American College of Physicians- Minnesota Chapter Annual Abstract Competition

Poster Session

October 27, 2023

Abstracts Submitted for Competition

Medical Students	
	Research/ Quality Improvement - Medical Students
Ivy Nguyen Allison Carr **Abstract Winner**	*The Role of Autophagy-Related Gene 5 (ATG5) in Sensitizing Cancer Cells to Immune-Mediated Cell Death* Background: Autophagy is a highly conserved process that helps maintain cellular homeostasis. Studies have shown that autophagy can have a duplicitous role in cancer cell survival, often suppressing tumor initiation, while also promoting tumor survival later on in progression. The role of autophagy in immune-mediated cancer cell death and survival remains uncertain. The goal of this study is to delineate the role of cancer cell autophagy processes in cytokine mediated cell death. Our study focuses on the role of ATG5, an autophagy protein that has surfaced in numerous cancer cell screens, investigating proteins that may be protective to tumor cells and help them resist immune-mediated cell death. Using a mouse melanoma cell line model (B16F1), we tested wild-type (WT) and ATG5 knockout (KO) cells in their cell death and proliferation response to TNFÎ± and IFNÎ³, two common cytokines which are not typically thought to be cytotoxic. We hypothesized that in the absence of autophagy, a protective mechanism of cancer cells, ATG5-KO B16 would have a higher level of cell death in response to TNFÎ± and IFNÎ³ compared to the wild-type B16. Methods: ATG5 was knocked out in mouse melanoma cell line (B16F1) using CRISPR/Cas9 and was confirmed with western blot. Control and treatment groups of both WT and ATG5-KO B16 were seeded in triplicates at 4000 cells/well at 37 degrees C. After 24 hours, the treatment groups were stimulated by either 100 ng/mL TNFÎ± alone, 100 ng/mL IFNÎ³ alone, or 100 ng/mL combination of both TNFÎ± and IFNÎ³. Cell confluence and death analysis were done in the IncuCyte S3 for 48 hours. 1 mM YOYO3-iodide dye was added at 1:2000 ratio for cell death detection. Cell death markers including cleaved Caspase 3, cleaved Caspase 8, cleaved Gasdermin D, and phosphorylated mixed lineage kinase domain-like protein (pMLKL) were detected using western blot. Results/Discussion: The ATG5-KO tumor cells were found to be more sensitive to cytokine induced cell death in comparison to WT, and were particularly sensitive at 100 ng/mL TNFÎ±+IFNÎ³ combo treatment. Cleaved Caspase 3 was seen in ATG5-KO B16 under both 10 ng/mL TNFÎ±+IFNÎ³ combo treatment and 100 ng/mL treatment but not seen in WT B16. Cleaved Caspase 8 was seen in both WT and ATG5-KO B16 under both treatments. Evidence of cell death through the cleavage of Caspase 3 and 8 suggested the

	possible involvement of apoptosis. Cleaved Gasdermin D and pMLKL were not detected, ruling out pyroptosis and necroptosis, respectively. Conclusion: Results from our study suggest that interference with cellular autophagy, specifically the loss of ATG5 protein, may be a mechanism of sensitizing cancer cells to TNFÎ± and IFNÎ³ mediated cell death. Therefore, targeting ATG5 through gene therapy could offer a promising strategy to decelerate tumor progression. In the next steps, we will investigate the specific mechanisms leading to this cell death in the absence of ATG5.
Virginia Hsiao Hongbo Wang Ross Cromarty Zachary Davis Jeffrey Miller	*Utilizing Tri-specific Killer Engagers Against B7H3 to Overcome Dexamethasone-Mediated Immunosuppression of NK Cells* Background: Glioblastoma Multiforme (GBM) is a difficult to treat, aggressive brain tumor, with an incidence of 4 in 100,000 people and represents around 14% of all primary brain tumors. The current standard of care involves surgical resection, followed by radiation therapy with or without temozolomide. Despite this, the median survival remains 14 months, as challenges posed by the blood brain barrier, heterogeneity of tumor cells, and the fast-growing nature of GBM limit treatment efficacy. Thus, novel therapies are urgently needed for the treatment of GBM. Natural killer (NK) cells offer a promising avenue for an accessible off-the-shelf targeted immunotherapy that can be applied in the context of GBM, as they can mediate an immune response without prior sensitization to viral or tumor antigens. Dexamethasone, which is a potent glucocorticoid given to GBM patients to treat cerebral edema, poses a potential challenge to the efficacy of immunotherapies, given their immunosuppressive nature. Here, we evaluated the suppressive effects of Dexamethasone on NK cells compared to T-cells. Further, we tested the potential ability of IL-15 or tri-specific killer engagers (TriKEs), which have a humanized camelid nanobody CD16, an IL-15 molecule, and a camelid nanobody against B7H3, to overcome immune suppression, and examined the impact of Dexamethasone on primary expanded NK cells derived from healthy individuals (PBNK), as well as adaptive NK cells (NKG2C+CD57+) derived from CMV seropositive individuals. Results: Dexamethasone exposure to NK and T cells did not robustly impact activation or phenotype markers. Functionally, dexamethasone decreases cytokine production (i.e., INF and TNF) in expanded PBNK and Adaptive NK cells, but does not impact degranulation via CD107a, while T-cells show suppression for both degranulation and cytokine production. In this setting, TriKE partially improves the function of NK cells, compared to controls exposed to dexamethasone. Increasing IL-15 concentrations partially improved functionality. Real-time imaging assays showed that dexamethasone slowed the killing kinetics for Adaptive NKs, and TriKE restore killing kinetics in this setting. Furthermore, cell proliferation assays demonstrated that dexamethasone impairs cell proliferation of expanded PBNKs, which is abrogated by TriKE. Overall, our data shows that NK cells can maintain partial functionality when exposed to dexamethasone, improved with TriKE, which suggests clinical potential for NK cells for GBM. Conclusions: Future experiments will seek to repeat these experiments and dive deeper into the impact of Dexamethasone with the complex tumor microenvironment (e.g., hypoxia, TGF-Î²). Additionally, future directions can also include specific testing using tumors isolated from GBM patient samples.

Austin Hoeg Kavisha Shah Ellen Gessford Elizabeth Case	*Improving Variation in the Pediatric Echocardiography Lab* Background: Transthoracic echocardiography (TTE) is the primary diagnostic tool for congenital heart disease. Because of how nuanced cardiac anomalies may present, eliciting accurate views and proper technique are essential for detailed assessment and treatment. As there has been significant growth in the University of Minnesota's congenital heart program and echocardiography lab, establishing protocols is critical to maintain the completeness and quality of the studies and reduce variability. Methods: Pediatric echocardiography technologists' workflow was observed while performing two separate TTEs, and areas of process improvement were identified. The measured areas of improvement were used to establish guidelines for TTE protocol intervention. A minimum of three pre-intervention and fifteen post-intervention echocardiograms from each technologist were assessed for image quality and exam completeness. After data were collected, one-way ANOVA and paired t-tests were performed to investigate variability across technologists and explore differences between quality and completeness. Results: In the analysis of the pre- and post-intervention data, there were statistically significant increases in total image quality (83% to 85%; $p<0.02$) and exam completeness (77% to 86%; $p<0.0001$). R-Squared values also decreased for image quality (0.86 to 0.67) and exam completeness (0.75 to 0.53), suggesting that the protocol may have reduced variation across different technologists. Additionally, F-values increased for image quality (15.84 to 18.14) and exam completeness (7.94 to 9.68), indicating that the protocol may have reduced variation within individual technologists. Conclusion: Establishing a standardized protocol was vital to reduce the variability of study completeness and quality in our pediatric echocardiography lab. These improvements will lead to more accurate diagnosis and treatment and improve patient care by preventing the need for repeat imaging and unnecessary procedures. In the next phase of this project, we will refine the pediatric TEE order to designate exact indications across study requisitions and establish protocols for specific congenital heart lesions.
Clinical Vignette - Medical Students	
Katie McLaughlin **William Miller** Dr. Christopoher Van Hove Morgan Kelly Nicholas Boettcher Dr. Amy Holbrook **Winning Abstract**	*US-Guided Ethanol Ablation of an Insulinoma in a 43-year-old Woman with Refractory Hypoglycemia Non-Responsive to Pharmacological Intervention and Non-Suitable for Surgery* Introduction: Insulinomas are rare neuroendocrine secretory tumors, for which the definitive treatment modality is surgical resection. However, diagnosis is commonly delayed as the symptoms of elevated circulating insulin and hypoglycemia are nonspecific. In this case, despite 8 years of documented recurrent hypoglycemic episodes, normal A1C, elevated proinsulin, and worsening morbid obesity, the patient did not receive a definitive diagnosis or appropriate therapy. This resulted in worsening comorbidities, specifically worsening obesity and increasing debility, rendering her a non-candidate for surgical intervention. Case Presentation: A 43-year-old female with a medical history of hypoglycemia, morbid obesity (BMI 62.54), chronic opioid use for knee pain

	and Crohn's disease presented to the emergency department with symptomatic hypoglycemia involving altered mental status, tremors, diaphoresis, vision changes and incontinence. Labs were remarkable for elevated serum insulin 768, C-peptide 27.90 and proinsulin 448.9. Endoscopic ultrasound (EUS) with fine needle aspiration (FNA) identified a 30 mm x 25 mm mass in the uncinate process of the pancreas, which was confirmed to be a neuroendocrine tumor staged at T2N0M0 (Stage 2). Upon chart review, the patient had symptomatic hypoglycemia 8 years prior to admission. Outpatient workup showed fasting blood glucose 65, HgbA1C 5.2% and proinsulin 142.8. Patient met with endocrinology; however, there was no further workup or definitive cause for her hypoglycemia. Patient self-treated symptoms with high oral intake of sugar, resulting in a 200lb weight gain. She continued to monitor home blood glucose regularly and noted multiple checks in the 50s leading up to admission. Due to significant surgical risk posed by morbid obesity, the interdisciplinary tumor board decided to initiate pharmacologic treatment with diazoxide, octreotide, and everolimus. A prolonged hospitalization of more than one month followed, with multiple complications including profound hypercapnic respiratory failure, severe drug-related rash, and thrombocytopenia. The patient remained dependent on continuous D10 infusion with multiple failed attempts at weaning, even with several weeks of pharmacologic treatment. The care team decided to pursue EUS-guided ethanol ablation as an alternative treatment option. The then 35 mm x 35 mm tumor was successfully injected with 6.5 mL of 98% alcohol for tumor destruction. The patient had no further episodes of hypoglycemia during the hospitalization and was discharged with outpatient follow-ups with a multidisciplinary team of gastroenterology, endocrinology and a referral to bariatric surgery. Discussion: With a tumor resulting in refractory hypoglycemia despite treatment with continuous dextrose infusion, diazoxide, long- and short-acting octreotide, and Everolimus, the EUS-guided ethanol ablation was critical in the management of insulinoma in this patient. This case highlights the importance of an interdisciplinary approach to caring for medically complex individuals as well as highlights the importance of considering EUS-guided antitumoral therapies for the treatment of pancreatic masses. To our knowledge, this case is the largest insulinoma to be treated successfully by EUS-guided ethanol ablation.
Emmanuel Fale Dr. Jessica Hane Dr. Hemin Lee Benjamin Shoults	*From Foraging to Liver Failure: A Case of Amanita Mushroom Poisoning* Introduction: Amanita toxin or amatoxin is a toxin found in mushrooms responsible for 95% of fatal mushroom poisoning cases. Exposure is not infrequently faced in hospitals or poison control centers, but guidelines for management of patients in acute liver failure from amatoxin poisoning, who are not candidates for transplantation, are not well-known. Case Presentation: A 74 year old male with a past medical history of Coronary Artery Disease (CAD), Type II diabetes, Renal Cell Carcinoma 11 years post nephrectomy, and stage II Chronic Kidney Disease presented to the ED with complaints of right upper quadrant pain, vomiting, and diarrhea approximately six hours after ingestion of mushrooms harvested from lawn. On examination the patient was afebrile, tachycardic, and hypertensive.

	Physical exam findings demonstrated a patient alert and oriented to self time and place noted to have extreme tenderness to light palpation along the epigastric area with right upper quadrant pain being greater than left upper quadrant, warm dry skin, and ant-icteric sclera. Labs were significant for elevated anion gap of 20 mmol/L, normal BUN of 22 mg/dl, and creatinine of 1.42 mg/dl at baseline of 1.2 - 1.40 mg/dl. Additionally, on presentation, AST and ALT levels were 43 U/L and 33 U/L respectively. Initial treatment in the external emergency room included opioid pain control, antiemetics, and close outpatient follow-up. 48 hours after initial ingestion, patient developed worsening abdominal pain, nausea and vomiting. Follow up labs demonstrated an anion gap of 19 mmol/L, BUN of 49.6 mg/dl, creatinine of 2.66 mg/dl, AST greater than 7000 U/L, and an ALT of 5,598 U/L. Ultrasound and CT imaging of the abdomen and pelvis showed evidence of fatty liver but were negative for acute hepatic and gallbladder abnormalities. With history, labs/imaging results, and physical exam findings, a high suspicion for poisoning secondary to unknown source was suspected and IV established for aggressive rehydration with 1500 cc over the following 60 to 120 minutes, IV N-Acetylcysteine, and zofran for nausea. Poison control consulted with photos of suspected poisoning agent and stipulated that liver transplant is required given the high mortality rate associated with patients who present similarly. Patient was not a candidate d/t age, extensive coronary artery disease, previous renal cell carcinoma, and TIID. Therefore, treatment continued with IV N-Acetylcysteine and IV Silibinin while monitoring liver function tests BID until AST and ALT were less than 1000 following poison control consultation. Treatment course occurred from 08/31 - 09/05, discontinued due to ALT being 624 U/L and AST being 51 U/L. Discussion: This case illustrates a typical presentation of amatoxin poisoning with 3 stages. Additionally, Current indications for transplantation are rapid progression to hepatic encephalopathy, hepatorenal syndrome, or coagulopathy. Otherwise, treatment is primarily supportive with a combination of IV N-acetylcysteine and Silibinin, and activated charcoal. While liver transplant is the primary indication in this population, continuous administration of IV silibinin and N-acetylcysteine, until ALT and AST levels are below 1000, HD followed by 48 hours of high dose CRRT with oxins administration should be investigated further.
Stefan Farrugia Dr. Mark Enzler Dr. Richard Silbert **People's Choice Award Winner**	*Fibrin Sheath Following Dialysis Catheter Removal: Difficult to Manage Infectious Source* Introduction: A known complication of indwelling central venous catheters is the formation of fibrin sheaths. They are formed by endothelial cell damage, leading to recognition of the catheter as a foreign body and creation of an overlying layer composed of fibrinogen, albumin, gamma-globulin, lipoproteins, and coagulation factors. Fibrin sheaths can remain after the removal of catheters and can serve as a nidus for infection. There is a paucity of published literature guiding management of retained fibrin sheaths. Case Presentation: We present the case of a 79-year-old female who presented to the emergency department from her nursing home with confusion, shortness of breath, nausea, and pain at her tunneled right internal jugular vein dialysis catheter site. Tenderness and purulence were noted at her catheter site. Both peripheral blood cultures and cultures from her catheter grew methicillin-resistant Staphylococcus aureus (MRSA). She was started on vancomycin and her infected hemodialysis catheter was removed.

	Transesophageal echocardiogram (TEE) was performed to evaluate for endocarditis. While no indwelling central venous catheters were in the patient at the time of TEE, she was noted to have an echodensity resembling a catheter at the superior vena cava - right atrium junction. This was felt to be a fibrin sheath retained after catheter removal. There was concern that the open end of the fibrin sheath could harbor bacterial vegetations that would not sufficiently be treated with antibiotics alone without further source control. Cardiovascular surgery, interventional cardiology, and interventional radiology were consulted and recommended observation rather than surgery or endovascular removal. She was discharged on IV vancomycin and anticoagulation. Follow-up TEE performed three months later showed the persistence of a mobile echodensity in the superior vena cava that appeared less mobile and was no longer protruding into the right atrium. Patient had no recurrence of MRSA bacteremia but unfortunately had a repeat episode of line-associated bacteremia attributed to methicillin-susceptible Staphylococcus aureus. Discussion: Blood stream infections occur in up to 70% of patients with central venous catheters. The presence of a fibrin sheath increases the odds of catheter-related bacteremia by over 60%. There is limited evidence to guide management of fibrin sheaths as infectious sources after catheter removal. While lytic enzymes and fibrin sheath stripping are employed to remove sheaths on indwelling catheters, these are costly options, and in the case of stripping, invasive. In the prevention of forming a fibrin sheath, anticoagulation does not have a significant benefit. However, once a fibrin sheath forms, the goal of anticoagulation is to aid the breakdown of the sheath and prevent complications from the clot. This is especially important in this patient's case to allow for antibiotic access to the interior of the sheath. The choice of antibiotic therapy and duration of treatment for this unique infectious scenario has limited reported evidence and should be managed on a case-by-case basis. Management should be guided by a multidisciplinary team including cardiology, infectious diseases, and interventional radiology.
Hamza Hai Pouria Pourzand Mithun Suresh	*Flecainide Toxicity Occurring in The Setting of Acute on Chronic Kidney Disease* Case Presentation: A 59-year-old male patient with a complex medical history, including heart failure with preserved ejection fraction, tachycardia-bradycardia syndrome, paroxysmal atrial fibrillation managed with a dual-chamber pacemaker, and ongoing maintenance therapy with flecainide, as well as stage 3b chronic kidney disease, presented with symptoms of dyspnea and orthopnea. Initial laboratory findings revealed elevated creatinine at 5.2 mg/dL (compared to a baseline of 2.0 mg/dL) and a markedly elevated B-type natriuretic peptide (BNP) of 885 pg/mL (normal range < 100 pg/mL). A subsequent chest X-ray demonstrated bilateral pleural effusions and pulmonary edema. Consequently, the patient was admitted with a diagnosis of acute decompensated heart failure compounded by acute-on-chronic kidney disease, and a diuretic infusion was initiated. Later in the day, follow-up blood testing indicated a worsening of renal function, and this was accompanied by oliguria. The patient also started to experience sustained episodes of a wide QRS complex rhythm with prolonged PR intervals, raising concerns of ventricular tachycardia. Concurrently, the patient reported lightheadedness during these arrhythmias. Intriguingly, pacemaker

	interrogation revealed normal device function and notably, did not detect any arrhythmias. Cardiology was consulted, and they expressed concern regarding flecainide toxicity as the potential cause. Flecainide was promptly discontinued, and a cautious sodium bicarbonate infusion was initiated after considering the patient's volume-overloaded state. The patient remained hospitalized for several additional days, during which the arrhythmias gradually resolved, and his overall clinical condition improved. Upon discharge, it was decided to permanently discontinue flecainide. Discussion: Flecainide is a class Ic anti-arrhythmic medication used to manage both supraventricular and ventricular arrhythmias. Its mechanism of action involves blocking myocardial sodium channels, resulting in a reduced rate of cardiac myocyte depolarization and slowed conduction within the cardiac conduction system. Electrocardiographically, flecainide may lead to widened QRS complexes and prolonged PR intervals. As the dosage increases, it can also cause bradycardia and heart block (first- and second-degree). In patients with impaired renal function, flecainide can accumulate, elevating the risk of flecainide toxicity, particularly in those with chronic kidney disease. This toxicity can manifest as an increase in ventricular arrhythmias. Common symptoms include dizziness or blurred vision. In addition to symptom control, treatment of flecainide toxicity consists of medical management with sodium bicarbonate, which is the first-line strategy due to its ability to block flecainide binding and facilitate dissociation from sodium channel receptor binding sites. In severe cases, adjunctive strategies may include intravenous lipid emulsions and mechanical circulatory support. Conclusions: Hospitalists should maintain vigilance for flecainide toxicity when caring for patients taking this medication, especially those with chronic kidney disease. The possibility of flecainide toxicity should be considered if patients develop wide QRS complex arrhythmias alongside acutely worsening renal function. Prompt recognition and management of flecainide toxicity are crucial to prevent further complications and improve overall patient outcomes. Information related to this case may have been changed per institutional policy.
Madi Sundlof Dr. Jacob Kohlenberg	*Hypoglycemia Challenges in Cancer: Exploring Nonislet Cell Tumor Hypoglycemia* Introduction: Nonislet Cell Tumor Hypoglycemia (NICTH) is a rare syndrome resulting in hypoglycemia. NICTH is most commonly a result of tumor production of incompletely processed insulin-like growth factor-2 (IGF-2), leading to increased glucose utilization and inhibition of glycogenolysis and gluconeogenesis. The tumor may also replace hepatic parenchyma, reducing hepatic glycogen reserves. Case Description: A 34-year-old man presented with recurrent episodes of low glucose and confusion. His medical history includes solitary fibrous tumor of the brain with diffuse pulmonary, osseous, and hepatic metastases. He underwent two resections of the primary parietal brain mass in 2015, gamma knife radiation as well as embolization of liver lesions in 2019, and stereotactic body radiotherapy and chemotherapy in 2020 due to liver mass enlargement. Due to disease progression, he subsequently received multiple lines of systemic therapy. His BMI was 20.54 kg/m2 and he appeared cachectic. Physical exam was significant for bilateral upper and lower extremity muscle wasting and

	hepatomegaly. On admission, his venous glucose was 29 mg/dL. His IGF-1 and IGF-2 were measured at 41 (Reference Range [RR] 82-242 ng/mL) and 84 (RR 180-580 ng/mL), respectively. Beta-hydroxybutyrate (BHB) was 0.39 (RR 0.27 mmol/L) with a negative hypoglycemic agent screen for sulfonylureas and meglitinides. Insulin, proinsulin, insulin antibody, and C-peptide levels were undetectable. ACTH stimulation was normal. Following administration of glucagon, his plasma glucose increased by >35 mg/dL and his confusion resolved. Abdominal imaging confirmed extensive hepatic metastases. His presentation was consistent with NICTH with multifactorial hypoglycemia resulting from both tumoral IGF-2 secretion and hepatic metastases. He was not a candidate for debulking of his hepatic metastases. To mitigate hypoglycemia he treated with prednisolone 25 mg BID. Despite high-dose glucocorticoid therapy, glucagon, calorie intake every two hours, and continuous glucose monitoring with real-time alerts, recurrent hypoglycemia persisted. Outside of recombinant growth hormone, which can stimulate further cancer proliferation, therapeutic options to mitigate hypoglycemia in NICTH are limited. Based on the mechanism of his hypoglycemia, an off-label trial of Alpelisib was initiated and titrated to 100 mg daily. Alpelisib, a kinase inhibitor of phosphatidylinositol-3-kinase (PI3K), frequently leads to hyperglycemia by directly interfering with the insulin signal transduction pathway. When insulin binds its receptor, activation of PI3K, ultimately facilitates the movement of GLUT 4-containing vesicles to the cell membrane. Alpelisib was well tolerated and the frequency of severe hypoglycemia improved. Discussion: The diagnosis of hypoglycemia requires fulfillment of Whipple's triad: neuroglycopenic symptoms, laboratory-confirmed low plasma glucose, and resolution of neuroglycopenic symptoms upon raising plasma glucose. The diagnosis of NICTH relies on this documentation, alongside clinical and biochemical findings. Laboratory evaluation should indicate suppressed BHB, C-peptide, and insulin at the time of hypoglycemia. Plasma glucose response to glucagon is typically >25 mg/dL. Elevated IGF-2/IGF-1 ratio, though common, is not mandatory for diagnosis. Treatment options include treatment of primary disease, glucocorticoids, glucagon, or growth hormone. Alpelisib was chosen in this case as a novel off-label use alternative due to recurrent hypoglycemia refractory to conventional approaches.
Quality Improvement - Medical Students	
Austin Hoeg Kavisha Shah Ellen Gessfor Elizabeth Case	*Improving Variation in the Pediatric Echocardiography Lab* Background: Transthoracic echocardiography (TTE) is the primary diagnostic tool for congenital heart disease. Because of how nuanced cardiac anomalies may present, eliciting accurate views and proper technique are essential for detailed assessment and treatment. As there has been significant growth in the University of Minnesota's congenital heart program and echocardiography lab, establishing protocols is critical to maintain the completeness and quality of the studies and reduce variability. Methods: Pediatric echocardiography technologists' workflow was observed while performing two separate TTEs, and areas of process improvement were identified. The measured areas of improvement were used to establish guidelines for TTE protocol intervention. A minimum of three pre-intervention and fifteen post-intervention echocardiograms from each technologist were assessed for image quality and exam completeness. After

	data were collected, one-way ANOVA and paired t-tests were performed to investigate variability across technologists and explore differences between quality and completeness. Results: In the analysis of the pre- and post-intervention data, there were statistically significant increases in total image quality (83% to 85%; $p<0.02$) and exam completeness (77% to 86%; $p<0.0001$). R-Squared values also decreased for image quality (0.86 to 0.67) and exam completeness (0.75 to 0.53), suggesting that the protocol may have reduced variation across different technologists. Additionally, F-values increased for image quality (15.84 to 18.14) and exam completeness (7.94 to 9.68), indicating that the protocol may have reduced variation within individual technologists. Conclusion: Establishing a standardized protocol was vital to reduce the variability of study completeness and quality in our pediatric echocardiography lab. These improvements will lead to more accurate diagnosis and treatment and improve patient care by preventing the need for repeat imaging and unnecessary procedures. In the next phase of this project, we will refine the pediatric TEE order to designate exact indications across study requisitions and establish protocols for specific congenital heart lesions.
Transitional Medical Graduates	
Research – Medical Transitional Graduates	
Max Guarda Andrew Hanson Hannah Langenfeld Cynthia Crowson Jigisha Rakholiya Cristian Labarca Cornelia Weyand Kenneth Warrington Matthew Koster **Winning Abstract People's Choice Award**	*Concordance of First Relapse Symptoms and Second Relapse Symptoms with Initial Baseline Presentation Features among Patients with Giant Cell Arteritis: An Analysis and Comparison of Four Patient Cohorts* Introduction: Relapse is common in patients with giant cell arteritis (GCA). The frequency of relapse has been repeatedly evaluated at a cohort level, but individual patient level assessment of the concordance of symptoms between diagnosis and relapse remains unknown. The purpose of this study was to evaluate GCA symptom concordance between baseline symptoms to first relapse and first relapse to second relapse in patients with GCA across four patient cohorts. Methods: Three previously described cohorts of patients with GCA were utilized. Cohort 1 (C1) 286 patients with biopsy-proven GCA treated without tocilizumab (TCZ), Cohort 2 (C2) 110 patients with biopsy negative GCA treated without TCZ, and Cohort 3 (C3) 114 patients with biopsy-proven or imaging-proven GCA treated with TCZ. An aggregate of all patients from C1-C3 was also evaluated and termed cohort 4 (C4, n=510). Clinical symptoms at diagnosis and relapse were grouped into five categories: constitutional, musculoskeletal (MSK), cranial (non-visual), visual, and large vessel (LV). Patients could have more than one category present at each time point. Odds ratios and conditional probabilities were calculated. Concordance was evaluated between baseline to first relapse and first relapse to second relapse. Results: In C1, C2, C3, and C4, 183/286 (64%), 62/110 (56%), 58/114 (51%), and 303/510 (59%) patients had at least one relapse, respectively. In C4, the merged cohort, 20% of patients reported a new symptom category at first relapse that was absent at baseline. In C4, at first relapse 67% of patients

	had symptoms in a single category, versus 10% at baseline. In C1, presence of LV symptoms at baseline resulted in a 48-fold increased risk of LV symptoms at first relapse. In C4, presence of LV symptoms at baseline resulted in a 25-fold increased risk of LV symptoms at first relapse and a 15-fold increased risk of developing LV symptoms at second relapse if LV symptoms were present at first relapse. In C3 (TCZ), the conditional probability of developing LV relapse with baseline LV involvement was 29%, versus 48%, 57%, and 44% in C1, C2, and C4, respectively. In C4, presence of visual symptoms at baseline resulted in a 15-fold increased risk of developing visual symptoms at first relapse and a 30-fold increased risk of developing visual symptoms at second relapse if present at first relapse. In C1 and C3 no patient without visual symptoms at baseline developed visual symptoms at first relapse. Conclusion: In our study, approximately 1 in 5 patients reported a new symptom domain at relapse that was not present at baseline. Visual relapses were overall uncommon, but risk was high on second relapse if present at first relapse. The likelihood of LV symptoms at relapse among patients with LV involvement at baseline was comparatively lower among patients on TCZ. The significance of this study lies in its potential to aid clinicians in providing patient-level data regarding relapsing GCA that will ultimately translate into better patient education, increased awareness of patients regarding what symptoms to look for, and overall better monitoring of the disease.
Tagbo Nduka Abdulkareem Lukan Valentine Nriagu Rukayat Otulana	*Impact of July Effect on In-Hospital Outcomes in Patients Admitted for Atrial Fibrilation* Introduction: Atrial Fibrillation is an abnormal heart rhythm characterized by rapid and irregular beating of the heart's atrial chambers. Hospitals across the United States see a high spike in medical errors due to the influx of new residents, fellows, and attending in July. This is called the July effect. This abstract aims to establish the impact of this on in-hospital outcomes in patients admitted for atrial fibrillation. Methods: We analyzed the National Inpatient Sample (NIS) database for 2018. The NIS was searched for hospitalization of adult patients with Atrial Fibrillation using ICD-10 codes. The exposure was patients admitted in July vs not in July. The outcomes were ventricular arrhythmia, Cardiogenic shock, cardiac arrest, acute kidney injury, cerebrovascular accident, acute congestive heart failure, and length of stay. Multivariate logistic and linear regression analyses were used accordingly to adjust for confounders. Results: Cohorts admitted in July vs. Not in July had the following characteristics, male (48.55% vs. 47.71%), female (51.45% vs. 52.29%), Non-Hispanic white (81.22% vs. 80.93%), Non-Hispanic black (8.39% vs. 8.16%), Hispanic (6.05% vs. 6.07%), Asian and Pacific Islander (1.90%vs1.65%) and Native Americans (2.44% vs. 2.57%), Rural hospital (11.85% vs11.46%), Urban non-teaching hospital (22.82%vs23.32%) and Urban Teaching hospital (65.33%vs65.22%). Compared to patients that were not admitted in July, patients admitted in July had no statistically significant difference in the following outcomes, Ventricular arrhythmia (3.36% vs. 3.12% OR 1.05, CI 0.9-1.24,p-0.48), Cardiogenic shock (0.53% vs. 0.57%, OR 0.91, CI 0.62-1.38,p-0.66), Cardiac arrest (0.36% vs. 0.37%, OR-1.00, CI 0.62-1.63, p-0.98), AKI (12.78% vs. 12.32%, OR-1.05, CI 0.96-1.15, p-0.26), Acute CVA (0.47% vs. 0.58%, OR 0.81, CI 0.53-1.23, p-0.32), Acute CHF

	(11.46% vs. 12.22%, OR 0.93, CI 0.85-1.01,p-0.11), LOS (3.2 days vs 3.2 days, OR -0.25, CI -0.19-0.14, p-0.762) Conclusion: This study has established that there is no statistically significant difference in outcomes in the management of atrial fibrillation in July compared to other months of the year. We should continue to improve the education and training of medical students, residents, and fellows in managing atrial fibrillation to reduce morbidity and mortality further.
Anusha Parisapogu Dr. MJ Binnicker Dr. ES Theel Dr. M Mahmood Dr. MJ Kasten	*Validation of Self-Collected Mail-in Swabs for Sexually Transmitted Infection Testing to Facilitate Virtual Preexposure Prophylaxis* Background: Preexposure prophylaxis (PrEP) is essential in HIV prevention, and adherence to PrEP is vital for its effectiveness. Routine sexually transmitted infection (STI) testing every 3-6 months is recommended for most people on PrEP care. Frequent in-person clinic visits are a deterrent to PrEP adherence. To address this challenge, we aimed to pilot a remote PrEP approach, wherein patients engage with healthcare providers or pharmacists remotely, self-collect STI samples, and reduce the need for face-to-face clinic visits. This approach has been successful at other institutions, increasing the number of patients starting and adhering to PrEP. Prior studies have demonstrated that self-collected vaginal swabs are more sensitive than provider-collected swabs for nucleic acid amplification testing (NAAT) of STIs. We hypothesized that this would also hold for self-collected rectal and pharyngeal swabs. Methods: We enrolled 65 participants to evaluate gonorrhoea and chlamydia NAAT testing using samples collected by trained healthcare providers and self-collected rectal and pharyngeal swabs. Participants were guided through the self-collection process with written and pictorial instructions, enabling them to perform the sample collection independently. Concurrently, healthcare providers collected the same specimens that were submitted for laboratory testing. To determine the acceptability and feasibility of self-collection, we administered a structured questionnaire to a subset of the participants. The questionnaire covered aspects such as ease of self-collection, confidence in the process, comfort levels during self-sampling, and overall interest in utilizing self-collection as a testing method. Results: Among the 65 patients, 46 had self-collected and provider-collected rectal swabs, with 41 (91.1%) showing concordant results. All 65 patients had self-collected and provider-collected pharyngeal swabs, with 63 (96.9%) matched samples yielding concordant results. We observed positive trends regarding self-collected and clinician-collected swabs for rectal and throat specimens submitted for gonorrhea and chlamydia testing. A substantial percentage of participants, 82% for rectal and 90% for throat swabs, found the self-swab instructions easy to follow. Comfort levels were positive, with 55% feeling comfortable or very comfortable during rectal self-swab and 66% during self-collection of a throat swab. Confidence in self-collection was notable, as 88% felt confident or very confident in obtaining appropriate specimens for both rectal and throat samples. A significant majority, 52% and 66%, respectively, expressed feeling confident or very confident in their ability to self-collect specimens at home if provided with instructions, potentially eliminating the need for clinic visits.

	Conclusion: The results of this study demonstrate a strong correlation between self-collected and provider-collected swabs for STI testing. Overall, self-collection exhibited higher sensitivity and demonstrated better patient acceptance compared to provider-collected specimens. This validation study supports the feasibility and effectiveness of self-collected STI swabs as a component of remote PrEP adherence monitoring approaches, potentially enhancing patient engagement and reducing the barriers to PrEP.
Clinical Vignette – Transitional Medical Graduates	
Mohamed Eldesouki Hazem Abosheaishaa	*Altered Mental Status on Top of Anaplasmosis-Induced Severe Rhabdomyolysis: A Rare Clinical Presentation* Introduction: Human granulocytic anaplasmosis (HGA) is a disease caused by tick-borne infection of Anaplasma phagocytophilum. The typical symptoms are fever, malaise, and body aches accompanied by abnormal blood tests such as leukopenia, thrombocytopenia, and transaminitis. Some rare complications may occur, especially in patients living in heavily wooded areas, with a mean age of 70 years. Case Presentation: We present a case of a 67-year-old male who was admitted for lower abdominal pain, fever, and diarrhea with derangement of his blood tests. Despite treatment, his condition deteriorated and complicated rhabdomyolysis and acute kidney dysfunction. Discussion: Empiric treatment including doxycycline was initiated while waiting for the infection blood work results. PCR came back positive for HGA. Empiric therapy was narrowed down to doxycycline for 14 days, and the patient's condition began to improve gradually and steadily. Aggressive hydration markedly improved rhabdomyolysis and, in turn, kidney function. Conclusion: Our case underscores the importance of considering HGA in ambiguous clinical scenarios and highlights the value of early diagnosis, empiric treatment, and intravenous hydration, especially in the presence of rhabdomyolysis.
Pouria Pourzand Hamza Hai Mithun Suresh	*Desmoplakin Cardiomyopathy Diagnosed after an Episode of Myocarditis* Case Presentation: A 29-year-old otherwise healthy female presented with left-sided chest pain of six days duration without any associated or prodromal symptoms. ECG showed normal sinus rhythm. On initial evaluation, Troponin I was elevated at 73.6 ng/mL (normal < 0.04 ng/mL), while the rest of the work-up, including blood work and CXR, was unremarkable. The patient was admitted with a presumptive diagnosis of myocarditis. The echocardiogram revealed an ejection fraction of 60%-65% with no wall motion abnormalities and no valvulopathy. CT coronary angiogram further revealed normal coronary arteries with no significant stenoses. Infectious work-up, including SARS-CoV-2 testing, was negative. Throughout admission, her symptoms improved, and the patient was discharged on hospital day 4. Cardiac MRI could not be initially obtained due to technical difficulties but was performed early after discharge and demonstrated no evidence of myocardial edema or inflammation. At follow-up six weeks later, the patient reported no recurrence of symptoms; however, repeat Troponin I was higher at 75.9 ng/mL. Repeat cardiac MRI revealed a focal area of increased myocardial edema and inflammation and dense epicardial delayed enhancement involving the basal inferior wall consistent with acute

	myocarditis; colchicine and losartan were initiated. Genetic testing later revealed a pathogenic nonsense mutation in the desmoplakin (DSP) gene, supporting a diagnosis of DSP cardiomyopathy. The patient was referred for ICD implantation and since then, has been asymptomatic. Discussion: DSP cardiomyopathy is a rare autosomal dominant condition caused by mutations in the DESM gene, encoding the DSP protein that anchors intracellular filaments, leading to abnormal DSP function. Notably, this condition is associated with myocarditis and frequently, is diagnosed soon after an episode of myocarditis like in our case. Accordingly, evaluation for this condition typically begins with the same workup performed for myocarditis. Initial symptoms may only consist of mild chest pain, but some patients may present with decompensated heart failure. A basic work-up typically includes ECG, cardiac enzymes, chest imaging, and blood work to assess for inflammatory markers such as white blood cell count, c-reactive protein, and erythrocyte sedimentation rate. An echocardiogram is commonly performed to evaluate for reductions in left ventricular function and cardiac MRI may subsequently be performed to assess for myocardial edema, inflammation, necrosis, and scarring. With the broader availability of genetic testing, DSP cardiomyopathy is increasingly recognized as a cause of myocarditis. However, there is little data on the management of this cardiomyopathy, but it usually consists of supportive care for the myocarditis episode with traditional pharmacological therapies and consideration of ICD implantation due to the fibrosis of the left ventricle, which can occur and lead to dangerous arrhythmias with this cardiomyopathy. Conclusions: Herein, we reported a case of myocarditis secondary to DSP cardiomyopathy, a rare condition diagnosed in this patient following a recent episode of myocarditis. Given the genetic basis of this condition, genetic testing is essential to appropriately diagnose such patients and provide the necessary counseling. Information related to this case may have been changed per institutional policy.
Residents	
Quality Improvement - Residents	
Mackenzie Maki MD Dr. Sarah Prebil	*Right Patient, Right Procedure and the Right Orders* Medical errors cost approximately $20 billion dollars a year to the U.S. healthcare system. A substantial portion - around 90% - of these errors can be attributed to systems failures and inadequacies in procedures. This case centers on a patient with presumed spinal osteomyelitis. All biospecimen orders necessary for diagnosis were not placed prior to the procedure, necessitating a repeat vertebral bone biopsy. A root cause analysis was performed revealing that providers lacked clarity for who is responsible for entering biospecimen orders. This is particularly problematic when multiple specialties are involved. Additionally, it was found that the majority of radiology-guided procedures occur without biospecimen orders in place at the start of the procedure. Standardized order sets have been shown to improve patient safety, ensure clarity in medical orders, and enhance workflow. They can also ‘nudge’ providers to do the right thing, especially when ordering something infrequently. Multiple standardized order sets were created for radiology-guided procedures. Order sets combined the radiology procedure with the possible biospecimen tests needed. This will help

	accomplish the goal of improving patient safety and promoting cost-effectiveness within the healthcare system by decreasing the need for repeat procedures.
Research - Residents	
Abdulsabur Sanni MD **Winning Abstract**	*COVID-19 Mortality and Outcomes in Hospitalized Patients with Sickle Cell Disease: A Retrospective Cohort Study using the National Inpatient Sample Databse (NIS)* Purpose: To investigate the mortality and outcomes of COVID-19-infected adult patients with sickle cell disease in 2020. Methods: Retrospective cohort study utilizing the Nationwide Inpatient Sample (NIS) database to select patients with a principal ICD-10-CM diagnosis of COVID-19. The sample consisted of 535 patients with sickle cell disease and 1,050,720 patients without. A P value of 0.05 was considered significant in this study. Results: Patients with sickle cell disease were more likely to be female, younger in age, had lower comorbidities at baseline, and were predominantly Black. In terms of outcomes, the total in-hospital mortality rate for COVID-19 patients was 11.1% in 2020, of which 0.02% had sickle cell disease. Patients with sickle cell disease had a 61% decrease in the odds of mortality compared to those without sickle cell disease, which remained insignificant after adjusting for confounders (OR 0.780 {95 % CI 0.321-1.899} p=0.586). There was no significant difference in the intubation rate between the two groups after adjusting for age, gender, and comorbidities. Furthermore, patients with sickle cell disease had a shorter length of stay compared to COVID-19 patients without sickle cell disease. Conclusions: Compared to COVID-19 patients without sickle cell disease, sickle cell disease does not increase the odds of mortality in hospitalized patients with COVID-19. Based on our data, the values obtained were statistically insignificant. Clinical Implications: Sickle cell disease although known to have pulmonary complications like acute chest syndrome, we cannot confidently say that sickle cell disease does not increase the mortality of patients admitted for COVID-19 pneumonia in the year 2020.
Maroun Chedid	*Effectiveness of Temporary Pacemakers with Active Fixation Compared to Conventional Balloon-Tipped Temporary Pacemakers* Background: Conventional Temporary Pacemakers (CTPM) are the most used temporary pacemakers; however, they are associated with a risk of dislodgment and thromboembolism. Recently, Temporary Permanent Pacemakers (TPPM) have become increasingly used. Evidence of better outcomes with TPPM compared to CTPM is still scarce. Methods: Data of patients who underwent TPPM or CTPM (n=61 each) was retrieved. Our primary endpoint was a composite of in-hospital death from cardiovascular causes, thromboembolism, and new in-hospital atrial/ventricular arrhythmias. Results: Males accounted for 54.1% of TPPM patients and 50.8% of CTPM

	patients. Most patients in were Caucasians (60.7% in TPPM vs 65.0% in CTPM), and the mean age at pacemaker insertion was 66.6 and 70.9 years, respectively. The most common indication for pacemaker in both groups was atrioventricular node dysfunction. The subclavian vein was the most common site of access in patients with TPPM (88.5%), femoral access was mostly used for CTPM (60.7% vs 0% in TPPM). Ambulation was possible only in patients with TPPM (57.4%, $p<0.01$). Lead dislodgement and/or loss of capture happened more frequently in the CTPM group (24.6 vs 8.2%, $p=0.01$). The study primary outcome occurred less in the TPPM group (OR= 0.05, $p<0.01$) (table 1). The median (min-max) number of days paced outside the ICU was 2 (0-10) in the TPPM group vs zero in the CTPM group ($p<0.01$). Conclusion: TPPM are associated with more favourable outcomes as well as a shorter ICU pacing duration compared to CTPM.
Clinical Vignette- Residents	
Ionnis Kournoutas, MD Dr. Joelle Friesen Dr. Adam Sawatsky **Abstract Winner**	*Skincredible Findings: A Case of Paraneoplastic Dermatomyositis* Case Presentation: A 66-year-old woman with medical comorbidities significant for metastatic ovarian cancer status post total abdominal hysterectomy with bilateral salpingo-oophorectomy and actively receiving chemotherapy with carboplatin, paclitaxel, and bevacizumab was admitted for generalized weakness. Four weeks prior to presentation, she developed a diffuse erythematous rash involving the bilateral upper extremities, trunk, and hips. She was otherwise asymptomatic and had no dermatologic or autoimmune history. The rash was presumed to be toxic erythema of chemotherapy and steroids were prescribed. During the subsequent weeks the rash improved significantly, however, the patient developed new dysphagia and progressive weakness. On admission, the physical exam was notable for proximal extremity weakness in the bilateral shoulders and hips. Laboratory studies were significant for a creatinine kinase (CK) of 2,662 (reference range: 26-192) and lactic acid of 3.1 (reference range: 0.5-2.2); vitals were notable for tachycardia. The patient was started on IV fluids and empiric antibiotics due to concern for sepsis and was admitted. Over the next three days her clinical status improved; infectious workup was unremarkable and antibiotics were de-escalated. However, the CK continued to uptrend and significant proximal weakness persisted. Rheumatology was consulted due to concern for an autoimmune process. Electromyography demonstrated diffuse myopathy proximal greater than distale consistent with myositis. Review of the images from the initial encounter revealed a rash that was morphologically consistent with the shawl sign, V-sign,and holster sign. Furthermore, rheumatologic investigation was significant for a positive speckled antinuclear antibody (1:320), positive Ro52/Ro60 IgG antibodies, and an elevated aldolase at 17.4 (reference range: <7.7). Review of a skin biopsy obtained at initial outpatient presentation demonstrated vacuolar interface changes with apoptotic keratinocytes and dermal melanophages, consistent with an inflammatory myositis. A diagnosis of paraneoplastic dermatomyositis was made. A regimen of IVIG and oral

	prednisone was initiated and over the next 6 months she gradually improved, ultimately returning to baseline strength with complete resolution of dysphagia. Conclusion: Dermatomyositis (DM) is an autoimmune inflammatory myopathy that is characterized by distinct cutaneous manifestations that often precede muscle weakness; the latter is only found on presentation in 50-60% of cases. When present, the distribution of muscle weakness is symmetric and affects proximal musculature. Additionally, as seen in our patient, when the striated muscle of the proximal esophagus is involved, patients may present with dysphagia. Other less common clinical features include interstitial lung disease and cardiac involvement (e.g., myocarditis). While DM is often idiopathic, there is a known association with underlying malignancy; the cancer may occur before, simultaneously with, or after the onset of DM. If not present at time of DM diagnosis, investigation of possible occult malignancy is warranted. Classic laboratory findings, as seen in our patient, include elevated CK, aldolase, and myositis-associated autoantibodies. Initial therapy involves treatment with glucocorticoids; however, in patients with refractory disease or high-risk features such as the presence of dysphagia or associated malignancy, additional treatment with IVIG, methotrexate, or azathioprine may be considered. Once remission of DM is achieved immunosuppressive medications are typically tapered and the patient is monitored closely for recurrence.
Abdallah Nadhem MD Dr. Mark Linzer Dr. Abdilahi Mohamoud	*Things to Think about Before Removing a Tricuspid Valve* Case Presentation: We present a case of a 29-year-old male with a past medical history of IV drug use and recurrent tricuspid endocarditis status post two TV replacements who was admitted to the ICU at HCMC for acute hypoxic respiratory failure after suspected heroin overdose. Post intubation the patient was found to be volume overloaded secondary to severe tricuspid stenosis due to recurrent vegetations and subsequently developed MRSA bacteremia. CTS was consulted and deferred a redo valve replacement as he was not an appropriate operative candidate due to his ongoing active IV drug use. Following aggressive diuresis, he was extubated and despite ongoing antibiotic therapy he developed recurrent bacteremia with cultures growing multiple organisms including mycobacterium abscessus. This was suspected to be secondary to PICC line infection which seeded to the TV and despite broad spectrum antimicrobial therapy he continued to have persistent hypoxia due to persistent valvular disease and a right to left shunt from a PFO. Given that his hypoxia would not improve without valve intervention, CTS recommended a valvectomy procedure followed by an eventual bioprosthetic valve replacement given maintenance of sobriety. Post valvectomy he was transferred back to HCMC for ongoing antibiotic therapy. However, despite his bacteremia resolving he continued to have worsening right sided heart failure and subsequently developed right brachial artery thrombus and a DVT in his right internal jugular vein. He was then started on apixaban for a 3-month period yet continued to develop worsening left upper extremety swelling with repeated upper limb ultrasounds remaining unchanged from his previous ones. Due to his unresolving nonocclusive DVTs in his left brachial and right internal jugular veins the approach to his anticoagulation was modified two months later and he was transitioned to enoxaparin with a plan to bridge warfarin. Hypercoagulability workup by hematology at that time was negative and the patient was kept on warfarin achieving a therapeutic INR.

	Unfortunately, weeks after starting his warfarin he continued to develop new clots in his upper limb veins. More workup revealed warfarin failure likely secondary to chronic liver congestion from severe tricuspid regurge and as such he was transitioned back to subcutaneous enoxaparin, eventually leading to a resolution of his non occlusive venous and arterial upper limb thrombi. After six months without IVDU he underwent a repeat tricuspid valve replacement with a bioprosthetic valve. Conclusion: This case challenges tricuspid valvectomy patient safety while on oral anticoagulants promoting awareness about the condition and why treatment failure should be on the differential diagnosis of patients presenting with recurrent upper limb DVTs even if they are anti-coagulated in what seems to be an appropriate manner.
Dzhalal Agakishiev MD Dr. Stephen John Dr. M. Colin Turner	*Medication Roulette: GLP1 Shortage Leading to Severe Acute Kidney Injury* Case Presentation: The case involves a 74-year-old female patient with a complex medical history, including Type 2 Diabetes Mellitus, Coronary Artery Disease, Hypertension, and Obstructive Sleep Apnea. She presented with symptoms of nausea and malaise, which manifested shortly after transitioning her GLP-1 agonist to high-dose semaglutide. Her symptoms included nausea, vomiting, non-bloody diarrhea, loss of appetite, left-sided chest pain, decreased urination, weakness, and dizziness upon standing. While her physical examination revealed no abnormalities in her cardiac, pulmonary, or musculoskeletal systems, she displayed intermittent jerking movements in her upper extremities. Laboratory findings indicated a new elevation in creatinine to 6.88 with prior baseline of 1, necessitating prompt intervention, including IV fluids and hemodialysis. Subsequently, her creatinine improved to 1.5, leading to discharge. Focused Diabetes History: She was initially diagnosed with T2DM over 2 decades ago. Her disease was complicated by retinopathy, neuropathy, nephropathy and CAD. Her A1c ranged from 8-9% . She was briefly on empagliflozin 10 mg but this was stopped due to inadequate glycemic control and recurrent UTIs. She was switched to GLP-1 agonists for her T2DM; the actual medication changed depending on what is available from her pharmacy. Briefly, she was on semaglutide (1mg weekly) â†' dilaglutide (0.75mg weekly) â†' liraglutide (1.8mg daily) â†' semaglutide (2 mg weekly; started 1 day prior to admission). Glucagon-Like Peptide-1 (GLP-1) agonists have gained prominence in Type 2 Diabetes management in recent years, offering modest A1C reduction but remarkable weight loss benefits, comparable to surgical interventions. This heightened interest in GLP-1 agonists, fueled by celebrity endorsements and their adoption for non-diabetic weight loss, has led to substantial demand and medication shortages. Notably, individuals with means have sought these medications for weight loss purposes, potentially exacerbating the scarcity for diabetic patients. Conclusion: Although adverse effects associated with GLP-1 agonists are shared within the class, distinctions in incidence rates, especially between oral and subcutaneous formulations, exist. Exenatide, for instance, has exhibited the highest risk of renal complications. Notably, limited data exists on the association between specific GLP-1 agonists and Acute Kidney Injury (AKI). Recent efforts have explored pharmacovigilance databases to analyze Reporting Odds Ratios (ROR) and Information Component (IC) scores, highlighting elevated frequencies of reported AKI cases with semaglutide and lixisenatide. However, these findings require cautious interpretation, as

	reporting frequency disparities do not inherently establish relative risk or causal relationships. The case in question underscores the consequences of the ongoing GLP-1 agonist shortage, compelling patients to switch medications based on availability. The limited data available regarding the risks associated with switching between GLP-1 agonists underscores the unpredictability of patient responses and the potential for severe side effects, such as stage 3 anuric acute kidney injury.
Nehaal Ahmed MD Dr. Dhruv Sarma Dr. Kyla Lara Breitinger	*Percutaneous Coronary Intervention in Coronary Artery Anomalies* Introduction: Coronary artery anomalies (CAAs) are a diverse group of conditions with a spectrum of clinical presentations. In rare cases, CAAs may cause acute coronary syndrome (ACS) or sudden death. We describe a case of a type 1 non-ST elevation myocardial infarction (NSTEMI) in a patient with an anomalous left circumflex (LCx) artery arising from the proximal right coronary artery (RCA). Case Presentation: A 46-year-old man was admitted for central chest pressure at rest with associated intermittent nausea and diaphoresis. His past medical history included obesity (BMI 31kg/m2) and recently diagnosed Type 2 diabetes mellitus (Hemoglobin A1c of 11.8%) being treated with only lifestyle measures. Evaluation was notable for an initial Troponin T of 34ng/L with a repeat Troponin T of 47ng/L at 6 hours. Serial ECGs revealed dynamic T-wave changes in leads V3-V6. He received high dose Aspirin and Clopidogrel and was started on an intravenous heparin infusion. He underwent coronary angiography and was found to have severe stenosis (90% thrombotic obstruction) of the proximal portion of an anomalous LCx arising from the proximal RCA, with only mild atherosclerosis of his other coronary arteries. He received a drug eluting stent to the proximal portion of the anomalous LCx. Post-procedure transthoracic echocardiography demonstrated a normal left ventricular ejection fraction without regional wall motion abnormalities. He was treated with a high intensity statin, dual antiplatelet therapy, metformin, subcutaneous insulin and discharged with a plan to initiate a GLP-1 agonist in the outpatient setting for diabetes and obesity. Case Discussion: We present a case of a type 1 NSTEMI due to severe stenosis of an anomalous LCx arising from the proximal RCA. Notably, the patient was young and had minimal coronary atherosclerosis in non-culprit vessels. There has been conflicting data on whether CAAs as a whole increase the risk of coronary atherosclerosis1. However, certain anomalies such as an intramural course (where the coronary artery traverses the myocardium rather than the subepicardial fat layer) or an acute angle of origin have been associated with ACS and sudden death presentations1. The patient is currently scheduled to undergo outpatient CT Cardiac Angiography to further define his coronary artery anatomy, as invasive coronary angiography cannot reliably confirm the presence of an intramural arterial course. The literature has demonstrated that PCI to anomalous left circumflex arteries, although technically challenging, can be performed successfully with favorable short- and long-term outcomes, potentially avoiding the need for surgical intervention2-4. This case highlights the principles of management of ACS both with and without CAAs, and the importance of considering CAAs as an etiological factor for ACS in younger patients. If an intramural arterial course is suspected, further workup with CT

	coronary angiography should be undertaken to fully characterize coronary anatomy5.
Joseph Akambase MD	*Is it Acute Fatty Liver of Pregnancy or an Atypical Form of HELLP Syndrome?* Introduction: Acute fatty liver of pregnancy (AFLP) is a rare obstetric emergency characterized by variable maternal liver dysfunction possibly related to defective fatty acid metabolism that can lead to complications for both mother and fetus, including death. The initial clinical findings are typically nonspecific; hence diagnosis of early disease is rarely straightforward and requires a high index of suspicion. Case Presentation: A 23-year-old woman, G1P0 at 38 weeks with no significant past medical history, admitted with nausea, vomiting, headaches, lower abdominal pain and urinary frequency of 2 days duration. At presentation, patient was notably tachycardiac to the 120s, otherwise hemodynamically stable, alongside an unremarkable physical exam. Laboratory evaluation was notable for a leukocytosis of 17.36 cell/ÂµL, platelet count 133 000 cell/ÂµL, creatinine level 1.5 mg/dL, protein to creatinine ratio 0.57, glucose 67 mg/dL, albumin 1.6, total protein 2.6, total bilirubin 9.0, with direct bilirubin 7.0 mg/dL, AST 159, ALT 146, ALP 689 and Ammonia 45. Coagulation studies demonstrated; INR 7.1, PT 80.4, PTT 84.0, and undetectable Fibrinogen. Additional liver and hemolysis studies included negative viral hepatitis panel, ferritin 176.0, LDH 580, haptoglobin < 3, peripheral blood smear with no red cell fragment population, acetaminophen level 8.5, ceruloplasmin 20, negative antinuclear, smooth muscle, mitochondrial, HSV 1/2 IGG antibodies, undetectable Epstein Barr virus PCR. A right upper quadrant ultrasound of the abdomen showed normal sonographic evaluation of the liver with small volume peritoneal ascites. She underwent immediate delivery on Day 2. Hospital course complicated by profound coagulability, acute blood loss anemia, and acute kidney injury, requiring transfer to the ICU and activation of massive transfusion protocol. In spite of immediate delivery and supportive management, hypoglycemia persisted and coagulability worsened, which informed a transfer out to a Transplant Hepatology center for evaluation for liver transplant where she improved remarkably and was discharged home after a few days. Conclusion: This case demonstrates the key clinical overlap between AFLP and other more commonly encountered pregnancy-induced liver disorders such as HELLP syndrome and severe preeclampsia which makes differentiating among them difficult. The Swansea criteria is a useful tool in late diagnosis, but its clinical utility is limited in patients with evidence HELLP syndrome or preeclampsia. Though an LCHAD deficiency was not confirmed, a presumptive diagnosis of AFLP was supported by; multiple system involvement, including acute kidney injury, coagulopathy, pulmonary edema, leukocytosis, persistent hypoglycemia, pronounced hyperbilirubinemia as well as rapid clinical decline. Assuming a broad differential diagnosis while pursuing targeted investigation to detect early liver dysfunction, hall mark of acute fatty liver of pregnancy, as well as immediate delivery are key to preventing associated maternal and fetal mortality.
Alexandra	*Caring For Complex Patients Discharging on Hospice*

AllmanMD Dr. Cory Ingram	Introduction: The number of patients enrolled in hospice continues to grow, with 1.72 million patients cared for by hospice services in 2020. While most Americans wish to die at home, complex illnesses, difficult social scenarios, and financial barriers can make caring for these patients at home challenging. Different hospice agencies have varying resources and abilities, however when patients strongly prefer to spend their remaining time at home, dedicated Hospice and Palliative Care Physicians can be invaluable with coordinating complex logistics and medical care. Case Description: A 26-year-old female with past medical history of sarcoma with metastases to the spine, pelvis, and chest with an Intrathecal Drug Delivery System (IDDS) for cancer related pain presented in acute pain crisis. Unfortunately, her tumor burden had progressed despite multiple lines of chemotherapy and immunotherapy. Oncology, radiation oncology, and surgery had no further treatments available without risk of significant side effects. She was admitted directly to the Inpatient Palliative Care Service. She initially required a dexmedetomidine infusion which was transferred to a ketamine infusion, and pain management was consulted which resulted in a lidocaine infusion, ketamine infusion with boluses, methadone, pregabalin, hydromorphone, and concentration of her intrathecal medications. After a goals of care conference, she elected to move forward with an end-of-life care plan at home to focus on quality of life. She strongly preferred to spend her remaining time at home with family, and the family and hospice agency discussed how to feasibly discharge her home. Initially, she planned to discharge with a Ketamine patient-controlled analgesia (PCA) pump with as needed ketamine bolus, in addition to other oral pain medication. Although hospice could accommodate this PCA, there was a nationwide shortage of IV ketamine, and she was transitioned to oral ketamine. IV Lidocaine infusions in the home, without typical continuous EKG monitoring, were also explored. This would be a first for our institution, however the infusion policies were still being reviewed at the time of discharge. So, she was discharged home with hospice on oral pain medications. Discussion: People are more ill than ever before as they near the end of life. The increased complexity of patients' medical care can lead to increased complexity of discharges, even when discharging on hospice. This patient with a complicated pain regimen required a multidisciplinary team of physicians, social workers, pharmacists, RNs, and leaders in the hospice agency to coordinate her discharge. This dedicated team worked to creatively explore solutions that could work for her such as a Ketamine PCA, lidocaine infusions at home, and continuous midazolam infusions. Often, pumps and IVs are not thought of as being compatible with hospice but with the aim of reducing pain, these can be possible. This discharge process was further aided by the palliative care team acting as her primary inpatient service, a team that is more experienced with hospice discharges. When new and complex patient cases arise, creativity and communication with hospice teams about discharge possibilities can allow patients to spend their valuable remaining time left at home.
Talal Almasri MD	*Severe Caffeine Toxicity Masquerading as Alcohol Withdrawal Syndrome in a Cirrhotic Patient: A Diagnostic Challenge* Introduction: Caffeine, a widely consumed psychoactive substance, pervades

modern society through coffee, tea, energy drinks, and medications. While celebrated for its stimulating effects, the distinction between beneficial use and toxicity can be nebulous, obscured by the ubiquity of caffeine-containing products. Excessive caffeine intake can induce cardiovascular disturbances, central nervous system stimulation, and gastrointestinal symptoms. We present a case of severe caffeine toxicity in a patient with cirrhosis and a history of alcohol use, initially masquerading as alcohol withdrawal syndrome.

Case Presentation: A 57-year-old male with a complex medical history, including severe alcohol use disorder, severe alcohol withdrawal syndrome, cirrhosis, and seizure disorder, presented with palpitations and tremors. He was recently discharged for severe alcohol withdrawal syndrome but claimed abstinence since his prior hospitalization. He complained of tremors, palpitations, diarrhea, nausea, vomiting, and decreased oral intake. Initial concerns included hallucinations at the skilled nursing facility. Remarkably, he disclosed concurrent consumption of 10-15 Monster energy drinks daily for the past 10 days.

In the ED, vital signs revealed a heart rate of 120 and a blood pressure of 161/80 mmHg. Physical examination unveiled an anxious patient with generalized tremors. Initial lab results showed mild leukocytosis (10.54), hypokalemia (K: 2.1L), severe hyperglycemia (464), and an elevated lactate level of 8.2. Thyroid function was normal, ethanol was undetectable, and caffeine levels were pending. A positive urine drug screen indicated phenobarbital and benzodiazepines administered in the ED. The patient received intravenous fluids and potassium supplements totaling 180 mg (120 mg orally and 60 mg intravenously). Subsequent potassium measurement was 1.8. The patient also received 270 mg of Valium, exacerbating agitation, tremors, and mental status, necessitating intubation for airway protection. Consequently, the patient was admitted to the MICU for close monitoring.

During hospitalization, complications included refractory hypokalemia, aspiration pneumonia, and sustained hypertension and tachycardia. Neurological consultation with EEG yielded no seizure activity. By days 6-8, the patient was extubated but exhibited delirium, weakness, and supraventricular tachycardias. By days 9-11, his condition improved, and he was discharged to the SNF. Subsequently, serum caffeine levels were reported at 120 mg/L.

Discussion: This case underscores the diagnostic complexities associated with severe caffeine toxicity, especially when clinical presentations overlap with other medical conditions, such as psychostimulant overdose, alcohol withdrawal syndrome, and thyrotoxicosis. The concurrent use of energy drinks, compounded by cirrhosis, further complicated the clinical picture. Notably, caffeine levels exceeding 50 mg/L can precipitate serious complications, with levels surpassing 80 mg/L carrying lethal potential. Timely recognition of caffeine toxicity in such cases is crucial for prompt intervention and effective management, particularly when confirmatory testing entails protracted turnaround times.

Conclusion: Caffeine toxicity should be contemplated in patients displaying symptoms resembling alcohol withdrawal syndrome, particularly in the context of excessive energy drink consumption. Swift recognition and appropriate management are pivotal to ensuring patient safety, especially in individuals with underlying medical conditions such as cirrhosis. This case

	underscores the significance of thorough history-taking and the necessity for a heightened index of suspicion when confronted with intricate clinical presentations.
Kareem Al-Qadi MD Laura Tucker Dr. Megan Kristan	*Turning Weakness into Strength: Grave's Disease and Paralysis* Introduction: Thyrotoxic Hypokalemic Periodic Paralysis (TPP) manifests as recurrent sudden painless paralysis associated with hypokalemia and hyperthyroidism, primarily in East Asian men aged 20-39. This presents as sudden generalized weakness and is exacerbated by high carbohydrate intake, strenuous exercise, alcohol, stress, and steroids. Proximal muscles and lower extremities are typically more affected. The pathogenesis involves increased Na-K ATPase activity causing hypokalemia, typically < 2mmol/L during attacks. To diagnose TPP, it must be separated from other causes of periodic paralysis including Andersen syndrome, myasthenia gravis, metabolic myopathies, or other causes of initial paralysis like Guillain-Barre syndrome. Case Presentation: A 22-year-old male without significant medical history and a family history of Grave's disease presented with two months of intermittent bilateral upper/lower extremity weakness, 20lb weight loss, and intermittent palpitations. On admission, his potassium was 1.7 mmol/L, magnesium normal, TSH undetectable, free T4 elevated at 4.0 ng/dL and free T3 elevated at 14.7 pg/mL. Creatinine was normal with low urine potassium of 9 mmol/L, and AM cortisol was normal at 11.6 ug/dL. On exam he couldn't lift his lower extremities off the bed and had no other neurological deficits. His thyroid US showed a normal sized thyroid without nodules. Thyrotropin receptor antibody was positive at 15. Given his weakness, hyperthyroidism, and hypokalemia without clear renal wasting etiologies, he was diagnosed with Grave's TPP. He was given IV and then PO potassium with quick improvement in his weakness. He was discharged with potassium supplements, methimazole, metoprolol tartrate, and instructions to avoid triggers like alcohol, high carbohydrate intake, and strenuous exercise. His TSH normalized after 5 weeks, with low free T4 (0.55 ng/dL) and low total T3 (74 ng/dL) concerning for swinging hypothyroidism. After improvement of his hyperthyroidism, his potassium remained normal without recurrence of his weakness. Ultimately, he underwent I-131 ablation to prevent remission and eventually required levothyroxine for subsequent hypothyroidism. Discussion: TPP is a clinical diagnosis in patients with painless weakness, hypokalemia, and hyperthyroidism without other causes. An EMG or muscle biopsy isn't required for diagnosis. In TPP, thyroid hormone increases tissue sensitivity to beta-adrenergic stimulation, which increases Na-K ATPase channel activity on skeletal muscle membranes. This causes potassium influx into muscle cells, hyperpolarization, and muscle weakness. Epinephrine and insulin further augment the Na-K ATPase pump, explaining why exercise or carbohydrate-heavy meals can precipitate weakness. Unlike hypokalemic periodic paralysis which has autosomal dominant inheritance, TPP is sporadic and presents in adulthood. Unlike hypokalemic periodic paralysis where treatment is potassium-sparing diuretics and carbonic anhydrase inhibitors, in TPP treatment focuses on restoring a euthyroid state with methimazole, potassium supplementation, beta-blockers, radioactive iodine, or surgery to prevent further attacks. Patients with TPP on treatment are monitored by checking thyroid and potassium levels to assess for signs of remission. In cases of remission, there is benefit to discontinuing methimazole and pursuing more definitive treatments such as thyroidectomy or radioactive iodine ablation to reduce risk of recurrence of TPP. In these

	patients, surgical management is more likely to prevent any future episodes of TPP.
Nader James Al-Shakarchi MD Dr. Lena Ayari Dr. Ikram Ul-Haq	*Pel-Ebstein Fever and Cyclical Pancytopenia: An Unusual Presentation of Peripheral T-Cell Lymphoma* Introduction: Fevers of unknown origin continue to remain a formidable challenge, but subtle clues including fever pattern can provide vital diagnostic information. Pel-Ebstein fever is a rare condition, defined by cyclic fevers that rise and fall every 1 to 2 weeks, reported in patients with lymphoma. However, concomitant cyclical pancytopenia has only been reported on three occasions. Case Report: A 34-year-old previously healthy female presents high fever and abdominal discomfort following a 2-year episodic history of fever, anemia, and pancytopenia. The symptoms began in February 2020, presenting with high fevers to 39°C, night sweats, nausea, vomiting, and chills. The episodes used to last for 3 to 4 days but have progressively lengthened up to 10 days. She endorses a 40 lb weight loss since the start of the symptoms in February 2020 and an associated macular rash with the episodes, but she denies arthralgias, oral ulcers, Raynaud's phenomenon, or photosensitivity. No other family members of have developed similar symptoms, and there is no family history of autoimmune or hematologic pathology. She denies a history of cigarette smoking, illicit drug use, or current alcohol usage. Travel history was significant for travel to the Caribbean between 2012 and 2020, but otherwise unremarkable from February 2020 onwards. Upon admission in the emergency department, vital signs were significant for pyrexia (39.5°C), tachycardia (125 beats per minute), and tachypnea (18 breaths per minute). Physical examination revealed non-tender splenomegaly and a livedoid rash. Laboratory evaluation demonstrated a decreased hemoglobin (9.8 g/dL), thrombocytopenia (81 x 109/L), and leukopenia (3.0 x 109/L) with a decreased CD4+ cell count (76 /mm3). Hepatic function testing demonstrated an unconjugated hyperbilirubinemia (total bilirubin: 1.9 mg/dL, unconjugated bilirubin: 1.4 mg/dL), raised ALT (53 I/U), and AST (60 I/U), as well as an unremarkable ALP (87 I/U). CT scan with IV contrast with revealed a 22 cm splenomegaly. Following transfer to Internal Medicine, consultative input from hematology, infectious disease, and rheumatology services were obtained. Infectious and rheumatologic evaluation were negative, including Karius testing. PET-CT revealed a marked hypermetabolic spleen and bone marrow biopsy showed no abnormalities. Given the suspicion for splenic pathology and otherwise unremarkable laboratory evaluation, the patient was administered asplenia vaccinations in view of a diagnostic and therapeutic splenectomy. Pneumocystis jirovecii prophylaxis was deferred due to the patient's known history of transient pancytopenia. Pathologic examination of the spleen revealed a CD30+ T-cell lymphoma consistent with anaplastic large cell lymphoma. Discussion: Our case highlights the need to carefully consider fever patterns in patients with a fever of unknown origin. Although Pel-Ebstein fever is typically associated with Hodgkin's lymphoma, it has been described in other lymphoproliferative pathologies and in the context of infection. Clinicians should incorporate these findings with other suggestive clues, such as the

	presence of intravascular hemolysis and the PET-CT findings in this case, to reach a diagnosis.
Zach Anderson, MD	*Steroid-induced Esophageal Herpesvirus Reactivation* Introduction: This abstract presents a clinical vignette of a patient with a complex medical scenario who experienced oropharyngeal and esophageal ulceration due to herpesvirus reactivation secondary to steroid therapy. Case Presentation: The patient is a 53-year-old Asian-American male who was being treated with high-dose prednisone for pyoderma gangrenosum (PD) of the right lower extremity. PD is a rare, inflammatory skin condition which can form erythematous ulcerations. The mainstay of treatment for PD is anti-inflammatory and immunosuppressive agents including local and systemic agents. The patient was placed on high-dose prednisone and initially demonstrated improvement in the appearance and margins of the ulceration. Within 2 weeks of therapy, the patient acutely developed severely painful ulcers on his tongue and oral mucosa. Upper endoscopy demonstrated ulceration of the oropharynx, epiglottis, and esophagus. Punch biopsy and staining was consistent with herpesvirus (HSV) and the patient had HSV1 detectable in his serum. The reaction of HSV1 was thought to be attributable to the immunosuppressive effects of his steroid therapy. This compromised his immune response and allowed reactivation of HSV1, eliciting painful ulcerations throughout his oropharynx and esophagus, diminishing his ability to tolerate oral intake, thus requiring hospital admission. Conclusion: This case demonstrates the delicate nature of treating the patient's primary condition and balancing the risk of immunosuppression and related consequences. Clinicians need to monitor and remain vigilant concerning reactivation of previous infections, such as HSV1 amongst others, for patients receiving immunosuppressive treatment. Risks and benefits of steroid therapy should be discussed and tailored to the patient, particularly in those with preexisting infections and immunologic vulnerabilities.
Rachel Anderson, MD	*West Nile Encephalomyelitis Masked by Multiple Systemic Infections* Introduction: West Nile virus is now a common cause of mosquito-borne illness in the United States. Neuroinvasive disease presents a diagnostic challenge however, as it can cause a variety of syndromes including meningitis, encephalitis, and/or acute flaccid paralysis. Case Description: A 74-year-old female rancher with past medical history of hypertension presented to the emergency room in August for abdominal pain. She was seen at an outside ED one week prior for flu-like symptoms. She was diagnosed with a UTI and given Cefalexin. Her symptoms improved for the first two days, but worsened again four days ago when she developed fever, abdominal pain, and diarrhea. She was hyponatremic and hypokalemic, and CT abdomen/pelvis revealed watery stool throughout the colon. She was admitted to the hospital and found to be positive for C. Diff for which she was started on PO Vancomycin 125 mg QID. On the second hospital day, the patient developed encephalopathy with disorientation, periods of somnolence, and myoclonus in the upper extremities. Neurologic exam revealed cogwheel rigidity in the left upper extremity, flaccid tone in the right upper extremity, and hyperreflexia with clonus in the lower extremities bilaterally. She remained afebrile and hemodynamically stable with a normal CBC, renal function panel, hepatic function panel, lactate, and venous blood

	gas. The patient had blood and urine cultures with no growth to date, a negative Tick-borne panel, and a normal TSH. CT head was normal. Empiric acyclovir was started to cover for HSV/VZV encephalitis, but her neurologic function continued to deteriorate with decreased alertness, orientation only to self, and profound weakness in all four extremities. MRI brain revealed nonspecific dural thickening. However, MRI cervical spine showed anterior horn cell hyperintensity and lumbar puncture revealed elevated total nucleated cells of 10/mcL with lymphocytic predominance, elevated protein of 180 mg/dL, and normal glucose. Diagnosis was confirmed when the CSF and serum returned positive for West Nile virus IgM. Acyclovir was discontinued after CSF VZV/HSV PCR returned negative. The patient was treated with supportive cares, and her mental status improved over the next 48 hours allowing a more comprehensive motor assessment. Her exam showed moderately severe patchy flaccid paralysis in her limbs consistent with multifocal anterior horn cell injury. The patient was counseled that despite improvement in her encephalopathy, she will likely experience some permanent motor impairment as the viral damage to the anterior horn cells is largely non-reversible. She continued to work with PT and OT, and placement was arranged at a SNF for ongoing therapy. Discussion: This case demonstrates typical risk factors for West Nile virus infection: occurrence in late summer in a patient who is frequently outdoors and from the Midwest. It also illustrates that attention to the neurological exam provides clues to nervous system injury that could otherwise be masked by metabolic/infectious encephalopathy. Given the variety of syndromes West Nile virus can produce and its increasing incidence, providers should have a low threshold for including this virus on the differential for any case with a presentation in this realm.
Allison Angeli MD Dr. Audrey Blazek Dr. Christopher Stephenson	*Abscessed with Liver Lesions: A Case of Pyogenic Liver Abscesses in the Setting of Granulomatous Inflammation* Introduction: A hepatic abscess is a pus-filled cavity in the liver due to invasion of microorganisms. In Western countries, 80% are pyogenic, or due to bacteria, most commonly Escherichia coli, Klebsiella pneumoniae, Streptococcus species, and Enterococcus species. We present a case of a patient with pyogenic liver abscesses in the setting of hepatic granulomatous inflammation. Case: A 64-year-old female with hypertension, hyperlipidemia, and recent cholecystectomy presented to an infectious disease clinic with four months of right upper quadrant abdominal pain, episodic fevers, and a ten-pound weight loss. Exposures included turkeys, goats, cats, a bearded dragon, eating medium-rare game meats, and travel to Grenada and Mexico. Prior workup included unremarkable CBC, CMP, INR, ANA, AMA, and celiac panel. CT imaging four months prior revealed a 4.0x3.4x3.5 cm right hepatic lobe lesion, and a biopsy demonstrated inflammation with fibrosis. Interval CT at two months showed increased size to 5.2x4.9x3.9 cm, and repeat biopsy revealed necrotizing and non-necrotizing granulomas. The biopsies' cultures had no growth. After negative infectious workup and symptom persistence despite an antibiotic course, prednisone was initiated for symptomatic hepatic granulomatous inflammation. However, due to fever at the infectious disease appointment, she was transferred to the emergency department, where she was hypotensive (80s/50s mmHg) and tachycardic (127 bpm). Physical exam

	was remarkable for right upper quadrant abdominal tenderness. Labs were notable for WBC 15.5x10(9)/L, ESR 49 mm/h, and CRP 72 mg/L. A CT demonstrated now-two hepatic lesions, measuring 6.5x5.2x6.7 cm (superior) and 7.9x5.3x6.8 cm (inferior). Theseâ€¯multi-loculated lesions with perilesional enhancement were concerning for abscesses. Empiric antibiotics were initiated. Broad bacterial, mycobacterial, parasitic, fungal, and viral studies were negative. Extensive workup for malignant, autoimmune, inflammatory, and other systemic etiologies was unremarkable. Two percutaneous drains were placed for source control, and fluid from both abscesses grew Escherichia coli. Another biopsy re-demonstrated necrotizing and non-necrotizing granulomas. The patient was discharged with 4 weeks of IV ceftriaxone and oral metronidazole, and ultimately required right hepatic segmentectomy. Histology was consistent with an abscess and necrotizing granulomatous inflammation. Two-month post-operative CT showed no recurrence. Discussion: The etiology and acquisition of this patient's hepatic granulomatous inflammation and E. coli abscesses remain undetermined. Hepatic granulomas are found in 3-10% of all liver biopsies, and may be infectious, malignant, autoimmune/inflammatory, idiopathic, or drug-induced. An E. coli abscess would be an atypical presentation. Many (10-36%) granulomatous liver lesions remain idiopathic after extensive workup, as demonstrated in this case. E. coli hepatic abscesses are most commonly acquired by biliary spread. Our patient may have acquired the infection from manipulation of her biliary tract during cholecystectomy 1.5 years prior, or from the recurrent hepatic biopsies. Treatment of pyogenic liver abscesses requires 2-6 weeks of antibiotics and source control. Abscesses < 3cm should be managed with antibiotics alone, 3-5cm can be aspirated, and > 5cm require catheter drainage. If abscesses are not responsive to antibiotic therapy, are not amenable to drainage, multiple in nature, or there is evidence of rupture, liver resection should be considered, as was in this patient.
Lena Ayari MD Dr. Nader James Al-Shakarchi Dr. Ikram-Ul Haq	*Is it still Still's Disease? The Mimicry of Macrophage Activation Syndrome* Introduction: Macrophage activation syndrome (MAS) is a subtype of hemophagocytic lymphohistiocytosis and a life-threatening, severe inflammatory syndrome. It is frequently underdiagnosed and confused with sepsis, adverse effects of anti-arthritic drugs, and progression of rheumatologic or infectious diseases. Case Description: A 52-year-old woman with a history of adult-onset Still's disease presented to the emergency department with persistent fever (Tmax 38.4°C). Three weeks prior to her attendance, she had been diagnosed with COVID-19 and experienced a Stills disease flare requiring a 5-day admission. At that time, she denied respiratory symptoms, reported mild improvement of her symptoms with a methylprednisolone taper, and received outpatient rheumatology follow-up. Despite outpatient management, due to ongoing fever, profound fatigue, and joint pain, the patient returned to the emergency department. Upon presentation, the patient endorsed a rash, joint pain, right upper quadrant pain, and intermittent bilateral retro-orbital headache. Vital signs showed a hemodynamically stable blood pressure (149/88 mmHg), borderline tachycardia (112 beats per minute), tachypnea (19 breaths per minute), and pyrexia (38.4°C). Physical examination revealed a diffuse morbilliform rash, right upper quadrant tenderness with a negative Murphy's

	sign, and splenomegaly. Admission laboratory values were significant for hyponatremia (127 mmol/L), mild thrombocytopenia (119 x 109/L), elevated ALT (1698 U/L) and AST (3198 U/L), hypertriglyceridemia (152 mg/dL), marked hyperferritinemia (87720 ng/mL), and raised inflammatory markers (ESR: 51 mm/hr, CRP: 90.2 mg/dL). Due to ongoing concern for acute liver injury, hepatotropic virus serologies, an acetaminophen level, and right upper quadrant ultrasonography were obtained. To rule out sepsis, blood cultures were obtained. With a high index of suspicion for MAS, an H-score was calculated at a pre-test probability of 15% and a cytokine panel was collected. When transferred to the Internal Medicine floor, a 1g IV solumedrol infusion was initiated overnight and the following day rheumatology, hematology, and dermatology consults were instituted. Daily intramuscular dexamethasone 20 mg and oral hydroxychloroquine 200 mg twice daily were initiated, which improved the patient's retro-orbital headache and morbilliform rash. As per hematology recommendations and abnormal cytokine panel, a bone marrow biopsy and CT scan with contrast of the head, chest, abdomen and pelvis were performed, which demonstrated occasional hemophagocytic cells and 14 cm splenomegaly respectively. No evidence of lymphoma, active EBV infection, or CMV intranuclear inclusions were observed; fungal and bacterial cultures on the bone marrow biopsy showed no growth. Following downtrending CRP (18.4 mg/dL), D-Dimer (14617 ng/mL), and resolving transaminitis (ALT: 1271 I/U, AST: 1038 I/U), the patient was discharged with an oral prednisolone taper, outpatient rheumatology follow-up, and repeat laboratory testing. Discussion: Early diagnosis of MAS is critical to avoid significant morbidity and mortality. Its clinical presentation can mimic sepsis, lymphoproliferative disorders, and multisystem inflammatory syndrome, which have conflicting management strategies. Our case demonstrates the diagnostic utility H-score and the value of thorough diagnostic work-up.
Jacob Bauss, MD Dr. Caroline Matchett Dr. Victor Chedid	*A Case of Jejunal Hemangiolymphangioma Presenting as an Obscure Gastrointestinal Bleed* Introduction: Hemangiolymphangiomas are benign tumors of mesenchymal origin composed of dilated lymphatic and blood vessels. They most commonly appear on the skin early in life and regress spontaneously. Occurrence in the gastrointestinal tract is a rare phenomenon. Endoscopy remains the gold standard for diagnosis; however direct visualization can be difficult without more advanced endoscopic techniques due to their midgut predilection. We present a case of obscure gastrointestinal bleeding caused by a jejunal hemangiolymphangioma. Case Presentation: A 69-year-old male with a medical history of chronic obstructive pulmonary disease, hypertension, and coronary artery disease status-post coronary artery bypass grafting was admitted to the hospital with abdominal discomfort and melena. Laboratory results showed a hemoglobin of 6.9 mg/dL (reference range: 12-16 mg/dL). The patient's baseline hemoglobin was 9.3 mg/dL. His blood urea nitrogen was 32 mg/dL (reference range: 6-21 mg/dL) and creatinine was 1.12 mg/dL (reference range: 0.59-1.04 mg/dL). Physical exam was notable for pallor with mild, diffuse abdominal tenderness. He was transfused 2 units of packed red blood cells and started on an intravenous proton pump inhibitor (PPI). Esophagogastroduodenoscopy (EGD) showed gastric erosions without active

	bleeding. Colonoscopy showed blood throughout the colon without an identified source. Subsequent abdominal CT angiography disclosed active extravasation in the jejunum. Double balloon enteroscopy revealed an actively bleeding 0.8 x 0.6 x 0.4 cm semi-sessile polypoid lesion in the proximal mid-jejunum. Hemostasis was achieved with epinephrine injection, hot snare polypectomy, and clip closure. Histopathology confirmed the diagnosis of hemangiolymphangioma. The patient's hemoglobin stabilized following endoscopy and he was continued on a PPI for 6 weeks due to the gastric erosions. Conclusion: The presentation of gastrointestinal hemangiolymphangiomas varies widely from asymptomatic to chronic anemia from occult bleeding or brisk hemorrhage requiring aggressive resuscitation. Conventional endoscopy with esophagogastroduodenoscopy (EGD) or colonoscopy can miss these lesions due to their anatomic preference for the jejunum. Further investigation with capsule endoscopy, CT angiography (as in our case) or balloon enteroscopy might be required. Capsule endoscopy has the highest yield within the first 48 hours of active bleeding (1), however, it lacks therapeutic capability. The mainstay treatment for hemangiolymphangiomas is complete resection to prevent further bleeding, limit local recurrence, and prevent complications such as bleeding, intestinal obstruction, or intussusception. In the first two years, local recurrence varies between 10-27% for complete resections (2). Suspicion should be high for recurrence in patients with previously resected hemangiolymphangiomas presenting with worsening iron deficiency anemia, melena, or hematochezia.
Caroline Bergeron, MD Dr. Deepi Goyal	*ACEi: The Diagnosis of Non-specific Abdominal Pain: ACEi-Induced Intestinal Angioedema* Introduction: Angiotensin converting enzyme inhibitors (ACEi) are commonly prescribed for hypertension, cardiovascular disease, and diabetes mellitus. ACEi cause accumulation of vasoactive peptide bradykinin, des-Arg9-BK, and neuropeptide Substance P, stimulating vasodilation, increasing vascular permeability, and potentially resulting in angioedema. Incidence is estimated at 0.1-0.7%. Clinical features usually involve edema of the face, mouth, and upper airway. Visceral angioedema has also been described but is less well recognized. Case Presentation: A 50-year-old woman with a history of hypertension and chronic constipation presented to the emergency department with acute onset, diffuse, intermittent, cramping abdominal pain following dinner. The patient was born in Somalia and immigrated to the US in 2010 from Botswana. Medical comorbidities included pulmonary and hepatic echinococcal hydatid cysts treated with 3 months of albendazole and right lower lung lobectomy 12 years prior, and laparoscopic cholecystectomy 4 years prior. Six days before her emergency department presentation, her lisinopril was increased from 20 to 40mg for uncontrolled hypertension. She had previously been evaluated for a similar episode of abdominal pain 1.5 years prior to her current presentation following a lisinopril dose increase from 5mg to 10mg, and imaging revealed non-specific acute enteritis. She was afebrile with normal vital signs. Physical examination was notable for a soft abdomen with mild, diffuse tenderness and no guarding. Laboratory work-up, including CBC, BMP, liver function tests, lipase, and urinalysis was normal. CT of the abdomen and pelvis with IV contrast revealed non-specific, enteritis involving a long segment of jejunum without obstruction.

	ACEi-induced intestinal angioedema was diagnosed. She was given oral diphenhydramine, and her abdominal pain resolved. ACEi therapy was discontinued, and alternative antihypertensive agents were considered. Discussion: ACEi-induced intestinal angioedema should be considered in patients taking ACEi who present with non-specific abdominal pain. ACE-induced angioedema is 3 times more likely among black patients and slightly more common in women. CT imaging findings include dilated loops of bowel, thickened mucosal folds, and mesenteric edema. Involvement of long bowel segment is common, and the jejunum is usually involved. Treatment involves supportive care and cessation of ACEi therapy.
Nicholas Bergeron, MD	*No Methicillin Resistance? Still a Problem. A Case of Severe MSSA Pneumonia* Introduction: Methicillin-susceptible Staphylococcus aureus (MSSA) is a relatively uncommon but severe cause of pneumonia. As an uncommon cause of community or facility acquired pneumonia, adequate oral empiric treatment can be challenging. Case Presentation: An incarcerated 61-year-old man with a past medical history of Chronic Obstructive Pulmonary Disease (COPD) presented to the hospital with sepsis due to pneumonia and concerns for empiric treatment failure. He reported a 4-day history of respiratory symptoms inclusive of productive cough, shortness of breath, and fever. He was seen at the facility healthcare center and was given 1 dose of IV methylprednisone and a prednisone taper for presumed COPD exacerbation and levofloxacin for presumed pneumonia. Despite initial improvement, he experienced worsening productive cough, fevers to 39.5° C, hypotension, and new oxygen requirement prompting transfer to the hospital for further management. Antibiotics were broadened to vancomycin and cefepime for severe community-acquired pneumonia. CT scan demonstrated left lung consolidative process consistent with infection and diffuse centrilobular emphysema with large bullae, some of which contained fluid levels. S. aureus/MRSA nares with no evidence of staph bacteria. Vancomycin was discontinued and cefepime monotherapy was continued. Blood cultures demonstrated S. aureus in 2/2 bottles with MecA gene not detected, sputum culture consistent with MSSA resistant to levofloxacin. Infectious Disease (ID) was consulted and recommended cefazolin monotherapy, transesophageal echocardiography (TEE) to be completed 5 days after initial positive blood cultures. Three days after admission, with worsening respiratory status, repeat noncontrast chest CT scan demonstrated worsening of left upper lobe pneumonia, new layering fluid densities suspicious for bullous infection and worsening consolidation in the left lung base. Interventional pulmonology was consulted for bullous infection drainage, however risk of bronchocutaneous fistula was felt to be unacceptably high. Pulmonology and ID felt that pneumonia could be polymicrobial and antibiotics were broadened to cefepime and metronidazole. Blood cultures did not demonstrate any further growth of bacteria 3 days post-admission. TEE was performed and demonstrated linear strands on valves, felt likely to be degenerative however endocarditis could not be excluded. Tenuous respiratory status 8 days post-admission led to a repeat CT of the chest which demonstrated worsening of pneumonia, increased bullous fluid layering. Repeat sputum culture grew MSSA. Interventional pulmonology and thoracic surgery were consulted regarding intervention for source control; both declined due to unacceptably high risk of fistula formation. Patient was

	then transitioned back to cefazolin monotherapy per ID. The pneumonia slowly improved, and he was later discharged back to his facility for an additional 6 weeks of IV cefazolin for presumed endocarditis with close ID follow up as an outpatient. Conclusion: This case illustrates the importance in covering S. aureus pneumonia empirically, especially for patients who fail outpatient antibiotic therapy. Additionally, it demonstrates that nasal swabs for S. aureus/MRSA have error associated with the test and a high level of clinical suspicion should remain for patients with severe necrotizing pneumonia. Once gram-positive cocci have been detected in blood cultures, infectious disease consultation is paramount for effective management.
Yvonne Bui, MD Dr. Rachel Suen Dr. Amos Lal	*The Tonsillar Abscess That Couldn't Be Drained* Introduction: Peritonsillar abscesses are severe deep neck infections that generally follow tonsillitis or pharyngitis. They most commonly present as children or young adults with unilateral swollen tonsil, fever, and sore throat. The majority of patients recover with a course of antibiotic therapy. Atypical presentations of peritonsillar abscesses should raise suspicion for underlying malignancy such as squamous cell carcinoma or lymphoma. Here, we present a case of advanced diffuse large B-cell lymphoma (DLBCL) appearing initially as tonsillar abscesses. Case Presentation: A 73-year-old woman with rheumatoid arthritis on methotrexate presented with two days of cough, sore throat and fever. She was found to have bilateral pharyngeal and palatine tonsillar masses on CT, concerning for abscesses. The patient was started on antibiotics and steroids, especially with concern for airway compromise. Tonsillar aspirate was performed with minimal return for cultures which were positive for Serratia fonticola. Despite a week of appropriate antibiotic therapy, she developed worsening mental status and acute hypoxic and hypercarbic respiratory failure requiring intubation. Multiple laryngoscopies demonstrated airway edema without purulence. She underwent incision and drainage with tonsillar pathology revealing EBV-positive B-cell lymphoma. PET-CT showed FDG-avid lymph nodes above and below the diaphragm, as well as FDG uptake in the tonsils. Bone marrow biopsy was without immunohistochemical features of lymphoma. She was started on lymphoma-directed therapies with dexamethasone followed by rituximab. Follow-up PET-CT one month after initiation of therapies showed complete response. Discussion: This case highlights a unique presentation of DLBCL in an immunocompromised patient. DLBCL typically presents as a rapidly growing mass lesion in lymph nodes of the neck, abdomen, or mediastinum. Clinical manifestations tend to vary based on organ involvement. Prior case reports discuss similar tonsillar involvement leading to misdiagnoses as infections, particularly in immunocompromised patients. Careful diagnostic evaluation should occur with atypical presentations. Bilateral peritonsillar abscesses are rare and should initiate consideration of a broader differential.
Ahsan Butt, MD Dr. Julia Pearson Dr. Julia Liberto Dr. M. Nadir Bhuiyan	*Linitis Plastica: A Diagnostic Challenge* Introduction: Linitis plastica, a subtype accounting for approximately 5% of primary gastric cancer cases, predominantly presents as poorly differentiated signet-ring cell carcinoma. Its prognosis is often grim, primarily due to advanced disease at diagnosis and a proclivity for metastasis. Within this

	clinical case report, we describe a complex diagnostic conundrum that ultimately led to the diagnosis of signet-ring cell carcinoma after two sequential endoscopic evaluations. The case is further complicated by the unusual presentation of periprosthetic reactive synovitis, termed metallosis. Case Presentation: A 63-year-old male with a history of hip arthroplasty presented with a month-long constellation of symptoms including dysphagia, nausea, and abdominal distention. One week prior to admission, he developed left-sided abdominal pain, which prompted an evaluation at an external medical facility, where EGD/Colonoscopy with biopsies yielded unremarkable findings. He subsequently presented to our hospital. Upon admission, laboratory investigations revealed an elevation in total bilirubin (3.4), direct bilirubin (3.3), ALT (570), AST (288), Alkaline Phosphatase (475), and CRP (42.2). CT Abdomen/Pelvis demonstrated a significant periprosthetic fluid collection adjacent to the site of a prior total hip arthroplasty, exerting a mass effect on the bladder. A swallow study unveiled marked abnormalities, prompting further exploration of the periprosthetic fluid collection. Our suspicion hinged on the potential for metallosis-induced infiltrative processes that could account for transaminitis and possible esophageal involvement. However, fluid collection analysis was unremarkable, and testing for cobalt and chromium in both the collection and serum returned negative results. Given worsening liver enzyme abnormalities, our focus pivoted toward a meticulous evaluation of the liver. A RUQ ultrasound demonstrated mild dilatation of intra/extrahepatic biliary ducts, corroborated by a subsequent MRCP that unveiled abnormal biliary cutoff and mild periductal wall thickening, raising suspicion of perihilar cholangiocarcinoma. EUS/ERCP was subsequently conducted, revealing thickened gastric folds, arousing concern for an infiltrative neoplastic process, specifically linitis plastica. Biopsy results from endoscopic examination of the gastric mucosa confirmed the presence of poorly differentiated gastric adenocarcinoma with signet-ring cell features a diagnosis that had eluded previous biopsy attempts. In contrast, ERCP biopsy results from the biliary tract returned negative for malignancy, suggesting extrinsic compression of biliary ducts attributed to the mass effect of the infiltrative tumor. The patient was subsequently evaluated by an Oncology team, with plans for initiation of FOLFOX and Nivolumab. Discussion: This clinical case underscores the intricacies inherent in the diagnostic process. Initially, our attention was captivated by a conspicuously large and complex periprosthetic fluid collection (8 cm x 13 cm x 15 cm), leading us to consider it the linchpin of the patient's presentation an application of Occam's razor. However, this imposing entity ultimately proved to be a red herring. Instead, the primary driver of the patient's clinical manifestation was revealed to be diffuse gastric cancer, a pathological entity which often evades detection via biopsy due to its lack of superficial involvement. This case underscores the imperative recognition of the potential for false-negative outcomes in endoscopic biopsies for diffuse-type gastric cancer, necessitating a heightened index of suspicion to attain a definitive diagnosis.
Sophie Campbell, MD	*Anorexia...Analgesia...Acidosis*

Dr. Peter Lund	Introduction: Acetaminophen is a commonly used over-the-counter analgesic considered benign if used as directed. However, it can cause acidosis even when used at a standard dosage. Case Presentation: A 62-year-old female with a history of heart failure and chronic lower extremity edema presented to the emergency department with generalized pain for about 1.5 months. Vital signs were normal. She was notably somnolent and confused. Initial evaluation revealed acute kidney injury with elevated anion gap metabolic acidosis. Venous blood gas demonstrated pH 7.18, CO2 34. Acute kidney injury was presumed cardiorenal in the setting of CHF, however the etiology of the acidosis remained unclear. Lactate, beta-hydroxybutyrate, urine toxicology, salicylate, acetaminophen levels, serum osmolality, liver function tests were all unremarkable. Patient denied alcohol use or other ingestion. She reported daily 2 gram analgesic use for pain, which was ultimately presumed to be related to edema from CHF exacerbation vs. lower extremity cellulitis. Intravenous bicarbonate and N-acetylcysteine therapy were started empirically with a working diagnosis of 5-oxoproline as the cause of the acidosis given patient's reported daily acetaminophen use and no other clear explanation. Organic acid analysis ordered. Her mental status returned to baseline overnight. Upon further discussion, she reported anorexia for some time, and no appetite for one week in the setting of increased pain. A thorough nutritional work up was performed, and demonstrated low phosphorus, zinc, vitamin-D, magnesium, and iron deficiency anemia. Patientâ€™s kidney function and acidosis normalized with bicarbonate and time. Organic acid analysis revealed minimal to moderate elevation of 5-oxoproline, effectively confirming the diagnosis of elevated anion gap acidosis secondary to 5-oxoproline. Conclusion: This case illustrates a rare cause of elevated anion gap metabolic acidosis, 5-oxoproline. 5-oxoproline is a molecule in the gamma-glutamyl pathway, the metabolic cycle responsible for generating glutathione and membrane transport of amino acids into the cytosol (1). Normal glutathione levels are required for feedback suppression of this pathway (1). Acetaminophen use decreases glutathione stores, which can be further depleted by malnutrition or sepsis. Without sufficient glutathione to inhibit this pathway, 5-oxoproline levels accumulate and can lead to acidosis. This case exemplifies the risk that comes with therapeutic acetaminophen use in the setting of underlying poor nutritional status. Although this is ultimately a rare diagnosis, it is a good reminder that over the counter medications are not benign. It also emphasizes the importance of looking at the whole patient, as this patient's acetaminophen use was appropriate, however when viewed holistically, her anorexia in association with her analgesia use predisposed her to this rare cause of acidosis.
Patricia Carey, MD Dr. Nadir Bhuiyan	*A Case of Recurrent Inguinal Lymphadenitis* Introduction: Extrapulmonary tuberculosis (EPTB) often presents with (or as) lymphadenitis. We present a case of non-tender recurrent inguinal lymphadenitis presumed to be extrapulmonary tuberculosis. Case Presentation: A 73-year-old male from Mexico presents for evaluation of recurrent inguinal lymphadenitis. Patient developed a small, non-tender left inguinal lymph node resolving in under a month. The lymph node enlarged again one month later and surgical excision with biopsy revealed necrotizing panniculitis and granulomatous lymphadenitis. A month later,

	symptoms recurred, and repeat excision was performed with negative microbiologic studies. Further workup was negative for autoimmune processes, sarcoid, endemic mycoses, bartonella and chlamydia. He was initiated on empiric doxycycline and amoxicillin. However, he was found to have a positive T-SPOT tuberculosis test and was initiated on rifampin daily for presumed latent tuberculosis. He developed new right non-tender inguinal lymphadenopathy and presented to our institution for second opinion. He reported unintentional weight loss and denied fever, chills, or night sweats. CT chest demonstrated no findings concerning for active tuberculosis but stable findings likely secondary to prior granulomatous disease. He underwent FNA of the right inguinal lymph node with pathology revealing necrotizing granulomatous inflammation negative for malignancy and a negative AFB smear. QuantiFERON Gold test at that time was positive. He was initiated on standard four-drug therapy for presumed culture-negative, extrapulmonary TB presenting as recurrent inguinal lymphadenitis. Additionally, he also was found to have a high PSA. Subsequently, he underwent imaging of his prostate, which demonstrated PIRADS 5 lesions in the prostate, the enlarged right inguinal lymph node, and evidence of a small left-sided transsphincteric perianal fistula. Imaging five months later following drug therapy revealed that the inguinal lymphadenopathy had resolved. Discussion: This case demonstrates the need for thorough workup for lymphadenitis and to have high suspicion for tuberculosis, especially in patients from high endemic areas. There are many other causes of chronic lymphadenitis, including nontuberculous mycobacteria, toxoplasma and bartonella species, fungi, neoplasms, sarcoidosis, drug reactions or nonspecific hyperplasia. In this case, workup was performed without any significant findings for other etiologies of lymphadenitis. As in this case, tuberculosis lymphadenitis typically presents as painless swelling of lymph nodes lasting 1-2 months. As it is usually painless, patients may not initially seek medical care and often have long delays in diagnosis and treatment. Most patients will have a positive tuberculosis skin test and normal findings on chest radiography. In addition, draining sinus tracts have been reported in 5-10% of patients. Bilateral lymphadenopathy is present in approximately 20% of cases. The gold standard of tuberculosis diagnosis is made by culturing the mycobacterium tuberculosis organisms from the specimen. However, diagnosing remains difficult with prolonged wait times for cultures to grow and yields of mycobacterial culture ranging from an estimated 30% to 80%. EPTB is often presumed with a histopathological diagnosis of typical findings including the presence of granulomas and caseation in the appropriate clinical context. This case demonstrates presumed EPTB due to the significant histological findings on biopsy, patient's clinical context, and negative workup for other etiologies of lymphadenitis.
Maroun Chedid, MD Dr. Abdul Sanni Dr. Michelle Carlson	*Lyme Carditis Presnting as Recurrent Syncope in a Prviously Healthy Young Adult* Background: Lime disease has been associated with myocarditis and conduction abnormalities in the early stage of the disease. We present a case of Lyme carditis (LC) causing paroxysmal high degree atrioventricular (AV) block in a previously healthy adult. Case Presentation: 35-year-old healthy male was brought to the emergency

	department for syncope with profound bradycardia and hypotension. Initial EKG demonstrated a complete heart block with a slow junctional escape rhythm. He experienced 5 similar episodes of syncope since the age of 28. He did not recall any history of tick bites; however, he lived in a wooded area in the Midwest. Decision making: 1 mg of Atropine was administered without any improvement. Transcutaneous pacing was initiated and led to significant hemodynamic improvement. A temporary pacemaker (TPM) was inserted on the second day of hospitalization. Considering his social history, the patient was started on Ceftriaxone 2 g IV daily for LC. Lyme western blot IgG and IgM returned positive, confirming the diagnosis. An electrophysiology study was done due to the history of recurrent syncope and showed no infrahissian disease (normal H-V interval). The TPM was removed on day 3. Ceftriaxone was continued for 6 weeks as an outpatient. Eventually the patient regained intrinsic electric activity with a residual 1st degree AV block. Conclusion: LC should remain an important differential diagnosis in patients with syncope in endemic areas, even without other specific features of Lyme disease.
Sunnia Chen, MD Dr. Aurelio Vargas Dr. Hannah Nordhues	*A Rare Multiorgan Mimicker: HHV-8 Negative Castleman Disease* Case Description: A 21-year-old obese but otherwise healthy female presented with 3 weeks of unintentional 25 lb weight gain, progressive dyspnea on exertion, and generalized abdominal pain. Her symptoms rapidly progressed to include swelling in the upper and lower extremities, pain throughout the chest and abdomen, dark-orange urine and decreased urination, and dizziness upon standing. Admitted to an outside hospital, she was found to have rising alkaline phosphatase (ALP) peaking at 404 U/L, leukocytosis (14.2x109/L), normocytic anemia (hemoglobin 10.0 g/dL), thrombocytopenia (platelets 100x109/L), acute kidney injury (AKI) with creatinine 2.27 mg/dL, and C-reactive protein>320 mg/L. CT revealed diffuse lymphadenopathy, hepatosplenomegaly, body wall edema, and right perinephric stranding. She was treated with antibiotics for presumed pyelonephritis and an intra-abdominal infection causing reactive lymphadenopathy. She improved and was discharged. When she returned for follow-up a week later, labs again identified ALP of 415 U/L and AKI (Cr 2.18 mg/dL). She was admitted for recurrence of her symptoms. Repeat imaging demonstrated innumerable indeterminant lymph nodes. Phosphorus was 5.4 mg/dL, uric acid was 15.8 mg/dL, and lactate dehydrogenase was 323 U/L, raising concern for tumor lysis syndrome (TLS). Empiric treatment for TLS was initiated with initial improvement. PET-CT showed mildly FDG-avid nodes in the neck, axilla, iliac, and inguinal regions, with mild splenomegaly. Axillary lymph node fine-needle biopsy was obtained; however, results were negative for malignancy. Extensive workup for infectious, rheumatologic, malignant, and infiltrative causes remained negative; treatment for TLS was discontinued. Bone marrow biopsy showed nonspecific hypercellularity without evidence of malignancy. Kidney biopsy was considered due to nephrotic-range proteinuria but deferred due to improvement in kidney function. Ultimately, the patient underwent excisional lymph node biopsy demonstrating evidence of HHV8-negative Castleman disease, clinically consistent with TAFRO (thrombocytopenia, ascites, reticulin fibrosis, renal

	dysfunction, organomegaly) syndrome. She was initiated on steroids and siltuximab with significant improvement. Discussion: Castleman disease is a spectrum of lymphoproliferative disorders distinguished by regions of lymphadenopathy with characteristic histopathology and systemic inflammatory symptoms due to excessive production of pro-inflammatory cytokines. Multicentric Castleman disease (MCD) is classified based on HHV-8 status. HHV-8 positivity is commonly associated with immunocompromised states. It is unknown what causes HHV-8-negative, idiopathic MCD (iMCD), though many cases are associated with elevations in IL-6. While symptoms are often nonspecific, patients with iMCD may present with TAFRO syndrome or POEMS syndrome (polyneuropathy, organomegaly, endocrinopathy, monoclonal immunoglobulin spike, skin changes). In the absence of POEMS syndrome, treatment involves IL-6 inhibitors and high-dose steroids. Chemotherapy may be used for refractory disease. POEMS syndrome in general may be treated with radiation, chemotherapy, and/or stem cell transplant. As demonstrated in this case, iMCD is not easily diagnosed. Clinicians must maintain a high index of suspicion and pursue a multisystem evaluation. Diagnosis requires excisional lymph node biopsy, and all other inflammatory and/or lymphoproliferative diseases must be ruled out. Given the spectrum of organ dysfunction, patients should be referred early to tertiary care centers for expedited workup.
Christine Chen, MD Dr. Olivia Man Dr. Jenna Davidson Dr. Melissa Kerkellis Dr. Amos Lal **Abstract Finalist**	*Disseminated Mycobacteria in an Immunocompetent Man from Micronesia* Introduction: Tuberculosis (TB) is one of the leading causes of infectious-related deaths worldwide, though less commonly in the US. TB typically affects the lungs but can also include disseminated involvement in high-risk patients. Disseminated TB is rare in immunocompetent hosts who receive prompt treatment. Here we present a case of disseminated miliary TB in an immunocompetent patient in the US. Case Presentation: A 37-year-old man was admitted to the ICU with acute hypoxic respiratory failure accompanied by three months of malaise, abdominal pain, weight loss, and night sweats. He was malnourished, cachectic, and had moved to the US from Micronesia several years ago. One year prior to this admission, he presented to another facility for presumed pneumonia with empyema. He underwent thoracotomy with right lung decortication, thoracentesis with lymphocytic-predominant exudate, and bronchiolar lavage showing HSV1 and Enterobacter cloaca but negative AFB smear and mycobacterial culture. No TB PCR was performed. During admission to our ICU, Computed Tomography (CT) of chest was notable for innumerable bilateral pulmonary nodules. CT abdomen/pelvis was notable for diffuse nodular peritoneal thickening, enlarged mesenteric/retroperitoneal nodes, and bowel wall thickening. Thoracentesis was negative for malignancy. Sputum sample returned positive for rare acid-fast bacilli (AFB) on smear. PCR identified mycobacterium tuberculosis sensitive to rifampin. He was diagnosed with miliary TB and initiated on rifampin, isoniazid, pyrazinamide, and ethambutol, with rapid improvement in respiratory distress. However, serum drug levels were sub-therapeutic for three of his four medications, requiring therapeutic drug monitoring to ensure adequate dosing. Cultures eventually grew TB. All other bacterial/fungal workups were negative. Given his disseminated TB, workup for etiologies of

	underlying immunosuppression was undertaken but was largely unrevealing. He was leukopenic but HIV-negative. Autoimmune/immunology workup was unremarkable, including cytokine panels to assess for anti-cytokine autoantibody syndrome and other immunosuppressive states. He was discharged after 19 days, with close follow-up from the Department of Health to ensure ongoing therapy and prophylaxis for his family. Discussion: Our case highlights the presentation of disseminated TB in the lungs, pleura, intestines, and peritoneum in a previously healthy patient. His duration of infection without treatment likely contributed to his extensive presentation. He was exposed in Micronesia (TB prevalence of 80/100,000, compared to 3/100,000 in the US), though was not diagnosed until at least 4 years after arriving in the US. His malnourishment was another risk factor for disseminated TB. Furthermore, his negative AFB smear and culture at the outside hospital did not exclude diagnosis given the difficulty growing the organism. Indeed, his AFB smear during our admission was weakly-positive, but PCR revealed TB. Additionally, his sub-therapeutic medication levels were likely driven by TB bowel involvement increasing malabsorption, as well as poor nutritional status affecting pharmacokinetics. While he had early positive response to RIPE treatment, therapeutic drug monitoring in such high-risk patients is a critical yet often under-utilized tool to prevent future treatment failure and drug resistance. Overall, this case highlights the importance of integrating a patient's full history, disease state, and risk factors into effective TB diagnosis and treatment.
Anne Christiansen, MD Dr. Amy Holbrook	*From Pink Eye to ECMO: A Severe Case of Adenovirus* Introduction: Adenovirus is a well-known and common cause of mild febrile illnesses, particularly in young children. Most infections are self-limited, however, they can be severe in immunocompromised patients. Upper respiratory infections are the most common presentation, though other manifestations from adenovirus are possible. These include pneumonia as well as ophthalmologic, neurologic, genitourinary, gastrointestinal, and disseminated infections. Case Presentation: A 34-year-old female with history of type 2 diabetes, idiopathic orbital inflammatory syndrome (on daily mycophenolate and prednisone) presented for 1-2 weeks of cough, shortness of breath, fever, recent conjunctivitis, and diarrhea. She initially was diagnosed with pneumonia in the ER and discharged on cefuroxime and doxycycline. She returned for worsening symptoms and was found to be hypoxic and febrile. Physical examination showed moderate respiratory distress, diffuse coarse lung sounds, normal oropharynx and eye exams. CTA chest showed a dense left lower lobe consolidation. She was transitioned to levofloxacin and admitted. She subsequently developed worsening hypoxia and repeat imaging showed worsened infiltrates. Antibiotics were broadened and steroids were started. She ultimately required admission to the ICU for intubation. Her respiratory status deteriorated further, progressing to ARDS. She was started on ECMO. Thorough infectious workup was done including fungal studies, bronchoscopy with alveolar lavage, and cultures. Respiratory panel was positive for adenovirus and serum adenovirus PCR indicated a high viral load. With no other pathogen found, she was started on cidofovir and IVIG for adenovirus pneumonia. She slowly improved over about 5 weeks, although developed other complications including superimposed bacterial pneumonia, septic shock, and RV failure. Adenovirus PCR viral load slowly trended down. She was decannulated from ECMO and

	underwent tracheostomy. She remained on cidofovir until resolution of viremia, a total of 40 days. She was discharged to a long-term acute care hospital for ventilator weaning and successfully weaned to nasal cannula. She continued to need rehab for severe deconditioning. Conclusion: With the range in clinical presentation and illness severity in adenovirus infections, it is important to recognize adenovirus as a possible etiology for a severe infection. This is especially important in immunocompromised hosts where adenovirus infection can have considerable morbidity and mortality. Antivirals and often adjunctive IVIG can be used to treat severe illness. Cidofovir is the most commonly used antiviral, however, it requires close monitoring for adverse effects such as nephrotoxicity. Little is known about how long to treat with antivirals, but current practice is to typically treat until resolution of symptoms or viremia.
Lauren Clements, MD Dr. Breanna Zarmbinski Dr. Anya Jamrozy	*Cardiac Tamponade as Presentation of Undifferentiated Connective Tissue Disorder* Introduction: Definitive diagnosis in rheumatologic disease is challenging as serologic testing is often difficult to interpret and there is considerable overlap of disorders. Case Presentation: A 35-year-old woman presented with five days of worsening dyspnea one month after giving birth at 36w4d following a pregnancy complicated by pre-eclampsia. The patient had a history of ambiguous rheumatologic diagnoses self reporting distant diagnoses of Lupus and Rheumatoid Arthritis. The patient had six first trimester pregnancy losses but negative antiphospholipid antibodies. Available records showed a prior diagnosis of Lupus after findings of positive ANA (1:160) and anti-centromere antibodies (1:640); the patient was then lost to follow-up. Subsequently, consideration of systemic inflammatory disease was considered but the patient was ultimately diagnosed with fibromyalgia and chronic pain disorder. At that time labs included ANA 1:160, Negative RF, Sm, RNP, SSA, SSB, Scl-70, dsDNA, B2GP, aCL, MPO, PR3. At the time of presentation, the patient reported cough, lightheadedness, muscle cramps, chest heaviness, fatigue, and new areas of skin hypopigmentation in addition to her chief complaint of dyspnea. Exam revealed a tired appearing female, distant lung sounds, 3+ pitting edema in bilateral legs, regular heart rate and rhythm, pain on palpation of legs and hands, and hypopigmented areas of her forehead, nose, and cuticles. Labs significant for anemia (8.5), leukopenia (3.2), elevated d-dimer (2.93), and urinalysis with >300 protein and 11-25 red blood cells. TTE showed moderate-large circumferential pericardial effusion with findings of early tamponade. A pericardiocentesis removed 640 cc of transudative fluid and persistent effusion required drain placement. Cardiac MRI showed myocardial edema without significant fibrosis--findings not typical of myocarditis but were attributed to ongoing inflammation. Significant proteinuria and LE swelling raised concern for lupus nephritis vs scleroderma crisis, but renal biopsy revealed changes consistent with preeclampsia/severe HTN. Further lab workup revealed elevated ESR (1.51) and CRP (32), ANA 1:1280 with nucleolar staining, and negative rheumatologic workup including ANCA, CCP, RF, Scl-70 antibodies, C3, C4, anti-dsDNA, SM antibody, RNP antibody, ro52, Ro60, ss-B antibody and lupus anticoagulant. She was diagnosed with undifferentiated connective tissue disease and was discharged home with planned multidisciplinary follow up. She was hospitalized ~2

	months later with proximal muscle weakness followed by worsening cardiac function leading to multiple PEA arrests and ECMO cannulation. Her disease eventually evolved to fit a diagnosis of mixed connective tissue disease and was initiated on prednisone, Cellcept and Rituximab. She survived and continues to slowly recover. Conclusion: This case illustrates the importance of maintaining a broad differential in rheumatologic workup as early definitive diagnosis can limit repeat serologic testing that might be indicated by evolving disease. High ANA titers or presence of cytopenias at baseline are the leading factors associated with evolution to definite connective tissue disorder (CTD), but many patients remain undefined or have overlap syndromes. CTD has a high risk of preeclampsia, thromboembolism and death. It is possible that maintaining a broad differential with repeat serologic testing may have led to earlier identification of this patient's rheumatologic disease.
Brett Cornforth, MD Dr. Charlie Reznikoff	*Oritavancin Infusions for MRSA Tricuspid Endocarditis in a 27-Year-Old with IV Substance Use Disorder: Reducing Extended Hospital Stays Solely for IV Antibiotic Administration* Background: Infectious endocarditis (IE) is infection of the endocardium most often affecting the left-sided heart valves, while the tricuspid valve is affected in around 10% of cases. Tricuspid endocarditis typically occurs in patients with intravenous (IV) drug use, implanted cardiac device, or underlying structural pathology. IE typically requires extended antibiotic therapy following discharge, and patients who require IV antibiotics to complete the course often require peripherally inserted central catheter (PICC) lines for antibiotic delivery or frequent outpatient infusions. In populations where the above outpatient antibiotic plans are not possible, hospital stays are extended to deliver IV antibiotics. Case Presentation: 27-year-old woman with history of IV drug use including methamphetamine and fentanyl, untreated sexually transmitted infections, and homelessness presented with opioid withdrawal and was found to have Methicillin-Resistant Staphylococcus Aureus (MRSA) bacteremia. There was high concern for tricuspid valve endocarditis which was confirmed on transesophageal echocardiogram with a 2.2 cm vegetation identified. She also had septic emboli to the bilateral lungs, left shoulder, and left sacroiliac joint which were related to a persistent foramen ovale. Her MRSA infection was initially treated with Vancomycin which was switched to Daptomycin due to safety concerns as she declined daily labs. She had 2 weeks of negative blood cultures before becoming febrile and having positive cultures which were Daptomycin-resistant and Vancomycin-intermediate. Antibiotics were switched to Ceftaroline with plans for 6 weeks of treatment from the last positive cultures. Her hospital course was further complicated by a positive urine drug screen for fentanyl attributed to in-hospital IV drug use. Due to these concerns as well as her significant IV drug use history, she was not considered a candidate for PICC line placement. She became medically stable while on Ceftaroline treatment, and following debridement of her septic left shoulder, was only remaining in the hospital to receive IV antibiotics, with a further 5 weeks of treatment needed after she attained medical stability. She was accepted to residential chemical dependency (CD) treatment pending discharge, but her facility was not going to allow her to miss treatment for lengthy antibiotic infusions. After 1 week of complicated discharge planning, Oritavancin infusions were approved following susceptibility testing. 2 infusions were given inpatient which gave her 4

	weeks of antibiotic coverage, which completed her treatment course. Approval of Oritavancin shortened her hospital stay by 26 days and allowed for seamless transition to her CD program. Discussion: Infectious endocarditis typically requires weeks of antibiotics after discharge from the hospital. When the disease is further complicated by antibiotic resistance and contraindications for PICC placement and infusion availability, discharge planning can become difficult and extended hospital stays become necessary to facilitate completion of IV antibiotics. Antibiotics such as Oritavancin represent a solution to this problem as a two dose course in the hospital can provide 4 weeks of MRSA coverage without the need for a PICC or infusions after discharge. In addition to reducing health system costs by significantly reducing length stays, giving patients earlier access to CD treatment improves treatment outcomes.
Teni Davis, MD Dr. Katrina Willamson Dr. Ali Garcia Duarte	*Anakinra: An Overview of Use in Acute Gout with Individuals Who Failed or Have Contraindications to Conventional Therapy* Introduction: Acute gout flares may be treated with NSAIDs, colchicine, or systemic or intra-articular glucocorticoids. These treatment options are effective for most healthy individuals without multiple comorbidities. However, alternative therapy may be necessary for elderly patients, those with prednisone intolerance, poor kidney function, or complex comorbidities. Anakinra is an interleukin 1 (IL-1) inhibitor FDA-approved for moderate to severe rheumatoid arthritis but has shown to be safe and effective in the management of acute gout flares, especially in patients with contraindications to conventional therapy. Case Description: A 96-year-old woman with medical comorbidities of hypertension, coronary artery disease, heart failure with preserved ejection fraction, and chronic venous stasis edema presented with bilateral foot pain, worse with weight-bearing, that started after an abrasion to the right malleolus. Vitals were normal aside from a temperature greater than 38°C. Exam demonstrated a tender, edematous, and erythematous right foot with pain worse at the 1st MTP, skin desquamation, and tenderness over the plantar surface of the left foot. Notable labs included uric acid of 10 mg/dL, erythrocyte sedimentation rate 69 mm/hr, C-reactive protein 327 mg/L, and Creatinine 1.42 mg/dL. X-ray of bilateral feet showed diffuse soft tissue swelling of the right and left feet with severe arterial calcifications. Aspiration of the 1st MTP and midfoot was unsuccessful. Dual-energy CT revealed gout arthropathy with moderate scattered monosodium urate burden in the right foot clustered in the 1st MTP and 1st IP joint along with small erosive changes. Mild scattered monosodium urate burden was present in the left foot along the soft tissue of the medial ankle, medial talus, and plantar surface of the 2nd MTP without erosions. Calcium pyrophosphate deposition was also present in both feet without findings of osteomyelitis. The patient and family endorsed significant delirium complicating prior prednisone exposure. NSAIDs and colchicine were contraindicated due to her renal disease. Her acute gout flare was managed with a five-day course of Anakinra, dosed every other day due to her low creatinine clearance, with subsequent improvement in bilateral foot symptoms. Discussion: First-line therapy for acute gout flares includes systemic or intra-articular glucocorticoids, colchicine, or NSAIDS. However, in patients with contraindications to these therapies or severe, refractory gout, IL-1 receptor antagonists, like anakinra or canakinumab, may be preferred. Anakinra is

	preferred in cases complicated by significant renal dysfunction, with prior studies demonstrating safety and efficacy in this population and in those with concomitant infection[1]. Reduced frequency dosing is recommended in those with severe renal impairment or end-stage renal disease with a creatinine clearance of less than 30 mL/min[2].
Jenna Davison, MD Dr. Christine Chen Dr. Laura Kek Dr. Olivia Man Dr. Hilary Dubrock Dr. Henry Cajigas	*A Gastroenteritis Prodrome and the Differential Shadow Cast by Rhabdo* Introduction: Rhabdomyolysis is the dissolution of skeletal muscle leading to electrolyte abnormalities and renal failure. Common causes include traumatic injury, overexertion, drugs, toxins, and genetic defects. While respiratory and gastrointestinal infections have been shown to trigger rhabdomyolysis in pediatric or immunodeficient adults, cases in immunocompetent adults are rare. A review of the last 25 years of case reports found four cases of enterovirus induced rhabdomyolysis in healthy adults resulting in acute organ failure. Case Presentation: A 27-year-old male with no medical history presented with three weeks of progressively worsening vomiting, diarrhea, sore throat, proximal muscle weakness, and myalgias. Initial laboratory studies demonstrated an elevated CK 40,000 U/L (39-308 U/L), ALT 205 U/L (7-55 U/L), and AST 2017 U/L (8-48 U/L). The patient denied any overexertion, ingestion of drugs or toxins, or family history of neurological or muscular disorders. He developed oliguria, nephrotic syndrome, and hyperkalemia to 5.9 mEql/L (3.6-5.2 mmol/L) and required renal replacement therapy. Subsequently, due to uncontrolled hyperkalemia of 7.5 mEq/L, he suffered a PEA arrest, with return of spontaneous circulation after effective CPR. Follow-up labs demonstrated a potassium of 5.5 mEq/L, creatinine of 2.21 mg/dL (0.74-1.35 mg/dL), and CK of 115,560 U/L. An extensive workup included but was not limited to negative testing for a urine drug screen, ANA, ANCA, CMV, EBV, HIV, Histoplasmosis, beta glucan, syphilis, Bartonella, Hantavirus, tick-borne illnesses, Babesia, Legionella, Hepatitis A/B/C, blood cultures, and leukemia/lymphoma. Karius, a microbial cell-free DNA sequencing test, was unrevealing. He had a positive respiratory PCR test for Enterovirus/Rhinovirus. Enterovirus was considered to be the culprit due to its stronger association with gastrointestinal illness. He received three days of IVIG and seven days of broad-spectrum antibiotics. His CK continued to rise and peaked at 183,140 U/L, 10 days after the initial presentation. Persistent hypotension requiring vasopressors was present through day 13. A muscle biopsy was delayed due to the poor diagnostic yield during active rhabdomyolysis. He recovered satisfactorily after a prolonged hospitalization. Discussion: This case represents an uncommon cause of rhabdomyolysis. The leading theory is a potential autoimmune or genetic susceptibility triggered by acute enterovirus infection resulting in severe rhabdomyolysis and multisystem organ failure. Common complications of enterovirus, specifically Coxsackie A and B, include encephalitis, aseptic meningitis, and myocarditis. Rhabdomyolysis is an extremely rare complication in an immunocompetent adult. It is postulated that the pathogenesis of viral myositis is due to invasion and necrosis, or cytokine storm. The receptor in which Coxsackie B binds has been found in renal tissue, which could explain our patient's acute renal failure; however, his enterovirus was unable to be speciated. Diagnosis of enterovirus can be performed through PCR in serum and urine. Physicians should be aware of the potential life-threatening complications of enterovirus infection.

Jose De Melo, MD Dr. Konstantinos Siontis Dr. Malcolm Bell	*Coronary Vasospasm as a Cause of Sudden Cardiac Death* Introduction: Coronary vasospasm can be a cause of acute coronary syndrome in patients with otherwise non-obstructive or absent coronary artery disease (CAD). Rarely, it may also be the cause of malignant ventricular arrhythmias and sudden cardiac death. Case presentation: A 59-year-old male with no past medical history was referred for evaluation of recurrent polymorphic ventricular tachycardia (PMVT). Two months prior to his presentation, he experienced an episode of syncope with a prodrome of resting dyspnea and left biceps pain. A week later, he suffered an out-of-hospital cardiac arrest preceded by similar resting symptoms. Inpatient cardiac telemetry demonstrated episodes of PMVT. Coronary angiogram revealed only mild obstructive CAD with a tubular 60% lesion in the proximal right coronary artery (RCA), and cardiac magnetic resonance imaging showed no structural abnormalities or delayed gadolinium enhancement. Genetic testing demonstrated no pathogenic variants. Repeat coronary angiogram showed severe diffuse vasospasm involving the proximal and mid RCA during intracoronary assessment with Optical Coherence Catheter which resolved after intracoronary nitroglycerin. An implantable cardiac defibrillator was implanted. Nitrates and amlodopine were initiated but the patient experienced recurrent PMVT requiring multiple ICD shocks prior to referral to our institution. Review of cardiac monitoring information was suggestive of ST segment elevation preceding the episodes of PMVT, confirming the suspicion of coronary vasospasm and acute ischemia as the cause of PMVT. He underwent repeat coronary angiography and intravascular ultrasound-guided percutaneous coronary intervention with a drug-eluting stent of the 60% proximal eccentric RCA plaque. He was initiated on twice-daily high dose long-acting diltiazem, isosorbide mononitrate was up titrated to maximum tolerated dose, and high dose atorvastatin was started. At 6-week follow-up, he had complete resolution of anginal symptoms and no recurrent arrhythmias. Discussion: Coronary vasospasm should be considered in young patients presenting with cardiac arrest. The causal association between atherosclerosis and coronary vasospasm is well documented. Generally, medical management of vasospasm is recommended. However, in treatment-refractory cases where focal spasm is documented in an atherosclerotic coronary segment, additional consideration of stent placement may be necessary.
Madeline DeAngelo, MD Dr. Jill Huber **Abstract Finalist**	*When Should Sinusitis Make Us Pause?* Case Presentation: A 34-year-old man presented to clinic with a one-month history of sinus pressure exacerbated by bending forward and exertional shortness of breath. He had a history of childhood asthma and allergic rhinitis. He had been seen in urgent care five days prior to presentation where he was prescribed a prednisone burst and albuterol for presumed acute asthma exacerbation without symptom improvement. In clinic, he was prescribed amoxicillin-clavulanic acid for presumed bacterial sinusitis. He subsequently returned to clinic with worsening positional sinus pressure across the frontal region and a non-productive cough. Review of symptoms was negative for fever, orthopnea, weight changes, rash, joint pain, muscle aches. Lab work was unremarkable, including a complete blood cell count, a

	comprehensive metabolic panel, and a c-reactive protein. A MRI brain was ordered. He was prescribed a course of doxycycline for presumed ongoing bacterial sinusitis. Nine days later, he was reevaluated in urgent care for continued sinus symptoms and a new episode of dizziness. He was prescribed azithromycin again for bacterial sinusitis. Three days after being prescribed the course of azithromycin, he returned to our internal medicine clinic. At the time of this visit, it was three weeks from initial urgent care presentation. He endorsed new, atypical lightheadedness with vertiginous features, neck fullness, and facial flushing after standing from being supine. On physical exam there was no tenderness to palpation of the sinuses, appreciable lymphadenopathy, or hepatosplenomegaly. Lungs were clear to auscultation, but the patient coughed while supine. Given persistent symptoms despite treatment and progressive symptoms of sinus pressure, facial flushing, and neck fullness, a chest x-ray was obtained to look for venous obstruction. This demonstrated a large heterogeneous mediastinal mass. A chest CT followed showing a large anterior mediastinal mass measuring 9 x 12 x 19 cm with obstruction of the superior vena cava. CT-guided biopsy of the mass demonstrated primary mediastinal diffuse large B cell lymphoma. The patient was admitted for induction chemotherapy. Conclusion: Superior vena cava syndrome is caused by an obstruction or restriction of blood flow via direct invasion, external compression, fibrosis, or indwelling device. Intrathoracic malignancies are responsible for the vast majority of superior vena cava syndrome, most commonly lung cancer, lymphoma, thymic neoplasms, primary mediastinal germ cell neoplasms, and lymph node metastases. As seen in this patient, primary mediastinal diffuse large B cell lymphoma remains an aggressive subtype of non-Hodgkin's Lymphoma that frequently presents with superior vena cava syndrome. Symptoms of superior vena cava syndrome often include dyspnea, face or neck swelling, headache, visual or auditory disturbances, and a feeling of fullness of the head exacerbated by bending forward or lying down. Confirming superior vena cava syndrome often begins with imaging with a radiograph or CT to determine the compartment of the mass, with more specialized labs or biopsy following. As with this and any patient, we must pursue further workup of worsening sinus type symptoms that are refractory to multiple treatments.
Naba Farooqui, MD Dr. Jose De Melo Dr. Jeffrey Yang Dr. Adam Sawatsky	*Prosthetic Valve Fungal Endocarditis: Lessons for the Internists* Background: Histoplasma endocarditis is rare and accounts for less than 1% of all cases of endocarditis and 2% of prosthetic valve endocarditis. It may present with fevers, new-onset murmurs, pancytopenia and splenomegaly, however, majority of the clinical findings overlap with systemic histoplasmosis. There are currently no guidelines regarding screening for endocarditis in cases with disseminated histoplasmosis. Case Presentation: A 74-year-old male with coronary artery disease and stage 3b chronic kidney disease presented with subacute generalized weakness and weight loss. Past history was notable for a 2-vessel coronary artery bypass graft surgery with concomitant bioprostethic aortic valve (AV) replacement and left atrial appendage ligation 2 years prior to presentation. Six weeks prior to presentation, he developed abrupt-onset fatigue, exertional dyspnea, dry cough, evening rigors, and intermittent fevers to 101oF. He also had a 7-pound unintentional weight loss.

	Physical exam was notable for cachexia. Laboratory workup revealed stage I acute kidney injury and mild hypercalcemia. Monoclonal gammopathy screen was negative. Diffuse pulmonary groundglass opacities and splenomegaly were noted on CT imaging, with positive serum antibodies, and urine antigen tests for Histoplasma capsulatum. He was initiated on itraconazole for histoplasmosis and due to lack of clinical improvement and in view of prosthetic AV, a transesophageal echocardiogram was performed, demonstrating a 6 mm prosthetic AV vegetation. He was subsequently noted to have cerebellar infarcts, superior mesenteric artery occlusion, and splenic infarcts on subsequent CT and MR imaging. Blood cultures ultimately grew Histoplasma capsulatum after a few weeks. Due to poor surgical candidacy, our patient was medically managed with liposomal amphotericin B and itraconazole with protracted symptoms and limited recovery. Discussion: Fungal endocarditis carries mortality rates approaching 50% and as seen in our patient, history of open heart surgery and residence in an endemic area are important risk factors. Our case offers an example of how Histoplasma endocarditis can present, and the importance of considering an echocardiogram in every patient with disseminated histoplasmosis particularly with prosthetic valves and organomegaly, irrespective of reassuring cardiac exam and initial blood culture results, as this can be key in determining prognosis and governs the duration and type of treatment in such cases.
Iuri Ferreira Felis, MD Stefan Farrugia Dr. Jason Szostek	*The Abscess is Where? A Seminal Case Report* Introduction: Seminal vesical abscesses (SVA) are extremely rare urologic infections. They commonly present with fever, dysuria, suprapubic or perineal pain, and prostate tenderness on digital rectal examination (DRE). Risk factors include indwelling catheters, immunocompromised states, malnutrition, alcoholism, and benign prostatic hyperplasia (BPH). Case Presentation: We present the case of an 89-year-old male who presented to the emergency department (ED) with progressive left lower quadrant (LLQ) abdominal pain. His past medical history was notable for chronic kidney disease stage IV, BPH with obstruction requiring scheduled in-and-out urinary catheterizations, and recurrent urinary tract infections, including a left scrotal abscess two years prior. He reported 3 weeks of non-radiating LLQ pain, worsening in the last 4 days, with tenesmus as his only other symptom. He denied an inciting event, fevers, rigors, night sweats, nausea, vomiting, or other gastrointestinal symptoms. Notably, the patient had a history of elevated PSA since 2014, peaking at 38 ng/mL (reference range 7.2 ng/mL) in 2016, and most recently elevated at 28 ng/mL in 2017. He had repeatedly declined diagnostic and therapeutic procedural interventions. On presentation, his temperature was 36.6 °C, pulse 69 bpm, and blood pressure 116/67 mmHg. Physical exam was significant for left lower quadrant pain and an enlarged, non-tender prostate with a prominent left-sided apical nodule. Labs demonstrated leukocytosis of 12.9 x109/L, (reference range 3.4 to 9.6 x109/L), patient's chronically elevated creatinine of 2.54 mg/dL (reference range 0.74 to 1.35 mg/dL), elevated C-reactive

	protein (CRP) of 84.9 mg/L (reference range <5.0 mg/L), and PSA of 104 ng/mL. Urinalysis was positive for leukocyte esterase, nitrites, pyuria, bacteriuria, and proteinuria. Urine gram stain was positive for many gram-negative bacilli. CT with contrast demonstrated cystitis, prostatitis, and a 4 x 4 cm left seminal vesicle abscess. Urology was consulted and recommended no acute intervention. An indwelling urinary catheter was placed, he was started on intravenous ceftriaxone, and scheduled for percutaneous abscess drainage. On the medicine floor, he developed a fever of 38.1 oC, which resolved with his LLQ pain following left transgluteal percutaneous abscess aspiration and drain placement. Blood cultures remained negative. Urine cultures grew Klebsiella oxytoca, Raoultella ornithinolytica, and Raoultella planticola while abscess aspirate cultures grew Escherichia coli. The patient was switched from ceftriaxone to oral ciprofloxacin with plans for a 6-week course. He was discharged from the hospital, and the abscess drain was removed 17 days following placement. Ambulatory prostate ultrasound showed marked BPH (80cc). A repeat PSA and prostate MRI are scheduled for mid-October. Discussion: There are very few cases of SVA reported in the literature. To our knowledge, this is the first case reported of an immunocompetent patient with no chronic indwelling catheter. Diagnosis requires a high index of suspicion and typically necessitates pelvic CT, MRI, or transrectal ultrasound. DRE is usually non-specific and may show prostate tenderness and enlargement. Treatment typically includes abscess drainage and an extended course of prostate-penetrating antibiotics covering gram negative bacteria.
Joelle Friesen, MD Dr. Matthew Howard Dr. Terin Sytsma	*Of All the Nerve: An Uncommon Cause of Progressive Neuropathy and Myopathy* Case Presentation: A 67-year-old man with medical comorbidities including hypertension, hyperlipidemia, and depression was admitted with progressive weakness. Symptoms started 4 months prior to presentation with bilateral lower extremity numbness and weakness that progressed from distal to proximal. By the time of admission, he was no longer able to walk due to weakness and ataxia. He also endorsed early satiety with a 70-pound weight loss and three weeks of urinary retention requiring placement of an indwelling catheter. He denied fevers, chills, night sweats, paresthesias, myalgias, saddle anesthesia, lightheadedness, headaches, back pain, or radicular pain. Initial physical exam was notable for mild diffuse weakness in the upper extremities, proximal greater than distal weakness in the lower extremities, length-dependent sensory changes in the lower extremities to the bilateral knees, and absent reflexes at the bilateral ankles and the right knee. In short, the exam demonstrated progressive flaccid quadriparesis. On hospital day 4, he also developed new diplopia, left sixth nerve palsy, and dysarthria. Initial laboratory workup was unrevealing, including negative infectious and autoimmune evaluations. MR imaging of the brain and spine was significant for abnormal patchy leptomeningeal enhancement of the bilateral cerebral and cerebellar hemispheres, brainstem, fifth-eighth cranial nerves, and cervical, thoracic, and lumbar spine. PET CT showed FDG-avid subcarinal, porta hepatic,

	pericaval, and peritoneal adenopathy. Electromyography demonstrated nonspecific generalized neuropathy. Lumbar puncture revealed a normal opening pressure, elevated total nucleated cells (437; normal 0-5) with 78 percent lymphocytes, elevated total protein (318; normal 0-35), hypoglycemia (<20 with serum glucose 92; normal approximately 60 percent of serum glucose), and elevated oligoclonal bands (5; normal <2). Bacterial, mycobacterial, fungal, herpes simplex, Histoplasma, Blastomyces, and Cryptococcus testing as well as a meningitis-encephalitis panel, neuroimmunology panel, and cytology were negative. One of the FDG-avid retroperitoneal lymph nodes was biopsied; this revealed extensive fibrosis and non-caseating granulomas consistent with sarcoidosis. He was initiated on a 5-day course of IV methylprednisolone followed by a prednisone taper as well as infliximab once infectious and neoplastic workups were negative. Symptoms gradually improved with this regimen, and he was discharged to a rehabilitation facility with neurology follow-up. Conclusion: Sarcoidosis is an immune-mediated disease characterized by granulomatous inflammation. Pulmonary involvement is most common and is seen in approximately 90 percent of cases, but approximately 5-10 percent of patients have neurologic involvement. Neurosarcoidosis has a variety of presentations, including peripheral neuropathy, cranial neuropathies, seizures, radiculopathy, meningitis, hydrocephalus, ataxia, and myopathy. Neurosarcoidosis can follow a relapsing-remitting, monophasic, or progressive course. There is no singular diagnostic marker; typically, histology demonstrating noncaseating granulomas is confirmatory. CSF testing can yield a variety of abnormalities, including elevated total protein, pleocytosis, low-normal glucose, elevated IgG index, and oligoclonal bands. Contrast-enhanced MRI is necessary to visualize neurologic inflammation secondary to sarcoidosis. Additionally, PET can be helpful in identifying sites of involvement. As sarcoidosis is an inflammatory process, corticosteroids are the mainstay of treatment. Additionally, immunosuppressants, biologic agents such as infliximab, and low-dose radiation may be used in severe or refractory cases.
Cameron Gmehlin, MD Dr. Grace Hagan Dr. Alex Liu Dr. Aditya Devalapalli	*Superficial Venous Thrombosis as a Complication of Diabetic Nephrosclerosis* Introduction: Thromboembolism is a potentially life-threatening complication of nephrotic syndrome seen in up to 25% of patients. The pathophysiology is thought to be multifactorial and occurs in the setting of hemostasis-related protein loss with compensatory increase in synthesis of hemostasis-related proteins, leading to a prothrombotic state. Here we present a case of nephrotic syndrome that was diagnosed after a patient developed bilateral lower extremity superficial venous thrombosis (SVT). Case Presentation: A 40-year-old male presented for evaluation of bilateral lower extremity pain and swelling. His medical history included Type 2 Diabetes complicated by cataracts and diabetic retinopathy, CKD Stage 3 (creatinine 1.7), Hypertension on Amlodipine and Lisinopril, and Obesity. He reported development of progressive lower extremity swelling, fatigue, and shortness of breath over one month. More acutely, he developed bilateral thigh and knee pain. In the Emergency Department, he was hypertensive to the 140s/100s mmHg and tachycardic to 110 bpm but otherwise afebrile and saturating well on room air. The physical exam was remarkable for inspiratory crackles to bilateral lung bases, 4+ pitting edema to his bilateral

	lower extremities, varicose veins, and a palpable tender cord of the left inner thigh. Initial laboratory analysis revealed microcytic anemia, elevated creatinine to 4.65 with an eGFR of 15, and albumin of 1.5. A lower extremity ultrasound was obtained, showing extensive thrombosis of the right great and small saphenous vein approaching their confluence with the femoral vein and the left anterior accessory saphenous vein. He was started on unfractionated heparin IV infusion and admitted to the floor. Upon admission, urine studies were obtained which showed free fat/oval fat bodies, granular/waxy casts, and a predicted 24-hr protein of 16,826 mg. Renal ultrasound was obtained which showed normal appearing kidneys. PLA2 receptor/thrombospondin A2 receptor antibodies and HBV/HCV/HIV PCR testing were negative. Nephrology was consulted and kidney biopsy was performed, which showed diffuse diabetic glomerulosclerosis. He was subsequently transitioned to warfarin and discharged with close outpatient follow-up. Discussion: Patients with nephrotic syndrome are at considerable risk for thromboembolic events. In a Danish registry study, one-year absolute risk for arterial and venous thromboembolism was 4 and 3% respectively, and 10-year absolute risk was 14% and 8%. In the case of SVT involving the great saphenous vein, anticoagulation is indicated if the thrombus is extensive (5 cm) and/or approaches the deep veins (3 cm). Otherwise, conservative treatment with compression and NSAIDs can be attempted. Currently, clinical trials support the use of fondaparinux and rivaroxaban at prophylactic doses for treatment of symptomatic, high-risk SVT. However, both medications have significant renal excretion. In our patient's case, we chose to start anticoagulation with unfractionated heparin with transition to oral warfarin prior to discharge due to his creatinine clearance. Current guidelines recommend a total course of 3-6 months or continuation until the underlying illness has resolved. In summary, clinicians should maintain a high degree of suspicion for nephrotic syndrome in patients with longstanding diabetes, no matter their age, who present with findings suspicious for thromboembolic events.
Grant Goss, MD Dr. Sam Ives	*Severe Blood Loss Anemia a Few Drops at a Time* Introduction: Bedbug infestation is a known, but extremely rare cause of anemia. Here we present a case of recurrent anemia due to bedbug infestation where many alternative causes were evaluated and ruled out. Case Presentation: A 65-year-old man with schizophrenia presented to clinic with fatigue and lightheadedness. He was found to have normocytic anemia with a hemoglobin of 6.4 g/dL and was directly admitted. He had been admitted 3 years prior for anemia at which time he had had a bedbug infestation at his apartment. His prior workup included EGD and colonoscopy that were negative. His anemia had improved after decontamination of his apartment. On this presentation, he again underwent evaluation by the GI team to ensure he did not have occult blood loss. He had a negative EGD, colonoscopy, and capsule endoscopy. He was also seen by hematology and underwent a bone marrow biopsy that did not show any malignancy or an alternative cause of the anemia. He was again noted to have a bedbug infestation during this admission including bedbugs on his clothes. The patient and his home were

	decontaminated and he was discharged without requiring transfusion. Conclusion: This case demonstrates recurrent severe anemia where many potential causes were evaluated before confirming anemia secondary to bedbug infestation. Providers should be aware that while an individual bedbug bite causes minimal blood loss, repeated bites can add up to a profound blood loss.
Christin Grech, MD	*Acute Hepatitis B: To Treat or Not to Treat* Introduction: While there are estimated 14000 - 20000 cases of acute hepatitis B per year; only 2000-3000 cases are reported. [1] This discrepancy is due to the subclinical nature of the disease. [3] It is estimated that >95% of acute hepatitis B cases in immunocompetent adults clear infection spontaneously and <1% develop fulminant liver failure. [2] Given low rates of presentation and rapid clearance of virus the data is limited around antivirals utility in acute infection. Case Presentation: A 23-year-old female with an unremarkable past medical history presented with 5 days of progressive abdominal pain, nausea, and malaise. She was seen in ED 4 days prior and was diagnosed with an uncomplicated UTI. She was subsequently started on TMP-SMX and was discharged home. While she continued to take her antibiotics as prescribed, pain did not improve and approximately 2 days after being seen in the ED she noticed yellowing of her eyes, ongoing poor oral intake, and darkening of her urine. At which point she returned to the ED per direction of a coworker. Of note, she had no history of IV drug use or tattoos. She had no recent travel and did not consume uncooked meat. She is sexually active with one male partner. And has received all her childhood vaccinations. In the ED, liver enzymes were found to be significantly deranged with ALT 5009, AST 2145, and total bilirubin 9.9. Her INR was 1.9. Fortunately, her mental status remained intact. Initially work up was unrevealing for a cause of these significant abnormal labs; but ultimately she was found to have acute hepatitis b with positive core and surface antigen along with a positive IgM core antibody. Her case was discussed at length by the GI team for possible indication of treatment for acute hepatitis b, especially given her INR of 1.9. Ultimately, given her intact mental status and suspicion that the INR elevation was impart nutritional it was decided to monitor closely without antiviral treatment. Over the next few days; liver enzymes, oral intake, and INR improved without antiviral intervention. She was discharged home with close follow up. Discussion: At this point and time there are not clear guidelines for when to treat acute hepatitis b with antivirals; this may in part be due to the rarity of presentation as many cases are subclinical. While there are typical practices: INR > 1.5, prolonged period of jaundice, and acute liver failure it is ultimately up to the discretion of the team. [2] Furthermore, there is a theoretical benefit from not using antivirals as such medications may inhibit the production of neutralizing antibodies and therefore increase risk of chronic disease. [3]
Grace Hagan, MD Dr. Cameron	*Painful Ascites in the Setting of Known Systemic Lupus Erythematosus*

Gmehlin Dr. Alex Liu Dr. Mathias Palmer Dr. Chris Aakre	Introduction: Patients with Systemic Lupus Erythematosus (SLE) may present with ascites due to associated constrictive pericarditis, glomerulonephritis, CHF, portal hypertension. Extremely rarely, patients with SLE may present with ascites due to active Lupus Peritonitis (LP) in the absence of other SLE complications. Here, we present a case of LP in the setting of new-onset ascites in a patient with SLE and no known history of CHF, portal hypertension, constrictive pericarditis, or glomerulonephritis. Case Presentation: 46 y.o. female presented to the emergency department for evaluation of nausea, vomiting, fever, abdominal pain, body aches, bloating and neck pain. She had recently been experiencing increased urinary urgency, frequency, and dysuria for two weeks before these new symptoms developed. Patient had a past medical history of Roux-en-Y, SLE on hydroxychloroquine and leflunomide, HFpEF (LVEF 59%), fibromyalgia, and multi-substance use disorder. On physical exam, abdomen was soft, moderately distended, extremely tender in right upper quadrant with diffuse guarding. The patient had anicteric sclera, no jaundice on exam and no other cirrhotic skin findings. No asterixis. Kernig and Brudzinski signs were both positive. Patient was afebrile, tachycardic, normotensive, not tachypneic, normal oxygen saturation on room air. Lab results: lipase 6, lactate 1.8, Cr 3.14, Na 127, K 2.6, ALP 350, Protein 5.7, Albumin 2.8, PT of 36.5, INR 3.2, WBC 11.7, Hgb 10.9, Plt 91. UA positive for leukocyte esterase, >100 WBCs with bacteria present. CT resulted with large new volume fluid collection/loculated ascites surrounding the liver and tracking into the right paracolic gutter. The collection was associated localized mass effect with displacement of multiple loops of small bowel and the right kidney across midline. Blood cultures resulted in 3/3 bottles positive for E coli bacteremia. Liver Doppler was performed, which ruled out portal vein thrombosis. Given concern for possible meningitis, possible SBP, and active E Coli bacteremia with likely urinary source, all in the setting of immunocompromised host, patient was covered with broad spectrum antibiotics and acyclovir. Lumbar puncture did not show concern for meningitis. Paracentesis showed <50 PMNs, protein 4.0, negative Gram stain, SAAG 0.4. Despite findings of hepatic dysfunction, this indicated absence of portal hypertension and pointed toward exudative ascites. In the absence of bacteria within the fluid and in the setting of known SLE, it was determined that this was an auto-inflammatory exudate. We continued ceftriaxone for bacteremia. Her renal function improved and electrolytes normalized after volume expansion with colloid, and she was discharged in stable condition. Discussion: Rapid onset ascites in patients with known SLE, in absence of other diagnoses which result in development of ascites, should prompt consideration of LP. The pathophysiology of LP is poorly defined but includes the probable increase in vessel permeability due to immune complex deposition and circulating complement factors. LP has a lifetime occurrence rate of 8-11% in SLE patients. Previous case studies discuss the benefit of high dose steroid treatment in the setting of LP. LP is a rare, yet possibly fatal sequelae of SLE, and thus swift diagnosis and management should be prioritized.

Madison Hahn, MD Dr. Breanna Zarmbinski	*Coronary Compression Post Transcatheter Pulmonic Valve Replacement; A Rare but Serious Complication* Introduction: Transcatheter pulmonic valve replacements (TPVRs) are an effective treatment for patients with right ventricular outflow tract obstruction [1]. Transcatheter replacements involve placement of self or balloon expandable bioprosthetic valves delivered via central venous access [1]. TPVR can be utilized as an alternative to traditional cardiothoracic surgical interventions in the appropriate candidates. The most common complication of TPVR is a stent fracture, but other complications include infective endocarditis, conduit rupture, and, in 0 to 1 percent of cases, coronary artery compression [2]. Case Presentation: This case depicts a 27-year-old female with a history of ventricular septal defect (s/p patch disclosure), pulmonary valvotomy, PDA ligation, PFO closure, TPVR with 25 mm Harmony Valve (one month prior) who presented for evaluation of acute onset chest discomfort. The patient described the discomfort as a midline burning sensation worse with exertion that was associated with headache, shortness of breath and jaw pain. Initial EKG showed no evidence of ST changes, subsequent with anterolateral and inferior ischemic changes. Troponin rose from 100 to 345. Echocardiogram showed new left ventricular systolic dysfunction with EF 40-45%, apical hypokinesis and mild right ventricular systolic dysfunction. CT PE was negative. CTA showed impingement of the ostial left main artery by the TPVR. The patient underwent redo sternotomy with explantation of the Harmony valve, replacement with Inspiris 29 mm valve. She required monitoring in the cardiac ICU for 48 hours post-operatively. Post-operative coronary angiogram showed 20% stenosis in left main coronary artery (LMCA), normal left anterior descending artery (LAD), and normal left circumflex artery. She spent a total of 6 days in the hospital before discharging home with self-care. Conclusion: This case illustrates one of the less common complications of pulmonic valve replacements and emphasizes the importance of considering coronary pathology in patients who would otherwise be considered low risk (no history of hypertension, hyperlipidemia, diabetes, or other typical risk factors for coronary disease). Though stent fracture and endocarditis are more common complications of valve replacements, coronary compression can occur and lead to fatal outcomes if not promptly recognized [1,2].
Brian Harrison, MD Dr. John Ratelle	*Non-Traumatic Intramuscular Hematoma as an Atypical Cause of Leg Pain* Introduction: Pain is one of the most significant drivers of patients presenting to the emergency department, and even when pain is localized, the differential diagnosis can be vast. Trauma and its related sequelae are often easy to identify, but a non-traumatic hematoma can be an unexpected source of pain, and it should be considered in patients taking therapeutic anticoagulation. Case Presentation: We present the case of a 79-year-old woman who presented to the emergency department with two days of progressively worsening right shin and groin pain. An ultrasound of the bilateral lower extremities was obtained and demonstrated acute deep vein thromboses of both the right popliteal and femoral veins. Additionally, a large (approximately 7 x 8 x 13 cm) fluid collection was seen in the groin consistent with hematoma. She has a history of several malignancies

	(urothelial carcinoma, lung adenocarcinoma, colon adenocarcinoma) and was taking enoxaparin 150 mg daily due to a splenic infarct diagnosed in February, and clopidogrel related to her history of transcatheter aortic valve replacement. The patient's anticoagulation and antiplatelet therapies were held, and her leg was wrapped. An IVC filter was subsequently placed. Due to down-trending hemoglobin and persistent severe pain, interventional radiology and general surgery were consulted. CT imaging of the right lower extremity continued to demonstrate mild active venous extravasation with a question of a small focus of arterial bleeding for which the patient underwent embolization. The patient had subsequent stability of her symptoms, was eventually transitioned to apixaban 2.5 mg twice daily, and discharged in stable condition with outpatient follow-up to determine when to transition back to therapeutic anticoagulation. Conclusion: This case highlights the complexity and nuance of bleeding and thrombotic complications in a patient taking therapeutic anticoagulation. There are no clear guidelines on managing a spontaneous hematoma in the background of DVT, splenic infarct, and malignancy. Significant technical considerations also come into play because tamponade effect from the hematoma itself can confound attempts to identify a culprit bleeding lesion, and there are considerations regarding healing with any attempts at percutaneous evacuation. Extensive discussions with surgery, Interventional Radiology, and Cardiology were held to optimize this patient's anticoagulation and care, ultimately leading to clinical stability and a safe discharge.
Laura Hauff, MD Dr. Amy Holbrook	*An Uncommon Presentation of Achalasia Reveals Common Cognitive Bias* Introduction: Achalasia is an uncommon disease with prevalence of ~10/100,000 persons yearly. It results from degeneration of inhibitor neurons in the esophageal wall leading to lower esophageal sphincter failure and aperistalsis in the distal esophagus. The etiology of primary achalasia is unknown. It often develops gradually over years with patients often misdiagnosed with GERD that is refractory to PPIs. Dysphagia and regurgitation are the most frequent symptoms. Aspiration while recumbent occurs in approximately 8% of cases. Here we describe a case of probable achalasia presenting as an uncommon severe and acute cavitary pneumonia. Initial focus on this primary pulmonary process in the case lead to delay in care. This case demonstrates the harm of cognitive bias in medicine. Case presentation: 19-year-old female with history of asthma and GERD presented with one day of pleuritic chest pain, fever for three days, and two weeks of dry cough. Intermittent GERD symptoms for years. No wheezing or SOB. Moved from Arizona three months prior. No TB exposure. No drug use. Patient tachycardic (HR 112) and febrile (101.4F) on admission, but hemodynamically stable with normal O2 saturation on room air. WBC elevated at 17, with normal lactate, BMP, LFTs. EKG showed sinus tachycardia. D dimer was elevated, and subsequent CT PE study without pulmonary embolus, but did show a 4 cm thick-walled cavitary lesion in left lower lobe with surrounding airspace disease and small hydropneumothorax. Patient started on broad-spectrum antibiotics and placed in airborne precautions. Fever and leukocytosis resolved. Broad infectious disease workup: HIV, Covid-19, Quantiferon gold, AFB pleural fluid x 3, blood culture, histoplasma and blastomyces antigens, and coccidioides antibody - all negative. Pulmonology consulted and commented on dilated esophagus. Reassessment of radiologist's findings on CT noted esophagus distended

	with debris up to the level of the thoracic inlet (however, not noted in the impression). Bronchoscopy negative for neoplasm. On hospital day 4, patient developed recurrent fever and dyspnea. Chest imaging with increased hydropneumothorax and mediastinal shift. Urgent chest tube placed. EGD with food in middle and lower thirds of esophagus and abnormally tight lower esophageal sphincter, injected with botulism toxin. Pleural studies demonstrated exudative effusion, eventually growing coagulase negative staphylococcus. On hospital day 6, patient underwent VATS decortication, empyema drainage, and insertion of two large bore chest tubes without complication. Post-surgical CXR with expanded lung and no retained empyema. Started on soft dysphagia diet. Patient discharged on POD 4 with antibiotics for two weeks and doing well at follow up. GI plans barium esophagram and esophageal manometry to confirm presumed achalasia. Discussion: While achalasia is a rare condition, this case demonstrated an uncommon presentation with aspiration resulting in severe cavitary pneumonia with empyema. Additionally, this case also displayed common physician cognitive biases that delayed the ultimate diagnosis. The case provides a useful reminder for internists to review radiology reports and images thoroughly, as well as to keep in mind rare etiologies (i.e. achalasia) when treatment for common causes of symptoms (i.e. GERD) fail.
Mason Hinke, MD	*Klebsiella Meningitis and Ventriculitis in the Setting of Urosepsis and Bacteremia* Introduction: Klebsiella meningitis, while more common in Southeast Asia, is a rare cause of community-acquired meningitis accounting for less than 3% of cases annually. It is more commonly seen as a cause of hospital-acquired meningitis in post-neurosurgical patients. Case Presentation: Patient is a 54-year-old male with a history of muscular dystrophy who was admitted with altered mental status and shortness of breath. He was found to have Klebsiella pneumoniae and Pseudomonas bacteremia secondary to a urinary tract infection in the setting of a chronic suprapubic catheter. He was hypotensive upon admission but rapidly improved from a hemodynamic standpoint and was weaned off pressors. Due to persistent challenges with weaning sedation, MRI brain was ordered which showed fourth ventricle ventriculitis and basilar meningitis. Subsequent lumbar puncture confirmed this concern, with Klebsiella pneumoniae isolated on culture (sensitive to cephalosporins). He was then diagnosed with Klebsiella meningitis, despite being on ceftazidime for the previous five days. His antibiotics were broadened to meropenem. Despite aggressive antibiotics, he remained febrile and had a worsening neurological exam with concern for herniation. CT head was negative for worsening intracranial pathology. Neurosurgery was consulted and extraventricular drain was placed to allow for initiation of intraventricular gentamicin. Patient initially had improvement with IVT gentamicin and was able to follow commands. Unfortunately, his mental status slowly worsened and was no longer following commands or withdrawing to pain. Repeat MRI showed worsening hydrocephalus. He was transitioned to comfort care and ultimately passed away. Discussion: Here we discuss a rare case of klebsiella meningitis and

	ventriculitis in the setting of urosepsis and bacteremia. It is presumed that the patient developed a urinary tract infection and had bacteremia that resulted in seeding of his CNS. He has a remote history of T3-sacral spine fusion due to his history of muscular dystrophy, and while this was not confirmed on imaging, it is presumed that this may be playing a role as to why he developed meningitis. The role of intraventricular antibiotics can be beneficial. For individuals with multi-drug resistant organisms, implanted devices that cannot be removed or for those who do not respond to initial therapy, placement of an EVD should be considered.
Kanchan Hulasare, MD Dr. Amy Holbrook	*Disseminated Histoplasmosis* Introduction: Histoplasmosis is caused by the dimorphic fungus Histoplasma capsulatum. The soil-based fungus is endemic to the Ohio and Mississippi River valleys and in the southeastern United States. It can cause disseminated disease in the immunocompromised with the ability to affect multiple organ systems. In immunocompetent individuals Histoplasma is typically asymptomatic or a self-limiting pulmonary infection, however disseminated disease is possible. Disseminated disease can present with vague symptoms including anorexia, fatigue, weight loss, lymphadenopathy, skin lesions. Interestingly, adrenal involvement is common in disseminated disease. Case Description: A 52-year-old male with a past medical history of thrombophilia, femoral artery occlusion status post left above knee amputation presented to the emergency department with six months of worsening painful mouth sores, lip ulceration, dysphagia, hoarse voice, and weight loss. He also reported three days of left arm paresthesias, shortness of breath, and lightheadedness. Initial lab workup revealed sodium 129, potassium 2.5, lactate 2.4, HIV negative, rapid strep A negative. Exam was notable for large ulcerations on both lips with swelling and a larger crusted lesion on the lower lip and white plaques on the roof of the mouth. ENT was consulted, fiberoptic scope showed distortion of epiglottic contour but was limited due to amount of saliva and exudate. HSV swab of the lip was negative. A punch biopsy of the lip ulceration was taken and he was empirically started on prednisone. CT Neck showed diffuse edema of lips, soft palate, epiglottis, and aryepiglottic folds. CT Chest/Abdomen revealed centrilobular pulmonary nodules, some of which were cavitary as well as new bilateral adrenal masses. Additional workup included negative AFB sputum testing, negative antiSmith and ds-DNA, negative treponema pallidum, and negative ANCA. Biopsy pathology showed an ulcerated abscess with granulomatous inflammation and budding yeast consistent with Histoplasma. He was started on itraconazole for treatment of disseminated histoplasmosis. His lip ulceration and oral pain significantly improved after initiation of treatment and he was discharged home on hospital day 9. Conclusion: This case illustrates the importance of considering Histoplasmosis in patients residing in endemic regions who present with fatigue, weight loss, and other nonspecific symptoms. New unilateral or bilateral adrenal enlargement on imaging may be a clue to the diagnosis of Histoplasmosis.

Jack Inglis, MD	*Swelling Mystery: A Case of Urticarial Vasculitis Masquerading as Hereditary Angioedema* Introduction: Hereditary angioedema (HAE) is a rare genetic disorder characterized by recurrent episodes of severe swelling in the subcutaneous and submucosal tissues, often manifesting as facial edema and abdominal pain. Urticarial vasculitis (UV) is a small vessel vasculitis that presents with persistent, painful urticarial lesions that may be accompanied by systemic involvement of the kidneys, lungs, and GI tract. While these two conditions typically have distinct clinical presentations and objective findings, they can share key features. Here, we present a case of a young man with a history of hereditary angioedema who did not improve with typical therapy, leading to the broadening of the differential and, ultimately, to the diagnosis of urticarial vasculitis. Case description: A 32-year-old male with a history of hereditary angioedema presents with one day of worsening edema, abdominal pain, and a diffuse rash. He was hypertensive on arrival (systolics ~170s). On exam, he was noted to have swelling of the lips, tongue, and hands in addition to an annular, erythematous rash involving the trunk and extremities. Initial labs were significant for mild AKI, elevated ESR and CRP, and decreased C3/C4. Allergy was consulted due to a concern for HAE flair and he was started on a C1 esterase inhibitor infusion. The patient had not improved by day three of therapy and he suffered from painful, migratory edema and rash. The differential was broadened and rheumatology and dermatology were consulted. He was started empirically on steroids. Further workup demonstrated hematuria with nephrotic range proteinuria, ANA 1:80 with speckled pattern, (+) C1q antibody with suppressed C1q levels, and diffuse lymphadenopathy on CT. A skin biopsy at the rash site revealed leukocytoclastic vasculitis. After a multidisciplinary discussion, a diagnosis of urticarial vasculitis was favored. A renal biopsy was recommended to enhance diagnostic certainty, however, the patient declined and elected to leave the hospital following clinical improvement. Discussion: In this patient with known HAE presenting with abdominal pain and angioedema, initial treatment for HAE was reasonable. However, certain aspects of the case were not typical of HAE, particularly the lack of response to C1 esterase inhibitor infusion, diffuse annular rash, and glomerular disease. Early re-evaluation and multidisciplinary involvement resulted in a new diagnosis with distinct pathophysiology and treatment. Due to incomplete records and complicating social factors, it remains unclear if this patient's prior angioedema episodes were, in fact, due to UV, or if he had a true congenital C1 esterase deficiency and developed superimposed UV. Regardless, this case highlights the importance of vigilant diagnostic re-evaluation, especially when a given condition is not improving with standard therapy.
Brandon Jocher, MD Dr. Floranne Ernste	*A Drop in Hemoglobin is More Than Just a Drop in the Bucket with Microscopic Polyangiitis* Introduction: Antineutrophil cytoplasmic autoantibody (ANCA)-associated vasculitides are a group of disorders that predominately affect small vessels, have an association with ANCA, and may present with multi-organ involvement. Microscopic polyangiitis (MPA) is a p-ANCA associated

	vasculitis that classically involves the kidneys, lungs, and skin. We present a nuanced case of MPA and review the typical pulmonary manifestations. Case Description: A 76-year-old-man with a past medical history of hypertension, type 2 diabetes with nephropathy, and chronic kidney disease (CKD) stage 5 presented to the hospital with weakness and shortness of breath. Vitals were notable for a blood pressure of 179/112 and an oxygen saturation of 89%. Initial labs revealed a creatinine of 10.2 mg/dL (previous baseline 4 - 6 mg/dL), and a c-reactive protein of 72 mg/L. Urinalysis showed red blood cells > 100 /hpf, predicted 24-hour protein of 14.6 g/24h, and occasional granular casts. Hemodialysis was initiated for volume overload and electrolyte abnormalities with minimal improvement in respiratory symptoms. The patient developed hemoptysis with a decline in hemoglobin from baseline 11 to 7.0 g/dL. CT chest angiogram illustrated diffuse interlobar septal thickening and bilateral ground glass opacities suggestive of fluid accumulation. Bronchoalveolar lavage (BAL) revealed progressive bloody return diagnostic for diffuse alveolar hemorrhage and negative cultures. Further lab work was notable for myeloperoxidase antibodies > 8 U and positive p-ANCA. A diagnosis of microscopic polyangiitis was made with clinical features of diffuse alveolar hemorrhage and glomerulonephritis. High dose steroids and rituximab were started with improvement in symptoms. Discussion: The presented case illustrates a nuanced manifestation of MPA. MPA may present with pulmonary manifestations of cough, dyspnea, and hemoptysis. In this case declining kidney function was superimposed upon severe CKD making diffuse alveolar hemorrhage key in establishing the diagnosis. Diffuse alveolar hemorrhage can be evaluated with BAL, and progressive bloody return is diagnostic.
Simranjit Kahlon, MD Dr. Sujith Puskoor	*To Bleed or Not to Bleed: Navigating a Rare Case of Hypodysfibrogenemia* Introduction: Congenital bleeding and thrombotic disorders are prevalent in the general population, of which fibrinogen disorders are rare. Fibrinogen disorders can be classified into two groups including hypofibrogenemia (low fibrinogen levels) and dysfibrogenemia (dysfunctional fibrinogen). When these disorders occur simultaneously is known as hypodysfibrogenemia. We present a case of a patient with a history of recurrent bleeding episodes who was found to have a new deep venous thrombosis (DVT) and was ultimately diagnosed with hypodysfibrinogenemia. This narrative seeks to shed light on the clinical presentation, diagnostic hurdles, and management strategies to employ when encountering this perplexing disorder. Case Presentation: A 59-year-old female with a medical history of multiple surgeries and vaginal tear complicated by excessive bleeding, papillary thyroid carcinoma s/p hemithyroidectomy, parathyroid adenoma s/p resection, and parietal meningioma presented with a DVT that was indeterminate for being provoked or unprovoked. The patient's maternal uncle and his offspring have an unspecified thrombotic disorder. There is no family history of hemophilia. Due to the history of malignancy and a recent COVID-19 vaccination, it was suspected that her DVT was provoked. Hematology was consulted and hypercoagulable and hemophilia workup was initiated. Labs were negative for Factor V Leiden, Protein C/S deficiency, antithrombin III deficiency, prothrombin gene mutation, antiphospholipid syndrome, and von Willebrand disease. Platelet function assay, INR, and

	LFTS were within normal. Her prothrombin time was slightly elevated. Her fibrinogen levels were low on multiple draws and MGUS was incidentally found via small IgG kappa monoclonal protein on SPEP. Fibrinogen antigen testing was normal while her fibrinogen activity was low. Genetic testing revealed a FGB mutation. Therefore, the diagnosis was confirmed as hypodysfibrogenemia with a mixed thrombotic and hemorrhagic phenotype. Prior to future surgeries, it was recommended to have RiaSTAP, a human fibrinogen concentrate, be given to achieve adequate fibrinogen levels. It was also advised to utilize postoperative thrombophylaxis for 30 days due to her thrombotic phenotype. Conclusion: Hypodysfibrogenemia remains an incredibly rare and underdiagnosed bleeding disorder. Management of this disorder requires a multidisciplinary approach involving hematologists and genetic counselors to help tailor patient treatment plans. The wide range of fibrinogen gene mutations present can lead to a spectrum of variable clinical manifestations, thereby making the diagnosis of congenital hypodysfibrinogenemia challenging. Diagnosis of hypodysfibrogenemia relies on a comprehensive approach involving coagulation testing, fibrinogen assay to assess dysfunctional fibrinogen, fibrinogen levels and genetic testing. Management of bleeding episodes involves replacing dysfunctional or low levels of fibrinogen with fibrinogen concentrate rather than fresh frozen plasma (FFP) or cryoprecipitate. The use of the latter products may lead to increased transfusion to attain adequate fibrinogen levels and therefore possible transfusion related risks. Swift assessment and recognition of this disease is crucial due to its implications on effective patient management during bleeding or thrombotic episodes, surgical complications and pregnancy. The process by which our patient was diagnosed and managed, especially in the setting of her hybrid phenotype, portrays the challenges physicians may encounter with such patient presentations.
Victoria Kalinoski-Dubose, MD Dr. Joelle Friesen Dr. Bibek Saha Dr. Deborah Setter Dr. Darin Carabenciov	*Not Your Typical Headache: A Case of Cerebral Amyloid Angiopathy-Related Inflammation* Introduction: Cerebral amyloid angiopathy-related inflammation (CAA-ri) is a rare clinical entity that is important to recognize as it has a favorable prognosis if diagnosed and treated early. Case Presentation: A 57-year-old male comorbid with hypertension and benign prostatic hyperplasia presented to the emergency department with a 1-month history of worsening bifrontal headaches and progressive cognitive decline. The headaches were increasing in both frequency and severity, occurring throughout the day and night. He also endorsed nausea, light sensitivity, word-finding difficulty, and new memory impairments. Prior to this, he denied any history of headaches. His wife noted he was speaking more slowly with frequent pauses. Examination was significant for blood pressure of 195/130 and delayed speech initiation, but was otherwise normal, including high level cognitive functions. Serum chemistries, liver function tests, and complete blood count were within normal limits. CRP was mildly elevated at 5.5 (nl < 5) but erythrocyte sedimentation rate was normal. Lyme disease testing was negative. Cerebrospinal fluid analysis showed 2 total nucleated cells (54% lymphocytes), glucose 63 mg/dL, protein 38 mg/dL (normal <35). Brain MRI

	with and without contrast demonstrated extensive T2 hyperintensity in the right temporal parieto-occipital region and underlying cerebral microhemorrhages on SWAN with vascular and leptomeningeal enhancement. Vessel wall imaging did not demonstrate evidence of angiitis. A diagnosis of CAA-ri was made by clinical manifestations and MRI findings. The patient was prescribed IV methylprednisone 1000 mg daily for three days and was to complete a 6-week oral prednisone taper, however, steroids were discontinued after 7 days of therapy due to significant steroid-induced mania. Antihypertensives were titrated to a systolic blood pressure goal of < 140 mm Hg. A follow-up MRI 2 months later demonstrated markedly improved T2 hyperintensity in the right temporal parieto-occipital region. The patientâ€™s clinical outcome at follow-up was improved. Conclusion: CAA-ri is a rare and aggressive small-vessel disease characterized by an inflammatory response to deposits of amyloid protein in the cortical or leptomeningeal vessel walls. Clinical presentation includes acute or subacute onset progressive headaches, memory impairment, multidomain cognitive decline, behavioral changes, and seizures. Although the gold standard for diagnosis is brain biopsy, this is often deferred given the invasive nature of this testing. The diagnosis is often made clinically based on symptoms and suggestive brain MRI findings, which classically reveal asymmetric, patchy white matter hyperintensities on T2-weighted sequences, leptomeningeal enhancement, and scattered microbleeds on gradient echo sequences. A set of clinical diagnostic criteria have also been proposed that have high sensitivity and specificity (82% and 97% respectively). The mainstay of treatment is an initial burst of high-dose steroids followed by a taper over 6-12 weeks with interval MRI brain and clinical examination to evaluate recovery. Imaging is completed 4-6 weeks post-treatment to monitor for treatment response. Relapses occur relatively frequently and some patients are therefore transitioned to long term immunosuppressants. Although still rare in clinical practice, it is encountered with increasing frequency and will be particularly important to recognize as the novel amyloid targeting therapies can result in a similar inflammatory response.
Alexis Keef, MD Dr. Kellen Albrecht	*Missing More Than Stones- Severe Pulmonary Hypertension Presenting as Acalculous Cholecystitis* Introduction: Left unrecognized and untreated, pulmonary hypertension can lead to progressive right ventricular dysfunction and failure. Right heart failure symptoms can include abdominal symptoms secondary to hepatic congestion. This is a case of previously undiagnosed end-stage pulmonary hypertension presenting with acalculous cholecystitis, highlighting the importance of diagnostic vigilance and preoperative care. Case Description: A previously healthy 18-year-old female presented with 10 days of intermittent abdominal pain, nausea, and vomiting. On exam she was tachycardic but otherwise vitally stable, with right upper quadrant tenderness. Labs were notable for normal CBC and creatinine, transaminases in the 200s, Na 133, HCO3 17, and normal lipase. Abdominal US and CT imaging noted gallbladder thickening, negative sonographic Murphy's sign, hepatic congestion, and ascites. CTA chest was negative for pulmonary embolism but noted an enlarged pulmonary artery and small bilateral pleural effusions. EKG showed sinus tachycardia, right axis deviation, and nonspecific anterior ST abnormalities. The patient was admitted to medicine and underwent a laparoscopic cholecystectomy for acalculous cholecystitis

	the next day. Postoperatively, she required supplemental oxygen after having received 3 liters of IV fluid, and labs were notable for a new AKI, rising LFTs to >700, BNP >1000 and lactic acidosis. Transthoracic echocardiogram showed normal LVEF 60%, severe RA and RV dilation, and severely reduced RV systolic function with an estimated pulmonary artery systolic pressure of >85mmHg. The patient was diagnosed with acute right heart failure and cardiogenic shock prompting transfer to the ICU for diuresis and inotropic support. After extensive workup, the patient was diagnosed with end-stage pulmonary veno-occlusive disease (PVOD), an exceedingly rare form of pulmonary hypertension, and was promptly listed for and underwent a successful combined heart and bilateral lung transplantation. Discussion: This case highlights several important concepts for the internist including conducting thorough histories and physical exams, carefully analyzing all available objective data, and considering a broad differential diagnosis. These steps not only yield higher diagnostic accuracy but are necessary to avoid anchoring bias. In this case, recognizing acalculous cholecystitis as an entity most often associated with severe systemic disease should have raised clinical suspicion for additional underlying pathology. Additionally, preoperative cardiovascular risk assessment should be routinely performed to inform perioperative risk-benefit discussions and help identify when alternative interventions should be explored. Preoperative clues of right heart failure as the underlying etiology of gallbladder edema for this patient included imaging and lab evidence of hepatic congestion, volume overload with ascites and pleural effusions, an enlarged pulmonary artery, and EKG abnormalities.
Laura Kek, MD Dr. Griffin Reed Dr. Matthew Koster **Abstract Finalist**	*A Rare Case of Curative Colectomy for Takayasu Arteritis* Introduction: The co-occurrence of Takayasu arteritis (TAK) and inflammatory bowel disease (IBD), such as ulcerative colitis (UC) is notably rare with under 200 reported cases. Herein we present a case of complete TAK remission after subtotal colectomy without requirement of ongoing immunosuppression for either condition. Case Presentation: The patient is a 52-year-old female with a past medical history of depression, anxiety, and prior pulmonary embolism who presented with pleuritic chest pain, diaphoresis, and 1 week of bright red rectal bleeding. She had taken 1200 mg of ibuprofen every 6 hours for her chest pain. The patient denied any abdominal pain, dizziness, fever, or fatigue. Initial workup was significant for anemia (10.7 g/dL) and leukocytosis (15.7 10^3L). CT pulmonary angiogram showed aortitis in the ascending and descending aorta, CRP of 338.1 mg/L, and ESR of 117 mm/hr. Patient was febrile to 101F, tachycardic to 201 bpm, and was started on prednisone 60 mg with significant symptomatic improvement. ANA was positive to a titer of 1:1280 with a homogenous pattern, but the remainder of her immune and infectious workup was unrevealing. Temporal artery biopsy was performed and negative. Follow-up CT angiogram demonstrated resolution of the aortitis and ESR and CRP levels normalized. She received 40 mg of prednisone for 1 month but self-discontinued thereafter. She continued to have hematochezia despite discontinuing NSAIDS with progressive anemia (hemoglobin 8.7 g/dL). Upon prednisone discontinuation, her chest pain symptoms returned and repeat imaging demonstrated recurrent large-vessel vasculitis (LVV) involving the ascending and descending thoracic aorta, proximal left carotid artery, and brachiocephalic artery, a distribution felt more consistent with TAK. A colonoscopy was performed, showing diffuse

	severe mucosal changes with biopsy consistent with UC. She was treated with IV methylprednisolone 60 mg daily, 1 dose of Infliximab 10 mg/kg with oral prednisone taper, and a plan for outpatient elective colectomy. However, she was lost to follow up for 11 months before being hospitalized for progressively severe anemia (hemoglobin 6.5 g/dl), at which time she underwent an urgent subtotal colectomy with end ileostomy. Two months after surgery, follow-up imaging with PET-CT showed complete resolution of her aortitis, off all immunosuppressive therapy. The patient has remained symptom-free for three years without any immunosuppression for either TAK or UC. Discussion: TAK and IBD are chronic immune-mediated diseases and often require ongoing immunosuppression. Extra-intestinal manifestations associated with IBD present prior to surgery will persist in about half of patients even after colectomy. Historically, patients with TAK and IBD have still required immunosuppression to manage their vasculitis following colectomy. To our knowledge, we present the first case of resolution of LVV associated with IBD following colectomy without the need for ongoing immunosuppressive therapy, highlighting a unique association between IBD and LVV which requires further investigation into a common etiopathogenic mechanism.
Daniel Khan, MD	*Chronic Cough Due to Bronchobiliary Fistula* Introduction: Bronchobiliary fistula can be acquired due to malignancy, biliary stenosis, trauma, cholangiolithiasis, hepatic echinococcosis, or other conditions inducing chronic inflammation. As this is a rare condition, the presence of this fistula may go undiagnosed for many years. Case Description: In 2018, a 60-year-old man with a significant past medical history of cirrhosis secondary to hepatitis B was found to have an abnormal 3cm mass in the liver on routine cirrhosis surveillance via ultrasound. Biopsy demonstrated cholangiocarcinoma, and he underwent cholecystectomy, hepatic resection, and adjuvant gemcitabine chemotherapy. In late 2020, he developed worsening cough, shortness of breath, and green mucus production. CT scans demonstrated right lower lobe groundglass opacities with bronchiectasis, leading to multiple courses of antibiotics to treat presumed aspiration pneumonia. In 2021, a biloma was identified, and concerns were raised for possible bronchobiliary fistula. His symptoms improved after CT guided drain was placed, but it was unclear whether this was due to his antibiotics or the drainage of the biloma. The biloma recurred after the drain was removed, and he reported recurrence of respiratory symptoms. Over the same interval, he had multiple bronchoscopies performed, which demonstrated concerns for histoplasmosis and strep species as possible explanations for his respiratory symptoms. In 2023, he continued to develop a worsening cough, so he presented to the emergency department once again for further evaluation. He was found to be afebrile, hemodynamically stable and saturating well on room air. However, he was found to be intermittently tachypneic up to 30 breaths per minute and was ill appearing on exam. Respiratory exam demonstrated diminished breath sounds on the right lung base, and a harsh-sounding cough. He was found to have bilious appearing nasal discharge and serum bilirubin was measured and found to be 2.3. This led to an admission where a bronchobiliary fistula was further investigated with an MRI study. MRI abdomen with and without

	contrast demonstrated suspicion for a fistula between the bile ducts at the dome of the liver to the right lung base. Further imaging with MRCP confirmed bronchobiliary fistula with filling of multiple posterior right lower lobe bronchi with the hepatobiliary contrast agent. After this diagnosis was confirmed, an ERCP was performed where a biliary stricture was identified, leading to a balloon dilation and deployment of 3 stents. A multidisciplinary evaluation between gastroenterology, hepatobiliary surgery, and thoracic surgery determined the decision to proceed with right hepatectomy followed by thoracotomy for right lower lobe decortication and lobectomy. Followup ERCP will be needed in 3 months to remove these temporary stents. Discussion: This case illustrates a sample presentation of bronchobiliary fistula. Many patients with recurrent localized lung opacities and infectious symptoms are presumed to have recurrent infection/aspiration. However, groundglass opacities are a nonspecific finding for a variety of etiologies including pus, blood, or water. Symptoms refractory to antibiotics may prompt consideration of additional sources of infection or inflammation that may be driving recurrence of respiratory symptoms.
Nooreen Khan, MD Dr. Mohammed Khalid	*Thoracic Endometriosis Causing Recurrent Pleural Effusions* Case Presentation: 43 y/o F presents to the clinic with recurrent pleural effusions that have worsened in the past year. She associates it with a sharp pain on the right side of her chest. The pain typically occurs a few days before the onset of her menstrual period and persists for several days during her period. She rates the pain intensity as 8/10 on a numerical rating scale. HPI: Pt reports that her symptoms started approximately 2 years ago. Initially, she attributed the chest pain to muscle strain or anxiety. However, as the pain became more severe and consistently coincided with her menstrual cycle, she sought medical attention. She has tried over-the-counter pain relievers and heating pads, which provided minimal relief. Past Medical History: Pt has a history of menorrhagia, which was diagnosed during her early twenties. She has previously had a uterine polypectomy. She had a pleural effusion in 2019 and 2020 which self-resolved. She had 2 in 2022, one in Africa where 1.5L was drained, and another in America where 1L was drained. Both were bloody. Menstrual History: Pt has irregular menstrual cycle. She reports moderate to severe dysmenorrhea and often experiences heavy menstrual bleeding. She is not currently on any hormonal contraceptives. Social History: Pt is a non-smoker and does not consume alcohol or recreational drugs. She is in a monogamous heterosexual relationship and is nulliparous. She does not have any significant occupational exposures. Family History: There is no known family history of endometriosis or other reproductive disorders. Physical Examination: On physical examination, pt appears well-nourished and in no acute distress. Her vital signs are within normal limits. Abdominal and pelvic examinations are unremarkable. Notably, there diminished breath sounds on the R side. No masses or abnormalities are appreciated on chest wall examination. There are no signs of respiratory distress or cardiac abnormalities.

	Workup notable for recurrent moderate to large pleural effusions on CXR. MRI notable for extensive endometriosis in the pelvis and thoracic chest with associated moderate to large pleural effusion. OBGYN was consulted and per the literature, pt was started on Leuprorelin therapy. Despite hormonal suppression, pt continues to have recurrent hemorrhagic effusions though the duration of treatment before results are seen has not been established. She otherwise has improved from a symptomatic perspective. Conclusion: This case illustrates a challenging clinical presentation of thoracic endometriosis, which should be considered in patients with a history of endometriosis who present with cyclic chest pain. Thoracic endometriosis is suspected in this patient due to her history of endometriosis, cyclic chest pain, and hemorrhagic effusions. If her symptoms do not resolve with leuprorelin therapy alone, can consider VATS with pleurodesis. Appropriate recognition of this condition can tailor adequate therapy, and ideally prevention of further pleural effusions.
Nathaniel Klair, MD Dr. Aditya Chauhan Dr. Kristina Krohn	*Diabetes Mellitus and Insipidus: A case of HHS and untreated DI* Introduction: Hyperosmolar hyperglycemic state (HHS) is a complication of type 2 diabetes mellitus (T2DM) caused by elevated glucose. Central diabetes insipidus (DI) is a deficiency of antidiuretic hormone that leads to inability to concentrate urine. Both cause volume depletion and the additive effect can cause severe hypernatremia and hyperosmolality. Case presentation: A 57-year-old female with a history of T2DM and central DI secondary to neurosarcoidosis was brought into the hospital for altered mental status and hyperglycemia. She had been discharged two weeks prior and had not taken her desmopressin (DDAVP) since discharge. At presentation her glucose was 749 mg/dL, her corrected sodium was 177 mmol/L, and her calculated serum osmolality was 380 mOsm/L. She was awake, oriented to self and place, and able to provide single word answers to questions. Her workup also revealed serum ketones of 0.60 mmol/L, normal serum bicarbonate and pH, and an AKI. A head CT revealed a stable hypothalamic mass related to her neurosarcoidosis. She was volume resuscitated with 2L of crystalloid fluids, then treated with an HHS protocol consisting of an insulin drip and maintenance IV fluids for approximately 18 hours until her blood glucose normalized. Her corrected sodium had improved by 11 mmol/L and her calculated serum osmolality had improved by 34 mOsm/L. She was transitioned to a subcutaneous insulin regimen and allowed free access to fluids. DDAVP was initially held to prevent overcorrection. When restarted, it was given on a PRN basis based on sodium levels until her levels normalized and she was returned to her prior twice daily dosing. Her sodium level returned to normal range after about 60 hours with an overall correction rate of 0.62 mmol/L/hr and a serum osmolality correction rate of 1.38 mOsm/L/hr. She returned to her neurologic baseline. She did not develop headaches or other signs of cerebral edema. Discussion: Most sources recommend targeting a sodium correction rate of 0.5mmol/L/hr in hypernatremia to minimize risk of cerebral edema, though technically it's the change in effective osmolality or tonicity that matters. The

	risk decreases with age due to cerebral atrophy. Overcorrecting hypernatremia carries less risk than in hyponatremia. In fact, several studies have shown worse outcomes from correcting too slowly in hypernatremia. Additionally, while symptoms of osmotic demyelination syndrome from overcorrecting hyponatremia are delayed, in an awake patient cerebral edema should cause real time symptoms that would allow for slowing of the correction rate. This case required prioritizing treatments to correct the sodium at an appropriate rate. We initially treated the HHS with IV fluid resuscitation and an insulin drip while closely monitoring the sodium levels. We then allowed her to drink to thirst and slowly restarted DDAVP, dosing it to control the rate of sodium correction. With the extreme starting point of her hypernatremia, normalization of her labs over 2.5 days led to an appropriate rate of correction.
Regina Koch, MD Dr. Ashley Zhou Dr. John Bundrick **Abstract Finalist**	*Sustained Oculo-Cardiac Reflex in a Patient with Severe Panophthalmitis* Case Presentation: A 73-year-old woman presented with right eye pain and decreased vision. On exam she was hypotensive, hypoxemic, and tachycardic with irregular rhythm. She had right periorbital edema, conjunctival injection, and corneal opacification. Laboratory studies revealed Streptococcus pneumoniae bloodstream infection and meningitis. MRI/MRV Brain and Orbits showed leptomeningeal enhancement, fourth ventricle and occipital horn debris consistent with ventriculitis, and right orbit/globe inflammatory changes consistent with panophthalmitis. Intravenous and intravitreal antibiotics were initiated. On day 3 of hospitalization, the patient developed intermittent bradycardia (HR 20-30s) and sinus pauses lasting 10-20 seconds. Repeat ophthalmology exam revealed severely elevated intraocular pressures (52mmHg, normal range: 10-21 mmHg). She was given intravitreal dexamethasone and maximal topical pressure-lowering agents. As her intraocular pressures decreased over the next few days, her sinus pauses shortened, and her bradycardia resolved. The patient improved clinically over the following week with medical management of her infectious syndromes. On day 10 of hospitalization, she developed bradycardia (HR 20-30s), alternating with atrial fibrillation with rapid ventricular response (HR 150-160s). That day, she also began complaining of increasingly severe right eye pain, requiring fentanyl infusion and ultimately sedation. Intraocular pressures were once again elevated (34mmHg). Intraocular pressure drops were restarted, with improvement in her sinus pauses and bradycardia. The patient continued to suffer from intractable eye pain, and enucleation was performed. She did not experience issues with bradycardia or sinus pauses for the remainder of her hospitalization. Discussion: The oculo-cardiac reflex is defined by a >20% decrease in heart rate following direct globe pressure or traction on the extraocular muscles. This reflex usually causes sinus bradycardia but has been associated with several arrhythmias, including sinus node dysfunction and atrial fibrillation. Classically, this reflex is considered a transient and exhaustible phenomenon usually encountered intraoperatively. However, there have been reports of the oculo-cardiac reflex as a sustained phenomenon. One case described episodic bradycardia for several years that resolved after surgical removal of an old intraocular foreign body.1 Another case described persistent bradycardia following repair of a globe perforation that did not resolve until surgical removal of an orbital foreign body.2 In our case, there was a clear temporal

	association of her bradycardia with elevated intraocular pressure. Unfortunately, we cannot offer definitive evidence of this suggested mechanism as we did not attempt to alter vagal tone with anticholinergics such as atropine. It's possible her bradycardia and sinus pauses could be partly explained by increased vagal tone in the setting of severe pain. However, her rate and rhythm improved with intraocular pressure control despite inadequate pain control. We considered meningitis-induced Cushing reflex as a mechanistic explanation of her bradycardia but felt this was less likely given she remained normotensive without elevated pulse pressure. Here we describe a patient with suspected oculo-cardiac-mediated arrhythmia in the setting of severe right globe infection and inflammation. This case adds to a limited body of literature suggesting that oculo-cardiac reflex may manifest as a prolonged phenomenon. In this setting, use of intraocular pressure lowering agents may aid in the management of these arrhythmias.
Jenna Langer, MD Dr. Daniel O'Leary	*A Case for Chemotherapy in the Treatment of Monoclonal Gammopathy of Renal Significance* Introduction: Monoclonal gammopathy of renal significance (MGRS) is a relatively new concept that defines a B cell or plasma cell clonal disorder that does not meet the criteria for malignancy but produces a monoclonal immunoglobulin that leads to renal injury. MGRS is used to describe patients who would otherwise meet the criteria for monoclonal gammopathy of undetermined significance (MGUS) but have additional renal injury secondary to the underlying monoclonal protein. This distinction is quite important as patients with MGUS are not usually treated whereas those with MGRS may benefit from treatment. Patients with MGRS can present broadly with nephrotic syndrome, hypertension, hematuria and acute or chronic renal failure. A renal biopsy demonstrating immunoglobulin deposition is required for the diagnosis. In this report, we highlight the case of a 54-year-old male who presented with severe renal failure due to MGRS and demonstrated remarkable recovery of renal function after four cycles of cyclophosphamide, bortezomib and dexamethasone (CyBorD). Case Description: This is a 54-year-old male with a past medical history of atrial fibrillation, depression, tobacco use, alcohol abuse and COPD who presented for dyspnea and lower extremity edema. Initial labs were remarkable for a creatinine of 2.09 mg/dL, urine protein to creatinine ratio of 16 g/g and 24-hour urine collection of 28 g of protein. Urine immunofixation showed no monoclonal protein or free light chains. A renal biopsy showed monoclonal immunoglobulin deposition with IgG1 lambda restriction and a diffuse proliferative and membranous pattern of glomerular injury. Following the biopsy, a hematologic workup was pursued. A bone marrow biopsy showed normocellular bone marrow with 5% of plasma cells. Concurrent flow cytometry showed polytypic B cells and plasma cells. Serum immunofixation showed an elevated monoclonal peak at 0.1 g/dL with an elevated kappa to lambda ratio of 2.21. Imaging included a total body CT scan and PET scan which were unremarkable. Based on the labs and renal biopsy demonstrating immunoglobulin deposition, the patient was diagnosed with MGRS. The patient's creatinine peaked at 6.51 mg/dL and CyBorD was started for a total of 4 cycles. Following treatment, the patient's renal function improved significantly to a creatinine of 1.46 mg/dL and a urine protein to creatinine ratio of 3.41 g/g.

	Discussion: Treatment of MGRS is determined by the nature of the clone, either a B cell or plasma cell, that produces the monoclonal immunoglobulin. In this case, the patient had a bone marrow biopsy showing 5% plasma cells, serum immunofixation showing a monoclonal spike with an elevated kappa to lambda ratio and a renal biopsy showing immunoglobulin deposition. Because this was determined to be a plasma cell-related MGRS, the National Comprehensive Cancer Network recommended following the algorithm for management of multiple myeloma. Therefore, CyBorD was initiated which led to clinical improvement and marked renal recovery. This case offers evidence that MGRS-related kidney injury may be reversible with chemotherapy treatment that was previously restricted to patients with overt hematologic malignancies.
Julia Liberto, MD Dr. Ahsan Butt Jennifer Pearson Dr. M. Nadir Bhuiyan	*Acute Parvovirus B19 Infection in an Adult Patient: A Clinical Case Report* Introduction: Parvovirus B19, a single-stranded DNA virus, primarily targets erythroid progenitor cells in the bone marrow, inducing erythropoiesis inhibition. While typically benign in immunocompetent individuals, it can manifest with a diverse array of clinical presentations. We report an unusual presentation of acute Parvovirus B19 infection in a 33-year-old female healthcare worker, emphasizing the importance of clinical recognition in atypical cases. Case Presentation: A 33-year-old female healthcare worker presented with fever, arthralgia, headache, night sweats, inguinal lymphadenopathy, and an erythematous maculopapular rash, which had been ongoing for approximately one week prior to hospital admission. On admission, her vital signs were notable for a temperature of 102.7°F and a heart rate of 117 bpm. Laboratory findings revealed Hgb 11.5 g/dL, WBC 3.0 KL, platelet count of 116 KL, ALT at 178 U/L, AST at 146 U/L, and Alk-Phos at 132 U/L. Given the severity of her headache, a lumbar puncture was performed, yielding results that were unremarkable for signs of meningitis. An MRI of the brain displayed no discernible abnormalities. Diagnostic findings revealed a positive monospot test, positive Parvovirus B19 PCR and Parvovirus B19 IgG assay. Her hemoglobin levels decreased to 8.7 g/dL, with a nadir in white blood cell count at 3.0 KL and platelet count of 96 KL. A low reticulocyte index alluded to bone marrow suppression. The patient's clinical course necessitated several days of hospitalization due to an incapacitating headache that required intravenous medications. Fortunately, following a regimen of supportive care, the patient experienced gradual resolution of her symptoms over the course of several hospital days. Subsequent outpatient evaluation confirmed the complete normalization of her laboratory parameters. Discussion: Parvovirus B19 infection is typically mild in adults. However, acute infection can manifest with diverse clinical presentations. This case exhibited a spectrum, including fever, rash, headache, lymphadenopathy, pancytopenia, and transaminitis, with spontaneous resolution. Her risk factors included recent exposure to children and healthcare occupation. Transmission routes encompass respiratory, vertical, and hematogenous spread. Parvovirus B19 operates through the replication within erythroid progenitor cells, precipitating erythropoiesis suppression. The initial phase of Parvovirus B19 infection results in cytotoxicity towards red blood cells (RBCs), while rash and joint symptoms may potentially be mediated by immune system activation. In individuals with compromised immunity, cytotoxicity against

	RBC precursors can lead to transient aplastic crisis. Detection of Parvovirus B19 infection involves serologic testing and PCR. Typically, IgM antibodies become detectable around 10-12 days post-infection, with IgG antibodies emerging after two weeks. IgG antibodies are not particularly helpful given 50% of asymptomatic patients can be positive. Humoral immunity plays an important role in infection clearance, making intravenous immunoglobulin (IVIG) a consideration for immunocompromised patients. Of note, the positive monospot test was likely a false-positive, due to its poor specificity and cross-reactivity with other infections. Our case report underscores the importance of considering acute Parvovirus B19 infection in adults, despite most infections being mild and self-limiting Fortunately, while comprehensive diagnostic evaluations may be warranted in some instances, the cornerstone of management remains grounded in supportive care.
Mackenzie Maberry, MD Dr. Alessandra Tomasi	*Don't Hold your Breath Over Antimicrobial Prophylaxis* Introduction: The decision regarding antimicrobial prophylaxis initiation is a commonly encountered one. While guidelines exist for specific clinical scenarios, others remain less clear. Additionally, sentinel events involving missed prophylaxis unfortunately remain a frequent and important cause of iatrogenesis in modern healthcare. Case Presentation: Here we present the case of a 57-year-old male with recently diagnosed brain lesions of likely metastatic malignant origin who was admitted to the medical intensive care unit (ICU) for acute hypoxic respiratory failure. Briefly, the patient presented to an outside institution after 7 weeks of dysarthria and cervical arthralgia and was found to have multiple supratentorial, infratentorial, and spinal cord CNS lesions thought to be metastatic in origin. He was placed on high-dose dexamethasone (6mg every 6 hours) to decrease cerebral edema with subsequent improvement of his neurologic symptoms. The patient then unfortunately left the hospital against medical advice without a taper plan nor Bactrim prophylaxis. His dexamethasone was then filled at the same dose by an outpatient prescriber a few weeks later. The patient re-presented for a staging biopsy that was unable to be completed due to significant dyspnea during bronchoscopy, for which he was sent directly to the emergency department. He required positive pressure ventilation and was admitted to the MICU for further evaluation and management. A chest CT at that time was suggestive of multi-cavitary pneumonia that was not seen on imaging just one month prior. Cultures from a bronchoscopy subsequently confirmed pulmonary aspergillosis. The patient also had positive Beta D-glucan serum studies, suggestive of disseminated fungemia. His immune suppression workup including HIV and a bone marrow biopsy were otherwise negative and it was thought the patient's disseminated fungal infection was iatrogenic in nature. Notably, his PJP smear and PCR were negative. The patient was treated with voriconazole but unfortunately required tracheostomy due to prolonged clinical course. A metastatic lesion was biopsied and found to be consistent with metastatic adenocarcinoma of unknown origin. Conclusion: This patient receiving immunosuppression with high dose steroids was at elevated risk for opportunistic infections, including fungal. Prophylaxis for systemic fungal infections is not routinely given to HIV-

	negative patients, except in those with neutropenia, planned bone marrow transplant, or actively undergoing treatment for AML. In these clinical scenarios, micafungin and posaconazole or fluconazole are the first line prophylactic agents of choice for prophylaxis against both Candida species and Aspergillus. In other clinical scenarios, Bactrim is the gold standard antimicrobial prophylaxis agent, covering major opportunistic pathogens including Toxoplasma gondii, Listeria monocytogenes, Nocardia species, and Pneumocystis jirovecii (PJP). Indications for prophylaxis in HIV-negative patients on high-dose steroids are not well established for many clinical scenarios. Our institution currently recommends PJP prophylaxis initiation at 20mg prednisone (or equivalent) for greater than 4 weeks' duration; although, this is an area of opportunity for further research.
Sanjoyita Mallick, MD	*Clozapine Induced Cardiomyopathy* Background: Clozapine, an atypical antipsychotic, is known for its efficacy in treating treatment resistant schizophrenia. However, its use is associated with a wide range of adverse effects, including rare but potentially life-threatening cardiotoxicity. Clozapine is associated with myocarditis and cardiomyopathy with an estimated absolute risk of 0.01% to 0.19% (Citrome et al., 2016). Psychiatrists tend to focus on the well-known side effects like agranulocytosis and the development of metabolic syndrome when monitoring a clozapine-treated patient, yet the cardiac risk is not well understood and often overlooked. Case Presentation: A 32-year-old male with a history of long standing, treatment- resistant schizophrenia was initiated on Clozapine during a hospital inpatient psychiatry admission. This patient had no significant history of cardiovascular disease or risk factors. The patient was started on 25 mg and gradually titrated up by 25 mg daily to a therapeutic dose of 250 mg over the span of approximately 9 days. He was scheduled to be discharged from inpatient psychiatry about a week after reaching therapeutic dose. Surrounding the days approaching his discharge, he was noted to be persistently tachycardic and hypotensive. He denied chest pain, shortness of breath, or leg swelling. On physical exam, patient was tachycardic, without JVD, peripheral edema, and lungs were clear to auscultation. Electrocardiogram showed sinus tachycardia and possible left atrial enlargement. Further investigation revealed that his troponins were elevated and peaked at 466. On echocardiography, his left ventricular ejection fraction was reduced to 32%, confirming the diagnosis of cardiomyopathy. Cardiac MRI was obtained and ruled out myocarditis. Clozapine was promptly discontinued, and the patient was started on an ACE inhibitor. Beta blocker (metoprolol XL) was initiated prior to discharge per heart failure guideline directed medical therapy. Discussion: Clozapine induced cardiomyopathy is a rare but potentially life-threatening adverse effect of clozapine therapy. The exact pathophysiology remains unclear, but it is believed to be multi-factorial. Symptoms vary depending on severity, time of onset, and pre-existing comorbidities, but may include shortness of breath, fatigue, chest pain, palpitations, or lower extremity edema. It is also more likely when the drug is rapidly titrated up. Essentially, the presentation of clozapine-induced cardiomyopathy resembles acute heart failure. However, 40-83% of patients are asymptomatic thus complicating the diagnosis and likely an underestimation of the actual prevalence (2). The onset of symptoms can also vary, ranging from months to years after initiation. Unlike clozapine-induced myocarditis, which typically

	presents in a few weeks, cardiomyopathy usually presents after at least eight weeks of treatment (3). It is important to note that although it is less common, cardiomyopathy can occur at any point after clozapine initiation. This was the case in our patient, for whom presented with symptoms a few days after initiation. Tachycardia is also one of the more common side effects of clozapine and reported to occur in 25 out of every 100 people treated (6). In our case, tachycardia was the only initial presenting symptom, and it is often unclear how best to navigate this in practice.
Paulina Marell, MD	*Water, pus, blood, cellsâ€¦and protein! A Case of Pulmonary Alveolar Proteinosis* Background: Pulmonary alveolar proteinosis (PAP) is a rare pulmonary condition in which surfactant lipids and proteins accumulate in the alveoli and small airways. The majority of cases are primary PAP with the development of auto-antibodies against granulocyte-macrophage colony-stimulating factor (GM-CSF). Rarely, PAP can be congenital or associated with an underlying condition that affects alveolar macrophages, such as infections, immunodeficiencies, or hematologic diseases. Case Presentation: Here we present the case of a 71-year-old male never-smoker who initially presented to pulmonary medicine clinic with over a year of exertional dyspnea and previous abnormal imaging findings. He began developing a cough and exertional dyspnea in early 2021. Symptoms persisted for several months, and he received multiple rounds of antibiotic therapy with minimal effect. He underwent a chest computed tomography (CT) scan in August 2021, which showed bilateral patchy ground-glass opacities with interlobular septal thickening. He underwent bronchoscopy with bronchoalveolar lavage and endobronchial biopsy, with pathology results revealing expanded alveoli with proteinaceous fluid and cholesterol clefts and interstitial amyloid deposition. Mass spectrometry identified ATTR (transthyretin)-type amyloid deposition. While undergoing further evaluation, he presented to a local hospital with worsening dyspnea and hypoxia and was transferred to this facility's medical intensive care unit (MICU) for further management. CT chest with contrast showed a cavitary lesion in left upper lobe measuring about 2 cm with progressive bilateral patchy pulmonary infiltrates. Sputum cultures returned positive for Nocardia farcinica. He was treated with trimethoprim-sulfamethoxazole, linezolid, and imipenem-cilastatin. He underwent an extensive autoimmune work-up which revealed an auto-antibody to GM-CSF, normal monoclonal protein assays, and negative connective tissue disease serologies. He underwent bronchoscopy with whole lung lavage and was discharged on high dose trimethoprim-sulfamethoxazole. After discharge, he met in the pulmonary medicine clinic and was started on sargramostim, inhaled recombinant GM-CSF. About 6 months later, he developed both clinical and radiographic progression of PAP. In June 2023, he was admitted for planned repeat whole lung lavage. He underwent general anesthesia in his MICU room and was intubated using a double-lumen endotracheal tube. The right lung was de-aired and 10 liters of saline were instilled and subsequently drained; the same procedure was performed on the left lung. He tolerated the procedure without complications and was discharged the next day. In follow-up outpatient appointments, the patient reported significantly improved dyspnea and was advised to decrease his dose of dose trimethoprim-sulfamethoxazole to

	secondary prophylaxis dosing. The patient was a musician in a band and reported that he was able to start playing the trumpet again. Discussion: This patient developed autoimmune pulmonary alveolar proteinosis likely secondary to pulmonary nocardia infection with unclear contribution of pulmonary ATTR amyloidosis. The patient twice underwent whole lung lavage with significant improvement in his symptoms in addition to treatment with inhaled GM-CSF. For autoimmune PAP, whole lung lavage and GM-CSF are the mainstays of treatment. The prognosis for autoimmune PAP is variable and may include spontaneous resolution in up to a quarter of patients, persistent symptoms, or a progressive disease that ultimately leads to respiratory failure.
Rebecca Martinez, MD	*Acute Psychosis Caused by Thyrotoxicosis in a Patient with Untreated Graves' Disease* Introduction: Thyrotoxicosis is associated with many severe and often life-threatening complications. This clinical vignette highlights a case of psychosis that was initially thought to be due to underlying psychiatric disease, but in turn demonstrates a rare, as well as some more common complications of severe hyperthyroidism. Case description: A 54-year-old patient with no known medical or psychiatric history was initially admitted to the psychiatric unit after she was found in the community exhibiting disorganized behavior with severe paranoia. Admission labs obtained after a large goiter was noted on the patient's physical exam revealed a significantly suppressed TSH, positive TSI, and an undetectably high free T4 (>7.7). Given lack of known psychiatric history, there was concern that her thyrotoxicosis was the main driver of her psychosis. She was initiated on methimazole and atenolol per Endocrinology recommendations however given her paranoia she refused oral medication. It was determined that due to her psychosis, the patient lacked capacity to make medical decisions. She was transferred to the ICU to facilitate her treatment, eventually requiring intubation and sedation due to agitation. Given the severity of her symptoms, the patient was treated for impending thyroid storm. Her medications were switched from methimazole and atenolol to PTU and propranolol. Despite this, free T4 remained undetectably high. She was additionally started on iodine and cholestyramine. Due to hemodynamic instability, the patient was also started on IV steroids for 10 doses. The patient's hospital course was complicated by atrial fibrillation with RVR that was refractory to medical treatment with metoprolol. Given the refractory nature of her condition despite maximal medical therapies and with a new complication of atrial fibrillation, nephrology was consulted to initiate plasma exchange. The patient underwent plasma exchange daily for 3 days with improvement in free T4 to 6.6. Given the refractory nature of the patient's thyrotoxicosis despite maximal medical therapy and plasma exchange, it was determined that the most definitive form of treatment would be thyroidectomy and ENT was consulted. On day 10 of admission, the patient converted from atrial fibrillation to sinus rhythm. She received a total of 7 rounds of plasma exchange and underwent a total thyroidectomy on day 15 of hospitalization without complication. Her anti-thyroid therapies were discontinued and decadron tapered off. Her post-surgical free T4 was 3.3. She was extubated 3 days later and subsequently transferred from the ICU.

	Discussion: Though psychosis is a rare complication of thyrotoxicosis, it can be an underlying cause. This case highlights the importance of a thorough physical exam and clinical evaluation in a patient exhibiting psychosis. By noting a large goiter on exam in this patient, we were able to identify an underlying medical cause of acute psychosis in a patient without any known psychiatric history and provide the appropriate medical treatment with improvement in her symptoms. This case also shows that in a patient with severe thyrotoxicosis refractory to initial treatment, a multidisciplinary approach is necessary for adequate treatment of the patient.
Abdilahi Mohamoud, MD	*Inferior Wall Inferno: Recurrent Stent Thrombosis* Introduction: Eptifibatide is an antiplatelet medication commonly used during percutaneous coronary intervention (PCI) for patients with acute coronary syndromes. It binds the glycoprotein IIb/IIIa receptor on platelets, inhibiting platelet aggregation and thrombus formation. Eptifibatide has been shown to reduce ischemic complications in patients undergoing PCI, however, it carries a risk of thrombocytopenia and thrombosis. We present a case of recurrent in-stent thrombosis attributed to eptifibatide-induced profound thrombocytopenia in a patient treated with PCI for ST-elevation myocardial infarction (STEMI). Case Presentation: A 61-year-old female with no known past medical history presented to the emergency department after she developed acute chest pain. EKG showed ST elevation on lead II, III, AVF, V4, V5, and V6 with ST depression on lead I and AVL as well as high-grade AV Block. Vitals were notable for bradycardia, hypotension, and hypoxia. The Cardiac Catheterization lab was activated, ASA 324 mg was given, and the patient was intubated due to acute hypoxic respiratory failure. Emergency cardiac catheterization showed 100% occlusion of the proximal right coronary artery (RCA) which was stented with a drug-eluting stent (DES). The patient was started on dual antiplatelet therapy with aspirin and ticagrelor as well as eptifibatide infusion. Within an hour, she again developed high-grade AVB. Repeat angiography showed in-stent thrombosis complicated by RCA dissection. Two additional DES were placed. Twelve hours after initiation of eptifibatide, the patient developed profound thrombocytopenia with platelets dropping from 224 to 36 k/cmm (baseline: 150-400). Recheck of platelets in citrate tube remained low indicating true thrombocytopenia. Due to concern for possible thrombocytopenia secondary to drug reaction, eptifibatide was discontinued and the patient was switched to heparin infusion. The patient was also found to have bleeding in the conjunctiva and around IV sites, requiring platelet transfusion to maintain platelets above 50 k/cmm. Her hospitalization course was complicated by cardiogenic shock leading to in-hospital cardiac arrest with return of spontaneous circulation following multiple rounds of CPR and defibrillation. Over the next 10 days, the patient experienced four more episodes of recurrent inferolateral ST-elevation on EKG and chest pain concerning for recurrent stent-thrombosis. She underwent a total of five emergency angiographies and interventions including thrombectomy and multiple stenting to RCA. Extensive hypercoagulability workup was unremarkable. Her thrombocytopenia eventually resolved during her hospitalization. A permanent pacemaker was placed due to symptomatic pauses and sinus arrest. The patientâ€™s course improved and was able to be discharged with apixaban along with dual antiplatelet agents of aspirin and clopidogrel with follow-up at Cardiology Clinic.

	Conclusion: This case highlights a rare but serious complication of eptifibatide therapy of thrombocytopenia with recurrent stent thrombosis. The thrombocytopenia was likely an idiosyncratic reaction, as it occurred rapidly after initiation of eptifibatide. With profound thrombocytopenia, the patient developed recurrent stent thrombosis despite adequate management. This case reinforces the need for close monitoring of platelet counts after initiation of eptifibatide. Discontinuation of the drug and treatment with platelet transfusion reversed thrombocytopenia. Alternative parenteral antiplatelet agents, such as tirofiban or cangrelor, could be considered in future cases to avoid the recurrent thrombosis associated with eptifibatide.
Pornthira Mutirangura, MD Dr. Kellen Albrecht Dr. Anthony Prisco Dr. Andrea Elliott	*From Crisis to Clarity: A Rare Case of Pheochromocytoma Presenting as Cardiogenic Shock in an Otherwise Healthy Young Woman* Background: Pheochromocytoma, a rare neuroendocrine tumor that produces catecholamines, typically manifests with paroxysmal hypertension as a prominent feature. However, these patients rarely (2%) present with cardiogenic shock and associated hypotension. This leads to diagnostic challenges and increased mortality risk if not recognized and treated promptly. In this case report, we present the clinical details of a 35-year-old female diagnosed with pheochromocytoma, who initially presented with cardiogenic shock. Case presentation: A 35-year-old active female with no significant medical history presented to an emergency room with shortness of breath. Upon arrival, she was febrile, tachycardic, hypotensive, and hypoxic. Shortly after arrival, she developed hemodynamic instability and respiratory failure, requiring intubation and vasopressor support (norepinephrine 6 mcg/min, vasopressin 0.03 unit/min). EKG showed sinus tachycardia and Troponin I was elevated to 49,882 ng/L. An echocardiogram revealed biventricular failure with a left ventricular ejection fraction (LVEF) of 10-15%. Coronary angiography demonstrated no coronary artery disease. Her hemodynamic status continued deteriorating with rising pressors (norepinephrine 8 mcg/min), rising lactate of 7.2 mmol/L, and significant kidney injury (creatinine 3.4 mg/dL). Subsequently, an Impella© was inserted in the left ventricle. An abdominal computed tomography (CT) scan revealed a large right suprarenal mass measuring 7.5 x 6.2 x 9.1 cm with an intralesional bleed. The patient's parents revealed she has had recurrent episodes of transient headache, profuse sweating, and palpitations in the past 2 months. Further blood tests revealed elevated metanephrine (2.92 nmol/L), normetanephrine (33.6 nmol/L), and chromogranin A (4263 ng/mL). Notably, these labs can be confounded by acute illness making diagnosis challenging in this setting. The combination of history, presentation, CT scan, and laboratory results raised strong suspicion for pheochromocytoma. Surgical resection was planned as a diagnosis and definitive treatment. However, pre-medications with alpha and beta blockers were required prior to surgery. As the patient's hemodynamics were unstable, pre-medications and surgery were impossible then. After a week of inotropes and mechanical circulatory support, the patient's blood pressure and respiratory status improved significantly, leading to successful extubation and removal of the Impella device. Repeat echocardiogram demonstrated improvement of LVEF to 60-65%. She was premedicated with phenoxybenzamine and carvedilol for 7 days. Subsequently, laparoscopic right adrenalectomy was performed. The

	pathology results confirmed the diagnosis of pheochromocytoma. Conclusion: The diagnosis of pheochromocytoma is challenging due to the variability of presenting symptoms. The classic triad consisting of episodic headache, sweating, and tachycardia accounts for only 20 percent of these patients. This case emphasizes the significance of considering pheochromocytoma in young patients presenting with cardiogenic shock, where the excess catecholamines can lead to profound cardiomyopathy. Early recognition and prompt definite treatment of surgical resection are required to prevent morbidity and mortality. However, immediate surgery might not be possible due to hemodynamic instability and the inability to pre-medicate with alpha and beta blockers to prevent intraoperative hypertensive crisis. In such cases, hemodynamic support, including inotropes and mechanical circulatory support, remains a valuable tool for maintaining cardiovascular function and organ perfusion until stability is achieved.
Jordan Nunnelee, MD Dr. Ashley Egan **Abstract Finalist**	*Uncovering the Unexpected: Influenza-Induced Rhabdomyolysis in a Patient with Undiagnosed AL Amyloidosis* Case Description: A 51 y.o. female patient with a history of depression and suicide attempts presented to the ED with myalgias, cough, and emesis. She was diagnosed with influenza A infection, admitted for symptom management, and started on oseltamivir. Over the next 48 hours, she developed progressive lower extremity weakness, paresthesias, and encephalopathy. She had rapidly progressive mottling of her lower extremities from her feet to midback. She was noted to have acute kidney injury and decreased urine output in the setting of rhabdomyolysis. She was transferred to the MICU. Her lower extremities and left upper extremity became tense on exam. Compartment syndrome was confirmed via elevated compartment pressures. Orthopedic Surgery performed urgent fasciotomies of her extremities. Due to ongoing muscle necrosis, she required multiple debridements. Peak creatine kinase was 52,000. She eventually required hemodialysis due to uremic encephalopathy. Multiple subspecialty teams were consulted including Rheumatology and Hematology. She underwent extensive evaluation investigating an underlying etiology including autoimmune disease (APLS, vasculitis), infection, and toxin ingestion, which was unrevealing. Skin biopsy of a mottled-appearing area showed non-specific findings consistent with a small occlusive vasculopathy. Hematologically, she was found to have a monoclonal IgG kappa proteinemia. A fat aspirate was negative for amyloidosis, but muscle biopsy demonstrated amyloid deposition in connective tissue, intramuscular blood vessels, and skeletal muscle. Mass spectrometry confirmed AL amyloid. Bone marrow biopsy demonstrated <5% clonal plasma cells and amyloid in the periosteum. Her renal function recovered allowing for hemodialysis discontinuation. Due to extensive tissue necrosis, she tragically underwent bilateral lower extremity below-knee-amputations. She was discharged to inpatient rehabilitation. She started daratumumab in the outpatient setting for treatment of AL amyloidosis. Discussion: Rhabdomyolysis is a diagnosis with a wide range of causes. Determining etiology is imperative to allow for disease-directed therapy in the hope of hastening recovery and decreasing the risk of recurrence. However, treatment is similar, including aggressive hydration and close monitoring of electrolytes due to the potential need for dialysis. When a patient is suffering from severe rhabdomyolysis, it is important to consider

	advanced muscle inflammation leading to compartment syndrome and subsequent tissue necrosis. Astute and frequent clinical examinations should be performed. The etiology of this patient's rhabdomyolysis was likely an acute viral myositis with a predisposition to extreme muscle inflammation in the form of previously undiagnosed AL amyloidosis involving the musculature. It is unlikely, due to the acuity of her symptoms, that her viral infection preceded her amyloidosis and caused rapid amyloid deposition, though this was considered. There are 12 reported cases of rhabdomyolysis secondary to influenza A infection (PMID: 25839005). However, there are few cases of amyloid myopathy presenting as rhabdomyolysis (PMID: 16919949, PMID: 3115035). This complicated case reflects a rare complication of influenza in the form of myositis, severely impacted by an undiagnosed underlying malignancy. While rhabdomyolysis in this setting will not be frequently encountered, it is important to consider the relationship between rhabdomyolysis, myositis, and compartment syndrome, in which early diagnosis and intervention is paramount to salvage affected limbs.
Shilpa Paul, MD	*Young Adult with Acute Pleurisy* Introduction: Chest pain history, physical examination, CAD risk factors and initial EKG compose the immediate data available to determine probability of ACS/AMI in a patient with typical chest pain. Atypical chest pain is more elusive. Pleuritic or positional nature and pain reproduced by palpation are likely non-ACS, likelihood ratio- of 0.2 to 0.3 (as evidenced by studies). Differentiating life threatening causes from benign is crucial because of the consequences of misdiagnosis in either direction. Case Presentation: 48 yr. male with hypertension, tobacco use disorder presented with chest pain that began around noon on the day prior to admission while sitting at his computer. He passed out for some time and woke up with his head on the keyboard. After that, felt cold sweats and was worried that he was having a heart attack. Informed a friend who called EMS on his behalf. In the OSH, labs showed wbc-17 and D-dimer elevated to 2.81. CT pulmonary angiography negative, but with moderate pericardial effusion, peri bronchial thickening and ground glass opacities. Troponin I 0.07, 0.13, 0.12. Pleuritic pain and CT findings were thought to be due to myocarditis /pericarditis. He was started on ibuprofen, antibiotics. Transferred to ANW on the next day, as echocardiography was not available at the local hospital. On presentation to ANW, patient endorsed ongoing pleuritic chest pain that was worse when lying down. Vitals stable. Bedside US with small/moderate effusion. Abnormal movement of right atrial wall on US raised concern of early tamponade and Cardiology consulted immediately. After review of OSH chest CT by cardiologist, there was concern for Type A aortic dissection. Chest CTA dissection protocol was positive for dissection starting from distal to the aortic root and terminating in the distal arch. He was transferred to the ICU after starting esmolol and nitro drips for BP control and evaluated for emergent surgical repair. He underwent aortic dissection/hemiarch repair with ascending aorta graft as well as repair of the R axillary artery. Post operative course complicated by atrial fibrillation with

	RVR and cardiogenic shock. Cardiovascular meds titrated for optimal HR and BP control and recovered well post-operatively. Per vascular medicine, tortuosity of proximal cervical segment in right vertebral artery on CTA of head/neck was not consistent with fibromuscular dysplasia. Family history of brain aneurysms and cardiomegaly on maternal side raised concern for connective tissue disorder (Loeys-Dietz Syndrome). Longstanding untreated HTN / possible connective tissue disorder seem to be the apparent reasons for his aortic dissection. Further genetic counseling /screening is ongoing. Conclusion: Aortic dissection is a rare disorder with 2000 cases/ year in US and mortality is as high as 1% per hour if left untreated. Prompt medical surgical intervention significantly improves mortality. Key concerns are misdiagnosis and over testing. Although, there are no standardized diagnostic strategies, pretest probability assessment tools like Aortic dissection risk detection risk score and D. dimer can be helpful in decision making.
Daniel Pollmann, MD Dr. Peter Lund Dr. John Lesser	*An Unexpected Cause of Embolic Stroke with a Striking ECG* Case description: A 78-year-old woman with untreated hyperlipidemia presented to the Emergency Department with acute onset positional dizziness in the setting of multiple weeks of bilateral neck pain. Her neurologic exam was unremarkable except for dizziness upon standing. Brain MRI showed subacute infarcts of the posterior cerebral hemispheres. On initial evaluation, high-sensitivity cardiac troponin was stably elevated at about triple the ULN, and ECG showed diffuse T wave inversions deepest in the anteroseptal leads (up to 10 mm in amplitude). She denied any typical or atypical anginal symptoms. She was admitted to the hospital for workup of presumed embolic strokes. CT angiography of the head and neck revealed no significant carotid or vertebral artery stenosis or dissection and no large vessel occlusion. Cardiac telemetry remained normal sinus rhythm with ongoing T wave inversions. Transthoracic echocardiogram revealed new apical septum segment and apical anterior segment wall motion abnormalities. Thus, a cardiac ischemic evaluation was undertaken. CT cardiac coronary arteries showed a >70% first diagonal stenosis, positive remodeling and low-attenuation plaque with associated regional hypokinesis of mid anterior and anteroseptum and entire apical segment's findings consistent with recent acute myocardial infarction. No LV or LA thrombi were observed. Cardiac catheterization and coronary angiogram showed moderate-sized first diagonal branch with 50% stenosis on tortuous segment with normal flow. The patient's posterior strokes were ultimately considered cardioembolic from a now absent LV mural thrombus which resulted from a silent myocardial infarction. The patient was discharged with high-intensity statin therapy, low dose aspirin, and apixaban. Discussion: This case illustrates the possible causes of diffuse T wave inversions with anteroseptal predominance on ECG. Initially, these findings were attributed to cerebrovascular disease, an association that is well described in both hemorrhagic and ischemic stroke and thought related to increased myocardial stress leading to cardiomyopathy during these events. Her lack of anginal symptoms and stable (though modestly elevated) high-sensitivity troponins argued against acute myocardial ischemia. Eventually, convincing evidence arose for recent acute myocardial infarction causing apical wall motion abnormality and presumed mural thrombus. Thus her ECG findings may more correctly be attributed to myocardial ischemia which can produce the same pattern, though typically with LAD lesions.1 This is an

	important pattern to recognize on ECG and of which to thoroughly consider the possible etiologies.
Sanjina Rajput, MD	*Pyogenic Liver Abscess in an Immunocompromised Patient* Introduction: Liver abscesses are the most common type of visceral abscess, annual incidence being 2.3 cases per 100,000 people, affecting males more than females, with higher reported incidence in East Asian countries. They are classified into pyogenic or parasitic, with a small number being fungal in origin. There is significant mortality risk and complication rate in untreated patients. Case presentation: A 65-year-old male with past medical history of colonic adenomatous polyps presented to the emergency department with a 7-day history of fevers, altered mental status, muscle aches, nonproductive cough and abdominal pain. He was noted to be febrile to 38.9 C, tachycardic, tachypneic, diaphoretic, requiring 5L oxygen via nasal cannula. He was only oriented to self and place. Pertinent physical exam findings included oral candidiasis, and abdominal tenderness in the right upper quadrant and right flank. No focal neurological deficits were present. Labs revealed leukocytosis of 25.9 with neutrophilic predominance, elevated AST 114, ALT 98, total bilirubin 1.2, direct bilirubin 0.9, elevated lactate 3.2, anion gap metabolic acidosis, and new acute kidney injury. CT head imaging showed a 1.6 cm mass noted as likely meningioma. CT abdomen/pelvis showed several heterogeneously hypoattenuating hepatic lesions suspicious for metastatic disease versus atypical infection. Blood cultures were obtained, patient was started on ceftriaxone, metronidazole and vancomycin and admitted to the ICU. Diagnostic workup was pursued including HIV serology which returned positive with viral load 3750000, CD4 51. Antibiotics were escalated to daptomycin and piperacillin-tazobactam due to clinical deterioration. Ultrasound guided liver biopsy yielded 3 samples of purulent fluid, liver aspirate and tissue returned positive for gram positive cocci later determined to be strep intermedius, corroborated with broad range PCR. No anaerobic, fungal or acid-fast growth was noted. Blood cultures also grew streptococcus intermedius. Negative studies included testing for CSF infections via lumbar puncture, hepatitis, echinococcus, E. histolytica, syphilis, sputum cultures and leukemia/ lymphoma flow cytometry. Transthoracic echocardiogram was normal. Source control of hepatic abscesses was achieved with percutaneous aspiration and ultrasound-guided drain placement of 3 different sites, which largely resolved within a 1-month period. Antibiotics were deescalated first to ampicillin-sulbactam, followed by homegoing regimen of ceftriaxone and metronidazole for 2 weeks along with Biktarvy and Bactrim due to new HIV diagnosis. Discussion: Pyogenic liver abscesses usually develop secondary to biliary pathology, portal vein pyemia, and hematogenous seeding with comorbidities such as diabetes and an immunocompromised state. They are typically polymicrobial, including E. coli, Klebsiella, Streptococcus, Staphylococcus and anaerobic species. Presentation includes fever, RUQ abdominal pain, and systemic symptoms. Laboratory evaluation reveals leukocytosis, transaminitis, bilirubinemia, and elevated alkaline phosphatase. Evaluation includes imaging with CT or ultrasound, aerobic and anaerobic blood cultures, and abscess fluid aspiration sent for gram stain, bacterial, fungal and

	mycobacterial culture. Optimal therapy is a combination of antibiotics and percutaneous drainage via catheter placement or needle aspiration depending on the size, number and complexity of lesions. Empiric antibiotics pending abscess microbiologic results should cover streptococci, enteric gram-negative bacilli, anaerobes as well as E. histolytica until it is ruled out as a cause.
Thomas Reimann, MD Dr. Wojciech Kraszkiewicz	*The Joint Condition: SLE and Gonococcal Arthritis in a Young Woman* This clinical vignette describes a case of gonococcal arthritis in a young woman with systemic lupus erythematosus. It will touch on the overlapping symptoms of these two disorders and how they initially made the accurate diagnosis much more elusive. Additionally, we explore key factors that increase the risk of gonococcal disease progressing from a fairly pedestrian and easily treated infection to the more morbid disseminated state.
Alex Rivero, MD	*Unmasking Chronic Empyema: A Cystic Masquerader* Introduction: Empyema is defined as the collection of pus in the pleural space. The most common cause of empyema is bacterial pneumonia. Other causes of empyema include chest trauma, post-procedural (thoracotomy, thoracentesis, pleural biopsy), and bronchogenic carcinoma. Presentation of an empyema is variable and can depend on the pathologic stage of the condition. Case presentation: A 52-year-old man with a past medical history of diabetes and hypertension presented to clinic with an approximately 1-year history of progressive shortness of breath and decreasing exercise tolerance to the point where he could barely walk up a flight of stairs. Over the last 5-6 months, he developed a dry cough, significant left anterior chest pressure and pain. On physical exam, breath sounds were absent over the left lung. A chest X-ray showed a large peripherally calcified mass in the left thorax with an associated rightward shift of the mediastinum. Chest CT showed mass dimensions of 18.8 x 16.7 x 21.9 cm and no intrinsic enhancement, suggesting cystic contents. Labs were remarkable for eosinophilia. The patient was born and raised in rural Ecuador (Canar province). His family were farmers and agrarians, and he had significant domestic animal exposure, including sheep, goats, and dogs. He reported a remote history of a prolonged hospital stay when he was 13 years old, requiring multiple chest tubes for clinically suspected Paragonimus infection. He moved to the United States in 2000 and has not traveled outside the country since. Given his history and findings, there was initial concern for pulmonary Echinococcosis causing the formation of a large hydatid cyst. Echinococcus serology was negative. He was admitted to the hospital and underwent a left thoracotomy and debridement/drainage of the left pleural cavity with pulmonary decortication and repair of air leak with reinforcement using an intercostal muscle transposition flap. Approximately 2.5L of thick, brown-appearing fluid was aspirated, and a densely calcified rind was seen covering the visceral and parietal pleura. No evidence of endocyst or cyst membrane was seen intraoperatively, ruling out pulmonary Echinococcosis. Cultures from fluid and tissue obtained intraoperatively were negative. The overall etiology of the patient's presentation remains unclear, although it is likely related to his history of chest tube placement.

	Discussion: The American Thoracic Society defines any empyema lasting four weeks and beyond as chronic. In the third pathogenic stage of empyema (organizing stage), the pleural fluid begins to organize, forming granulation tissue and an inflammatory process, which in late stages, can cause a fibrinous pleural covering (fibrous peel or pleural rind), encasing and compressing the visceral pleural surface, and preventing expansion of the lung (i.e., trapped lung). This case highlights an atypical presentation of empyema. Chronic late/stage empyema is a diagnosis that should be entertained when evaluating a patient with a lung mass or cyst.
Cassandra Roeder, MD Dr. Breanna Zarmbinski	*When No Spleen Leads to Gangrene. A Life-Threatening Case of DIC and Bacteremia in an Asplenic Patient* Introduction: Though splenectomy is curative for several conditions, it is not a procedure without risks. A rare yet serious complication of splenectomy is overwhelming post-splenectomy infection (OPSI) which only occurs in 0.1-0.5 %of splenectomy patients yet has up to a 50% mortality [4]. Purpura fulminans (a severe thrombotic subtype of DIC) is also more likely to develop in asplenic patients [1]. Case Presentation: A 34-year-old male with history of prior splenectomy from immune thrombocytopenic purpura presented to the emergency room after one day of vomiting, malaise, rigors, and fevers up to 105 degrees F. On presentation, he was profoundly hypotensive (BP 70/50), hypoxic, and febrile. His exam was notable for duskiness on his nose and cold hands and toes. His labs showed a lactate of 7.9, platelets of 15, elevated transaminases, and a creatinine of 3.7. CT of his chest showed diffuse patchy ground glass opacities within the lungs. He was admitted to the ICU for pressor support, fluid resuscitation, and broad-spectrum antibiotics for presumed septic shock secondary to pneumonia. With his hemodynamic instability and duskiness of his digits and nose, DIC was suspected and confirmed with laboratory evaluation, and he received cryoprecipitate and FFP. His hemodynamics improved over the next several days and blood cultures returned positive for Streptococcus pneumoniae. Infectious Disease was consulted for assistance with antibiotic management, and he was transferred to the floor. His toes and fingers became more purple in appearance and more painful, so vascular surgery was consulted. Vascular felt he had ischemia secondary to microthrombi in the setting of DIC and that the extent of injury may not be known for weeks. His hospital course was also complicated by hyponatremia from SIADH and hypoxia secondary to fluid overload. He was ultimately discharged home with 2 additional weeks of antibiotics. Primary care referred him to orthopedics, occupational therapy, and physical therapy for rehabilitation of the acrocyanosis of his hands and toes. He received updated vaccinations and has continued to follow up with infectious disease, wound care, and podiatry with nearly full recovery of his digits besides for osteomyelitis of one of his toes. Conclusion: Though complications of asplenia are rare, this case demonstrates how vigilant care providers and patients following splenectomy must be. Prevention of OPSI involves vaccination against S. pneumoniae, N. meningitidis, Hib, and influenza [3,4]. Individuals should also carry an emergency supply of antibiotics in case of sudden illness [3,4]. The treatment of purpura fulminans and symmetrical peripheral gangrene are topics of sparse evidence-based guidelines [1,2]. There is consensus to treat the underlying cause, prevent further infection, and remove non-viable tissue [2].

	For the outpatient provider, treating symmetric peripheral gangrene should be a multidisciplinary approach including consultation of surgical specialties, hematology, and dermatology [2].
Justin Sachs, MD	*Chronic Eosinophilic Pneumonia, an Uncommon Cause of a Cough* Introduction: Chronic eosinophilic pneumonia is a rare interstitial lung disease that typically presents as insidious onset cough and dyspnea. It is further characterized by bilateral, peripheral opacities on imaging, peripheral blood eosinophilia, and a bronchoalveolar lavage eosinophil count >25%. Frequency and cause are unknown; however, it is twice as common in women, typically presents in a patient's 30s and 40s, and most have a history of atopy and/or asthma. As this case proves, presentation can, at times, be atypical hence chronic eosinophilic pneumonia should remain on the differential across all populations. Case Description: A 72-year-old man with past medical history of HTN, DM2, allergic conjunctivitis, and remote history of childhood asthma presented to clinic with a 2-week history of dry cough. Symptoms began 1 week after chopping wood at his cabin in southeastern Minnesota. The patient had no recent changes in medications, he was prescribed lisinopril, but he has taken it for many years. He denies any history of acid reflux. On presentation patient was hypertensive, all other vitals were within normal limits including an oxygen saturation of 95%. Physical exam was unremarkable, no abnormal findings appreciated on lung auscultation. A chest X-ray was significant for bilateral peripheral upper lobe infiltrates, right more than left. CT confirmed the presence of upper lobe predominant nodular and consolidative opacities. Initial lab work revealed an elevated white blood count (11.81 K/ccm) neutrophil predominant with elevated eosinophils (1.5 K/ccm). He was Covid-19 negative. Pulmonology was consulted for further evaluation. A broad workup was negative for bacterial, viral, or fungal etiology. ANCA (Anti Neutrophilic Cytoplasmic Antibodies) testing was negative. Bronchoalveolar lavage finally revealed an elevated eosinophil count of 39% (normal 0-8%). He was diagnosed with chronic eosinophilic pneumonia and was started on prednisone. The treatment course was complicated by a hospital admission for hyperglycemia. Respiratory symptoms resolved and the patient was weaned from corticosteroids with resolution of symptoms. Discussion: Chronic eosinophilic pneumonia is an uncommon interstitial lung disease that can be easily misdiagnosed at first presentation. In this case there were multiple instances in which clinicians could have pursued incorrect diagnostic and treatment pathways for presumptive diagnosis of atypical bacterial or fungal infections. A broad differential and methodical approach allowed for early recognition and treatment for this individual.
Stephanie Saey, MD Dr. Alessandra Tomasi	*Don't Mess with the Pancreas: A Case of ICI-Associated Diabetic Ketoacidosis* Introduction: Immune checkpoint inhibitors (ICIs) have transformed cancer treatment over the last decade. Pembrolizumab, an ICI that blocks the programed cell death 1 (PD1) receptor, restores immune system function by releasing the brakes on T-cell activation. This mechanism makes Pembrolizumab an effective anti-tumor agent but also predisposes individuals

	to a host of immune-related adverse events. Here we present a case of diabetic ketoacidosis (DKA) secondary to Type 1 diabetes mellitus in the setting of Pembrolizumab use. Case Presentation: An 86-year-old woman presented to the Emergency Department (ED) with a six-day history of progressively worsening nausea, weakness, â€œbrain fog, polydipsia, and polyuria. She denied abdominal pain, diarrhea, vomiting, infection, or corticosteroid use. The patient had recently been diagnosed with Stage IV lung adenocarcinoma and had begun a 3-week regimen of Pembrolizumab 2mg with palliative intent. The most recent dose was 2 weeks prior to her presentation. Baseline endocrine workup during cycle 1 was notable for prediabetes (postprandial glucose 191 mg/dL, A1c 5.9% 3 months prior) and subclinical hypothyroidism (TSH 5.8, free T4 1.1, TPO antibodies <15). Family history was positive for Type 2 Diabetes Mellitus and unremarkable for autoimmune endocrinopathies. Her personal medical history included hypertension, fibromyalgia, and osteoarthritis; medications included Acetaminophen as needed. Upon arrival to the ED she appeared well and was hemodynamically stable though slightly tachypneic (respiratory rate 22). Laboratory evaluation was consistent with DKA: blood glucose 441 mg/dL, pH 7.14, bicarbonate 13 mmol/L, anion gap 19, and beta hydroxybutyrate 6.2 mmol/L. A1c was 7.5%. She responded well to intravenous insulin and fluids and was admitted to the hospital where she transitioned to basal/bolus insulin. She was discharged home on a multiple daily doses of insulin regimen. The patient established care with Endocrinology in the outpatient setting and continues to have suboptimal glycemic control requiring ongoing insulin titration. Notably, despite recent PET imaging showing excellent treatment response, Pembrolizumab has not been resumed. This decision was patient preference, as she has experienced significant psychological burden from the diabetes diagnosis. The patient described the events to her Oncology team as ‘traumatic’ and now has considerable daily anxiety and depressed mood. She is concerned that continuing Pembrolizumab will lead to additional adverse effects. Discussion/Conclusion: Type 1 diabetes mellitus with ketoacidosis is an uncommon adverse reaction in patients treated with Pembrolizumab, and our case adds to the literature of heterogenous presentations. Not only is ketoacidosis life-threatening, but ICI-induced diabetes is irreversible. Therefore, in addition to counseling patients on the signs and symptoms of hyperglycemia for earlier detection, the psychological impact of managing a chronic illness should be routine discussion in patient education prior to initiating ICI therapy. While certain risk factors for the development of ICI-induced diabetes have been suggested, further research into the pathophysiology is necessary to refine risk stratification tools that may guide shared-decision making, especially in the context of palliative treatment.
Bibek Saha, MD Dr. Nerissa Collins **Abstract Finalist**	*Significant Unintentional Weight Loss in a Sixty-Nine-Year-Old Diabetic* Introduction: Type 2 diabetes mellitus (DM2) affects more than 8.5% of Americans and the prevalence is expected to continue to rise along with obesity. Metformin, an effective first-line medication for DM2, has a well-known, typically beneficial, side effect of weight loss. Patients with DM2/prediabetes and overweight/obese patients (BMI 27kg/m2) without diabetes were shown to lose an average of 6.5% and 5.6% of body weight,

	respectively, after 6 months of metformin. However, here we report a case of a 69-year-old woman who lost 22.9% of her body weight unintentionally within 6 months of metformin initiation. Case Presentation: A 69-year-old female with DM2 presented with a 6-month history of unintentional weight loss. She reported decreased appetite, dizziness and fatigue since starting metformin. Specifically, she could not eat solid foods anymore and mainly ate a liquid diet. She was diagnosed with DM2 (HgbA1c 6.7) 1 year prior to presentation (PTP) and was started on Metformin 2g daily 6 months PTP as her HgbA1c rose to 7.9. At that time, her weight was 51.5kg (BMI: 27.04kg/m2). Three months PTP, her HgbA1c was 6.2. However, since starting metformin, her weight had decreased to 44.9kg and so the metformin dose was reduced to 1g daily. She stated that her appetite slightly improved after the dose reduction. She denied fever, chills, night sweats, dysphagia, choking on food, sensations of food getting stuck in her throat, abdominal pain, back pain, diarrhea, hematochezia, and melena. However, she reported a 3-month history of constipation and a 3-week history of 'pain in her bones' diffusely. She denied any family history of malignancy including esophageal, gastric, and colorectal cancer. On exam, her vitals were normal, and she weighed 39.7kg (BMI: 20.4kg/m2). She was thin but not cachectic with no concerning skin rashes/lesions, no lymphadenopathy, no abdominal pain on palpation, and no foot ulcers. Metformin was discontinued. The 22.9% weight loss, especially in a non-obese individual, appeared too high to be attributed to metformin as per the literature prompting investigation for hyperthyroidism, TB, CLL, HIV, malignancy (multiple myeloma, breast cancer, colon cancer), celiac disease and adrenal insufficiency. Labs showed: normal CBC with differential, overall unremarkable CMP, normal TSH (1.8), negative QuantiFERON-Tb Gold assay, negative HIV test, negative celiac serologies, normal AM cortisol (18), normal CRP (<3), slightly elevated ESR (33), and negative monoclonal gammopathy panel. Additionally, she had a normal mammogram 6 months PTP, and colonoscopy performed after the visit was not suggestive of cancer. Ten days after metformin discontinuation, her appetite improved, and she was eating solids again and felt more energetic. Her weight was 41.4kg. On follow-up, approximately 6 months after metformin discontinuation, her weight was 45.8kg. Given the overall negative workup as well as improved symptomology and weight after metformin discontinuation, her weight loss was attributed to metformin. Discussion: While metformin is typically associated with mild-moderate weight loss, it should be kept in the differential for significant unintentional weight loss, especially if it occurs within a short time frame. However, more dangerous causes should also be worked up and excluded.
Jason Sakizadeh, MD Dr. Lauren Fontana	*What's the (White) Matter?* Introduction: Progressive multifocal leukoencephalopathy (PML) is a fatal demyelinating disease of the central nervous system (CNS). This case illustrates the importance of considering PML in post-lung transplant recipients with neurologic deficits. Case Presentation: A 69-year-old man with a history of end-stage chronic obstructive pulmonary disease status post bilateral lung transplantation on tacrolimus and mycophenolate mofetil developed lethargy, gait instability,

	delayed speech, and progressively worsening pulmonary function tests (PFTs) seven months post-transplant. His worsening PFTs raised concern for acute cellular rejection (ACR), and he was started on empiric treatment for ACR with intravenous steroids followed by an oral steroid taper. The patient progressively developed short term memory loss, behavior changes, episodes of staring into space, confusion, and limited verbal responses over the course of 2-3 weeks. His gait instability and aphasia acutely worsened approximately 3 weeks after the initiation of empiric steroids. There was concern that he may have suffered a stroke and he had an outpatient computed tomography (CT) of the head that demonstrated bifrontal, temporal deep and subcortical white matter hypoattenuation which was thought to represent a subacute white matter process. He was sent to the emergency department for expedited workup and was subsequently admitted to the hospital. Neurological exam was notable for inattention, delayed response to questions, intermittent following of commands, tremors, and a few beats of clonus bilaterally. Brain magnetic resonance imaging revealed abnormal white matter signals throughout the bilateral frontal, parietal, and temporal lobes. There was initial concern for calcineurin inhibitor-induced toxic leukoencephalopathy, and his tacrolimus was held and basiliximab was administered for immunosuppressive therapy bridging. Lumbar puncture was performed on hospital day 2. Cerebrospinal fluid (CSF) showed a nucleated cell count of 2/uL, glucose of 52 mg/dL, protein of 74.9 mg/dL. CSF cytology, Toxoplasma gondii polymerase chain reaction (PCR), Epstein-Barr virus PCR, herpes simplex virus 1 and 2 PCR, and venereal disease research laboratory tests were negative. The CSF JC virus PCR was positive with 4,100 copies/mL. The tacrolimus level was in a therapeutic range. Repeat lumbar puncture on hospital day 15 was obtained for further evaluation of new fevers with a repeat CSF JC virus PCR demonstrating an increase in the CSF viral load at 8,000 copies/mL. The constellation of findings including rising CSF JC viral loads, radiographic imaging, and neurologic changes made PML secondary to JC virus the most unifying diagnosis. His mentation and speech gradually worsened throughout the hospitalization, and he was ultimately discharged with hospice on hospital day 25. The patient expired 3 days after discharge. Discussion: PML should be suspected in immunocompromised patients including lung transplant recipients presenting with subacute neurological deficits. Brain MRI can show white matter changes that should make one consider a PML diagnosis, and quantitative JC viral PCR values from CSF can be valuable diagnostic tools with potential value in prognostication as well.
Abdulsabur Sanni, MD	*Hyper-Hemolytic Syndrome (HHS); A Morbid and Underrecognized Complication of Frequent Transfusions in Sickle Cell Disease* Introduction: Patients with Sickle cell anemia often require multiple blood transfusions throughout their lifetime, exposing them to various risks associated with frequent transfusions including transfusion reactions, iron overload-related complications such as liver cirrhosis, restrictive cardiomyopathy, hypogonadism, and rarely delayed hemolytic transfusion reactions (DHTF) or Hyper-Hemolytic syndrome (a subtype of DHTF). This is why physicians should be conservative with blood transfusions unless

	clinically indicated. Case description: We present the case of a 23-year-old female with Sickle cell anemia (HbSS genotype) who sought medical attention due to an acute vaso-occlusive pain episode. She presented with severe bilateral lower extremity pain. Laboratory investigations revealed a critically low hemoglobin level of 3 g/dL (baseline 6-7), increased lactate dehydrogenase (LDH), decreased haptoglobin, high reticulocyte count, and a positive direct Coombs (DAT) test with anti-D antibodies and evidence of hemolysis on a peripheral blood smear, despite having a B-positive blood group. Notably, she had received blood transfusion about 2 weeks prior with a negative DAT for anti-D antibodies at the time. The patient received multiple matched B-positive blood transfusions to correct her anemia, but her hemoglobin levels failed to improve, and markers of hemolysis remained elevated, raising suspicion for hyper-hemolytic syndrome (HHS). Given the clinical presentation and laboratory findings, concerns for a delayed hemolytic transfusion reaction were raised and the emergence of anti-D antibodies and anti-Kpa antibodies on the DAT/eluate prompted a diagnosis of Hyper-hemolytic transfusion reaction. The patient was initiated on high-dose steroids and a five-day course of intravenous immunoglobulin (IVIG), but her anemia persisted, prompting a transfer to an outside hospital for further management. At the receiving facility, she was administered erythropoietin (EPO), Rh-negative matched red blood cells, and eculizumab. Subsequently, her clinical condition improved, and she was discharged in a clinically stable state. Discussion: This case underscores the significance of recognizing hyper-hemolytic syndrome as a rare and highly morbid complication of frequent blood transfusions in SCD. The distinction between hyper-hemolytic syndrome and delayed hemolytic transfusion reactions is essential, requiring a high index of suspicion and prompt medical management, as additional blood transfusions can exacerbate anemia and result in life-threatening consequences in HHS. The hyper-hemolytic syndrome (HHS) has been postulated to be associated with the pro-inflammatory state inherent to SCD leading to hemolysis of auto and allo red blood cells. Our case emphasizes the importance of raising awareness of this condition when managing patients with SCD, especially for internists who frequently manage these patients on the medical floors, particularly when confronted with refractory anemia following blood transfusions. Early recognition and appropriate intervention are crucial for improving outcomes in these patients. Bibliography; Menakuru SR, Priscu A, Dhillon V, Salih A. Acute Hyperhemolysis Syndrome in a Patient with Known Sickle Cell Anemia Refractory to Steroids and IVIG Treated with Tocilizumab and Erythropoietin: A Case Report and Review of Literature. Hematol Rep. 2022 Jul 21;14(3):235-239. doi: 10.3390/hematolrep14030032. PMID: 35893156; PMCID: PMC9326715.
Deepon Sarkar, MD	*An Uncommon Cause of Cavitary Lung Lesions* Introduction: A cavitary lung lesion is defined as a gas-filled space within a pulmonary consolidation, mass, or nodule, and it is produced by expulsion of a necrotic part of the lesion through the bronchial tree. Cavitary lung lesions found on chest imaging can present with a wide range of pathological

	processes. The differential for cavitary lung lesions involves both infectious, such as bacterial, mycobacterial, fungal, or parasitic etiologies, and non-infectious processes, including autoimmune, neoplastic, vascular, congenital, or traumatic. Case Description: A 54-year-old male from Ecuador with a history of treated latent tuberculosis and former tobacco use presented to clinic for evaluation of incidental lung nodules with cavitary lesions found on chest imaging. He reported having five years of persistent cough associated with dark sputum production but no B symptoms. He moved from Ecuador two years ago and previously worked in a goldmine for four years. While living in Ecuador, he would often eat seafood, including raw crab. Physical exam was unremarkable with normal vitals. Lab workup was notable for eosinophilia to 1.23 k/cmm. CT chest showed bilateral nodular opacities with foci of cavitation. Based on these findings, a broad workup was initiated. He had 3 negative sputum cultures for AFB, negative ANCA and rheumatoid factor antibodies, negative fungal serologies, negative strongyloidiasis serology, and negative ova and parasites in both the sputum and stool. A CT-guided core needle biopsy of a nodule was obtained and demonstrated evidence of eosinophilia. Serologies for Paragonimus, a genus of parasitic trematodes, were sent to the CDC and came back positive; thus, supporting a diagnosis of pulmonary paragonimiasis. He was initiated on Praziquantel 25 mg/kg three times daily for two days. During a follow-up appointment one month later, he reported improvement of his chronic cough, and lab workup revealed resolution of his peripheral eosinophilia. He was recommended to follow up in two months for repeat imaging to assess for improvement of the chest imaging abnormalities. Discussion: This case demonstrates an uncommon cause of cavitary lung lesions: pulmonary paragonimiasis. The patient's geographic exposure to raw freshwater shellfish in Ecuador, peripheral eosinophilia, and characteristic lung imaging findings with tissue biopsy showing eosinophils should prompt consideration for a parasitic infection and further workup for paragonimiasis. The combination of cavitations, irregular linear lesions, nodular opacities, or pleural effusions on chest imaging can aid as radiographic clues for the diagnosis of paragonimiasis given its known pathogenesis involving larval penetration through the diaphragm into the pleural cavity via burrowing tracts. Although sputum and stool ova and parasites were found to be negative, serologies for parasites should still be obtained because those tests have low sensitivities, especially for Paragonimus. This case also highlights the importance of obtaining a thorough social history, including country of origin, recent travel, work, environmental exposures, and diet, which allowed for early clinical suspicion of a parasitic infection. This resulted in timely identification and appropriate treatment of the patient's pulmonary paragonimiasis.
Reid Schlesinger, MD Dr. Alessandra Tomasi	*Difficulties in Diagnosis of Urogenital Tuberculosis and the Importance of Rapid Molecular Tests* Introduction: Urogenital tuberculosis (TB) is the second most common form of extrapulmonary TB. Symptoms are often non-specific and may not present until late in the disease course, leading to delays in diagnosis and treatment with devastating consequences for patient outcomes. Diagnosis requires evidence of Mycobacterium tuberculosis (Mtb) in the urine or on tissue culture. Urine culture for Mtb remains the gold standard, but results can take

	weeks to return, and sensitivity is variable. Urine PCR, a newer molecular assay, has a significantly faster turnaround time and high sensitivity and specificity, however, it is often used as an adjunct to conventional tests. Case Presentation: A 56-year-old woman from Vietnam with a history of latent TB, systemic lupus erythematosus, lupus nephritis type IV on chronic mycophenolate and prednisone, and Evans syndrome was admitted with one month of bilateral flank pain and a year-long history of suprapubic pain accompanied by persistent sterile pyuria and hematuria. During previous hospitalizations for similar symptoms and declining kidney function, empiric treatment for presumed recurrence of lupus nephritis was trialed but unsuccessful. On admission, she was hemodynamically stable with bilateral flank and suprapubic tenderness on exam. Labs were notable for creatinine 1.68 mg/dL (baseline 1.1 mg/dL), WBC 19.8 x10(9)/L, urinalysis with subnephrotic proteinuria, leukocyte esterase, >100 WBCs per hpf, 51-100 RBCs per hpf, but negative nitrite and bacterial urine culture. CT scans revealed significant cystitis, early bilateral pyelonephritis, and a calcified right upper lobe nodule. The patient was started on cefepime for empiric treatment of bacterial pyelonephritis but did not improve significantly. Additional history revealed uncertainty about whether the patient had been adequately treated for latent TB, and, in fact, QuantiFERON Gold and urine Mtb PCR returned positive shortly after. Antituberculosis therapy was deferred while awaiting confirmatory testing, including urine cultures, urine acid-fast smears, and sputum studies, which did not show evidence of Mtb initially. One week later, urine cultures returned positive for Mtb, and she was promptly started on rifampin, isoniazid, pyrazinamide, and ethambutol. Two weeks after that, sputum culture also demonstrated growth of Mtb. The patient was discharged but readmitted for a multi-drug resistant urinary tract infection and complications from the antituberculosis treatment. Her condition continued to worsen, and after thoughtful discussions with the patient and her family, she opted for comfort measures in favor of continued treatment, before passing away days later. Conclusion: This case underscores the importance of timely treatment for urogenital TB and the pitfalls of conventional TB testing, which can be slow, thereby delaying treatment initiation. It also highlights the value of rapid molecular tests and the challenges posed by diagnostic uncertainty when initial test results are discordant. Given the severe consequences associated with delayed treatment for urogenital TB and the accuracy of many urine Mtb PCR tests, antituberculosis therapy should be strongly considered if PCR testing is positive, even without confirmation from more conventional tests. This is especially true in patients with high pretest probability for urogenital TB, such as those with latent TB, immunosuppression, and chronic renal disease.
Elizabeth Schroer, MD Dr. Breena Zarmbinski	*Pulmonary Blastomycosis: A Case of ARDS Requiring ECMO* Introduction: Blastomyces is an endemic fungus to the Great Lakes with the some of the highest infection rates in Minnesota that can cause granulomatous pulmonary disease by inhalation of fungal spores found in the soil [1, 3]. The spectrum of disease in Blastomycosis ranges from asymptomatic to acute respiratory distress syndrome in up to 10% of cases with mortality rates as high as 89% [1, 2, 5]. However, this relatively

	uncommon disease often has delays in diagnosis of greater than 4 weeks in 40% of cases, emphasizing the importance of increasing awareness about this condition [4]. Case Presentation: A 24-year-old male with a past medical history of ADHD presented to the emergency department with four weeks of cough, wheezing, and shortness of breath. A week prior he was seen in the emergency department with similar symptoms; chest x-ray showed a dense opacification of the right upper lobe. He was diagnosed with community acquired pneumonia and treated with cefuroxime and azithromycin. He did not have significant improvement with antibiotics and developed night sweats so he returned to the emergency room. The patient met sepsis criteria with tachycardia, tachypnea, and leukocytosis to 20.4. Lactate was normal. CT chest showed dense alveolar consolidation with cavitation in the right upper lobe with extensive bronchopneumonia in the lower lobes. Notably, there were several known cases of tuberculosis in the community he lived in within the last year. The patient was started on ceftriaxone and azithromycin and placed in airborne precautions for possible tuberculosis. Infectious disease recommended broadening to meropenem as well as obtaining AFB cultures, sputum cultures, and blastomycosis urine antigen. He was persistently tachycardic and febrile despite several days of antibiotics, so was started on itraconazole empirically due to concern for possible fungal infection. The following day the patient decompensated with worsening dyspnea, hypoxia requiring heated high flow, tachycardia, and hypotension requiring ICU transfer and intubation. KOH prep returned showing broad-based budding yeast and he was empirically started on amphotericin B. Blastomycosis antigen eventually returned positive; AFB cultures were negative. Developed ARDS with worsening P/F ratio and hypercarbia, ultimately met criteria for cannulation to venovenous ECMO. Underwent tracheostomy and remained on ECMO for 17 days prior to decannulation. He continued to improve and was eventually discharged to inpatient rehab. Conclusion: This case illustrates the potential rapid progression of severe blastomycosis in an otherwise young and healthy individual. Clinicians must have a high degree of clinical suspicion for possible blastomycosis in patients not responding to typical community acquired pneumonia treatment [4]. Additionally, transfer to an ECMO capable facility should be considered due to possible benefit for rescue treatment in cases of severe ARDS [1].
Alexandra Skovran, MD	*Concussion or Invasive Fungal Infection: A Rare Case of CNS Blastomycosis* Introduction: Blastomycosis is a fungal infection that has growing prevalence in the midwest. While it often presents as pneumonia, CNS involvement is uncommon in immunocompetent hosts, occurring in less than five percent of cases. Case presentation: A 39-year-old female with no past medical history presented to an outside facility three days after a motor vehicle accident, complaining of persistent headaches after a motor vehicle accident. Head CT showed new brain mass with mild shift and hydrocephalus, and she was transferred to a higher level of care. Upon presentation, she complained only of headaches and intermittent night sweats. History significant for pulmonary blastomycosis (06/2020) treated with Itrazonazole for approximately 5 months. Brain MRI showed a 4.7cm left thalamic lesion, favoring high-grade glioma. Neurosurgery performed a

	biopsy two days later which was relatively unrevealing and she was discharged. At a two-week clinic visit, repeat MRI showed multiple ring-enhancing nodules, some increased in size from her previous. Infectious process was favored and she was readmitted for repeat biopsy and potential ventriculostomy. Biopsy was significant for large amounts of pus, consistent with fungal infection. Infectious disease (ID) was consulted and this was thought to be recurrent blastomycosis. She was started on liposomal amphotericin B IV, which unfortunately caused an acute kidney injury and electrolyte disturbances. She was switched to Voriconazole, and later to Itraconazole due to intolerance, and followed regularly with ID with repeat imaging showing improvement in lesions. Discussion: Blastomycosis is a rare fungal infection that most often presents as a pulmonary infection after inhalation of spores (1). It is unique compared to other deep fungal infections, in that it occurs in both immunocompromised and immunocompetent hosts. CNS blastomycosis is a rare complication that occurs in only 5-10% of cases. The recommended treatment is six weeks of amphotericin followed by a twelve-month course of azole therapy, along with close follow up with infectious disease.
Kevin Stanko, MD Dr. Alessandra Tomasi	*A Case of Leukocytoclastic Vasculitis in the Setting of MSSA Bacteremia* Introduction: Leukocytoclastic vasculitis is a rare, small vessel vasculitis characterized by immune complex deposition in dermal capillaries and venules. The annual incidence is approximately 45 cases per million individuals. Clinical features include palpable purpura involving the lower extremities, small vessel involvement, and extracutaneous involvement in approximately 30% of patients. Common triggers include infection with Mycobacterium, Staphylococcus aureus, Neisseria, Chlamydia, and HIV. Leukocytoclastic vasculitis has also been associated with nearly all classes of drugs, though penicillins, cephalosporins, sulfonamides (including most loop and thiazide-type diuretics), phenytoin, and allopurinol have been most frequently implicated. Less common triggers include underlying malignancy and connective tissue disease, although 50% of cases are idiopathic in nature. Case Presentation: A 64-year-old male with medical comorbidities notable for dual-chamber permanent pacemaker placement for high grade AV block was admitted for fevers, chills, and weakness. Blood cultures demonstrated high-grade methicillin-susceptible Staphylococcus aureus (MSSA) bacteremia with TEE and PET-CT demonstrating pacemaker lead infection as well as mobile irregular echodensities concerning for tricuspid valve endocarditis. The patient was started on cefazolin 2 g every 8 hours and his cardiac device was extracted on hospital day 8. On hospital day 10, a new non-pruritic, non-painful, petechial rash was appreciated involving the bilateral feet and lower extremities. The majority of lesions were non-palpable, albeit with few focal raised areas. Notable laboratory values included hemoglobin 9.8 g/dL (stable), white count 9.4 x 109/L (eosinophils 0.12 x 109/L), platelet count 252 x 109/L, creatinine stable at 0.67 mg/dL. ANCA serologies were negative with complement levels within normal limits. Over the following days, the lesions progressed to involve scattered areas on the bilateral hips and flanks, prompting biopsy. Pathology was consistent with leukocytoclastic vasculitis, with immunofluorescence pattern showing IgA granular deposition within the

	walls of superficial dermal vessels. Outcome: In this case, cutaneous leukocytoclastic vasculitis was most likely secondary to MSSA bacteremia. Given that the rash was slowly progressive, and the patient demonstrated no evidence of systemic involvement, observation was recommended. Moreover, because the rash was not consistent with a classic drug eruption, which has a morbilliform morphology and begins with truncal involvement before spreading to the extremities, no changes in antimicrobial therapy were recommended. Symptoms resolved over the following days to weeks. Discussion: Patients with leukocytoclastic vasculitis syndromes should be monitored for signs/symptoms of systemic involvement including serial monitoring for renal, gastrointestinal, pulmonary, or neurologic manifestations that may be more consistent with microscopic polyangiitis or polyarteritis. Once systemic involvement has been excluded, treatment of cutaneous leukocytoclastic vasculitis is largely symptom-based, and mild cases of do not require immunosuppressive agents. Renal or systemic involvement may warrant high dose steroids or immunosuppressive agents, though underlying infectious triggers should certainly be considered in this setting. In cases of drug-induced leukocytoclastic vasculitis, discontinuation of the offending agent should bring about symptom resolution in days to weeks. Conclusion: The differential diagnosis for palpable purpura involving the lower extremities is broad. In this case we discuss the presentation, workup, and diagnosis of leukocytoclastic vasculitis, which is a rare cause of these symptoms.
Williams Swain, MD Dr. Courtney Arment	*From Mouth to Myocardium: A Rare Case of Systemic Sarcoidosis in Multiple Organ Systems* Case Presentation: A 45-year-old male presents after experiencing witnessed cardiac arrest with return of spontaneous circulation. Two years prior, he had focal periodontal disease that was biopsied during dental work. This showed mucosa overlying fibrous connective tissue with granulomas composed of histiocytes and giant cells. No further workup was undergone despite a history of heart palpitations in the antecedent 5 years and a single syncopal episode. After stabilization, echocardiogram showed mild structural disease and coronary catheterization was unremarkable. Cardiac MRI showed areas of epicardial late gadolinium enhancement in the inferior and anterior walls in a noncoronary distribution. Chest CT showed peribronchovascular pulmonary nodularity as well as splenic and hepatic lesions. The lung was biopsied, showing fibrosis with granulomata and numerous multinucleated giant cells with negative GMS and fite stains. An implantable cardioverter-defibrillator was placed, and mexiletine, amiodarone, metoprolol, methotrexate, prednisone, and Bactrim were started. Cardiac PET CT scan done 1 month later showed no focal FDG uptake in the LV myocardium. Since starting treatment, he has not experienced any ICD shocks or had any abnormal rhythms on Holter monitoring. Discussion: Sarcoidosis is a disease of noncaseating granulomas, and variably affects different organ systems. With the exception of a specific disease patterns definitive diagnosis requires pathology findings with negative fungal and mycobacterial stains. In this case, this patient's gingival

	pathology was done years prior to developing complications of systemic sarcoidosis at an outside dental office and did not prompt further workup at that time. Our patient's history of (probable) oral sarcoidosis suggests that earlier investigation may have prevented his arrest. As of 2005, there were only 68 cases in the literature describing oral sarcoidosis. This may present as nodules or ulcers in the buccal mucosa, gingiva, palate, tongue, lips, jaw bones, and glands. Roughly half of the time, it can precede other systemic manifestations.6 To our knowledge, this case is the first in the literature that describes oral sarcoidosis preceding cardiac arrest. In the setting of sarcoidosis and myocardial imaging involvement, this patient also meets criteria for cardiac sarcoidosis. Myocardial involvement may be seen in as many as 25% of autopsy reports of those with systemic sarcoidosis, although the prevalence of clinical disease is likely lower. American Thoracic Society guidelines advocate baseline EKG as well as additional testing based on presence of cardiac symptoms following diagnosis of extracardiac sarcoid. These additional diagnostics include holter monitor findings and echocardiogram, which may predict the involvement of cardiac disease better than routine electrocardiogram (ECG). Additional evaluations for cardiac sarcoidosis include cardiac MRI and/or cardiac PET scans. PET scans can only identify active inflammation as in our case. Conclusion: While sarcoidosis is a rare disease, and oral manifestations an even more rare subset of patients, it is important for clinicians to have a low threshold to complete recommended baseline screens when encountering a patient with new diagnosis of sarcoidosis. As illustrated in this case, the completion of proper workup may have prevented a catastrophic health event.
Emily Syverud, MD	*How Sweet it is: A Unilateral Pleural Effusion in a Patient on Ambulatory Peritoneal Dialysis* Introduction: Pleural effusion is a common etiology for shortness of breath and has a wide differential diagnosis. Here we present an unusual cause of pleural effusion that is unique to patients with end-stage renal disease (ESRD) who use peritoneal dialysis (PD). Case Presentation: An 80-year-old woman with a past medical history significant for ESRD on PD, atrial fibrillation, and pulmonary embolism on long-term anticoagulation with warfarin presented to the emergency department (ED) with shortness of breath. She had been feeling progressively more short of breath for 3-4 days and in the ED had a new oxygen requirement of 3-4L of nasal cannula O2. She was not tachycardic or hypotensive and denied any lower extremity swelling, recent upper respiratory symptoms, or chest pain. Her exam was significant for diminished breath sounds on the right without crackles, wheezes, or rhonchi. Bedside cardiac ultrasound showed grossly preserved left ventricular function and A-line predominance. Her initial chest X-ray demonstrated a possible right-sided effusion or atelectasis with mild pulmonary edema. A follow-up chest CT showed a large right pleural effusion causing compressive atelectasis throughout the right lung. A bedside thoracentesis was performed and 1L of fluid was drained, the fluid was clear and yellow-tinged. The patient had improvement in her shortness of breath with a decreased oxygen requirement following the procedure.

	Analysis of the pleural fluid showed a transudative effusion based on Light's criteria with a glucose content of 218 mg/dL, a concentration almost identical to that of her peritoneal dialysis fluid. The patient was seen by the nephrology team, who discussed a trial of decreasing her PD exchange volumes in an effort to decrease re-accumulation of the effusion. Unfortunately, she returned within 2 months with another right-sided effusion and was transitioned to hemodialysis. Conclusion: Pleural effusion is an uncommon complication of peritoneal dialysis that can result in acute hypoxic respiratory failure. The effusion is most commonly caused by a small diaphragmatic defect that allows for transdiaphragmatic leakage of dialysis fluid. While not performed in this case, the defect can be located using technetium-99m (Tc-99m) peritoneal scintigraphy. Treatment options include surgical repair of the diaphragmatic defect or pleurodesis. Patients may also elect to trial smaller volumes of peritoneal dialysis or transition to hemodialysis either temporarily or permanently. This case demonstrates the importance of thoracentesis in diagnosis, as a high glucose content in the pleural fluid from the peritoneal dialysis is pathognomonic for PD-associated hydrothorax.
Kristna Techar, MD Dr. Hayley Turch Dr. Amy Holbrook	*Don't DILI-dally Over Losartan Induced Liver Injury* Case description: A 23-year-old woman was diagnosed with hypertension and was started on losartan. About one month later, she presented to clinic with an asymptomatic elevation of her liver enzymes found on routine screening done by her employer with an AST of 85 and an ALT of 129. Labs were repeated and showed worsening of ALT to 2,124 and AST to 1,063 and she was admitted to the hospital. Her losartan was held on admission and her transaminases continued to trend downwards throughout her stay and her liver synthetic function remained intact with INR 1.1 and albumin of 4.0. Further testing showed ANA titer of 1:2560, with negative anti-mitochondrial antibody, anti-smooth muscle antibody, Alpha-1 antitrypsin, HIV, CMV, hepatitis A, B and C and a normal ceruloplasmin and positive EBV IgM and IgG suggesting old infection. Her liver biopsy showed a lobular hepatitis with clusters of ceroid laden macrophages and some accentuating in zone 3 that suggested a drug-induced liver injury. She continued to improve and was discharged with a plan to follow up with rheumatology and hepatology as an outpatient. Her losartan was discontinued and her liver enzymes returned to normal without additional therapy. Discussion: Based on her labs and presentation the most likely diagnoses were a drug induced liver injury vs autoimmune hepatitis. However, the pathology strongly favored a drug induced liver injury. Drug induced liver injury (DILI) from angiotensin receptor blockers (ARB) is a known but uncommon adverse effect. One study reported a 2% rate of abnormal liver function tests after starting ARB therapy. (1) There are also multiple other case reports showing the same pattern of injury with similar time lines of enzyme elevation and resolution. (2,3). Interestingly, the patient's only medication was losartan, which was started weeks before her lab tests became abnormal. Often, patients are on multiple medications and it is hard to confirm which is the culprit. This case illustrates the potential for severe drug-induced liver injury from a commonly prescribed medication, and raises awareness of the possibility of an uncommon adverse effect from ARB therapy.
Nadia Toumeh,	*Is it the Lungs or the Heart? A Case of Hypoxemia in a Patient with Stage IV*

MD Dr. Franklyn Wallace Dr. Kyla Lara-Breitinger	*NSCLC on Osimertinib* Introduction: Since its FDA approval in 2017, the tyrosine kinase inhibitor osimertinib has emerged as a first-line treatment for patients with locally advanced or metastatic non-small cell lung cancer (NSCLC) who have identified epidermal growth factor receptor (EGFR) mutations. Due to its expanding applications, it is essential for the general internist to familiarize themselves with its potential adverse effects. Here, we present a severe case of osimertinib-associated pneumonitis and the diagnostic challenges associated with its heterogenous presentation. Case Presentation: A 78-year-old female with stage IV EGFR-mutated NSCLC on osimertinib, heart failure with preserved ejection fraction (HFpEF) secondary to ischemic heart disease, and moderate mitral valve regurgitation presented to the emergency department with three days of progressive dyspnea and generalized fatigue. She denied having any cough, chest pain, fevers, chills or recent sick contacts. Presentation vital signs were significant for hypoxia (requiring 3-4 L NC), tachypnea (RR 30-40), and tachycardia (100-110 bpm). NT-pro-BNP was elevated around 5,000 pg/mL, and CT of the chest with IV contrast revealed diffuse ground glass opacities with interlobular septal thickening consistent with pulmonary edema versus an infectious or inflammatory process. Echocardiogram revealed an enlarged IVC with reduced inspiratory collapse. She was admitted to the general cardiology service, wherein she was initially managed with IV diuretics and broad-spectrum antimicrobials for coverage of community-acquired pneumonia and consequent exacerbation of HFpEF. Despite treatment, hypoxemia worsened gradually over the subsequent days. Medical oncology was consulted for consideration of osimertinib-associated pneumonitis. Their team recommended initiating 1 g/kg of prednisone daily, which resulted in gradual resolution of hypoxemia over the coming days prior to hospital dismissal. Prednisone was rapidly tapered in the outpatient setting, and follow-up imaging in one month revealed radiographic resolution of pneumonitis. She was transitioned thereafter to afatinib for management of NSCLC. Case Discussion: We present a case of osimertinib-associated pneumonitis which was initially mistaken for an exacerbation of heart failure. Osimertinib-associated lung toxicity is relatively rare (< 2% of individuals using the drug), has highly variable clinical/radiographic features, and is caused by yet-uncertain mechanisms. Although most cases are mild, our case demonstrates that an important minority of patients may benefit from prompt immunosuppressive therapy with high-dose prednisone. Our case highlights the importance of early involvement by medical oncology for patients presenting with respiratory failure while on novel tyrosine kinase inhibitors such as osimertinib.
Laura Tucker, MD Dr. Kareem Al-Qadi Dr. Megan Kristan	*Fluctuating Post-Partum DDAVP Needs for Central Diabetes Insipidus* Introduction: Central diabetes insipidus (CDI) is a disorder characterized by polyuria and hypernatremia. Is it caused by deficiency of the antidiuretic hormone arginine vasopressin (AVP), which is released by the posterior pituitary gland and acts on V2 receptors in the kidney to promote reabsorption of free water. CDI is managed with desmopressin (DDAVP), a synthetic form of AVP. Dosing of DDAVP is titrated per individual based on urine output and serum sodium and osmolality.

	During pregnancy, the placenta produces an enzyme called vasopressinase which causes accelerated degradation of endogenous AVP but is inactive against the synthetic analogue DDAVP due to it being deaminated at the N-terminus. If patients with CDI have partial function of endogenous AVP, the action of placental vasopressinase can necessitate an increased dose of DDAVP during pregnancy to compensate for the degradation of AVP. Case Presentation: A 39-year-old woman was admitted for a caesarean delivery of her first pregnancy. Her medical history is significant for panhypopituitarism and chronic CDI after surgical removal of a non-functioning pituitary macroadenoma at age 35. For her CDI, prior to pregnancy she had been on a stable dose of DDAVP nasal spray 10mcg twice daily. During pregnancy this dose was increased to three times daily based on her self-reported increase in urinary output. She remained normonatremic during pregnancy using this dosing and her sodium 1 week prior to delivery was normal at 138 mmol/L. After delivery, the patient's DDAVP dose was immediately reduced back to the pre-pregnancy dose of twice daily in anticipation of the reduced effect of placental vasopressinase. The day after delivery, her sodium reduced slightly to 134 mmol/L and her urine output was 1.3L over 24 hours. Two days after delivery the patient reported feeling off and had a significant headache; her sodium was found to be 124 mmol/L, necessitating hypertonic saline administration and fluid restriction. Serum/urine osmolalities were not obtained at the time. DDAVP was held completely on this day and she produced 6.4L of urine over the following 24 hours. The following day her sodium normalized to 140 mmol/L. She was then resumed on twice-daily dosing and remained normonatremic by time of discharge the following day and on outpatient follow-up labs. Discussion: This patient required increased DDAVP dosing to manage her CDI during pregnancy and demonstrated a rapidly reduced need after delivery. Because placental vasopressinase is believed to degrade only endogenous AVP and not exogenous DDAVP, this suggests that the patient may have only partial CDI and retains some AVP production contributing to her free water homeostasis. In the days after delivery, reduction in placental vasopressinase caused greatly reduced degradation and therefore transient increased sensitivity after recovery of her AVP. In patients with CDI who have some suspected remaining AVP production, DDAVP requirement needs to be considered carefully during and immediately after pregnancy. Patients may require inpatient monitoring of urine output and serum sodium and osmolality in the days after delivery in order to understand safe new dosing requirements of DDAVP.
Hayley Turch, MD Dr. Anya Jamrozy	*NSTEMI in a 17-Year-Old Woman* Introduction: Myocardial infarction is a rare but life-threatening complication of Kawasaki disease. In young adults with minimal risk factors of cardiac disease presenting with chest pain, non-atherosclerotic etiologies of coronary disease should be considered. Case Presentation: A 17-year-old female presented to the Emergency Department with non-reproducible, substernal chest pain after eating dinner. She had exerted herself at lacrosse practice hours earlier without anginal symptoms. EKG showed t-wave inversions in leads III and V1. An initial

	high sensitivity troponin was 373 (reference 6-10 ng/L). It then rose to 460 in 2 hours, and 497 in 4 hours. C-reactive protein and sedimentation rate were negative, and CT did not show a pulmonary embolism. Lipid panel was grossly normal with LDL of 40 (reference <=130 mg/dL), and normal lipoprotein A. The initial differential included myocarditis, pericarditis, spontaneous coronary artery dissection, and vasospasm given patientâ€™s age and low-to-minimal risk factors. CT of the coronary arteries showed complete occlusion of the mid left circumflex artery with a perfusion defect in the first obtuse marginal distribution consistent with infarct. There was no evidence of coronary dissection. Cardiac MRI showed a discrete acute transmural myocardial infarction in the first obtuse marginal distribution with hypokinesis. The pericardium was normal, and no myocarditis was seen on cardiac MRI. Findings were thought to be due to an embolic event, and she proceeded with invasive coronary angiogram. She was found to have a large amount of thrombus in the distal circumflex and proximal first marginal branch which was removed via aspiration thrombectomy. She was also found to have an aneurysmal segment in the mid-circumflex that subsequently had evidence of dissection requiring angioplasty. The proceduralist opted against stenting given the patient's young age and need for multiple stents. The patient was pain-free after the procedure, and repeat EKG showed resolution of t-wave inversions in lead III. Transthoracic echocardiogram was done post-operatively which showed normal left ventricular systolic function (estimated EF 55-60%) and no pericardial effusion. Workup for arterial thrombophilia causing thrombosis was negative including lupus anticoagulant, beta 2 glycoprotein, and cardiolipin antibodies. The etiology of thrombus is thought most likely due to a Kawasaki-related aneurysm given findings of a focal area with proximal ectasia and no atherosclerosis. She was treated with apixaban and clopidogrel for 3 months and low-dose metoprolol. This case demonstrates the sequela of Kawasaki disease manifesting as a myocardial infarction in an otherwise healthy young adult. Based on 2 studies, 5-6% of young adults, 40 years of age or younger, presenting with myocardial infarction developed disease from suspected sequelae of Kawasaki disease. Thrombotic coronary lesions from Kawasaki disease can manifest as chest pain in young adults.
Allison Valerius, MD	*Atorvastatin-Induced Rhabdomyolysis in Nonalcoholic Cirrhotic Liver* Case Presentation: This case reviews and discusses a rare but severe complication of atorvastatin use in a 79-year-old female with a history of nonalcoholic liver cirrhosis. The patient was started on atorvastatin 40 mg and aspirin 81 mg daily for a presumptive transient ischemic attack four weeks before presenting to the emergency department with one week of generalized weakness. She was noted to be jaundiced. Laboratory testing found an elevated creatine kinase of 20350, bilirubin of 7.9, AST of 1717 (baseline 36), ALT 444 (baseline 17), prothrombin time 18.1 and a creatinine of 3.4 (baseline 0.96). She was diagnosed with statin-induced rhabdomyolysis, further complicated by an acute kidney injury, an acute liver injury, hypotension requiring two vasopressors and new onset atrial fibrillation. She responded well to extensive intravenous fluids and diuresis. A workup for other causes of acute liver injury was negative.

	Discussion: Identified risk factors of statin-induced rhabdomyolysis include age >65 years, twofold risk increase among females, engaging in strenuous exercise, pre-existing renal impairment, and combination of statin with a medication which inhibits cytochrome P450 3A4 enzymes. Although not established as a risk factor, liver disease is associated with decreased expression of SLC01B, the gene responsible for encoding the anion transporter that regulates statin absorption in the liver. Therefore, this patient's history of nonalcoholic liver cirrhosis may have increased her risk for statin-induced myopathy, resulting in rhabdomyolysis. A review on the pharmacokinetics and safety profiles of statins in patients with cirrhosis found that atorvastatin showed pronounced pharmacokinetic changes in patients with cirrhosis. Maximum plasma concentration of atorvastatin in patients with Child-Pugh class B score had a 16-fold increase in plasma concentration. These findings are concerning for an increased risk of statin side effects in patients with cirrhosis. Conclusion: This case highlights the importance of assessing statin complication risk factors prior to prescribing the medication as well as the importance of patient education regarding potential side effects.
Aurelio Vargas, MD Dr. Dongni Yi Dr. Sunny Chen Dr. Cordelia McGehee	*A Breach in the Wall: Risk and Remedy in Birt-Hogg-Dube-Associated Pneumothorax* Case Presentation: A 42-year-old female with a history of Birt-Hogg-Dube syndrome and ongoing tobacco use presented with a significant left-sided pneumothorax. Following successful thoracostomy tube insertion, her lung achieved full re-expansion. She remained hemodynamically stable and the tube was removed without complications after several days. For pain management, an unsuccessful trial of intrapleural lidocaine was initially attempted, before resorting to a PCA pump, which was later discontinued. Given her heightened risk for recurrent pneumothorax, thoracic surgery consultation advised an outpatient follow-up to consider elective pleurodesis. She was discharged in a stable condition with a scheduled follow-up with the thoracic surgery team. Unfortunately, a later outpatient chest X-ray disclosed a recurrent pneumothorax, prompting an emergency department visit for another thoracostomy tube placement. Ultimately, she underwent uncomplicated Video-Assisted Thoracoscopic Surgery (VATS) for a wedge resection and mechanical pleurodesis, and was discharged with successfully re-expanded lungs and plans for close monitoring by her primary care physician. Discussion: Patients with Birt-Hogg-Dube syndrome (BHD) have a higher propensity for developing spontaneous pneumothorax due to the cystic changes in the lung parenchyma that this syndrome engenders. The syndrome is an autosomal dominant disorder characterized by skin fibrofolliculomas, renal tumors, and multiple lung cysts. Lung cysts are commonly seen in adulthood, particularly between the ages of 40 and 50, and tend to be fragile, putting these patients at increased risk for pneumothorax. Compared to the general population, patients with Birt-Hogg-Dube (BHD) syndrome face a risk of pneumothorax that is approximately 50 times higher, with recurrence rates between 75% and 82% and an average of 3.6 episodes(1). This elevated risk requires a more proactive management strategy to minimize recurrences. Pleurodesis is a procedure that creates adhesions between the visceral and parietal pleura to prevent recurrent pneumothorax. Chemical pleurodesis

	using agents like talc is often employed, but given this patient's history, a surgical approach involving VATS (Video-Assisted Thoracoscopic Surgery) wedge resection followed by pleurodesis was conducted. This is in alignment with guidelines that recommend surgical intervention for recurrent pneumothorax, especially when underlying lung disease is present, to achieve better outcomes and lower recurrence rates. In summary, given this patient's underlying conditions and propensity for recurrent pneumothorax, the choice to proceed with surgical pleurodesis appears well-founded. The case underscores the importance of proactive and multidisciplinary management in patients with Birt-Hogg-Dube syndrome who are at high risk for recurrent pneumothoraces
Charles Warner, MD Dr. Amy Holbrook Dr. Victor Cheng	*SCAD, No Longer Just the Young Person's Disease* Introduction: Spontaneous Coronary Artery Dissection (SCAD) is a condition characterized by sudden onset of acute coronary syndrome with resulting myocardial injury related to the dissection of a major coronary artery, classically thought of as disproportionately affecting younger females. While the disease process is fairly well defined, the frequency is increasing, especially in older and post-menopausal women. A 62-year-old female with past medical history of hypertension, hyperlipidemia, type 2 diabetes presented to the ER with substernal chest pain after playing with her grandson that radiated to the arms and jaw. Her ECG was normal, and troponin was initially mildly elevated, though it plateaued and trended down. Due to concern for acute coronary syndrome, a heparin and nitroglycerin drip were started, and she was transferred for further workup. Chest pain continued overnight requiring aggressive titration of the nitro drip, prompting CT coronary artery scan. CT showed severe stenosis of the mid and distal LAD with a calcium score of 0, a pattern most suggestive of SCAD. Given the severe hypoperfusion of the LAD territory, and sudden rise in troponin, she was taken for invasive coronary angiogram which confirmed the CT findings, and reaffirmed the lack of CAD in the other coronary arteries. Percutaneous coronary intervention was considered, however the risk of propagating the dissection prompted consultation with CV surgery to discuss bypass grafting. Due to unclear suitability of the distal LAD for a graft anastomosis, CV surgery deferred grafting in favor of medical management. Due to known association, CTA of the head and neck was recommended for fibromuscular dysplasia (FMD) evaluation, notable for findings consistent with FMD in the carotid arteries, prompting vascular medicine consult. CTA of the chest, abdomen, and pelvis was without FMD in any other major vessel. Echocardiogram noted a hypokinetic septum and akinetic apex, with normal appearing EF. Cardiac MRI showed significant edema to the affected LAD territory, in addition to a 6 mm mural thrombus in the left ventricle. She was placed on aggressive GDMT and anticoagulation and discharged from the hospital after 6 days without evidence of further dissection propagation with planned follow up to cardiology and vascular medicine. This case illustrates a more classic presentation of SCAD confirmed by multimodal cardiac imaging in a less classic patient demographic. While CT imaging was useful in this case, it is not the recommended first line imaging modality for suspected SCAD diagnosis. From an etiology standpoint, even though exertion such as lifting a grandchild can trigger SCAD, this specific etiology tends to be more common in males, and the FMD seen here is the

	much more likely contributor. Keeping SCAD on the differential even in an uncommon patient demographic can be vital for the timely initiation of appropriate treatment.
Leah Williams, MD Dr. Anya Jamrozy	*The Scintillating Sensory Symptoms of Superior Semicircular Canal Dehiscence* Introduction: Superior semicircular canal dehiscence (SSCD) is a rare condition of the inner ear that results in significant auditory and vestibular symptoms. The syndrome is caused by a bony defect of the superior semicircular canal, which creates a third window between the canal and middle cranial fossa. This allows for sound waves traveling through the cochlea to be transmitted to the labyrinthine system, which can cause hearing loss and cause minor sounds to be perceived as vertigo and disorienting vibrations by the brain. These symptoms can be very debilitating and are generally only resolved with surgical repair. Case Presentation: A 51-year-old man with a history of hyperlipidemia, hypothyroidism, and obstructive sleep apnea presented to his primary care provider with symptoms including loss of equilibrium, head pressure, frequent morning emesis, and unusual auditory sensations. He described reverberations in the right side of his head when speaking or with brisk walking. Physical exam was unremarkable with normal ear pinnae, patent ear canals, and clear tympanic membranes. Rinne testing was inconclusive. Brain MRI was ordered, but patient never completed this due to cost concerns. He was evaluated by audiology and otolaryngology 4 months later. At that time, patient described worsening right ear reverberations, sometimes even causing vibrations in his vision, especially when driving over bumps or hearing his own voice. The otolaryngology provider did consider the possibility of SSCD and offered a CT, but patient did not get this completed. When evaluated by his PCP six months later, he decided to go forward with the original brain MRI, which was unremarkable and gave no explanation for his symptoms. After many more months lost to follow up, he subsequently completed the recommended CT scan, which revealed right superior semicircular canal dehiscence. Three years after the onset of symptoms, patient finally underwent surgical repair involving right middle fossa craniotomy with plugging and overlying grafting of the dehiscence. Symptoms immediately improved with only a residual low hum remaining in the right ear. The patient's postoperative course was complicated by poor wound healing at the right temple due to pseudofolliculitis. Repair of this defect dehisced after the patient's cat scratched the wound. The patient underwent two additional surgeries to attempt reclosure. Ultimately due to recurrent infection and concerns for osteomyelitis, removal of the hardware was recommended. Conclusion: This case illustrates one of the rarer disorders to consider when patients present with unusual auditory or vestibular symptoms without any notable findings on physical exam. High resolution CT imaging of the temporal bones is key to diagnosing this condition, as MRI can miss the subtle pathognomonic bony abnormalities. This case also highlights the potential complications that can result from the surgical repair, including hardware infection and wound dehiscence. While SSCD affects only 2% of the general population, knowing the unique presenting symptoms can prompt providers to order the most sensitive imaging test, expediting accurate diagnosis.

Alexander Xiao, MD Dr. Gordon Ruan Dr. Rachel Suen Dr. Kebede Begna	*A Vexing Case* Introduction: VEXAS syndrome is characterized by inflammatory and hematologic manifestations resulting from mutations in the UBA1 gene. Its recent description has shed light on the connection between seemingly unrelated adult-onset inflammatory syndromes. Better understanding of VEXAS is important as its prevalence and mortality are higher than previously thought, with multiple therapies under investigation. Case Presentation: A 68-year-old man with a history of recurrent fever, maculopapular rash, inflammatory eye disease, ear and nose chondritis, wrist pain, periorbital edema, right-sided pleural effusion, macrocytic anemia, and thrombocytopenia is admitted for worsening fevers, rash and fatigue. He also has a medical history notable for sensorineural hearing loss, splenomegaly, neutrophilic dermatosis and granulomatous vasculitis. This constellation of symptoms began in 2011, at the age of 57. At that time, a diagnosis of presumed atypical Cogan's syndrome was made, and subsequent disease exacerbations were treated with prednisone. Review of prior records notably reveal a 2015 bone marrow examination with hypercellularity and vacuoles in myeloid and erythroid precursors. With these findings and patterns within his inflammatory syndrome, suspicion was raised for VEXAS syndrome. Genetic testing for the UBA1 gene mutation, pathognomonic for VEXAS syndrome, returned positive and hematopathology re-interpretation of his 2015 slides was consistent with VEXAS syndrome. He was diagnosed with VEXAS syndrome, treated again with good response to steroids and followed closely thereafter with Rheumatology and Hematology for consideration of additional therapies and clinical trials. Discussion: VEXAS syndrome was first described in 2020. Recent research has found its prevalence be as high as 1 in 4000 men. VEXAS syndrome is characterized by the presence of vacuoles in bone marrow cells and results from mutations in the UBA1 gene. UBA1 is located on the X chromosome, and mutations are somatically acquired. Thus, VEXAS syndrome predominantly affects men aged 40 and older. Patients with VEXAS syndrome can present with a broad spectrum of inflammatory symptoms, including fever, periorbital edema, vasculitis, painful rashes, inflammatory arthritis, inflammatory eye disease, myocarditis and pleural effusions. Hematologic abnormalities are common and may include cytopenias, multiple myeloma, splenomegaly and DVTs. VEXAS syndrome is often also associated with relapsing polychondritis, polyarteritis nodosa, sweet syndrome, and myelodysplastic syndrome. The mortality rate remains high, with roughly half of patients passing within 5 years of diagnosis. Treatment primarily involves the use of steroids, but additional therapies are actively being investigated.
Yuan Yao, MD Dr. Binav Baral Dr. Lynn Cornell Dr. Silvia Titan	*Low M-Spike, High Stakes: Identifying Monoclonal Gammopathy of Renal Significance as a Serious Renal Threat* Introduction: Monoclonal gammopathy of renal significance (MGRS), a term coined in 2012 (1), describes a spectrum of kidney diseases induced by monoclonal immunoglobulin. It applies to patients who fulfill the diagnostic criteria of monoclonal gammopathy of undetermined significance (MGUS) but also exhibit kidney damage due to monoclonal proteins. Here, we present a case of MGRS managed successfully through multidisciplinary

	collaboration, bringing attention to the importance of prompt recognition of this condition. Case presentation: A 67-year-old male with past medical history of atrial fibrillation, hypertension, prediabetes and CKD3a presented with acute kidney injury and fluid overload, initially attributed to acute decompensated heart failure and cardiorenal syndrome. Despite aggressive diuresis to euvolemia, creatinine levels failed to improve (baseline 1.6 mg/dL, peak 2.5 mg/dL). Urine analysis showed >100 RBC/hpf, and 24-hour protein was 4.7 g. Kidney ultrasound showed changes consistent with chronic parenchymal disease. Further workup revealed SPEP with small monoclonal M-spike (IgG lambda) of 0.7 g/dL, urine M-spike of 407 mg and remaining glomerulonephritis workup was unremarkable. A kidney biopsy showed results consistent with MGRS pathology, specifically the subtype - proliferative glomerulonephritis with monoclonal immunoglobulin deposits (PGNMID). Further tests, including a bone marrow biopsy showing 5% plasma cells, ruled out multiple myeloma and other lymphoproliferative diseases, and the patient was diagnosed with MGRS/PGNMID. To prevent further immunoglobulin deposition and preserve renal function, he was initiated on chemotherapy with cyclophosphamide, bortezomib and dexamethasone. After 6 cycles, his creatinine improved to 1.2 mg/dL and 24-hour urine protein decreased to 0.3 g. Discussion and conclusion: MGRS refers to a group of renal diseases caused by monoclonal immunoglobulin, when the monoclonal disease burden is not enough to fulfil criteria for hematological malignancies such as multiple myeloma or Waldenstrom macroglobulinemia. Both MGUS and CKD are common, affecting up to 5.8% of adults over the age of 50 and up to 14% of US population, respectively (2, 3), a fact that imposes diagnostic challenges. MGRS can reside at the intersection of these two prevalent conditions and poses a risk for progressive renal damage, potentially leading to ESRD. It is crucial that the medical community can distinguish between MGUS patients (therefore without kidney damage) or MGUS in the setting of CKD of other etiology from, MGRS patients at risk for renal function decline, since risk of CKD progression and treatment options are very different in these two groups. Despite a low M-spike level, as observed in our patient with a 0.7 g/dL reading, MGRS can cause significant renal injury, manifesting acutely, subacutely, or chronically. Internists must remain vigilant, especially when renal impairment seems disproportionate to the severity of background diseases. Diagnosis is confirmed through kidney biopsy, and effective management typically involves chemotherapy. Early recognition by internists, along with a multidisciplinary approach involving internal medicine, nephrology and hematology, is vital for successful outcomes.