American College of Physicians- Minnesota Chapter Annual Abstract Competition

Virtual Poster Session

October 29, 2021

Abstracts Submitted for Competition and Winning Abstracts

Medical Students	
Research - Medical Students	
Kristene Tadese Dr. LaPrincess Brewer Sarah Jenkins Dawn Aycock Clarence Jones Dr. Sharonne Hayes Dr. Lisa Cooper Dr. Christi Patten **2021 MN-ACP winner Medical Students-Research**	*Factors Facilitating Academic-Community Research Partnerships with African-American Churches* Background: African-Americans (AAs) continue to be underrepresented in medical research and clinical trials. This can lead to inadequate access to novel medical therapies and innovations among this population, thus undermining health equity. AA churches are valuable partners in implementing health promotion interventions to combat disparities. This study evaluates church characteristics associated with enrollment into the FAITH! Trial, a community-based, cluster randomized trial for cardiovascular health (CVH) promotion among AA churches. Methods: Churches attending recruitment events in Minneapolis-St. Paul and Rochester, MN were invited to complete an electronic survey and telephone interview. These instruments assessed church characteristics and readiness to engage in health promotion programming (HPP) as determined by the Brand's PREACH (Predicting Readiness to Engage African American Churches in Health) assessment tool. The primary outcome of interest was church enrollment in the FAITH! Trial. Key predictors of interest included congregation size, average congregation member age and church readiness to engage in HPP as defined by PREACH scores and stage classifications (1-3), denoting low to high capacity for engagement. Results: Of the 30 churches attending recruitment events, 26 (93%) completed the screening survey and follow-up interview, and 16 (53%) enrolled into the trial. Of churches surveyed, 7 (27%) were classified as PREACH Stage 3; 14 (54%) as Stage 2 and 5 (19%) as Stage 1. No statistically significant associations between the predictors with enrollment in the FAITH! Trial emerged, but the data demonstrated important differences in characteristics between enrolled and non-enrolled churches. Twelve (75%) of enrolled churches had >75 members vs 6 (60%) of non-enrolled churches; 12 (80%) of enrolled churches had an average congregation member age ≤54 years vs 6 (66.7%) of non-enrolled churches; and 5 (31.3%) of enrolled churches were Stage 3 vs 2 (20.0%) of non-enrolled churches. PREACH scores (enrolled vs non-enrolled averages) showed that enrolled churches had higher personnel scores (8.8 vs 5.6), funding scores (7.3 vs 4.8) and overall PREACH scores (36.1 vs 30.2). Conclusion: AA churches enrolling into a community-based CVH

	intervention study had greater infrastructure for HPP, larger congregations and members of younger age. Institutions seeking to increase accessibility to clinical trials and medical therapies to AA communities by partnering with AA churches must provide greater support to smaller congregations with members of older age through adequate personnel and funding for optimal outcomes.
Christina Gu Dr. Patricia Jewett Dr. K. Robin Yabroff Dr. Rachel Vogel Dr. Helen Parsons Dr. Himal Purani Dr. Anne Blaes	*Regional Clustering of Financial Hardship in the United States Among Cancer Survivors by Age, Sex, and Race/Ethnicity* Background: Innovative treatments have improved cancer survival but also increased financial hardship for patients. While demographic factors associated with financial hardship among cancer survivors are known in the United States (US), the role of geography is less clear. Methods: We evaluated financial hardship (self-reported foregoing care due to cost within 12 months) by US Census region (Northeast, North Central/Midwest [NCMW], South, and West) in combination with demographic factors (age, sex, race/ethnicity) among 217,981 adult cancer survivors aged 20 to 82 years who participated in the 2015-2019 Behavioral Risk Factor Surveillance System survey. We summarized region-and group-specific prevalence of medical financial hardship and used multivariable logistic regression models to compare financial hardship by region. Results: Financial hardship was most prevalent in the South (19-38% among those aged <65 years; 4-21% among those aged ≥65; odds ratios [OR] of financial hardship adjusted for demographics, NCMW versus South, OR: 0.63 [0.56-0.71]; Northeast versus South, OR: 0.63 [0.55-0.73]; West versus South, OR: 0.73 [0.64-0.84]). Across all US regions, including regions with broad Medicaid expansion, younger, female, and persons of color experienced the most financial hardship. Conclusion: Medical financial hardship among cancer survivors is regionally clustered in the US, and greatest in the South. Underlying factors likely include both differences in regional population compositions, wealth inequities, and contextual factors such as Medicaid expansion and different social policies. Disproportionate financial hardship among persons of color in all regions highlight systemic barriers throughout the US, underscoring the need to improve health care access for those most vulnerable.
Clinical Vignette - Medical Students	
Kimberly Wang Dr. Hadiyah Audil Dr. Yuan Yao Dr. James Howick Dr. Camilo Bermudez Noguera Johnny Dang Dr. Chris Aakre **2021 MN-ACP winner Medical Students-Clinical Vignette**	*Stones, bones, groans, and psychiatric moans during COVID overtones?* Introduction: Severe hypercalcemia (>13 mg/dL) can present with a wide array of clinical manifestations including nephrolithiasis, anorexia, constipation, weakness, bone pain and osteopenia, fatigue, confusion, bradycardia, and ECG changes. The most common cause of hypercalcemia is primary hyperparathyroidism, which typically manifests as calcium levels <13 mg/dL. We present a case of primary hyperparathyroidism causing severe hypercalcemia and altered mental status. Case Description: An 87-year-old man with a history of hypothyroidism, hypertension, hyperlipidemia, and depression presented to the emergency department for evaluation of hypercalcemia (total calcium 14.6 mg/dL) discovered during routine lab work. His family described a history of altered

	mental status, weakness, increased urinary frequency, mood changes, gait changes, fatigue, constipation, and shoulder pain coinciding with the timing of his recent COVID-19 infection. On physical examination, he was afebrile with a heart rate of 51-58 beats per minute and blood pressure of 173/70 mmHg. He endorsed myalgias and arthralgias. Lab workup revealed phosphorus 1.7 mg/dL and PTH 167 pg/mL; the free light chain ratio was normal. CT chest angiogram demonstrated evolving pulmonary fibrotic changes consistent with a previous COVID-19 pneumonia and noted mild thoracic lymphadenopathy that was atypical for COVID-19 pneumonia. Following admission, he was given IV fluids, calcitonin, and zoledronic acid; he was started on a low calcium diet, his home thiazide diuretic (which may have contributed to his presentation) was discontinued, and a sestamibi scan was ordered to evaluate for parathyroid adenomas that may be amenable to surgical intervention. Discussion: When hypercalcemia is noted on lab work, the first step is to confirm the diagnosis with a repeat measurement and to correct for albumin. Once severe hypercalcemia is diagnosed, it is important to initiate prompt treatment with IV fluids, calcitonin, and bisphosphonates. Lactated Ringers (LR) is preferred over normal saline (NS) for fluid resuscitation; although it contains calcium, LR is less likely to exacerbate hyperchloremic acidosis compared to NS. For most patients, prompt treatment with an IV bisphosphonate, zoledronic acid, is preferred because it is more potent than other agents, particularly for hypercalcemia of malignancy. However, for patients who have renal impairments, IV pamidronate is the next best option. Amid the COVID-19 pandemic, many generalized symptoms of primary hyperparathyroidism overlap with those of the post-COVID-19 syndrome including weakness, fatigue, confusion, and muscle and body aches. Clinicians must therefore have a high level of clinical suspicion in ruling out a patient for hypercalcemia.
Allison Angeli Dr. Bryant Megna Dr. Marshall Mazepa Dr. Zalaya Ivy Dr. Shahnaz Sultan Dr. Joshua Sloan	*Transfusion-Dependent Anemia Secondary to Vitamin C Deficiency* Introduction: Scurvy, or the syndrome of vitamin C deficiency, is rare in industrialized nations. Characteristic manifestations include coiled hair, perifollicular hemorrhage, petechiae, and gingivitis. Mild anemia occurs in up to 80% of patients. This clinical vignette details a noteworthy presentation of scurvy with six weeks of profound transfusion-dependent anemia. Case Presentation: A 57-year-old man presented to the emergency department with anemia, fatigue, arthralgias, myalgias, and easy bruising. He was hypotensive at 72/54 mmHg. Physical exam demonstrated bilateral lower extremity tenderness, edema, and ecchymoses. Hemoglobin was 5.1 g/dL, and reticulocyte count was 0.15 mil/mm3 (0.03-0.08 mil/mm3). Elevated total bilirubin and lactate dehydrogenase were noted due to a known resolving quadriceps hematoma. Haptoglobin, DAT, PT, aPTT, and fibrinogen were normal. No bleeding was identified on extensive gastrointestinal, vascular, and musculoskeletal imaging. Peripheral smear showed hypochromic, normocytic anemia. Bone marrow biopsy showed intact trilineage hematopoiesis, no dysplasia or blasts, and hypocellular marrow (5-25%). He required 20 units of pRBCs over a 26-day-long admission. He was transferred to a tertiary care center for further management. A capsule endoscopy was unremarkable. Assessments for infections (HIV, EBV, CMV, parvovirus, HBV, HCV), coagulopathies (INR, aPTT, PT, fibrinogen), hemolysis (haptoglobin, LDH, DAT), pernicious anemia (parietal cell antibodies),

	autoimmune processes (C3, C4, CH50, anti-Jo antibody, CRP, ESR), and factor deficiencies (factor 10 assay, factor 13 assay) were normal. The total bilirubin remained elevated (2.0 mg/dL). A repeat peripheral blood smear and bone marrow biopsy were not significantly changed. He required 10 more units of pRBCs. Upon further interview, the patient described months of a micronutrient-poor diet consisting universally of simple carbohydrates. Physical exam revealed persistent lower extremity echymosses and edema, subcutaneous hemorrhages, perifollicular changes, coiled body hair, and gingival erythema. Two vitamin C levels were < 5 umol/L. Vitamin C supplementation was initiated at 250 mcg twice daily. Three days later, no further pRBC transfusions were necessary. The vitamin C level and hemoglobin continued to rise until time of discharge. Discussion: In addition to highlighting the importance of a robust history and physical exam, this case also illustrates an unusually profound degree of transfusion-dependent anemia with an unclear pathophysiology in a patient with scurvy. The mechanism of scurvy-associated anemia is multifactorial and may involve: hemorrhage from improper collagen synthesis, hemolysis from unopposed oxidant injury in erythrocytes, impaired erythropoiesis, and associated folate deficiency. Given the persistent hyperbilirubinemia and low reticulocyte counts relative to the degree of anemia, this patient's scurvy-associated anemia may have been due to hemolysis and insufficient erythropoiesis. Further research is needed to understand the role of vitamin C deficiency in hemolysis and inadequate erythropoiesis.
Johnny Dang Dr. Hadiyah Audil Dr. James Howick Dr. Yuan Yao Dr. Camilo Bermudez Noguera Kimberly Wang Dr. Christopher Aakre	*A Severe Case of SSRI-Induced Hyponatremia* Introduction: Hyponatremia affects up to 30% of hospitalized patients, with the most common cause being drugs including thiazides, antipsychotics, and SSRIs. Severe hyponatremia is a life-threatening emergency that can lead to confusion, seizures, and even death. Hyponatremia secondary to SSRIs most commonly occurs within the first week of treatment initiation. Case Presentation: A 71-year-old female was recently started on buspirone and escitalopram by her PCP for anxiety. Sodium prior to medication initiation was 129. Four days later, she presented to the ED with anorexia, anxiety, and insomnia. She was reassured that SSRIs can cause these nonspecific symptoms and was discharged without further workup. She returned to the ED a week later with progressive malaise and lethargy. Workup revealed a sodium of 110. Serum osmolarity was 241, urine osmolarity was 404, and urine sodium was 61. TSH was normal at 1.0. AM cortisol levels were normal. She was given hypertonic saline and subsequently admitted to the ICU, where her sodium normalized with normal saline administration, free water restriction, and salt tablets. Discussion: Given the laboratory findings mentioned above, the differential for hyponatremia included SIADH, adrenal insufficiency, and hypothyroidism. Further workup showed normal cortisol and TSH, therefore her presentation was thought to be due to SIADH caused by her SSRI. Her buspirone and escitalopram were discontinued, and her sodium remained normal and stable on discharge.
Patrick Duggan	*Hiding in (Not So) Plain Sight: Spontaneous Tumor Lysis Syndrome due to*

Dr. Saurabh Zanwar	*Intravascular Large B Cell Lymphoma* Case Presentation: A seventy-six-year-old female presented with two weeks of generalized weakness and confusion. Associated symptoms included fever, night sweats, and a 10 lb (12.5%) weight loss over 3 months. She denied pain or easy bruising and vitals were normal. On exam she was oriented only to person. There was no lymphadenopathy, hepatosplenomegaly, or focal neurological deficits. Laboratory workup revealed thrombocytopenia (98x109/L), acute kidney injury (Cr 1.18 mg/dL; baseline 0.78 mg/dL), hyperkalemia (5.3 mmol/L), hypercalcemia (corrected 10.9 mg/dL), hyperuricemia (15.6 mg/dL), and normal serum phosphorus (3.9 mg/dL). Serum lactate dehydrogenase (LDH) was markedly elevated (1,507 U/L). A peripheral smear revealed abnormal lymphocytes without schistocytes. Despite normal phosphorus levels and hypercalcemia, hyperuricemia and hyperkalemia in a patient with B symptoms, AKI, and markedly elevated LDH are concerning for tumor lysis syndrome (TLS). It is an oncologic emergency, most often occurring in hematologic cancers undergoing cytoreductive therapy [1,2]. Resulting complications include renal failure, new-onset seizure, cardiac arrhythmia, and sudden death. Overall in-hospital mortality in one study was 21% [3]. While our patient did not meet the Cairo-Bishop definition for TLS, she never received cytoreductive therapy and instead met 3 of 4 criteria proposed by Weeks et al for spontaneous TLS (elevated uric acid, LDH, AKI refractory to volume resuscitation) [1]. Normal saline and rasburicase were administered. CT of the chest, abdomen, and pelvis with IV contrast did not reveal lymphadenopathy or splenomegaly. Serum protein electrophoresis and free light chains were normal. A healthy adult has approximately 1.4 kg of bone marrow that could “hide” tumor burden significant enough to cause spontaneous TLS [2]. Bone marrow biopsy revealed large atypical cells arranged in a snake-like intravascular distribution with co-expression of CD20, CD5, and BCL6 by immunohistochemistry, and partial CD10 by flow cytometry, strongly supporting a diagnosis of intravascular large B-cell NHL (IVLBCL). A PET-CT scan revealed diffusely increased FDG uptake in the bone marrow, skull base, and calvarium. Conclusion: The differential for an intravascular distribution of large atypical B cells includes DLBCL-not otherwise specified and IVLBCL. They can be delineated by location and extent of extravascular involvement, with IVLBCL being predominantly located within blood vessels [4]. Within IVLBCL, there are 3 variants: classic, cutaneous, and hemophagocytic. Classic and cutaneous variants are more prevalent in Western countries. Organ involvement (CNS, bone marrow, lung, spleen) in addition to the skin differentiates classic from cutaneous variants. The hemophagocytic variant is most common in Asian countries and is characterized by reticuloendothelial activation and hemophagocytic histiocytes. Historically, IVLBCL is associated with a poor prognosis [5]. Since the addition of rituximab to CHOP (R-CHOP) for CD20+ IVLBCL, prognosis improved to an 88% complete remission rate, 91% overall response rate, and 81% 3-year OS [6]. Possibly the most important prognostic factor is CNS involvement at diagnosis, making early diagnosis paramount to good outcomes [7].
William Dyke Dr. Stephen John Dr. Afua Nti	*Pepsi: A Doubly Detrimental Drink for a Diabetic- A Case Report* Introduction: A felon infection of a digit is not an uncommon presentation to

	the hospital, and can lead to life threatening sepsis. Cuts, scrapes, and puncture wounds are all potential entry points for an infection and can be caused by a variety of traumatic mechanisms that disrupt the skin barrier. Diabetics with neuropathy and poor vascularity are at increased risk of unidentified foot wounds, but the hands also remain at risk for injury secondary to activities of daily living. Case Presentation: A 69 year old male with history of uncontrolled type 2 diabetes mellitus, hypertension, atrial fibrillation, and bilateral transtibial amputations secondary to chronic foot ulcers, presented to the hospital with altered mental status. Non-con CT of the head did not reveal acute intracranial hemorrhage, electrolytes within normal limits, but labs were notable for elevated white count, CRP, ESR, lactate, and hyperglycemia. Patient was febrile, tachycardic with irregularly irregular rhythm. Right thumb markedly swollen, erythematous, and warm with redness extending into the thenar eminence and paronychia. The patient is unable to recall any injury to his thumb. A clinical diagnosis of sepsis secondary to right thumb cellulitis was made. Blood cultures were obtained and broad-spectrum antibiotic coverage with vancomycin and zosyn was started. Xray of the thumb was negative for gas, foreign body, or osteomyelitis. Orthopedic surgeons performed bedside irrigation and debridement. Collateral history was obtained from the patient's wife who noted that the patient injured his thumb nail while opening a can of Pepsi Cola. Blood cultures returned 4/4 bottles positive for Methicillin Susceptible Staph aureus and antibiotics were narrowed to IV cefazolin. The patient's altered mental status and vital abnormalities improved with antibiotic treatment. He was discharged on a 6 week course of antibiotics (extended treatment duration as he had a cardiac pacemaker). Conclusion: While the treatment of bacteremia with IV antibiotics is commonplace, we found the mechanism of injury to be unusual- suffering onycholysis from the tab of a pop can leveraging the nail from the nailbed. Ironically, regular consumption of sugar beverages likely exacerbated his uncontrolled diabetes, which increased his infection risk from the Pepsi can injury. It is important to understand the synergistic nature of diabetic neuropathy leading to progression of infection that affected blood glucose management which further hindered prompt treatment in this patient. Learning points: Impaired sensation from diabetic neuropathy can lead to delayed presentation from injury because of lack of sensation. As his thumb infection worsened and became bacteremia, he was unable to keep up with insulin to manage his diabetes. His altered mental status can be attributed to infection, but was likely also exacerbated by hyperglycemia. Additionally, based on this experience we can only recommend twist tops for at risk soda drinkers.
Salma Hassan Khuaten Maaneb de Macedo	*PEA as a Rare Cardiac Presentation of Pheochromocytoma* Introduction: Pheochromocytomas are rare, catecholamine-producing tumors with a typical clinical presentation including refractory hypertension, headaches, palpitations, and diaphoresis. The initial presentation may include cardiac complications such as heart failure, ischemic heart disease, myocardial infarction, or arrhythmias. Although studies have linked rare incidences of cardiac arrest as the initial presentation of pheochromocytoma, pulseless electrical activity (PEA) as an initial presentation has not been

	reported. We discuss a patient who was diagnosed with pheochromocytoma during the workup of an episode of PEA. Case Presentation: A 60-year-old male with a history of hypertension, anxiety, and cervical myelopathy underwent an anterior cervical corpectomy and fusion. Postoperatively, the patient appeared disoriented and short of breath. Ativan was administered for anxiety. Within an hour, he developed a recurrent cough and subsequently became unresponsive to sternal rub. He was found to be apneic and in PEA arrest. One round of CPR was initiated with epinephrine, followed by intubation. Return of spontaneous circulation was achieved within 5 minutes. The patient continued to be persistently tachycardic and hypertensive. The cause of the PEA was unknown; it was initially thought to be secondary to hypoxemia due to possible aspiration. An EKG showed no signs of ischemia. No abnormalities were noted on echocardiogram. Electrolytes were within normal limits. The CT chest was unremarkable. However, a CT abdomen/pelvis revealed a right adrenal mass. His plasma free normetanephrine level was 27.20 (0.00-0.89) and his plasma norepinephrine was 4,473 (80-520), supporting a diagnosis of pheochromocytoma. The patient later underwent laparoscopic adrenalectomy, which he tolerated well without complications. He was discharged home on lisinopril with a plan to continue outpatient follow-up. Discussion: This case highlights a patient with longstanding hypertension who was diagnosed with pheochromocytoma after PEA arrest. In patients who present with cardiac abnormalities, particularly those with hypertension, pheochromocytoma should be considered when developing a differential diagnosis. Although initial presentations of cardiogenic shock or arrest are rare, about 12 percent of patients with pheochromocytomas present with some form of cardiac complication.2 Multiple pathogenic mechanisms have been described regarding the cardiovascular complications of pheochromocytomas. Various arrhythmias, including ventricular fibrillation, have been described to have an association with surgery in patients with pheochromocytomas.5 However, episodes of PEA as a presenting sign have not been well documented. Pheochromocytomas require prompt recognition to prevent morbidity and mortality. In retrospect, the patient had a two-year history of excessive sweating and intermittent palpitations along with a 10-year history of hypertension. A thorough history may have resulted in earlier consideration of pheochromocytoma. Detecting pheochromocytoma requires a high degree of clinical suspicion and early consideration, thus, pheochromocytoma should be included in the differential diagnosis when patients present with cardiac abnormalities.
Maryam Omar Dr. Meghan Hill	*Bones, Groans, Thrones, and Immune Checkpoint Inhibitor Overtones* Introduction: Immune checkpoint inhibitors have become a common treatment for many malignancies. Though these therapies can be highly effective, they carry the risk of many potential adverse effects. Case Presentation: A 72-year-old woman presented with two weeks of progressive weakness, fatigue, and confusion. Her medical history was notable for metastatic Merkel cell carcinoma and chronic kidney disease with a remote history of two renal transplants.

	Methods: The patient received her first infusion of pembrolizumab, an immune checkpoint inhibitor (ICI), two weeks prior to presentation and) after progression of her malignancy on other therapies. She presented to the emergency department due to profound weakness and a fall. The patient was hemodynamically stable at presentation. Initial laboratory testing was notable for a total calcium of 13.7 mg/dL (increased from 9.6 mg/dL 11 days prior), ionized calcium of 6.8 mg/dL, and parathyroid hormone (PTH) of 8.6 pg/mL. The patient was treated with intravenous normal saline, zoledronic acid, and calcitonin for her severe and symptomatic hypercalcemia. Evaluation for the etiology of her hypercalcemia included repeat PTH, phosphorous, and vitamin A levels which were low. Thyroid-stimulating hormone, 1,25-dihydroxy vitamin D, 25-hydroxy vitamin D, and early morning cortisol were within normal limits. Reconciliation of the patient's medications, diet, and supplemental vitamin and mineral intake did not suggest increased calcium consumption. Parathyroid hormone-related peptide (PTHrP) level returned elevated at 19 pg/mL, indicating ICI-induced hypercalcemia. The patient's calcium normalized, and she eventually required calcium supplementation for hypocalcemia. Discussion: The most common cause of hypercalcemia is primary hyperparathyroidism. Hypercalcemia with a low PTH typically suggests a malignant etiology: humoral hypercalcemia of malignancy (PTHrP-mediated), granulomatous diseases (1,25-dihydroxyvitamin D-mediated), or plasma cell dyscrasias (mediated by osteolytic lesions). Conclusion: Hypercalcemia arising from a malignancy has a poor prognosis and is found in up to 30% of cancer patients. Thus, it is critical to obtain PTHrP and vitamin D levels in the evaluation of patients with hypercalcemia and a history of malignancy. Our patient had Merkel cell carcinoma with severe hypercalcemia and elevated PTHrP. There has only been one reported case of Merkel cell carcinoma producing PTHrP. Our patient was treated with Pembrolizumab shortly before exhibiting symptoms of hypercalcemia. Pembrolizumab, an immune checkpoint inhibitor, is a monoclonal antibody targeted at programmed cell death protein-1 (PD-1) receptors, preventing cancer cells from utilizing it to evade detection. Several cases of ICI-driven PTHrP production have been described, leading us to believe that our patient's case of hypercalcemia is likely due to ICI-mediated PTHrP.
Alexander Roth Dr. J. Michael Bostwick	*Biopsychosocial Analysis of Non-MDD Suicidality in the Setting of Malignant Alcohol Use and Dependency* Introduction: The psychiatric condition most classically associated with suicidality is major depressive disorder (MDD). However, too strong a reliance on this association to identify or treat suicidality may be misguided. This case illustrates how depression does not have to figure prominently in suicidality, and how a psychosocial approach may offer more useful tools to treat non-MDD suicidality than a biological one, particularly in the acute treatment phase. Case Presentation: TD was a 47-year-old man presenting to the ED after morphine overdose while intoxicated. On presentation, TD was hypoventilatory, tremulous, and actively suicidal. Serum alcohol was 263 mg/dL, and UDS revealed opioids. Thus he was admitted to the medical floor. During medical stabilization, frank suicidality resolved. Due to continued hopelessness, TD was transferred to inpatient psychiatry.

	Biologically, TD did not meet criteria for MDD, but rather complicated grief. Excessive drinking—especially while alone—was associated with the overdose and two previous suicide attempts as well as gastrohepatitis and BMI of 18.2. Genetically, two cousins had committed suicide. Psychologically, he was grappling with several severe stressors, including his wife's protracted cancer death, repeat DUI charge with job loss, and loss of visiting privileges with his children. A main coping mechanism was alcohol use, but he at first adamantly denied the link between his episodic catastrophic thinking and rapid decompensation. Socially, he lived alone. His support network included three ex-wives, a sober friend, and a tightknit Mormon family. However, in the setting of DPD traits of submissiveness, clinginess, and codependent self-worth, he felt lost. He saw value in his ex-son-in-law's willingness to move in to keep him company. Services rendered included safety planning; mental health OT for coping skills; and motivational interviewing including validation, reframing, and reflection. A Rule 25 chemical dependence (CD) assessment recommended residential CD treatment. Discussion: During his hospitalization, TD progressed from hopelessness to confidence while developing a comprehensive safety plan. He also gained significantly improved insight into alcohol's deleterious role in his life—e.g., he proactively evaluated CD treatment options and voluntarily committed to a residential program. Notably, psychosocial rather than biological interventions drove his rapid improvement; he learned skill-based tools for combatting isolation, loneliness, and poor coping. While antidepressants e.g. SSRIs could help decrease vulnerability to future stressors, direct modification of current stressors in acute non-MDD suicidality may prove to be sufficient, effective treatment. Many providers seem to reflexively prescribe SSRIs for suicidality, whether ± MDD. This may be rooted in educational failure or a mistaken belief that antidepressants are standard of care. Implications: PCPs should evaluate for suicidality, independent of the presence of MDD, and consider psychosocial rather than biological interventions in MDD's absence. Further, psychosocial therapy for non-MDD suicidality should be highlighted in medical curricula, and providers should improve their awareness of the construct. Lastly, further research into treatment for non-MDD suicidality may improve outcomes in this lesser studied group.
Alexander Schmidt Dr. Meghan Hill	*A Connection That Took Her Breath Away* Introduction: Chronic pancreatitis is common and can manifest complications associated with significant morbidity, including pancreatic insufficiency, biliary obstruction, pleural effusion, vascular complications, and increased risk of malignancy. Case Presentation: A 77-year-old woman presented to the emergency department for evaluation of shortness of breath. She reported four weeks of epigastric pain with abdominal bloating and two weeks of progressive exertional dyspnea. Her medical history was significant for recurrent pancreatitis, alcohol use disorder, intraductal papillary mucinous neoplasm, cholecystectomy, hypertension, and obstructive sleep apnea. On physical examination she was afebrile, blood pressure was 147/95 mmHg, heart rate

	106 beats per minute, respiratory rate 19 to 30 per minute, and saturating 89% on room air. Initial laboratory studies were notable for leukocytes 13x10(9)/L, sodium 126 mmol/L, lipase 573 U/L, and c-reactive protein 153.9 mg/L. Chest x-ray showed a large left pleural effusion. Computed tomography imaging of the chest and abdomen confirmed a large left pleural effusion with near complete collapse of the left lung, marked pancreatic inflammation, and multiloculated rim enhancing fluid collections in the left paracolic gutter. A CT-guided drain was placed into the abdominal fluid collection and thoracostomy tube was placed to control the pleural effusion. Intravenous piperacillin-tazobactam was subsequently initiated to treat possible intraabdominal abscesses. Pleural fluid analysis suggested an exudative process, per Light's criteria. Pleural fluid amylase was significantly elevated at 12,900 U/L (serum amylase 186 U/L), suggestive of pancreaticopleural fistula (PPF). Hypoxia and dyspnea resolved with resolution of the pleural effusion and antibiotics were discontinued due to negative fluid culture. The patient subsequently underwent successful ERCP-guided pancreatic stent placement and was discharged with close outpatient follow up. Discussion: While some pancreatic fistulas are iatrogenic in nature, others may be a rare complication of recurrent or chronic pancreatitis. Fistulas form following disruption of the pancreatic duct leading to pancreatic fluid leakage and surrounding tissue erosion. Based on the anatomic position of the ductal disturbance, pancreatic fluid can drain to a variety of locations, including the pleural cavity. For patients who develop PPFs, symptoms might include cough, dyspnea, dysphagia, chest pain, and unilateral or bilateral pleural effusions. Although our patient presented with epigastric pain, dyspnea, and a history of recurrent pancreatitis, it is difficult to diagnose a rare complication like PPF without proper laboratory evidence. Pleural effusions secondary to PPF are usually exudative, but it was the significantly elevated pleural amylase that made the diagnosis of PPF. Providers must maintain a high index of suspicion for possible PPF in patients with pancreatic disease and pleural effusion in order to obtain the necessary diagnostic testing and initiate the appropriate interventions.
Katie Schmitz Dr. Kirsten Shaw **2021 MN-ACP Medical Student People's Choice Award**	*Stress…Congest…Repeat: Super-Recurrence of Takotsubo Cardiomyopathy* Introduction: Takotsubo cardiomyopathy (stress cardiomyopathy) is the etiology of 1-2% of troponin positive presentations of ACS. It typically affects the apex of the heart, sparing the base, appearing as an asymmetrical balloon or octopus trap (takotsubo) on imaging and the majority of cases are reversible. This condition typically occurs as an isolated event, but recurrence has been documented. Knowing that recurrence is a possibility, internists can speed diagnosis and help provide focused management. Case Presentation: A 72-year-old female with a past medical history of NSTEMI s/p DES, takotsubo cardiomyopathy, hypertension, hypercholesterolemia, angina, chronic systolic congestive heart failure with CRT placement, type 2 diabetes, and anxiety presented to the emergency department with 3 hours of chest pressure not relieved by nitroglycerin and associated radiation to the jaw and nausea. In the ED, she was stable and EKG showed electronic pacing and initial troponin I was normal. She was admitted and her troponin I steadily increased, peaking at 0.333, IV heparin was initiated and cardiology was consulted for possible angiography.

	Methods: Echocardiogram revealed moderately reduced global systolic function with an estimated EF of 35% and multiple segmental wall motion abnormalities, suggestive of takotsubo cardiomyopathy. Coronary CT angiography showed patent coronary arteries with no evidence of an ischemic event. She was medically managed for her takotsubo cardiomyopathy with beta blockers, and isosorbide dinitrite in addition to her chronic aspirin, ranolazine, and rosuvastatin for comorbid CAD. She improved and was discharged to home after three days. One month post discharge her echocardiogram was normal with no wall motion abnormalities and EF of 55%. Upon further chart review, it was discovered that this was the patient's 8th documented episode of takotsubo cardiomyopathy. She reported that she typically has one to two episodes of cardiomyopathy per year that are linked with significant emotional stressors in her life (her brother's death, daughters' marital trouble, dog's illness etc.). To our knowledge, this is the highest number of recurrent episodes of takotsubo cardiomyopathy documented in the medical literature. Conclusion: Takotsubo cardiomyopathy is an important clinical diagnosis whose incidence is increasing. A JAMA study found that incidence of takotsubo cardiomyopathy has increased by roughly 8% in the setting of the COVID-19 pandemic. Takotsubo cardiomyopathy disproportionately impacts women and one study found 55.8% of patients with takotsubo cardiomyopathy had psychiatric or neurological disorders compared to 25% of patients with ACS. This case demonstrates that although stress cardiomyopathy recurrence is rare, it should be included in the differential. Additionally, these patients should have multifactorial management that treats not only the physiological symptoms of the cardiomyopathy, but also targets the psychological stressors that caused the pathology. Proper management of patient's symptoms may help prevent recurrence.
Anja Touma Dr. Georgios Manousakis Dr. Prabhjot Nijjar	*A Rare Case of Severe Cardiomyopathy Associated with Myotonic Dystrophy Type 2* Background: Myotonic dystrophies (DM) are multisystemic diseases characterized by muscle weakness and myotonia. DM2 (myotonic dystrophy Type 2; proximal myotonic myopathy; PROMM) is an autosomal dominant multisystemic disorder caused by a microsatellite repeat expansion within intron 1 of the zinc finger protein 9 (ZNF9; also known as CNBP) gene. DM2 is generally characterized by a less severe phenotype than DM1 (myotonic dystrophy type 1). Despite a growing appreciation for the cardiovascular manifestations in DM1, cardiac involvement in DM2 has been less well characterized. In patients with DM2, cardiomyopathy has rarely been described. Case Presentation: This clinical vignette describes a rare case of DM2 associated cardiomyopathy in a 56-year-old male with DM2 who presented with palpitations, fatigue, reduced exercise tolerance, atypical chest pain, and lower extremity edema. He also reported proximal progressive muscle weakness for the past 20 years. Genetic testing confirmed DM2 due to a ZFN9 mutation. Physical exam showed normal vital signs, no jugular venous distention or edema, clear lungs, regular heart rate and rhythm without murmurs, and mild proximal muscle weakness. He additionally had hypertension, dyslipidemia, prior tobacco use (15 pack-years), well-controlled

	type 2 diabetes mellitus, and hypothyroidism. The differential diagnoses included coronary artery disease (CAD), ischemic cardiomyopathy, muscular dystrophy associated cardiomyopathy, other non-ischemic cardiomyopathies, and heart failure with preserved ejection fraction. He had an elevated creatine kinase and N-Terminal Pro BNP, but normal Troponin I. An ECG demonstrated normal sinus rhythm with inverted T waves in V4 and flat T waves in V5-6. A 72-hour Zio Patch monitor recorded sinus rhythm with rare ectopy and no recorded symptoms. Cardiac magnetic resonance imaging (CMR) confirmed a severely enlarged left ventricular cavity with a left ventricular ejection fraction of 28% consistent with severely reduced global systolic function. The lateral wall epicardium exhibited late gadolinium enhancement in a pattern seen in myotonic dystrophy-related cardiomyopathy. Regadenoson stress perfusion imaging showed no ischemia. His non-ischemic myotonic dystrophy-related cardiomyopathy was treated with goal-directed, maximally tolerated neurohormonal therapy including lisinopril, metoprolol succinate, and spironolactone. Ultimately the patient also received an ICD for sudden cardiac death primary prevention. Conclusion: This case highlights the potential for significant cardiovascular involvement in myotonic dystrophy Type 2, as well as, the importance of cardiovascular screening, including cardiac magnetic resonance imaging (CMR), and therapy in the myotonic dystrophy patient population.
Beija Villalpando	*Warfarin Skin Necrosis in a Patient with Acquired Protein C Deficiency* Case Presentation: A 67-year-old male with cirrhosis secondary to primary sclerosing cholangitis (PSC), ulcerative colitis (UC), and distant history of venous thromboembolism with pulmonary embolism currently on warfarin, presented to the emergency department for a painful area of redness and bruising on his left upper arm after his wife lightly touched his arm two days prior. He denied left upper extremity numbness, chest pain, or shortness of breath. He did report greater than ten episodes of daily non-bloody loose stools beginning five days prior, which he treated as an UC flare. His physical exam showed tender, erythematous to violaceous, plaques on his left posterior upper arm concerning for warfarin skin necrosis. Warfarin was discontinued and replaced with an intravenous heparin drip. Lab work showed supratherapeutic INR of 4.7, fibrinogen of 185, and D-dimer of 31775. His coagulation factors II, VII, IX, and X and protein C and S were decreased. After receiving three units of fresh frozen plasma and vitamin K throughout his hospitalization, a repeat fibrinogen panel was normal, and D-dimer and INR were downtrending daily. His clinical presentation remained consistent with warfarin skin necrosis with acquired protein C deficiency secondary to his significant liver disease. Given the patient's prior history of autoimmune conditions, the differential diagnosis was broadened to include autoimmune vasculitis. The extensive immunology panel was only positive for perinuclear anti-neutrophilic cytoplasmic autoantibodies, which was previously positive when he was diagnosed with PSC. To evaluate for calciphylaxis, parathyroid hormone, calcium, and phosphorus levels were obtained. His parathyroid hormone was normal, while calcium, 8.4, and phosphorus, 2.3, were minimally lowered. Ultimately, a deep, wedge excisional biopsy down to the fascia was obtained from his left upper extremity wound to evaluate for warfarin skin necrosis, vasculitis, and calciphylaxis. The final pathology report showed dermal and subcutaneous

	fibrin thrombi and extravasation of red blood cells consistent with warfarin necrosis. With a definitive diagnosis, warfarin was discontinued indefinitely. A direct-acting oral anticoagulant was avoided given the patient's Child Pugh B score. Unfortunately, a direct thrombin inhibitor was cost prohibitive. Ultimately, Lovenox 90 mg twice daily was chosen. Follow-up was scheduled with wound care and Thrombophilia Clinic to discuss long-term anticoagulation. Discussion: Warfarin skin necrosis is related to low levels of protein C, a vitamin K-dependent plasma protein with anticoagulant properties (1). Acquired protein C deficiency can result from chronic UC, which predisposes UC patients using warfarin to warfarin skin necrosis (2,3). Late onset warfarin skin necrosis lesions, such as this patient's presentation, can be difficult to diagnose (4). Thus, it is important to maintain a broad differential. When anticoagulation options are limited, as with this patient, a biopsy may be obtained to confirm warfarin necrosis and guide future anticoagulation management.
Micaela Witte Dr. Savannah Liddell	*Hemoptysis: to bronch or not to bronch?* Introduction: Hemoptysis is a common condition with a broad differential diagnosis including respiratory infection, malignancy, and pseudo-hemoptysis due to epistaxis. This diagnostic complexity may pose a significant risk to those with previously treated non-small cell lung cancer due to a high recurrence risk. Case Presentation: An 81-year-old female was admitted to inpatient medicine with a two-week history of coughing up bloody sputum. Her medical history was significant for 20 pack-years of smoking; stage 2B squamous cell of the lung treated with curative intent with lobectomy, radiation, and chemotherapy one year prior; malignancy-associated dermatomyositis currently quiescent on IVIG; multinodular goiter status-post thyroidectomy; acute bronchitis; and allergic rhinitis with recent addition of fluticasone nasal spray. The hemoptysis had progressively worsened, and she was expectorating one-half teaspoon of blood four times daily. She had also noticed nosebleeds, increased sputum production, shortness of breath, and subjective fevers. She had no recent travel history, sick contacts, weight loss, or history of blood clots. She was taking 81 mg of aspirin daily, but no other antiplatelet or anticoagulating drugs. Vital signs were within normal limits. Methods: Head and neck exam revealed no lymphadenopathy or oronasal lesions. Pulmonary exam was remarkable for intermittent wheezing in the right lung fields without crackles. Labs showed an unremarkable CBC and basic metabolic panel, normal INR, negative COVID swab, and negative antineutrophil cytoplasmic antibodies (ANCAs). CT chest angiogram showed no pulmonary embolism, infiltrates, masses, or lymphadenopathy. Because symptoms were suggestive of respiratory infection, ceftriaxone and azithromycin were started. After 24 hours of stability with minimal hemoptysis, she experienced seven episodes of tablespoon-size, bright red sputum over six hours. She remained hemodynamically stable and continued to breathe comfortably on room air. Because of increasing hemoptysis, bronchoscopy was performed and revealed a small bleeding lesion suspicious for malignancy. The site was biopsied and treated with argon plasma coagulation. Pathology results revealed local recurrent squamous cell carcinoma of the lung that was 40% PDL1 positive. She began palliative

	pembrolizumab therapy. Conclusion: This case illustrates the need for a broad differential in the diagnosis of hemoptysis as well as the high suspicion for recurrence in patients with previously treated non-small cell lung cancer. Although infection is the most common cause of hemoptysis, the possibility of recurrent malignancy is quite high for patients with non-small cell lung cancer even with appropriate treatment. Initial diagnostic evaluation for hemoptysis may include chest x-ray and CT chest. However, bronchoscopy should be considered if this testing is negative and the index of suspicion for recurrence is high.
Catherine Yang Dr. Christopher Dinh	*The Rollercoaster of Central Diabetes Insipidus - where only bedside clinical data can apply the brakes* Introduction: Central diabetes insipidus (DI), characterized by hypotonic polyuria of ≥3 liters in 24 hours despite fluid restriction, is caused by vasopressin deficiency from the posterior pituitary gland. Patients with central DI classically present with polyuria and polydipsia, which can result from a variety of causes including tumors, surgery, and most commonly, idiopathic. Desmopressin, the treatment for central DI, can be administered orally, intranasally, or parenterally. The newer oral form is preferred by most patients due to ease of administration. The most common complication of desmopressin treatment is hyponatremia, which evidence suggests is less common with the oral than the intranasal formulation. Case Presentation: A 43-year-old female group home resident was admitted from an acute rehabilitation unit with subacute progressive headache and confusion. Her medical history was notable for intellectual disability and craniopharyngioma, status post distant surgical resection and radiation, complicated by panhypopituitarism and hypothalamic dysfunction. Her central DI had been previously well-managed with oral desmopressin until two hospitalizations within the last month for both hyper and hyponatremia. At the time of presentation, sodium was 151 mmol/L from 129 mmol/L earlier that day. For her symptomatic hypernatremia, she received 5 liters of D5W and 80 mcg of intranasal desmopressin, which corrected sodium to 135 mmol/L. Over the following days, desmopressin was titrated to the lowest effective dose by monitoring hourly urine output and frequent serum sodium level. Due to frequent hyponatremia after desmopressin administration, each dose of intranasal desmopressin was administered only when both urine output increased and serum sodium normalized. In this manner, the desmopressin dose was progressively decreased to 10 mcg every 12 hours. On this regimen, she maintained normal sodium without polyuria and was discharged with endocrinology follow-up. Ultimately, it was felt that irregular absorption of oral desmopressin led to her labile serum sodium. Discussion: This case of central DI with rapid and somewhat unpredictable swings between both hypernatremia and intervention-induced hyponatremia illustrates the importance of careful desmopressin dose titration by monitoring both serum sodium and urine output. Close monitoring is important for two reasons. First, polyuria in patients with central DI can lead to rapid increases in serum sodium and subsequent osmotic demyelination syndrome. Second,

	intranasal desmopressin cannot be empirically dosed and must be titrated to each patient based on urine output due to wide individual variability in drug pharmacokinetics and pharmacodynamics. Specifically, intranasal desmopressin is approximately 10 to 40-fold more potent than oral desmopressin and its clinical effect lasts between 5 to 24 hours. Given the critical importance of measuring and communicating urine output in an accurate and timely fashion to prevent adverse outcomes, this case also highlights the importance of teamwork and communication between bedside nurses and the medical team.
Residents	
Quality Improvement - Residents	
Kristen Westenfield **2021 MN-ACP winner Resident-Research/Quality Improvement**	*Under Pressure...Ambulatory Blood Pressure Monitoring in the Diagnosis and Management of Hypertension* Background: Ambulatory blood pressure monitoring (ABPM) is an increasingly accessible clinical tool used to assess blood pressure. ABPM provides a more accurate physiological description of systemic blood pressure throughout a 24-hour time period than isolated office measurements. It can be used to diagnose sustained hypertension, exclude white-coat hypertension, and identify masked hypertension. The results of an ABPM can lead a provider to alter a medication regimen. In this project we looked at ABPMs performed at two Allina Health primary care clinics between November 2020 and August 2021. All ABPMs met criteria for hypertension and the indications for performing the ABPMs included elevated office blood pressure readings without diagnosis of hypertension, question of white coat hypertension, and uncontrolled previously diagnosed hypertension. Hypertension was defined by the European Society of Hypertension practice guidelines as blood pressure greater than 130/80 mmHg, awake greater than 135/85 mmHg and/or asleep blood pressure greater than 120/70mmHg. Methods: In total, a 131 ABPM evaluations were performed. The average patient was 64 years old and 50% were females. From the 131 ABPM evaluations, there were 65 new diagnoses of hypertension. Based on the results of the ABPM, a medication change was made in 78% of the cases with the addition of a new medication, dose adjustment, and cessation of a medication in 65.4%, 12.8%, and 0.8% of the time, respectively. The most common new antihypertensive class added to a patient's medication regimen was an angiotensin converting enzyme inhibitor or angiotensin receptor blocker (added 31% of the time). The second most frequent addition, a calcium channel blocker, was added in 28% of cases. Conclusions: Overall, this data demonstrates that providers are frequently making changes to patients' medical regimen based on the results and findings in ABPM studies. Limitations to this project include a predominantly caucasian patient population. In the future, this project could expand to include interval follow up ABPMs to see if blood pressure was adequately treated based on the initial medical changes.
Lisa Peng Dr. Breanna Zarmbinski Jacob Langness, Pharm D	*10 Pills a Day Does Not Keep the Doctor Away* Background: Polypharmacy (defined as > 5 medications) has been associated with higher morbidity and mortality and increased risk for adverse medical outcomes. Risk factors for polypharmacy include older age, multiple chronic

	conditions, seeing multiple subspecialists, and poor medical record keeping in the healthcare system. To help improve outcomes in patients with excessive polypharmacy in our resident clinic, we enrolled patient's with >10 medications to undergo a series of comprehensive medication reviews with our pharmacist and their primary care provider (PCP) in an effort to maintain an accurate medication list. Methods: Patients could be included in the study if they were on >10 medications between October 2020 and August 2021. A comprehensive medication review was completed by our clinic pharmacist and total number of medications and any medication discrepancies were noted. The patients then completed a clinic appointment with their primary care provider dedicated to medication list review and education. Medications were again reviewed via pharmacist phone visit at 1 month and 6 months following PCP visit and discrepancies and total number of medications were noted again. Frequency of medication discrepancies, common categories of medication discrepancies and change in total number of medications were compared over the series of follow ups. Results: 45 patients are actively enrolled in this study. 51% of these patients did not complete their dedicated PCP visit immediately following their initial pharmacist visit, 57% of them due to needing an acute visit (clinic, ED or hospitalization) first before their PCP visit. Of the patients enrolled, 100% of them had >1 medication discrepancy following initial pharmacist review. The most common reasons for medication discrepancies were: no longer taking a medication(s) (56%), medication(s)/supplement(s) added (36%), and dose adjustment(s) (22%). Of the 9 patients that completed the study at the time of this review, 33% had a smaller medication list at 1-month follow up compared to the start and 22% had a smaller medication list at 6-month follow up compared to the start. Conclusions: As the literature has shown, polypharmacy is a risk factor for increased morbidity and mortality. Our study demonstrated this finding because more than half of patients in this study required an acute visit (clinic, ED or hospitalization) before they were able to see their PCP for their dedicated polypharmacy visit. We also found excessive polypharmacy was associated with medication list inaccuracy and that the use of collaborative pharmacy review was able to capture many of these inaccuracies. However, it was not associated with a reduction in total number of medications at follow up in the majority of our patients.
Research - Residents	
Ashley Draisey Dr. Kristen Westenfield Dr. Brian Swiglo	*Hypophysitis due to Checkpoint Inhibitors* Introduction: Immunotherapy has changed the field of oncology allowing for treatment of previously untreatable cancers. Checkpoint inhibitors (CPIs) block checkpoint proteins from binding with partner proteins, allowing for T-cell activation and destruction of cancer cells. While CPIs such as anti-program death (PD-1) and anti-cytotoxic T lymphocyte antigen-4 (CTLA-4) produce beneficial anti-tumor responses, they can result in immune-related adverse events (irAEs). Endocrine organs are commonly affected, resulting in conditions including hypophysitis. We report two cases of males who developed hypophysitis after treatment with a CPI. The first patient had a history of melanoma with metastasis to the

	lung and was initially treated with nivolumab (PD-1 inhibitor) and later impilimumab (CTLA-4 inhibitor). He developed autoimmune cutaneous toxicity so impilimumab was stopped. Nivolumab was continued for an additional 11 cycles before he was hospitalized for ongoing nausea, vomiting, and a 40 lb weight loss. The second case was a man with merkel cell carcinoma admitted to the hospital with fatigue, anorexia, and nausea with vomiting after receiving 7 cycles of pembrolizumab (PD_1 inhibitor). In both cases laboratory evaluation was consistent with secondary adrenal insufficiency and hypophysitis from CPI therapy. The CPI therapy was stopped. Both patients were started on glucocorticoid replacement therapy with complete resolution of symptoms at follow up visits. Conclusion: These cases demonstrate an increasingly common development of irAEs associated with CPIs. Given the growing number of patients treated with CPIs, providers will need to be aware of potential irAEs such as adrenal insufficiency which can be severe and potentially life threatening.
Reid Eggleston Dr. Thomas Hartman Dr. Laura Walkoff Dr. Eunhee S Yi Dr. Jay H Ryu Dr. Misbah Baqir	*Pulmonary ATTR Amyloidosis: Clinical, Radiologic and Pathologic Features and Outcomes* Introduction: Transthyretin amyloidosis is a disorder characterized by deposition of a misfolded conformation of the transport protein transthyretin (ATTR) most commonly in cardiac and nervous tissue causing clinical disease. Pulmonary amyloidosis, or deposition of ATTR in lung tissue, is a poorly characterized manifestation of this disease. We present the clinical course, imaging characteristics, pathology results, and outcomes of a cohort of patients diagnosed with pulmonary amyloidosis at an academic institution. Methods: We performed a retrospective descriptive review of 28 patients at Mayo Clinic between 2005 and 2020 with pulmonary ATTR amyloidosis (89% male, age at diagnosis 50-99 years). Data collected included patients' demographics, subjective symptoms, tissue biopsy results, and pulmonary function testing, imaging, and treatment data. Results: Patients were typically diagnosed after persistent dyspnea and abnormal chest imaging resulted in bronchoscopy yielding ATTR amyloidosis on biopsy. Most had a pre-existing diagnosis of cardiac ATTR amyloidosis. A similar number of patients had wild-type and hereditary disease. Patients typically had mild restrictive lung dysfunction with moderate reduction in diffusion capacity for carbon monoxide. Ninety-three percent had chest computed tomography imaging most commonly showing diffuse nodularity. Several patients had pulmonary cysts, pleural effusions, or noncalcified lymphadenopathy. Almost all patients had pulmonary vascular involvement and half had interstitial involvement on tissue biopsy. One-third received either anti-amyloid pharmacotherapy or organ transplant. Half of patients died by the time of study inclusion [Jan. 1, 2005-Dec. 31, 2020]. Conclusion: Pulmonary disease is a less common, but clinically important manifestation of ATTR amyloidosis. While there is heterogeneity in the presentation of these patients, there is a common symptomatology and disease course. Anti-amyloid therapies are a promising development with associated mortality benefit.
Benjamin Krehbiel Dr. Adam Wolfe Dr. David Tierney	*Outcomes and Clinical Characteristics of COVID-19 in Patients with Tuberculosis* Introduction: COVID-19 illness severity and mortality is associated with

	certain comorbidities, such as increased age, diabetes, cardiovascular disease, and chronic obstructive pulmonary disease. Tuberculosis (TB) disease severity and outcomes correlate with similar comorbidities. Literature on COVID-19 infection in patients with TB is limited. This study evaluated the clinical characteristics, illness severity, and mortality of COVID-19 infection in patients with TB compared to patients without TB. Methods: This retrospective cohort study, conducted with the Allina Health COVID-19 registry included all in- and outpatients with a (+) COVID PCR within the Allina Health System between March 1, 2020 and January 14th 2021. The TB cohort included patients with an accompanying new or prior diagnosis of TB. Assessment of disease severity for the TB and non-TB cohorts included rates of clinic and emergency department visits, hospitalization, intensive care unit (ICU) care, length of stay (LOS), repeat hospitalizations, and mortality (in-hospital and 30-days). Univariate analyses were conducted on both cohorts. Results: A total of 33 patients (67% female, mean age 56 years) and 8711 patients (53% female, mean age 54 years) were included in the TB and non-TB groups, respectively. Rate of hospitalizations was 42% vs. 36% in the TB and non-TB cohort, respectively. Patients requiring ICU care was 15% vs 8% (31% vs. 22% of hospitalizations). Mean LOS was 11.9 days (SD±14.5) vs. 8.08 days (SD±7.39). Rate of multiple hospitalizations was 6.1% vs. 5.2%. In-hospital mortality was 3.0% vs 4.1% (p=1.00), and 30-day mortality was 6.1% vs. 5.7% (p=0.71). Prevalence of various comorbidities did not differ significantly between cohorts. Conclusion: In this retrospective cohort study, PCR-confirmed COVID-19 disease and history of TB was associated with higher rates of hospitalization, need for ICU care, and LOS compared to patients without TB. However, a history of TB did not correlate with higher in-hospital or 30-day mortality rates. Limitations of the current study include a small TB cohort sample size and lack of matched cohort. Future studies will include examination of matched cohorts for patient characteristics, therapies, and medical conditions.
Clinical Vignette- Residents	
Sara Medina-Bielski **2021 MN-ACP winner Resident-Clinical Vignette**	*What The A1c Doesn't See* Introduction: Diabetes mellitus is a common disease which primary care providers encounter on a regular basis, and managing diabetes requires close monitor of hemoglobin A1c. Although A1c revolutionized monitoring diabetes, there are factors that can falsely alter the results. Case Presentation: A 65-year-old male came into primary care clinic for follow up on diabetes. He was initially diagnosed with diabetes in 2014 when he was noted to have elevated fasting glucose and an A1c of 8.3%. He and his primary care provider decided to trial lifestyle modification with success as his A1c decreased to 6.9% on repeat measurement. Patient was lost to follow up for several years and when he returned to PCP in 2018, his A1c increased to 11.2% and he was started on metformin. He was seen one year later with an increase in A1c to 14.4% and subsequently started on nightly Lantus and given patient education regarding glucose monitoring. His A1c decreased to 7.7% five months after initiation of insulin. He continued to check his glucose periodically but would not do so regularly due to cost of testing supplies. Over the next two years, his A1c increased to 12%. On evaluation,

	he denied any symptoms of polydipsia, polyuria, vision changes or weight changes. He was adamant that his fasting glucose was 100-130 at home, which did not correlate with the elevated A1c. A fructosamine was therefore obtained which was mildly elevated at 312, correlating to an A1c of roughly 7.5%. As the A1c and fructosamine were discordant, the conclusion was the A1c was an unreliable test in this patient. An initial workup to evaluate for cause of falsely elevated A1c included thorough medication review and social history, lipid panel, BMP and CBC. As the workup proceeds, the patient is being evaluated for possible continuous glucose monitor and will have fructosamine testing done in the future instead of A1c. Conclusion: The A1c is often a reliable test, but this case demonstrates the potential imperfection of the test. The A1c relies on a non-enzymatic reaction between hemoglobin and glucose, so there are many factors that may impact hemoglobin and therefore A1c accuracy. These factors include inherited thalassemias, acquired anemias, certain supplements/medications, renal disease and several other causes. Although false A1c is relatively uncommon, it may be underdiagnosed. It is therefore important to correlate A1c with fasting glucose measurements and use alternative laboratory biomarkers as another measurement if there are any underlying disease processes that may interfere with A1c accuracy.
David Jonason Dr. Guru Trikudanathan **2021 MN-ACP Resident-Clinical Vignette finalist**	*A Case of MALT Lymphoma Masked as Gastric Varices Confirmed by EUS* Introduction: Gastric varices (GV) can cause gastrointestinal bleeding and increase morbidity and mortality in patients with cirrhosis. Upper endoscopy with direct injection of tissue adhesive glue or sclerosing agents is commonly utilized whenever there is no endoscopic ultrasound (EUS) expertise. However, alternative diagnoses can masquerade the endoscopic appearance and presentation of GV leading to suboptimal management and delays in diagnosis. We present a case of MALT lymphoma masked as GV on endoscopy and later correctly identified by Doppler EUS. This case highlights the importance of EUS in the evaluation of GV to aid appropriate management. Case Presentation: A 60-year-old male with a history of alcohol and NSAID use was admitted following a pre-syncopal episode. Physical exam was significant for tachycardia and hypotension. Labs were significant for a hemoglobin of 6.9 g/dL. Upper endoscopy showed isolated large gastric varices with stigmata of recent bleeding. However, there were no signs of cirrhosis with portal hypertension or gastric varices on CT scan. He was referred for EUS-guided coil/glue embolization during which he was found to have a large subepithelial lesion (SEL) in the gastric fundus. Doppler interrogation excluded underlying varix but revealed a large intramural hypoechoic mass. Two smaller SELs were also found, one with a deep ulceration and a visible vessel. Hemostasis was achieved with injection of Epinephrine and placement of hemostatic clips. Ulcers on biopsy with immunohistochemistry showed neoplastic CD 20 positive cells consistent with low grade B-cell lymphoma such as mucosa-associated lymphoid tissue (MALT) lymphoma. H.pylori was negative. He was subsequently referred to oncology for radiation treatment. Discussion: EUS exploration should be offered in cases where direct visual diagnosis of gastric varices with endoscopy is unclear or when there is no evidence of portal hypertension on cross-sectional imaging or in the absence of cirrhosis to exclude alternative diagnosis. EUS can distinguish varices

	located in the submucosa from other causes for prominent gastric folds. In this case, EUS aided in making a timely diagnosis of gastric SEL masquerading as varices which further impacted management. EUS should be preferred over conventional endoscopy for GV as it offers many conceptual diagnostic and therapeutic advantages including real-time imaging that enables detection and precise targeting of perforating and collateral vessels which often accounts for uncontrolled or recurrent bleeding.
Stephanie Kuhlman **2021 MN-ACP Resident-Clinical Vignette finalist**	*Severe Methemoglobinemia After Sodium Nitrite Ingestion* Introduction: 39-year-old female presented after an intentional sodium nitrite ingestion in an attempt to kill herself. Case Description: A 39-year-old female with a history of major depressive disorder, post-traumatic stress disorder, and borderline personality disorder presented to the ED by EMS after a suicide attempt. She reported intentionally ingesting 4 tablespoons of sodium nitrite and quickly after that called 911. She arrived to the ED about 20 minutes after the initial reported time of ingestion. The medic reported that she looked blue/gray and "dead" but was sitting up talking to him, so he rushed her to the ED. On arrival she was briefly awake and speaking but became lethargic and then began to have a seizure. She received lorazepam and Keppra and was intubated for airway protection. She initially received activated charcoal for the ingestion. Her methemoglobin level came back at 73 so she received methylene blue (2 doses total). Her skin was bluish gray on presentation and when the ED physicians placed an arterial line her blood was dark brown. Her blood pressures began to drop and she required at one point up to 3 pressors to maintain adequate blood pressure. Her lactate was 11.7, pH 7.15, and bicarb 11 initially. The SpO2 persistently read in the 60s. In the ED after receiving methylene blue, her methemoglobin level was improved to 29. She was admitted to the ICU and remained intubated. When sedation was stopped, she began to have occasional and focal myoclonus, so propofol was restarted. This developed into full body myoclonus vs seizure activity. She was started on a versed drip at that time with concern for seizure. An EEG was obtained that did not show any seizure activity. About 36 hours after admission she was weaned from sedation and able to follow commands. She was extubated successfully. She continues to have suicidal ideation and is pending admission to inpatient psychiatry. She does not appear to have significant neurologic damage. Discussion: A methemoglobin level reports the percentage of hemoglobin that has been oxidized to the ferric state and cannot carry oxygen. She presented with an initial level of 73% which means only 27% of the hemoglobin in her body was capable of carrying oxygen. With this high of a level it is interesting that she did not have any kidney or liver damage on admission. She appeared to initially have hypoxic brain injury with the myoclonus and non-responsiveness of of sedation, although this now appears to be a toxic effect of the sodium nitrate more so than a hypoxic injury as she demonstrated good neurologic recovery.
Nermine Abdelwahab Dr. Emily Ewan Dr. Kristin Inman	*That Blood Pressure, You Gotta be Kidney-ing Me!* Case Presentation: A 50 year old female with history of bilateral renal cell carcinoma (RCC) s/p partial left nephrectomy, ESRD on hemodialysis, and hypertension was admitted for hypertensive emergency with a blood pressure

	of 240/135 and associated shortness of breath, chest pain, and headache. Labs were notable for elevated troponin and BNP. Initial blood pressure control was obtained with a nitroglycerin drip, however, this was ultimately discontinued due to worsening headache. The patient required initiation of multiple medications including carvedilol, losartan, clonidine, doxazosin, minoxidil, hydralazine, and bumetanide in addition to challenging her dry weight with hemodialysis. Despite the above medications, the patient's blood pressure remained elevated >200/110 requiring transfer to the ICU for nicardipine drip. An inpatient secondary hypertension workup was pursued. Primary aldosteronism, pheochromocytoma, and renal artery stenosis were ruled out. After several multidisciplinary discussions, the patient's hypertension was hypothesized to be driven by RCC in the setting of advanced renal disease, and bilateral nephrectomy was posited as a treatment option. Patient agreed and subsequently underwent surgery. Following post-operative recovery, the patient demonstrated significant improvement in her blood pressure and discharged home on a three drug regimen. Discussion: The pathophysiology for hypertension--and specifically hypertensive crisis--is complex and unclear. Two of the most common mechanisms include: 1) failure of autoregulation, defined as the organ's ability to maintain blood flow regardless of perfusion pressure, and 2) cyclical activation of the renin-angiotensin system. More specific to our patient, patients with ESRD are at increased risk of developing hypertension. Additionally, some research suggests that arterial hypertension in patients with RCC could be driven by secretion of vasoactive peptides. Conclusion: In a patient adherent to an anti-hypertensive regimen of three or more medications, secondary causes of hypertension must also be investigated including primary hyperaldosteronism, renal artery stenosis, Cushing's syndrome, and pheochromocytoma. In this case, there was inability to control blood pressure despite seven antihypertensive medications and hemodialysis. This degree of resistance led to the consideration of invasive procedures. The most common invasive procedures used in the treatment of resistant hypertension include renal denervation, for which there is limited clinical data and is not currently FDA approved in the United States, and nephrectomy. Prior studies have demonstrated significant improvement in blood pressure following nephrectomy in patients with RCC and pediatric patients with renal hypertension. Nephrectomy was likely effective in this case due to the resultant decrease in renin release and subsequent deactivation of the RAS system as well as the possible decrease in vasoactive peptides from RCC. Although bilateral nephrectomy was a high-risk surgery in a patient with residual kidney function, the quality of life desired by the patient, as well as their RCC and exhaustion of conservative options made it a risk worth taking.
Aldo Acosta-Medina Dr. Ronald Go	*VEXAS: A New Constellation to Consider in the Male Patient* Case Presentation: A 57-year-old male with past medical history of type 2 diabetes mellitus was evaluated due to right hemifacial erythema, pain, trismus, and periorbital edema of two days of evolution. Ambulatory antibiotic treatment was initiated due to suspicion of facial cellulitis and was admitted 24 hours later due to progression of symptoms. Maxillofacial imaging was suggestive of infectious sinusitis and broad-spectrum IV antibiotic coverage was initiated. No infectious agent was isolated and, given clinical improvement, patient was discharged to complete a 7-day course of antibiotics.

	Over the next 48 hours, he developed progressive headache with concomitant edema in the right side of his face and neck. Imaging evidenced markedly progressed edema of deep neck spaces leading to readmission and reinitiation of broad-spectrum IV antibiotics. His course mildly improved but was then noted to have progressive increase in volume of right submandibular gland on day +5 of antimicrobial coverage. High-dose steroids were given due to concern for immune-mediated sialadenitis in the setting of highly elevated CRP (106 mg/L). Through this admission he was first noted to have a normocytic anemia (Hb 11.9 g/dL) and leukopenia with borderline neutropenia (2.8 and 1.59 x109/L). Facial and submandibular swelling markedly improved and patient was discharged. The patient was reassessed 3 weeks later given progressive redevelopment of facial swelling, new onset of night-drenching sweats, and unintentional weight loss. CT imaging showed subcentimeter lymphadenopathy in neck and thorax indeterminate for infection versus occult neoplasm. PET CT showed no evidence of high-grade malignancy, ill-defined uptake in the right upper neck and diffuse bone marrow uptake. Extensive laboratory workup including ANA, CCP, SS-A, SS-B, ANCA vasculitis panel, terminal complement levels, and attempts at identification of monoclonal protein were negative. Bone marrow biopsy was performed with subsequent initiation of empiric steroids given prior sialoadenitis response. Near-complete resolution of symptoms was noted upon 48 hours of 10mg prednisone. Bone marrow evaluation demonstrated hypercellularity, left shifted granulopoiesis, no increase in blasts, and minimal maturation atypia with occasional cytoplasmic vacuolation on erythroid and granulocytic elements. Given the patient's clinical course, rapid response to steroid therapy, and bone marrow findings, peripheral blood sequencing was performed demonstrating a Met41Val pathogenic variant in UBA1. Vacuoles, E1 enzyme, X-linked, autoinflammatory, somatic (VEXAS) syndrome was diagnosed. Patient and family have been HLA-typed in anticipation for possible future allogeneic bone marrow transplantation in case of myeloid malignancy. Steroid-sparing strategies for his long-term management, including copper supplementation and ruxolitinib, are being explored. Conclusion: The VEXAS syndrome is a recently described, monogenic entity characterized by inflammatory findings highly responsive to steroids which should be considered when in the unexplained constellation of macrocytic anemia and thromboses in a male patient. Bone marrow evaluation should be pursued in all cases of unexplained bicytopenia.
Chine AK Dr. Christopher Dinh Dr. Jennifer Gile Dr. Zongming Chen	*Amyloidosis: An Unusual Case of Gastrointestinal Hemorrhage* Introduction: Gastrointestinal involvement is common in amyloid light chain (AL) amyloidosis, however gastrointestinal hemorrhage secondary to amyloidosis is rare. Case Presentation: A 70-year-old man with a known history of AL amyloidosis and acquired factor X deficiency presented to the ED with melena and hematochezia. Physical examination was notable for blood pressure of 68/44 mm Hg and a pulse of 114 beats/min. His abdomen was soft, non-distended and diffusely tender to palpation. Rectal exam revealed bright red blood. Laboratory evaluation revealed hemoglobin 6.3 g/dL, platelets 127 x109/L, blood urea nitrogen (BUN) 46 mg/dL, creatinine 2.1 mg/dL and INR 1.9. The patient received 3 units of packed red blood cells, 2

	units of fresh frozen plasma (FFP) and 2 L of crystalloid for resuscitation from hemorrhagic shock. He underwent emergent EGD which revealed mucosal friability throughout the stomach and duodenum but did not show any active bleeding. The following day the patient underwent a colonoscopy which revealed multiple very large rectal ulcers with adherent clot. Histologic staining with Congo red stain revealed positively staining deposits in the vasculature of the ulcer bed that exhibited apple-green birefringence under polarized light consistent with amyloidosis. Given diffuse involvement and no intervenable site, no endoscopic intervention was performed. Hematology recommended use of FFP or recombinant factor X to correct coagulation abnormalities if the patient had ongoing bleeding. The patient's hemoglobin remained stable and he did not have recurrent bleeding during the hospitalization. Conclusion: Amyloidosis can be associated with life-threatening gastrointestinal hemorrhage. In patients with AL amyloidosis, acquired hemostatic abnormalities, including coagulation deficiencies (most commonly a factor X deficiency), hyperfibrinolysis, and platelet dysfunction, are considered the primary pathogenic factors. Amyloid angiopathy with increased fragility of blood vessels and impaired vasoconstriction may also promote bleeding. Early endoscopic intervention is indicated in patients with amyloidosis. Patient should be continued or initiated on systemic treatment for amyloidosis which may prevent or mitigate future episodes of gastrointestinal bleeding.
Nadia Akhiyat Dr. David Brennan	*Pain in the Neck: Headache with an Elusive Etiology* Case Presentation: A previously healthy 41-year-old man presented to primary care clinic with eight days of progressive headache. The constant left hemicranial headache suddenly developed while golfing one afternoon and progressed in intensity over days. He slowly developed intermittent slurring of his speech, weakness of the left side of his tongue and slight drooping of his left eyelid. While his headache mildly improved during waking hours with ibuprofen, he reported waking up multiple times from sleep with a severe left hemicranial pain. He denied nausea, vomiting, or previous episodes of headache. A cranial nerve exam was unremarkable other than mild left tongue weakness [BDNM1] . Tongue deviation and fasciculation were absent. He had no dysarthria. Signs of Horner Syndrome were not present. The remainder of his exam was normal. Magnetic resonance (MR) imaging of the brain with and without gadolinium contrast was normal except for an incidental note of chronic lacunar infarcts in the right cerebellum. Methods: Conservative management and close specialty outpatient follow-up was arranged. During the 48 hours after initial ambulatory evaluation, the patient experienced worsened slurred speech and inability to coordinate his tongue while eating. With increased suspicion for arterial dissection, the patient was empirically prescribed daily aspirin 325mg and clopidogrel 75mg. Physical activity involving recoil or neck twisting was not advised. Closer examination of brain MR imaging revealed indeterminate signaling of distal left carotid artery. MR brain and neck angiogram with and without contrast revealed an acute and nearly occlusive dissection of the left distal cervical internal carotid artery (ICA) with some extension of the dissection intracranially. Conclusion: ICA dissections are a significant etiology for ischemic stroke in young patients and should be suspected in the right clinical scenario. Clinical

	diagnosis can be challenging and a broad differential of headache in patients who present to ambulatory clinic is essential. Classic signs and symptoms (pain, Horner syndrome, cranial nerve palsy, cerebral ischemia) or a high index of suspicion as in this case should prompt angiography for diagnosis. The management of carotid artery dissection is focused on preventing thromboembolic complication. Our patient was treated with daily aspirin 325mg and clopidogrel 75mg with a follow up MR angiogram in three months to determine duration of therapy.
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 (1). In 2019 alone, there were 23,453 per 100,000 confirmed cases in the US (2). Lyme carditis remains an uncommon complication in the early disseminated phase of the disease. In this report, we hope to shine a light on the importance of cardiac workup in this population. Case Presentation: A 41-year-old female with a past medical history of class 3 obesity, OSA, HTN, PCOS, saphenous vein thrombosis, and splenectomy post-MVA, presented to the clinic feeling fatigued for 1 month after returning from a Cabin trip to Connecticut. A week before presentation the patient had low-grade fevers, a disseminated rash on her abdomen, and low home blood pressure and heart rate readings. Initially, she denied palpitations, chest pain, shortness of breath, lightheadedness, or syncope. Her heart rate was in the lower 50s bpm but her vitals were otherwise normal. Erythema migrans was noted on the upper abdomen, but the exam was largely unrevealing. Her initial EKG showed evidence of intermittent high degree AV block. Initial workup was negative for COVID-19, electrolytes and hepatic derangements, and thyroid dysfunction. Her WBC count was 11.36k/cmm and her neutrophils 7.99k/cmm. 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. A temporary cardiac pacemaker was placed and the patient was started on 3g IV ceftriaxone after a presumptive diagnosis of Lyme carditis was made. On the 3rd day, Lyme IgG & IgM western blots were positive and the patient was continued on IV ceftriaxone. After 7 days of antibioticcs, the patient was reassessed and 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 travel history to endemic areas who present with bradycardia or AV block with or without cardiac symptoms,

	allows for timely management which includes temporary cardiac pacing and antibiotics to prevent negative outcomes. 1. (CDC,2021), https://www.cdc.gov/lyme/index.html 2. (CDC, 2019), https://wonder.cdc.gov/nndss/static/2019/annual/2019-table2j.html
Hadiyah Audil Dr. Jennifer Duke Dr. Patricio Escalante	*Pulmonary-Cutaneous Fistula Associated with Empyema Necessitans due to M. Avium Complex* Case Presentation: A 67-year-old woman with a history of right breast cancer status post chemoradiation, recent longstanding right-sided pneumonia (diagnosed at outside facility, unsuccessfully treated for months with oral antibiotics), and BMI 13 presented with acute development of a purulent-draining cutaneous fistula on her right chest wall. CT chest showed a right pulmonary-cutaneous fistula (PCF), pleural effusion, and pneumothorax. An occlusive dressing was placed over the fistula and she was started on vancomycin and piperacillin-tazobactam. Thoracic Surgery was consulted and recommended creation of an open thoracic window, as her malnutrition precluded muscle flap creation; the patient declined this in favor of drain placement. Cultures of the drainage grew Mycobacterium avium complex (MAC), and she was started on rifampin, ethambutol, azithromycin, and amikacin with outpatient follow up. Discussion: To our knowledge, PCFs have not been previously reported with MAC. Risk factors for PCF development include prior thoracic surgery/procedures; thoracic radiation; malignancy; malnutrition; and empyema necessitans associated with chronic lung infections (particularly with M. tuberculosis, M. abscessus complex, and Actinomyces), wherein extension of the infected pleural space to the chest wall causes skin/soft tissue erosion. Patients should initially be placed on airborne precautions given the association with tuberculosis. CT chest should be obtained to evaluate for co-existing intrathoracic pathologies. Thoracic Surgery should usually be consulted for consideration of surgical treatment. If drainage is observed, bacterial/fungal cultures should be obtained, and broad-spectrum antibiotics should empirically be started. Although morbidity and mortality can be high for untreated PCF, prognosis is usually good with appropriate management.
Connor Buechler Dr. Tom Freeman	*A Tale of Two Blisters* Introduction: Systemic Lupus Erythematosus (SLE) is a chronic autoimmune connective tissue disorder of unknown origin with wide-ranging clinical manifestations. In the absence of widely accepted diagnostic criteria, diagnosis is generally guided by classification criteria, recognition of classic features, and exclusion of alternative diagnoses. Porphyria cutanea tarda (PCT) is a metabolic disorder caused by altered activity of the heme biosynthetic enzyme uroporphyrinogen decarboxylase (UROD) in the liver, and is much rarer in clinical practice than SLE. Blistering skin lesions are the predominant clinical manifestation of PCT, and over 80% of cases are due to an acquired deficiency rather than inborn mutation. Case Presentation: A 64 year old male with a past medical history of coronary artery disease presented with a rash beginning on his trunk one month prior to presentation. A dermatology visit at that time noted a widespread annular erythematous and edematous eruption. Initial biopsy showed a robust

	interface dermatitis with apoptotic basal keratinocytes, moderately dense superficial perivascular lymphocytic infiltrate, and no alteration of dermal collagen. The patient was placed on topical triamcinolone cream which proved ineffective. The patient presented to the ED three weeks later with progressive desquamation and mucositis prior to ultimately being transferred to our hospital with concern for toxic epidermal necrolysis. On admission, he had a severe erythematous blistering and erosive rash with occasional tense vesicles and bullae covering >75% of the body surface. He was additionally noted to have oral ulcers, severe darkening of sun-exposed skin, synovitis, and nonscarring alopecia. His laboratory work-up was notable for pancytopenia, low levels of complement, elevated ESR and CRP, and positive ANA and anti-dsDNA. Repeat biopsies were non-specific. Paraneoplastic pemphigus was considered, but no neoplasm was found after extensive workup. Infectious workup, including HCV and HIV, was negative. The patient was started on a course of high-dose corticosteroids, leading to gradual improvements to blistering rash and complement levels, but no change to skin darkening and photoaccentuated desquamation. Urine and serum porphyrins from day of admission then returned dramatically elevated, with much higher uroporphyrin and heptaporphyrin than coproporphyrin levels in the urine, a pattern consistent with PCT. He was ultimately discharged on prednisone 30 mg daily and hydroxychloroquine 100 mg twice weekly for presumed overlap of severe cutaneous SLE and PCT. On followup, his diffuse rash and hyperpigmentation had dramatically improved. Further treatment is ongoing, with serial phlebotomy planned. Conclusion: This case illustrates that acute presentations do not always arise from a single disease process. Establishing one diagnosis does not preclude another, and a collaborative interdisciplinary team is vital to the evaluation and treatment of complex, multi-system presentations. Here, our recognition and treatment of both the acute SLE flare and acquired PCT led to resolution of both disease processes.
Claire Carlson	*Poor Concentration as Presenting Sign of Severe Cardiac Amyloidosis* Case Presentation: A 58-year-old female with past history of hypertension, hyperlipidemia, sleep apnea, anxiety, intermittent SVT and recent diagnosis of AL light chain renal amyloidosis in the setting of persistent nephrotic range proteinuria presented to the emergency department with 2-3 weeks of fatigue, extremity swelling, decreased appetite and poor concentration. She was visibly frustrated in ED stating “I can’t get any work done” due to inability to focus on tasks. Methods: Expectedly anasarcic on exam, however no hypoxia or clinical evidence of pulmonary edema. Labs showed normal renal function, elevated BNP >2000, troponin>100. EKG with normal sinus rhythm. Seen by cardiology with concern for systemic amyloidosis and associated inflammatory myocarditis. Transthoracic echocardiogram showed progressive severe concentric left ventricular hypertrophy with apical-anterior hypokinesis and 2.3 cm partially enhancing LV mass with mild reduction in LV ejection fraction to 50%. Aggressive IV diuresis and therapeutic heparin were started. Subsequent cardiac MRI showed diffuse infiltrative myopathy, acute LAD territory infarction and confirmed large LV thrombus. Emergent PCI and LAD thrombectomy were performed. MR of the brain showed multifocal late acute right MCA infarcts including large area of ischemic injury to the right insula consistent with embolic stroke, with LV thrombus as likely source. The patient had a prolonged hospitalization complicated by VT cardiac arrest

	requiring eventual ICD placement, recurrent pulmonary edema and progressive renal failure despite maximal therapies and initial response to chemotherapy. Ultimately discharged with family on hospice cares on hospital day 27. Conclusion: The relatively poor prognosis of primary AL amyloidosis compared to other fibril types is largely related to the frequency of cardiac involvement, including a higher incidence of LVH and intracardiac thrombosis [1]. Unfortunately atypical systemic symptoms can be presenting features of AL amyloidosis progression. Early goals of care discussions and involvement of palliative medicine should be considered given higher risk for sudden fatality. References: [1] Choukair MO, Halawi A, Nehmeh A, Kazma H. Role of Echocardiography in the Diagnosis of Light Chain Amyloidosis: A Case Report and Review of Literature. Cureus. 2020;12(11):e11377. Published 2020 Nov 8. doi:10.7759/cureus.11377 [2] Kastritis E, Dimopoulos MA. Recent advances in the management of AL Amyloidosis. Br J Haematol. 2016 Jan;172(2):170-86. doi: 10.1111/bjh.13805
Karan Chohan Dr. N. Nora Bennani	*A Case of Cartilage-Hair Hypoplasia and Non-Hodgkin Lymphoma* Introduction: Cartilage-hair hypoplasia (CHH) is a rare autosomal recessive chondrodysplasia often characterized by short-limbed short stature, hypoplastic hair, combined immunodeficiency, and an increased risk of malignancy, especially non-Hodgkin lymphoma. There are recommendations for regular lymphoma screening in CHH, however, there are currently no guidelines on how to safely treat a lymphoid malignancy in patients with CHH and concurrent severe immunodeficiency. The standard frontline treatment of diffuse large B-cell lymphoma consists of the cytotoxic multi-agent chemotherapy R-CHOP (rituximab, cyclophosphamide, doxorubicin, vincristine, and prednisone). Although well-tolerated in the general population, it can cause significant immunosuppressive effects with B-cell depletion, reduction in serum IgG levels, and decreased CD4+ count. Here we present a case of an aggressive B-cell lymphoproliferative disorder in the context of CHH and severe immunodeficiency. Case Presentation: A 51-year-old male with CHH was found to have a new asymptomatic well-circumcised 2.6 cm nodular opacity over the right lower lung on annual screening chest x-ray. His medical history included severe humoral (IgG subclass 2, 3, and 4 deficiencies) and cellular immune deficiency, maintained on intravenous immunoglobulin (IVIG). Initial lab workup revealed a normal CBC with mild lymphopenia, absolute lymphocyte count (ALC) of 0.67, normal serum chemistry, appropriate organ function laboratories, LDH 185, and 25-OH-Vit D level of 29. Furthermore, a CD4 count of 256, CD8 count at 62 with helper suppressive cell ratio of 4.1, a severe IgM deficiency at 12, IgA of 213, and IgG of 1090 with replacement. A workup with a chest CT, PET-CT scan, and wedge resection revealed a diagnosis of stage IVE immunodeficiency-associated EBV lymphoproliferative disorder (diffuse large B-cell lymphoma, non-germinal center phenotype). He received full dose R-CHOP chemotherapy for 6 cycles with growth factor support along with IVIG every 3 weeks with a goal to maintain IgG trough level >1000. He was carefully followed due to the higher risk for severe infection and complications with his prior immunosuppression. He tolerated therapy well, achieved remission, and remained disease-free beyond 24 months post-treatment.

	Discussion: In this case, we illustrated that standard treatment with full-dose cytotoxic chemotherapy, growth factor support, IVIG, and close monitoring resulted in good outcomes for a patient with CHH, aggressive B-cell lymphoma, and severe combined immunodeficiency. With a significant risk of severe infections related to the use of cytotoxic chemotherapy, balanced against treating aggressively a highly curable disease, there is limited guidance in the literature surrounding chemotherapy use in patients with pre-existing severe combined immunodeficiency in the context of CHH. Unlike in post-transplant lymphoproliferative disorders (PTLD) where immunosuppression is acquired and could be decreased when a lymphoid malignancy occurs, the immunosuppression in CCH is irreversible which places patients at continued higher risk of lymphoma recurrence. Hence, these patients need to be monitored closely for relapse.
Donna Coetzee Dr. Ayesha Behfooz Dr. Anne Bantle	*Central Adrenal Insufficiency in a Patient on Pembrolizumab* Introduction: Since the first immune checkpoint inhibitor (ICI) was approved a decade ago, this class of anticancer drugs has revolutionized cancer treatment and has become widely used against many types of malignancies. Due to their action on the immune system, ICIs are implicated in immune-related adverse events (irAEs), of which endocrinopathies are some of the most common. With the increasing frequency of ICI use, general internists will more often be exposed to patients presenting with the sometimes vague complaints of endocrine disorders. It will be important that irAEs of ICIs are on the differential for these patients. Case Presentation: A 65-year-old man with history of metastatic melanoma with known brain metastases and currently being treated with pembrolizumab was admitted to the hospital for expedited workup of fatigue, anorexia, and weight loss. Two months prior to this he had been doing well with his cancer treatment when he woke up one morning severely fatigued with nausea and vomiting. Over the next several weeks he continued to have very little appetite, frequently had dry heaves, and lost almost 30 pounds. His oncologist held a dose of pembrolizumab due to these symptoms, and when the symptoms continued hospital admission was recommended. On admission, the patient was found to have a low morning cortisol level and a cosyntropin stimulation test demonstrated essentially no cortisol response. An ACTH level was undetectable, consistent with central adrenal insufficiency. The most likely etiology was felt to be hypophysitis caused by his ICI. Other pituitary hormone axes were screened without evidence of abnormality. The patient was started on intravenous hydrocortisone and had rapid improvement in symptoms. By the next day he was able to transition to oral hydrocortisone. Discussion: Awareness of ICI induced endocrinopathies is essential for the general internist. The increasing frequency with which ICIs are being used makes it very likely that this is something that most will see in their practice. This patient presented with non-specific complaints that it would be relatively easy to attribute to his underlying malignancy. However, identifying the endocrinopathy early is important because they are very treatable with hormone replacement, generally result in rapid symptomatic improvement, and – importantly – do not preclude the patient from resuming ICI cancer therapy once stable. Rapid diagnosis and treatment of ICI endocrinopathies is critical to improve quality of life for patients, decrease morbidity and

	maximize longevity by potentially allowing continuation of effective cancer treatment regimens.
Sara Cuadra Aruguete Dr. Kevin Chang Dr. Terri McBride Billy Boua	*Platelets of Zero: An Interesting Case of ITP* Introduction: Immune thrombocytopenic purpura (ITP) is an autoimmune disorder characterized by isolated thrombocytopenia. It is a rare condition with a prevalence of approximately 12 per 100,000 adults. Patients may be asymptomatic or present with any form of bleeding ranging from mild mucosal bleeding to severe, life threatening hemorrhage. Here we present a case of ITP with an initial chief complaint of acute onset black lesions on the tongue. Case Presentation: A 69 year old male with a past medical history significant for Hodgkin's Lymphoma treated with chemotherapy and radiation in 2002, hypertension, obstructive sleep apnea and gout presenting with one day of acute onset black lesions on his tongue as well as an episode of epistaxis that resolved spontaneously. He also noticed red spots in his lower extremities for two weeks prior to presentation without any other constitutional symptoms. A month prior he had experienced URI symptoms after attending a gathering. He had no history of liver diseases or prior autoimmune disorders. He had no recent medication changes or travels. Initial vitals were within normal limits. Physical exam was significant for multiple, non-tender, black purpura scattered throughout the tongue and oral mucosal membranes. Petechiae were also noted from the dorsal aspect of his feet extending up to his knees bilaterally. Labs were notable for hemoglobin 13.6, platelet 0, INR 1.0, LDH 244, total bilirubin 2.0 and direct bilirubin 0.6. Additional labs including fibrinogen, haptoglobin, reticulocyte count, complement levels, vitamin B12 and folate were within normal limits. ANA was positive with a homogeneous pattern of 1:320. Infectious work up including HIV, hepatitis panel, as well as COVID-19 were negative. Imaging studies including chest x-ray was negative and abdominal ultrasound showed no splenomegaly. No evidence of recurrent lymphoma was seen on bone marrow biopsy. Patient was treated for presumed ITP immediately and required two doses of intravenous immunoglobulin, six doses of methylprednisolone, and one dose of rituximab over seven days. At the time of discharge, the oral purpura had significantly decreased in size and faded in color, and his platelets were 30K. He was later transitioned to a prednisone taper and continued weekly rituximab infusions in clinic. Conclusion: Thrombocytopenia has a broad differential. Platelets may be low secondary to increased destruction, decreased production, or sequestration. Because ITP is a diagnosis of exclusion, there must be a broad initial work-up to identify the underlying cause. This is necessary in severe cases of thrombocytopenia where there is a risk for mucosal bleeding or spontaneous intracranial hemorrhage. As with our patient, a new diagnosis of platelets of less than thirty thousand who are asymptomatic or have minor mucosal bleeding warrant management with corticosteroids at minimum.
Austin Cudak Dr. Diaa Osman	*Case Report of Secondary Autoimmune Bone Marrow Fibrosis Treated with Rituximab after Failing Glucocorticoid Treatment due to Se* Introduction: Autoimmune myelofibrosis is a rare cause of bone marrow

	fibrosis and has been found to be quite steroid responsive, however no second line treatment has yet been established. Case Presentation: We present a case of a 77-year-old woman with a history of seropositive rheumatoid arthritis and Sjogren's syndrome who presented with unexplained macrocytic anemia noted during routine monitoring labs. She was seen by her rheumatologist for her autoimmune arthritis which had been well controlled with methotrexate, adalimumab, and as needed ibuprofen for many years. Complete blood count was ordered for monitoring while on methotrexate and she was found to have precipitous drop in her hemoglobin over the past three months. Gastrointestinal loss in the setting of ibuprofen use was first considered, however the patient refused endoscopic workup and was eventually referred to hematology. At that time, she had elevated inflammatory markers with a significantly elevated sedimentation rate and c-reactive protein. In addition, she was noted to have an elevated ferritin and iron studies which showed a mixed picture. Because of this, a broad workup was ordered including a bone marrow biopsy. The bone marrow biopsy results showed a hypercellular marrow with moderate reticulin fibrosis consistent with secondary autoimmune myelofibrosis associated with rheumatoid arthritis. After obtaining these results she was started on high dose prednisone for treatment. Unfortunately, the patient subsequently developed a pseudomonas septic arthritis of her sacroiliac joint and required prolonged treatment with intravenous antibiotics, along with discontinuation of high dose prednisone and adalimumab due to the infection. She was thus started on treatment with rituximab after discussion between her hematologist and rheumatologist. This did eventually lead to a modest improvement of her hemoglobin level. Discussion: Autoimmune myelofibrosis is a clinically rare disease that must be differentiated from other causes of myelofibrosis. A clonal malignant disease is seen in patients who have gain of function mutations resulting in disease such as janus kinase 2 (JAK2) or calreticulin mutation (CALR). Autoimmune myelofibrosis can be either secondary, being related to another autoimmune disease such as systemic lupus erythematosus or Sjogren syndrome, or it can be primary, presenting in the absence of a clear diagnosed autoimmune disease. Differentiation these entities is important because it affects treatment options, where autoimmune myelofibrosis is typically very responsive to glucocorticoids. Second line treatments and steroid sparing agents have been used in case reports such as mycophenolate mofetil, methotrexate, and rituximab. However, no second line therapy has been clearly identified to treat autoimmune myelofibrosis.
Hanah Cushen	*When Nausea is Neurologic: A Case Report of Neuromyelitis Optica* Introduction: Neuromyelitis optica spectrum disorders (NMOSD) are progressive immune-mediated demyelinating diseases of the central nervous system. Though prototypical presentation is weakness or vision loss, the following case demonstrates an incidence where nausea and vomiting can be among the first symptoms to occur. Case Presentation: A 47-year-old female presented with a four-week history of intractable nausea, vomiting, and generalized weakness. Initial labs were remarkable for hyponatremia secondary to hypovolemia in the setting of her poor oral intake and vomiting. When given intravenous fluids to correct this imbalance, her sodium increased rapidly from 119 to 130 in under 24 hours, despite aggressive monitoring and attempts to slow the correction. Within the

	following 24 hours, she developed ophthalmoplegia, limb ataxia, bilateral nystagmus, urinary retention, and tremors. Initial differential was highly concerning for pontine osmotic demyelination syndrome and imaging was ordered to evaluate. MRI demonstrated a nonspecific T2 hyperintensity of the medial hypothalamus and periaqueductal gray, and spinal MRI demonstrated an abnormal diffuse signal edema. Neurology was consulted overnight due to the new neurologic findings and this abnormal imaging. Osmotic demyelination was not consistent with her imaging and was ruled out. A lumbar puncture was performed by neurology and the cerebral spinal fluid was remarkable for white blood cell count of 161, and elevated IgG. Given the absence of infectious symptoms, there grew an increasing suspicion for an underlying rheumatologic process. Approximately one week into admission she developed cranial nerve VII palsy, and progressive lower extremity weakness. Relevant serology studies were positive for ANA, anti-Ro, and anti-aquaporin-4. History, imaging, and serology findings solidified a diagnosis of neuromyelitis optica spectrum disorder (NMOSD) with possible comorbid Sjogren's Syndrome. Her initial hyponatremia was secondary to a combination of hypovolemia as well as SIADH caused by her NMOSD. Immediately following diagnosis, she was started on plasmapheresis and 1g daily of methylprednisolone for five days, followed by a prolonged prednisone taper over at least six months. She received a Rituximab infusion, with plans to undergo infusions every six months as an outpatient. The patient was ultimately discharged to an acute rehabilitation facility after a four-week admission. Since initiation of treatment she has had moderate improvement of upper extremity weakness, however continues to suffer from profound lower extremity weakness, neurogenic bladder, and neuropathic pain. Discussion: Though atypical, intractable nausea and vomiting can be the only presenting symptoms of NMOSD. This progressive disease is characterized by periods of relapse leading to cumulative disability, which can be fatal. It is critical to recognize this presentation, given that earlier initiation of immunotherapy has proven to decrease rate and frequency of relapse, and improve long-term outcomes. Therefore, it is important to remember that not all nausea is just nausea, sometimes it's neurologic.
Jissy Cyriac	*I'm Backed Up: Renal Failure and Myocardial Infarction Secondary to Bladder Incarceration* Background: Obstructive uropathy can lead to urinary retention and acute kidney injury. We present a case of obstructive uropathy secondary to inguinal hernia with bladder incarceration leading to renal failure and myocardial infarction. Case Presentation: An 80-year-old male with hypertension and chronic lower extremity edema presented with a three-week history of nausea, vomiting, and poor oral intake along with a 6-month history of worsening painful testicular swelling and urinary retention. Initial exam was notable for 15x20 cm left groin swelling and computed tomography of the abdomen/pelvis revealed direct left inguinal hernia with bladder incarceration, ureteral obstruction and

	bilateral hydronephrosis. Labs were notable for elevations in creatinine (14.83 mg/dL), blood urea nitrogen (180 mg/dL), and potassium (6.3 mmol/L). His initial troponin was elevated at 141 ng/dL with decrease to 128 ng/dL at 2 hours and this was thought to be secondary to renal failure. Electrocardiograms (EKGs) showed mild lateral wall ST segment depression and T-wave inversion and patient denied anginal symptoms. He underwent bilateral nephrostomy tube and foley catheter placement with relief of urinary obstruction. His troponins continued to elevate up to 1700 ng/dL without significant changes from initial EKG findings. Echocardiogram showed ejection fraction of 27% with severe mitral and tricuspid regurgitation and regional wall motion abnormalities concerning for non-ST elevation myocardial infarction (NSTEMI). Emergent catheterization was deferred in the setting of acute renal failure and he was medically optimized. Coronary angiogram was completed on hospital day 7 and revealed 99% stenosis in the left circumflex artery which was treated with a drug eluting stent. His creatinine continued to downtrend during his hospital course and he continued to maintain good urine output. Given his recent percutaneous coronary intervention and need for dual antiplatelet therapy, hernia surgery was delayed for 6 months from stent placement. He was given a hernia Truss to avoid repeat herniation in the interval period. His serum creatinine stabilized at 3.9 mg/dL with estimated glomerular filtration rate (GFR) of <15 mL/min with stable urinary output through nephrostomy tubes for management of chronic bilateral hydronephrosis while awaiting hernia repair and arteriovenous fistula placement for dialysis. Conclusions: Approximately 1-5% of inguinal hernias can cause bladder incarceration and obstructive uropathy leading to renal failure, hydronephrosis, and severe metabolic derangements. Diagnosis of myocardial infarction is challenging in the setting of renal failure due to baseline troponin elevation secondary to decreased renal clearance of this cardiac biomarker. Higher cutoff values for high sensitivity troponin measurements may be appropriate in the setting of renal failure to diagnose myocardial infarction but the exact thresholds remain unclear. Serial troponins and EKG should be utilized to assess for dynamic changes which would warrant further evaluation with echocardiogram and cardiac catheterization.
Atharva Dhole Dr. Marinos Kosmopoulos Dr. Kellen Albrecht	*An Unusual Transformation of Left Hemiblocks in Consecutive ECGs* Introduction: Cardiac conduction abnormalities are frequent contributors to cardiogenic syncopal events. Workup of such events, including advanced imaging and electrophysiologic study, is triggered by aberrant ECG findings and symptomatology. Case Presentation: The case described here presents a rare combination of a Right Bundle Branch Block (RBBB) and the transformation of a Left Posterior Hemiblock to a Left Anterior Hemiblock. Such uncommon ECG findings could pose diagnostic and management equipoise for clinicians. Conclusion: Subsequently, we advocate for precise adherence to the 2018 American College of Cardiology Guidelines for Bradycardia, which, for this patient, resulted in the appropriate course-of-care trajectory and implantation of a permanent pacemaker.
Christopher Dinh Dr. Neel Shah	*A "Toxoc" Cause of Hypereosinophilic Syndrome* Introduction: Peripheral eosinophilia presents a challenging diagnostic

	dilemma for internists due to its infrequent clinical presentation and wide differential diagnosis that includes allergic, infectious, rheumatologic, neoplastic, and less commonly idiopathic disorders. Eosinophilia is defined as an absolute eosinophil count (AEC) of ≥500 eosinophils/μL in peripheral blood. Related disorders include hypereosinophilia (≥1,500 eosinophils/μL) and hypereosinophilic syndrome (hypereosinophilia plus end-organ dysfunction). We present a case of hypereosinophilic syndrome with pulmonary and renal dysfunction. Case Presentation: A 76-year-old Somali-immigrant was admitted to an outside hospital with several weeks of progressive dyspnea and wheezing. His past medical history was significant for ischemic cardiomyopathy, type 2 diabetes mellitus, and chronic kidney disease. His social history was significant for having lived in Somalia, Yemen, and Indonesia prior to immigrating to Minnesota five years earlier. At the outside hospital, he was treated for community-acquired pneumonia without improvement. Due to his severe eosinophilia of >10,000 eosinophils/μL, he was administered glucocorticoids and transferred to our hospital for further evaluation. During his hospitalization, a broad evaluation was obtained to identify the etiology of his hypereosinophilia. His evaluation was significant for a peak absolute eosinophil count of 10.6K eosinophils/μL, sputum eosinophil 21% (normal <3%), elevated IgE to 817 kU/L (normal <214), positive toxocara antibodies, and peak creatinine 2.66 mg/dL (baseline creatinine 1.3-1.4). The remainder of his allergic, infectious, rheumatologic, and hematologic evaluation was unrevealing. He was treated with albendazole for toxocariasis and inhaled corticosteroids for his wheezing. His eosinophilia downtrended and clinical symptoms resolved. Bone marrow biopsy was deferred due to his clinical improvement. Discussion: We present a case of hypereosinophilic syndrome secondary to toxocariasis. The clinical approach to peripheral eosinophilia should begin with a broad multisystem evaluation to identify the underlying etiology. Clinicians should also evaluate for evidence of end-organ involvement bearing in mind hypereosinophilic syndrome most commonly involves the pulmonary, cardiac, renal, gastrointestinal, central nervous, and/or cutaneous systems. Toxocariasis is an uncommon parasitic infection caused by the accidental ingestion of eggs shed by the roundworm parasites Toxocara canis and Toxocara cati. The two most common clinical syndromes are visceral larva migrans (whereby larvae migrate through the systemic circulation and directly cause end-organ dysfunction) and ocular larva migrans (localized larval ocular infection), though mild infections may cause eosinophilia as the primary symptom. The diagnosis of toxocariasis is made with a positive Toxocara serology along with a compatible clinical and exposure history. Stool O&P may not be sensitive as humans are the accidental host and the organism may not complete a full life cycle within the gastrointestinal tract. Toxocariasis is treated with albendazole and while symptoms often resolve rapidly, eosinophilia may take months to resolve due to ongoing antigenic stimulation from dead larvae.
Michael Downey Dr. Andrew Olson Dr. Kellen Albrecht	*Primum Non Nocere: Selection of Beta Blocker Therapy in Methamphetamine Associated Cardiomyopathy* Case Presentation: A 35-year-old male presented with recurrent acute

	respiratory distress with volume overload. Investigation during hospitalization one month prior resulted in a diagnosis of heart failure with reduced ejection fraction (HFrEF) due to methamphetamine associated cardiomyopathy (MACM). Medications at the time of discharge included initiation of baseline diuresis and guideline directed medical therapy (GDMT) for heart failure with beta blockade (metoprolol succinate) and ACEi; a mineralocorticoid antagonist was deferred per patient preference. Since his initial discharge, he reported complete adherence to medications with no anginal symptoms. He continued to use methamphetamine. Upon readmission, his beta blockade was held in the setting of cardiogenic shock, and he responded robustly to diuresis – ultimately not requiring ionotropic nor mechanic circulatory support. During his second hospitalization, he declined to meet with members of the chemical dependency team. He was discharged with re-initiation of his previous GDMT, with plans to up titrate to prior to admission doses in the outpatient setting as hemodynamically tolerated. Since his second discharge, he has required 4 additional hospitalizations for acute heart failure exacerbations (total of 6 inpatient admissions in as many months). Discussion: This case raises the question of potential unintentional harm in the setting of prescribing beta-blocker therapy within the MACM subpopulation who plan to continue methamphetamine, especially from a harm reduction standpoint. Owing to the adrenergic effects of methamphetamine, there exists a theoretical concern for interplay between a prescribed beta blockade and methamphetamine resulting in "unopposed \alpha-agonism" with subsequent hypertensive crisis. Drawing parallels from the literature on cocaine-induced cardiomyopathy and myocardial infarctions, there may (or may not) be a harm reduction afforded via mixed \alpha/\beta blockade over a chronic \beta selective beta-blocker therapy in patients who continue to use methamphetamine with their prescription pharmaceuticals. Thus, while patients with HFrEF due to MACM will likely benefit in vacuo with any evidence-based beta blocker (bisoprolol, carvedilol, or metoprolol) might those patients who both: continue to plan to use methamphetamine and concurrently continue to utilize and follow outpatient treatment plans for MACM be safer on chronic carvedilol therapy (over metoprolol or bisoprolol)? And, perhaps most importantly, for those patients who continue to willingly engage in healthcare, could open discussions around this unique patient/pharmacodynamic interplay serve as a platform to further partner with the patient with hopes of decreasing (or extinguishing) future methamphetamine use?
Mitchell Dumais Dr. Benjamin Kopecky Dr. Thomas "Nick" Smith Dr. Scarlett Cao Micaela Witte	*Pain in the Neck: Erosive Esophagitis due to Migrating Spinal Fusion Hardware* Introduction: Cases of hardware-associated complications often hide in plain sight during initial patient presentation. Subtle clues regarding associated symptoms, imaging, or physical exam can be easily missed if there is not some degree of initial clinical suspicion. This report highlights a case of anterior cervical fusion hardware that quietly contributed to a presentation of MSSA bacteremia and acute blood loss anemia. Case Presentation: A 65-year-old male with a history of juvenile onset rheumatoid arthritis on prednisone and hydroxychloroquine and C4-7 discectomy with spinal fusion presented to outside emergency department with neck pain and lower extremity weakness. He had been transferred from an outside hospital with concerns for cervical osteomyelitis after presenting with five months of lower extremity weakness and pain. During his previous

	hospitalization, while CT and MRI studies of the cervical spine did not clearly demonstrate infection, he had been found to have MSSA bacteremia and leukocytosis of 38 x 109/L prompting osteomyelitis concerns. The patient had also developed left lower extremity swelling, with negative DVT ultrasound, thought to be a new cellulitis. While admitted, the patient's hemoglobin precipitously dropped from 15.5 to 7.9 secondary to a gastrointestinal bleed which was treated supportively. He was transferred due to concern for higher level of care. After transfer, the patient continued to have significant neck tenderness and lower extremity pain. He was treated with IV cefazolin for the MSSA bacteremia. Reexamination of the prior CT and MRI imaging of the cervical spine demonstrated evidence of unattached anterior cervical hardware from the spine. The patient continued to have significant hemoglobin drop, prompting investigation with EGD and colonoscopy. EGD provided direct visualization of his cervical hardware eroding into the upper third of the esophagus, with follow-up esophagram negative for extravasation around this hardware erosion. TTE showed no evidence of endocarditis or infected thrombi. He was planned for complex esophageal repair and anterior cervical plate removal. Discussion: For patients with indwelling hardware, malfunction always remains on the differential. For this patient, his chronic inflammatory state and prednisone use may have contributed to migration of spinal hardware. While his GI bleed resulted in escalated care, more subtle symptoms of this erosion were present such as progressive dysphagia and more recent neck pain. It is important to evaluate indwelling devices and hardware, especially in patients with localized symptoms.
Aoife Feighery	*The Cecal Bascule: A rare form of large bowel obstruction* Introduction: A cecal bascule is a rare form of bowel obstruction where the cecum folds upon itself in an anterior-caudal direction along the ascending colon. Cecal bascules account for only 0.05-0.4% of large bowel obstructions. In contrast to other forms of mechanical obstruction, the cecum in a bascule folds along a transverse line, without torsion. This movement is likened to a see-saw, the French word for which is "bascule". Symptoms resemble those experienced in other forms of bowel obstruction: abdominal pain, distension, constipation, and occasionally respiratory distress due to abdominal hypertension. Diagnosis usually involves an abdominal Xray showing a dilated cecum in the right upper quadrant without the axial twisting or the "coffee bean" sign seen in cecal volvulus. Computed tomography (CT) may be utilized for further characterization and identification of perforation or ischemia. Initial management incudes colonic decompression, pain control, and respiratory support. Ultimately, the treatment of a cecal bascule is surgical, typically right hemicolectomy. Case Presentation: An 80-year-old male with a history of COPD, congestive heart failure, paroxysmal atrial fibrillation, and chronic kidney disease presented to the emergency department with worsening dyspnea and was admitted to a medicine service for management of presumed COPD exacerbation. The patient soon became progressively hypoxic with worsening abdominal pain. X-ray imaging showed a large air collection in the right upper quadrant with marked dilation of the hepatic flexure of the colon. The "coffee bean" sign was absent". He was subsequentially transferred to the intensive care unit (ICU) for an increasing oxygen requirement and concern

	for impending bowel perforation. Upon arrival to the ICU, the patient required bilevel positive pressure ventilation. His abdomen was firm, distended, and markedly tender in the right upper quadrant. Bowel sounds were diminished. CT showed a dilated cecum flipped into the right upper quadrant, consistent with a cecal bascule. There was no free air, no upstream bowel dilation, and no cecal wall thickening or mesenteric edema to suggest acute volvulus. A nasogastric tube was placed for decompression. An aggressive bowel regimen was initiated without effect. Hypaque enema and endoscopic colonic detorsion were unsuccessful. After exhausting non-operative options, the patient was taken for explorative laparotomy, which confirmed a cecal bascule with impeding rupture. Right hemicolectomy with end ileostomy was performed. Conclusion: Bowel obstruction is common within both inpatient and outpatient settings. Typically, non-mechanical or functional obstruction is managed medically, while mechanical obstruction, such as colonic mass, volvulus, and intussusception requires surgical intervention. Cecal bascule is an uncommon form of mechanical obstruction not responsive to aggressive medical or endoscopic therapy. It is paramount for physicians to identify such cases early in the clinical course and to involve a surgical team for definitive intervention to prevent intestinal ischemia, necrosis, or perforation.
Nouran Felo Dr. Siri Urquhart Ddr. Sumedh Hoskote	*When the Patient Reads the Textbook – A Classic Case of Thrombotic Thrombocytopenic Purpura* Introduction: Thrombotic thrombocytopenic purpura (TTP) is a thrombotic microangiopathy characterized by thrombocytopenia, microangiopathic hemolytic anemia (MAHA), and organ ischemia; a medical emergency that should be recognized quickly to initiate prompt medical treatment. Although ADAMTS13 activity, which can confirm the diagnosis of TTP takes time to result, TTP can be recognized clinically. One should have a high index of suspicion for TTP when a patient presents with severe thrombocytopenia and MAHA, and should consider initiation of plasma exchange (PLEX) therapy. Case Presentation: A 21-year-old gentleman presented to the ED for evaluation after a fall with seizure-like activity and development of AMS in the setting of a 5-day-history of dark stools, hematuria, nausea, vomiting, headaches, dizziness, and jaundice. On initial presentation, he was tachycardic but otherwise hemodynamically stable. Imaging studies including head CT, RUQ ultrasound, and CT abdomen/pelvis were without acute abnormalities. Laboratory investigations were significant for thrombocytopenia-platelet count 6x10(9)/L, anemia-hemoglobin 7.8g/dL, indirect hyperbilirubinemia-8.8mg/dL, increased reticulocytes-7.38%, elevated fibrinogen-398mg/dL, elevated LDH-1712U/L, elevated CRP-20.5mg/L, and elevated creatinine-1.94mg/dL. Peripheral blood smear showed presence of bite cells. He was jaundiced with minimal petechiae/purpura on bilateral lower extremities. He was febrile to 38.4º C and PLASMIC score was 7, indicating high risk for severe ADAMTS13 deficiency. Given high probability for TTP, he was admitted to the ICU for emergent PLEX. Next day labs included a decreased haptoglobin at <14 and final interpretation of the peripheral blood smear showed moderate schistocytes & helmet cells. His ADAMTS13 activity was severely reduced at <5% and positive for the

	inhibitor, confirming his diagnosis. He was continued on daily PLEX until platelet count recovered and was started on high-dose steroids, caplicizumab, and rituximab. He tolerated treatment well and has been closely monitored by hematology. Discussion: This case highlights the importance of maintaining a high index of suspicion for TTP and initiating treatment with PLEX immediately when a patient presents with the classic TTP pentad: thrombocytopenia, MAHA, fever, renal failure, and severe neurologic findings. Prior to the introduction of PLEX, there was a 90% mortality associated with TTP and with current treatment, there is still a high mortality rate at 10-15%. Even in the absence of all components of the pentad, key lab findings increase the likelihood of diagnosis allowing for early treatment. The PLASMIC score risk stratifies and acts as an aid in decision-making prior to treatment. Thrombocytopenia, a peripheral blood smear with schistocytes, indirect hyperbilirubinemia, and increased LDH with absent haptoglobin in the setting of normal coagulation studies can be sufficient for diagnosis. Further evaluation should include: CBC, BMP, reticulocyte count, and ADAMTS13 testing. If there is a high enough suspicion for TTP, hematology should be consulted for assistance in consideration of initiation of PLEX therapy to minimize morbidity and mortality.
Caitlyn Gallagher Dr. Nikhil Kolluri Dr. Samuel Asirvatham Dr. Meir Tabi	*Giant Cell, Bigger Problems* Introduction: Giant cell myocarditis is a rare but life-threatening condition. Timely diagnosis is beneficial as the disease progresses rapidly, and its treatment differs from other types of myocarditis. Diagnosis is often challenging due to clinical overlap with other cardiovascular conditions, but with prompt identification and treatment, prognosis improves significantly. Case Presentation: We present a case of a 74-year-old woman with a history of polymyalgia rheumatica and temporal arteritis controlled on low-dose prednisone who presented to the hospital with two days of progressive dyspnea, weakness, and midsternal chest heaviness. On examination, she had increased respiratory effort, tachypnea, diffuse crackles in her lungs bilaterally, and minimal pitting edema in her bilateral lower extremities but no murmurs, rubs, or gallops on cardiovascular exam. ECG and telemetry showed new intermittent right bundle branch block, nonsustained polymorphic ventricular tachycardia, and nonsustained monomorphic ventricular tachycardia. Troponins were elevated and changing, and BNP was >18,000. Chest X-ray did not show cardiomegaly but did show increased interstitial markings suggestive of volume overload. She was admitted with concerns for NSTEMI, started on a heparin drip, and given 324 mg of Aspirin. She was also started on IV lidocaine for her arrhythmias and was sent for cardiac catheterization, but this did not show significant coronary artery disease. Transthoracic echocardiogram showed ejection fraction of 20%, and there was concern for myocarditis or cardiomyopathy given the degree and location of regional wall motion abnormalities. Given this, she underwent cardiac MRI, which showed scattered areas of subendocardial delayed enhancement in the basal and left ventricle in a non-vascular distribution compatible with giant cell myocarditis, which was confirmed on endomyocardial biopsy. Diuresis and guideline-directed medical therapy were used to treat her acute heart failure. She continued to have arrhythmias, so IV Amiodarone was started in addition to IV Lidocaine and ultimately transitioned to oral

	Mexiletine and Amiodarone. An ICD was not placed as per patient preference. For treatment of the giant cell myocarditis, she was started on pulse-dose IV steroids followed by high-dose oral steroids with a prolonged taper. Cyclosporine and Azathioprine were also initiated for immunosuppression. She was discharged in improved condition with appropriate cardiology follow-up. Conclusion: Though giant cell myocarditis is rare, its diagnosis is important as the treatment differs from other forms of myocarditis. For giant cell myocarditis, prompt treatment with a combination of immunosuppressive medications provides improved prognosis. Even with treatment, the risk of ventricular arrhythmias remains elevated. Ultimately, cardiac transplantation is often warranted, though recurrence is common.
Vanessa Gow-Lee Dr. Elie Berbari	*EBV Meningitis as Manifestation of Infectious Mononucleosis* Introduction: Although Epstein-Barr virus in the central nervous system has been well-characterized in immunocompromised patients, CNS involvement in immunocompetent hosts with primary infection is not. Furthermore, the significance of detecting EBV by PCR in the cerebrospinal fluid is not always clear, especially in immunocompetent patients. EBV has been described to cause a variety of CNS syndromes, including meningitis, encephalitis, cerebellar ataxia, acute disseminated encephalomyelitis, and myelitis. We describe here a case of mononucleosis in an immunocompetent adult who presented with aseptic meningitis which was attributed to EBV. Case Presentation: Our patient was a 27 year-old woman with minimal comorbidities (ADHD, chronic sinusitis) who presented with several weeks of fatigue, myalgias, left-sided abdominal pain, and a severe occipital and retro-orbital headache with associated neck stiffness, photo- and phonophobia. She denied any rash, pharyngitis, any new romantic/sexual partners, or high-risk sexual activity. She had nuchal rigidity on exam, but no lymphadenopathy or other focal neurological deficits. Laboratory tests showed a lymphocytic leukocytosis (WBC 14.6 x 10^9 cells/L with >80% lymphocytes, with smear showing atypical lymphocytes) with a new transaminitis and hyperbilirubinemia (AST 324 U/L, ALT 387 U/L, tBili 3.8 mg/dL, dBili 2.8 mg/dL). EBV serologies revealed positive IgM and negative IgG, indicating acute infection. Abdominal imaging showed splenomegaly of 17.2 cm. MRI of the brain was normal without parenchymal changes or hydrocephalus. LP revealed an elevated opening pressure (>40 cm H2O) and lymphocytic pleocytosis (75 cells with 81% lymphocytes), slightly elevated protein (66 mg/dL), and normal glucose (52 mg/dL). Gram stain and culture were negative and a broad-range PCR panel was positive only for EBV. She was initially treated conservatively with pain control as well as Acetazolamide due to the increase in intracranial pressure. By the time EBV PCR was found to be positive, she was already having symptomatic improvement and her transaminitis were downtrending spontaneously. She was not treated with antiviral treatment nor with steroids. She was discharged in good condition three days later and subsequently had complete normalization of her LFTs. Conclusion: It is not fully clear how often infectious mononucleosis involves the CNS, with case studies reporting a wide range from 1–18%. Isolation of EBV in the cerebrospinal fluid is of special concern in immunocompromised patients given its association with lymphoproliferative disease or can suggest reactivation of latent EBV in the CNS space. However, as in our patient, EBV

	meningitis can be considered in patients with primary infection. There is no good evidence for the use of Acyclovir in patients with CNS involvement. Therefore, while EBV meningitis is very possible, the tenets of therapy are to rule out other pathologies and then to treat EBV meningitis with supportive care.
Alexandra Hall Dr. Forum Kamdar	*Heart Failure Management in Becker Muscular Cardiomyopathy: A Need for Interdisciplinary Neuromuscular Heart Failure Care* Background: Becker muscular dystrophy (BMD) is an X-linked recessive disorder resulting from mutations in the structural cytoskeletal Dystrophin gene. The resulting neuromuscular disorder typically presents with musculoskeletal weakness, but cardiomyopathy remains the number one cause of death in these patients. Here we present a patient with longstanding BMD associated cardiomyopathy with cardiogenic shock who successfully underwent orthotopic heart transplant. This case illustrates the need for proactive treatment as well as an early interdisciplinary approach to best care for patients with BMD associated cardiomyopathy. Case Presentation: A 50 year-old wheelchair-bound man with a history of BMD and associated cardiomyopathy (LVEF 10-15%) was referred to the hospital for increasing shortness of breath, decreasing exercise tolerance, leg swelling, and intermittent nausea. Vital signs: temperature 97.6, heart rate 115, blood pressure 85/63, respiratory rate 20, oxygen saturation 92% on ambient air. Exam was notable for elevated JVD, 1+ bilateral lower extremity edema, and cool extremities. Labs were notable for sodium of 121, ALT 169, AST 102, lactic acid 3.3, and NT-BNP of 3826. He was admitted for cardiogenic shock and started on inotrope therapy with dobutamine and milrinone; due to continued poor perfusion, an intra-aortic balloon pump was placed. Urgent transplant evaluation was undertaken with interdisciplinary input from neuromuscular neurology, cardiac surgery, pulmonary medicine, and PM&R. The patient successfully underwent orthotopic heart transplantation (OHT) and at 2 years of follow-up he is doing well without any rejection, infection, or interim hospitalizations. Discussion: BMD affects one in 18450 males. These patients are at high risk for heart failure with dilated cardiomyopathy occurring in an estimated 70%. Onset of cardiomyopathy tends to occur in adulthood with symptomatic heart failure presenting in the third decade of life or later. To date, echocardiography has been the primary modality use for screening, but cardiac MR might better fully characterize the severity of myocardial damage. Recognition of heart failure symptoms in patients with BMD can be challenging due to physical inactivity and obscuring respiratory complaints, and there is growing evidence that evaluation and treatment before overt cardiac symptoms appear afford patients the best opportunity for impacting mortality. As such, internists should ensure BMD patients are followed by a cardiologist to ensure heart failure therapies are initiated early. Advanced therapies, including heart transplantation should be considered in selected patients with interdisciplinary input. Prior studies have suggested that clinical outcomes after cardiac transplantation in selected patients with muscular dystrophy are similar to those seen in those with non-ischemic cardiomyopathy. Our case demonstrates a successful OHT in a patient with BMD with clinical benefit demonstrated at two years follow-up.
Kelly Hallowell	*Hematuria as an Unusual Presentation of Bladder Paraganglioma*

	Introduction: Paragangliomas are exceedingly rare neuroendocrine tumors derived from extra-adrenal autonomic paraganglia. Indistinguishable from pheochromocytomas at the cellular level due to their shared embryonic origins from the embryonic neural crest, they can present with the same symptoms of catecholamine excess including headaches, sweating, and palpitations. Paragangliomas arising from the bladder have interestingly been reported to present with hematuria and symptoms of catecholamine surge during micturition or sexual intercourse. This is a case of a young man presenting with hematuria found to have a bladder paraganglioma. Case Presentation: A 27-year-old healthy man presented to the emergency department for evaluation of hematuria and urinary retention. He described an episode of frank hematuria followed by a complete inability to pass urine for the last day. He had a similar episode four years prior and was found to have a bladder mass which was resected and reportedly benign. A foley was placed in the emergency department and drained frank blood. A CT urogram was obtained and revealed a large, enhancing soft tissue mass protruding from the anterior bladder with infiltration concerning for urothelial malignancy. Urology was consulted and recommended a transurethral resection of the bladder tumor. Upon attempted resection, the mass started bleeding profusely, and the patient developed significant hypertension resulting in the procedure being aborted. The urologist obtained outside records and discovered his prior resection was also aborted due to bleeding and hypertensive crisis which raised concerns for possible paraganglioma. Endocrinology was consulted. Although a pathologic diagnosis was not yet made, the patient was treated presumptively for catecholamine-secreting pheochromocytoma with phenoxybenzamine followed by propranolol. Robotic partial cystectomy was then performed to resect the lesion. Additional imaging revealed an enhancing subcarinal nodule concerning for metastatic disease, however the patient was lost to follow up before a definitive diagnosis could be made. Conclusion: This is a case of a young patient who was found to have a paraganglioma of the bladder presenting as hematuria. Paragangliomas are a rare tumor with a wide spectrum of presentations ranging from asymptomatic to life-threatening. Sympathetic paragangliomas are highly active and more commonly found in the abdomen and pelvis while parasympathetic paragangliomas are usually asymptomatic and located in the head and neck and symptoms are due to mass effect. When suspected, treatment prior to surgery includes careful initiation of alpha-receptor antagonists to reduce tumor-secreted catecholamines from causing blood pressure fluctuation. This should be followed by beta-receptor antagonists to counteract the tachycardia of nonselective alpha-blockade.
Ikram Haq	*Cardiac Sarcoid Heart Block - What to do?* Case Presentation: A 47-year-old previously healthy male presented to the ED with sudden onset palpitations. On examination, he was normotensive but tachycardic with a heart rate of 177. Labs were unremarkable and his ECG revealed a wide complex tachycardia concerning for ventricular tachycardia. He was cardioverted with his repeat ECG revealing new complete heart block. Reversible causes including myocardial ischemia, infections, electrolyte derangements and hypervagotonia were ruled out. A transthoracic echocardiogram revealed an ejection fraction of 59%, no regional wall motion abnormalities and no valvular heart disease. A 18-FDG PET/CT revealed a large perfusion deficit with associated FDG uptake in the apical, septal and inferior segments. There were numerous hypermetabolic nodules noted in the

	lungs and axillary lymph nodes. Biopsy revealed non-caseating granulomatous disease concerning for sarcoidosis. Discussion: Sarcoidosis is a multisystem disorder defined histologically by the presence of noncaseating granulomas. Cardiac involvement is a common sequela of disease with recent autopsy studies suggesting 25% of patients with systemic sarcoidosis have cardiac involvement. Cardiac manifestations include atrioventricular blocks, ventricular arrhythmias and heart failure. Three major guidelines exist to aid diagnosis: World Association of Sarcoidosis And Other Granulomatous diseases (WASOG), 2014 Heart Rhythm Society (HRS) and Japanese Ministry of Health and Welfare (JMHW). The WASOG and HRS Guidelines required histological evidence of disease. The JMHW Guidelines do not require histological confirmation but rely on PET/CT and Cardiac MRI as diagnostic tools. Late gadolinium enhancement in the mid ventricular wall and subepicardial regions with multifocal uptake on a cardiac MRI is suggestive of sarcoidosis. Focal 18-FDG accumulation in the septum and ventricles is indicative of an inflammatory process such as sarcoidosis. Treatment involves a combination of immunosuppression, treating the arrhythmia and preventing sudden cardiac death. The aim of immunosuppression is to control inflammation and prevent fibrosis. Steroids remained the most used immunosuppressive agent and are often combined with methotrexate, azathioprine, leflunomide or mycophenolate. Our patient was treated with leflunomide 20 milligrams once a day, a Prednisone taper over 6 months and ICD implantation. The 2018 ACC/AHA/HRS guidelines recommend ICD implantation is a class 2A recommendation in patients with cardiac sarcoidosis who have an indication for a permanent pacemaker implantation. This is because during active inflammation, ventricular tachyarrhythmias may occur and following resolution of inflammation, the subsequent scar may be a nidus for life-threatening reentrant ventricular arrhythmias. A PET/CT is useful in monitoring cardiac inflammation, treatment response and guiding further treatment. Our patient was followed up every six months in clinic with a PET/CT scan. His 6-month scan showed qualitatively and quantitatively reduced inflammation and his 12 month scan showed complete resolution of the inflammation. He was continued on the leflunomide and his prednisone was discontinued.
Naima Hashi Dr. Amrit Kamboj Dr. Vijay Shah	*When the Liver Goes Viral* Introduction: Epstein-Barr virus (EBV) is a herpes virus that affects more than 90% of the world's population and is most commonly transmitted via saliva. Classically, acute EBV infection presents with fever, lymphadenopathy, and pharyngitis. It can also result in mild transaminitis but rarely causes jaundice. Most cases are self-limited and resolve without any intervention. Case Description: An 18-year-old woman, employed as a childcare worker, with a past medical history of mild intermittent asthma was admitted to the hospital with a 2-week history of fevers, malaise, myalgias, and right upper quadrant abdominal pain. She also reported a 1-week history of dark urine and jaundice. At presentation, she was febrile to 38.1°. Laboratory studies were notable for total bilirubin 8.9 (mg/dl), direct bilirubin 7.8 (mg/dl), ALT 348 (U/L), AST 246 (U/L), and alkaline phosphatase 426 (U/L). Additionally, there was mild lymphocytosis present on the differential and peripheral smear

	showed atypical lymphocytes. A CT abdomen and pelvis was performed which revealed gallbladder wall thickening suspicious for cholecystitis without biliary dilatation, as well as mild splenomegaly. A right upper quadrant ultrasound showed similar findings of gallbladder wall thickening with a small amount of pericholecystic fluid and a positive Murphy's sign concerning for acalculous cholecystitis. Given the liver enzyme abnormalities and concern for choledocholithiasis, an MRCP was obtained which showed diffuse hyperintensity of the liver with periportal edema and prominent gallbladder wall thickening consistent with hepatitis. An extensive serologic evaluation was obtained to further work-up the liver enzyme abnormalities, including work-up for infectious and autoimmune etiologies. Hepatitis B and C serologies, iron studies, antinuclear antibody, smooth muscle antibody, antimitochondrial antibody, and ceruloplasmin were unremarkable. Monospot test was negative. EBV nuclear antigen was elevated at 1560 (IU/mL) and EBV antibody panel showed positive IgM and negative IgG/EBNA which was consistent with an acute EBV infection. She was discharged in stable condition with supportive care. Discussion: This case demonstrates the importance of keeping a broad differential when patients present with right upper quadrant abdominal pain, jaundice, and liver test abnormalities. In this case, there was initial suspicion for choledocholithiasis given the degree of liver enzyme elevations and gallbladder abnormalities, but no biliary stone was seen on MRCP making this less likely. Although EBV-driven cholestatic hepatitis is rare, it should be considered in young patients. Hepatic involvement occurs in 10% of young adults. The diagnosis is made by ordering the EBV nuclear antigen and antibody panel. It is important to know the heterophile (Monospot) test has a 25% false-negative rate in the first week and around 5% by the third week. Treatment is usually supportive care, but in cases of severe disease, steroids and antivirals can sometimes be utilized.
Christopher Heinrich Dr. Mark Wieland	*Dyspnea in the COVID-19 Pandemic: Avoiding Recency Bias* Introduction: Pneumocystis pneumonia (PCP) is an infection caused by Pneumocystis carinii, which commonly colonizes the lungs. Infection under immunocompromised states, the most common of which is human immunodeficiency virus (HIV) infection. However, in HIV negative patients, this can be more difficult to elucidate and treatment can be challenging. Case Presentation: A 78-year-old male with rheumatoid arthritis on methotrexate, squamous cell carcinoma of the tongue status post resection and radiation, and obstructive sleep apnea presented to the outpatient clinic with progressive dyspnea on exertion, fever, and night sweats. He was tested for COVID-19 pneumonia four different times, with negative results. After one month, he presented to the emergency department for persistent dyspnea and was found to have bilateral pulmonary emboli and was initiated on apixaban. However, his clinical condition continued to worsen and was accompanied by a 10-pound weight loss. He presented again to the emergency department where repeat CT scan showed resolved pulmonary emboli but demonstrated parenchymal changes consistent with COVID-19 pneumonia. Other diagnostic considerations included hypersensitivity pneumonitis, rheumatoid lung disease, and methotrexate induced lung injury. He was persistently hypoxic despite supplemental oxygen and he was started on azithromycin for atypical pneumonia. A PET CT was obtained which demonstrated faint sclerotic FDG avoid osseus lesions concerning for possible metastatic

	squamous cell carcinoma. He then underwent bronchoscopy with bronchoalveolar lavage (BAL) with the immunocompromised diagnostic panel. BAL returned positive for pneumocystis pneumonia and he was transitioned from azithromycin to high dose IV trimethoprim sulfamethoxazole and prednisone 80 mg daily with plans for total of 3 weeks of corticosteroids. The etiology of his immunocompromise was unclear but initial thought was given to recurrence of his squamous cell carcinoma, rheumatoid arthritis (or a concomitant inflammatory condition), long-term methotrexate use, or hematologic malignancy. His initial work up demonstrated anemia and thrombocytopenia. He underwent bone marrow biopsy which demonstrated a new large B cell lymphoma which was the most likely source of immunocompromise and contributed to his dyspnea, fatigue, and weight loss. After learning of this diagnosis, he elected to pursue comfort measures and was discharged to hospice, where he passed away surrounded by his family. Conclusion: This case demonstrates several key lessons. First, it is important to maintain a broad differential diagnosis for dyspnea even during the midst of a pandemic where recency bias (a cognitive bias that favors recent diagnoses over historic ones) can cloud clinical judgement. Second, any patient diagnosed with PCP pneumonia needs to undergo evaluation for underlying immunocompromise, with focus on recent steroid administration, hematologic malignancy, underlying inflammatory conditions, or other solid malignancies if the patient has tested negative for HIV. Lastly, it has been demonstrated that PCP in patients without HIV is more difficult to treat and carries a higher mortality.
Jeffrey Heller Tanisha Ronnie	*Rheum for Improvement - A Case of Transverse Myelitis and the Need for Early Rheumatologic Workup* Introduction: Transverse myelitis is characterized by weakness, sensory loss, pain, and bowel and bladder dysfunction secondary to an inflammatory lesion of the spinal cord. It is a common initial presentation of systemic lupus erythematosus (SLE). In fact in 50-60% of patients with SLE and transverse myelitis, transverse myelitis is the initial presentation, prior to other more common sequelae of the disease (1,2). Thus, diagnostic evaluation for rheumatological causes should be pursued when transverse myelitis is suspected. Case Presentation: An otherwise healthy 31 year-old woman, with no significant past medical history, presented to the hospital with three weeks of worsening nausea and vomiting. On day 3 of her hospitalization, the patient developed severe weakness in her lower extremities, urinary retention, and bowel incontinence. MRI showed diffuse inflammation in the spinal cord, suggesting extensive longitudinal myelitis, defined as transverse myelitis involving three or more contiguous vertebral segments of the spinal cord. PLEX therapy and IV steroids were initiated on day 6, given the unknown etiology of spinal inflammation. On hospital day 8, there was no significant improvement in her symptoms with CBC notable for worsening pancytopenia. With the transition of the medical team, further history taking revealed a 6-month history of joint pain and eye inflammation. The patient was not successfully diagnosed with SLE until hospital day 10, and treatment was started on hospital day 12, approximately one week after the development of extensive longitudinal myelitis. Once the diagnosis was established, the targeted treatment for SLE proved effective. Over the course of 6 months, the patient regained her ability to walk, void, and stool spontaneously.

	Conclusion: This case highlights the importance of thorough history taking and pursuing a rheumatologic workup in patients presenting with transverse myelitis. When considering the differential for transverse myelitis there are a number of etiologies that must be considered including vascular lesions, structural lesions, postinfectious, post-vaccination, paraneoplastic, autoimmune, demyelinating and metabolic causes (3, 4). A detailed history with questions asking about visual changes, constitutional symptoms, dermatologic findings, joint and muscle pain, history of pregnancy loss, and overall timing and onset of symptoms can guide the workup. Imaging is the first step in the workup and is twofold in its purpose. It provides radiologic confirmation of the diagnosis and can also indicate whether a compressive lesion is present. Spinal MRI with gadolinium enhancement is the preferred imaging modality. (5, 6) In our patient, transverse myelitis presented as a sudden and life-altering process. Recognition of the etiology through thorough history taking is critical, as the correct diagnosis allows for targeted treatment and resolution of symptoms over time.
Jason Hoard	*MRSA Epidural Abscess in the Setting of Precipitated Opioid Withdrawal: A Case for Buprenorphine Microdosing* Introduction: Bacterial infections due to injection drug use are increasing. Untreated opioid use disorder (OUD) is a major contributing factor in this trend. Although healthcare systems are starting to recognize the importance of early OUD treatment while patients are hospitalized, there are challenges and risks associated with starting buprenorphine. Improper buprenorphine induction can cause precipitated withdrawal, leading to discharges against medical advice, relapse, untreated bacterial infections, and poor clinical outcomes. Case Presentation: A 44 year-old male with history of opioid use disorder (OUD) and injection drug use was admitted to the hospital with methicillin-resistant Staphylococcus aureus (MRSA) bacteremia. He also had thoracic back pain concerning for vertebral osteomyelitis. Appropriate antibiotics were initiated. The patient was started on 8mg-2mg buprenorphine-naloxone (Suboxone), and immediately went into severe precipitated opioid withdrawal. The Suboxone dose was then increased to 10mg twice daily. His withdrawal symptoms worsened, and he left the hospital against medical advice. He returned 5 weeks later with bilateral lower extremity weakness and numbness. He was found to have MRSA bacteremia as well as T8-T9 discitis/osteomyelitis with MRSA epidural abscess causing severe canal stenosis. He required emergent surgical drainage and laminectomy for spinal decompression. On day 2 of admission, microinduction of buprenorphine was initiated at 75 micrograms four times daily. As his post-operative pain improved, buprenorphine was slowly titrated up with good response. After 4 weeks in the hospital, the patient was discharged to an acute care facility to complete 8 weeks of IV antibiotics. His Suboxone dose at discharge was 8mg in the AM, 4mg at noon, and 4mg in the PM. Discussion: This case highlights the risk of precipitated withdrawal during buprenorphine induction in patients with OUD. This is due to the competitive partial agonist effect of buprenorphine, which readily displaces other μ-agonists. Offering buprenorphine to hospitalized patients with OUD is a critical treatment tool. However, initiating buprenorphine has become more challenging due to changes in the illicit drug supply. Heroin has largely been

	replaced with illicit analogs of fentanyl, which are highly lipophilic, distribute widely to adipose tissue, and lead to protracted excretion. This pharmacokinetic profile increases the risk of precipitated withdrawal after starting buprenorphine. Microinduction, also known as microdosing, is an effective method for starting buprenorphine in the hospital setting that decreases the risk of precipitated withdrawal while offering acute pain control with other opioids. With improved symptom control, patients with OUD are less likely to leave against medical advice, leading to improved clinical outcomes.
Abel Hooker	*Prolonged QT: A Case of Cardiac Arrest in a Young Woman* Introduction: A corrected QT interval (QTc) of >440ms in men or >460ms in women characterizes the long QT syndrome (LQTS), a disorder of myocardial repolarization. This syndrome is associated with an increased risk of polymorphic ventricular tachycardia, also known as torsades de pointes. LQTS can be acquired or congenital. The former is usually a result of pharmacologic therapy. The risk of having arrhythmias related to LQTS increases with hypokalemia, hypomagnesemia and bradycardia. Presented below is a case of a young female with a sudden cardiac arrest from polymorphic ventricular tachycardia in the setting of drug-induced LQTS. Case Presentation: A 35-year-old woman with a medical history of alcohol and cocaine use disorder was brought to the ED after being found unable to care for herself at a bus stop. During her stay in the ED, she received ondansetron and droperidol as she was experiencing profound nausea and vomiting. Several minutes later, she was found to have agonal respirations and was in cardiac arrest with initial rhythm of polymorphic VT. ACLS was started and she received 1mg of epinephrine and defibrillation with ROSC. After the arrest, an EKG tracing showed evidence of prolonged QTc (498-544ms) without signs of myocardial ischemia. Potassium and magnesium levels were 2.6mEq/L and 1.9mEq/L, respectively. Repletion of potassium and magnesium resulted in correction of her QTc. During her hospital stay, she received olanzapine and hydroxyzine to treat insomnia. Subsequent EKGs again revealed prolonged QTc (544ms). On her ninth day of hospital stay she underwent EP study with epinephrine challenge without QT prolongation which essentially ruled out LQT1 and LQT2, the most common channelopathies that cause LQTS. After discontinuing QT-prolonging medications, her QT appropriately corrected and was discharged home with the diagnosis of drug-induced LQTS and with close follow-up. Discussion: Prolonged QT interval can be inherited or acquired. Anti-psychotics, anti-emetics, and antihistamine medications are known to prolong the QT interval. A large observation study found that use of QT prolonging medication was associated with an increased risk of sudden cardiac death (adjusted OR 2.7). The feared consequence of a prolonged QT interval is polymorphic ventricular tachycardia, also known as torsades de pointes. Many causes of long QT are multifactorial with medications and electrolyte abnormalities being implicated. These cause an abnormality in myocardial repolarization which causes patients to be prone to the “R on T” phenomenon which causes torsades de pointes. It is important to be aware of the synergistic effects that medications and electrolyte abnormalities have on prolonging the QT interval and putting patients at risk for deadly arrhythmias.
Sara Hooshmand	*Dysphagia as a side effect: The disease or the medication?*

Dr. Emily Olson	Background: Extrapyramidal symptoms (EPS) are well-recognized adverse effects of antipsychotic medications. Symptoms include acute dystonia, akathisia, parkinsonism, and tardive dyskinesia. A less commonly recognized symptom of EPS is dysphagia, which is critical to recognize, as it can lead to aspiration pneumonia, choking, and coughing. Other rare functional etiologies for dysphagia include thyrotoxicosis, myositis, and myasthenia. In this report, we describe a case of dysphagia in an adult patient on neuroleptic medications and recently diagnosed with thyrotoxicosis. Case Report: A 40-year-old man with schizoaffective disorder and bipolar II disorder on paliperidone and haloperidol presented to the emergency department with dysphagia, coughing, tremors, and palpitations. His exam was significant for resting tremor, tachycardia, and diaphoresis. There were no facial dyskinesias noted on the exam. He had copious secretion in the oropharynx. Chest X-ray identified a focal opacity in the right lower lobe, concerning for pneumonia. A video fluoroscopic swallowing study confirmed oropharyngeal dysphagia, and the patient began a modified diet. His complete blood count, urea nitrogen, electrolytes, plasma glucose, and creatinine phosphokinase were within normal limits. He had a serum TSH level of <0.03 (normal 0.3 - 4.2 μIU/ml), free T4 of 4.0 ng/dl (0.9–1.7 ng/dl), total T3 of 203 (80-200 ng/dl), negative thyroid stimulating immunoglobulin (TSI), negative thyrotropin receptor autoantibodies (TRAb), and elevated thyroglobulin of 9.0 (normal undetectable). Based on the above results, the patient was diagnosed with thyrotoxicosis of undetermined etiology and initiated methimazole and metoprolol. However, the patient continued to have worsening tremors and dysphagia that ultimately required nasogastric tube feeds, prompting further evaluation. Thyroid ultrasound was unremarkable, and radioactive iodine uptake scan was consistent was silent thyroiditis. Due to the lack of improvement in his symptoms, methimazole was discontinued. Other etiologies of dysphagia were considered, and he was ultimately treated for EPS. In addition to decreasing the neuroleptic medications, he was started on benztropine and diphenhydramine. His symptoms improved and a repeat swallow study ten days later showed complete improvement of his dysphagia. Conclusion: Our case describes a patient on neuroleptic medications for underlying psychiatric conditions who was diagnosed with dysphagia and thyrotoxicosis. Unfortunately, the patient's symptoms did not improve with the treatment of thyrotoxicosis but did have remarkable improvement with treating EPS. We hope this case increases awareness of unique presenting symptoms of EPS, as prompt recognition is critical to prevent complications.
James Howick Dr. Hadiyah Audil Dr. Yuan Yao Dr. Camilo Bermudez Johnny Dang Kimberly Wang Dr. Chris Aakre	*A Case of May-Thurner Syndrome in an Otherwise Healthy Young Female* Introduction: May-Thurner syndrome is a rare syndrome in which the right common iliac artery compresses the left common iliac vein against the lumbar spine, causing iliofemoral deep vein thrombosis (DVT). Although infrequently diagnosed, it has an estimated prevalence in the general population of 20%. Treatment with anticoagulation is typically insufficient given the extent of the clot burden, and often requires intervention to prevent progression or extension. Case Presentation: A 38-year-old otherwise healthy female presented to the emergency department with back pain and left lower extremity numbness.

	Lumbar x-ray showed no fracture. The patient was thought to have sciatica and was discharged with Flexeril and Lidoderm patches. The following day, she noted a considerable increase in left-sided back pain and left leg swelling, prompting return to the ED. Ultrasound revealed an extensive, nearly occlusive thrombus throughout the entire lower extremity, from the common femoral through peroneal veins and extending into the common iliac vein. CT abdomen pelvis venogram demonstrated the DVT completely occluding the left common, internal, and external iliac veins and extending to involve the lower 5 cm of the inferior vena cava. She had no prior history of venous or arterial thrombosis or miscarriages, and family history was negative for the same. Her history was significant for a recent 24-hour automobile trip and 10 pack-year history of smoking; she was on contraception with progesterone-only Depo-Provera shots. Given the extensive nature of the clot, further thrombophilia workup was sent. A heparin drip was started, and Interventional Radiology was consulted. The patient underwent venogram with thrombectomy, at which time imaging and intraoperative findings were consistent with the rare diagnosis of May-Thurner syndrome. Post-procedure, the patient reported a drastic decrease in symptoms. The following morning, her pain and swelling were markedly improved. The patient was discharged from the hospital on long-term anticoagulation the following day and scheduled for a follow-up left lower extremity ultrasound in 4 to 6 months. Conclusion: This case demonstrates the importance of quickly recognizing the diagnosis of May-Thurner syndrome, particularly in young females in the second or third decade of life, due to the large extent of clot burden; indeed, complications of untreated May-Thurner syndrome include massive pulmonary embolism and phlegmasia (in which extensive venous clot burden compromises arterial flow and can threaten limb viability), which could potentially be fatal. Early recognition and appropriate, aggressive management are therefore of vital importance.
Jack Inglis Dr. Mary Bernton	*The Sound of Heartbreak* Introduction: Post-MI mechanical complications, including rupture of the free wall, papillary muscle, or intraventricular septum (IS), are relatively rare but devastating sequelae of NSTEMI and STEMI. While uncommon, internists who care for hospitalized MI patients are likely to encounter several post-MI mechanical complications throughout the course of their careers. Early recognition of these complications is critical given the high mortality and need for intervention. Case Description: A 63-year-old man with a history of stroke, hypertension, and hyperlipidemia presented to the emergency department with 6-7 hours of severe, left-sided chest pain and shortness of breath. He had 2 weeks of episodic, typical chest pain but otherwise no cardiac history. Troponins were elevated and EKG was consistent with inferior STEMI. The patient was taken to the cath lab and coronary angiography revealed multi-vessel disease and 99% stenosis of the right posterolateral AV branch which was stented. Upon arrival to the floor, the patient was hemodynamically stable and initially said his chest pain had improved significantly. However, 30 minutes later, he reported sudden-onset chest pain and dyspnea. On physical exam, he was alert and well-perfused. Auscultation revealed a new, 4/6 holosystolic murmur heard best at the left, lower sternal border. Echocardiography revealed a large VSD with left-to-right shunt and an inferior wall aneurysm. He was started on a nitro drip for afterload reduction and transferred to the ICU for management of impending cardiogenic shock. An intra-aortic balloon pump was placed and

	the patient was transferred to an outside hospital for surgical VSD repair, aneurysmectomy, and CABG. The patient passed away two weeks later. Discussion: This case illustrates a typical course for the most common post-MI mechanical complication: rupture of the IS. Because infarcted myocardium is not amenable to surgical repair, the prognosis is poor even with prompt identification. However, early diagnosis can help expedite clinical decisions and improve the chances of a good outcome. A large majority of post-MI septal ruptures result in a characteristic holosystolic murmur, and therefore diligent cardiac auscultation can result in diagnosis prior to the onset of cardiogenic shock and associated complications.
Isla Johnson Dr. Gretchen Colbenson	*Taming of the Sprue: Celiac Disease-Associated Neutropenia as Cause of Prolonged Neutropenia* Introduction: Neutropenia is a hematologic abnormality with a broad differential diagnosis, and especially in hospitalized patients, can often be attributable to illness or medication effect. However, prolonged neutropenia merits extensive workup as it confers increased risk of infection and, with strict neutropenic precautions, limits patients' ability to return to normal life. Physicians should be aware of uncommon etiologies, such as Celiac Disease (CD), during workup for neutropenia. Case Presentation: A previously healthy 42-year-old man was hospitalized for norovirus gastroenteritis after presenting with 8 days of severe diarrhea. During this admission, workup revealed a positive anti-tissue transglutaminase IgA concerning for CD. On admission, he was found to be neutropenic with a white blood cell count of 1.5x109/L and an absolute neutrophil count (ANC) of 0.18x109/L; other cell lines were within normal limits. He remained persistently neutropenic throughout admission. He had no prior history of hematologic abnormalities or of recurrent infections. He was discharged on neutropenia precautions and followed up in Hematology Clinic 3 weeks later where neutropenia had improved but not resolved, with an ANC of 0.3x109/L. He had no significant occupational exposures, alcohol or drug use, or B-symptoms and was not on any medications associated with neutropenia. Lab workup, including CMV, EBV, nutritional labs, peripheral smear and peripheral flow cytometry were all normal. ANA was weakly positive at 1.3; he did not have other symptoms concerning for autoimmune disease. He underwent bone marrow biopsy which did not show any evidence of malignancy, myelodysplasia or other abnormalities. During this period of several weeks, he remained neutropenic with ANC <0.5x109/L so he was unable to return to work. Given the persistent neutropenia, it was considered unlikely to be secondary to his now-resolved gastroenteritis. He underwent upper endoscopy where duodenal biopsies were consistent with celiac sprue. Given this confirmed diagnosis of Celiac Disease with an otherwise unremarkable hematologic workup, he was considered to have CD-associated neutropenia. He started a gluten-free diet. He remains neutropenic but, as this is CD-associated without recurrent infection, has been able to return to work. Conclusion: Celiac Disease (CD), a gluten-sensitive enteropathy, is associated with a wide variety of hematologic abnormalities. Classically, it is associated with anemia secondary to nutrient deficiency. CD-associated leukopenia and neutropenia is uncommon and thought due to either autoimmune destruction or production issues due to deficiencies in folate, B12 or copper. A gluten-free diet is only occasionally associated with improvement in the neutropenia. In previous analyses, the severity of ANC did not correlate with incidence of

	infection (Vijayvargiya et al., Blood 2015). In the case of severe infection, G-CSF responses have been favorable, and this can be used if needed.
David Kazadi Dr. Michael Eastman	*Vasopressin, Vasopressinase and the Woman: A Tale of Polyuria and Diabetes Insipidus in a Pregnant Woman with Bipolar Depression* Introduction: Diabetes Insipidus (DI) is a disorder of urine concentration that can lead to electrolyte abnormalities and a heavy polyuria burden. It results from the unavailability of — or resistance to — antidiuretic hormone (ADH), also known as vasopressin. In central DI, ADH production or availability is impaired, while tubular response to ADH is intact. In peripheral DI, ADH is available and active, but tubular response to ADH is deficient. Here we present the case of a woman with polyuria likely from central, vasopressinase-related transient DI of pregnancy and lithium-related peripheral DI, who arguably also had a component of primary polydipsia. Case Presentation: A G1P1 37-year-old woman was admitted to the obstetrics service for elevated blood pressures at 35 weeks and is being evaluated for increased water intake, polyuria, and DI. She is POD3 from cesarean section for a pregnancy complicated by preeclampsia. Her water intake increased significantly during pregnancy. She has also experienced increasing polyuria, with significant impact on her life. She has no known personal or family history of kidney disorder. Her history is significant for bipolar depression type I, well controlled on lithium for 15 years. After a 1.5-liter/day fluid restriction, her morning serum sodium increased to 149 mmol/L from 145, and her urine osmolality increased to 248 mOsm/kg from 170. She was challenged with 0.1 mg of desmopressin, and her urine osmolality further increased to 309 mOsm/kg. Interestingly, her serum sodium on admission was 131 mmol/L and her serum osmolality 268 mOsm/kg. At that time, her urine osmolality was 73 mOsm/kg and her ADH level was 1.2 pg/mL. A daily desmopressin regimen was initiated; the patient's polyuria and polydipsia subsided, and she ultimately stopped needing desmopressin. Aripiprazole replaced lithium for bipolar depression treatment. Discussion: Transient diabetes insipidus of pregnancy due to placenta-produced vasopressinase is high on the differential diagnosis for this patient. Her history of preeclampsia, low-normal ADH level on admission, and symptomatic improvement following delivery argue for this view. Her ability to increase urine concentration following water restriction points to residual native production of — and response to — ADH. Chronic lithium use could certainly have impaired adequate functioning of collecting tubule water channels, leading to (sub) clinical decline in urine concentrating faculty, and creating a diathesis for renal resistance to ADH and nephrogenic DI, exacerbated during pregnancy. Her limited response to vasopressinase-resistant desmopressin supports this argument. Admission studies point to a component of primary polydipsia as well, though no substantial use of phenothiazines could be ascertained. There was low concern for other causes of central DI, including sarcoidosis, postpartum pituitary gland necrosis, diabetes mellitus, hypoaldosteronism, and use of anticholinergic medication.
Ahmad Khalaf	*Presentation of Chest pain with a Challenging Diagnosis* Case Presentation: a 37-year-old gentleman was admitted to a health care facility on 4/13/2021 with a history of Left-sided pleuritic chest pain, cough, and low-grade fever. The physical exam was unremarkable. The respiratory

	viral panel and COIVD test were negative. Basic labs were unremarkable. Chest X-ray was negative, CTPA was unremarkable except for mildly enlarged left hilar lymph nodes most likely reactive. ECG showed nonspecific subtle diffuse ST-segment abnormalities with normal sinus rhythm, exercises stress echo was negative for ischemia or pericardial effusion. The patient did not receive antibiotics and improved with NSAIDs. Impression was a mild viral illness, and he was discharged with a plan of near follow-up. On 4/18 presented again to our ER with persistent fever (reaching 102.5), cough, chest pain, and skin rash. He denied associated SOB, diaphoresis, N/V. The rash started on his face and then elbows. Physical exam was remarkable for fever and non-scaly maculopapular rash over the elbows and the face. Labs showed neutrophilic leukocytosis, normocytic anemia, and high CRP. ECG was unremarkable but he was found to have mildly elevated troponin of 77 ng/L which was trending down. The cardiology team was consulted and he was started on high dose ibuprofen and colchicine for possible myopericarditis. Cardiac MRI showed no cardiac abnormality but incidentally found enlarged infrahilar lymph node and mild splenomegaly with bilateral minimal pleural effusion. He had a negative workup for blood cultures, infectious mononucleosis, HIV, Quantiferon, hepatitis B and C, syphilis, streptococcal infection, CMV, EBV, parvovirus B19, and Autoimmune workup. Based on the above findings rheumatology team was consulted for suspicion of adult-onset still's disease. Further workup showed very high ferritin of >12500ng/mL (upper limit is 400), patient was started on prednisolone 0.5mg/kg daily. His fever improved gradually but not fully. PAN CT scan done on 6/17/2021 showed worsening lymphadenopathy and splenomegaly with sizable Left axillary lymph node. Left axillary LN Biopsy was negative for TB, fungal infection, and lymphoma. He was recently started on Anakinra by the rheumatology team. Discussion: This is a challenging case presented with chest pain. Based on the above clinical, laboratory, imaging studies, and after ruling out infectious, neoplastic, and rheumatologic diseases, our patient was diagnosed with Adult-onset Still's disease (AOSD). AOSD is a rare inflammatory disorder that affects multiple organ systems. The etiology is unknown. The major clinical features include daily high spiking fever, Evanescent salmon-colored rash, arthritis or arthralgia. Diagnosis is clinical, and other infectious, neoplastic, and rheumatologic diseases must be excluded. Treatment includes NSAIDs, glucocorticoids, DMARDs such as methotrexate, and IL-1 inhibitors. Most patients respond to therapy, and the prognosis of AOSD with treatment is good.
Mohammed Khalid	*Hidden in Plain Sight: A Case of Amiodarone Pulmonary Toxicity (APT)* Introduction: Amiodarone is an antiarrhythmic agent commonly used to treat supraventricular and ventricular arrhythmias. This drug is an iodine-containing compound that tends to accumulate in several organs, including the lungs. It has been associated with a variety of adverse events. Of these events, the most serious is amiodarone pulmonary toxicity. Although the incidence of this complication has decreased with lower doses of amiodarone, it can occur with any dose. Because amiodarone is widely used, all clinicians should be vigilant of this possibility(1). Case description: An 89 y.o. Male with a medical history of HFpEF,

	Pulmonary HTN, A-fib/A-flutter (was on amiodarone 200 mg daily for 4 months and was then stopped 1 month before presentation), CAD, CKD, T2DM, and HTN. His home nurse saw him and noted an O2 sat in the 80s, usually requiring 1-2L but now requiring 4L O2. He presented to the ED reporting acute on chronic SOB. The patient was tachypneic, and normotensive. Initial labs with BMP significant for Cr 2.3 mg/dl. VBG significant for pH 7.42, CO2 of 47 mmHg, Bicarb of 30 mEq/L. CBC showing Hgb of 6.7 g/dl. NT proBNP of 1720 pg/mL, with unremarkable troponins. PT of 35.2 sec and INR of 3.0. Initial imaging was consistent with pulmonary edema. The patient received 1 unit of RBCs and was started on aggressive diuresis with no response. He went from 4L O2 NC to requiring BiPAP and was transferred to CMIC for that reason. Cardiology and pulmonology were consulted. Additional workup included TTE, which was unremarkable. Follow-up CXRs with worsening opacities. ABG showed a pH of 7.28, CO2 of 81, and Bicarb of 35. Follow-up VBGs with persistently elevated CO2 ranging from 70s-90s Bicarb in the 40s. The patient's SOB, hypoxia, and worsening CXRs were most likely due to amiodarone pulmonary toxicity. He was started on glucocorticoids, resulting in remarkable improvement in SOB. Discussion: Amiodarone pulmonary toxicity incidence ranges between 5-13%(2). Typically manifests as diffuse interstitial pneumonitis with varying degrees of fibrosis. Risk factors for APT include a high dose (>400 mg/day), duration > 2 months, increased age, and preexisting lung disease(1). Treatment is stopping amiodarone and initiating glucocorticoids. This case shows that while it does not have to be at the top of the differential diagnosis, it should still be relevant in patients exposed to amiodarone. Being a widely used medication, clinicians should be aware of the possibility that despite stopping amiodarone, its adverse effects may still present later, resulting in a need to consider targeted monitoring allowing for early diagnosis and management, minimizing morbidity and mortality. References: 1. Wolkove N, Baltzan M. Amiodarone pulmonary toxicity. Can Respir J. 2009;16(2):43-48. doi:10.1155/2009/282540 2. Schwaiblmair M, Berghaus T, Haeckel T, Wagner T, von Scheidt W. Amiodarone-induced pulmonary toxicity: an under-recognized and severe adverse effect? Clinical research in cardiology: official journal of the German Cardiac Society. 2010;99(11):693-700. doi:10.1007/s00392-010-0181-3
Jonathan Knott Dr. Caroline Matchett Dr. Matthew Koster	*Treatment-Resistant Autoinflammatory Disease: A Unique Case of VEXAS Syndrome in a 60-Year-Old Male* Introduction: VEXAS syndrome is a newly identified adult-onset inflammatory syndrome.1 It is characterized by a somatic mutation affecting methionine-41 in the UBA1 gene resulting in diminished cellular ubiquitylation and hyperinflammation affecting multiple organs, including the skin, lungs, blood vessels, and cartilage.1-2 VEXAS syndrome is associated with considerable morbidity and mortality as symptoms are refractory to typical treatments including steroids and steroid-sparing agents.2 Case Description: A 60-year-old male, non-smoker originally presented to an outside hospital with chest pain, left-sided spontaneous pneumothorax, and diffuse ground glass opacities (GGO). He then developed recurrent fever, thrombophlebitis, urticarial dermatosis, episcleritis and cytopenias with elevated acute phase reactants. Comprehensive infectious evaluation was

	negative. Bone marrow was hypercellular but did not disclose overt malignancy. He responded to high-dose glucocorticoids, but symptoms returned on tapering below 20mg/day. Trials of dapsone and omalizumab were unsuccessful for control of his rash. He presented to Mayo Clinic for evaluation with active symptoms while on mycophenolate 1000 mg twice daily and prednisone 20 mg. A repeat bone marrow revealed vacuolated erythroid and granulocytic precursors. He was hospitalized due to worsening dyspnea, fever and rash. Chest CT revealed progressive diffuse GGO. Bronchoscopy with broad infectious workup was again negative. Skin biopsy showed neutrophilic dermatosis. The constellation of symptoms was concerning for VEXAS and genetic testing confirmed pMet41Val mutation on UBA1. The patient's symptoms initially improved with methylprednisolone, but his disease remained refractory to steroid tapering and steroid-sparing agents. Discussion: VEXAS syndrome is a severe, progressive disease with clinical features of both rheumatologic and hematologic conditions. A diagnosis of VEXAS syndrome should be considered in patients with treatment-refractory inflammatory disease with associated hematologic abnormalities. Genetic testing is required for confirmation. High-dose glucocorticoids are only transiently effective and carry significant toxicity. Steroid-sparing agents also have a temporizing treatment effect, and none have been effective in improving cytopenias or myelodysplastic features.2 Randomized clinical trials are needed to evaluate different therapeutic strategies to improve outcomes in patients with VEXAS syndrome.3 1. Beck DB, Ferrada MA, Sikora KA, et al. Somatic Mutations in UBA1 and Severe Adult-Onset Autoinflammatory Disease. New England Journal of Medicine. 2020;383(27):2628-2638. 2. Grayson PC, Patel BA, Young NS. VEXAS syndrome. Blood. 2021 Jul 1;137(26):3591-3594. doi: 10.1182/blood.2021011455. PMID: 33971000. 3. Bourbon E, Heiblig M, Gerfaud Valentin M, Barba T, Durel CA, Lega JC, Barraco F, Sève P, Jamilloux Y, Sujobert P. Therapeutic options in VEXAS syndrome: insights from a retrospective series. Blood. 2021 Jul 1;137(26):3682-3684. doi: 10.1182/blood.2020010177. PMID: 33619558.
Marcela Kuijpers	*Where it Leads, Bacteria will Follow: A Case of Lead-Associated Bacterial Endocarditis* Introduction: Cardiac implantable electronic devices are becoming more commonplace with an aging population and expanding number of indications. Pacemaker lead endocarditis is a rare but serious complication with high morbidity and mortality. This case represents how pacemaker endocarditis presents with unspecific symptoms, often leading to a delay in diagnosis. Case Presentation: A 72-year-old woman with end stage renal disease (ESRD) on hemodialysis and heart failure with reduced ejection fraction (EF 35%) with an ICD placed 5 years ago was brought into the emergency department with one day of generalized body pain, shortness of breath and chest pain. History was provided by the patient's care giver as the patient was obtunded on arrival to the ED. The patient's care giver added that she was in her usual state of health up until one day ago. She was found to be hypotensive and hypoxic on initial exam without any focal findings. Notably, there was no fluctuance or erythema surrounding the ICD pocket. Additional work up

	revealed a WBC count of 20,000 k/cmm and a lactate of 3.9 mmol/L. Imaging studies including an EKG, CXR, and CT scans of the head, chest, abdomen, and pelvis were unremarkable except for scattered nodules in the right upper lobe of the lung on CT scan. Patient was treated empirically for possible community acquired pneumonia. Blood cultures resulted with methicillin sensitive Staphylococcus aureus and antibiotics were narrowed to Cefazolin, Nafcillin was deferred due to allergy. An echocardiogram was obtained and showed no evidence of bacterial endocarditis or vegetation on the ICD lead. Despite appropriate antibiotic treatment, she had ongoing encephalopathy with worsening leukocytosis. Repeat CT scan of the chest showed bilateral opacities consistent with septic embolic and CT scan of the spine was notable for L5-S1 osteomyelitis. A TEE obtained showed multiple, independently mobile echogenic masses attached to the pacemaker wire consistent with infective vegetation, confirming a diagnosis of lead endocarditis. In addition to ongoing antibiotic treatment, extraction of pacemaker was essential for resolution of infection. She underwent extraction of Bi-V system and Angiovac of lead vegetations. The cardiac device was not replaced as she was not pacer dependent. Clinical status worsened and decision was made to transition to comfort cares. Discussion: Prevalence of IE is documented anywhere between 3–10 cases per 100 000 per year. Unfortunately it is initially misdiagnosed in over 2/3 of older patients as the initial symptoms are vague such as malaise and musculoskeletal pain. Musculoskeletal symptoms are present in 23-44% of all cases. Joint pain can represent spread of infections and/or infarction from immune complex deposition. As evidenced in this case, a TEE must be performed for accurate diagnosis. In addition to appropriate antibiotic therapy, extraction of infected material is needed for the cure.
Kenzie Lee	*Anterior Mediastinal Mass: A Diagnostic Evaluation* Introduction: Mediastinal masses are relatively uncommon with a prevalence of 0.73 to 0.9 per 1000 and nearly 60% occurring in the anterior mediastinum. The most common etiologies of anterior mediastinal masses (AMM) include thymic malignancy or thymoma 40%, lymphoma 25%, thyroid and endocrine tumors 15%, teratoma 10%, and germ cell malignancies 10%. AMM can be challenging to diagnose; gender and age are important characteristics as certain pathologies are more common based on demographic group. In females less than 40 years, lymphoma is most common with Hodgkin and large cell non-Hodgkin lymphomas predominating. Case Presentation: A previously healthy 30-year-old female presented with two months of non-productive cough, shortness of breath, and intermittent, progressive left-sided pleuritic chest pain. Initial urgent care evaluation was notable for negative COVID-19 testing. Chest x-ray (CXR) showed opacification of the left hemithorax with moderate pleural effusion. She completed a course of empiric antibiotics for community acquired pneumonia with minimal improvement followed by prednisone burst for presumed bronchospasm with mild improvement. After completion of therapy, symptoms worsened prompting reevaluation in the emergency department. She reported associated 7 kg unintentional weight loss, night sweats, diarrhea, and myalgias. Repeat CXR identified progressive opacification in the left chest. CT angiography of the chest showed a large lobulated heterogeneous left anterior mediastinal mass measuring 7.3 cm by

	10.9 cm by 7.3 cm, as well as a large pleural effusion and moderate pericardial effusion. Laboratory evaluation was notable for hemoglobin 10.1 g/dL and leukocytosis of 23.57/μL with 91% neutrophils. Alpha-fetoprotein (AFP) and beta-HCG were normal and lactate dehydrogenase was elevated. Cytology by CT-guided mediastinal biopsy was consistent with large B-cell lymphoma with extensive necrosis. Pleural fluid was positive for malignant cells. PET-CT scan showed FDG-avid large anterior mediastinal mass and mediastinal lymphadenopathy without other FDG-avid disease. Bone marrow biopsy and lumbar puncture were negative for malignancy. She met with reproductive endocrinology for fertility preservation prior to starting appropriate chemotherapy. Discussion: This case illustrates the clinical presentation and diagnostic evaluation of an anterior mediastinal mass in a young female patient. Minimal response to antibiotics for pneumonia in young patients should prompt further imaging. Appearance on high-resolution CT is important in differentiating etiology of AMM. This patient had a large heterogeneous mass with associated pleural effusion, suggesting the more aggressive large B-cell lymphoma, especially in the setting of "B" symptoms. Comparatively, teratomas are diagnosed by imaging alone, and a homogeneous, lobulated mass raises suspicion for thymoma. For the general internist evaluating an anterior mediastinal mass, demographic information, clinical symptoms, and appearance on imaging are critical for appropriate diagnosis and staging prior to oncology involvement.
Katy Lehenbauer	*Stress Cardiomyopathy Within the COVID-19 Pandemic* Introduction: COVID-19 has become a primary suspect diagnosis in many patients who present acutely ill during the pandemic. Interestingly, the incidence of stress cardiomyopathy has increased significantly compared to pre-pandemic times, from less than 2% up to 7.8% in patients with acute coronary syndrome, suggesting an association between collective public stress triggered by COVID's presence and individuals' physiologic stress manifesting as stress cardiomyopathy.1 Case Presentation: A previously healthy 41 year-old female presented to the ED with acute onset of subjective fevers, palpitations, and emesis. She was found to be profoundly hypoxic with a third heart sound and bilateral crackles. CTA chest demonstrated patchy and nodular opacities in bilateral lungs, ground-glass attenuation, and interlobar septal thickening, suspicious for a viral pneumonia such as COVID-19. Bedside echocardiogram showed a severely reduced ejection fraction of 25-30%, with mid-anteroseptal, inferior, and lateral hypo- to akinesis. Labs were notable for elevated inflammatory markers, troponin, and BNP. Despite two negative COVID-19 tests, the patient remained a PUI given her unvaccinated status and profound illness. Evolving serial EKGs showed new T wave inversions and later a left anterior fascicular block concerning for a lesion in the left anterior descending artery. Emergent coronary angiography showed normal coronary arteries and an elevated left ventricle end diastolic pressure. The patient was transferred to the nearest ECMO-capable facility in anticipation for deterioration from a suspected acute myocarditis. Borderline oxygen saturations were maintained on high-flow nasal cannula and BiPAP; only after a third negative COVID test was the patient's isolation status lifted. Aggressive diuresis generated a rapid improvement in respiratory status.

	Cardiac MRI showed no evidence of infarction, fibrosis, infiltrative disease, or edema consistent with myocarditis. Ejection fraction had normalized to 55% with no persistent structural abnormalities. The patient was discharged on hospital day four with a clinical picture most consistent with stress cardiomyopathy and acute heart failure with recovered ejection fraction. Discussion: This case represents how the COVID-19 pandemic has contributed to physician anchoring bias, while also exemplifying its propensity to cause detrimental public health effects by its mere presence. This unvaccinated patient was tested for COVID-19 three times during her hospitalization, despite studies showing rapid antigen testing sensitivity of approximately 93%.2 Conclusion: Not only are hospitals being filled continuously with those diagnosed with COVID-19, they are also being occupied by patients who become ill as a potential result of simply existing within the pandemic's unprecedented times. Citations: [1] Jabri A. Incidence of stress cardiomyopathy during the coronavirus disease 2019 pandemic. JAMA Netw Open. 2020;3(7):e2014780. doi:10.1001/jamanetworkopen.2020.14780. [2] Pilarowski G, et al. Performance Characteristics of a Rapid Severe Acute Respiratory Syndrome Coronavirus 2 Antigen Detection Assay at a Public Plaza Testing Site in San Francisco. J Infect Dis. 2021;223(7):1139-1144. doi:10.1093/infdis/jiaa802.
Savannah Liddell Dr. Megan Dulohery Scrodin	*Epithelioid Hemangioendothelioma: A Case of Mistaken Identity* Introduction: Epithelioid hemangioendothelioma (EHE) is an extremely rare vascular sarcoma that has a wide variety of clinical presentations including as a mediastinal mass. Similarly, fibrosing mediastinitis is an inflammatory process which often presents as an insidious mediastinal mass composed of scar tissue that extends into surrounding structures. Presented here is an unfortunate case of EHE mistaken for fibrosing mediastinitis. Case Presentation: A 51-year-old women originally presented to her primary care physician with the chief complaint of a 6-month history of shortness of breath, cough, and facial swelling that occurred with exercise. Physical exam revealed distended neck veins and facial edema. A CT scan revealed an infiltrative mediastinal mass encasing the trachea and superior vena cava (SVC). The compression resulted in severe narrowing of the SVC at the level of the carina. These findings were thought to be secondary to fibrosing mediastinitis. Infectious disease evaluation was negative for Histoplasma antibodies and Aspergillus IgE. The patient was treated with SVC angioplasty and stenting. The procedure was complicated by embolism of the stent to her right pulmonary artery resulting in hemopericardium that required hospitalization for a pericardial drain. Repeat imaging 7 months later showed narrowing of the right upper bronchus and continued SVC stenosis. Her ongoing SVC syndrome resolved after a repeat intervention for stenting of the SVC. Unfortunately, over the course of the next month the patient began to experience progressive shortness of breath. Bronchoscopy was performed and revealed an infiltrating process involving the distal trachea and right main bronchus. The right upper lobe was occluded by severe stenosis. Repeat CT imaging suggested enlargement of the infiltrative mass. The patient was sent for second opinion at our institution for further work up. Physical exam was significant for rhonchi throughout the right lung and progressive dyspnea. A second bronchoscopy was performed and identified severe narrowing and

	necrosis of the distal trachea and proximal right mainstem bronchus. This was treated with a Y-stent. The patient's symptoms shortness of breath were markedly improved. Biopsy revealed epithelioid hemangioendothelioma (EHE). Unfortunately, the tumor was surgically unresectable. Treatment was initiated with radiation therapy. Conclusion: This case of mistaken identity reveals the absolute importance of ruling out malignancy prior to the diagnosis of fibrosing mediastinitis. Fibrosing mediastinitis and EHE can both present as mediastinal masses that can compress surrounding structures. Evaluation of patients with suspected fibrosing mediastinitis should include testing for histoplasmosis, immunoglobulin G4-related disease, and malignancy. Tissue biopsy is necessary to exclude malignancy and IgG4-positive plasma cells. EHE can arise anywhere in the body and is often resistant to systemic chemotherapy. Surgery is preferred treatment when complete resection is possible; however, EHE is radiosensitive. Radiation can be used adjuvant or for palliative treatment.
Alex Liu	*A Cancer Syndrome in a Leather Bottle* Introduction: Cancers with widespread metastatic involvement can present atypically. Here, we present a case of stage IV diffuse gastric cancer which presented with skeletal involvement, back and leg pain, and blood dyscrasia, but notably without GI symptoms. Case Presentation: Our patient is a 59-year old healthy male with hyperlipidemia who presented with 7 weeks of low back pain as well as sharp pain which radiated down his left leg. Also, in the past 3 weeks, he endorsed spontaneous bruising and 25-pounds of unintentional weight loss. He denied other symptoms, including chest pain, shortness of breath, and any GI symptoms. Physical exam was significant for sporadic ecchymoses and pain on palpation of the lower spine. MRI showed hypointense lesions in the sacrum, ilia, and diffuse involvement of the vertebral bodies. PET CT showed PET-avid lesions throughout the skeleton but no evidence of a primary lesion. Serum sodium was 119 mEq/L, hemoglobin 7.6 g/dL and platelets 65,000. Peripheral smear showed rare schistocytes, Carcinoembryonic antigen was 15.3 (RR: <=3), CA 19-9: 179 (RR:<35), and CA 125: 36 (RR:<46). Calcium, SPEP and UPEP were normal. Bone biopsy showed poorly cohesive signet ring adenocarcinoma. CT chest, abdomen, and pelvis showed diffuse thickening of the stomach wall. EGD with biopsy matched the result of the bone biopsy. We diagnosed the patient with linitis plastica with metastatic spread. Family history revealed that the patient's mother had metachronous breast cancers in both the right and left breasts, as well as some form of stomach cancer. This combination suggests the hereditary cancer syndrome of Hereditary Diffuse Gastric Carcinoma (HDGC), though without knowledge of the mother's cancer histology, pedigree information was insufficient to meet diagnostic criteria and genetic testing was not done. He was started on palliative radiation and chemotherapy. Discussion: We present a case of an atypically presenting linitis plastica. The absence of GI symptoms with cytopenias and extensive bone involvement without evidence for a primary solid tumor on PET, initially made us consider multiple myeloma. However, tumors with signet ring pathology are often not PET-avid. Given the implications for the patient and his blood relatives of a

	possible hereditary cancer syndrome affecting both male and especially female relatives, further investigation into HDGC by obtaining prior medical records and slides, and/or CDH1 genetic testing, should be considered. There are screening and preventive treatment implications for the rest of the patient's family, which include detailed 30-minute upper endoscopy every 6-12 months with multiple random biopsies and biopsies of subtle lesions, beginning 5-10 years prior to the earliest cancer diagnosis in the family along with H. pylori screening, prophylactic total gastrectomy, as well as breast cancer screening and prophylactic surgery.
Rosemary Liu Dr. Rena Singleton	*REMAPping COVID-19* Introduction: Effective goals of care conversations have become increasingly important with the onset of the COVID-19 pandemic, particularly considering that COVID-19 can cause significant morbidity and mortality even in relatively healthy individuals. The REMAP framework provides guidance for how to navigate these complex conversations in a patient-centered way. Case Description: A previously healthy 80 year old male presented with four days of shortness of breath, dry cough, dizziness, fatigue, decreased appetite, and low-grade fever. After being found to be hypoxic to 85-80% on room air during a visit to his primary care doctor, he was sent to the ED where vitals were temperature of 100.7F, respiratory rate of 18, pulse of 89, blood pressure 121/77, and oxygen saturation of 78%. Physical exam revealed crackles in bilateral lower lung fields. CXR demonstrated bilateral interstitial changes concerning for COVID pneumonia, and COVID testing returned positive. Initial workup was notable for leukocytosis of 12.9, elevated AST (106), BNP (367), CRP (205), LDH (449), ferritin (823). Due to elevated D-dimer (1.59), CT chest was obtained which showed bilateral multilobar pulmonary space disease without evidence of pulmonary embolism. He was started on dexamethasone, remdesivir, azithromycin, ceftriaxone, therapeutic DVT treatment and high flow nasal cannula. Due to rising inflammatory markers, tocilizumab was initiated and he was broadened to Zosyn for antibiotic coverage. He ultimately required intubation for worsening hypoxia. His course was subsequently complicated by severe ARDS, pneumomediastinum, pneumopericardium, hypotension requiring pressor support, and hospital acquired pseudomonal pneumonia. With persistent hypoxia and tenuous respiratory status, he was initiated on Flolan. Eventually palliative care was consulted for goals of care discussion support in light of lack of clinical improvement. In discussing patient goals with his family members, they shared that he was a man who, up until infection with COVID-19, had continued to be highly independent and was actively working on his greater than 100 acre family farm. A care conference with the family was held using the REMAP framework to support goals of care discussion. His family ultimately decided to transition to comfort cares and the patient died. Discussion: Unfortunately, such cases are not unique in our current global pandemic. This case illustrates an important and often overlooked approach to goals of care conversations. Clinicians may rush into making recommendations without including steps like exploring a patient's own values and care goals and aligning with these. The REMAP framework (Reframe, Expect emotion, Map out patient values, Align with values, and Propose a plan) is a useful tool both for exploring goals of care and when

	transitions in care are anticipated. It provides a template for conducting effective goals of care conversations that promote shared decision making with patients and their families. 1. Suresh, E., et al., Acute pancreatitis with pseudocyst formation in a patient with polyarteritis nodosa. J Rheumatol, 2005. 32(2): p. 386-8. 2. Flaherty J, Bradley EL 3rd. Acute pancreatitis as a complication of polyarteritis nodosa. Int J Pancreatol. 1999 Feb;25(1):53-7.
Caroline Matchett Dr. Lillian Wang Dr. Nikita Rafie Dr. Laura Raffals	*A Case of Spontaneous Bacterial Peritonitis due to Lactobacillus rhamnosus in Cirrhosis* Introduction: Lactobacilli are ubiquitous commensals of the normal human flora and are rarely found in clinical infections. We present a rare case of a cirrhotic patient with Lactobacillus as the sole pathogen in spontaneous bacterial peritonitis. Case Presentation: A 53-year-old male with alcoholic cirrhosis presented to the emergency department with abdominal pain. CT scan of the abdomen and pelvis showed new ascites. On admission, he had normal vital signs. His abdomen was distended with a positive fluid wave without peritoneal signs. Laboratory results revealed a leukocytosis 16,000 cells/mL, mild worsening of liver function tests, and a creatinine 0.96 mg/DL. His Model of End-Stage Liver Disease (MELD-Na) was 27 on presentation. He was started on empiric ceftriaxone for possible spontaneous bacterial peritonitis (SBP). Paracentesis revealed cloudy peritoneal fluid with 29,105 white blood cells with 84% neutrophils. Given significant neutrophils, there was concern for secondary bacterial peritonitis. Repeat CT scan of the abdomen and pelvis demonstrated mild enhancement of his peritoneal lining, but no evidence of gastrointestinal perforation. Peritoneal fluid and blood cultures grew Lactobacillus rhamnosus as the sole pathogen isolate. The patient progressively worsened despite antibiotic therapy, prompting early transition to Zosyn for broader Lactobacillus coverage. Subsequent serial paracenteses showed marked improvement in his nucleated cell count, and his peripheral leukocytosis resolved. However, the patient's creatinine continued to rise despite albumin administration. He ultimately developed Type 2 Hepatorenal Syndrome. He remains hospitalized with multiorgan failure. Discussion: Spontaneous Lactobacillus peritonitis in cirrhotic patients is rare. Our case illustrates the importance of early microbiologic studies and treatment of atypical pathogens in patients presenting with spontaneous bacterial peritonitis and cirrhosis.
Alesandra Mendoza	*When Opportunity Arises: A Complicated Diagnosis of AIDS-Defining Illness* Introduction: The incidence of AIDS-related opportunistic infections in the United States has decreased significantly with the use of antiretroviral therapy (ART). In the absence of ART and appropriate prophylactic coverage for opportunistic infections, patients with HIV infection can present with life-threatening illnesses. Case Presentation: A 42-year-old male from Guatemala with a 15-year history of untreated HIV presented for several days of fatigue and confusion found to be febrile and encephalopathic. Brain MRI revealed extensive rim-enhancing lesions with vasogenic edema. Chest CT showed innumerable scattered thin-walled pulmonary cavities bilaterally. Absolute CD4 count was 59/mm3 and HIV-1 viral load was 159,400 copies/ml. The differential for these brain and

	lung lesions was extensive with both unifying and separate diagnoses considered. With a history of positive toxoplasmosis IgG, compatible brain imaging findings, and toxoplasmosis being the most common cause of rim-enhancing lesions with a CD4 count <100, CNS/pulmonary toxoplasmosis appeared most likely. Mycobacterial infection, nocardiosis, bacteremia with septic emboli, fungal (i.e. Histoplasma, Cryptococcus, Pneumocystic jiroveci pneumonia (PCP)) or viral infections, lymphoma, or a combination of these were among other investigated diagnoses. He was empirically started on high-dose Bactrim and intravenous vancomycin and ceftriaxone, with the latter two discontinued after negative blood cultures and normal echocardiogram. Unfortunately, lumbar puncture was contraindicated due to edema, but serologies, cultures from sputum and bronchoalveolar lavage (BAL), and molecular tests were obtained. BAL was positive for P. jiroveci and toxoplasmosis. After two weeks on Bactrim, repeat brain MRI showed significant reduction in lesions and edema, while chest CT seemed to demonstrate progression of disease. Rare Candida albicans on sputum culture only was unlikely to be contributing to his lung disease, but nonetheless fluconazole was started for oral thrush. Testing for endemic mycoses, Aspergillus, Mycobacterial infections, and Echinoccocus was unremarkable. In consultation with pulmonary and radiology it was determined that the chest imaging was evolving in conjunction with treatment for presumed PCP, instead of worsening, and the patient remained on Bactrim at discharge after 5 weeks. His cognition slowly improved but he was still not at his baseline. Discussion: In patients with untreated HIV with new illness, it is important to consider possible opportunistic infections or malignancies based on CD4 count. This patient with a CD4 count <100 would particularly be at risk for Toxoplasma and cryptococcal infections, in addition to other infections seen at higher counts. While positive serology and rim-enhancing brain lesions that improve with appropriate therapy confirm the diagnosis of CNS toxoplasmosis, it is notable that toxoplasmosis more rarely causes cavitary pulmonary lesions, demonstrating that multiple opportunistic diseases can present at once. If improvements had not been seen, next steps would include brain biopsy for possible lymphoma or other infectious causes.
William Minteer Dr. R. Ramkissoon Dr. X.J. Wang	*Holy Hematopoiesis!* Case Presentation: A 70-year-old man with severe aortic stenosis recently diagnosed with systemic mastocytosis and chronic monomyelocytic leukemia presented for evaluation of a cholestatic liver injury. He was recently started on hydroxyurea for marked leukocytosis. His liver chemistries were previously within normal limits. On presentation, his alkaline phosphatase (ALP) was elevated to 1147 U/L, alanine aminotransferase to 67 U/L, aspartate aminotransferase to 43 U/L, and total bilirubin to 4.9 mg/dL, predominantly direct. Physical exam was notable for conjunctival icterus and jaundice, without stigmata of cirrhosis or volume overload. Imaging identified hepatosplenomegaly and new, small-volume ascites. Ascites fluid studies demonstrated a serum ascites-albumin gradient (SAAG) >1.1 and total protein of 3.1 g/L. Skeletal survey was recently negative. Liver doppler showed patent vasculature but a decrease in peak portal vein velocity from 32 cm/s to 7 cm/s during a period of two weeks. A magnetic resonance cholangiopancreatography excluded extra-hepatic biliary pathology. Hepatic venous pressure gradient (HVPG) was obtained, which demonstrated a right atrial pressure of 1 mmHg, free hepatic pressure of 5 mm Hg, wedged hepatic venous pressure of 35 mm Hg, equating to a HVPG of 30 (normal <5). Liver biopsy revealed extramedullary hematopoiesis within sinusoids and portal

	tract infiltration by mastocytes, with stage 3-4/4 fibrosis. Esophagogastroduodenoscopy (EGD) revealed small esophageal varices (<5mm) for which daily nadolol was initiated. Patient was started on cladribine with significant improvement in liver function tests within weeks. Discussion: While this case presents a rare entity of systemic mastocytosis with hepatic involvement and sequelae of non-cirrhotic hepatic fibrosis and portal hypertension, it highlights the importance of a systematic framework for common problems facing the general internist: cholestatic injury pattern and ascitic fluid analysis. Differentiating between extra- and intra-hepatic cholestasis in a patient with active hematologic malignancy, while considering other confounding features including risk factors for elevated bone ALP, drug induced liver injury, infiltrative liver disease, heart failure, and viral hepatitis, added to the complexity of this case. The high SAAG and protein ascites required evaluation of post-sinusoidal causes of portal hypertension. Hepatic infiltration and post-sinusoidal portal hypertension secondary to systemic mastocytosis is a rare phenomenon that was first described 1978. The pathophysiology is thought to be related to infiltration of inflammatory mast cells within the portal vein and hepatic sinusoid compromise. Hepatic mast cell infiltration is a feature of aggressive disease and confers a poor prognosis. Lastly, this case highlights the importance of EGD to screen for esophageal varices in portal hypertension regardless of cirrhosis state.
Mitra Moazzami Dr. Jennifer Clark Dr. Rashi Sandooja Dr. Diana Dean	*A Rare Cause of Dyspnea and Chylous Effusion in a Man with Enlarging Neck Mass* Introduction: Mantle cell lymphoma is a non-Hodgkin's lymphoma that has a varied presentation with the potential to affect any organ. In the thyroid, primary thyroid lymphoma (PTL) has an indolent presentation requiring a high degree of suspicion. Hashimoto's thyroiditis has been shown to increase the risk of PTL by 60 times the general population. Case Presentation: We present the case of a 70-year-old man with a history of coronary artery disease, hypothyroidism on levothyroxine for 10 years, type-2 diabetes, and prostate cancer status-post distant brachytherapy who presented for evaluation of a multi-nodular goiter. He had noticed enlarged appearance of the lower neck with palpable mass about 5 months prior. Associated symptoms included progressive dyspnea with cough, intermittent hoarseness, and unilateral non-pitting edema of the left face and upper extremity. Upon evaluation with his local primary care clinician, computed tomography (CT) imaging revealed marked thyroid enlargement, left greater than right, with some airway compression but no compromise. He was also found to have a pleural effusion which was chylous and recurrent despite multiple pleurocenteses. Multiple fine needle aspiration (FNA) biopsies of the thyroid were performed and found to be benign. Upon evaluation at our center, ultrasound thyroid notable for marked hypoechogenicity with scattered calcifications and no discrete nodules. Left-sided adenopathy was noted, most suspicious at the left supraclavicular area. Core biopsy was performed and found to be positive for mantle cell lymphoma (CD5+, CD20+, cyclin D1+, with Ki-67 of 60%). Although lymphangiogram was not performed in light of this diagnosis, it was thought likely that the enlarged thyroid was obstructing the thoracic duct at the level of the mediastinum, causing the chylous effusion. The patient was initiated on chemotherapy with bendamustine, rituximab, venetoclax, with reduction in thyroid size and resolution of recurrent pleural effusion over the subsequent weeks.

	Discussion: PTL is a rare malignancy accounting for less than 5% of all thyroid malignancies. Mantle cell lymphoma can present in the thyroid as an extra-nodal disease in only about 1% of cases. Furthermore, in cases of PTL presenting as an enlarging neck mass, only 1/3 of patients experience actual compressive symptoms of dysphagia, stridor and hoarseness. A high index of suspicion is needed to diagnose PTL, and core needle biopsies (rather than FNA) or excisional biopsy of an affected lymph node are needed to reliably diagnose lymphoma. Concern for PTL is raised in the setting of marked hypoechogenicity and rapid enlargement of the thyroid gland. Once diagnosed, chemotherapy is the treatment of choice for PTL.
Danny Mohama Dr. Antenneh Zewde	*Acquired Acrodermatitis Enteropathica and Other Signs and Symptoms of Nutritional Deficiency Following Bariatric Surgery* Introduction: Micronutrient deficiencies are common following bariatric surgery. These often include water-soluble vitamin, trace metal, and Vitamin D insufficiencies. Signs and symptoms of these deficiencies are numerous. However, a skin examination is an underutilized and important tool toward their identification. In the developed world, it is rare for these nutritional deficiencies to be present in severe and longstanding ways to cause dermatologic changes. Bariatric surgery patients remain at risk with restrictive and malabsorptive states, especially with difficulty adhering to proper nutritional supplementation. This case exhibits skin changes associated with multiple micronutrient deficiencies – most notably acquired acrodermatitis enteropathica associated with zinc deficiency. Case Presentation: TM is a 45 year old woman with medical history of severe malnutrition, pancreatitis s/p total pancreatectomy and islet cell transplantation, and Roux-en-Y gastric bypass who presented with 3 weeks of progressive generalized weakness, distal neuropathy, and ongoing, recently progressive full-body, pruritic and painful, scaly and erythematous rash. The rash was distributed principally over her abdomen, hands and feet, and spared her face. She was admitted 1.5 months prior with generalized weakness, found to have severe malnutrition complicated by sensorimotor axonal peripheral neuropathy and was started on naso-jejunal (NJ) tube feeds, which unfortunately failed shortly after discharge. Over the prior 9 months she had lost 19.5% of her weight. Labs were notable for low copper, zinc, and vitamin A levels. She was given copper gluconate IV 2 g daily, restarted on regular zinc and Vitamin A supplementation, and given regular NJ and subsequent percutaneous gastrostomy feeding. The rash improved in appearance, with reduction in its erythematous extension over her abdomen and healing scale in her lower extremities. Discussion: Diagnosis of zinc deficiency is clinical, though aided by measuring levels of plasma zinc and its transporters – alkaline phosphatase and albumin. Dermatologic manifestations present in moderate to severe deficiency, often as a psoriasifiorm dermatitis involving the hands, feet, and knees, as was observed here. Skin manifestations respond rapidly to treatment, often several days to weeks following supplementation at 1-3 mg/kg/day. Of note, vitamin A and copper deficiencies were also observed here, which likely affected skin integrity. Her distal neuropathy was thought to have been connected with her copper deficiency and poor night vision with her Vitamin A deficiency. This case demonstrates the importance of measuring and supplementing vitamins and trace minerals in patients with malabsorptive or restrictive gastrointestinal states, especially those with a history of bariatric surgery. Zinc plays catalytic, structural, and regulatory

	roles in human growth and development. The prevalence of zinc deficiency in the United States is between 1-3% and up to 20% worldwide. Deficiency is less common in developed countries due to its prevalence in dairy products, green leafy vegetables, and whole grains.
Jwan Naser Dr. Nandan Anavekar	*Left Ventricle Wall Rupture: Protected from Tamponade?* Case Presentation: A 86-year-old man presented with sharp central chest pain radiating up his neck associated with diaphoresis and dyspnea. He had a remote history of coronary artery bypass graft, atrial fibrillation and sinus node dysfunction requiring dual chamber pacemaker. On physical exam, there was no evidence of abnormal heart sounds or volume overload. ECG showed paced rhythm. Troponins were elevated and flat. He was admitted to the cardiac intensive care unit. Given improvement of chest pain and hemodynamic stability, an ischemia-guided approach was considered. Transthoracic echocardiography (TTE) showed ejection fraction of 45%, regional wall motion abnormalities mainly affecting the inferoseptal, anteroseptal and anterior walls, and a loculated pericardial fluid collection adjacent to the anterolateral left ventricular (LV) wall. Contrast TTE showed extension of the contrast agent from the main LV cavity to the adjacent pericardial fluid cavity, consistent with contained rupture of the LV wall or psuedoaneurysm. Cardiovascular surgery recommended computed tomography angiography of the thoracic aorta and coronary arteries which confirmed LV myocardial rupture along the anterolateral wall with active extravasation of contrast. Unfortunately, the patient's hospital stay was complicated by acute kidney injury. In the setting of prior cardiac surgery and increased risk for dialysis, the patient and his family opted for a conservative approach in management. The patient remained hemodynamically stable and therefore pericardiocentesis was not indicated. Pre-dismissal TTE one week later re-demonstrated contained LV anterolateral wall rupture with systolic and end-diastolic flow through the myocardial defect and showed an ejection fraction of 30%. Discussion: LV pseudoaneurysms develop when LV free wall rupture is contained by adherent pericardium or scare tissue and is most commonly secondary to myocardial infarction(1). Other causes include cardiac surgery, trauma, and infection. Most common presentations include dyspnea, chest pain, congestive heart failure although it can also present with sudden cardiac death. Frequently, the diagnosis is difficult due to the nonspecific nature of symptoms(1). In one case series, the mean interval between myocardial infarction and diagnosis of pseudoaneurysm was 7 months and ranged between 1-11 months(2). Patients are at risk of complete rupture, hemopericardium and tamponade. Mortality rate is high both with surgically (23%) and medically (48%) managed patients. Our patient's prior cardiac surgery was thought to provide him with protection against complete rupture due to scar tissue formation along the pericardium. 1. Frances C, Romero A, and Grady D. Left ventricular pseudoaneurysm. J Am Coll Cardiol 1998;32(3):557-61. 2. Eren E, Bozbuga N, Toker ME, Keles C, Rabus MB, Yildirim O et al. Surgical treatment of post-infarction left ventricular pseudoaneurysm: a two-decade experience. Tex Heart Inst J 2007;34(1):47-51.
Neela Nataraj Dr. Gretchen Colbenson	*Transaminitis, Fever, and Joint pain: An Unusual Presentation of Sarcoidosis* Introduction: Sarcoidosis is a granulomatous disease affecting multiple organ

	systems, frequently presenting with hilar adenopathy, pulmonary infiltrates, and dermatologic manifestations. Less commonly, sarcoidosis can involve other organ systems, including ocular, cardiac, and hepatobiliary. Evaluation involves a comprehensive work-up to eliminate alternate diagnoses, while also gathering data to support sarcoidosis. Case Presentation: A 53-year-old male with rheumatoid arthritis on methotrexate and adalimumab, COPD, OSA, and non-alcoholic fatty liver disease, presented with a 3-month history of dyspnea, cough, acute arthralgias, and intermittent fevers. Prior to presentation, he sought care in the outpatient setting and was treated with multiple courses of antibiotics for his fever of unknown origin without improvement. Due to recurrent fevers to 104F and worsening dyspnea, he re-presented for evaluation, and was noted to have a new transaminitis, right axillary adenopathy, and asymptomatic hypercalcemia to 10.7 mg/dL. A Chest CT was performed, which revealed numerous enlarged axillary lymph nodes, diffuse parenchymal nodularity, fibrosis, and traction bronchiectasis, consistent with an infectious or inflammatory etiology. Infectious disease was consulted, and the patient underwent another broad infectious work-up, along with a bronchoscopy with bronchoalveolar lavage. This was unrevealing for a microbial source, gross airway abnormalities, or evidence of malignancy. Transthoracic echocardiogram showed no systolic or diastolic dysfunction. A work-up of hypercalcemia revealed an appropriately suppressed PTH, low 1,25-vitamin-D, normal ACE, and normal PTHrP. The hepatobiliary team was consulted to assist in the workup of his elevated alkaline phosphatase (peak 1025 U/L) and transaminitis (AST and ALT 100s). An MRCP revealed hepatosplenomegaly with mild fibrosis but was otherwise unremarkable. Axillary lymph node biopsy ultimately revealed granulomatous disease, suggestive of sarcoidosis. With the constellation of symptoms of adenopathy and polyarthritis, a subtype of sarcoidosis, Lofgren's syndrome, was considered. He didn't have skin findings consistent with erythema nodosum, the third characteristic sign of Lofgren's syndrome, but given the negative infectious workup and granulomatous findings on biopsy, he was diagnosed with sarcoidosis with hepatobiliary involvement. He was initiated on a steroid taper, with improvements in symptoms, normalization in liver enzymes and calcium, and resolution of fevers. Discussion: Sarcoidosis is a diagnosis of exclusion, as demonstrated by this case. It is imperative to initiate a broad infectious workup, rule-out other causes for granulomatous disease, and to screen patients for cardiac and ophthalmologic involvement due to risk of fatal or permanent impairments. Lofgren syndrome is a rheumatologic manifestation of sarcoidosis that presents with acute arthritis, bilateral hilar lymphadenopathy, and erythema nodosum. Presence of all three findings is 95% specific for Lofgren syndrome, and confirmatory biopsy is often not required for diagnosis of sarcoidosis. Though our patient did not have findings of erythema nodosum, his other presenting findings were consistent and not explained by an alternative cause after exhaustive workup.
Nathan Nelson Dr. Monazza Chaudhry	*A Case Where the Type of Testing Makes the Difference for Legionella* Introduction: Legionella pneumophila is a common cause of community

Dr. James Leatherman	acquired pneumonia (CAP). Legionella pneumophila can be difficult to diagnose because of multiple serogroups and subtypes not detected by the standard urine antigen test. This case describes the hospital course of a patient with CAP due to a legionella subtype with a negative urine antigen test and complicated by Fanconi's syndrome. Case Presentation: A 65-year-old female with a past medical history of multiple myeloma s/p chemotherapy and autologous stem cell transplant on Lenalidomide at admission, who initially presented with chest pain. Her workup revealed a right lower lobe opacity. Her pulmonary workup included negative serologies for histoplasmosis, blastomycosis, coccidioides, aspergillus, cryptococcus, and tuberculosis. She was discharged home on Augmentin, however returned three days later with fevers, non-productive cough, nausea and vomiting. She was septic with hypotension, leukocytosis, and high grade fever. She was started on IV Vancomycin, Cefepime, and Azithromycin. She had blood cultures drawn and a urinary Legionella antigen test which was negative. Urine analysis also showed microscopic hematuria. ID and pulmonology were consulted. She had persistent fevers, and began requiring low levels of supplemental oxygen, so a bronchoscopy was performed with AFB, fungal, and bacterial cultures and pneumocystis DFA, all of which would return negative. Legionella PCR was also obtained. Azithromycin was discontinued after three doses and coverage was broadened to Vancomycin, Meropenem and Voriconazole. Repeat CT of the chest showed worsening lower lobe opacities. The patient's course was further complicated by hypokalemia, non-nephrotic range proteinuria, and severe hypophosphatemia meeting criteria for Fanconi's syndrome. After multiple days the Legionella PCR came back positive. She was started on Azithromycin for seven days and able to discharge home on oxygen. Follow up chest CT showed marked improvement of the right lower lobe opacity and she was on room air. Her hypophosphatemia and proteinuria also resolved and was suspected to be from her infection. Discussion: Legionella pneumophila has several subtypes, and serotype group 1 consists of about 80% of Legionella infections and is identified by the urinary antigen test. Serotype groups 2-15 are less common causes of Legionella and have poor sensitivity with only urine testing. Bronchoscopy testing with Legionella PCR is far more sensitive at identifying serotypes 2-15 and other subtypes, and should be performed when a diagnosis is uncertain. Lastly, Fanconi's syndrome is a rare complication, but has been documented in cases of Legionella.
Philippe Nyembo Dr. Joseph Steffens Dr. Rehan Karim	*An Ignored Cause of Presyncope and Syncope: A Case of "Tri-Fascicular Block"* Introduction: A tri-fascicular block is a disorder characterized by abnormal intraventricular conduction diagnosed on surface EKG. In clinical practice, the condition is defined by the presence of varying combinations of EKG abnormalities suggesting a disease process involving all three conduction fascicles - Right Bundle Branch Block (RBBB), Left Anterior Fascicular Block (LAFB), and Left Posterior Fascicular Block (LPFB). In addition to that, a combination of a bi-fascicular block (either RBBB plus LAFB or RBBB plus LPFB) along with prolonged PR interval can represent disease involving the third fascicle and is also sometimes referred to as "Tri-fascicular block." The condition could progress to intermittent complete AV block, often presenting with lightheadedness and syncopal episodes, as seen in the following case.

	Case Presentation: A 68-year-old man with a history of schizoaffective disorder was evaluated in the clinic for recurrent lightheadedness and pre-syncopal episodes with transient bradycardia for several months. EKG in the clinic showed RBBB with LAFB in addition to prolonged PR interval ("Tri-fascicular block") and felt stable from previous. The patient was discharged from the clinic with a plan for outpatient workup. Given EKG abnormalities with reported symptoms, the patient was called back for close observation and further workup per cardiology recommendation. The patient was admitted with ongoing cardiac telemetry. Transthoracic echocardiogram did not reveal any significant structural abnormalities to explain clinical symptoms. On the second day, the telemetry revealed a 37 seconds episode of ventricular asystole due to a complete AV block, followed by a ventricular escape rhythm at approximately 30 beats per minute, later followed by the resumption of 1:1 AV conduction. The event was associated with a transient loss of consciousness, and an urgent temporary trans-venous pacemaker was placed. The patient subsequently received a dual-chamber permanent pacemaker. The patient was seen six weeks after discharge with no recurrence of lightheadedness or syncope. Discuss: Our case highlights the importance of recognizing "Tri-fascicular block" on EKG in patients presenting for evaluation of syncope and presyncope. Although the mere presence of "Tri-fascicular block" on EKG without symptoms does not predict a high rate of progression to complete AV block, it is essential to note that when symptoms are associated with this EKG abnormality, there is an increased likelihood of progression to complete AV block, which could be intermittent. In one study by Marti-Almor et al., syncope was a predictor of progression to complete AV block in patients with tri-fascicular block. Management of patients presenting with syncope and tri-fascicular block involves clinical and electrophysiological evaluation of AV conduction system and consideration of a permanent pacemaker.
Kevin O'Donnell Dr. Peter Lund	*Heartbreaker: A Case of AML Relapse Presenting as Myocardial Infarction* Introduction: Acute chest pain in young healthy individuals is unlikely to be due to cardiac ischemia. Nevertheless, clinicians must consider this pathology in the differential. Prompt diagnosis of ischemia can expedite treatment and avoid further sequalae. Case Presentation: A 21-year-old male with history of NPM1 mutated high risk AML in remission after chemotherapy who presented with three days of episodic chest pain radiating to left arm. He had been evaluated at a virtual visit and diagnosed with chest wall pain when symptoms first started. Chest pain occurred again with new symptom of diaphoresis prompting call to EMS. Patient given ASA and nitroglycerin with improvement in pain. Two EKGs en route were concerning for STEMI. Chest pain continued in the ED. CT PE study was negative for acute findings. Taken to cath lab emergently when initial troponin I returned at 0.737(ref. <0.034). Subsequent troponins peaked at 360.8. Found to have total occlusion of proximal LAD due to clot. Treated with thrombectomy, angioplasty and placement of a drug-eluting stent in the proximal LAD. Ejection fraction was estimated at 25% during angiogram with akinesis involving the multiple wall segments. Initial lab studies were also notable for a Hgb 9.9, MCV 100, Plt 239, and WBC of 8.8 with a differential showing 52% blasts. Fibrinogen 365, D-dimer >4, INR 1.3, prothrombin 15.6 and homocysteine 9.6. Thrombophilia and

	COVID-19 testing were negative. Hematology consulted and a bone marrow biopsy found a hypercellular bone marrow with 85% blasts. Treated with induction chemotherapy. EF remained 35-40% on subsequent imaging studies with development of left ventricular thrombus. After discharge, patient later underwent allogenic bone marrow transplantation from sibling with 6 month follow-up bone marrow showing no evidence of disease. Conclusion: This case represents a rare presentation of acute myocardial infarction due to AML relapse in a young individual. A 2012 review found that only 12 of 774 (1.6%) cases of AML presented with myocardial infarction at time of diagnosis. Hyperleukocytosis causing leukostasis is proposed as one mechanism, however this was unlikely to be present in our case due to WBC on admission of 8.8. Other proposed etiologies in the literature include effects of prior chemotherapy, disseminated intravascular coagulation, hyperhomocysteinemia and deficiency of coagulation factors. References: 1. Lukenbill, Joshua, et al. "Impact of Myocardial Infarction On Survival in Acute Myeloid Leukemia." Blood 120.21 (2012): 4321. 2. Chang, Hung, et al. "Acute myeloid leukemia associated with acute myocardial infarction and dural sinus thrombosis: the possible role of leukemia-related hyperhomocysteinemia." Journal of the Chinese Medical Association 71.8 (2008): 416-420. 3. Jachmann-Jahn, U., et al. "Acute anterior myocardial infarction as first manifestation of acute myeloid leukemia." Annals of hematology 80.11 (2001): 677-681.
Jessica Padniewski	*Nettling Nodules; A Case of Kaposi's Sarcoma- Associated Immune Reconstitution Inflammatory Syndrome (KS-IRIS)* Introduction: The widespread use of highly active anti-retroviral therapy (HAART) in patients with HIV infection has led to increased quality of life and prolonged survival in patients with HIV infection (Stekler). However, initiation of HAART may result in reactivation of indolent infections known as immune reconstitution inflammatory syndrome (IRIS) which is a well reported phenomenon and carries a high mortality rate (Novak). HIV related Kaposi's sarcoma (KS) is associated with human herpes virus 8 (HHV-8) and remains the most common tumor in patients with HIV. KS may be reactivated as an IRIS-like syndrome that can also result in rapid clinical progression of KS on initiation of HAART (Connick, Bower). Herein we report a case of IRIS-like syndrome in a patient initiating HAART therapy. Case Presentation: A 29-year-old African-American, homosexual, male with a past medical history of HIV not on HAART therapy, presented to infectious disease clinic to establish care. Labs revealed a CD4 count of 9 and HIV RNA 375,000 and he was promptly initiated on Biktarvay. He was noted to have diffuse papulonodular lesions and an urgent referral to dermatology was placed for evaluation. One week later, the patient presented to dermatology clinic and reported that he noticed the first papule on his arm a couple months ago and since that time developed diffuse papulonodular lesions to the face (Figure 1), trunk and extremities. The patient consented to skin biopsies of the lesions, but unfortunately, they were compromised, and he was asked to return the following week. Two weeks after initiation of HAART, he returned to dermatology clinic and was noted to have diffuse facial swelling primarily involving the periorbital and submandibular region (Figure 2). Repeat biopsies were obtained in clinic and the patient was admitted for expedited work up and close monitoring of new onset facial edema. While awaiting

	biopsy results, he was started on vancomycin and Zosyn empirically. Anti-fungal treatment was deferred. On ROS, he denied any dysphagia, odynophagia or shortness of breath. He reported abdominal pain associated with eating, early satiety and a 20 lbs weight loss over the preceding 4 months. He reported recent travel throughout US, Jamaica, Costa Rica, Puerto Rico, and Italy. He denied any other relevant exposures or risk factors. Initial laboratory survey was notable for HGB 7.8 (N 13.1-17.5), elevated alkaline phosphatase 234 (N 40-129) with BMP, CBC and LFTs otherwise unremarkable. Bacterial and fungal blood cultures, Cryptococcal antigen, urine histoplasma and sputum AFB were all negative. CMV IGG was positive. Bronchoscopy was performed and returned negative for pneumocystis with no growth otherwise. An inguinal lymph node biopsy was performed without immunophenotypic evidence of non-Hodgkin lymphoma. Imaging studies including CT neck revealed subcutaneous soft tissue nodules throughout the head, face, and neck with prominent cervical lymph nodes and tonsillar hypertrophy concerning for disseminated fungal infection or IRIS. CT chest, abdomen and pelvis also noted diffuse skin and subcutaneous nodules particularly in the abdomen and pelvis as well as multiple enhancing nodules in the lungs, pleural space and thoracic paraspinal area with extensive mesenteric and retroperitoneal adenopathy. Skin biopsy returned positive for HHV8 and CD34 on immunohistology stains confirming a diagnosis of Kaposi sarcoma. CD68 was negative ruling out a granulomatous inflammation. Acid fast, GMS and PAS special stains were negative ruling out disseminated mycobacterial and fungal infection respectively. Conclusion: Commonly implicated causes of IRIS including mycobacterial, cryptococcus, pneumocystis, cytomegalovirus, herpes, hepatitis B and C were ruled out further supporting the diagnosis of IRIS-like syndrome in our patient. It is postulated that IRIS occurs in >10% of patients with KS, tuberculosis, or cryptococcus infection and visceral involvement leads to considerable morbidity and mortality (Achenbach). We present this case to increase awareness of IRIS-like syndrome and stress the importance of maintaining close clinical supervision in patients with KS while initiating, changing or resuming HAART (Leidner).
Jessica Park	*Small Bowel Obstruction Caused by Carcinoid Tumor* Conclusion: As cancer therapies advance to personalize treatment and target specific immune responses, it is important for providers to be aware of the potential adverse effects such as the potential for increased production of PTHrP leading to hypercalcemia.
Nisha Patel Dr. Angela Grassi Dr. Mariana Restrepo-Holguin Dr. Sunnia Chen Dr. Laura Maciejko	*Acalculous cholecystitis and splenic infarction: An unusual presentation of acute Q fever infection* Introduction: Q fever is a rare zoonotic infection caused by Coxiella burnetii with a diverse range of presenting symptoms and clinical manifestations, including flu-like symptoms, atypical pneumonia, and hepatitis. In this abstract, we describe an atypical presentation of Q fever that presented with acalculous cholecystitis and splenic thrombosis in the setting of positive antiphospholipid antibodies.

	Case Description: A 47-year-old male sanitation worker presented with a two-week history of fevers, right upper quadrant pain, and jaundice. Labs were significant for elevated liver enzymes in a hepatocellular pattern and thrombocytopenia. Imaging revealed sludge in the gallbladder with mild wall thickening, diffuse hepatic steatosis, and multiple wedge-shaped hypoenhancing areas in the spleen suggestive of infarcts. The patient was admitted due to concern for acute cholangitis and incidentally found splenic infarcts. During hospitalization, MRCP showed no biliary ductal dilation or filling defects, which ruled out acute cholangitis, and TEE was negative for evidence of infectious endocarditis. The patient continued to have elevated liver enzymes and persistent fevers despite antibiotic coverage with piperacillin-tazobactam. Additional workup was significant for elevated antiphospholipid antibodies in the setting of splenic infarcts. With no clear explanation for the patient's presentation, the differential diagnosis was broadened to include a variety of atypical infectious causes of acute hepatitis. Liver biopsy subsequently revealed non-necrotizing lobular and fibrin granulomas. Due to suspicion for Q fever infection, the patient was empirically started on doxycycline with subsequent clinical improvement. Coxiella burnetii titers and PCR resulted positive in a manner consistent with acute Q fever infection, and the patient was started on hydroxychloroquine as well. His hospitalization was also complicated by upper GI hemorrhage secondary to anticoagulation in the setting of positive antiphospholipid antibodies. EGD was performed, revealing four duodenal ulcers and an actively bleeding pre-pyloric ulcer, which was clipped. Discussion: This case highlights the importance of considering a broad differential diagnosis, including Q fever, in patients with signs of hepatobiliary disease. Q fever has a variable presentation and can often resemble other infections or autoimmune diseases. While hepatitis is commonly associated with Q fever, acalculous cholecystitis is a very rare presentation. In addition, although transient antiphospholipid antibodies are a known phenomenon of Q fever, they are rarely associated with independent thrombotic complications in patients without prior diagnosis of antiphospholipid antibody syndrome or evidence of underlying Q fever endocarditis. However, our patient suffered an extensive thrombotic event necessitating anticoagulation, despite relatively low level positivity of anticardiolipin IgG (60.9). Our findings suggest that patients with Q fever should be routinely tested for antiphospholipid antibodies, with further workup for thrombosis and consideration of anticoagulation as needed. Furthermore, due to the significant morbidity Q fever patients may suffer secondary to thrombosis, close follow-up with vascular medicine specialists is warranted.
Marissa Paulson Dr. Abdulsabur Sanni	*Initial Concern for One in a Million Vaccine Side Effect leads to Diagnosis of Acute Promyelocytic Anemia* Introduction: When this 41 year old patient began developing bruising around her abdomen and her typical migraines increased in severity, her initial concerns were not for her eventual diagnosis of cancer, but of an exceedingly rare diagnosis that was being splashed across media at the time- thrombotic thrombocytopenia. This patient had received the Johnson and Johnson (J & J) Ad26.COV2.S vaccine against severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) two months prior to symptom onset. She presented to care due to concerns that she was displaying the symptoms of thrombotic thrombocytopenia, a condition seen in less than 1 in 1,000,000 vaccine

	recipients but was eventually diagnosed with Acute Promyelocytic Leukemia (APML). Case presentation: This 41-year-old woman presented to clinic complaining of fatigue, headaches, dyspnea with exertion, vaginal bleeding, and bruising over her abdomen and bilateral lower extremities. She had received the J & J vaccine in March 2021, two months before presentation. She had recently gotten a MR Venogram for worsening migraine headaches which was unremarkable. Her initial workup showed a hemoglobin of 6.8, platelets of 13, WBC of 6.3, fibrinogen of 83, and elevated PT/INR consistent with DIC. A peripheral blood smear showed blasts and she underwent a bone marrow biopsy and was found to have 15:17 translocation on FISH consistent with APML. She started chemotherapy with ATRA and arsenic trioxide. Throughout her admission, her WBC increased rapidly and this coupled with ongoing weight gain raised concern for differentiation syndrome. This was treated with hydroxyurea and dexamethasone. Discussion: Thrombotic thrombocytopenia is a diagnosis of thrombosis, commonly cerebral venous sinus thrombosis (CVST), associated with severe thrombocytopenia and DIC. This diagnosis has been noted as a rare side effect of the J&J vaccination that can occur 7-14 days after vaccination. During the clinical trial program for this vaccine a single case of CVST occurred and during ongoing surveillance, six additional cases were observed. This led to a pause in vaccination. After study, it was shown that that CVST cases documented in Johnson and Johnson vaccine recipients were occurring within the range of published background incidence and there was insufficient evidence to establish a causal relationship between the vaccine and a diagnosis of CVST. The media coverage of this rare side effect did help educate the public of symptoms to be aware of that could signal a rare and potentially devastating side effect. Despite the rare incidence of thrombotic thrombocytopenia and CVST, it is an important diagnosis to consider in the right clinical context but should not cause providers to anchor and a broad differential diagnosis still needs to be considered.
Leslie Pensa Dr. Daniel Mueller Dr. William Valente Dr. Taylor Hand	*Scurvy, Not Just a Diagnosis on Boards* Introduction: Vitamin C (ascorbic acid) is essential for life as it is an important reducing agent for many biochemical processes. Even relatively short term deficiencies of vitamin C can lead to the development of scurvy. Scurvy is best known for the characteristic skin changes, gingival bleeding, and impaired wound healing. However, with profound vitamin C deficiency, anemia is also an important finding. Case Presentation: A 56 year old male with history of spontaneous coronary artery dissection and coronary artery disease (on dual antiplatelet therapy), left ventricular apical thrombus (no longer on anticoagulation), hypertension, & tobacco use was admitted to an outside hospital after near syncopal episode. Syncope workup was unrevealing but incidental findings of new acute anemia (hemoglobin of 9.6 g/dL) and bilateral lower extremity edema with overlying ecchymosis (right greater than left) were appreciated. The patient endorsed being struck in the right thigh by a softball roughly two weeks prior but denied any other traumas. Lower extremity ultrasound was negative for deep vein thrombosis and iron panel was normal. Decision was made to stop Clopidogrel and discharge home with close outpatient follow up.

	Hemoglobin on post-hospital follow up 5 days later was 5.7 g/dL, prompting re-admission to the outside hospital. Despite repeated blood transfusions (total of 20 units), the anemia persisted. Extensive imaging to look for indolent bleed including endoscopy, colonoscopy, tagged red blood cell study, CT angiography of abdomen/pelvis/lower extremities, CT chest/abdomen/pelvis, MRI right femur, and repeat lower extremity ultrasound with doppler was unrevealing. Labs including infectious studies, hemolysis labs, peripheral smear, and bone marrow biopsy were also inconclusive. He was subsequently transferred to our institution for further workup/management of refractory anemia. Upon transfer, Rheumatology was consulted for comprehensive evaluation. During our initial exam, corkscrew hairs, perifollicular hemorrhages, and purpura were appreciated, raising suspicion for scurvy and prompting us to obtain an in-depth dietary history. He described a diet completely devoid of fruits or vegetables and reported eating only one meal per day prior to hospitalization, which was frozen and processed. Otherwise, he reported snacking almost exclusively on chips. A vitamin C level was checked and returned undetectably low. Dermatology and Hematology/Oncology were also consulted and agreed that scurvy was the most likely diagnosis. He was treated with intravenous and oral ascorbic acid with significant improvement in cutaneous findings and no further need for blood transfusions. Conclusion: This case highlights the importance of a thorough physical exam and history in reaching a diagnosis. Albeit rare, scurvy does still occur in modern times and should be considered in patients with poor diet and characteristic exam findings. This case also demonstrates the lesser appreciated finding of anemia, which is also characteristic of scurvy.
Angela Phillips Dr. Breanna Zarmbinski	*Does Stool Really Matter? An Atypical Presentation of Life-Threatening Fulminant Clostridium Difficile Infection* Introduction: Clostridium Difficile is a leading cause of health care associated infections in the United States. It carries significant risk of mortality leading to 15,000 - 20,000 deaths per year with an estimated mortality rate of 67% in patients with multisystem organ failure [1,2,3]. There is a well-defined association between antibiotic use and the incidence of Clostridium difficile infections (CDI), stressing the ongoing importance of antibiotic stewardship specifically in high-risk geriatric populations [1, 4]. Case Presentation: A 64-year-old male with a past medical history of type II diabetes, congenital aortic stenosis status post replacement, COPD, alcohol use disorder, neurogenic bladder and BPH presented to the emergency department with somnolence, confusion and weakness. Review of systems was negative for diarrhea. One month prior, the patient was admitted for sepsis caused by a complicated urinary tract infection related to obstructive uropathy and required ureteral stent placement. On admission, the patient met sepsis criteria with a fever, tachycardia and tachypnea. Labs revealed significant leukocytosis, AKI, metabolic acidosis and borderline hyperkalemia. Lactate was normal. Urine analysis (UA) showed moderate bacteria, negative nitrates and large leukocyte esterase. Exam revealed no abdominal tenderness. Patient was started on Vancomycin and Cefepime for sepsis with suspected urinary source given recent stent placement. Urology was consulted and did not feel that he had a urinary tract infection. Patient decompensated on hospital day one with worsening septic shock. Repeat labs revealed worsening lactate, white blood cell count and acidosis. He was transferred to the ICU and was eventually intubated for respiratory failure. CT

	abdomen and pelvis showed pan colitis after which patient was started on oral vancomycin and metronidazole for suspected CDI. Patient did develop diarrhea following admission and a stool sample returned positive for Clostridium Diffcile. General surgery was consulted and discussed surgical intervention which included total colectomy with end ileostomy for fulminant CDI. After this discussion, it was felt that the patient would not want to continue with aggressive measures, and he was subsequently transitioned to comfort cares and died several hours later. Conclusion: This case illustrates the diagnostic challenges associated with fulminant CDIs specifically in patients who present without diarrhea which can occur in 20% of cases [1]. Health care providers need to have a high level of suspicion for a possible CDI in any patient presenting with acute encephalopathy and sepsis with an unidentified source. Early consultation to general surgery is key as fulminant CDI is an indication for surgery in a patient with multisystem organ failure not responding to medical management [1].
Aaron Pickrel	*A Rare Cause of Spontaneous Pneumothorax* Introduction: Sporadic lymphangioleiomyomatosis (LAM) is a rare multisystem disease that typically affects young women in their 30s to 40s. It should be considered with patients that present with respiratory complaints, including dyspnea on exertion for unknown etiology or recurrent pneumothorax. Case presentation: A healthy 39-year-old female presented with a 5-day history of an upper respiratory infection. She also complained of a long history of dyspnea on exertion. Patient's cough progressed, and she developed acute onset pleuritic chest pain and shortness of breath. Patient presented to the emergency department and a CT chest demonstrated a large, right tension pneumothorax, as well as innumerous thin-walled cysts throughout her lungs bilaterally, consistent with LAM. A chest tube was placed with reinflation of the right lung and improvement of the patient's shortness of breath. Patient had a negative work-up for autoimmune diseases and other cystic lung diseases including negative UPEP, SPEP, free light chains, ANA, RF, Sjogren's, anti-CCP, and alpha-1 antitrypsin. VEGF–D was unable to be completed due to limited testing availability. CT chest/abdomen/pelvis included in the work-up and confirmed a 4.8 cm right renal mass, consistent with renal angiomyolipoma. Patient had persistent air leak of her right pneumothorax throughout the hospitalization. Case was discussed with multiple specialists and video-assisted thoracoscopic surgery with biopsy and mechanical pleurodesis was considered, but ultimately the patient deferred. Pneumothorax stabilized after chest tube remained to suction for 8 days. Chest tube was able to be removed without recurrent pneumothorax and patient discharged home. She has since had her renal angiomyolipoma embolized as an outpatient. Patient's diagnosis of lymphangioleiomyomatosis was a clinical diagnosis made from her presentation, lung imaging, and renal angiomyolipoma. The patient is planning to have pulmonary function tests performed, with consideration of a lung biopsy and initiation of Sirolimus. Discussion: This case demonstrates a common presentation of a rare disease, sporadic lymphangioleiomyomatosis. It is expected that less than 1000 people in the United States have LAM. LAM is a rare multisystem disease

	that typically affects young women in their 30s to 40s. Patients most often present with signs and symptoms of cystic lung disease including pleural effusions, spontaneous pneumothorax, and progressive dyspnea with exertion. Other symptoms can be associated with extrapulmonary sites related to renal angiomyolipoma or lymphatic vasculature including chylothorax. Diagnosis of LAM is typically a clinical diagnosis which includes characteristic thin-walled, well defined, diffusely distributed pulmonary cysts, VEGF–D level>800 pg/ml, chylothorax, and renal angiomyolipoma. Definitive diagnosis is made with lung biopsy via VATS. There is currently no cure for LAM, but mTOR inhibitors like Sirolimus can help slow the progression of the disease.
Sumar Quint Dr. Samuel Ives	*Hold Your P: Renal Phosphorous Wasting, a Rare Side Effect of IV Iron Infusion* Introduction: Iron deficiency anemia (IDA) is a prevalent disease, affecting up to 20% of women worldwide. Treatment with parenteral iron is common due to oral iron side effects and absorption difficulties. An underrecognized but serious side effect of a common IV iron formulation, ferric carboxymaltose (FCM), is hypophosphatemia. This adverse effect is thought to be caused by increased circulating levels of fibroblast growth-factor-23 (FGF-23) which leads to inappropriate renal excretion of phosphorous. Risk factors for developing severe hypophosphatemia after FCM therapy include patients who are chronically malnourished or have malabsorption syndromes. Case Presentation: A 36-year-old female with past medical history of bulimia nervosa and IDA presented to the emergency department for chest pain, she was found to have phosphorous of 1.0 initially thought to be due to malnourishment and refeeding syndrome. She was briefly admitted and given oral replacement. The patient re-presented two days later after labs at a residential eating disorder facility showed continued hypophosphatemia. Symptoms included headache and muscle weakness. In the ED the patient's phosphorous was 1.4, she was admitted for IV repletion and further work up. Parathyroid hormone (PTH), 25-OH Vitamin D level, and ionized calcium were within normal limits. The patient did not have any other persistent electrolyte disturbances. Over the course of one week the patient's phosphorous was continually repleted, with only transient improvements in serum levels. A 24-hour urine phosphorous was found to be high at 1718 mg/day (400-1300 mg/day), suggesting renal wasting of phosphorous. Given no other endocrine or lab abnormalities, prior medications were investigated. The patient had been seen by hematology three weeks prior to admission and given two doses of FCM infusion for IDA. The suspicion of FCM causing phosphorous wasting was confirmed with an inappropriately normal FGF-23 level of 122. The patient was discharged with a PICC line for outpatient infusion of phosphorous, and follow-up with endocrinology. Discussion: FCM infusions are thought to cause hypophosphatemia in susceptible patients by increasing FGF-23 levels by a mechanism that is not fully understood. FGF-23 is a bone-derived hormone that inhibits phosphate transporters in the renal tubules and decreases activation of 1,25-OH Vitamin D, leading to both renal wasting and inability to absorb phosphorous. Risk factors for hypophosphatemia secondary to FCM include malnourishment, hyperparathyroidism, malabsorption syndromes, and vitamin D deficiency. In the case presented the patient had a long history of disordered eating and was likely deficient in total body phosphorous before the FCM infusion. The side effect of severe hypophosphatemia should be better known to clinicians, as

	patients with risk factors for hypophosphatemia are often the most common patients to have IDA. Treatment consists of frequent lab checks and replacement of phosphorous and most cases resolve within 3 months.
Alison Raffman	*Non-Cardiogenic Pulmonary Edema Following Naloxone Use for Heroin Overdose* Introduction: Naloxone is an opioid antagonist medication that is commonly used as a reversal agent in cases of opioid overdose. This medication is generally considered as safe to give for this purpose, however it has been associated with several uncommon complications. Non-cardiogenic pulmonary edema following Naloxone administration has been reported as a rare adverse event associated with use of this medication. Case Presentation: A 32-year-old male with prior history of opioid abuse presented to the emergency department after reported IV heroin overdose approximately one hour prior to arrival. The patient's significant other found the patient unresponsive at home, called EMS and initiated bystander CPR at the scene. She also administered 1.2mg IM Naloxone with subsequent immediate improvement in patient's mental status to his baseline. The patient subsequently developed worsening shortness of breath, was found by medics with oxygen saturations in the 80s, and required oxygen via non-rebreather en route to the hospital. On arrival to the emergency department the patient was alert and oriented, reporting shortness of breath but otherwise without complaints. Significant initial vitals included HR 129, RR 29, oxygen saturation 89%, the patient was otherwise normotensive and afebrile. His exam was notable for coarse breath sounds bilaterally (greater on the left). Bedside cardiac ultrasound did not demonstrate any significant abnormalities, and lung ultrasound demonstrated B-line predominance bilaterally. Chest x-ray demonstrated bilateral infiltrates (moreso on the left). CT chest again showed bilateral infiltrates that were more prominent on the left, consistent with asymmetric pulmonary edema. The patient was monitored in the emergency department and was eventually transitioned from non-rebreather to 3L O2 via nasal cannula, however after four hours of monitoring the patient was trialed on room air and experienced worsening tachypnea with desaturation to 90%. The patient was admitted to medicine for observation, and after several hours the patient was successfully transitioned to room air without significant hypoxia or increased work of breathing, with clear breath sounds noted on subsequent examinations. The patient was able to be discharged home in good condition. Discussion: Non-cardiogenic pulmonary edema has been previously reported as a rare complication of Naloxone administration following opioid overdose. There has been some controversy regarding Naloxone's role in the pathophysiology of pulmonary edema in such cases, however a dose-response relationship between Naloxone and subsequent pulmonary complications has been described previously. Overall, this case highlights a case of respiratory distress associated with non-cardiogenic pulmonary edema following Naloxone administration for opioid overdose, and demonstrates successful management of this condition with observation and supportive care measures.
Nikita Rafie Dr. Caroline Matchett	*Nutritionally Variant Streptococcal Endocarditis: A Case Report* Introduction: Infective endocarditis is an infection of the endocardial surface of the heart, typically involving at least one heart valve or an intracardiac

device. Complications of endocarditis are vast and include cardiac valve destruction, perivalvular extension, systemic embolization, metastatic spread of infection, and immunologic manifestations[1]. Infective endocarditis also carries a high mortality rate. In hospital mortality rate for patients with infective endocarditis is 18-23%, and the six-month mortality rate of patients with infective endocarditis is 22-27% [2-4]. Given the significant complications and high rate of mortality of infective endocarditis, prevention and prompt treatment carry the upmost importance.

Case Description: The patient is a 61-year-old male who presented with 10 days of weakness, myalgias, increasing dyspnea on exertion, and fevers. Relevant medical comorbidities include severe aortic stenosis secondary to bicuspid aortic valve status post aortic valve replacement with a bioprosthetic valve eight years prior. On physical exam, a III/VI systolic ejection murmur was appreciated at the right upper sternal border. There were no peripheral manifestations of endocarditis or embolic phenomenon appreciated on physical exam. A transesophageal echocardiogram showed a 3 mm echodensity on the anterior leaflet of prosthetic valve and thickening of the mitral-aortic intervalvular fibrosa. A CT cardiac angiogram showed a para-aortic phlegmon. Blood cultures grew Granulicatella adiacens, a streptococci species. He initiated intravenous vancomycin and ceftriaxone for presumed endocarditis. The patient then underwent surgical debridement of previously implanted aortic valve and entire root complex and aortic root replacement utilizing a 25-mm On-X composite valved conduit. Extensive purulent material was noted intraoperatively. He continued vancomycin for six weeks then transitioned to oral penicillin for suppression for at least 12 months.

Discussion: This patient's age, sex, and valvular disease with prosthetic valve put him at risk of infective endocarditis. The significant mortality of infective endocarditis makes this diagnosis a medical emergency requiring prompt hospitalization, acquisition of blood cultures, empiric antibiotic therapy, and expedited cardiac and systemic imaging to confirm the diagnosis and evaluate for complications. Granulicatella is a nutritionally variant streptococci, meaning it grows best on media supplemented with thiol or pyridoxal [5]. It is a catalase-negative, oxidase-negative, facultative anaerobe and is normally present in oral flora and rarely manifests as invasive infections [6]. Interestingly, Granulicatella has only been implemented in up to five percent of cases of streptococcal endocarditis [5]. Targeted antimicrobial therapy is essential to ensure elimination of bacteremia and treatment of infective endocarditis.

References:

1.Mansur,A.J.,etal.,TheComplicationsofInfectiveEndocarditis:AReappraisalinthe1980s.ArchivesofInternalMedicine,1992.152(12):p.2428-2432.
2. Hasbun,R.,etal.,ComplicatedLeft-SidedNativeValveEndocarditisinAdultsRiskClassificationforMortality.JAMA, 2003.289(15):p.1933-1940.
3. Hill,E.E.,etal.,InfectiveEndocarditis:changingEpidemiologyandPedictors of 6-month mortality: a prospective cohort study†. European Heart Journal, 2006. 28(2): p. 196-203.
4. Wang, A., et al., Contemporary Clinical Profile and Outcome of Prosthetic Valve Endocarditis. JAMA, 2007. 297(12): p. 1354-1361.
5. Gupta, S., et al., Granulicatella adiacens abscess: Two rare cases and review. Journal of laboratory physicians, 2018. 10(1): p. 121-123.
6. Cargill, J.S., et al., Granulicatella infection: diagnosis and management. Journal of Medical Microbiology, 2012. 61(6): p. 755-761.

Beau Rigstad	*Chest Compressions During CPR as a Cause of Chylothorax* Introduction: Chylothorax can occur with any disruption of the flow of chyle through the thoracic duct. There are many well-described etiologies of chylothorax, with the most common non-traumatic cause being malignancy, and the most common traumatic causes being thoracic surgery, motor vehicle accidents, assaults, or falls. This case describes the development of a chylothorax in a hospitalized patient following chest compressions during a cardiac arrest. Case Presentation: A 60-year-old man with a history of type 2 diabetes mellitus and hypertension was initially admitted for a diabetic foot infection which required a right-sided below the knee amputation. He had a prolonged hospitalization with multiple complications including delirium, intubation for airway protection in the setting of altered mental status, and an in-hospital cardiac arrest. He also had a right-sided chest tube placed for a simple pleural effusion. The patient had a second in-hospital cardiac arrest which resulted in 20 minutes of ACLS and chest compressions. The patient was again intubated and transferred to the ICU. The morning following this cardiac arrest, after enteral feeding had been resumed, the pleural fluid output from the right chest tube was noted to be a milky white color and had previously been serosanguineous in appearance. A pleural fluid sample was collected and the triglyceride level was found to be 599 mg/dL, diagnostic of chylothorax. Subsequent chest imaging revealed fractures of ribs 2-6 on the right side as well as a possible non-displaced sternal fracture. Given his clinical course, the most likely etiology of this new chylothorax was blunt chest trauma from chest compressions following his most recent cardiac arrest. Due to the patient's critical condition the chylothorax was treated conservatively with the chest tube set to suction and a low-fat enteral feeding formula. For the next 5 days, the right sided chest tube put out between 380-1,070cc of milky-appearing fluid. After 5 days of conservative management the pleural fluid returned to a serosanguineous appearance, and the repeat triglyceride level of the fluid on the 6th day was 55 mg/dL. Discussion: Pleural effusions are a very common problem in hospitalized patients that can be due to many different underlying diseases. Thorough differential diagnosis and evaluation is important as management and prognosis of pleural effusions are largely dependent on their cause. This case highlights the importance of thorough differential diagnosis in patients with pleural effusion, and is an example of blunt chest trauma from chest compressions as an uncommon but possibly overlooked etiology of chylothorax.
Alexandria Roy Dr. Emily Olson	*Blacklegged Tick Madness: Babesia Microti and Borrelia Burgdorferi Coinfection* Introduction: Babesiosis is a rare disease caused by infection of human erythrocytes with parasitic tickborne protozoans of the Babesiidae genus. Babesiosis can range in presentation from asymptomatic to life threatening; manifestations and complications can include flu-like symptoms, hemolysis from parasitemia, splenic rupture, acute respiratory failure, congestive heart failure, renal failure, shock, and death. Babesia is spread through the Ixodes scapularis (Blacklegged) tick, which can also carry Borrelia burgdorferi, Anaplasma phagocytophilum, and Powassan virus. High suspicion for tick-borne illness in endemic areas is essential, especially with the possibility of co-infections.

	Case Presentation: A 78-year-old male presented to the Emergency Department with two-days of fevers, nausea, vomiting, confusion, and generalized weakness. Physical examination revealed a diaphoretic, ill-appearing patient with a petechial rash across his bilateral lower extremities. There was no known tick bite or erythema migrans, although he did spend time outdoors. Initial laboratory studies were significant for acute hemolysis superimposed on hypoplastic anemia, thrombocytopenia, and mild hepatic transaminase elevation. Work up for tick-borne illness was initiated based on his presentation and endemic area. A peripheral blood smear revealed intracellular trophozoites with a parasitemia burden of 0.5%. Serum polymerase chain reaction was positive for amplification of Babesia microti DNA. Serum indirect fluorescent antibody testing was positive for Babesia specific immunoglobulin gamma (IgG) at a titer of 1:1024. These results were consistent with moderate Babesiosis due to acute infection with Babesia microti. Evaluation for co-infection was significant for positive Lyme antibodies on screening enzyme-linked immunosorbent assay. Confirmatory Lyme IgG and IgM immunoblots were positive for greater than five and two Borrelia burgdorferi proteins, respectively. These results were consistent with early disseminated Lyme disease caused by Borrelia burgdorferi coinfection. His evaluation for complications of Lyme disease, including facial palsy, radicular neuropathy, Lyme carditis, and neuroinvasive borreliosis was negative. Given his relatively low Babesia parasitemia burden and absence of severe features, he received a seven-day course of Azithromycin and Atovaquone as well as a fourteen-day course of Doxycycline for Lyme co-infection. His clinical status and mentation improved significantly with treatment. Discussion: This case highlights the necessity to consider co-infection of tick-borne illnesses, as co-infection can have important implications for management and prognosis. For example, treatment regimens differ between Babesia and Borrelia. Co-infection is a risk factor for severe illness and an increased risk of death. When patients do not present with erythema migrans, confirmatory testing is necessary to diagnose Lyme disease. Thorough evaluations for Lyme complications should be part of the diagnostic work-up.
Sehrish Sardar Dr. Ashley Hickman	*Urine for a Surprise: A Case of Renal Calyceal Rupture* Introduction: Renal calyceal rupture (RCR) is a rare complication of obstructive uropathy. RCR is caused by increased renal pelvis pressure from urine impaction leading to calyceal rupture. 75-80% of RCR cases are caused by acute, obstructing renal stones. Rare causes include pregnancy, urinary retention, iatrogenic injury, and malignancy. Malignancy as a cause of RCR is rare and is only described in a few case reports of genitourinary cancers. In this case, we describe an unusual presentation of RCR secondary to locally invasive cervical cancer with extensive urinoma presenting as thigh pain. We also discuss the treatment of urinoma. Case Presentation: A 69-year-old female without significant medical history presented for three days of upper abdominal pain associated with constipation, emesis, 20 kg unintentional weight loss, and years of postmenopausal bleeding. Physical exam was significant for right upper quadrant abdominal tenderness. Abdominal ultrasound showed right sided hydronephrosis. CT abdomen and pelvis revealed a pelvic mass invading surrounding structures including the right ureter, with atrophy of right renal parenchyma. Biopsy revealed high grade squamous cell cervical carcinoma. Placement of a

	nephrostomy tube was discussed but ultimately deferred due to an atrophic right kidney with presumed trivial function and a decompressed ureter. The patient was discharged home with outpatient oncology follow up scheduled in one month. During her oncology appointment, she described increased right thigh pain and swelling, otherwise denied new symptoms. Staging PET scan showed rupture of the upper pole of the right kidney due to hydronephrosis from progressive malignant obstruction complicated by an extensive urinoma extending from the posterior right kidney along the retroperitoneum and psoas/iliopsoas musculature into the muscles of the proximal medial right thigh. She was readmitted to the hospital, and these findings were confirmed with a CT Urogram with the urinoma measured to be 36 cm. Percutaneous drains were placed to the right perinephric, right retroperitoneal, and right upper leg fluid collections. Nephrostomy tube was deferred due to urinoma infection with E. coli. A CT urogram was repeated 48 hours after tube placement and showed interval decrease in all three fluid collections. She was discharged home on antibiotics with plans for future nephrostomy tube placement. She was initiated on radiation therapy to shrink the tumor and relieve obstruction. Discussion: This is a rare case of locally advanced cervical cancer causing ureteral obstruction leading to hydronephrosis, calyceal rupture, and an extensive urinoma presenting as right thigh pain. RCR is more commonly seen with acute renal stone impaction, and is less common in malignancy, potentially secondary to the subacute/chronic obstruction from tumor burden. Treatment of urinoma includes percutaneous drain placement with or without nephrostomy tube to decompress the kidneys. Nephrostomy tube placement should be delayed in infected urinomas, until infection resolves.
Dhruv Sarma	*A Case of Non-Bacterial Thrombotic Endocarditis with Embolic Stroke* Introduction: Non-bacterial thrombotic endocarditis (NBTE) is characterized by the deposition of sterile thrombotic vegetations on native cardiac valves. It is associated most frequently with malignancy and systemic lupus erythematosus, and is diagnosed clinically by the presence of valvular vegetations in the absence of bacteremia or fungemia. There is a paucity of data regarding optimal management of this rare condition, particularly regarding the use of direct anticoagulants for treatment. Case Presentation: A 70-year-old lady with a background of metastatic lung adenocarcinoma, paroxysmal atrial fibrillation and previous deep vein thrombosis with pulmonary embolism presented to the emergency department with a one week history of progressive headache and mild gait unsteadiness. Pertinent medication history included apixaban 5 mg twice daily and chemotherapy with carboplatin, pemetrexed, and pembrolizumab administered one week prior to presentation. Clinical examination was significant for a soft pan-systolic murmur at the cardiac apex and mild-moderate difficulty with tandem walking. CT brain on admission revealed three hypo-attenuating lesions in the left cerebellar hemisphere and right temporal and occipital lobes, with surrounding edema. Subsequently, Diffusion Weighted MRI brain demonstrated large areas of restricted diffusion in these areas, but also revealed numerous smaller areas of restricted diffusion throughout both cerebral and cerebellar hemispheres, as well several tiny enhancing foci in bilateral frontal lobes. These findings were most in keeping acute or early sub-acute infarcts. Given suspicion for an embolic source of cerebral infarction, trans-esophageal echocardiography (TEE) was performed, which confirmed a thickened mitral valve with mobile

	echodensities present on both anterior and posterior leaflets, and moderate mitral regurgitation. In the absence of positive blood cultures or other symptoms or signs of infection, the diagnosis of NBTE was made. Notably, this developed despite anticoagulation with apixaban. Acknowledging the risk of hemorrhagic transformation of the patient's cerebral infarcts, anticoagulation with enoxaparin was initiated due to the high risk of further embolic infarction. She was subsequently discharged with minimal neurological deficit. Follow up TEE five weeks later showed that mitral valve thrombotic vegetations had resolved and the mitral regurgitation had decreased to mild. Discussion: This case illustrates the importance of excluding an intracardiac source of embolism in the work-up of stroke, especially when infarcts are widespread or in a non-vascular territory. Furthermore, the value of a thorough clinical examination is highlighted; clues to the diagnosis of endocarditis with systemic embolism were provided by the presence of ataxia and a new regurgitant murmur. Lastly, this patient developed thrombotic MV vegetations despite apixaban use, which resolved after five weeks of anticoagulation with enoxaparin. This may suggest that low molecular weight heparin is comparatively more effective than direct oral anticoagulants in the management of NBTE.
Thomas Schmidt Lucy Witchell Dr. Brandon Wesche	*To the Brink and Back: Blastomycosis and Acute Respiratory Distress Syndrome in the ICU* Introduction: Blastomycoses is a fungus that can be found in the environment in the Great Lake, Ohio and Mississippi river valleys. This fungus is found typically in decaying wood or leaves. Infection occurs when a host inhales spores of the fungi. Symptoms of infection can range from fever and cough to severe illness requiring hospitalization, even death. Case Presentation: A 65-year-old immunocompetent female with recent breast cancer diagnosis, DCIS with recent surgical resection, presented to the hospital following months of dyspnea and new profound hypoxia to 85, white blood cell count of 9.9, and a CT chest demonstrated bilateral infiltrates. She was empirically treated for a community acquired pneumonia, given bronchodilators upon discharge, and home oxygen. Following discharge from the hospital, she was requiring oxygen at ever increasing levels, ultimately came back to the hospital in acute on chronic hypoxic respiratory failure requiring intubation, admission to a local Intensive Care Unit. WBC of 12.8 and CRP of 31 on arrival to ICU. The PaO2 to FiO2 ratio in the ICU was 71. Upon initial admission, patient was found to have a recent EIA Histoplasmosis positive test from her previous admission. Given the profound hypoxia and acute respiratory distress syndrome, the patient required prone placement, four vasopressors, maximum ventilatory support in accordance with ARDS, and dialysis. Amphotericin B, vancomycin, piperacillin/tazobactam were empirically started. Corticosteroids were initiated and extra corporeal membrane oxygenation (ECMO) was entertained, no local ECMO capable hospitals had availability due to ongoing COVID-19 pandemic. Sputum results returned 2 days after admission with a high density of blastomycoses. Following administration of amphotericin B, the patient slowly recovered such that she no longer requiring vasopressors, able to tolerate intermittent hemodialysis.

	Discussion: While Amphotericin B is the main therapy to treat blastomycosis in severe infections. The robust inflammatory response that accompanies severe infection has led clinicians to utilizing corticosteroids. For patients with severe respiratory failure such that they cannot adequately ventilate or oxygenate their lungs while on mechanical ventilation, ECMO has been utilized. Corticosteroids and ECMO are logical choices in severe disease as these therapies are utilized in other settings of ARDS. It remains unclear as to the efficacy of these therapies in reducing morbidity or mortality as randomized control trials have not been conducted.
Leah Soderberg Dr. Benjamin Stultz Dr. Michael Mueller	*A Case of IgA Vasculitis with a History Worth Repeating* Introduction: IgA vasculitis (IgAV) is a rare small-vessel inflammatory condition primarily involving the skin, joints, GI tract, and kidneys. Early recognition and surveillance for potential complications, most commonly renal involvement progressing to chronic kidney disease, is critical to reducing IgAV-associated morbidity. There is a broad spectrum of known IgAV triggers and identifying these in patients with characteristic symptoms aids clinical evaluation and expedites diagnosis. Case Presentation: A 25-year-old male with no significant past medical history presented with a one-day history of non-blanching petechial rash in the distal lower extremities and pain and swelling of the knees and ankles. He had no recent fevers, upper respiratory symptoms, dyspnea, abdominal pain, nausea, vomiting, diarrhea, urinary symptoms, or recent febrile illness. He was sexually active but used barrier contraception and had no new partners. Ankle ultrasound revealed bilateral joint effusions, but subsequent synovial fluid studies were unremarkable. CRP was elevated to 8.8 but labs showed no abnormalities on CBC, normal renal and liver function, and negative ANA and ANCAs. Infectious workup was negative for HIV, HSV, hepatitis B and C, tuberculosis, syphilis, chlamydia, and gonorrhea. Urinalysis showed no proteinuria, hematuria, or pyuria. A urine drug screen was positive for THC, opiates, and amphetamines and a tick-borne illness panel had positive 1:128 titers for Anaplasma and Ehrlichia IgG. He was treated with 10 days of doxycycline. Two weeks later, he returned with worsening polyarthralgias. His rash had evolved to purpuric papules and erythematous targetoid lesions with overlying dark vesicles and necrotic centers covering his dorsal feet, legs, and buttocks. Histologic analysis of his skin biopsy was consistent with leukocytoclastic vasculitis and direct immunofluorescence demonstrated IgAV. The patient was treated supportively and had no sequelae or evidence of additional organ involvement. Discussion: Although most commonly seen in young children, up to 10% of IgAV cases occur in individuals over age 10. While there are many IgAV triggers, upper respiratory and gastrointestinal infections precede 75% of cases. Other triggers include allergies, medications (antibiotics, NSAIDs, TNF-alpha inhibitors), illicit drug use, vaccines, tick bites, and malignancy. IgAV complications are secondary to vasculitic organ inflammation and damage. Although progressive renal failure is the most prevalent, intussusception, GI and pulmonary hemorrhage, interstitial fibrosis, neurologic manifestations, and inflammatory eye conditions can also be seen. Surveillance for disease progression and emerging complications after diagnosis is critical to reduce morbidity and mortality. This case demonstrates multiple clues in the patient's history to raise suspicion for a vasculitic process including recent methamphetamine use and tick exposure. An excellent patient history that identifies potential triggers is critical to making a

	unifying clinical diagnosis of IgAV and initiating early surveillance for sequelae and complications.
Mackenzie Stice	*When Weakness isn't Deconditioning: A Case of Anti-HMG-CoA Reductase Necrotizing Myositis* Introduction: Anti-HMG-CoA reductase necrotizing myositis is a rare autoimmune myopathy, with a prevalence of less than four cases per 100,000 people1. Often related to statin use, it is an important diagnosis to consider in patients with proximal muscle weakness. Diagnosis is based on muscle biopsy, autoantibody testing and response to immunosuppression. Case: A 76-year-old male with past medical history of seizure disorder, CAD s/p CABG x4 presented with acute on chronic right hip pain and right lower extremity weakness. In the preceding month he had lost the ability to get out of his recliner or weight bear independently. He had no history of trauma or infectious symptoms. Emergency department exam was limited by right hip pain but distal strength in his lower extremities was intact. Hip radiograph was without acute fracture or dislocation. Basic labs were largely unremarkable apart from large blood on urinalysis with only 1 RBC on microscopy. Creatine kinase (CK) level was markedly elevated at 18,000 units/L. While there was not a convincing history of rhabdomyolysis, initial management included aggressive fluid resuscitation. After 3 days, CK was unchanged, prompting a rheumatology consult. Thorough exam demonstrated 3/5 hip flexor strength on the right, 4/5 on the left with 4/5 strength in bilateral shoulders. The combination of proximal muscle weakness with elevated CK raised concern for necrotizing myositis related to statin use, although statin medication had been held since admission. High dose corticosteroids were initiated. MRI revealed "extensive T2 signal/edema and enhancement throughout the musculature of both thighs and lower pelvis." Muscle biopsy of the right thigh was obtained and electron microscopy confirmed diffuse myofibril necrosis. The CK remained elevated after 5 days of pulse steroids, prompting two doses of intravenous immunoglobulins. CK gradually down trended and the patient demonstrated slow clinical improvement. Anti-HMGCR autoantibodies ultimately returned elevated at 138 (reference range <20). At discharge, he was prescribed a prolonged steroid taper with close rheumatology follow-up. Given his history of significant CAD, he was started on a PCSK9 inhibitor for lipid lowering given absolute contraindication to statin therapy. Discussion: Inflammatory myopathies are a collection of syndromes characterized by proximal muscle weakness, often elevated CK levels, and characteristic changes on muscle biopsy1. This case encourages the general internist to remain alert to acute pathology when building a differential diagnosis for subacute weakness, a relatively common cause of hospital admission. The physical exam remains a powerful tool for narrowing the differential in these cases. References: 1) Pinal-Fernandez, Iago et al. "Immune-Mediated Necrotizing Myopathy." Current rheumatology reports vol. 20,4 21. 26 Mar. 2018, doi:10.1007/s11926-018-0732-6

Michael Storandt Dr. Karna Sundsted Dr. Kathryn Eschbacher Dr. Teerin Liewluck Dr. Christopher Klein	*Immune-Mediated Necrotizing Myopathy Causing Rhabdomyolysis* Introduction: Immune-mediated necrotizing myopathy (IMNM), previously called necrotizing autoimmune myopathy, is a rare immune-mediated myopathy affecting 5 in a million persons. Hospitalization is common from subacute onset weakness with rhabdomyolysis. Case Presentation: A 78-year-old female presented to the emergency department with 10 days of progressive fatigue, weakness, dysarthria, with oliguria and leg edema. Medical history included chronic kidney disease stage III, transient ischemic attack (TIA), distant history of bilateral subdural hematomas, and Hepatitis B with undetectable viral load. On examination, she exhibited flaccid dysarthria, right lower extremity weakness of hip flexors and knee extensors, with profound lower extremity edema. Labs were remarkable for hyperkalemia, anion gap metabolic acidosis, acute kidney injury (AKI) with a creatinine of 10.58 mg/dL (RR 0.59-1.04 mg/dL), and transaminitis. She underwent noncontrast head CT, CT abdomen/pelvis, and chest x-ray, all unremarkable. Further work-up for AKI included creatine kinase (CK), which was elevated to 25,990 U/L (RR 26-192 U/L). She had recently been started on atorvastatin 40 mg following a TIA 5-months prior, without other risk factors for rhabdomyolysis. She began dialysis, with persistent CK elevations (>16K U/L) and progressive muscle weakness, now involving bilateral iliopsoas, gluteus maximus, quadriceps, hamstrings, deltoids, and biceps (MRC 3-4). An electromyogram demonstrated proximal myopathy with frequent fibrillation potentials and myotonic discharges. IMNM was considered utilizing an online validated calculator (www.imnm.info) for disease likelihood. This calculated 99% likelihood of IMNM, and she was initiated on high-dose methylprednisolone and intravenous immune globulin (IVIG), while myositis-specific and IMNM-specific antibodies [anti-HMG-CoA reductase (anti-HMGCR) and anti-signal recognition particle (anti-SRP)], were pending, and ultimately negative. On immunotherapy, her CK elevations immediately began improving. A subsequent muscle biopsy showed scattered necrotic fibers without inflammation supporting the diagnosis of seronegative-IMNM. She was initiated on Rituximab 375mg/m2 and 40mg prednisone, with IVIG and methotrexate not possible long-term because of her kidney disease. A repeat dose of rituximab was planned in 2 weeks, along with outpatient management of chronic immunosuppression and CT/PET whole body for exclusion of paraneoplastic causes. At time of discharge, her CK had downtrended to 205 U/L. She also had improved strength able to ambulate with a walker safely, with reduced dialysis requirements. Discussion: IMNM affects middle-age persons and rarely children, typically with subacute onset (2-4 weeks) of proximal>distal weakness, occasionally affecting bulbar musculature. Hospitalizations are common with rhabdomyolysis and associated kidney injury. Approximately 70% of patients are seropositive, either anti-HMGCR (statin exposure commonly) or SRP autoantibodies. The online calculator can assist in speeding confirmatory biopsy and serologic testing. Delays in diagnosis are associated with cardiorespiratory deaths and permanent neuromuscular disability. Steroids alone are typically ineffective at reducing CK, improving kidney function, or improving strength. Seronegative-IMNM may have a greater likelihood of underlying malignancy driving the process.
Rachel Suen Dr. Grant	*Immune Mediated Necrotizing Myositis: Don't Let Seronegativity Creep you from the Diagnosis*

Wintheiser Dr. Mohammed Bhuiyan	Introduction: Immune-mediated necrotizing myopathy (IMNM) is a rare inflammatory myopathy distinguished on pathology by necrotic muscle fibers without significant inflammation. It is associated with anti-3-Hydroxy-3-Methylglutaryl-CoA Reductase (HMGCR) and anti-signal related peptide (SRP) antibodies, although up to 20% of patients are seronegative. Like other immune-mediated myopathies, it presents with progressive muscle weakness and elevated serum creatine kinase (CK). Treatment consists of immunosuppression with some combination of steroids, immunomodulators, intravenous immunoglobulin (IVIG), methotrexate, and rituximab. Case Presentation: A 78-year-old woman presented with four days of bilateral lower extremity weakness. Physical exam revealed proximal muscle weakness in the deltoids and hip flexors. Labs demonstrated a CK of 26,000 unit/L and marked transaminitis (ALT 617, AST 1338). She also had a significant AKI (creatinine 10.51) and ultimately required dialysis. Notably, she had been started on statin therapy several months prior. This, combined with her worsening muscle weakness throughout the hospitalization and lack of alternative explanation for her rhabdomyolysis, prompted concern for an IMNM. Myopathy panel was negative for anti-SRP and anti-HMGCR antibodies. EMG showed fibrillation potentials and moderate myotonic discharges, suggestive of muscle denervation. Muscle biopsy showed necrotic and regenerating muscle fibers, suggestive of active muscle myopathy. Despite the negative antibody testing, she was diagnosed with IMNM. Treatment with high-dose IV steroids, IVIG, and methotrexate resulted in rapid improvement in her CK and transaminases. She was later switched to oral steroids and rituximab given her continued AKI. She was discharged on intermittent hemodialysis, with plans for outpatient PET-CT to evaluate for malignancy. Discussion: This case illustrates the importance of maintaining high clinical suspicion for IMNM in patients with myopathy, as it can cause rapidly progressive and severe muscle impairment if left untreated. Notably, up to 20% of patients do not have anti-HMGCR or anti-SRP antibodies.[i] The pathophysiology of seronegative IMNM is unclear, but the disease is associated with increased malignancy.[ii] Although the diagnosis can be strongly suggested on clinical grounds and laboratory testing, muscle biopsy is required for definitive diagnosis. The characteristic findings are myofibers in various stages of necrosis and regeneration with absence of inflammatory cells. Treatment of all types of IMNM includes immunosuppression.
Nuttavut Sumransub Dr. Keith Skubitz	*Malignancy-Associated Lactic Acidosis: A Case Series and Literature Review* Abnormal accumulation of lactic acid occurs secondary to increased production, decreased clearance, or a combination of both. Etiologies can be broadly categorized into type A, a state related to tissue hypoxia or hypoperfusion, and type B which refers to other causes not related to tissue hypoxia. Most reported cases of malignancy-associated lactic acidosis have been described with hematologic malignancy, however, some, have occurred in patients with solid tumors. Several mechanisms for malignancy-associated lactic acidosis have been proposed. Metabolism reprogramming is accepted to be one of the hallmarks of cancer, as exemplified by the Warburg effect, an upregulation of glycolysis even in aerobic conditions. Besides aberrant cellular metabolism, rapidly proliferative cancer cells with limited blood supply and oxygen can create a

	relative hypoxic environment further augmenting production of lactic acid. Decreased lactate utilization from extensive liver metastasis might also play a role in some cases. Malignancy-associated lactic acidosis is associated with a very high mortality rate and is usually fatal without the initiation of treatment for underlying malignancy. We report 6 selected cases of tumor-associated lactic acidosis, and describe 3 in detail that demonstrate interesting pathophysiology related to the condition. Localization of sites of production and excretion of lactic acid are presented in the case of a 71-year-old man with metastatic small cell lung cancer. Chronic hypokalemia and cancer cachexia are observed and discussed in our second and third cases of metastatic leiomyosarcoma. Lactic acid metabolism and the treatment of lactic acidosis in neoplastic disease are also discussed. The observation of this number of cases of lactic acidosis in solid malignancies in one practice suggests the syndrome is more common than appreciated. Awareness of the syndrome is critical to provide appropriate care.
Benjamin Swart Dr. Brooke McDonald	*Hickam's Purpura? Multiple Vasculitides Presenting in a 67-Year-Old Man* Introduction: The presence or absence of immune complexes is one of the major differentiating factors of acute glomerulonephritis (GN). Lupus nephritis is one of multiple GNs characterized by immune complex deposition. Pauci-immune vasculitides, in contrast, are characterized by a lack of immune complex deposition. This case report describes a patient with rapidly progressive GN secondary to both types of mechanisms. Case Presentation: A 67-year-old male with past medical history pertinent for lupus nephritis, CKD 3, hypertension, and hypothyroidism presented with five days of altered mental status and fever. Vitals at presentation were within normal limits. On exam he was obtunded, with a non-blanchable, petechial rash on his lower extremities, bilateral myoclonus in upper extremities, and cola-colored urine. Initial labs were notable for WBC count 24.51 cells/mm2, hemoglobin 11 g/dL, and platelets 302 cells/mm2. Basic metabolic panel revealed serum potassium 5.4 mEq/L, BUN 33 mg/dL and creatinine of 3.94 mg/dL (baseline of 1.5). Inflammatory markers were elevated with CRP of 225.7 mg/L and ESR of 46 mm/hr. Complements 3 and 4 were within normal limits. HIV, hepatitis B/C and cryoglobulins were negative. IgA and IgG were within normal limits and CSF was unremarkable. His urinalysis was notable for proteinuria and hematuria with no growth on urine culture. Head CT and renal ultrasound were both within normal limits. Lupus anticoagulant was positive. Anti-neutrophil cytoplasmic antibody (ANCA) was negative, however myeloperoxidase (MPO) antibody was strongly positive (46 AU/mL). With low suspicion for active infection, the patient was started on high dose corticosteroids and emergent dialysis in the setting of worsening uremia. Kidney biopsy was performed and demonstrated focal proliferative lupus glomerulonephritis (classes III and V) in addition to necrotizing small artery vasculitis consistent with MPO/ANCA vasculitis. Skin biopsy was positive for leukocytoclastic vasculitis without lupus involvement. The patient was started on monthly cyclophosphamide with mesna infusion for dual treatment of both lupus nephritis and ANCA vasculitis and hydroxychloroquine with prednisone taper. By the time of discharge, his kidney function had significantly improved.

	Discussion: Though rare, there are case reports of an overlapping syndrome of systemic lupus erythematosus and ANCA/MPO-associated vasculitis. It usually presents in women in the fourth decade of life and diagnosed via kidney biopsy. Treatment is not standardized, but can include cyclophosphamide, rituximab, hydroxychloroquine, plasma exchange, IV immunoglobulin, and steroids. When presented with conflicting lab values, be open to the fact that a single unifying diagnosis may not exist. In the search for parsimony, we should always be mindful of Hickam's dictum: "Patients can have as many diseases as they please."
Reema Tawfiq Dr. Christopher Tipton Dr. Akeem Lewis Dr. Naseema Gangat **2021 MN-ACP Resident People's Choice Award**	*Triple Negative Myelofibrosis with Pyoderma Gangrenosum and Sweet's Syndrome* Introduction: Myelofibrosis is a myeloproliferative neoplasm characterized by marrow fibrosis and extramedullary hematopoiesis. The median age of presentation is 67 years. In rare occurrences, it can affect younger adults. Commonly implicated driver mutations include JAK2, CALR and MPL. Triple-negative MF makes up 10-15% of patients with MF and lends to a higher rate of leukemic transformation and poorer survival. Other high-risk mutations linked to MF have been identified, such as ASXL1, EZH2, IDH1/2, SRSF2, and U2AF1. Case presentation: A previously healthy 29-year-old Nepali male presented with pancytopenia, recurrent fevers, night sweats, weight loss, and a left axillary skin lesion, presumed to be an abscess. Despite treatment, he continued to have high-grade fevers, drenching night sweats, rigors, and the wound continued to progress. He was transferred for further evaluation and management. On physical exam, he was thin and weak, with pale oral mucosa and conjunctiva. The left axilla had a large ulcer with irregular borders covered with granulation tissue mixed with areas of necrosis. On the right forearm and back, there were several tender erythematous violaceous plaques. Initial laboratory studies revealed pancytopenia hemoglobin 7 g/dL, platelet 52 x 109/L, and leukocytes 2.1 x 109/L without evidence of hemolysis or blasts; renal and liver function were normal but, inflammatory markers were considerably elevated: CRP 213 mg/L, ferritin 1300 mcg/L, IL-6 18.6 pg/mL and IL-2 soluble receptor alpha 2119 pg/mL. Exhaustive infectious and rheumatologic evaluation was negative. Dermatopathology elucidated two distinct noninfectious neutrophilic dermatoses. The pathology of the left axillary wound revealed pyoderma gangrenosum, and the erythematous plaques were consistent with Sweet's syndrome. PET-CT showed no organomegaly but increased FDG-activity in the lungs, and lavage revealed increased neutrophils with no underlying infection, suggesting likely pulmonary Sweet's syndrome. Bone marrow biopsy showed a hypercellular marrow with marked megakaryocytic hyperplasia, dysmegakaryopoiesis with grade 3 fibrosis, and no blasts. Next-Generation Sequencing resulted in a pathogenic U2AF1 mutation. The patient was diagnosed with primary myelofibrosis. He was initially treated with high-dose steroids and broad-spectrum antibiotics without improvement. Given the above findings, he was started on ruxolitinib, and continued steroids with clinical improvement. Discussion: This is a unique presentation of primary triple-negative myelofibrosis which is typically a chronic hematological malignancy. Despite the complex clinical manifestations, the bone marrow biopsy findings coupled

	with the presence of the U2AF1 mutation supported the diagnosis of primary myelofibrosis rather than reactive or autoimmune myelofibrosis. The pyoderma gangrenosum and Sweet's syndrome with pulmonary involvement was a secondary systemic inflammatory reaction to the primary myeloid neoplasm. The decision to start ruxolitinib, a JAK inhibitor and high-dose steroids was due to ongoing high-grade fevers. Ultimately, the patient is being considered for a matched sibling donor hematopoietic stem cell transplant which may offer prolonged survival.
Daniel VanDerhoef Dr. Thomas Meehan	*Not Your Typical Chest Pain: Chronic Necrotizing Pulmonary Aspergillosis* Introduction: Aspergillus osteomyelitis is a severe and rare form of invasive aspergillosis. Seen in immunocompetent and immunocompromised individuals, it is difficult to diagnose without tissue sampling given its rare and often insidious presentation. Most commonly it develops via hematogenous spread, but it has also been reported to occur via contiguous spread and direct inoculation. We present a case of a 77-year-old immunocompromised male who developed osteomyelitis of the sternum likely following contiguous spread from chronic necrotizing pulmonary aspergillosis. Case Presentation: A 77-year-old immunocompromised male with a past medical history of rheumatoid arthritis on rituximab, type-I diabetes mellitus on insulin, COPD, ischemic heart failure and post-traumatic axial skeletal deformity with chronic pneumothorax presented to outpatient pulmonary clinic with 18 months of a progressively worsening cough and reproducible chest pain. He had been intermittently treated for community acquired pneumonia over the preceding year with transient but incomplete resolution of symptoms. Chest CT prior to this evaluation demonstrated a left-upper lobe consolidated opacity, scattered air space opacities, pleural thickening and a usual interstitial pneumonia pattern. Initial labs were remarkable for a C-reactive protein (CRP) 37 mg/dL, with no leukocytosis. In clinic, his COPD was optimized on triple therapy, and repeat imaging in 8 weeks was planned. On follow up, his chest pain had improved but repeat imaging demonstrated a new left lower lung cavitary nodule and atelectasis, as well as erosive changes involving the sternum concerning for osteomyelitis. He was referred to infectious disease clinic, where after further evaluation he was not believed to have osteomyelitis given overall stability and unchanged clinical picture. Notably at that time he had a CRP of 35, as well as negative histoplasmosis and blastomycosis antigens. He was lost to follow-up. He presented to the hospital two months later after two months of chest pain and drainage from a rash on his chest. A repeat CT scan of his chest demonstrated new T12 burst fracture, increased inflammatory changes around site previously concerning for osteomyelitis and now involving the 6th rib. Inflammatory markers remained elevated, beta-D-glucan level was > 500 pg/mL, and repeat histoplasmosis and blastomycosis antigens were negative. Initial wound culture showed light growth of aspergillus, with subsequent confirmation of infection with removal of inferior aspect of sternal bone and subsequent culture growing Aspergillus fumigatus. He was treated with voriconazole with clinical improvement. Conclusion: Aspergillus is a rare cause of osteomyelitis, and treatment usually requires surgical debridement and antifungal treatment. Our case illustrates the importance of considering Aspergillus infections in the immunocompromised, as well as to consider Aspergillus infections as a cause

	of insidious infection in immunosuppressed individuals with chronic respiratory symptoms.
Elizabeth Wendt	*Two Uncommon Etiologies of Hypercalcemia - Adrenal Insufficiency and Hemosiderosis* Introduction: Treatment for hypercalcemia typically consists of lowering the serum calcium concentration and treating the underlying disease. But, what do you do when the underlying cause is difficult to discern? This case explores two uncommon etiologies of hypercalcemia - adrenal insufficiency and hemosiderosis. Case Presentation: A 55-year-old male with history of esophageal SCC s/p resection (now in remission) with recent prolonged hospitalization was admitted after routine clinic BMP revealed hypercalcemia to 14. The patient's only symptoms were 3-4 weeks of myalgias, arthralgias, and pruritis, which he initially attributed to other causes. He was not on any obvious causative medications including lithium or thiazide diuretics. He was not significantly volume deplete. PTH was found to be low-normal, indicative of non-PTH mediated hypercalcemia. PTHrP, 1-25-dihydroxyvitamin D, and 25-hydroxyvitamin D were all within normal limits. Despite this, given high suspicion for cancer recurrence, further evaluation with NM bone scan was conducted and unrevealing for any metastasis or new cancer. CTCAP was unrevealing for granulomatous disease and quant gold and sarcoidosis work up, including ACE and IL-2, was unrevealing. SPEP/UPEP/immunofixation, TSH, and vitamin studies were also unrevealing. At this point, adrenal insufficiency work up was pursued given patient's recent prolonged hospitalization during which time steroids were utilized, and results were notable for sub-optimal response to cosyntropin stimulation. Decision was made to initiate steroids to treat possible secondary adrenal insufficiency. Although the mechanism of hypercalcemia in adrenal insufficiency is not fully understood, it is thought that changes in GFR due to hypervolemia may alter the balance of calcium excretion and absorption; additionally, alpha-1-hydroxylase levels may be increased in adrenal insufficiency, leading to increased conversion of inactivated vitamin D to activated vitamin D and thereby increasing calcium resorption. Later in admission, the patient was found to have increasing alkaline phosphatase with elevated GGT. MRCP revealed hemosiderosis of the liver and spleen. Ultimately, it was felt that the hemosiderin deposition was secondary to multiple pRBC transfusions during the patient's previous admission. There is some evidence to suggest that iron accumulation may be associated with a bone loss due to both osteoblast inhibition and osteoclast activation. Since admission, the patient's calcium has remained within normal limits without ongoing steroid therapy. This suggests that the underlying process causing hypercalcemia is not ongoing, supporting theories that secondary adrenal insufficiency and/or secondary hemosiderosis were culprits. Discussion: Because calcium plays a critical role in myocardial function, coagulation, neural transmission, and other cellular functions, hypercalcemia can be a life-threatening condition. Primary hyperparathyroidism and malignancy account for >90% of all hypercalcemia cases; however, when the underlying etiology lies outside of those categories, the diagnosis can be elusive and may warrant evaluation for less common etiologies including

	adrenal insufficiency and hemosiderosis.
Rebecca Yao Dr. Ikram-Ul Haq	*A Case of Presumed Tuberculous Pericarditis Treated with Prednisone* Case Presentation: A 32-year-old physically active male immigrant from Cameroon presented with chronic persistent abdominal pain with distension, cough and shortness of breath. He had been previously hospitalized for these symptoms during their initial onset. Prior workup revealed elevated ALP (144), CRP (90.3), and BNP (974), along with a paracentesis which revealed a SAAG of 1.4 and total protein greater than 2.5, suggestive of cardiac ascites. Abdominal imaging showed hepatomegaly with possible cirrhosis and moderate bilateral pleural effusions. CT Chest was performed for his respiratory symptoms, revealing pulmonary infiltrates, and a sputum PCR was positive for Mycobacterium tuberculosis. He was promptly started on RIPE therapy and later discharged.
Yuan Yao Dr. Hadiyah Audil Dr. Kimberly Wang Dr. James Howick Dr. Johnny Dang Dr. Camilo Bermudez Noguera Dr. Christopher Aakre	*A Clear but Not So Clear Case of Type 4 Renal Tubular Acidosis* Introduction: Hypoaldosteronism, or type 4 renal tubular acidosis, could lead to persistent hyperkalemia and a mild hyperchloremic metabolic acidosis with normal anion gap. The etiologies of hypoaldosteronism include both acquired and inherited disorders that affect aldosterone synthesis and renin secretion. Presentation: A 92-year-old female presented with delirium secondary to a urinary tract infection and was found to have an elevated potassium at 6.1. She was also found to have incidental hyperkalemia when hospitalized 2 years ago, which resolved with temporizing measures during this hospital stay. Her medical history was significant for obstructive sleep apnea, congestive heart failure, hypertension, hyperlipidemia, coronary artery disease, hypothyroidism, chronic kidney disease and depression; she denied regular NSAID use. During her post-discharge follow up, labs revealed potassium 5.9, sodium 135, chloride 110, bicarbonate 17, anion gap 8. Further assessment showed aldosterone <4, renin activity <0.6, cortisol level normal at 9.7. TSH was 0.04. She was treated with polystyrene sulfonates (Kayexalate), with which her potassium improved. She was then started on torsemide 10 mg for hyperkalemia and heart failure symptoms. Her potassium level dropped to 5.3 at 2-month follow up. Discussion: Based on the findings, this patient was diagnosed with hyporeninemic hypoaldosteronism, which is the most common acquired cause of hypoaldosteronism. Review of her medications did not show any potential pharmacologic cause. Her hypoaldosteronism was attributed to her underlying CKD. However, stage III CKD does not entirely match the extent of her hyperkalemia. Although further testing was deferred in this patient due to her age and the favorable response to potassium lowering therapy, one could consider follow up workup for hereditary causes of hypoaldosteronism in such patients.
Lucas Zellmer	*No Obstruction, Still a Problem: A Myocardial Infarction with Clean Coronary Arteries* Introduction: Contemporary use of coronary angiography in the setting of acute myocardial infarction (AMI) has resulted in greater characterization of myocardial infarction with nonobstructive coronary arteries (MINOCA). In addition to meeting the standard diagnostic requirements for myocardial infarction, MINOCA is defined by the absence of obstructive coronary artery

	disease (<50%) and absence of another precipitating cause (ie. stress cardiomyopathy). Diagnosis, acute interventions, long-term management, and epidemiologic factors of AMI are well-established in the literature, however, MINOCA represents a relatively new working diagnosis that requires additional investigation. The case discussed herein provides a backdrop to review MINOCA and discuss the current literature on diagnosis and management of this syndrome. Case Presentation: A 58 year old woman with a history of hypertension and type II diabetes mellitus presents to the emergency department with a several hour history of substernal chest pain, nausea, and vomiting. The patient initially experienced chest pain the previous night that subsided after taking an aspirin and applying salve to her chest. After arriving at the emergency department, the patient was transferred to the stabilization room for evaluation; she was noted to be mildly hypertensive with an otherwise normal physical exam and bedside cardiac ultrasound was concerning for an inferior wall motion abnormality. Initial EKG showed ST elevation in the inferior leads and high-sensitivity troponins were increased to 1,698 ng/L (normal < 16) at two hours. The catheterization lab was activated and coronary angiography showed no obstructive coronary artery disease. Follow-up transthoracic echocardiogram in the hospital identified a lateral wall motion abnormality and ejection fraction of 40%. Differential diagnosis at that time included MINOCA/vasospasm, stress cardiomyopathy, and myocarditis. The patient remained vitally stable throughout her hospitalization with no other acute concerns. She was discharged on lisinopril, metoprolol, and atorvastatin and encouraged to follow-up with outpatient cardiology. Further outpatient imaging with cardiac MRI was pursued and did not indicate signs of scar or wall motion abnormality. The diagnosis remains unclear and the differential continues to include myocarditis and MINOCA/vasospasm, with stress cardiomyopathy less likely given initial EKG findings and lack of apical ballooning. The patient continues to follow with cardiology and a treatment plan centered around lifestyle modifications is being pursued. Conclusion: Thomas meehThe case described in this vignette portrays a common initial presentation of a common medicine diagnosis: ST elevation myocardial infarction. However, a patient without obstructive coronary disease in the setting of AMI forces providers to broaden their differential diagnosis to include less common etiologies for familiar cases. Moreover, this case illustrates the role of cardiac imaging in evaluating potential causes of AMI and serves as an excellent review of MINOCA.