nodal blocking medications with metabolic derangement leading to profound bradycardia and shock. Often, BRASH presentations are unrecognized. However, rapid diagnosis and proper management of the disorder is imperative to prevent cardiovascular collapse and subsequent organ failure.1-4

	Case Presentation: A 72-year-old male presented to the emergency department with weakness, symptomatic hypotension, and anuria after acute-onset non-bloody diarrhea. Comorbid conditions included hypertension (on metoprolol, lisinopril, and furosemide; stage 2 chronic kidney disease, and type 2 diabetes on metformin. Initial workup demonstrated hyperkalemia, acute renal failure, metabolic acidosis, and bradycardia with a junctional rhythm and peaked T waves. Temporizing measures were given, and patient was admitted to the ICU for further cares. Of note, he had been admitted to the ICU one month prior for a similar constellation of symptoms following an episode of diarrhea. While in the ICU, the patient's home antihypertensives were held, his bradycardia resolved after IV fluids were given, and he had appropriate urinary output after furosemide challenge. His potassium normalized and the patient was transferred to the floor. On the floor, patient's creatinine down trended with oral fluids. After two days, he was discharged to home in stable condition. He was advised to hold his glimepiride, lisinopril, and furosemide for a short time while his kidneys recovered. Additionally, he was counseled to permanently avoid metoprolol (or other AV nodal blocking medications) to prevent a third presentation of BRASH syndrome. Discussion: As displayed by the above case, BRASH syndrome represents an important constellation of symptoms that can be life-threatening and disproportionally impacts the elderly. BRASH is caused by the synergistic action of AV nodal blocking medications and hyperkalemia which both contribute to bradycardia. This bradycardia causes decreased cardiac output and worsens renal perfusion leading to worsening renal failure and hyperkalemia. This cycle can lead to multiorgan failure if left untreated.1,4 Treatment for BRASH typically includes management of the acute hyperkalemia and bradycardia. However, attention must be paid to the underlying pathophysiology. Internists should consider stopping AV nodal blocking medications to prevent disease recurrence, as seen in our patient. Bibliography: 1. Farkas JD, Long B, Koyfman A, Menson K. BRASH Syndrome: Bradycardia, Renal Failure, AV Blockade, Shock, and Hyperkalemia. J Emerg Med. 2020 Aug;59(2):216-223. 2. Grigorov, et al. The BRASH syndrome, a synergistic arrhythmia phenomenon. Proc (Bayl Univ Med Cent). 2020 Jul 10;33(4):668-670. 3. Bailuni, et al. BRASH Syndrome: A Case Report. Am J Case Rep. 2022 Jan 21;23:e934600. 4. Khan A, Lahmar A, Ehtesham M, Riasat M, Haseeb M. Bradycardia, Renal Failure, Atrioventricular-Nodal Blockade, Shock, and Hyperkalemia Syndrome: A Case Report. Cureus. 2022 Mar 25;14(3):e23486.
Steven Schull Dr. Maxwell Leither	*A Case Report of Pancytopenia and Anemia Caused by Copper Deficiency after Dialysis Initiation in a Patient with Gastric Bypass* Introduction: The link between copper deficiency and resulting anemia and neurologic deficits has previously been established. Copper deficiency is most commonly related to low dietary copper intake or gastrointestinal malabsorption, including in gastric bypass patients. Removal of copper from the serum during hemodialysis is another mechanism of copper deficiency, although it seems to be rare for hemodialysis patients to develop clinically consequential copper deficiency.

	Case Presentation: In this report, we describe a unique case of a patient with remote history of gastric bypass that developed acute pancytopenia with refractory, transfusion-dependent anemia related to copper deficiency after initiating hemodialysis. The patient underwent bone marrow biopsy which demonstrated a hypocellular marrow with pathologic ringed sideroblasts. Conventional cytogenetics and Floursecence in situ Hybridization were normal. The serum copper level of this patient was undetectable and supplementation resulted in resolution of pancytopenia, refractory anemia, and myelodysplastic changes in the bone marrow. Conclusion: There is an increasing prevalence of patients with morbid obesity and end stage renal disease. Referral for bariatric surgery in patients with advanced chronic kidney disease is likely to continue to increase given its proven benefits. Clinicians therefore should be aware that hemodialysis is a possible cause of life-threatening copper deficiency in patients with other risk factors such as gastric bypass history and should suspect copper deficiency in patients with neurological symptoms or refractory anemia with or without pancytopenia.
Kevin Stanko Dr. Kathryne Wood Dr. Katrina Williamson Dr. Clement Michet D. Zafer Keser	*To Behcet or Not to Behcet, an Atypical Presentation* Case Presentation: Mr. H is a 28-year-old Somali-born male with recent history of left sided Bell's palsy with residual facial muscle weakness and synkinesis and chronic migraine headaches presenting for progressive left-sided frontal headache and diplopia. Nine days prior, he experienced nonspecific dizziness accompanied by a bifrontal throbbing headache with diplopia that worsened with leftward gaze. Over the following days, his headache intensity worsened and he presented to the emergency department. He denied a history of fever, night sweats, weight change, arthritis, uveitis, oral/genital ulcers, blood clots, or neurological symptoms. Physical exam showed left eye abduction palsy with right eye horizontal nystagmus at left gaze consistent with complete left six nerve palsy, in addition to long-standing seventh nerve palsy. MRI/MRA head&neck vasculitis protocol revealed no parenchymal lesions though he had significant inflammation, wall thickening, and luminal narrowing in multi-vessel large arteries including the left common, cervical, and intracranial internal carotid arteries (ICA) along with the left subclavian artery and aortic arch. Inflammation involving the left cavernous segment of the ICA possibly extended to the sixth nerve as well. MR brain venogram demonstrated subtle venous enhancement without evidence of acute thrombosis but identified a potential chronic non-flow-limiting venous sinus thrombosis. CTA chest abdomen-pelvis show no other large vessel involvement. Serum studies for infectious etiologies, hypercoagulability, and vasculitis were notable only for mildly elevated inflammatory markers and positive QuantiFERON. CSF studies returned unremarkable and not suggestive of inflammation or infection. Discussion: This patient displays radiographic findings of large vessel vasculitis with venous and cranial nerve involvement. Despite the lack of classical symptoms there was strong suspicion for Behcet’s, with Takayasu considered but thought to be less likely. The clinical picture is further complicated by a positive QuantiFERON, though active TB and TB meningitis were ruled out with chest CT and CSF studies. Due to reactivation risk, we started Rifampin for latent TB plus high dose steroids for vasculitis. Additional steroid sparing immunosuppression was deferred. Thus, this

	constellation of findings does not fit neatly into a single diagnosis, yet initial treatment is corticosteroids for both conditions. Conclusions: The diagnosis and management of vasculitis in the cervical and intracranial arteries presents a multitude of challenges. Clinical manifestations are nonspecific and may be mimicked by other diseases. Thus, a broad differential must be considered. Moreover, such cases are extremely rare, with Behcet's disease and Takayasu arteritis occurring in the U.S at rates of 5.2 cases/100,000 and 2-3 cases/1,000,000, respectively. In this case, we present the diagnosis and management of a large vessel vasculitis presenting in an atypical fashion and further complicated by underlying latent TB. Overall, this unique situation illustrates the subtleties in presentation and treatment of large vessel vasculitis.
Erin Steiner Dr. Breanna Zarmbinski	*A Case of Amenorrhea and Macrocytic Anemia Unmasking Atypical Anorexia Nervosa and Alcohol Use Disorder* Introduction: Patients with eating disorders are known to be at increased risk of concurrent substance use disorders. Additionally, normocytic anemia and leukopenia are common hematologic abnormalities in patients with eating disorders. Patients with restrictive eating patterns who present with macrocytic anemia should be evaluated for both nutritional deficiencies and alcohol use disorder. Case Presentation: A 32-year-old woman with a history of anxiety and depression presented to her gynecologist with amenorrhea. Labs revealed leukopenia, macrocytic anemia with a hemoglobin of 8.9 and an MCV of 124, and elevated liver enzymes with AST greater than ALT. Iron studies were normal. A peripheral blood smear demonstrated macrocytic anemia with poikilocytosis consistent with B12 and folate deficiency and alcohol toxicity effect. At primary care follow up, patient endorsed a history of intermittent fasting with fifteen pound weight loss and restrictive eating patterns. Additionally, she reported increased alcohol use over the course of the COVID pandemic with consistent consumption of up to 750 mL of wine daily. She also noted worsening anxiety and depression despite continuing her SSRI. Further lab workup revealed low vitamin B12 and folate. She was started on daily B12 injections, and folic acid supplementation. For her alcohol use disorder and restrictive eating, she was referred for mental health treatment with psychiatry and psychology in addition to pharmacologic assistance for alcohol cessation with naltrexone which she declined. At two month follow up, her hemoglobin improved but the patient was unable to abstain from alcohol and naltrexone was prescribed. Unfortunately, the patient was subsequently lost to follow up and returned to clinic approximately eleven months after her initial presentation with weakness, syncope, and vomiting. She was found to have a profound anemia with hemoglobin 7.3, leukopenia, and elevated transaminases with hyperbilirubinemia. She was prescribed gabapentin for both alcohol cravings and anxiety and referred for virtual addiction assessment. One month later she requested assistance to enroll in an inpatient alcohol abuse treatment program but coverage was declined by her insurer. She remained anemic and was found to have new iron deficiency despite previously normal iron studies. This was suspected to be diet related as she had no reported blood loss. She had significant improvement in her anxiety and alcohol cravings wand was able to maintain sobriety with the use of gabapentin. Conclusion: This case illustrates the importance of screening for co-morbid

	alcohol use in patients with eating disorders associated with macrocytic anemia. It also demonstrates the utility of gabapentin in the outpatient treatment of alcohol use disorder at doses of up to 600 mg three times daily.
Rachel Suen Dr. Daniel DeSimone Dr. Kianoush Kashani	*Out with the Old, In with the New: Exchange Transfusion for Severe Babesiosis* Introduction: Babesiosis is a potentially life-threatening illness caused by the intraerythrocytic protozoan parasite Babesia microti transmitted by the Ixodes tick. It is most common in the Northeastern and Upper Midwestern United States during summer. Typical symptoms include fever, chills, myalgias, fatigue, and anemia after several weeks of incubation. Immunocompromised, asplenic, and elderly patients may develop severe disease with hemolytic anemia, kidney dysfunction, and pulmonary involvement. Here, we present a case of severe babesiosis treated successfully with azithromycin, atovaquone, doxycycline, and red blood cell (RBC) exchange transfusion (ET). Case Presentation: A 72-year-old gentleman with a history of insulinoma s/p distal pancreatectomy and splenectomy, gastric wedge resection, hyperlipidemia, prior history of prostate cancer presented with three weeks of myalgias, fatigue, icterus, anorexia, night sweats, fevers, and chills. He reported living in a forest and frequent mountain biking with multiple tick bites two months before his presentation. Initial labs were remarkable for acute kidney injury, anemia, thrombocytopenia, bilirubinemia, undetectably low haptoglobin, and tick-borne infection panel positive for Babesia. He was started on oral azithromycin and atovaquone but eventually required inpatient admission due to worsening nausea and inability to tolerate oral therapy. He developed severe parasitemia to 32%, acute kidney injury with a creatinine peak of 7.26 mg/dL, and hemolytic anemia with hemoglobin of 7.8 g/dL. After initial RBC ET, parasitemia decreased to 5.6% (82.5% reduction). Repeat ET resulted in reduced parasitemia to 1.3%, resolution of his hemolytic anemia, and improvement of his kidney injury without the need for dialysis. Parasitemia remained low throughout the remainder of his hospitalization. Due to his asplenia and severe infection, he was treated with intravenous azithromycin (transitioned to oral after symptom resolution and parasitemia decline) and atovaquone for six weeks, with plans to repeat a peripheral smear after therapy to ensure infection resolution. Discussion: This case illustrates the use of RBC ET in treating severe babesiosis. Asplenia is a risk factor for severe infection as splenic macrophages play a key role in phagocytosing bacteria and damaged erythrocytes from circulation. ET acts by replacing parasitized cells with noninfected erythrocytes. It is a therapy option for severe parasitemia in intracellular infections such as malaria and Babesia but is not well studied. Prior case reports show 40-95% reduction in Babesia parasitemia following ET, consistent with the reduction seen in this case. Some vulnerable adults have developed prolonged or relapsing infection even after ET, emphasizing the importance of continued monitoring. Nonetheless, ET can be a potentially life-saving treatment in patients with severe babesiosis.
Christopher Van Hove	*A Rare Cardiac Etiology of Exertional Dyspnea* Introduction: High-degree atrioventricular block is often a relatively straightforward diagnosis attained based on a classical history of dizziness, lightheadedness, fatigue, presyncope, and/or syncope with corresponding

	cardiac monitoring or electrocardiography. However, exercised-induced high-degree atrioventricular block is an exceedingly unusual diagnosis that poses a diagnostic dilemma to uncover as exertional symptoms are almost always appropriately pursued for obstructive atherosclerotic cardiovascular disease, structural disease, or valvular disease which, when negative, may lead to a pursuit of noncardiac causes. Case Presentation: Herein we describe a unique case of an otherwise healthy and active 64-year-old female who presented with complaints of exertional fatigue, pre-syncope, and dyspnea over the course of six months. Notably, her symptoms were absent at rest. Cardiac risk factors included a family history of premature coronary disease, and her physical exam was entirely unremarkable from a cardiopulmonary perspective. Initial evaluation with resting ECG showed sinus rhythm, first-degree AV block, an incomplete right bundle branch block, and non-specific ST-T wave abnormalities. Follow up transesophageal echocardiography was arranged to assess for possible structural, functional, and valvular disease which demonstrated a normal left ventricular ejection fraction of 55-60%, mild mitral valve prolapse, a mildly dilated aortic root, mild to moderate dilated ascending aorta, and mild to moderate aortic regurgitation which was not convincing for the etiology of her symptoms. Cardiac monitoring was then pursued with twenty-four hour Holter monitoring which showed occasional supraventricular and ventricular ectopy but no significant cardiac dysrhythmia to correlate her symptoms despite her report of reproduction of symptoms. Ultimately, exercise stress testing was undertaken revealing the development of left bundle branch block and high-degree AV node block with return of symptoms that resolved post-testing. Given concern for ischemia, subsequent computed tomography (CT) coronary angiography was negative for occlusive disease or atherosclerotic burden. It was presumed our patient had infranodal disease within the His-Purkinje system given her evidence of 1st degree AV block and incomplete right bundle branch block on resting ECG. With shared decision making, she proceeded with implantation of dual chamber permanent pacemaker for definitive management resulting in complete resolution of symptoms. Conclusion: This case illustrates an important concept in keeping a broad differential diagnosis to guide diagnostic workup, especially in the setting of an evolving field of evaluation for clinically significant atherosclerotic cardiovascular disease with some individuals obtaining non-stress inducing imaging such as CT coronary angiography. Although this diagnosis is quite rare, pursuit of testing that did not evoke a similar stress response with reproduction of symptoms may have missed a potentially ominous outcome.
Matthew Vincent Dr. Manal Abdelmalek Dr. Kathryn Schnidt	*A Rare Cause of Portal Hypertensive Ascites and Hepatorenal Syndrome in a Patient with Metastatic Renal Cell Carcinoma* Introduction: Malignant ascites is not an uncommon complication of metastatic renal cell carcinoma which is characterized by a high total protein with a low serum albumin ascites gradient (SAAG). Portal hypertensive ascites, however, is associated with complications including esophageal varices, spontaneous bacterial peritonitis, and hepatorenal syndrome. Here we present a case of metastatic renal cell carcinoma complicated by intrahepatic portal hypertension leading to ascites, esophageal varices, and hepatorenal syndrome. Case Presentation: Our patient is a 79-year-old male with a pertinent medical

	background of renal cell carcinoma status post unilateral nephrectomy with peritoneal metastases who presented to hospital with new-onset ascites and acute kidney injury. Initial labs were notable for a macrocytic anemia with a Hb of 9.9g/dL (baseline 11.2g/dL), and acute kidney injury with creatinine 3.9 g/dL (baseline 2.3 g/dL). Magnetic Resonance and ultrasound imaging of the abdomen showed known abdominal disease, but no evidence of liver involvement. He underwent paracentesis which revealed a total protein of 2.1 g/dL with a SAAG of 2.1. A transjugular hepatic vein pressure gradient was measured which revealed a transhepatic pressure gradient of 11 mmHg, consistent with intrahepatic portal hypertension. A liver biopsy was performed concurrently which revealed diffuse hepatic infiltration by metastatic renal cell carcinoma. An EGD was performed which revealed grade 1 esophageal varices. He was treated with IV albumin and frequent paracentesis to optimize renal perfusion for possible hepatorenal syndrome. Despite this, his kidney function continued to worsen, requiring increasing doses of Lasix to maintain adequate urine output. He had a dialysis catheter placed and was initiated on intermittent hemodialysis. Ultimately, given his multiorgan failure, it was felt that systemic anticancer therapy was not within his goals of care. He had an abdominal pleurX catheter placed to manage his ascites and was ultimately discharged to hospice. He passed away one week after hospital discharge. Discussion: This case outlines how in any patient presenting with new onset ascites, proper etiological workup is essential, even in cases where the pre-existing diagnosis suggests an obvious cause. In our patient, ascitic fluid studies led to the diagnosis of portal hypertension complicated by esophageal varices which had treatment implications. While renal cell carcinoma has been associated with malignant ascites, portal hypertension has only rarely been described as a complication from portal vein thrombosis. To our knowledge, this is the first case of hepatic infiltration by renal cell carcinoma leading to portal hypertension.
Yun Wang	*A Case of Severe B12 Deficiency Presenting with Pancytopenia and Hemolysis* Case presentation: A 67-year-old female with a past medical history of type 2 diabetes, hypertension, hypothyroidism was admitted to the hospital for evaluation of profound anemia (hemoglobin 4.6 g/dL). She initially presented to her PCP with one-month of fatigue, generalized weakness, and exertional dyspnea. She denied any bleeding history. She was not a vegetarian and denied alcohol use. Physical exam was remarkable for jaundice, scleral icterus, and pale conjunctiva. CBC additionally revealed low hematocrit (12.8%), macrocytosis (MCV 118.5 fL), thrombocytopenia (platelet 97 × 109/L) and leukopenia (WBC 2.5 × 109/L). BMP was unremarkable. Hemolysis labs showed unconjugated hyperbilirubinemia, elevated lactate dehydrogenase (3565 U/L), low fibrinogen (159 mg/dL), and low haptoglobin (<17 mg/dL). Absolute reticulocyte count was abnormally normal at 31.4 × 109/L (30.4 - 110.9 × 109/L) with a low reticulocyte index of 0.4. Peripheral blood smear revealed anisocytosis, polychromasia, presence of hypersegmented neutrophils, but no schistocytes. Coomb's test obtained prior to pRBC transfusion was negative. PT was 13.2s and aPTT was 24s. Serum iron and ferritin levels were normal. Following admission, the patient received 3 units of pRBCs, raising her hemoglobin to 8.1 g/dL. Further workup was remarkable for low vitamin B12 level of < 150 ng/L. She was started on daily vitamin B12 injections at 1000

	mcg/ml. Repeat absolute reticulocyte count showed immediate positive response with an increase to 63.6 × 109/L after the first dose of B12 injection. Pernicious anemia cascade returned after hospital discharge which revealed elevated methylmalonic acid level and the presence of intrinsic factor blocking antibody. The patient was diagnosed with pernicious anemia. Additional outpatient workup with CT chest, abdomen, and pelvis did not reveal malignancy, liver or spleen related pathologies that could explain her presentation. Follow-up labwork after the first week of B12 replacement showed improved hemoglobin at around 9 g/dL, normalization of WBC and platelet counts, and an increase in absolute reticulocyte count to 200.6 × 109/L. Discussion: Vitamin B12 deficiency is well-known to cause macrocytic and megaloblastic anemia. Pancytopenia is less common but can be seen due to impaired DNA synthesis leading to ineffective trilineage hematopoiesis. This patient additional presented with lab findings consistent with hemolytic anemia. However, her low normal reticulocyte count suggested the presence of a hypoproliferative process and therefore argued against hemolysis as a sole explanation for her anemia. Both intramedullary and extramedullary hemolysis associated with B12 deficiency have been observed and reported. Intramedullary hemolysis is due to the destruction of megaloblastic cells within the bone marrow, whereas the mechanism for extramedullary hemolysis is less clear. Reticulocyte count and index can be helpful in differentiating B12 associated intravascular hemolysis from thrombotic microangiopathy.
Alexander Xiao	*A Rare Case of Recurrent Lipoid Pneumonia* Introduction: Exogenous lipoid pneumonia is a rare condition caused by aspiration of lipid substances and classically characterized by fatty attenuation on chest CT. Diagnosis is difficult due to its insidious nature, nonspecific symptoms and clinical findings. A high index of suspicion and careful history taking is vital to helping guide appropriate treatment and reducing recurrence rates. Case Presentation: A 91-year-old man in his primary care clinic endorsed one year of intermittent dyspnea. Chest x-ray revealed bilateral lower lung opacities and CT chest showed large opacities in both lungs suspicious for lung cancer, lymphoma, or pneumonia. Further history revealed that for the last year, the patient had applying mineral oil to his CPAP mask to combat dry mouth. With this additional information, repeat CT chest identified classic findings of lipoid pneumonia including a “crazy-paving” pattern. The patient was diagnosed with lipoid pneumonia and counseled on ceasing mineral oil use and by follow up he was significantly improved. Three years later, he was admitted for hypoxia. He had recently been diagnosed with Zenker’s diverticulum and CT chest found both left lower lobe consolidation suspicious for aspiration, lung scarring and findings consistent with lipoid pneumonia. He now endorsed misting his mouth with an over-the-counter spray, which we found to contain oil, and he was softening food with olive oil. He was discharged after receiving appropriate antibiotic therapy for aspiration pneumonia and advised to avoid subtle triggers for lipoid pneumonia. Discussion: Lipoid pneumonia is an inflammatory response to lipid substances in the lungs and can lead to resultant fibrosis. Classically, it is associated with mineral oil use for constipation. Other common causes

	include use of oil nasal emollients, occupational exposure such as metal working, and e-cigarette use. The clinical history is challenging as patients are typically asymptomatic with incidental imaging abnormalities or they may present with insidious, nonspecific symptoms, usually cough, dyspnea, or fever. Aspiration may not be obvious due to oil's high viscosity which can depress the cough reflex. Unique radiological findings include low-density fat attenuation within areas of consolidation on CT chest (-30 to −150 Hounsfield Units) and a "crazy-paving" pattern. These imaging findings are often misread as carcinoma. Ultimately, diagnosis is made by the combination of clinical history and consistent radiographic findings which can be further supported by demonstration of lipid laden macrophages in bronchoalveolar lavage fluid or lung biopsy. The most important aspect of treatment is identification and avoidance of the offending agent. However, avoidance alone may not clear the aspirated material, so additional treatment such as whole lung lavage or systemic corticosteroids may be needed if clinical improvement is inadequate. However, the overall efficacy of these interventions remains uncertain.
Yuan Yao Dr. Katarina Fabre Dr. Alex Liu Dr. Rachel Suen Dr. Jonas Paludo	*A Case of Conjugated Hyperbilirubinemia Due to Epstein-Barr Virus-Associated B-cell Lymphoproliferative Disorder* Introduction: Hyperbilirubinemia is a common clinical problem with various causes. The pathophysiology includes bilirubin overproduction, impaired conjugation, biliary obstruction and hepatic inflammation. Initial laboratory tests include measuring total and conjugated bilirubin, liver enzymes, prothrombin time and albumin. Subsequent evaluation includes imaging (eg. ultrasound, ERCP, MRCP), hepatitis serology, autoimmune serology, thyroid function tests, iron studies and celiac test. Liver biopsy may be required to confirm the diagnosis. We present a case of conjugated hyperbilirubinemia due to EBV-associated B-cell lymphoproliferative disorder (LPD) that was diagnosed with biopsy. Case presentation: A 68-year-old male is being evaluated for hyperbilirubinemia. His past medical history includes rheumatoid arthritis, hypothyroidism, bipolar disorder, gastroesophageal reflux disease and pulmonary embolism. A CT of the body revealed a pancreatic mass, with nonspecific cervical, thoracic, and abdominopelvic lymphadenopathy. Lab workup revealed total bilirubin 5.6 mg/dL (conjugated bilirubin 3.4 mg/dL), ALT 374 U/L, AST 690 U/L, alkaline phosphate 471 U/L, albumin 2.8 g/dL, PT 15.6 s. He underwent ERCP and EUS which confirmed the mass was actually to be a duodenum diverticulum with diffusely mildly narrowed intrahepatic ducts; no discrete strictures were seen. During the hospital course, his hyperbilirubinemia worsened and peaked at 14.1 mg/dL (conjugated bilirubin 13.4 mg/dL) 10 days later. HIV, HAV, HBV, HCV, HSV1/2, VZV, and CMV tests were negative. Extended infectious workup including histoplasmosis, blastomycosis, legionella, anaplasma, Borrelia, Babesia, spotted fever, Ehrlichia and Quantiferon were negative. Antinuclear antibody, LKM1 antibody, ANCA, IgG4, SPEP was negative. Anti-mitochondrial antibody was weakly positive at 0.1 U. Anti-smooth muscle antibodies was initially positive at 1:40, however repeat SMA antibody was negative. Plasma EBV DNA was significantly elevated to 17,300 IU/mL. Porta hepatis lymph node and liver biopsy was pursued which showed EBV-positive, polymorphic B-cell lymphoproliferative disorder. He was started on rituximab and his EBV DNA decreased to 70 IU/mL after 4 doses, and his bilirubin completely normalized within 5 weeks. He underwent PET-CT scan

	after completion of rituximab treatment which showed complete response of the lymphoproliferative disorder. Discussion: The patient underwent liver biopsy for conjugated hyperbilirubinemia which showed polymorphic LPD. LPDs are a group of diseases that are frequently EBV positive and are commonly immunodeficiency-associated. In our patient's case, though he has no history of organ transplant and is not taking any immunosuppressant, age-associated immunosenescence and the comorbidity of rheumatoid arthritis could contribute to an immunodeficiency state which predisposes him to LPD. In patients with conjugated hyperbilirubinemia, infiltrative diseases such as LPD need to remain on the differentials. Liver biopsy should be offered to confirm the diagnosis when lab workups and imaging are inconclusive.
Dongni Yi Dr. Savannah Liddell	*Coexisting Colitis: A Diagnostic Conundrum* Introduction: Segmental colitis associated with diverticulosis (SCAD) is the inflammation of the interdiverticular mucosa without involvement of the diverticular orifices with unclear pathogenesis. Patients typically present with chronic diarrhea, abdominal pain, primarily in the left lower quadrant, and hematochezia, which are indistinguishable from ulcerative colitis (UC). Although the distribution of colitis helps differentiate SCAD and UC, they may coexist simultaneously. Flexible sigmoidoscopy is critical in determining the driving force to guide treatment. Here we present a case of UC and SCAD. Case Presentation: A 61-year-old male with UC previously well-controlled with sulfasalazine presented with abdominal pain, bloody diarrhea, and tenesmus. He completed a 10-day course of prednisone taper. However, symptoms persisted. He initiated the second course of prednisone and presented to hospital 6 days later due to worsening symptoms. Computed tomography (CT) on admission showed colonic diverticulosis, diffuse pancolonic wall thickening, most prominently involving the sigmoid colon, pericolonic stranding involving the entirety of the sigmoid colon, and multiple gas and fluid collections along the sigmoid margins, with the largest measuring 3.7 x 3.4 x 3.1 cm, concerning for colitis with contained perforations. The largest fluid collection was drained via CT-guided aspiration. Culture grew Streptococcus anginosus, Gram-positive and negative bacilli, and Candida albicans, treated with piperacillin/tazobactam and fluconazole. Methylprednisolone was started on top of sulfasalazine to treat presumed ulcerative colitis flare. He completed a 3-day course of methylprednisolone 60 mg daily. C-reactive protein (CRP) decreased by less than 50% from 171.4 to 136.6, suggesting an inadequate response to steroids. Abdominal pain worsened on hospital day 4 following initial improvement. Repeat CT showed non-drainable additional abscesses, treated conservatively with continued antibiotics and bowel rest. We proceeded with flexible sigmoidoscopy, which noted only mild colitis in rectum and more obvious inflammation around the abscesses, suggesting SCAD complicated by perforation with abscess is more likely to be the driving force than UC flare. Treatment for UC was de-escalated to home sulfasalazine, and steroid was discontinued. Symptoms improved with continued antibiotics, the first-line treatment for SCAD, and the patient was discharged in 2 weeks. Due to sinograms showing persistent fistulous communication between the bowel and the original abscess, although downsizing, partial colectomy was planned after the infection and inflammation got better controlled.

	Conclusion: The manifestation of SCAD is indistinguishable from UC. Failure to consider SCAD could set the patient on the wrong treatment pathway. Direct visualization on endoscopy is crucial as the distribution of the colitis helps to differentiate SCAD from UC. SCAD demonstrates inflammation in the mucosa between the diverticula and does not involve the distal rectum, whereas the rectum is always involved in UC. However, SCAD and UC may coexist, as in our case. Suspicion of co-existent SCAD should be raised when patients with UC fail to improve with glucocorticoids. Although rectal involvement cannot rule out SCAD, it provides valuable information regarding which is the driving force.