American College of Physicians- Minnesota Chapter Annual Abstract Competition
Poster Session
October 24, 2025
Abstracts Submitted for Competition

Medical Students	
Research - Medical Students	
Rebecca Heinze Yvonne Larson-Smith Tage Runkle Emma Behnken Dr. Gen Shinozaki Dr. Allyson Palmer	*Delirium Detection Using Point-of-Care Single-Channel EEG: A Pilot Study* Objective: Delirium is a common yet underdiagnosed medical emergency involving acute deficits in cognition and attention. It affects over 20% of hospitalized patients and up to 80% of ICU patients, with increased prevalence in older adults. Delirium is associated with increased mortality, prolonged hospitalization, long-term cognitive decline, and substantial healthcare costs. However, current screening tools are subjective and difficult to implement consistently. Traditional EEGs show diagnostic utility; however, they are costly and require specialized interpretation. Methods: We conducted a prospective pilot study evaluating a point-of-care, single-channel EEG device for delirium detection in hospitalized older adults. EEG spectra were utilized to calculate a 'bispectral EEG' (BSEEG) score reflecting the ratio of slow to fast brain waves. Twenty-two postoperative patients (mean age 75; 59.09% female) were assessed daily for up to seven days using both the BSEEG and the gold standard 3-Minute Diagnostic Interview for Confusion Assessment Method (3D-CAM). ROC analysis was utilized to determine an optimal threshold BSEEG score to indicate delirium positivity. Results: BSEEG scores were significantly higher in the 3D-CAM-positive group than in the 3D-CAM-negative group (1.60 and 1.45, respectively; $p < 0.05$). A BSEEG threshold of 1.5 demonstrated 77.78% sensitivity and 56.10% specificity for delirium. In contrast, routine clinical screenings (Delirium Triage Screen and Brief CAM) performed within 4 hours of 3D-CAM assessment showed 0.00% sensitivity and 96.30% specificity, failing to detect all true delirium cases. Conclusion: Although further validation in larger cohorts is needed, these findings suggest that the point-of-care single-channel EEG is a

	promising, non-invasive screening tool for delirium screening, particularly in fast-paced clinical settings.
Ronin Joshua Cosiquien Sai Anoop Dhamera Isaiah J. Whalen Pauline M. Cao Phillip Wong Dr. Ryan J Sorensen Dr. Anala V Shetty Dr. Shun-Qing Liang Dr. Clifford J. Steer	*Detecting Methylation Changes Induced by Prime Editing* Background: While prime editing offers improved precision compared to traditional CRISPR-Cas9 systems, concerns remain regarding potential off-target effects, including epigenetic changes such as DNA methylation. In this study, we investigated whether prime editing induces aberrant CpG methylation patterns. Methods: Whole-genome bisulfite sequencing revealed overall methylation similarity between Cas9-edited, and PE2-edited cells. However, localized epigenetic changes were observed, particularly in CpG islands and exon regions. The PE2-edited group showed a higher proportion of differentially methylated regions (DMRs) in some coding sequences compared to controls and Cas9-edited samples. Notably, CpG island methylation reached 0.18% in the PE2 vs. Cas9 comparison, indicating a higher susceptibility of these regulatory elements to epigenetic alterations by prime editing. Molecular function analyses including Gene Ontology and KEGG pathway analyses further revealed enrichment in molecular functions related to transcriptional regulation and redox activity in PE2-edited cells. Conclusion: These findings suggest that prime editing, while precise, may introduce subtle but functionally relevant methylation changes that could influence gene expression and cellular pathways. In summary, prime editing can induce localized DNA methylation changes in human cells, particularly within regulatory and coding regions. Understanding these epigenetic consequences is critical for the development of safer and more effective therapeutic applications of genome editing technologies.
Mahima Devarajan Rachel Meyer Gavin Fredrickson Dr. Douglas Mashek	*Fasting as a Chemotherapy Adjuvant: Mobilizing Lipids to Enhance Genomic Repair* Background: The integrity of our genome is dependent on a delicate balance between DNA damage and DNA repair. An imbalance of DNA damage over DNA repair contributes to the genomic instability that drives malignant transformation and aging. In support of the essential role of DNA repair in maintaining cellular and organismal health, mutations in DNA repair proteins result in early cancers and accelerated aging. In contrast, fasting and caloric restriction are arguably the most well-established interventions that extend lifespan and reduce cancer burden across organisms, a mechanism that is supported by evidence that fasting can enhance genomic stability. In a fasting state, activation of lipolysis leads to the breakdown of lipid droplets (LDs), and upregulation of lipolytic enzymes such as adipose triglyceride lipase (ATGL) that serve as a source of energy as well as contribute to multiple signaling cascades in the cell. Given the contributory role of LDs and ATGL in the fasting response and the

	link between fasting and genomic stability, our objective is to elucidate the mechanisms by which LD metabolism regulates DNA homeostasis. Methods: We show that LD ablation prior to DNA damage increases DNA damage, suggesting a protective role for LDs in the context of genotoxic stress. Importantly, we show that overexpression of ATGL (thus increasing lipolysis) reduces DNA damage, reduces the DNA damage response, and enhances DNA repair in response to both etoposide and ionizing radiation, suggesting that the protective role LDs play is due to enhanced lipolysis. These findings are recapitulated in mice; mice overexpressing ATGL are better able to resolve DNA damage after irradiation-induced DNA damage. Further, fasting – a process during which ATGL and lipolysis are upregulated – is protective when mice are given lethal doses of chemotherapy, allowing fasted mice to survive weeks longer than their fully fed counterparts. Results: Finally, our data indicate that pharmacological upregulation of lipolysis is able to enhance DNA repair, providing both proof of concept for the ATGL studies and an exciting potential intervention for populations undergoing large amounts of DNA damage (i.e., patients undergoing chemotherapy). Conclusion: Overall, these studies reveal a novel role for lipid metabolism in DNA damage and repair, thus unveiling a novel therapeutic target for chemotherapeutic management.
Peter Salama Conner Olson Dr. Scott Mikesell	*Trajectory of Left Ventricular Mass and Flow Patterns in Patients Undergoing Aortic Valve Replacement* Objective: To compare longitudinal changes in left ventricular (LV) remodeling and cardiac flow dynamics following surgical aortic valve replacement (SAVR) versus transcatheter aortic valve replacement (TAVR) in patients with aortic stenosis. Methods: A retrospective cohort study of 612 patients (312 TAVR, 300 SAVR) was conducted from October 2019 to December 2023. Echocardiographic measurements including stroke volume (SV), SV index (SVI), dimensionless index (DI), LV mass, LV mass index (LVMI), and LV ejection fraction (LVEF) were analyzed at pre-procedure, 30 days, 1 year, and ≥2 years post-procedure. Generalized linear mixed models were used to assess time-based changes and differences between procedure types, with controlling for body surface area (BSA), history of coronary artery bypass grafting (CABG), percutaneous coronary intervention (PCI), and chronic kidney disease (CKD). Results: Both TAVR and SAVR groups showed significant improvements in SV, SVI, DI, LVmass, LVMI, and LVEF over time (all p < 0.05). TAVR was associated with greater increases in SV and SVI and more rapid LV mass regression compared to SAVR. LVEF improved in both groups, though SAVR patients had a relative

	decline over time. CABG history and higher BSA correlated with improved SV, while CKD was associated with less favorable remodeling. Conclusion: TAVR is associated with more favorable and sustained LV remodeling compared to SAVR, likely due to reduced myocardial injury. These findings highlight the importance of patient-specific factors, including comorbidities and body size, in procedural selection.
Clinical Vignette - Medical Students	
Lucy Greenhagen Roon Mahboub Whitney Wenner Ravikanth Nathani Dr. Ramakanth Pata Dr. Mithun Suresh	*Immunotherapy Induced New-Onset Type 1 Diabetes Mellitus and Diabetic Ketoacidosis* Background: Immunotherapy has revolutionized cancer treatment by utilizing the patient's own immune system to identify and target tumor cells. Immune checkpoint inhibitors (ICI) are T cell targeting immunomodulators which restore the ability of T cells to recognize and respond to tumor cells. Without modulation, immune checkpoints suppress T cell activity, preventing the immune system from responding to tumor cells. ICI activate T cells to recognize and destroy tumor cells. Although immunotherapy frequently offers a more efficacious and less toxic option compared to other treatments, it has its own unique set of adverse events. New-onset type 1 diabetes mellitus (T1D) is an uncommon but documented adverse event to ICI. While the exact mechanism is unknown, it is theorized that ICI may contribute to the activation of autoreactive T cells that destroy the insulin producing beta cells in the pancreas, leading to new onset T1D which often presents as diabetic ketoacidosis (DKA), a life-threatening metabolic emergency. Its rarity presents a challenge for internists, who may be the first to evaluate these patients, making recognition of immunotherapy related complications valuable. Case Presentation: A 55-year-old female presented with nausea and vomiting. Her medical history was most notable for metastatic right renal clear cell carcinoma which was being treated with ipilimumab and nivolumab. She was tachycardic, appeared fatigued and her breath had a fruity odor. Initial labs revealed the following: blood pH 6.98, lactate 4.1 mmol/L, bicarbonate 7 mmol/L, anion gap 29.0 mmol/L, creatinine 1.72, sodium 126 mmol/L, glucose 563 mg/dL, WBC 17.6 thousand per uL, urine ketones were positive, phosphorus 1.9 mg/dL, beta-hydroxybutyrate 7.8. mmol/L, hemoglobin A1c 6.9 mmol/L. Following the diagnosis of DKA, she was treated with liberal IV fluid resuscitation, an insulin drip, and serial electrolytes with concurrent replacement. With the management plan as described, the patient progressively improved over several days. Upon discharge, all serum abnormalities and symptoms had resolved. Given the mechanism of action of immunotherapy, it was felt that the diagnosis of new-onset T1D may have been attributed to the use ipilimumab and nivolumab and that this diagnosis was permanent, given the mechanism by which it occurred.

	Conclusions: This case illustrates a life altering and uncommon adverse event to immunotherapy. New-onset T1D is reported in less than 1% of ICI therapies. Patients utilizing dual ICI therapies involving the use of both an anti-CTLA-4, such as ipilimumab, and an anti-PD-1/anti-PDL-1, such as nivolumab, are at an increased risk of T1D, though the overall risk remains markedly low. Considering the expanding role of immunotherapy in cancer treatment, recognition of ICI as the potential cause of hyperglycemia or DKA is critical when considering differential diagnoses for patients presenting with metabolic emergencies.
Allison Hanka Paige Stueve Dr. John Meisenheimer VII Joseph McGrath Dr. Noah Goldfarb	*Metastatic Lung Carcinoma Mimicking a Perianal Abscess and Fistula* Learning objectives: Review the differential diagnosis of conditions that present with perianal fistula. Recognize atypical signs of perianal abscess and fistula, which may warrant a biopsy. Case Presentation: A 75-year-old male with a history of non-small cell lung cancer of the right lung with multiple presumed local lung metastases status post stereotactic body radiation therapy, clear cell renal cell carcinoma status post nephrectomy and squamous cell carcinoma of the vallecula status post radiation therapy developed perianal pain and drainage during hospitalization for complicated urinary tract infection. Initial examination with colorectal surgery revealed an open perianal wound along the posterior aspect of the perineal region with minimal purulent drainage. No erythema, induration, fluctuance or palpable fistulous tract was noted at that time. A diagnosis of a perianal abscess was made. Over the following three months, the wound did not heal and a fistulous tract extending back to the scrotum developed. CT Pelvis demonstrated a 10.8x7.3cm thick-walled fistula extending into the left hemiscrotum. Rectal thickening was noted, suspicious for perianal fistula. Examination under anesthesia was performed and a seton was placed. A subsequent perineal CT-guided tissue biopsy was done and was consistent with metastatic pulmonary adenocarcinoma. A decision for palliative care was made due to the advanced progression of several carcinomas. Discussion: Perianal fistulas are most commonly the result of a perianal abscess with differentials including Crohn's disease, hidradenitis suppurativa (HS), pilonidal disease, diverticulitis, tuberculosis, and malignancy (Sohrabi et al., 2024). We present a case describing a patient with a perianal fistula caused by malignancy to highlight warning signs that might warrant a biopsy or other workup for underlying cancer. While malignancy is considered a rare cause of perianal fistulas, missed diagnosis can result in life-threatening complications from delayed cancer treatment. Personal history of malignancies, particularly of the pelvic organs, or indurated, bound

	down soft tissue masses in the perianal and pelvic region should raise suspicion of tissue infiltration by malignant carcinomas. Clinicians should look out for red flag symptoms, such as rapid unintentional weight loss, drenching night sweats, intermittent fevers, and pelvic lymphadenopathy, which may raise suspicion for cancer. Certain conditions like HIV increase risk for anal cancer, and should be screened for (Albuquerque et al., 2023). As always, when malignancy is on the differential, it is important to determine whether the patient is up to date on age-appropriate cancer screening. Conclusion: In patients with perianal fistula, clinicians should keep malignancy on the differential and consider a biopsy if the fistula does not respond to conventional treatment, particularly in patients with a history of malignancy. References: Sohrabi M, Bahrami S, Mosalli M, et al. Perianal Fistula; from Etiology to Treatment - A Review. Middle East J Dig Dis. 2024;16(2):76-85. doi:10.34172/mejdd.2024.373 Albuquerque A, Cappello C, Stirrup O, Selinger CP. Anal High-risk Human Papillomavirus Infection, Squamous Intraepithelial Lesions, and Anal Cancer in Patients with Inflammatory Bowel Disease: A Systematic Review and Meta-analysis. J Crohns Colitis. 2023;17(8):1228-1234. doi:10.1093/ecco-jcc/jjad045
Pareekshith Hirenallur Lohithaswa Dr. Samir Parekh Dr. Ram Subramanian Dr. Steven Keilin Dr. Sailaja Pisipati	*Post-Transplant Kaposi Sarcoma Presenting as Obstructive Hepatopathy with KSHV Inflammatory Cytokine Syndrome* Background: Kaposi Sarcoma (KS) is a vascular malignancy driven by Human Herpes Virus 8 (HHV-8), commonly occurring in immunocompromised states. Hepatic involvement in post-liver transplant recipients is rare and poorly characterized. An even rarer complication is Kaposi Sarcoma Herpesvirus Inflammatory Cytokine Syndrome (KICS), a systemic hyperinflammatory condition with high mortality rates. Case Presentation: A 59-year-old Caucasian woman developed progressive cholestatic liver dysfunction six months following orthotopic liver transplantation for primary biliary cirrhosis. She presented with malaise, fatigue, and mild icterus. Laboratory investigations revealed alkaline phosphatase 482 IU/L, ALT 104 IU/L, AST 122 IU/L, and total bilirubin 0.7 mg/dL. Magnetic resonance imaging demonstrated a soft tissue mass at the hepatic hilum encasing biliary and vascular structures. Endoscopic ultrasound-guided fine needle aspiration revealed spindle cells positive for ERG and HHV-8, confirming Kaposi Sarcoma. Endoscopic retrograde cholangiopancreatography identified biliary strictures requiring stenting.

	Her clinical course rapidly deteriorated with multidrug-resistant Pseudomonas aeruginosa and Klebsiella pneumoniae bacteremia, causing septic encephalopathy and acute kidney injury requiring inotropic support, mechanical ventilation, and continuous renal replacement therapy. Despite antimicrobial therapy, she experienced refractory shock and persistent inflammation. Quantitative PCR revealed a high HHV-8 viral load. Immunosuppression was modified from tacrolimus to sirolimus with dose reduction. Liver function deteriorated dramatically with total bilirubin exceeding 35 mg/dL, alkaline phosphatase 1477 IU/L, ALT 2377 IU/L, and AST 9206 IU/L. The combination of high viral burden, persistent inflammation despite infection control, and multi-organ dysfunction suggested KICS. She was ultimately transitioned to comfort care. Discussion: This case illustrates the aggressive nature of post-transplant hepatic KS and its potential evolution into KICS. The hilar presentation causing obstructive hepatopathy created diagnostic challenges and severely limited therapeutic options. Standard chemotherapy with PEGylated liposomal doxorubicin was contraindicated due to ongoing sepsis and hepatic impairment. Rituximab therapy for KICS was precluded by infection risk and potential KS flare. The pathophysiology of KICS involves cytokine dysregulation with elevated interleukin-6 and interleukin-10 levels, creating a hyperinflammatory state that can precipitate multi-organ failure. Management strategies remain limited, particularly when complicated by concurrent infections that hinder immunomodulatory interventions. Conclusions: Clinicians should maintain high suspicion for KS and KICS in post-transplant patients presenting with unexplained systemic inflammation and hepatic dysfunction. Early recognition and development of alternative therapeutic strategies are crucial for improving outcomes in this challenging clinical scenario.
Tasneem Issa Andrea Davis Dr. Ryan Kelly	*Viral Meningitis from West Nile Virus: A Clinical Case Report* Background: West Nile virus (WNV) is a positive-sense, single-stranded RNA flavivirus. It is transmitted through mosquito bites and most people are asymptomatic when infected with the virus. About 1 in 5 develop symptoms including fever, rash and muscle aches. Most cases are associated with mosquito exposure during summer months as well as traveling and participating in outdoor activities. In rare cases, WNV can cause serious cases of meningitis, encephalitis, acute flaccid paralysis, seizures and even coma. We present a case of viral meningitis caused by WNV in a young adult with no recent travel history or underlying medical condition. Case Presentation: A 27-year-old previously healthy male presented to the clinic with fever, progressive headache, vomiting, photophobia, and nausea. He denied recent travel, but had exposure to mosquitoes with evidence of bites on right upper extremity and back at St. Croix

	River Valley in Minnesota. Neurological exam revealed nuchal rigidity and cervical tenderness. Bacterial cultures were negative. The CSF viral PCR panel was negative for common pathogens such as H. influenzae, N. meningitis, E. coli K1, Listeria monocytogenes, and Strep pneumoniae. CSF protein level was elevated and serology was positive for West Nile virus IgG. The patient received supportive care with intravenous fluids and analgesics, and his symptoms improved over the following week. Discussion: Although West Nile Virus is known to cause viral meningitis, it is uncommon and thus is rarely considered as one of the primary differentials by providers in the absence of travel or obvious exposure risk. This case underscores the importance of considering WNV in the differential diagnosis of viral meningitis, particularly during mosquito season in endemic regions. Conclusions: Clinicians should consider WNV meningitis in young patients with no recent travel history and minimal medical history. Early diagnosis allows for appropriate supportive management and public health reporting, as well as preventing delay in treatment.
Joseph Juliette Dr. Erica Levine	*G6PD Who? Hemolytic Anemia with Dapsone use without G6PD Deficiency* Introduction: Dapsone is an anti-microbial drug commonly used in immunocompromised patients for the prophylaxis of Pneumocystis jirovecii pneumonia (PJP). It is known to cause hemolytic anemia through the oxidation of iron elements of hemoglobin. Dapsone-induced hemolysis is most commonly found in patients with deficiency of G6PD, an enzyme with a vital role protecting red blood cells against oxidative stress. Case Presentation: A 76-year-old female presented with a 10-day history of profound fatigue, weakness, and intermittent hypotension. Her medical history was significant for hypertension, SLE, iron deficiency, chronic anemia, endometrial carcinoma (treated with TAH/BSO and radiation in 2023), MGUS, and interstitial lung disease. The patient started dapsone one month prior for prophylaxis of PJP per pulmonology recommendation due to steroid use. She was negative for G6PD deficiency. On admission her labs revealed low hemoglobin (7.6 g/dL; 10.8 g/dL two months prior), low haptoglobin (<10) with elevated reticulocytes (9.04%), absolute reticulocytes (223,300/uL), and LDH (351), concerning for acute on chronic anemia secondary to acute hemolysis. The patient was never hypoxemic. Despite her negative screen for G6PD deficiency, dapsone was held out of concern for hemolysis. The peripheral blood smear was consistent with normocytic anemia. Hematology recommended a Heinz body study be performed, which showed elevated Heinz body formation with induction, supporting a hemolytic process. Given her history of SLE, a secondary autoimmune hemolytic anemia was

	considered. However, the patient was found to have negative DAT, which had previously been found to be positive during previous flares, thus lowering suspicion. While traditionally thought to affect patients with G6PD deficiencies, dapsone was the only new medication started within the timeframe of the new onset of symptoms and anemia, increasing suspicions that her presentation was due to dapsone-induced hemolysis. The patient's hemoglobin dropped as low as 6.5 g/dL, before stabilizing again soon after discontinuation of dapsone at ~7.5-8 g/dL. She was discharged at this level, with repeat hemoglobin testing one week after measuring at 9.8 g/dL. Internal medicine, rheumatology, and hematology agreed to start the patient on pentamidine as replacement prophylaxis for PJP, which the patient began during admission with no adverse effects. Discussion: While it is still possible the patient's acute anemia had another cause, it is most consistent with dapsone-induced hemolytic anemia. While primarily associated with G6PD deficiency, dapsone has been found to induce hemolysis in a variety of immunocompromised populations. A recent report found the development of dapsone-induced methemoglobinemia in addition to hemolysis in a patient with SLE as well. Given the possible contribution of an immunocompromised state to the hemolysis effect of dapsone with or without G6PD deficiency there is reason to monitor for and counsel patients on signs of the development of acute anemia in all patients taking dapsone, even in the setting of a normal G6PD enzyme test.
Olivia Karanja Dr. Rose Baumann Dr. Jonathan Kirsch Dr. Haitham Hussein	*Hope for Hypertension: A Case from a Mobile Health and Community Collaboration in an Underserved Population* Background: Hypertension is a significant risk factor for many cardiovascular diseases. The burden of cardiovascular diseases is unequally distributed among different populations, with a higher prevalence in African-American patients. Poor access to healthcare has been identified as a key factor in poorly controlled blood pressure. For several years, our Mobile Health Initiative (MHI) has partnered with multiple community organizations in planning free community health events where participants are screened for chronic conditions such as hypertension and diabetes. To maximize the benefit from and ensure continuity of care after these events, we are currently conducting a community participatory research study, titled Hypertension Identification using mobile Health Outreach ProjEct (HI HOPE), in collaboration with a community organization. In this study, we provide participants who have high blood pressure with a self-monitoring blood pressure (SMBP) device, hypertension education, and active facilitation of primary care follow-up. Here we present a case that illustrates the impact of HI HOPE on a participant who attended one of the community health events. Case Presentation: A 63-year-old African-American female with no known past medical history was found to have elevated blood pressure

	of 167/101 at the health event. She reported having no primary care doctor and expressed interest in receiving information for local clinics. She was eligible for the HI HOPE Study and was enrolled in the study after signing an informed consent. She was then provided with a free SMBP device along with a log to chart her readings. She also received education on blood pressure management and information for local clinics to establish primary care. During the follow-up calls, she reported use of her blood pressure cuff at least 3 times per week. She also stated that she has plans to make an appointment to establish primary care. Discussion: This case highlights the importance of community health events in screening for hypertension in underserved communities with limited healthcare access. The participant was unaware of her high blood pressure and was not connected to a primary care clinic for management. The health event enabled the participant to identify an unknown medical issue and, using the printed handouts, connect her to local clinics for long-term hypertension management. Additionally, incorporating the SMBP device provides an opportunity for the participants to become fully involved in their own health and gain a sense of autonomy in the management of their blood pressure. Beyond direct benefits for participants, the community events hosted by the mobile health initiative and the community organization offer broader advantages for the healthcare system. Conclusions: Overall, mobile health clinics demonstrate substantial cost savings, reduced emergency department utilization, and improved return on investment. By partnering with local organizations, mobile health clinics bring care directly to communities, which provides important public health benefits while helping reduce health disparities.
Roon Mahboub Whitney Wenner Lucy Greenhagen Ravikanth Nathani Dr. Ramakanth Pata Dr. Mithun Suresh	*Semaglutide Included Pancreatitis and Euglycemic Non-DKA* Case Presentation: A 36-year-old man with a past medical history of obesity presented to the ED with nausea, vomiting, and abdominal pain that started two days earlier. His only medication was semaglutide injections. He described his pain as sharp and located in the epigastric region of the abdomen, and upon examination, this area was tender. He was tachycardic and had a mildly elevated respiratory rate. Upon conducting laboratory tests, the notable results were elevated anion gap (36 mmol/L), elevated lipase (853 U/L), normal glucose (163 mg/dL), and positive urine ketone test. A CT of the abdomen and pelvis showed inflammation around the pancreas. While the results of the laboratory tests pointed to a diagnosis of diabetic ketoacidosis, the patient had no history of diabetes. Accordingly, this led to a diagnosis of EnDKA, and the imaging findings combined with the clinical history and elevated lipase led to the concurrent diagnosis of pancreatitis.

	Discussion: With increasing prevalence of obesity in the United States, GLP-1 agonist (semaglutide) injections, although initially used to treat diabetes, have become an increasingly popular way to treat obesity, with 12% of Americans self-reporting that they use a semaglutide injection to help manage their weight. Obesity is caused by a multitude of factors such as, lack of physical activity, unhealthy eating habits, lack of sleep, high stress, genetics, the environment where an individual resides, and a side effect of certain medications like antidepressants, antipsychotics, and beta-blockers, among others. Because obesity can cause adverse effects in afflicted individuals, the current treatment plan includes significant lifestyle changes, which includes weight loss, exercise, and a balanced diet or weight loss surgery. As a result, GLP-1 agonists are sought out due to their ability to treat obesity without requiring the patient to adhere to a strict lifestyle change. GLP-1, which stands for glucagon-like peptide-1, agonists bind to the GLP-1 receptors in the pancreas to induce insulin release into the body. This causes lower blood sugar, prevents glucagon release, slows digestion, and increases satiety. A side effect that is becoming notable is euglycemic non-diabetic ketoacidosis (EnDKA), which may occur if GLP-1 agonists overstimulate the pancreas and cause hyperplasia, which can lead to a decrease in serum insulin and an excess of glucagon. The glucagon excess causes lipolysis and increased ketone production, which promotes a decrease in pH and prompts an anion gap metabolic acidosis. This may be further exacerbated by nausea, vomiting, and poor appetite, which are frequently concurrent symptoms. Furthermore, because GLP-1 agonists can reduce appetite, lipolysis and ketone production can follow, and gluconeogenesis is slowed, this ultimately leads to EnDKA. Finally, from the FDA, there is a warning linking semaglutide to pancreatitis as a possible adverse event. Although the exact mechanism is still unknown, one possibility is related to pancreatic overstimulation, which is a similar proposed mechanism for EnDKA. Conclusion: With the increasing use of GLP-1 agonists, it is important for providers to be aware of potential side effects that may be related to their use.
Julia Meyer Dr. Sharon Li Dr. Alireza Nathani	*Return of Spontaneous Circulation After Cessation of Resuscitative Efforts: Electromechanical Dissociation as a Result of Intrinsic Positive End-Expiratory Pressure* Introduction: Intrinsic positive end-expiratory pressure (PEEP), or "autoPEEP", occurs due to incomplete exhalation and is common in mechanically ventilated patients with severe obstructive lung disease. We report a case of a patient with severe emphysema who suffered cardiac arrest due to autoPEEP and achieved return of spontaneous circulation (ROSC) after ventilatory efforts were halted.

	Case Presentation: A 75-year-old male with a history of metastatic lung cancer and chronic obstructive pulmonary disease (COPD) presented with increased work of breathing and fevers for three days. On examination, diffuse wheezing was observed. Initial laboratory workup revealed leukocytosis and acute respiratory acidosis (pH 7.27, PCO2 79, HCO3 36). RSV testing was positive, and chest radiograph showed bilateral airspace opacities consistent with RSV pneumonia and acute COPD exacerbation. Despite bilevel positive airway pressure (BiPAP) therapy, the patient remained hypoxic with oxygen saturations of 85-90%. He was admitted to the intensive care unit (ICU) and intubated for acute on chronic hypoxic hypercarbic respiratory failure. Shortly after intubation and initiation of mechanical ventilation, he became bradycardic and then pulseless. Pulseless electrical activity (PEA) arrest ensued, prompting advanced cardiac life support (ACLS). The patient was disconnected from the ventilator and ventilated manually with a bag-valve mask (BVM) at 8-10 breaths per minute using an expiratory PEEP valve and there was no difficulty with bagging. Despite 40 minutes of resuscitation, his rhythm deteriorated from PEA to asystole. A femoral arterial line showed no pulsatility after 50 minutes, and bedside ultrasound confirmed no cardiac activity. With family consent, resuscitative efforts ceased. After observing a moment of silence, cardiac monitoring was turned off, and the patient was disconnected from the BVM. However, during the formal death exam conducted within five minutes of cessation, a strong pulse and cardiac sounds were detected. The femoral arterial line displayed a mean arterial pressure of 40 mmHg. Mechanical ventilation was resumed, and fluids and vasopressors were initiated. His ICU course was complicated by severe respiratory acidosis and hemodynamic instability. He developed acute renal failure, and his family declined dialysis in accordance with his wishes. He expired ten days later. Discussion/Conclusions: Patients with severe airflow obstruction require adequate expiratory time to prevent hyperinflation and autoPEEP. During acute exacerbations, respiratory acidosis often necessitates increased minute ventilation. However, increasing the respiratory rate during mechanical ventilation or BVM ventilation shortens expiratory time, increasing the risk of autoPEEP. This elevated intrathoracic pressure impairs venous return, reduces cardiac output, and can ultimately lead to cardiac arrest. When positive pressure ventilation ceases, venous return may suddenly increase, sometimes leading to ROSC as seen with our patient. This case underscores autoPEEP as a reversible cause of cardiac arrest and highlights the importance of recognizing and managing this condition in patients with severe obstructive lung disease.
Passy Nabongho Dr. Meredith Kavalie Dr. Sashi Nair	*Shunt Sabotage; A rare case of M. Abscessus CNS Infection*

	Introduction: Mycobacterium Abscessus is a non-tuberculous mycobacterium found in soil and water, with evolving evidence suggesting it can colonize the GI tract. It is an emerging cause of community and hospital-acquired infections that has rarely been reported to cause central nervous system (CNS) infections related to VP shunts. We present a case of VP shunt–associated CNS infection due to M. Abscessus requiring shunt removal and prolonged antimicrobial therapy for infection control. Case Presentation: A 44-year-old male with a ventriculoperitoneal (VP) shunt placed in 2013 for post-traumatic hydrocephalus presented for an intraabdominal abscess near the peritoneal portion of the VP catheter while on antibiotic therapy. Initial cultures grew intraabdominal bacterial flora and he was treated with ceftriaxone and metronidazole. CSF analysis and cultures at the time of abscess development were not concerning for infection. A follow up CT scan of the abdomen and pelvis revealed an enlarging fluid collection in the abdomen, now involving the shunt catheter tip. His VP shunt was externalized, but not completely removed. Interventional Radiology placed an abdominal drain into milky white fluid. Acid fast bacilli (AFB) staining was positive, and at day 7 grew Mycobacterium Abscessus. Empiric therapy was initiated with amikacin, tigecycline, and azithromycin while awaiting susceptibilities. The patient tolerated therapy well until 7 days after initiation when he developed high-grade fevers, tachycardia, and headaches. Blood and urine cultures were negative, and respiratory panel testing along with chest and abdominal imaging revealed no fever source. VP shunt tap demonstrated new cerebrospinal fluid (CSF) findings: elevated nucleated cells (28/μL, 50% neutrophils), elevated protein (76.3 mg/dL), and normal glucose. The M Abscessus therapy was adjusted to account for CNS penetration and susceptibility results to high-dose cefoxitin, tigecycline, and azithromycin. Tigecycline was later discontinued due to intractable GI symptoms. Persistent high-grade fevers prompted repeat CSF studies that showed positive AFB stain, culture, and 16S ribosomal RNA, confirming VP shunt–related M. Abscessus CNS infection. The VP shunt was explanted, and an external ventricular drain (EVD) was placed. Therapy was continued with intrathecal amikacin in addition to systemic amikacin, high-dose cefoxitin, azithromycin, and linezolid, with a planned duration of 12 months. The EVD was subsequently removed due to low output, which he tolerated well. Repeat CSF cultures did not clear until 15 days after shunt explantation. At last follow up, the patient was afebrile and hemodynamically stable, without headaches, and continues to be hospitalized for management of other comorbidities with plans for at least 1 year of multidrug antibiotics. Discussion: VP shunt–associated CNS infections with Mycobacterium Abscessus, a slow-growing, multidrug-resistant nontuberculous mycobacterium are exceedingly rare and pose significant diagnostic and therapeutic challenges. The importance of early suspicion and intervention cannot be overstated. Management requires surgical

	intervention, typically shunt removal or externalization, and prolonged, combination antimicrobial therapy, including intrathecal agents. Conclusions: This case highlights the importance of considering NTM infections in patients with indwelling CSF devices who present with persistent fever with no alternative explanation. Timely source control (usually shunt removal) is essential to achieving infection eradication and favorable outcomes.
Peter Salama Kai Akimoto Dr. Pierre Tawfik	*Dupilumab Induced Acute Eosinophilic Pneumonia* Introduction: Acute Eosinophilic Pneumonia(AEP) consists of both pulmonary infiltrates and eosinophilia on bronchoalveolar lavage (BAL) within a time frame of less than one month(1). While it can be triggered by environmental exposures, medications, or infections, it is most often idiopathic(2). IL-4 inhibitors like Dupilumab are often used to treat eosinophilic asthma and has been linked with an eosinophilia surge which can rarely lead to intrapulmonary eosinophilic pathologies. This includes AEP, Eosinophilic Granulomatosis with Polyangiitis (EGPA), or Hypereosinophilic Syndrome (HES)(3). With this in mind, differentiating AEP from EGPA and HES leads to a diagnostic challenge. Here, we present the unique case of Dupilumab-Induced AEP. Case Presentation: A 47-year-old female with a four-year history of treatment resistant asthma, rhinosinusitis, and nasal polyps was started on dupilumab after failed treatments on budesonide, formoterol, and montelukast with intermittent usage of high dose corticosteroids for severe asthma attacks. On admission, labs were significant for normal eosinophil count of 400 cells/μL and negative MPO, PR3 and ANCA IgG. However, after dupilumab administration, she developed marked eosinophilia (3,400 cells/μL). A high-resolution CT scan showed upper-lobe predominant ground-glass opacities and consolidations. BAL revealed 31% eosinophils (516 total WBCs). Transbronchial biopsies showed mixed inflammation with lymphocytes and plasma cells in addition to eosinophils present in the airways. Transbronchial biopsies and nasal biopsies did not show vasculitis, but the sample quality of the former was limited. Conclusions: While eosinophilia is common is following dupilumab use, pathological manifestations are extremely rare. This case highlights this unique complication. The temporal association with dupilumab initiation strongly suggested medication induced AEP. By recognizing the signs and symptoms of this complication, clinicians must consider discontinuing this medication and offer an alternative treatment plan.

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Michael Tschida Dr. Morgan Kelly Dr. Krishnakumar Dr. Balakrishna Menon	*A Rare Culprit of Chronic Abdominal Pain: Idiopathic Myointimal Hyperplasia of the Mesenteric Veins Masquerading as Inflammatory Bowel Disease* Introduction: Idiopathic myointimal hyperplasia of the mesenteric veins (IMHMV) is an exceptionally rare, non-thrombotic, non-inflammatory veno-occlusive disease characterized by proliferation of smooth muscle cells within the intima of small-to-medium sized mesenteric veins, leading to progressive luminal narrowing and chronic intestinal ischemia. Patients typically present with abdominal pain and diarrhea, and the condition is frequently misdiagnosed as inflammatory bowel disease (IBD) in ~49% of cases due to overlapping clinical and endoscopic features. We report a case of IMHMV presenting as chronic abdominal pain, highlighting its potential to masquerade as IBD and need for histopathologic confirmation for definitive diagnosis. Case Presentation: A 45-year-old female with well-controlled HIV and a history of anorectal cancer status post chemoradiation, with recurrent hospitalizations for ileitis presented with acute-on-chronic abdominal pain. Her clinical symptoms and previous colonoscopy findings of non-specific active ileitis with ulceration, led to a presumed diagnosis of inflammatory bowel disease (IBD). Despite multiple courses of corticosteroids, her symptoms worsened. Magnetic resonance enterography demonstrated terminal ileum inflammation with narrowing with concerns for a stricture. She developed acute peritonitis and imaging demonstrating acute pneumoperitoneum, prompting emergent surgery. Intraoperatively, she was found to have a perforated ileum, ischemic cecum and extensive adhesions with a band near the terminal ileum. She underwent an ileocecectomy with end ileostomy creation. Histology of the ileum and right colon demonstrated mural fibrosis and idiopathic myointimal hyperplasia of the mesenteric veins (IMHMV). Vascular medicine recommended daily aspirin 81 mg. CTA of the head and neck excluded fibromuscular dysplasia. Her hospital course was complicated by sepsis, multiple subcapsular hepatic abscesses, and a bladder abscess. She was discharged with close vascular follow-up for her new diagnosis of IMHMV.

	Discussion: Idiopathic myointimal hyperplasia is a rare condition that leads to chronic colonic ischemia which is commonly mistaken for IBD due to overlapping features as seen in this case. The average time to diagnosis is four months, though it may be prolonged as pre-operative diagnosis remains challenging.1 Because radiographic and endoscopic findings are nonspecific, surgical histopathology samples are the most common method for definitive diagnosis.2,3 One notable feature of this case was ileal involvement, present in only 5% of IMHMV cases.1,2 While the ileum is often associated with Crohn's disease, the patient's refractory response to steroids, the standard of treatment for IBD, was an essential distinguishing factor.1 Delayed recognition can lead to life-threatening complications in 30% of IMHMV cases, such as bowel perforation and toxic megacolon. One misdiagnosis can lead to unnecessary immunosuppression or surgery, such as a superfluous appendectomy in this patient. Conclusion: While IMHMV is rare, this case highlights the importance of broadening the differential when clinical features and standard treatment responses do not align with the presumed diagnosis.
Whitney Wenner Lucy Greenhagen Roon Mahboub Ravikanth Nathani Dr. Ramakanth Pata Dr. Mithun Suresh	*Nitrous-Oxide Toxicity Causing Lower-Extremity Weakness and Neuropathy* Background: Nitrous oxide, commonly known as "laughing gas," is a colorless, odorless gas that has been used as a general anesthetic for over a century. It is also commonly used in culinary environments for food preparation. Its recreational use is globally widespread, ranking as the 10th most popular recreational drug in the world due to its euphoric and hallucinatory effects. Over the past 15 years, recreational use across the United States has been steadily increasing, with more significant increases observed in recent years. Nitrous oxide, commonly sold as "Whippit" tanks or "Galaxy Gas," is readily available for purchase in tobacco stores nationwide and can also be purchased online. Its chronic misuse is associated with bilateral extremity myeloneuropathy and low serum vitamin B12. As a neurotoxin, nitrous oxide works by inactivating vitamin B12, resulting in disrupted myelin sheath maintenance and leading to demyelination in both the peripheral and central nervous systems, which causes slowed or blocked nerve impulses. Case Presentation: A 30-year-old female with no relevant past medical history presented with bilateral lower extremity pain and weakness that she reported had been ongoing for one month before her visit. She described the pain in her legs as a pins-and-needles sensation that starts in her toes and travels up to her hips. Physical examination revealed bilateral lower extremity weakness that was more proximal than distal, with 4/5 strength with hip flexion/extension/abduction/adduction, left sided dorsi/plantar flexion and ankle eversion/inversion 4/5 compared to the 5/5 on the right side, diminished light touch, pinprick, and proprioception sensation in both feet below the ankles, sensory disturbance throughout both legs in

	scattered areas, and an unsteady gait. Laboratory workup noted low serum vitamin B12 levels. The NCS and EMG workup revealed prolonged distal onset latency as well as no response from the right fibular (EDB) motor, left tibial (AHB) motor, and the right sural sensory nerves, indicating severe sensorimotor neuropathy. Upon further questioning, the patient admitted to inhaling Whippit cannisters daily for the past several months. This knowledge, along with clinical findings, indicated nitrous oxide poisoning causing myeloneuropathy. She was started on cyanocobalamin (IM) for one week and then prescribed weekly injections for the next 3-6 months, followed by a maintenance dose of monthly injections. Discussion: In most cases of nitrous oxide-induced myeloneuropathy, patients are given intramuscular cyanocobalamin to replenish vitamin B12 stores in the body and work to reverse neurological damage. Most patients will experience improvement in their condition within a few months of treatment; however, it is common to continue experiencing sensory symptoms even after treatment ends. Conclusion: With the continued rise in nitrous oxide use throughout the country, it is necessary to be informed about the clinical presentation and associated treatments involved in managing this condition.
Drake Workman Dr. Margaret Crosby	*Atrial Standstill Leading to Thromboembolic Renal Infarction* Introduction: Atrial standstill (AS) is a rare arrhythmogenic condition characterized by the complete absence of both electrical and mechanical atrial activity, resulting in a lack of P waves on electrocardiography. It may be idiopathic or secondary to genetic mutations or structural cardiac diseases such as amyloidosis or myocarditis. Notably, atrial pacing does not elicit mechanical contraction, which leads to atrial stasis and a high risk of thromboembolism. Case Presentation: A 56-year-old male with a history of atrial standstill, VVIR pacemaker placement, embolic anterior STEMI on rivaroxaban, coronary artery disease, and hypertension presented with sudden-onset, severe left-sided abdominal pain and nausea. He reported a similar self-resolving episode one month prior. Labs were notable for acute kidney injury (creatinine 1.30 mg/dL), prolonged PT (26.5 sec), and INR of 2.2. CT abdomen and pelvis revealed multiple left renal embolic infarcts affecting 40–50% of the kidney. ECG showed a ventricularly paced rhythm with no additional abnormalities. He was admitted for management of renal infarction and associated AKI, and to determine the source of the embolisms. Notably, this event occurred while the patient was adherent with rivaroxaban therapy. As such, hematology was consulted and a hematologic workup was pursued. Hematology workup—including aPTT (67.8), DRVVT (1.34), and antiphospholipid antibody panel—

	was unrevealing. No clinical or imaging evidence suggested malignancy leading to a hypercoagulable state. Given his underlying AS, a cardiac source of embolism was suspected. Transesophageal echocardiogram revealed a large windsock variant of the left atrial appendage, spontaneous echo contrast with severely reduced inflow and outflow velocities, preserved left ventricular ejection fraction, and no overt thrombus. Due to presumed cardioembolic etiology, anticoagulation was transitioned to dabigatran per hematology recommendations. Warfarin was considered but was not a desired treatment option by the patient. Cardiology advised against surgical intervention as no procedural options are currently indicated for AS. Discussion: Atrial standstill is a rare but high-risk condition requiring lifelong anticoagulation. Owing to its rarity, there is a lack of evidence-based guidelines regarding optimal anticoagulation strategies. For this reason, current practice often mirrors that of atrial fibrillation management, though this case highlights the unique and persistent thromboembolic risk associated with atrial stasis, even with therapeutic anticoagulation. This underscores the importance of individualized treatment plans and close multidisciplinary collaboration. Further research is needed to establish standardized treatment protocols for atrial standstill.
Sarah Zhang Paige Stueve Dr. John Meisenheimer VII Dr. Noah Goldfarb	*Herpes Zoster Ophthalmicus Presenting with Hutchinson's Sign* Case Presentation: An 84-year-old male with a history of age related macular degeneration, diabetes mellitus type II, and non-melanoma skin cancer presented to dermatology for a routine skin check with a rash on the left side of his forehead ongoing for three days. The patient was up to date on vaccinations. The patient reported pain and numbness in the area of the rash, which consisted of papules and eroded vesicles overlying pink patches in the V1 dermatomal distribution without crossing the midline. The rash extended from the forehead to the nasal ridge, with accompanying involvement of the eyelid and mild periorbital edema and ptosis. The patient also reported left eye pain with extraocular movement and was referred to ophthalmology. Bilateral pupils were round and reactive to light without decrease in visual acuity and normal extraocular movement. Slit lamp exam showed punctate epithelial erosions in the left eye but was otherwise normal without dendrites or corneal edema. Intraocular pressure was normal. Fundoscopic exam showed mild bilateral cupping of the optic disk and bilateral drusen without evidence of retinal necrosis. The patient was diagnosed with herpes zoster ophthalmicus (HZO) and treated with valacyclovir 1000 mg three times a day for seven days. Two months later, on follow-up with ophthalmology, the rash and facial dysesthesia had resolved. The patient had no changes to visual acuity, and no evidence of persistent keratitis or retinal damage.

	Discussion: Herpes zoster (HZ) comes from the reactivation of the varicella zoster virus, which causes chickenpox during primary infection. The estimated lifetime prevalence of HZ is around 30%, but commonly affects older or immunosuppressed patients. HZ typically presents as dysesthesias followed by the development of a vesicular rash. Importantly, most cases of HZ are localized, occurring in a dermatomal distribution without crossing the sagittal plane. Treating HZ reduces the risk of the most common complication, chronic dysesthesia called post herpetic neuralgia. Approximately 10% of HZ cases involve the trigeminal nerve resulting in HZO. Hallmark symptoms of HZO include a unilateral rash in the V1 dermatome, burning pain, and headache. Differential diagnosis includes pre-septal cellulitis, post-septal cellulitis, and allergic contact dermatitis. Without timely treatment, up to 50% of HZO cases may result in ocular complications, including sequelae resulting in loss of visual acuity such as keratitis and acute retinal necrosis. Hutchinson's sign indicates involvement of the nasociliary branch of the trigeminal nerve, which innervates the nasal tip, alae, and ridge. Nasociliary involvement is associated with ocular symptomatology. The standard treatment for HZO is acyclovir, valacyclovir or famciclovir for seven days, ideally started within 72 hours of symptom onset. Conclusions: Painful, unilateral rash in the V1 distribution raises concern for HZO. Importantly, Hutchinson's sign includes rash over not just the nasal tip but also over the nasal ridge and nasal alae and portends ocular involvement. Recognizing HZO and administering the appropriate treatment within 72 hours of symptom onset can reduce the risk of long term neurologic and visual sequelae.
Quality Improvement - Medical Students	
Aarohi Shah Nissa Perry Dr. Rachael Mangaudis Dr. Jordan Marmet Dr. Kristina Krohn	*Improving Patient Safety Through Standardized Handoffs: Implementing the I-PASS Model* Background: The absence of a standardized approach to patient handoffs between shifts and units contributes to significant safety events. In the past two years at our hospital, communication gaps during these transitions have been identified as key factors in two immediate jeopardy events and roughly 20-30% of all serious safety incidents. Without standardization, critical information may be missed, and patients at risk of deterioration may not be identified in a timely manner. Method: To address these opportunities, we initiated a comprehensive quality improvement project to implement the I-PASS handoff model, which structures communication around Illness severity, Patient summary, Action list, Situational awareness and contingency planning, and Synthesis by the receiver. Beginning in May 2024, the initiative was rolled out in phases, starting with Pediatrics and OB/GYN. Key interventions included interactive training sessions for providers, the development of written handoff templates following the IPASS

	structure unique to multiple different specialties, and the development of faculty champions to observe and coach clinical teams. Two Plan-Do-Study-Act (PDSA) cycles guided implementation: the first established training and observation processes, while the second linked participation in observational feedback to Maintenance of Certification (MOC4) credit to promote sustained engagement and reinforce these practices. Our specific aim is to have 75% of handoffs across the institution utilize the I-PASS format by January 2026. Since the project started, the overall proportion of handoffs using the standardized I-PASS structure increased from 8% to 36% through July 2025. More specifically, the pediatric provider handoff rate reached 100% through May 2025 before decreasing to 40% in June 2025, and the internal medicine handoff rate similarly reached 100% through April 2025 before declining to 33% in June 2025. This shift in data suggests that as new trainees began in the summer, adherence to the I-PASS format dropped, likely reflecting the challenges of onboarding and integrating new staff into established workflows. This variation highlights the importance of continued monitoring once a goal has been reached and to watch for potential impacts from trainee turnover. Addressing these predictable dips by strengthening refresher training and ongoing support will be critical for maintaining and ultimately achieving our goal of 75% adherence by January 2026. In addition to pediatrics and internal medicine, we are also monitoring other teams, including the ICU and surgical specialties, to promote broader adoption of the I-PASS format. We have offered observation tools, training, and support to help integrate I-PASS into their workflows; however, these teams have not yet been able to fully engage in data collection and feedback cycles, likely due to competing clinical priorities and unique workflow challenges. Their limited participation highlights an opportunity for renewed engagement efforts and tailored strategies that reflect the specific needs of these specialties to advance institution-wide standardization. Conclusions: Overall, the I-PASS initiative has helped uncover persistent gaps, including naming the illness severity and allowing opportunity for the handoff recipient to synthesize information. Ultimately, by embedding a structured handoff into daily practice, we aim to reduce preventable harm associated with communication failures and improve patient safety throughout the hospital.
Transitional Medical Graduates	
Research - Transitional Medical Graduates	
Nidhi Lanka Dr. Sandeep Palakodeti Dr. O. Oluwasolabomi	*Effect of GLP-1 Receptor Agonists on Anthropometric, Metabolic, and Reproductive Health Parameters in Polycystic Ovarian Syndrome: A Systematic Review and Meta-Analysis*

Dr. R. Nalam Dr. G. Rangari	Introduction: PCOS is a complex condition with multifactorial etiology. GLP-1 RAs are used in insulin-resistant disorders like PCOS for their effects on weight and glycemic control. Recent studies suggest they may also improve follicular growth, endometrial health, ovulation, and fertility, independent of weight loss. This study evaluates the effects of GLP-1 RAs on anthropometric, metabolic, and reproductive parameters in PCOS. Methods: We followed PRISMA 2020 guidelines. We searched PubMed and Cochrane Library (2015-Feb 2025) using relevant keywords. We included RCTs and cohort studies with PCOS patients (per Rotterdam criteria) receiving GLP-1 RA monotherapy compared to placebo, metformin, or other interventions, reporting anthropometric, metabolic, or reproductive outcomes. Statistical analysis was performed using RevMan 5.4 with random-effects models, and heterogeneity was assessed using the I2 coefficient ($p<0.05$ considered significant). Results: We identified 73 studies, with 10 RCTs included (730 participants). Of these, 50.13% received GLP-1 RAs (49.5% liraglutide, 41% exenatide, 9.56% dulaglutide, 2.73% semaglutide), and 45.9% were controls (42.2% metformin, 20.3% placebo, others combination therapy). The meta-analysis findings are as follows: Anthropometric Outcomes: GLP-1 RAs caused significant reductions in BMI (9 studies, MD = -1.41, 95% CI: -1.73 to -1.10, $p<0.00001$, I2=100%) and waist circumference (8 studies, MD = -2.69, 95% CI: -4.06 to -1.32, p=0.0001, I2=100%). One study showed liraglutide reduced VAT (p=0.006). Metabolic Outcomes: GLP-1 RAs had a non-significant effect on HOMA-IR (7 studies, MD = -0.03, 95% CI: -0.82 to 0.76, p=0.94, I2=100%) and LDL (7 studies, MD = -0.45, 95% CI: -1.31 to 0.40, p=0.30, I2=100%). Three studies showed improvement in OGTT, and 1 showed a significant reduction in HbA1C (p=0.015). Reproductive Health Outcomes: GLP-1 RAs caused a significant decrease in total testosterone (10 studies, MD = -0.78, 95% CI: -1.02 to -0.55, $p<0.00001$, I2=99%). LH decreased non-significantly (4 studies, MD = -1.96, 95% CI: -4.18 to 0.26, p=0.08, I2=99%), while FSH increased non-significantly (4 studies, MD = 0.29, 95% CI: -0.69 to 1.27, p=0.56, I2=99%). Two studies showed significant improvements in menstrual regularity with liraglutide and exenatide (p=0.0001 and <0.001, respectively). Conclusions: Our findings suggest GLP-1 RAs could become key therapies for managing this condition. However, heterogeneity among studies—due to differences in design, drug used, control, and dosing—requires further studies to explore optimal dosages, durations, combination therapies, and their impact on ovulation and fertility.

Prajwal Shetty Dr. Prateek Kapur Jagruti Dasi Dr. D. H. Rao	*Knowledge, Attitudes and Practices Regarding the Measles Containing Vaccines During an Outbreak of Measles in an Urban Slum of a Metropolitan City: A Cross-Sectional Descriptive Study.* Introduction: Measles is a highly infectious disease and the city of Mumbai faced an outbreak in 2022, resulting in 600 cases and 17 deaths. Studies indicate the culprit to be a deficit in immunization coverage, but the reasons behind the same were yet to be identified. Methods: The study aimed to know the reasons behind missing the Measles Containing Vaccines (MCV), cited by caregivers of children reporting with febrile rash. The objectives included ascertaining reasons cited by caregivers and to assess the knowledge, attitudes and practices of the caregivers with respect to measles and MCV. In this cross-sectional qualitative study, caregivers of children aged 9 months to 5 years, reporting at Baiganwadi HP with febrile rash during a Measles outbreak, were surveyed. Following approval from the Ethics Committee and administrative clearance from MCGM, a peer validated, pilot-tested 30-item questionnaire was administered via telephone after verbal consent. Responses from 100 participants were recorded in Google Forms and analysed in MS Excel. Results: The study revealed that although 99% caregivers recognised measles, only 10% knew all key symptoms; and 53% were unaware of complications. Vaccination coverage was low: 50% unvaccinated, 18% unsure, 25% partially, and 7% fully vaccinated. Most common barriers included lack of awareness, concurrent illness, and fear of side effects. These results highlight critical gaps in caregiver knowledge and vaccination coverage. Conclusions: With half of the children not receiving a single dose of MCV, our study reveals major immunization gaps despite high awareness of Measles among caregivers. A targeted, community-specific response is needed to deliver full immunization coverage in vulnerable urban populations.
Mahsa Rezasoltani	*VEXAS Syndrome with Myelodysplastic Syndrome: A Case Highlighting the Intersection of Inflammation and Clonal Hematopoiesis* Background: VEXAS (vacuoles, E1 enzyme, X-linked, autoinflammatory, somatic) syndrome is a recently recognized disorder caused by acquired somatic mutations in the UBA1 gene. It most often presents in late adulthood with fever, recurrent inflammatory flares, cutaneous vasculitis, pulmonary infiltrates, cytopenias, and macrocytosis. Nearly half of patients develop concomitant myeloid neoplasms, most commonly myelodysplastic syndrome (MDS). Given its overlap with rheumatologic, dermatologic, and hematologic presentations, recognition of VEXAS is especially important for internists, who often serve as the first point of evaluation.

	Case Presentation: A 61-year-old man with no prior significant medical history presented with fever, malaise, and a diffuse lower extremity rash. Laboratory evaluation demonstrated elevated inflammatory markers (ferritin 771 ng/mL, ESR 94 mm/h, CRP 16.3 mg/dL). Skin biopsy revealed leukocytoclastic vasculitis, and symptoms improved with prednisone. Over the subsequent year, he developed progressive macrocytic anemia (hemoglobin ~10 g/dL, MCV 105 fL) and persistent hyperferritinemia. PET imaging demonstrated diffuse bone marrow uptake with pulmonary nodularity. Recurrent joint stiffness and systemic inflammatory flares required prolonged corticosteroids and multiple trials of steroid-sparing agents, including mycophenolate, rituximab, and hydroxychloroquine, with partial benefit. Bone marrow biopsy revealed MDS with ring sideroblasts (>15%), 4% blasts, and normal cytogenetics. Next-generation sequencing confirmed UBA1 p.Met41Val mutation (variant allele frequency 33%) establishing the diagnosis of VEXAS. Additional co-mutations included SF3B1, DNMT3A, and TET2, highlighting the link between oinflammation and clonal hematopoiesis. Despite a protracted clinical course, the patient has remained transfusion-independent with stable blood counts (hemoglobin 12 g/dL, WBC 4.5 $\times 10^9$/L, platelets 247 $\times 10^9$/L) and preserved functional status (KPS 90). His major morbidity stems from chronic steroid exposure, including weight gain, fatigue, and proximal muscle weakness. Evaluation for allogeneic stem cell transplantation was completed with insurance approval and donor search initiated; however, transplant timing remains under discussion given clinical stability and renal comorbidity. Conclusions: This case underscores the importance of considering VEXAS syndrome in patients with unexplained macrocytosis, cytopenias, and late-onset inflammatory or vasculitic manifestations. It illustrates the diagnostic challenge, the overlap between systemic inflammation and clonal hematopoiesis, and the therapeutic dilemma of balancing disease control with the risks of immunosuppression and transplantation. For internists, awareness of VEXAS is critical, as timely recognition can facilitate appropriate referral, genetic testing, and multidisciplinary management. Broader recognition and systematic reporting will be essential to guide prognosis and inform best practices.
Quality Improvement - Transitional Medical Graduates	
Arwa Bohra Saman Pathan Dr. Shubham Atal Dr. Santenna Chenchula Dr. Zeenat Fatima Dr. Ratinder Jhaj	*Exploring Polypharmacy: Defined Daily Doses as a Metric for Rational Inpatient Drug Use* Background: Polypharmacy remains a critical but inconsistently defined challenge in inpatient care. Unlike outpatient settings, where rational drug use can be benchmarked with WHO prescribing indicators, inpatient polypharmacy lacks standardized definitions or

Dr. Balakrishnan Sadasivam	assessment frameworks, complicating evaluation and stewardship efforts. Methods: This cross-sectional study reviewed treatment charts from 213 admissions across five wards of a tertiary-care teaching hospital in central India over six months. WHO core prescribing indicators were calculated, and drug consumption was standardized as Defined Daily Doses per 100 bed-days (DDD/100 BD). Potential drug–drug interactions (DDIs) were identified using Lexicomp®, and pharmacogenetically actionable medicines were documented based on CPIC, DPWG, and regulatory labelling. Results: Patients received a mean ± SD of 7.84 ± 1.80 medicines per admission, underscoring the burden of inpatient polypharmacy. Only 48.7% of drugs were prescribed generically, and 71.3% were from India's Essential Medicines List. Antimicrobials (20.7%), analgesics (14.6%), and vitamin–mineral supplements (13.5%) were the most frequent classes. Sedative–hypnotics had the highest DDD/100 BD (273), followed by ACE inhibitors (61), macrolides (33), and opioids (28). β-Lactams comprised 59% of 361 antimicrobial doses. Fourteen serious (X-level) DDIs and 25 medicines with pharmacogenetic-based recommendations were identified. Conclusions: Findings highlight that inpatient prescribing is characterized by high drug burden, extensive antimicrobial and injection use, and modest generic substitution. Yet, assessment remains ad hoc due to the absence of standardized inpatient polypharmacy definitions. Combining WHO indicators with DDD/100 BD, systematic DDI/PGx alerts, and clinical pharmacology feedback offers a pragmatic approach to standardizing monitoring, curbing inappropriate polypharmacy, and improving the safety and cost-effectiveness of inpatient care.
Residents	
Quality Improvement - Residents	
Abhishek Chandra Tanya Owens Dr. Kirsten Shaw Dr. Michael Miedema Dr. Kevin Harris	*Incidental Aortic Enlargement Noted on Coronary Calcium Scanning: How Often Do Patients Have Further Imaging and Specialist Referral?* Background: Incidental aortic enlargement is often picked up on coronary artery calcium (CAC) scans. There is a lack of data characterizing how often these patients have appropriate follow-up. We aimed to study follow-up patterns of aortic enlargement noted on CAC scans and factors associated with potential gaps in care. Methods: Consecutive patients with CAC scans showing dilated aortic dimensions were identified. Aortic size was stratified into three groups. Patient demographics, ordering provider vs self-referral status,

	presence of risk factors, and follow-up imaging and referral were collected. Results: From 3794 consecutive scans over 12 months, 200 patients (5%) were identified with aortic enlargement, dimensions were between 35-39.9 mm for 17, between 40-44.9 mm for 150, and > than 45 mm for 33 patients. 162 patients (81%) had follow-up imaging (68.5% within one year) and 38 patients (19%) had no follow-up imaging. 3 patients with aortic enlargement > 45 mm had no follow-up imaging. Mean time to follow-up imaging was shorter in patients with enlargement greater than 45 mm (7.9 months) compared to all others (15.8 months) ($p < 0.05$). Physician referral, vs self-referral for CAC scans was associated with greater likelihood of follow-up (85% vs 80% $p < 0.01$) as was having enlargement greater than 45 mm (91% vs 80%, $p < 0.05$). Male patients (82% vs 78%, $p < 0.05$) and those with history of tobacco use, hypertension and hyperlipidemia (all $p < 0.05$) were more likely to have follow-up imaging. In 42 patients (21%) the aorta enlargement led to referral to a cardiovascular provider within one-year of their initial CAC scan. Seven patients underwent aortic repair within five years. Conclusions: An incidental diagnosis of aortic enlargement was found in 5% of patients on CAC scans. There is inconsistent follow-up imaging of aortic enlargement noted on CAC screening with nearly 20% having no follow-up. Patients more likely to have follow-up include those with greater enlargement, referral by a healthcare provider, and males. This data highlights a need for structured reporting and referral protocols for patients with incidental aortic findings on CAC scans.
Justin Dunnell Dr. Emma Schaffer Dr. Caroline Davis Dr. David Flemig	*Code Connect: Communication Cirriculum for Healthcare Providers to Fight Burnout* Purpose: This quality improvement project aims to host a series of communication workshops at our county hospital in order to help providers feel more confident navigating moral dilemmas in medicine, which are some of the root causes of burnout. Background: Healthcare worker burnout has a negative impact on overall well-being and job satisfaction. High quality relationships between providers and patients as well as between interdisciplinary providers have a positive effect on burnout.1,2 Providers face moral dilemmas in patient care every day, and these situations are a major contributing factor to burnout. Good communication skills are essential in navigating these difficult situations to preserve relationships with patients and coworkers. Unfortunately, most providers do not receive adequate training in serious illness conversations. The core elements of effective serious illness communication include: assessing patient perspective, sharing information clearly, attending to emotion, eliciting goals and values, managing uncertainty, and shared decision-making. In an effort to

	improve mental health at the primary safety net hospital of Minneapolis, we will host a series of workshops for team members aimed at improving communication skills through structured didactics, teaching drills and simulations that mimic real world/bedside clinical practice. Methods: We will develop and present six trainings to an estimated 90 Physicians, Nurses, and Advanced Practice Providers. Five of the trainings will be skill-building workshops which will include skills of listening deeply, sharing information, recognizing and responding to emotion, and time management. Trainings will be held in 2-4 hour windows and can be delivered in flexible schedules to accommodate previously blocked educational time. After completing these trainings, we will hold patient simulations requiring participants to utilize these skills. Pre- and post-survey data will be gathered to assess empathy, burnout, job satisfaction, and confidence around communication before and after our curriculum. Conclusions: This project is currently in the "Do" phase of the IHI Framework Model for Improvement PDSA cycle. We have preliminary data from the pre-survey that indicates overall interest and excitement for this curriculum. 1 Fulmer T, Escobedo M, Berman A, Koren MJ, Hernández S, Hult A. Physicians' Views on Advance Care Planning and End-of-Life Care Conversations. J Am Geriatr Soc. 2018 Jul;66(6):1201-1205. doi: 10.1111/jgs.15374. Epub 2018 May 23. PMID: 29797314. 2 National Academies of Sciences, Engineering, and Medicine; National Academy of Medicine; Committee on Systems Approaches to Improve Patient Care by Supporting Clinician Well-Being. Taking Action Against Clinician Burnout: A Systems Approach to Professional Well-Being. Washington (DC): National Academies Press (US); 2019 Oct 23. PMID: 31940160.
Morgan Kelly Dr. Alyssa Welle Dr. Jacob Langness Dr. Peter Lund	*Delabel to Enable: Optimizing Antibiotic Stewardship Through an Outpatient Penicillin Allergy Delabeling Protocol* Background: Approximately 20% of patients report a penicillin allergy, but <5% have a true IgE-mediated reaction. Most true penicillin allergies wane over time with 80% of patients tolerating penicillin after a decade. Inaccurate labeling remains a threat to public health and antimicrobial stewardship. Alternative regimens are associated with unnecessary broad-spectrum antibiotic use, promotion of antibiotic resistance, more toxic side effects, increased rates of C. difficile infections and higher healthcare costs. For low-risk patients, a direct oral amoxicillin challenge is a safe and effective standard of care for confirming the absence of true penicillin allergy and enabling its removal from the medical record. Moderate risk patients may be evaluated further with skin testing.

	Purpose: Improve antibiotic stewardship by delabeling inappropriate penicillin allergies via a standardized protocol using the PENFAST assessment and an oral amoxicillin challenge. Implement the protocol in a primary care setting in an efficient manner to reduce the burden on clinician and patient time. Methods: Patients with a penicillin-class antibiotic allergy label from an outpatient clinic were identified through a population tool within the electronic health record. Clinicians completed PENFAST assessments with patients, delabeled intolerances (non-IgE-related reactions), referred low-risk patients (PENFAST score 0-2) for an oral amoxicillin challenge, and referred moderate to high-risk patients (PENFAST score 3-5) to allergists for skin testing and further assessment. A standardized outpatient amoxicillin oral challenge protocol for administration and documentation was developed, involving nursing, clinical pharmacy, and physicians. Patient education, healthcare worker education, and response to allergic reactions or complications involved documents and protocols were already created through Allina Health. Results: Of the 196 patients identified with a penicillin allergy label, 166 were eligible for evaluation. To date, 96 of 166 (57%) patients have been evaluated. Two patients declined to participate prior to any assessment. Fourteen patients were identified with non-allergic intolerances. 11 of 14 were delabeled, and 3 of 14 declined including two patients who had a non-allergic severe adverse reaction. Of the 50 low-risk patients, 19 (38%) completed an amoxicillin challenge with 18 of 19 (95%) successfully delabeled without an allergic reaction, 21 (42%) are pending, and 10 (20%) declined the challenge. Among 30 moderate-high risk patients, 7 (23%) completed skin testing without reaction, 12 (40%) are pending, and 11 (37%) declined. In total, 36 patients (38% of those evaluated so far) have been successfully delabeled to date. Throughout the process 26 of 96 (27%) of those evaluated have declined to follow through with the protocol at various stages. Conclusions: Complete evaluations for the remaining patients, improve the efficiency of the protocol as we standardize workflow, explore reasons for patients declining delabeling protocols, provide targeted education to patients and clinicians, and expand the protocol to additional outpatient clinics.
Research - Residents	
No Residents Presenting Research in 2025	
Clinical Vignette - Residents	
Mohammed Al Kurnas Dr. Jonas Paludo Dr. Kebede Begna	*Primary CNS Diffuse Large B-cell Lymphoma and Hodgkin Lymphoma in the Same Patient: A Rare Case of Composite Lymphoma*

	Background: Primary diffuse large B-cell lymphoma of the central nervous system (PCNSL) is a rare and aggressive subtype of diffuse large B-cell lymphoma (DLBCL). Classical Hodgkin lymphoma (HL), characterized by Reed-Sternberg cells, usually presents in supradiaphragmatic lymph nodes and spreads in a predictable pattern. While both are uncommon individually, the simultaneous presence of PCNSL and HL as a composite lymphoma is exceedingly rare and presents a unique therapeutic challenge. Case Presentation: A 66-year-old white male who initially presented with progressive neurological symptoms. MRI revealed brain mass, and biopsy confirmed to be PCNSL. Further staging with PET-CT identified FDG-avid cervical lymphadenopathy. Core needle biopsy of a cervical node demonstrated classical HL. Given the dual diagnosis, treatment was initiated with ten cycles of Methotrexate-Rituximab-Temezolamide (MRT) targeting the CNS lesions, followed by two cycles of high-dose methotrexate for consolidation therapy. Subsequent MRI of the brain revealed complete remission of PCNSL. The patient then received one cycle of Nivolumab-AVD (Nivo-AVD) for HL, but treatment was discontinued due to intolerance. He was deemed to be a candidate for high dose chemotherapy followed by autologous stem cell transplantation (ASCT). To deepen remission of HL before transplant, he was given a single cycle of bendamustine. He subsequently underwent high dose BCNU, Etoposide, Ara-C, and Melphalan chemotherapy (BEAM) followed by ASCT. The patient tolerated the transplant well. Subsequent MRI Brain and PET-CT four months after transplantation revealed continued remission of PCNSL and resolution of his lymphadenopathy with a Deauville score of 1 respectively. He has remained in complete remission for ten months, with no evidence of disease on follow-up imaging or neurological examination. Conclusions: Composite lymphomas involving PCNSL and HL are extraordinarily rare, with no established treatment guidelines. Our case illustrates the importance of addressing the more aggressive lymphoma first, in this case, PCNSL, while sequentially targeting HL. The treatment regimen resulted in a durable remission. As formal trials are unlikely due to the rarity of such cases, well documented case reports remain vital in guiding management strategies.
Kirsten Aspros Dr. Rose Baumann Dr. Ellen Overson	*A Case of Nonconvulsive Status Epilepticus as Manifestation of Cefepime Neurotoxicity* Background: Cefepime is a commonly used, broad spectrum, fourth generation cephalosporin. An increasingly recognized side effect of cefepime is neurotoxicity, which manifests as decreased level of consciousness, disorientation, myoclonus, nonconvulsive status epilepticus, seizures, and aphasia. It is theorized that cefepime induces neurotoxicity via crossing the blood brain barrier and antagonizing GABA receptor signaling. It is estimated that approximately 0.15% of all patients and 15% of ICU patients who are treated with cefepime will develop neurotoxicity. The greatest risk factor for developing

	cefepime-induced neurotoxicity (CIN) is renal dysfunction. Most patients will develop CIN within 4-6 days of starting cefepime. Case Presentation: A 63-year-old female with a history of T2DM complicated by diabetic foot ulcers, osteomyelitis, and right calcaneal fracture s/p closed reduction and percutaneous pinning on chronic suppressive cefepime, daptomycin, and metronidazole, as well as depression, anxiety, paroxysmal atrial fibrillation, and peripheral artery disease, was admitted for altered mental status (AMS) and acute kidney injury (AKI). Presenting history notable for 12 hours of increased fatigue followed by the patient being found down by her spouse, able to speak some words that became garbled and nonsensical. On exam, patient was stuporous, opened her eyes to voice, and did not follow commands. She repeatedly said "ouch". She had intermittent but frequent facial twitching similar to grimacing. Labs were notable for an AKI (Cr 3.1, baseline 1). Additional work up including head imaging and eventually lumbar puncture did not reveal an etiology. Initially she was thought to have AMS due to toxic accumulation of multiple CNS-altering medications in the setting of an AKI. However, mental status did not improve as her renal function improved. Neurology was consulted and performed an EEG that was immediately notable for generalized periodic discharges and triphasic waves consistent with non-convulsive status epilepticus (NCSE) and toxic metabolic encephalopathy. Her NSCE terminated briefly with a midazolam challenge. She was started on levetiracetam and later lacosamide was added. Due to concerns for CIN, cefepime was held. After several days of these treatments, her mental status returned to normal. Ultimately it was concluded that she developed NCSE as a result of cefepime neurotoxicity. Her presentation was unusual in that NSCE usually occurs within 4-6 days of initiation of cefepime, yet she had been on this medication for several weeks. This most likely occurred due to her AKI, the cause of which was not elucidated, or possibly missed doses of her home lorazepam and gabapentin while fatigued. Additionally, she may have been at greater risk for NSCE because of taking other medications associated with a lower seizure threshold (e.g. bupropion, citalopram, cyclobenzaprine, oxycodone). Conclusions: Cefepime neurotoxicity typically occurs within the first 4-6 days of initiation of therapy. However, for patients on prolonged therapy, it remains possible to develop severe neurotoxicity when renal function is impaired. NCSE should be considered as manifestation of neurotoxicity in a patient on cefepime. Regardless of the duration of therapy, for a patient on cefepime who is presenting with encephalopathy, cefepime neurotoxicity should remain on the differential diagnosis.
Jasson Barrios Dr. Brendon Herring Dr. Jonathan Day	*Acquired Hemophilia A Presenting as Spontaneous Bleeding in an Elderly Patient on Antiplatelet Therapy*

Dr. Aneel Ashrani Dr. Chris Aakre	Background: Acquired hemophilia A (AHA) is a rare bleeding disorder typically caused by autoantibodies directed against coagulation factor VIII. It predominantly affects elderly patients and can present with life-threatening hemorrhages if not managed adequately. We present a case of AHA in an elderly man with multiple cardiovascular comorbidities. Case Presentation: An 88-year-old man presented to the emergency department (ED) with dyspnea on exertion and left hip pain. Past medical history was significant for coronary artery disease with percutaneous coronary intervention 25 years prior, aortic valve replacement, heart failure with preserved ejection fraction, prior deep vein thrombosis and pulmonary embolism, type 2 diabetes, chronic kidney disease stage 3, and polymyalgia rheumatica. Upon presentation to the ED, patient reported a progressive three-month history of atraumatic spontaneous bleeding, resulting in hematomas affecting multiple extremities with active superficial oozing requiring frequent dressing changes. Patient was on Plavix for secondary prevention after stopping Eliquis three months prior. Notably, bleeding persisted despite the patient temporarily self-holding Plavix for a two-week trial. ED evaluation revealed a large retroperitoneal hematoma of unclear etiology. Patient promptly received emergent embolization of the left deep circumflex iliac artery and iliolumbar branch of the internal iliac artery. The patient required transfusion of two units of packed red blood cells and was admitted for further hematological workup. Diagnostic Workup: Hematology was consulted for suspected acquired bleeding disorder given spontaneous bleeding and history of thrombotic disease. Heyde syndrome was considered given his history of aortic valve replacement, but workup was largely unrevealing. The coagulation studies revealed a prolonged activated partial thromboplastin time of 66 seconds. Comprehensive factor analysis demonstrated critically reduced factor VIII activity of 1% with normal levels of factors IX, XI, XII, and von Willebrand factor. Mixing studies confirmed the presence of a factor VIII inhibitor, establishing the diagnosis of AHA. The differential etiology included medication-induced (Plavix), autoimmune disease, or occult malignancy. PET-CT imaging was negative for malignancy, and serologic evaluation for underlying autoimmune disorders was similarly unremarkable, suggesting drug-induced or idiopathic etiology. Notably, additional history revealed that he initially had an allergic response to Plavix that required desensitization therapy. Results: The patient was initiated on high-dose immunosuppression with prednisone 1mg/kg daily and rituximab 800mg administered on hospital days 5 and 12. Hemostatic therapy with emicizumab was initiated at 3mg/kg with subsequent weekly dosing. The patient demonstrated excellent clinical response with resolution of active

	bleeding following embolization and medical therapy. He remained hemodynamically stable without requiring further transfusions after the initial 48 hours of admission. Repeat chromogenic factor VIII assay on hospital day 12 showed persistent factor VIII activity of 1.2% with elevated inhibitor levels, though patient remained clinically hemostatic. Conclusions: This case outlines the importance of recognizing AHA in elderly patients with complex cardiovascular histories who present with spontaneous bleeding. Maintaining clinical suspicion for acquired coagulopathies in this population represents a diagnostic challenge, yet prompt diagnosis through comprehensive coagulation studies combined with immunosuppressive therapy and appropriate hemostatic agents can result in successful hemostasis and favorable outcomes.
Jacob Beery	*From Substance Use to Striatal Dysfunction: A Case of Methamphetamine-Induced Hemiballismus* Background: Chorea and ballism represent a spectrum of hyperkinetic movement disorders characterized by abrupt, involuntary, non-rhythmic muscle contractions. Chorea typically presents with distal, low-amplitude, flowing movements, whereas ballism involves large-amplitude, proximal, flinging motions, often unilateral as hemiballismus. These disorders arise from various etiologies, including genetic conditions, structural lesions of the basal ganglia, autoimmune and parainfectious processes, metabolic or toxic insults, infections, and drug-induced causes. Among drug-induced etiologies, substances such as antipsychotics, amphetamines, and other psychoactive agents are frequently implicated. Case Presentation: This case report describes a 43-year-old man with schizophrenia and a history of methamphetamine use disorder who presented with acute onset of uncontrolled, unilateral ballistic movements involving the left upper and lower extremities. Physical examination revealed normal cranial nerve, motor, and sensory function, but violent, flinging movements confined to the left side of the body. Initial laboratory workup and imaging, including CT angiography and MRI of the brain,were unremarkable. However, a urine drug screen confirmed recent methamphetamine use. Neurology and psychiatry consultations attributed his symptoms to methamphetamine-induced hemiballismus. The patient was treated supportively, advised on substance cessation, and discharged on a restarted antipsychotic regimen with follow-up arranged. His symptoms were resolved by the time of outpatient follow-up. The discussion emphasizes the importance of differentiating between primary (hereditary or idiopathic) and secondary (acquired) causes of chorea/ballism. While primary causes such as Huntington's disease typically follow a chronic, progressive course, secondary causes, including stroke, autoimmune disorders, and drug toxicity, present more acutely. Methamphetamine-induced chorea/ballism is a known

	but uncommon presentation, likely mediated by oxidative stress, microglial activation, and mitochondrial dysfunction within basal ganglia circuits. Diagnosis relies heavily on a thorough history and physical examination, supported by targeted laboratory testing and neuroimaging to exclude structural or ischemic lesions. Management of chorea/ballism is etiology-specific. In drug-induced cases, cessation of the offending agent can lead to full recovery, though symptoms may persist for weeks to months depending on the extent of neural damage. Symptomatic treatment may include second-generation antipsychotics such as risperidone or olanzapine, though evidence remains limited. Conclusion: This case underscores the clinical importance of recognizing methamphetamine use as a reversible cause of hemiballismus and highlights the need for comprehensive diagnostic evaluation to guide appropriate, etiology-directed management.
Christopher Behrend	*Hepatic Schistosomiasis Re-Infection in a Liver Transplant Recipient* Introduction: Schistosomiasis is a parasitic disease which is estimated to affect a worldwide population of 230 million through all forms. There are three primary species of schistosomes which affect humans and these species are primarily responsible for infections through two major forms, urogenital and gastrointestinal. Infections are caused through direct contact with surface water containing schistosomes in their free-living form. These species migrate via blood flow and can cause hepatic schistosomiasis through entry into the portal venous system, where they may mature. Eggs are deposited into tissue and can initiate a lifecycle within its host. Hepatic schistosomiasis can lead to a similar presentation to acute liver failure with evidence of hepatic fibrosis, portal hypertension, portosystemic collateral blood flow, massive ascites, and hepatic encephalopathy. Case Presentation: This clinical case involves a 41-year-old male with a past medical history of cirrhosis secondary to hepatitis B virus (HBV) and hepatitis D virus (HDV) coinfection, status post liver transplant with multiple prior episodes of acute cellular rejection. The patient had a prior hepatic schistosomiasis infection years after transplant that was treated at that time with praziquental. The patient presented to the hospital years later with generalized abdominal pain, scleral icterus, and jaundice. Initial liver chemistry testing was elevated, raising concern for a repeat episode of acute cellular rejection. On repeat liver biopsy, schistosomal granulomatous portal phlebitis superimposed on ductopenia likely from prior rejection injuries was seen in the absence of evidence of active rejection. The patient had not traveled to an endemic area or expressed medication nonadherence in the period of time between initial treated hepatic schistosomiasis infection and re-demonstration of schistocytes on liver biopsy. The patient was re-initiated on praziquental with repeat biopsy demonstrating evidence of chronic rejection in setting of nonadherence to immunosuppression, however, no evidence of schistocytes.

	Discussion: The patient in this case demonstrated re-infection of hepatic schistosomiasis post treatment course without travel to an endemic area or known infection contact. While the initial presentation was concerning for a repeat episode of acute cellular rejection in the setting of medication nonadherence, further workup yielded evidence of reinfection. Based on the life cycle of schistocytes and mechanism of praziquental acting on adult worms, it is hypothesized that schistosomal egg forms had matured in the time period and were not treated by initial praiquental. Conclusion: This case highlights an unusual case of re-infection and demonstrates the importance of keeping a broad workup with patient status post liver transplant presenting with signs concerning for acute cellular rejection.
Rebecca Caldwell Dr. Lucinda Gruber	*Masked by Motherhood: A Hidden Case of Autoimmune Adrenalitis* Background: New-onset adrenal insufficiency (AI) in the postpartum period is rare and typically occurs in the context of Sheehan syndrome or adrenal hemorrhage. Autoimmune adrenalitis causing primary AI in the postpartum period is not well quantified, though the postpartum window is recognized for unmasking autoimmune disease. The clinical presentation of AI is nonspecific, and symptoms may overlap with normal postpartum fatigue, weight changes, or depression, making diagnosis challenging. Adrenal crisis is a potentially fatal complication that can occur if diagnosis is delayed or missed. Prompt identification and treatment are critical, particularly for primary care providers (PCPs) who see patients routinely. Case Presentation: A 37-year-old woman, eight months postpartum with a history of primary hypothyroidism, presented to her PCP with a constellation of symptoms. She reported a 30-40lb weight loss over four months and early satiety. Her appetite was normal. She denied fatigue but noted decreased activity levels. She reported shortness of breath on exertion and light-headedness, with blacking out of her vision during position changes. She reported numbness in her arms and hands and darkening of moles on her arms. She denied salt cravings and changes in axillary or pubic hair. She was breastfeeding, and her menstrual cycles had not yet returned, but were regular prior to pregnancy. Vital signs showed hypotension (74/46). Physical exam revealed darkened moles and hair loss at the frontal scalp but was otherwise unremarkable. Laboratory workup revealed a morning cortisol 0.5, sodium 133, potassium 4.8, glucose 96, TSH 9.5, tissue transglutaminase antibody 76.6, and IgA 87. Repeat testing showed cortisol 0.4 and ACTH 2620. 21-Hydroxylase antibodies were positive, confirming autoimmune adrenalitis causing primary AI. Treatment was initiated with hydrocortisone and fludrocortisone, with significant clinical improvement in two weeks. Levothyroxine was not adjusted to prevent precipitation of an adrenal crisis. Moreover, the newly diagnosed celiac disease was thought to be affecting its

	absorption. She was advised to follow a gluten-free diet and the TSH normalized without adjustments to levothyroxine dosing. Discussion: The postpartum period is associated with increased risk of autoimmune disease, though rarely autoimmune adrenalitis. A careful history and physical exam are crucial when evaluating postpartum patients. Symptoms of AI are nonspecific and less than half of patients are diagnosed within the first six months of onset. Fatigue, weight loss, and gastrointestinal distress are often worse in the morning. Additional clinical clues for primary AI include orthostasis, hyponatremia, hyperkalemia, and hyperpigmentation, often involving the oral mucosa, palmar creases, or surgical scars. Over half of patients with autoimmune adrenalitis have a known autoimmune disease at the time of diagnosis. Autoimmune adrenalitis should be considered in postpartum patients presenting with multiple concerning symptoms and a history of autoimmune disease. Conclusion: To support early recognition in primary care, a clinical scoring system that integrates common presenting symptoms (i.e. fatigue, weight loss) with associated features of AI (i.e. orthostasis, electrolyte abnormalities, hyperpigmentation) may help clinicians risk-stratify patients and prompt timely diagnostic evaluation.
Kevin Cao	*Post-transplant Lymphoproliferative disorder* Case Presentation: We present a case of a 68/yo F who was discovered to have post-transplant lymphoproliferative disorder (PTLD). The patient's medical history includes: 2x deceased donor kidney transplant, previous MRDO E. coli infections, Epstein Bar viremia (EBV) and SLE. She was admitted for sepsis in the setting of chronic immunosuppression. Initially found to be febrile with an altered mental status, leukocytosis of 34k, and an elevated lactate. Sepsis protocol fluids and broad spectrum antibiotics were started. Initial diagnostics did not yield obvious evidence of urosepsis, bacteremia, skin-soft tissue infection or meningoencephalitis. Further imaging with CT chest-abdomen-pelvis ultimately showed diffuse lymphadenopathy in the retroperitoneum, mediastinum, axillae as well as ground-glass opacities in the bilateral lungs. She was empirically treated with a 5 day course of moxifloxacin with improvement for assumed pneumonia. Extensive infectious workup including HIV, blastomycosis, coccidioides, histoplasmosis, strongyloides IgG, rapid malaria test, SARS-CoV-2, Tb quant-gold, beta-D-glucan, and aspergillus galactomannan antigen were considered but were all negative. On further history, the patient noted 3 months of cyclic malaise and fatigue with an up trending EBV viral titer of 431k copies. The patient would be admitted multiple times for sepsis-like presentations. Her leukocytosis would climb to >50k, prompting an EBUS mediastinal lymph node biopsy and hematology/oncology consultation. On pathology, there was no evidence of lymphoma or

	metastatic carcinoma, shifting the differential towards possible plasma cell dyscrasias vs post-transplant lymphoproliferative disease. Eventually, a periaortic/common iliac lymph node biopsy showed atypical lymphoid polymorphic infiltrate with majority T-cells consistent with polymorphic PTLD. Upon discovery, the patient was trialed on Rituximab and then CHOP therapy. PTLD is a mixed group of plasmacytic proliferations that are commonly associated with solid organ or allogeneic hemopoietic stem cell transplantations. EBV infections drive the majority of the cases through host lymphocytic genetic aberration, but to note, EBV-negative PTLD has been documented. Recipients of multiorgan, lung, and heart transplants carry the highest relative risk of PTLD, while kidney transplants are markedly lower. In this case, the patient did receive 2 transplants during her life making her at higher risk. Discussion: Early diagnosis of this condition can confer earlier intervention, but there is current debate on when to intervene. There is varied data on intervention thresholds for EBV DNA titers and patient risk stratification for their respective cohorts. What has been shown is that preemptive rituximab therapy is highly effective in improving survival and disease burden. Conclusion: Serial EBV DNA monitoring is used in the first-year post-transplantation, but further testing of higher risk patients should also be considered given the overall mortality rate ranging from 40-60%. Suspicion of malignant processes in solid organ transplants should prompt PTLD on the differential.
Alexis Clark Dr. Muhammed Kizilgul Dr. Laura LaFave	*A Rare Case of Water Balance Reversal: Suspected AVP Deficiency Evolving into SIADH After Kidney Transplant* Background: Diabetes insipidus is a rare condition, only affecting approximately 1 in 25,000 people. It is typically described as a disorder impacting fluid regulation as a result of decreased vasopressin/ADH secretion by endocrine axis dysfunction in the hypothalamus or posterior pituitary gland (arginine vasopressin deficiency, previously known as central DI) versus decreased V2 receptor responsiveness in the kidneys (arginine vasopressin resistance, previously known as nephrogenic DI). Case Presentation: In the case of this 70-year-old female, arginine vasopressin deficiency was suspected as part of panhypopituitarism following treatment of a pituitary macroadenoma in the late 1990s, including transsphenoidal resection and late recurrence requiring subsequent XRT in 2004. She was treated with full pituitary hormone replacement including desmopression (DDAVP). The patient's medical history was complicated by concurrent bipolar 1 disorder treated for approximately 30 years with lithium, leading to progressive kidney disease and subsequent nonoliguric, dialysis-dependent ESRD. In 2025, she was able to undergo DDKT with brief perioperative

	cessation of her PTA DDVAP. Post-operatively the patient had unexpected hyponatremia in the setting of oliguria - a remarkable change from her preoperative diagnosis of arginine vasopressin deficiency in the absence of additional exogenous ADH administration. Medical work up included assessment of copeptin which was notably elevated (>21.4) - suggesting AVP resistance over previously diagnosed AVP deficiency. While the patient initially demonstrated symptoms of AVP deficiency following treatment for her pituitary macroadenoma with late recurrence, her current postoperative course strongly suggests chronic AVP resistance due to lithium exposure and ESRD. It is likely that kidney transplantation restored renal responsiveness to AVP, unmasking physiologic and possible stress-induced vasopressin activity, leading to transient hyponatremia and oliguria. In the postoperative course, the patient has continued to demonstrate controlled sodium levels, without instances of hypernatremia, and has remained off of DDAVP, further supporting this hypothesis. Conclusion: This case illustrates a rare resolution of presumed AVP deficiency (Central DI) with an SIADH-like presentation in the unique setting of long-term lithium exposure, ESRD, and pituitary pathology, highlighting the diagnostic uncertainty providers face in differentiating true AVP deficiency from resistance in complex endocrine and renal interactions.
Crosby Meg Drake Workman	*A Case of Recreational NO2 Induced Subacute Combined Degeneration* Introduction: Nitrous oxide (N_2O)–induced myeloneuropathy is a rare neurological condition characterized by subacute onset of distal, often symmetric paresthesias, limb weakness, sensory ataxia, and gait disturbances. In rare cases, it can cause bowel or bladder dysfunction. N_2O oxidizes the cobalt ion in cobalamin (vitamin B12), rendering it inactive and leading to a functional B12 deficiency. This mimics subacute combined degeneration (SCD) and may occur even when serum B12 levels appear normal. B12 is a crucial cofactor in myelin synthesis, so deficiency leads to the robust neurological consequences seen in SCD. Case Presentation: A 45-year-old nonbinary individual assigned male at birth, with a medical history of type II diabetes mellitus, alcohol use disorder in remission, cocaine use disorder in remission, tobacco use disorder, migraines, and obstructive sleep apnea, presented to the emergency department with two weeks of worsening numbness and tingling in the bilateral hands and feet. They also reported dizziness, particularly upon standing. This patient denied recent substance use, as well as infectious symptoms, joint pain, cold intolerance, weight changes, or rashes. On exam, the patient had a positive Tinel's sign bilaterally and gait instability. There was no gross motor weakness or sensory deficit.

	Initial workup, including brain MRI, was unremarkable. Labs revealed a blood glucose of 178 mg/dL, and both a BMP and CBC were unremarkable. Chart review showed a recent HbA1c of 10.6%. Additional testing included thyroid-stimulating hormone (2.50 mIU/L), erythrocyte sedimentation rate (0.25 mm/hr), C-reactive protein (10 mg/L), methylmalonic acid (1.52 µmol/L), iron-binding capacity (259 mcg/dL), transferrin (174 mg/dL), as well as unremarkable results for total urine protein, serum and urine protein electrophoresis, antinuclear antibody, vitamin B6, vitamin E, HIV, and RPR. A serum B12 level was not included. Neurology was consulted and agreed with the diagnostic workup. Initially, diabetic neuropathy was considered most likely, given the elevated A1c and bilateral carpal tunnel syndrome. However, the acute onset of symptoms was atypical. Upon further history-taking, the patient disclosed frequent nitrous oxide use. A cervical spine MRI revealed abnormal T2 hyperintensities in the dorsal columns from C2 to C6—findings consistent with subacute combined degeneration. Treatment was initiated with daily intramuscular high-dose B12 for one week, followed by weekly injections for four weeks. The patient was discharged to inpatient rehabilitation due to persistent gait instability and sensory symptoms, which had not improved significantly with initial therapy. At outpatient Neurology follow up, the patient reported being "95% better" at 8 weeks. Conclusions: This case underscores the importance of maintaining a broad differential diagnosis and conducting targeted history-taking. Nitrous oxide–induced myeloneuropathy, while uncommon, closely resembles subacute combined degeneration. Clinicians must specifically ask about nitrous oxide use when faced with compatible neurological symptoms.
Tyler Crowe	*Dilated Cardiomyopathy due to an Asymptomatic but Hemodynamically Significant Patent Ductus Arteriosus* Introduction: Cardiomyopathies are a heterogeneous group of diseases with a broad differential of etiologies including both common and rare causes depending on patient-specific factors. Presentations are highly variable, meaning correct diagnosis and treatment depend on the clinician's ability to have a differential that is both efficient and broad. This vignette explores a rare cause of cardiomyopathy and highlights the importance of the special considerations that must be for immigrant patients, especially from rural and low-resource regions. Case Presentation: A 25-year-old woman without known past medical history presented to the Emergency Department with acute abdominal pain. She had recently immigrated from Central America, where she did not have access to most medical services growing up. During her evaluation she was incidentally noted to have a grade IV/VI systolic

	murmur, and bedside ultrasound was concerning for mitral thickening. Surprisingly a formal TEE instead showed dilated cardiomyopathy with LV dimension of 6.6cm, as well as a large left-to-right extracardiac shunt consistent with a patent ductus arteriosus (PDA). Other findings include preserved ejection fraction (EF), LV thickening, and left atrial enlargement. A right heart catheterization confirmed moderate pulmonary hypertension and a tubular, 1.5cm PDA with a Qp/Qs of 3.5. Despite these findings, the patient was very active and without any dyspneic or anginal symptoms. Cardiac surgery evaluated the patient and recommended outpatient follow up, as well as PDA closure with Adult Congenital Heart Disease at the nearby tertiary center. She was started on an ACE inhibitor, and tolerated low doses to minimize future remodeling. A few months later she underwent transcatheter closure of her PDA with a VSD Occluder, but this was complicated by device embolization in PACU requiring repeat catheterization for retrieval. She subsequently underwent surgical ligation a few weeks later, which was well-tolerated and without complication. Her postoperative TTE showed a now reduced EF to 40%, as well as residual left-to-right PDA shunt with peak gradient of 107 mmHg. Sadly, she has been lost to follow up since. Although a reduction in EF is expected immediately post-procedure, the literature suggests that most adults will still have a mildly reduced EF after 12 months. Limited data exists on long term outcomes. Conclusions: This case highlights the importance of considering not only the additional exposures, but lack of screening in our younger immigrant patients when evaluating for heterogenous disease pathologies such as heart failure. Clinicians should not take the thorough screening that years of pediatric care provide for granted, and recall that young patients may be compensate physiologically for significant abnormalities when constructing their differential. Since adults typically have some degree of reduced EF after hemodynamically significant PDA closure, timely evaluation and referral for intervention is necessary.
Maria Diaz	*A Dopamine-Secreting Cervical Paraganglioma Masquerading as Recurrent Neck Mass: Diagnostic Challenges in an Asymptomatic Young Adult* Introduction: Paragangliomas are rare neuroendocrine tumors arising from extra-adrenal chromaffin tissue. While most catecholamine-secreting tumors produce norepinephrine and epinephrine, dopamine-secreting paragangliomas represent a particularly challenging diagnostic entity due to their often asymptomatic presentation. Head and neck paragangliomas rarely secrete catecholamines and when they do, dopamine is the predominant hormone produced. We present a case of a young patient whose recurrent cervical mass was ultimately diagnosed as a dopamine-secreting paraganglioma

	Case Presentation: A 31-year-old male presented to the emergency department with a 10-day history of right periorbital pain. His medical history was significant for a left cervical mass of unknown etiology that had been surgically resected in Ecuador in 2019. The prior surgery was complicated by a left posterior stroke affecting the middle cerebral artery, resulting in residual neurological deficits. Physical examination revealed multiple vesicular lesions in the right trigeminal nerve distribution consistent with herpes zoster ophthalmicus. Additionally, a 5-cm pulsatile, soft mass was palpated in the left neck without associated lymphadenopathy. The patient was admitted for intravenous acyclovir therapy. Given the palpable neck mass, CT scan was obtained to further characterize finding. CT angiography demonstrated a 5.4 × 3.9 × 5.6 cm heterogeneously enhancing mass in the left neck with hypervascularity and central necrosis. ENT consultation deferred FNA due to the highly vascular nature and proximity to critical neurovascular structures, recommending further imaging characterization. MRI confirmed a well circumscribed mass adjacent to the left carotid artery with imaging features highly suggestive of a carotid body tumor. This prompted endocrinology consultation for biochemical evaluation. Initial 24-hour urine metanephrines were within normal limits at 245 mcg/24h (normal <400mcg/24h). However, urine normetanephrines were elevated at 873 mcg/24h (normal <375mcg/24h) with corresponding elevated plasma normetanephrine levels at 1.8 nmol/L (normal <0.9 nmol/L). Most notably, plasma dopamine was elevated at 1,255 pmol/L (normal< 240 pmol/L). Given the patient's young age and family history of early-onset hypertension in a sibling, genetic testing for hereditary pheochromocytoma-paraganglioma syndromes was performed, which returned negative. PET scan demonstrated intense uptake in the left cervical mass with no evidence of metastatic disease or additional paragangliomas. Based on the combination of characteristic imaging findings, elevated dopamine and normetanephrine levels the diagnosis of dopamine-secreting cervical paraganglioma was established. The patient was initiated on alpha- adrenergic blockade with doxazosin followed by beta-blockade with metoprolol prior to planned surgical resection. Discussion: This case demonstrates three key clinical concepts. First, dopamine-secreting paragangliomas often present asymptomatically, lacking the classic catecholamine excess symptoms, which can lead to delayed diagnosis. Second, the incidental discovery during evaluation for an unrelated condition (herpes zoster ophthalmicus) highlights how systematic imaging can reveal significant underlying pathology in patients with complex medical histories. Third, the recurrence of a cervical mass following prior resection underscores the importance of adequate initial surgical planning and long-term surveillance, particularly in young patients where hereditary syndromes may be involved despite negative genetic testing.

Trinh Do	*Paraneoplastic Acquired Hemophilia A in a Patient with Small Lung Cell Carcinoma* Introduction: Paraneoplastic syndromes are a heterogeneous collection of clinical syndromes typically preceding or obscuring the diagnosis of the associated cancer. Small cell carcinoma of the lung (SCLC), an extremely aggressive neuroendocrine neoplasm with a strong propensity for early metastasis, is particularly notorious for its association with paraneoplastic phenomena. Although neurologic, endocrine, and metabolic syndromes are most adequately described, hematologic presentations are incredibly uncommon. Hemophilia, a genetic coagulopathy caused by the deficiency of coagulants VIII or IX, has been uncommonly described as an acquired paraneoplastic syndrome in association with SCLC. The intersection of a classically inherited coagulopathy and an acquired condition precipitated by a malignancy presents unique diagnostic challenges, particularly in the differentiation of paraneoplastic acquired hemophilia from other causes of bleeding in patients with cancer. We describe herein an uncommon case of hemophilia as a paraneoplastic syndrome associated with SCLC, and discuss its clinical relevance, diagnostic challenges, and therapeutic strategy. Case Presentation: A 61-year-old woman with a history of newly diagnosed SCLC was admitted for hemoptysis complicated by acute hypoxic respiratory failure. She developed an acute onset of hemoptysis one week after undergoing endobronchial ultrasound with biopsy of a large left infrahilar mass extending into the mediastinum, noted to be consistent with SCLC. The source of the hemoptysis was found to be from the invasion of SCLC into the left lower lobe pulmonary artery. She underwent clot removal with bronchoscopy followed by palliative radiotherapy for local control and hemostasis. She was responsive well to that treatment with improvement of hemoptysis and received inpatient chemotherapy with cisplatin and etoposide. Due to PICC line malposition, the line needed repositioning, but she was at significant risk for bleeding. As part of the workup for hemoptysis, her aPTT level was noted to be 72.8 on admission and continued to remain high during the hospitalization. She has no prior elevation of aPTT, personal clotting history, nor prior surgeries complicated with significant bleeding, though she has a sibling with a significant clotting disorder and a mother with SLE. She was found to have low factor VIII, but her aPTT did not completely correct during a 1:1 mixing study, which is not consistent with a factor deficiency. A Bethesda assay was then performed to detect factor VIII inhibitor, yielding 70 BU/ml, which was consistent with high levels of inhibitor. She was therefore diagnosed with acquired hemophilia A in the setting of SCLC. Treatment with prednisone was initiated with a notable improvement in aPTT levels. She continued to receive prednisone after discharge, with plans on transitioning to rituximab after completion of chemotherapy.

	Discussion: Acquired hemophilia as a paraneoplastic manifestation of small cell lung cancer is very rare but profoundly morbid and lethal when not detected. This case highlights the importance of maintaining a high index of suspicion for acquired bleeding disorders among cancer patients, particularly when laboratory work is noted to have an isolated elevation of aPTT. Diagnosis and combined management must be initiated early to improve patient outcomes and raise awareness of this very rare paraneoplastic syndrome.
Claire Embree Dr. Matthew Record Dr. Jairo Hernandez Dr. Reid Schlesinger Dr. Nadir Bhuiyan	*Dressed Down Dressler Syndrome* Case Presentation: A 62-year-old male presented to the emergency department with 4-5 days of dizziness, chest pain, and a cough, found to be hypotensive at 75/59. His past medical history was pertinent for hypertension, multiple strokes, tobacco use disorder, and a motorcycle accident in 2024 with extensive injuries, including a traumatic brain injury. On admission, the patient was afebrile and breathing comfortably on room air. He described the chest pain as sharp, persistent, and mild at rest, but worse with inspiration. He reported a dry cough at baseline, but felt it had worsened recently. Physical exam was pertinent for muffled heart sounds, clear lungs, no JVD, and no lower extremity edema. He had no known sick contacts. Initial troponins were mildly elevated at 37 with 2-hour reflex troponin down trending to 28. His EKG had T-wave abnormalities concerning inferior ischemia. Labs showed an elevated CRP at 237, a slight leukocytosis at 11.6, and a negative viral panel. A CT Chest Angiogram was significant for a new moderate right pericardial effusion. An urgent bedside transthoracic echocardiogram (TTE) demonstrated a mild, circumferential pericardial effusion with reduced ejection fracture of 56%, a severely enlarged right ventricle, and a/dyskinesis in the right ventricle. These findings were new from his last TTE in 7/2024. The decision was made to forgo a pericardiocentesis as his overall picture was not consistent with cardiac tamponade. Cardiology recommended a cardiac MRI due to concern for myocarditis and initiating treatment for pericarditis with colchicine and Aspirin. The cardiac MRI showed no evidence of myocarditis but found transmural enhancements of the left ventricle septum and the right ventricular wall. These findings raised concern for ischemia with possible Dressler Syndrome due to a silent right ventricle infarction. The patient was an overall poor historian, but did say he had some recent chest pain and chose not to seek medical care. The patient's hypotension improved with fluids and he was eventually discharged on the colchicine and aspirin with close follow-up in the cardiovascular clinic to further investigate the new right heart dilation and a/dyskinesia. Discussion: Dressler Syndrome, also known as Post Myocardial Infarction Syndrome, is a cardiac inflammatory condition that can occur anywhere from 2-6 weeks after a myocardial infarction. Typical

	presentation includes chest pain, pericarditis, pericardial effusions, and pleuritis. The mechanism for Dressler Syndrome is not completely understood, but is attributed to be an autoimmune response due to antigens released from damaged myocardial cells. As reperfusion during/after myocardial infarcts has greatly improved, Dressler Syndrome has become far less common. This is credited to a decrease in overall infarct size with a decrease in the body's exposure to myocardial antigens. Dressler Syndrome is now most commonly seen after large, silent infarctions, particularly inferior infarctions with right ventricular involvement, such as what we believe happened in this patient. Conclusion: In the end, we believe this case represents the new "classic" presentation of Dressler Syndrome from an untreated myocardial infarct. This is supported by the patient's worsened structural heart disease, EKG findings, cardiac MRI imaging, and clinical presentation.
Lauren Fang	*When the Colon is Clean but the Marrow is Mean: Clostridium Septicum as a Harbinger of Hematologic Malignancy* Background: Profound neutropenia in acute infection poses diagnostic challenges, potentially reflecting sepsis-related marrow suppression, drug effect, autoimmune destruction, or underlying hematologic disorder. Clostridium septicum and necrotizing soft tissue infections are rare but strongly associated with occult neoplasms, particularly colorectal adenocarcinoma and hematologic malignancies. Case Presentation: A 66-year-old man presented with fever (Tmax 103°F), weakness, fatigue, and myalgias. One month prior, he noted a small bite on his lower abdomen with surrounding rash but did not identify a tick. He lives on 2 wooded acres and spends considerable time outdoors. Admission labs showed WBC 1.0 K/μL, lymphocytes 0.38 K/μL, ANC 600/μL, hemoglobin 13.3 g/dL, and platelets 185 K/μL. Liver function tests revealed total bilirubin 1.6 mg/dL, direct bilirubin 0.9 mg/dL, AST 255 U/L, ALT 368 U/L, and lactate 4 mmol/L. Imaging demonstrated terminal ileum and cecal inflammation suggestive of typhlitis. Ultrasound and HIDA scans were negative for cholecystitis. Physical examination revealed a 3 cm erythematous rash on the left posterior thigh without fluctuance. Given leukopenia and potential tick exposure, Anaplasma/Ehrlichia infection was favored. He was started on cefepime 2 g IV, vancomycin 1,250 mg IV q12h, and doxycycline 100 mg PO BID. Despite antibiotics, the rash extended to his waistline and knee with progressive left lower extremity edema. CT femur demonstrated diffuse emphysema consistent with necrotizing fasciitis. Emergency surgical exploration revealed significant subcutaneous edema without murky dishwater fluid, patchy muscle hemorrhage with preserved

	contractility, and healthy intact fascia. Frozen sections were negative for necrosis. Antibiotics were changed to linezolid 600 mg IV/PO BID and piperacillin-tazobactam 3.375 g IV q6h. He underwent eight additional debridements with tissue cultures growing C. septicum. The patient demonstrated brisk but transient neutrophil recovery after G-CSF 480 mcg, then developed recurrent neutropenia and progressive anemia despite infection control. Granulocyte antibody testing was negative. While linezolid-associated anemia was considered, an underlying clonal process could not be excluded. Bone marrow biopsy revealed 40% cellularity with sideroblastic anemia (>15% ring sideroblasts) but no other dysplastic features. Next-generation sequencing showed TET2 mutation (9% VAF) and IDH1 variant of uncertain significance (50% VAF). Additional studies showed absolute reticulocyte count 74 K/μL, copper 152 μg/dL, vitamin B12 1,077 pg/mL, folate >20 ng/mL, and soluble transferrin receptor 2.8 mg/L. Monocyte distribution was inconsistent with chronic myelomonocytic leukemia. Colonoscopy was negative for polyps and malignancy. Discussion: Profound neutropenia may attenuate typical inflammatory responses and tissue necrosis, complicating clinical assessment. Additionally, unusual pathogens like C. septicum should prompt investigation for occult malignancy. While colorectal cancer is classically implicated, hematologic disorders must be considered when colonoscopy is unrevealing. This case illustrates the diagnostic complexity of distinguishing reactive neutropenia from myeloid neoplasms and precursor states such as clonal hematopoiesis of indeterminate potential and clonal cytopenia of undetermined significance (CCUS). Ultimately, the patient was diagnosed with CCUS. Conclusion: Clinicians should maintain high suspicion for underlying hematologic disorders when encountering unusual infections with significant neutropenia.
Yahye Farajuun Dr. Eric Lander	*Recreational Nitrous Oxide Use Leading to Severe Vitamin B12 Deficiency, Pancytopenia and Hemolytic Anemia* Introduction: B12 deficiency is commonly associated with pernicious anemia, gastric surgeries, and strict vegetarian or vegan diets. However, an increasing number of young, otherwise healthy patients are presenting with severe pancytopenia secondary to vitamin B12 deficiency caused by excessive recreational nitrous oxide use. Approximately 6% of individuals under age 60 and 20% of adults over the age 60 in the U.S. and UK1 are vitamin B12 deficient, compared to 42.2% of recreational nitrous oxide users, who have a circulating total vitamin B12 below the reference interval. Case Presentation: We present the case of a 24-year-old female with a past medical history of iron deficiency anemia, substance-use disorder, including fentanyl abuse and recreational nitrous oxide use. She

	presented to the emergency department with shortness of breath, weakness, fatigue, and recurrent episodes of fever, chills, myalgia, and lightheadedness. Vitals: BP 107/56 mmHg, HR 56 bpm, RR 18, T 98.4 F SpO2 100 % on room air. Physical exam: pale, ill-appearing female and no focal neurological deficit. Labs with WBC 3.2 (ANC 0.6, slight left shift), hemoglobin 3.5 on arrival (MCV 94) with prior baseline of 12.4 in 2021, platelet 15. Reticulocyte absolute 0.02. D-dimer 7.32, INR 1.4, PTT 26, fibrinogen 196. AST 56, ALT 20, total bilirubin 1.4, direct bilirubin 0.6, indirect 0.8, ALP 35. Cr 0.79. Infectious work up was negative for Treponema pallidum, strep pneumoniae, Legionella, mycoplasma, influenza, HIV, and RSV. CT head was unremarkable. CT chest, abdomen, and pelvis showed minimal early/developing infectious changes in the left upper lobe of the lung, mild periportal edema in the liver, and small amount of pelvic ascites. Patient was started on vancomycin and cefepime for suspected sepsis. She was admitted to the ICU and received 2 units PRBC and 1-unit platelets. Hemoglobin improved to 7.7 g/dL. Peripheral blood smear revealed severe normocytic anemia with circulating nuclear cells and rare schistocytes, marked absolute neutropenia with left-shifted neutrophils show toxic changes; marked absolute monocytopenia, eosinophilia, and basophilia; severe thrombocytopenia. Hemolysis labs with haptoglobin < 10 (30-200) and LDH 1800 (135-214). Inappropriately low reticulocyte counts 2.3. B12 level undetectably low. A bone marrow biopsy confirmed pancytopenia. Severe vitamin B12 deficiency was attributed to persistent Whippet (nitrous oxide) use, which led to pancytopenia and hemolytic anemia. Patient was treated with a vitamin B12 supplementation and discharged on hospital day 5. At one week follow-up with her primary care provider, pancytopenia had improved. Complete blood count showed hemoglobin 11.5 g/dL, WBC 5.6 and platelets of 155 Discussion: Excessive recreational nitrous oxide use significantly increases the risk of severe vitamin B12 deficiency, which can manifest as pancytopenia, hemolytic anemia, and exonal neuropathy. For many patients, cessation of recreational nitrous oxide uses and vitamin B12 supplementation are sufficient to reverse hematologic abnormalities. However, prolonged deficiency of vitamin B12 may result in irreversible neurological damage. Conclusion: This case underscores the importance of recognizing vitamin B12 deficiency in recreational nitrous oxide users to prevent hematological and long-term neurological complications.
Mannat Gill Shivansh Pandey Dr. Sophia Parente	*When Antibodies Go Off Script: A Case of IgA Nephropathy in Newly Diagnosed AIDS* Background: HIV-associated nephropathy (HIVAN) is a collapsing form of focal segmental glomerulosclerosis and the archetypal renal

	pathology in patients with HIV; however, the differential diagnosis for kidney injury in this population is broad. Case Presentation: We present a case of a young man with newly diagnosed AIDS whose severe kidney injury was due to IgA nephropathy (IgAN), illustrating how a definitive histological diagnosis is crucial to avoid clinical bias and guide appropriate therapy. In this case, we describe a 20-year-old man with no prior medical history who came into the ED with 3 weeks of worsening shortness of breath, cough, anasarca, and fatigue. He was found to have hypertensive emergency, acute oliguric renal failure, and acute hypoxic respiratory failure with associated anemia, thrombocytopenia, and severe hyperkalemia. On admission, his creatinine was noted to be 17 mg/dL with an unknown baseline along with a BUN of 108 mg/dL, potassium 6.4 mEq/L, nephrotic range proteinuria, and hematuria. Further lab testing revealed a new positive HIV test with a CD4 count of 53 indicating AIDS. Chest imaging showed ground glass opacities concerning for infection and bronchoscopy ultimately revealed PJP pneumonia. He was urgently started on hemodialysis, PJP pneumonia treatment, and antiretroviral therapy during admission. A renal biopsy was obtained and showed IgAN and treatment with high dose IV steroids was initiated. Further steroid-sparing agents were deferred due to concurrent HIV and PJP pneumonia. The patient was discharged on dialysis, antiretroviral therapy, and oral steroids with plans to initiate further steroid-sparing immunosuppression following treatment for PJP. Conclusions: Not only does this case illustrate that there are multiple etiologies of nephropathy that may be associated with HIV, but it also highlights how a definitive diagnosis via biopsy is paramount, as the management of IgAN (immunosuppression) is starkly different from that of HIVAN (cART-centric). Additionally, the importance of keeping an open differential and re-evaluating one's own biases, evident in this patient's lack of engagement in high-risk behaviors often associated with HIV.
Zachary Goldberg Dr. Grace Hagan Dr. Joshua Daum Dr. Kelsey Lynch	*Endocarditis Associated with Atrial Septal Occlusion Device* Background: Endocarditis with prosthetic valves is relatively common with surgical replacement of the valve being the ideal option for source control in most patients. There are limited options for source control in patients who are poor candidates for surgery and device anatomy limits endovascular options. Case Presentation: A 70-year-old man presented to the emergency department for uncontrolled back pain, altered mental status, tachycardia, and fever. He has a complex medical history including multiple strokes with patent foramen ovale (PFO) closure in 2022, inflammatory arthritis on chronic prednisone and tocilizumab, minimally displaced cervical fracture, right total shoulder arthroplasty, OSA, prostate cancer, diabetes, hypertension, and hyperlipidemia.

	The patient was admitted to the ICU with blood cultures growing Streptococcus mitis and CSF studies were significant for 1,903 total nucleated cells with neutrophilic predominance. He was started on Vancomycin and Ceftriaxone for bacteremia and suspected bacterial meningitis. A transthoracic echocardiogram (TTE) showed no evidence of endocarditis with plans for further workup pending clinical improvement. There was no evidence of infection seen on CT imaging of the maxillofacial region and 5 days after the TTE, a transesophageal echocardiogram (TEE) showed multiple masses adherent to the left atrial side of the septal occlusion device thought to be vegetation with possible superimposed thrombus. He developed new onset atrial fibrillation with rapid ventricular response following the procedure and was initially rate controlled with metoprolol before being placed on amiodarone overnight with conversion back to sinus rhythm. He continued to have bouts of atrial fibrillation during his course. He was started on heparin given the high likelihood of intracardiac thrombus, although there was no thrombus noted in the left atrial appendage. The following day, MRI brain demonstrated septic emboli to the left temporal lobe and ventriculitis. There were significant benefits of continuing anticoagulation given the evidence of intracardiac thrombi although there were clear risks of hemorrhagic conversion of the septic emboli. Anticoagulation with heparin was continued before being transitioned to a direct oral anticoagulant via shared decision making. He was continued on metoprolol for rate control of his intermittent atrial fibrillation. The patient was a poor candidate for surgical device removal via cardiothoracic surgery and interventional cardiology would be unable to attempt mechanical thrombectomy given the typical approach from the venous system through the atrial septum was blocked by the occlusion device. Given the lack of source control, this was treated similar to prosthetic valve endocarditis with IV antibiotics for 6 weeks followed by a minimum of 6 months of antibiotic suppression. The patient was recently discharged from inpatient rehabilitation and is receiving daily injections of ceftriaxone. Conclusions: This case is a rare example of an atrial septal occlusion device infection that was unable to be detected via TTE. There is clear utility in performing TEE in patients with cardiac devices when endocarditis is suspected. Shared decision making is important when discussing anticoagulation when there is high risk of concurrent emboli and hemorrhage.
Samarth Goyal	*Balancing the Mind, Breaking the Heart: Clozapine induced Myocarditis* Case Presentation: A 29-year-old patient, with past medical history of obstructive sleep apnea, schizophrenia, obsessive-compulsive disorder

	(OCD) and major depressive disorder (MDD) was admitted for evaluation of worsening psychosis characterized by paranoia in the setting of psychiatric medication noncompliance. He was found to be in decompensated schizophrenia and ongoing OCD. Given his inadequate response to multiple antipsychotic regimens, he was initiated on clozapine for treatment-resistant schizophrenia. Clozapine was started at 25 mg daily and gradually titrated. Approximately two weeks after initiation, when the dose had reached 300 mg daily, the patient developed non-radiating substernal chest pain associated with nausea and vomiting. He denied palpitations, shortness of breath, or diaphoresis. He was febrile (maximum temperature: 102°F) and mildly tachycardic. Laboratory evaluation revealed a significantly elevated C-reactive protein (CRP) level of 120 mg/L and an up-trending troponin, initially 104 ng/L and peaking at 280 ng/L. Electrocardiogram (ECG) was normal. Trans thoracic echocardiogram (TTE) showed a newly reduced ejection fraction (EF) to 20-25% with global hypokinesis. Infectious work up was largely negative. Given the clinical presentation and recent clozapine initiation, clozapine-induced myocarditis was suspected, and the medication was promptly discontinued. Cardiology was consulted and recommended cardiac magnetic resonance imaging (cMRI) for further evaluation. Due to logistical delays, cMRI was performed five days after clozapine discontinuation and showed normalization of LVEF to 60% with no regional wall motion abnormalities. CRP levels concurrently normalized, and the patient's chest pain and systemic symptoms resolved. Discussion: Clozapine is a well-known drug for treatment resistant schizophrenia. Unfortunately, it comes with several hematological, metabolic and cardiac side effects. One of the adverse effects, which is very rare but potentially life-threatening is clozapine induced myocarditis (CIM). CIM is defined as inflammation of the myocardium secondary to clozapine use. Due to early onset with clozapine use and presence of eosinophilic infiltrates in myocardium, this phenomenon is thought to be immune mediated though exact pathogenesis still remains unclear. Direct cardiotoxic effects with clozapine metabolites causing oxidative and metabolic stress has also been thought of. The incidence of CIM is the United States is as low as <0.1%. Most studies have demonstrated cardiac clinical manifestation within 2-8 weeks of initiation of this medication. The clinical presentation can vary widely, from ECG changes in individuals without symptoms to more subtle indicators of heart failure like fatigue. In more severe cases, patients may experience pronounced symptoms such as chest pain, shortness of breath, arrhythmias, or even sudden cardiac death. Notably, some individuals may remain entirely asymptomatic and show no obvious clinical signs. The wide range of clinical manifestations and often subtle, gradual onset of symptoms make it challenging to accurately assess the true incidence and prevalence of clozapine-induced myocarditis (CIM). Experts suggest gradual dose titration, serial EKGs and close monitoring of clinical

	symptoms to prevent / early diagnosis. Most reliable laboratory markers for diagnosis are CRP and troponin. Most effective treatment is to discontinue clozapine as soon as this phenomenon is suspected.
Nicholas Hable Dr. Sophie Harris Dr. Darin Carabenciov	*A Surprise in the Sinus: A Case of Lymphoma Presenting with Cavernous Sinus Syndrome* Background: Lymphoma, a broad category of malignancies involving lymphocytes, is further categorized into Hodgkin lymphoma (HL) and Non-Hodgkin lymphoma (NHL), with NHL estimated to make up roughly 90% of such cases. Various subtypes exist within NHL, one of which is mature B-cell neoplasms. One of the most common mature B-cell neoplasms is diffuse large B-cell lymphoma (DLBCL). DLBCL typically first presents as a mass in a lymph node, but can also impact the bone, testicles, intestines, spinal cord, or brain. We present a case below of DLBCL involving the bilateral cavernous sinuses. Case Presentation: A 65-year-old male experienced subacute onset progressive left-sided neck pain with dysarthria and numbness along the cheek. Medical assessment was notable for multiple cranial neuropathies affecting cranial nerves five and twelve. MRI brain with contrast identified multifocal T2 hyperintensities along the skull base and foramen magnum, with enhancement of the hypoglossal canal. MRI spine visualized multifocal enhancement and edema in the cervical and thoracic skeleton. PET scan revealed FDG avidity in the left neck cervical chain, supraclavicular lymph nodes, and osseous lesions. Biopsies were performed and ultimately thought to be non-diagnostic, although there was some reported suspicion for B-cell lymphoma in a right iliac crest biopsy. A short course of methylprednisolone resulted in profound symptomatic improvement, but he did not receive any subsequent treatment and symptoms recurred a few months later. Multiple cranial neuropathies re-emerged and he was reassessed after developing diplopia, facial numbness, and progressive headaches. Clinical examination noted CN III, IV, and VI palsy, right eye ptosis, decreased sensation in the right V1-V2 distribution, vertical diplopia with eye opening, and skew deviation. MRI brain revealed pachymeningeal thickening and enhancing soft tissue masses involving the cavernous sinuses bilaterally with extension into the sella on the left and the superior aspect of Meckel's cave on the right. A PET scan noted significant FDG avidity in the cavernous sinus and subtle FDG avidity in the testicle. He underwent a transsphenoidal cavernous sinus biopsy which revealed CD20+ germinal center diffuse large B-cell lymphoma. He was started on a regimen of rituximab, methotrexate, and temozolomide. However, given concurrent staph aureus endocarditis, temozolomide was deferred while he cleared the infection. He also received a three-day steroid burst, subsequently transitioning to an oral steroid taper. Conclusion: This case highlights a manifestation of diffuse large B-cell lymphoma resulting in cavernous sinus syndrome. Although this

	presentation is rare it should be considered as part of the differential diagnosis when encountering patients with multiple cranial neuropathies in clinical practice.
Dana Hamadi Dr. Laura LaFave Dr. Sreekant Avula	*When Weight Loss Comes at a Cost: A Case of Adrenal Insufficiency Linked to Ozempic* Background: Semaglutide, a glucagon-like peptide-1 (GLP-1) receptor agonist, is widely used for type 2 diabetes and obesity. It is generally well tolerated, with the most common adverse effects being gastrointestinal. Preclinical studies suggest GLP-1 analogs may transiently activate the hypothalamic–pituitary–adrenal (HPA) axis. However, no causal relationship between semaglutide and adrenal insufficiency has been established in the literature. Case Presentation: A 54-year-old woman with a past medical history of type 2 diabetes, hypothyroidism, and rheumatoid arthritis, presented with 3 days of persistent nausea, vomiting, watery diarrhea, chills, weakness, and abdominal pain. Symptoms began one day after an increase in semaglutide dose from 0.25 mg to 0.5 mg. Despite aggressive resuscitation, she developed worsening hypotension, hypovolemia, hypoalbuminemia, and multiorgan dysfunction. Infectious workup, abdominal imaging, and labs were unrevealing. A morning cortisol level was low at 2.6 µg/dL, consistent with acute adrenal insufficiency. Unfortunately, she was started on stress dose steroids and ACTH level was drawn post-steroids and therefore, could not be interpreted. Despite treatment, her condition deteriorated with respiratory failure, worsening renal function, and eventual death from multiorgan failure. Discussion: This case raises concern for a possible association between semaglutide and acute adrenal insufficiency, particularly following dose escalation. While adrenal crises have been previously described in patients with autoimmune comorbidities, including hypothyroidism and rheumatoid arthritis, this patient's symptoms appeared temporally related to semaglutide titration and occurred in the absence of infection, adrenal hemorrhage, or other common precipitants. Furthermore, the presence of autoimmune comorbidities may have rendered her adrenal reserve more vulnerable to pharmacologic or metabolic stressors. Though causality cannot be definitively established, this case highlights the importance of maintaining a high index of suspicion for adrenal insufficiency in patients presenting with unexplained shock and multiorgan dysfunction while on GLP-1 receptor agonists. Conclusion: This case illustrates a possible association between semaglutide and acute adrenal insufficiency, particularly following dose escalation. Further research is warranted to evaluate for any association between semaglutide and acute adrenal insufficiency.

Mikako Harata Dr. Ying Jin	*The Constellation Effect: A Rare First Presentation of SLE with APS, Lupus Nephritis, Libman-Sacks Endocarditis, and Bilateral Optic Perineuritis* Case Presentation: We present a complex case of a 27-year-old female with a past medical history of idiopathic intracranial hypertension, who was hospitalized with acute ischemic stroke and subsequently diagnosed with antiphospholipid syndrome (APLS) secondary to systemic lupus erythematosus (SLE) and biopsy-proven lupus nephritis. This case highlights a rare and multifaceted initial presentation of autoimmune disease involving neurologic, ophthalmologic, renal, hematologic, and cardiac systems. The patient initially presented to the emergency department with acute-onset left upper extremity weakness, and 2-week history of quotidian fevers. Imaging revealed multiple acute and subacute cerebral infarcts, as well as a splenic infarct and bilateral optic perineuritis. Laboratory evaluation was notable for thrombocytopenia, acute on chronic anemia, elevated inflammatory markers, and acute renal injury. Urinalysis showed significant proteinuria, hematuria, and pyuria. Serologic workup demonstrated low complement levels and multiple positive autoantibodies, including anti-dsDNA, anti-Sm, RNP, SSA, SSB, chromatin, TPO, and phosphatidylserine/prothrombin IgG antibodies. She also had a markedly elevated pANCA titer and a positive lupus anticoagulant panel, raising concern for concurrent APS. Transesophageal echocardiography revealed an 8x10 mm globular thickening of the posterior mitral valve leaflet. Though not characteristic of a single specific etiology, the absence of infectious signs or symptoms made non-infectious Libman-Sacks endocarditis the most likely diagnosis, though healed endocarditis and papillary fibroelastoma remained in the differential. A renal biopsy performed revealed class IV (diffuse proliferative) and class V (membranous) lupus nephritis, with a modified NIH activity index of 12/24 and chronicity index of 2/12. These findings confirmed active lupus nephritis with limited chronic damage. . Given the presence of arterial thrombotic events and serologic evidence consistent with APLS, the patient was initiated on therapeutic anticoagulation with warfarin. For treatment of perineuritis, Libman Sacks endocarditis, and lupus nephritis, she received high-dose intravenous methylprednisolone during hospitalization, followed by a tapering course of corticosteroids in the outpatient setting. Long-term immunosuppressive therapy was initiated with hydroxychloroquine and mycophenolate mofetil to target SLE and prevent further renal and systemic disease progression. Discussion: This case demonstrates the potential for SLE and its comorbidities to present with severe, multi-organ manifestations in previously undiagnosed individuals. The initial presentation with multi-territory brain and splenic infarctions, and cardiac valvular

	abnormality was highly suggestive of APLS and Libman-Sacks endocarditis, both of which are serious complications associated with SLE. Early recognition and a multidisciplinary approach involving neurology, rheumatology, nephrology, hematology, ophthalmology, and cardiology were critical in initiating appropriate therapy and preventing further complications. Continued close follow-up is necessary for monitoring therapeutic response, anticoagulation management, renal function, and cardiac surveillance.
Kiyan Heybati Dr. Sarah Abernathy Dr. Frank Cossio Bernabe Dr. Louise Asleson Dr. Giuseppe Maiocco Dr. Lindsay Howlett Dr. Issac Duggan Dr. David Rosenman	*TB or not TB: A Case of Incidental, Bilateral Cavitary Lung Lesions* Background: Hospitalists often encounter incidental imaging findings with varying levels of significance. Among these are cavitary lesions, which arise from a wide range of underlying conditions and are typically defined as a lucent area within a consolidation, mass, or nodule with walls greater than 4 mm in thickness. Case Presentation: A 33-year-old male presented to an outside ED with a large volume hemorrhage after sustaining a right arm laceration from a fall onto a broken light bulb. Past medical history included treated hepatitis C, nicotine dependence, and remote IV drug use (10 years sober). Initial hemoglobin was 5.5 g/dL with leukopenia (3.2 x 10^9/L) and thrombocytopenia (71 x 10^9/L), attributable to considerable blood loss. He received 2 units of packed red blood cells and was subsequently transferred with a tourniquet in place. CT angiogram of the right arm demonstrated a superficial soft-tissue laceration without arterial bleeding. Incidentally, imaging revealed cavitary lesions and nodular consolidations in the bilateral lung apices, raising concern for an infectious process. Per guidance from Infectious Diseases (ID), he was admitted for the rule-out of tuberculosis (TB). Further history included a several-year history of intermittent dry cough with mild improvement after switching from cigarettes to e-cigarettes approximately one year prior, as well as several months of night sweats he attributed to his bedding material. He denied hemoptysis, fevers, chills, dyspnea, dysuria, rashes, or other systemic symptoms. He had lived in Alaska for 23 years and was routinely tested for TB in grade school; he believed he received the BCG vaccine due to the high local prevalence. Social history included employment in construction and mining, travel to Arizona and Texas 13 years ago, and hunting of bear, moose, elk, rabbit, and waterfowl. In consultation with ID and Pulmonary Medicine, a broad infectious and inflammatory workup was pursued. Due to the inability to produce sputum, bronchoalveolar lavage (BAL) was performed with a negative acid-fast smear and mycobacterium tuberculosis PCR. Testing for histoplasmosis, blastomycosis, HIV, and vasculitis was also negative. Therefore, he was discharged with an ID and pulmonary follow-up. His BAL fungal culture later returned positive for coccidioidomycosis, while his mycobacterium tuberculosis culture was negative. Given his intermittent dry cough and night sweats, outpatient ID recommended a

	6-week course of fluconazole 400 mg daily followed by interval CT chest imaging. Conclusions: While a history of residence in high-prevalence regions (e.g., Alaska) may increase suspicion for TB, this case underscores the importance of maintaining a broad differential and avoiding diagnostic anchoring. Longstanding coccidioidomycosis infection can also present with cavitary pulmonary lesions and minimal symptoms, which may be overlooked and attributed to other causes. A systematic approach, with a thorough exposure history including remote travel, appropriate microbiologic and serologic testing, and multidisciplinary consultation, remains important for accurate diagnosis and management.
Nathan Holthaus Dr. Amy Holbrook	*Hereditary Pyoderma Gangrenosum Masquerading as a Necrotic Soft Tissue Infection* Background: Pyoderma gangrenosum (PG) is a rare inflammatory disorder that causes rapidly progressive skin ulcers, classically accompanied by a violaceous border. It is commonly associated with underlying autoimmune conditions such as inflammatory bowel disease, arthritis, and hematologic conditions. The diagnosis of PG is clinical and relies heavily on the exclusion of other disorders that closely resemble its presentation. Case Presentation: An 84-year-old female presented to the emergency department with a two-day history of right foot pain, swelling, and erythema. She was febrile to 102.1 °F with a notable leukocytosis of 17.4. A wound was present on the dorsum of her foot with concern for fluctuance. The patient underwent a bedside incision and drainage in the ED with no purulence expressed from the wound. Cultures were obtained during the procedure. She was started on IV cefazolin and admitted to the hospital for treatment of presumed right foot cellulitis. Throughout her initial days of hospitalization, the patient clinically decompensated. She developed worsening pain and skin bullae overlaying her right lateral foot, raising concern for a necrotic soft tissue infection. She experienced ongoing fevers as well as persistent leukocytosis. An MRI of her right foot was negative for osteomyelitis and demonstrated soft tissue edema with skin thickening. Infectious disease and podiatry were consulted, who performed a bedside debridement and skin biopsy. Despite escalation to broad-spectrum antibiotics with IV vancomycin and piperacillin-tazobactam, the patient's lower extremity continued to worsen. She developed an acute kidney injury in the setting of sepsis and was found to have significantly elevated ESR/CRP levels, thus prompting a rheumatology consultation. A soft tissue CT again demonstrated diffuse edema of the right lower extremity without the presence of gas. Her autoimmune laboratory workup was unrevealing aside from an atypical p-ANCA,

	suspected to be a false-positive. Given her prolonged clinical course without meaningful improvement following completion of IV antibiotics, the patient was started on high-dose methylprednisolone. In obtaining additional family history, multiple first-degree relatives had been diagnosed with pyoderma gangrenosum previously. Following the initiation of steroids, the patient began to improve clinically. The trend of her elevated CRP/ESR quickly returned to normal and her leukocytosis resolved. She was discharged from the hospital with a plan for a prolonged taper of prednisone. Conclusion: This case illustrates the importance of a detailed and thorough physical exam. PG may not be included in the initial differential diagnosis given its rarity of presentation, especially when it can resemble a necrotizing soft tissue infection or cellulitis in its early stages. Attention as to whether the patient is clinically improving with the current treatment regimen is of the utmost importance. Failure to demonstrate a positive response raises the question of treatment failure or whether the presumed diagnosis is correct. This case also highlights the importance of obtaining a detailed family history, as a hereditary predisposition has been reported in some occurrences of PG.
Hannah Irwin	*Under Pressure: Critical Aortic Stenosis in a Bicuspid Valve* Case Presentation: A 69-year-old woman presented for a routine echocardiogram for monitoring of a bicuspid aortic valve, previously known to have moderate aortic stenosis. She was found to have critical aortic stenosis with a mean gradient of 118mmHg. She reported minimal symptoms with some shortness of breath when walking up a full two flights of stairs. She denied pain and was still doing her normal activities. With her critical aortic stenosis, the patient also had aortic root dilatation and a large pericardial effusion. Cardiothoracic surgery was consulted, and she underwent a Bentall procedure with mechanical aortic valve replacement and drainage of the pericardial effusion. The post-operative course was complicated by atrial fibrillation with rapid ventricular response that improved with amiodarone. An extensive work-up was done of the pericardial effusion which was ultimately determined to be post-viral in origin. Following the procedure, she had a trace effusion with no recurrence since then. Conclusion: In patients with bicuspid aortic valves, ongoing surveillance is essential. This includes careful attention to the physical exam, as patients can be asymptomatic, or minimally symptomatic, despite significant stenosis. Evolution of valvular disease and aortic root dilatation can occur without symptoms and routine echocardiogram can help identify when intervention is needed.
Yazan Jabban Dr. Matthew Pinkleton	*Hypercalcemia as the Initial Presentation for Pneumocystis Jirovecii Pneumonia in a Kidney Transplant Recipient*

	Background: Pneumocystis jirovecii pneumonia (PJP) is a serious complication among solid organ transplant patients, with an incidence around 5-15% and mortality up to 38%. While respiratory symptoms are typically prominent at presentation, hypercalcemia as an initial manifestation is rare, and may precede pulmonary findings, leading to diagnostic delay. Case Presentation: We present a 71-year-old female with past medical history of kidney transplant (2020), polycystic kidney and liver disease, hypothyroidism, and hypertension, presented with a month-long history of fatigue, weakness, constipation, anorexia and intermittent dry cough, with worsening symptoms the week prior to presentation. Physical exam was unremarkable. Initial labs were notable for significant hypercalcemia at 16 mg/dl. Patient was admitted for further workup and started on IV fluids and calcitonin. Workup revealed suppressed PTH, 25-OH VitD and PTHrP were normal, but 1-25-OH VitD was markedly elevated at 257 pg/ml. Workup for multiple myeloma was unrevealing. Initial CXR and CT chest were unremarkable. Due to worsening of her dry cough, we decided to proceed with infectious workup which came back positive for sputum PJP PCR. Infectious diseases were consulted, who deferred treatment given the lack of imaging findings. Three days later, Beta-D-glucan returned positive, and repeated CT chest showed new ground glass opacities, consistent with PJP infection. She was then started on TMP-SMX. Despite initial treatment with IV fluids and calcitonin, the patient remained hypercalcemic, with a further rise in calcium levels after the effect of calcitonin subsided, prompting treatment with pamidronate. She improved clinically and was discharged on oral TMP-SMX with close follow-up in the transplant clinic. Of note, patient was started on belatacept one month prior to admission (was previously cyclosporine, which was tapered off due to recent biopsy showed hyalinosis and possible contribution to her hypertension). Discussion: Hypercalcemia as the initial presentation is exceptionally rare and challenging, especially in the absence of findings on chest imaging. Only few case reports and small case series have reported this atypical presentation. In addition, few cohorts and case reports have documented an association between the conversion from calcineurin inhibitor and increased risk of PJP, with some studies noting 1.6-4.3% increase in risk with belatacept compared to calcineurin inhibitors. Conclusion: In immunocompromised patients, PJP can present atypically, and hypercalcemia may precede respiratory symptoms or radiographic changes. Internists should maintain a high index of suspicion for PJP in such patients, even in the absence of classic pulmonary findings, to prevent delays in diagnosis and treatment.

Hisard Jack Dr. Jesse Abelson Dr. Emma Leirdahl	*Two Chambers, One Threat: The Biatrial Journey of IPDE* Background: Impending paradoxical embolism (IPDE) is a rare but life-threatening condition in which a venous thrombus straddles an intracardiac shunt—most commonly a patent foramen ovale (PFO)—with the potential to cross into systemic circulation and cause devastating consequences. This phenomenon poses risks to both pulmonary and systemic circulation, often presenting with overlapping symptoms that complicate diagnosis. While the gold standard for diagnosing IPDE is transesophageal echocardiography (TEE), a few case reports have described diagnosis via transthoracic echocardiography (TTE). This case describes a patient diagnosed with IPDE via TTE who underwent emergency AngioVac suction thrombectomy. Case Presentation: An 85-year-old man with hypertension and a history of squamous cell carcinoma presented with sudden-onset chest pain radiating to the upper back. On initial evaluation, his heart rate was 105 bpm and oxygen saturation was 89% on room air. High-sensitivity troponin levels were elevated, while pro-BNP was normal. The patient underwent CT pulmonary angiography, which revealed multiple bilateral occlusive pulmonary emboli without right heart strain. A heparin infusion was started. A lower extremity ultrasound revealed an acute right peroneal vein deep vein thrombosis. Given the elevated troponin and a Simplified Pulmonary Embolism Severity Index (SPESI) score of 2 (age > 80 and oxygen saturation < 90%), the patient was classified as having submassive PE with high-risk features. POCUS performed by the admitting internal medicine team showed possible right heart strain but no evidence of a clot in transit or IPDE. Due to ongoing concern for right heart strain, a formal TTE was ordered. The echocardiogram revealed a clot in the right atrium measuring 8 mm × 70 mm, spanning the tricuspid valve into the right ventricle. A similar large mass (7 mm × 50 mm) was seen in the left atrium, raising concern for a clot traversing a PFO. Interventional cardiology and cardiothoracic surgery teams were promptly consulted. The patient was emergently taken for a percutaneous suction thrombectomy. Given his high risk of decompensation, he was first cannulated for ECMO and subsequently underwent successful thrombectomy. A multidisciplinary discussion was held to evaluate the need for IVC filter placement, PFO closure, or lifelong anticoagulation. Lifelong anticoagulation was ultimately chosen. The patient remained hemodynamically stable and was discharged on apixaban. A TTE performed two months after hospitalization showed no residual thrombi, and the patient made a full recovery.

	Discussion: Impending paradoxical embolism is rare and carries a high 30-day mortality rate, estimated at ~18%, though likely underdiagnosed. It is typically identified following an initial PE diagnosis. Interestingly, this is the first published case of V-V ECMO cannulation for an IPDE thrombectomy. Given the rarity of IPDE, no guidelines exist regarding when IPDE should be considered, or risk factors for developing IPDE. Current guidelines do not recommend routine TTE for hemodynamically stable PE patients; however, they do note its importance for risk-stratification. Conclusion: This case highlights the importance of POCUS and TTE in the risk stratification and management of PE and IPDE, especially in patients with hemodynamic instability or signs of right heart strain.
Elizabeth Jensen Dr. Elizabeth Jensen Dr. Amy Holbrook	*A Twitchy Kind of Fever: Cefepime-Induced Myoclonus* Background: Neurotoxicity is a known potential side effect of cephalosporin therapy that can include altered level of consciousness, disorientation, seizure activity, and myoclonic movements. Known risk factors include high dose antibiotic therapy, renal dysfunction or preexisting neurologic disease. Case: A 47-year-old female with malignant pancreatic cancer with metastases to peritoneum and malignant ascites and PD catheter in place presented to the emergency department with 3 days of fever >101.7, hypotension, tachycardia and neutropenia (absolute neutrophil count of 1.2). She was admitted to the hospital for high-risk neutropenic fever of unknown etiology after starting a new chemotherapy regimen. She was started on empiric IV cefepime and vancomycin for her fever and morphine for pain control. Antibiotics were continued from hospital day 0-7 without source identification. During this time, the hospital course was complicated by AKI from ATN with a Cr 1.34 on hospital day 7, from a baseline of ~0.4 on presentation. Also on hospital day seven, the patient was noted to have myoclonic movements of upper and lower extremities, as well as fever to 101.3F. No metabolic causes for myoclonic movements were identified. Potential mediation culprits included cefepime and morphine. Antibiotics were changed to meropenem on hospital day 8 due to ongoing fevers. She was continued on morphine for pain control. Myoclonic movements slowly improved and resolved completely over the next 4 days after discontinuing cephalosporin antibiotic and with improvement in kidney function. She completed a total of 14 days of antibiotics for neutropenic fever of unknown etiology. She was discharged to home on oral levofloxacin while on chemotherapy to treat potential peritonitis while peritoneal catheter in place. Conclusion: This case demonstrates one of several neurotoxic symptoms that can be seen with cephalosporin treatment, which can be

	challenging to recognize due to the variety of presenting symptoms. There are several risk factors that increase the incidence of these complications, including AKI, high dose therapy and as well as potential polypharmacy with morphine, as seen in our patient. It is important to consider renally dosing antibiotics, especially in patients with fluctuating renal function after initial dose selection.
Muhammed Khalifa	*Topical Timolol-Induced Lupus Erythematosus: An Extremely Rare Case Emphasizing the Importance of Patient Perspective* Background: Drug-induced lupus erythematosus (DILE) is a lupus-like syndrome triggered by recent drug exposure and characterized by cutaneous and mild systemic manifestations with autoantibody positivity. Systemic medications such as hydralazine and procainamide are well-studied causes, whereas reports of DILE following topical agents remain scarce in the literature. We present a case of DILE secondary to topical timolol. Case Presentation: A 43-year-old male with a medical history of open-angle glaucoma and diabetes mellitus type 2 developed a 6-week history of low-grade fever, arthritis and a photosensitive annular erythematous rash. Laboratory evaluation revealed elevated CRP and ESR plus positive ANA, anti-histone antibodies and anti-Ro/SSA. A presumptive diagnosis of systemic lupus erythematosus (SLE) was made, and hydroxychloroquine (HCQ) plus NSAIDs were prescribed. Despite multiple discussions regarding the extremely low likelihood of systemic absorption, the patient firmly believed his symptoms were linked to the timolol eye drops, noting they began around 10 days after initiation of the timolol. He decided to discontinue both HCQ and timolol, and within two weeks, all his symptoms resolved and repeated labs normalized. He has remained without symptoms or lab abnormalities for the past year without timolol. These findings strongly support a diagnosis of DILE rather than SLE. Discussion: Although systemic absorption from topical agents is often considered negligible, this case demonstrates their potential to trigger even systemic autoimmune reactions. There is no definitive test for DILE, however, the temporal association, positive autoimmune serologies, complete resolution of symptoms and lab abnormalities after cessation of the offending agent and sustained remission support the diagnosis of DILE. Conclusion: This extremely rare case highlights the importance of listening attentively to patient concerns as the correct diagnosis was ultimately guided by his own observations. It also serves as an important reminder for clinicians to consider drug-induced etiologies of systemic manifestations even for topical agents, before establishing a diagnosis of a chronic condition. References: Bilewicz-Stebel M, Miziołek B, Bergler-Czop B, Stańkowska A. Drug-induced Subacute Cutaneous Lupus

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Connor Krumm Nolan Mclaughlin	*A Shocking Turn of Events* Background: Urinary tract infections are the fourteenth most common cause for hospital admissions in the United States resulting in over half a million admissions annually. Although the diagnosis and management of bacterial and fungal urinary tract infections are viewed as routine, maintaining a detailed knowledge regarding potential side effects of treatments is imperative to prevent adverse outcomes. Here we describe the case of a patient who developed a life-threatening cardiac side effect during treatment of a urinary tract infection. Case Presentation: A 77-year-old female patient presented to the emergency room from her nursing home for altered mental status and dysuria. Urine analysis obtained in the emergency department was concerning for urinary tract infection and urine cultures ultimately resulted positive for Candida glabrata. Due to the patient's prior cardiac history involving ventricular tachycardia and a previously implanted cardiac resynchronization device with a pacemaker, cardiac screening was obtained prior to starting anti-fungal therapy. At the time of anti-fungal therapy initiation, her QTc was 422 and she was started on fluconazole in addition to ongoing cardiac monitoring. Four nights after her initial admission, the patient endorsed a sensation of "being tasered." Review of her telemetry at this time was notable for Torsades de pointes. She was subsequently given 150mg of amiodarone, 4g of magnesium, and 100mg of lidocaine after which her arrhythmia resolved. Interrogation of her implanted cardiac device after the event was notable for ninety shocks. Manual calculation of her QTc after the events was found to be 521. She was subsequently transferred to the cardiac intensive care unit for amiodarone loading and further monitoring. She was ultimately transferred to medicine and was discharged without any further events. Conclusion: This case illustrates the importance of understanding medication side effects, which patients who require ongoing monitoring, and understanding of how to appropriately manage life-threatening situations when they arise. Although the incidence of Torsades de pointes is rare, the actions of the medical team in this case resulted in rapid identification and treatment preventing a possible cardiac arrest.
Alison LeVahn Dr. Peter Lund	*The Rule of 2's: A Surprising Case of Meckel's Diverticulum*

	Background: Meckel's diverticulum, a pediatric pearl for the internist. The incomplete obliteration of the omphalomesenteric duct (connecting the yolk sac to the embryo) at approximately 7 weeks gestation results in the most common congenital abnormality of the gastrointestinal tract, a meckel's diverticulum. Memorized by generations of medical students as the "rule of 2's": 2% of the population, 2 inches long, 2 feet from the ileocecal valve, 2 times as common in males, and 2 types of ectopic tissue (gastric or pancreatic). Complications in order of frequency include obstruction, diverticulitis, perforation, and hemorrhage. Case Presentation: A 39-year-old male with no significant past medical history presented to the emergency department with one week of intermittent bloody stools. He endorsed occasional ibuprofen use, occasional alcohol use and no steroid use. Review of systems was notable only for lightheadedness. He had presented to the emergency department two days earlier with notable hemoglobin of 12.9 and positive stool guaiac testing. He was instructed to follow up with gastroenterology. Increasing bloody stools prompted re-evaluation. On admission, he was vitally stable. The abdomen was soft and non-tender. His hemoglobin was 9.4. CT of the abdomen and pelvis was normal. He had ongoing hematochezia with hemoglobin drifting to 7.9 over 24 hours. Gastroenterology was consulted and he underwent colonoscopy and EGD. Colonoscopy revealed non-bleeding internal hemorrhoids, but no source of his hematochezia. No acute abnormality was found on EGD. Serial hemoglobin checks continued to decline, requiring a total of 6 units of transfused red blood cells over 3 days. Given ongoing evidence of bleeding with negative endoscopic evaluation and age, a nuclear medicine Meckel's scan was obtained which revealed abnormal radiotracer accumulation in the right lower quadrant consistent with a Meckel's diverticulum. General surgery was subsequently consulted, and the patient underwent surgical exploration. Intraoperatively, an easily identifiable diverticulum adherent in the mesentery adjacent to the bowel was identified and resected. Following surgery, serial hemoglobin checks remained stable, and his symptoms resolved. He was discharged home with general surgery follow up. Pathology ultimately confirmed the diagnosis of Meckel's diverticulum with gastric oxyntic type mucosa consistent with gastric heterotopia involving Meckel's diverticulum on pathology. Conclusion: This case illustrates the need to remain cognizant of unusual etiologies of gastrointestinal bleeding in younger to middle aged adults and to consider the potential of a Meckel's diverticulum. Clinicians must remain aware of the difficulty diagnosing Meckel's diverticulum especially in adults where the preferred imaging modality Technetium-99m scintigraphy is less sensitive and specific than in the pediatric population (sensitivity of 80%-90%, a specificity of 95% and

	an accuracy of 90% in children, in adults it is less reliable with a sensitivity of 62.5%, a specificity of 9% and an accuracy of 46%.).
Christine Loo	*Unusual Overlap of Ankylosing Spondylitis, Peri Orbital Myositis, and Cogan's Syndrome* Background: Cogan's syndrome is a rare systemic inflammatory disease characterized by interstitial keratitis and audiovestibular dysfunction, with potential systemic involvement. While atypical ocular manifestations of spondyloarthropathy have been described, the coexistence of Cogan's syndrome and spondyloarthritis is uncommon. Case Presentation: We present the case of a 33-year-old man with congenital sensorineural hearing loss who was admitted with recurrent eye pain in the setting of recently diagnosed orbital inflammatory syndrome. His symptoms improved partially with high-dose intravenous steroids but relapsed with tapering. Initial orbital MRI demonstrated inflammation of the left inferior rectus muscle but did not reveal other signs of vasculitis or CNS involvement. Rheumatologic work-up showed HLA-B27 positivity but was otherwise unremarkable. Imaging confirmed sacroiliitis, while repeat orbital MRI was negative for active inflammation. Given the constellation of findings—sensorineural hearing loss, ocular inflammation, and HLA-B27–positive sacroiliitis—the diagnosis of Cogan's syndrome overlapping with spondyloarthritis was considered. Although vestibular symptoms were absent, atypical cases have been described in which auditory and ocular manifestations present more than two years apart. The patient was treated with high-dose glucocorticoids and infliximab, with transient hearing improvement during IV steroid administration. Conclusion: This case highlights the diagnostic challenges of atypical Cogan's syndrome and emphasizes the importance of considering overlapping autoimmune diseases in patients with ocular inflammation and systemic features.
Brianna Lupo Dr. Leeore Levinstein	*Beyond the Bone Marrow: Extramedullary B-cell Acute Lymphoblastic Leukemia as a Mimic of Cholecystitis and Heart Failure* Introduction: B-cell acute lymphoblastic leukemia (B-ALL) is a malignancy of B lymphocytes most common in adults over the age of 60 or in pediatric patients. Typical clinical presentation involves fatigue, bone pain, fevers, night sweats, bleeding, or infection. Disease involvement is often limited within the bone marrow, though extramedullary disease including the visceral organs is possible. This case describes a patient who presented with shortness of breath and abdominal pain who was found to have visceral organ infiltration of B-ALL.

	Case Presentation: A 65-year-old female presented to the emergency department (ED) for dyspnea on exertion and postprandial right upper quadrant (RUQ) pain. Medical history was significant for B-ALL status post bone marrow transplant. In the ED, she was normotensive and mildly hypoxemic to 90% on room air. She was in rate-controlled atrial fibrillation. Exam was notable for bilateral lower extremity edema. Pro-brain natriuretic peptide was elevated to 48,803, high-sensitivity troponin was elevated but stable at 101 then 104, hepatic panel exhibited mildly elevated ALT of 39, AST of 41, and alkaline phosphatase of 127. Other labs were within recent baseline. She was admitted to the hospital for suspected acute decompensated heart failure and underwent diuresis on admission. Echocardiography showed new severely increased left ventricular wall and septal thickening compared to six months prior. Cardiac magnetic resonance imaging was planned to further characterize the new ventricular hypertrophy. Given ongoing postprandial abdominal pain and elevation in liver enzymes, RUQ ultrasound was completed and revealed gallbladder wall thickening with multiple gallstones and pericholecystic fluid, consistent with acute cholecystitis. General surgery planned for laparoscopic cholecystectomy. Unfortunately, prior to further evaluation or intervention, the patient unexpectedly developed pulseless electrical activity then cardiac arrest and expired. Autopsy later revealed diffuse cardiac B-ALL involvement and transmural B-ALL involvement of the gallbladder wall. Interestingly, bone marrow was negative for leukemia. Discussion: Cholecystitis and acute decompensated heart failure account for hundreds of thousands of hospital admissions annually in the United States. Internists are adept at diagnosing and treating these conditions. Rarely, these conditions are secondary to atypical infiltrative diseases such as B-ALL, which may not be discernible by standard imaging modalities such as ultrasound. To more promptly recognize this, patients with history of leukemia or with hematologic cell line abnormalities should be considered for extramedullary disease when presenting with single or multi-organ system diseases. Conclusion: While rare, this case highlights the importance of early identification of extramedullary B-ALL, as delay in recognition or treatment can be devastating.
Carolyn Lussenhop	*Lithium Lethargy: Slowing More Than Mood* Introduction: Lithium has been used as a mood stabilizing agent for psychiatric conditions for over a century. Its exact mechanism of action is poorly understood and its therapeutic index is narrow, which lends to a large proportion of patients on lithium therapy experiencing at least one episode of toxicity during treatment. Lithium toxicity can have wide ranging manifestations; primarily neurologic, renal, and gastrointestinal abnormalities. Rarely, it can have cardiotoxic effects.

	Case Presentation: A 60-year-old male with past medical history of bipolar disorder, stage 3 chronic kidney disease, type II diabetes, hypertension, hyperlipidemia, obstructive sleep apnea, chronic anemia, and prior stroke presented to with myalgias, global weakness, and decreased urination. He also had a history of several prior syncopal episodes. After his fourth syncopal event, he was hospitalized for telemetry monitoring, which was significant for sinus bradycardia. No dysrhythmias, heart block, or sinus pauses were observed. His metoprolol was discontinued and he underwent placement of an implantable loop recorder (ILR). Following ILR placement he did not have any recurrent episodes of syncope or observed significant brady or tachyarrhythmias, though he was lost to follow up with last ILR check about one year after placement. Upon presentation to the Emergency Department, he was bradycardic with heart rate in the 30s, but normotensive. Electrocardiogram demonstrated junctional bradycardia. Labs on presentation were notable for sodium 132, potassium 5.4, creatinine 3.33 (baseline 2.5 - 2.8), magnesium 3.4. Lithium level was elevated at 1.79 (therapeutic range is 0.6-1.2 mmol/L). It was felt that his presenting symptoms could be due to his arrhythmia, and the patient was transferred to a hospital with pacemaker implantation capabilities. On admission, lithium level recheck was confirmed to be 1.7. After consultation with Electrophysiology and Nephrology, emergent hemodialysis was initiated to treat presumed lithium induced cardiac toxicity. About two hours into his first hemodialysis run, he converted back to normal sinus rhythm with heart rate in the 70s. His lithium level returned to normal range after one hemodialysis run, and his symptoms significantly improved. Lithium was discontinued, and it was recommended that he avoid it in the future. The patient remained in normal sinus rhythm and did not require pacemaker implantation. Discussion: This case illustrates the importance of prompt recognition of lithium toxicity so that it can be appropriately treated, as it can be life threatening. Lithium toxicity classically presents with neurologic and/or gastrointestinal symptoms, but can rarely present with cardiac manifestations including dysrhythmias, cardiomyopathies, and myocardial infarction. The mechanism of cardiotoxicity from lithium is not well understood, but likely is a result of its interference with cation exchange across cardiac cell membranes, resulting in decreased spontaneous depolarization of the sinus node. Conclusion: The management of cardiotoxicity from lithium includes supportive care with intravenous fluids and hemodialysis in severe cases. Often, pacemaker placement can be avoided if dysrhythmias are present, as they typically resolve once lithium levels normalize.
Purvaja Marella Dr. Gaurav Suryawanshi	*From Womb to World: Vertical Transmission of Disseminated Histoplasmosis*

Dr. Thomas Leventhal	Disseminated histoplasmosis in pregnancy is rare, and vertical transmission is even more unusual. Case Presentation: A 27-year-old primigravida with HLA-B27–positive spondyloarthritis and prior infliximab exposure presented at 23 weeks' gestation with fever, cough, and hypoxia. Blood and tissue cultures confirmed disseminated Histoplasma capsulatum. She received prolonged liposomal amphotericin B and transitioned to posaconazole after delivery. Her infant, though asymptomatic, had a positive urine antigen and was treated with itraconazole. Conclusion: This case underscores the diagnostic challenges of disseminated histoplasmosis during pregnancy and documents the exceptionally rare occurrence of confirmed maternal–fetal transmission.
Richard Nguyen Dr. Amanda McIntyre	*Food-Dependent Exercise-Induced Anaphylaxis* Background: Food-dependent exercise-induced anaphylaxis (FDEIA) is a phenomenon characterized by the onset of anaphylaxis only when a food allergen is ingested within a few hours of exercise. Neither food allergen ingestion nor exercise in isolation is sufficient to provoke symptoms. Patients with FDEIA have IgE sensitization to the food that triggers symptoms. Exercise is thought to enhance intestinal permeability, which facilitates increased absorption of the food allergen and IgE binding. This amplifies mast cell degranulation and can result in a more severe allergic reaction, including anaphylaxis. The diagnosis of FDEIA is confirmed by IgE sensitization to the food that causes the reaction in close proximity to exercise. Management requires avoiding exercise at least a few hours before and after ingesting the food allergen and carrying an epinephrine autoinjector in case of reaction. Case Presentation: A 15-year-old male presented to allergy clinic after being treated in the ER for full body hives, facial swelling, and shortness of breath responsive to epinephrine. He had noted 3 weeks of preceding upper respiratory infection symptoms along with itching and some intermittent hives. When he was seen in allergy clinic, he reported that his symptoms occurred after waking up from a nap. He recalled eating some mango after his facial swelling occurred, but he had subsequently tolerated mango. He had also played soccer a few hours prior to his nap. It was thought that his week of preceding urticaria were related to viral illness with worsening of his symptoms due to exercise or another cofactor. Environmental allergy testing was positive to cat and dog, but he has no pets. He had a normal tryptase (to screen for systemic mastocytosis) and C4 and C1 esterase inhibitor studies (to screen for hereditary angioedema). Alpha-gal syndrome was considered as this

	can cause delayed anaphylaxis, but alpha-gal IgE was negative. Wheat is the most common cause of FDEIA. He was not sensitized to the gluten component most commonly responsible for FDEIA (omega-5 gliadin). Patient was advised to document exercise and food intake if any further reactions occurred. He had another episode of anaphylaxis about 3 months later after consuming shrimp and mango. He noted he had soccer practice a few hours prior. Allergy testing was positive to mango and negative to shrimp, consistent with FDEIA triggered by mango. He continues to tolerate mango as long as it is not consumed within a few hours of exercise. Conclusion: Food allergy reactions typically occur with every ingestion of the food allergen, which is not the case in FDEIA. This case highlights the important role of cofactors in allergic reactions. Common cofactors that can affect allergic reaction severity include exercise, NSAIDS, alcohol, and infection. When evaluating a patient for anaphylaxis, it can be helpful to ask about other cofactors.
Dil Patel	*When Infection Mimics Infarction: Unrecognized Endocarditis Leading to Cardiogenic Shock and Transplant Evaluation* Background: Mitral valve (MV) infective endocarditis (IE) presenting as ST-elevation myocardial infarction (STEMI) is a rare but life-threatening complication. It typically occurs when a fragment of infected valvular material embolizes into a coronary artery. Early diagnosis is often delayed due to nonspecific systemic symptoms and the limited sensitivity of transthoracic echocardiography (TTE) in detecting small vegetations. Case Presentation: A 42-year-old previously healthy woman with a 3-month history of night sweats and weight loss underwent preoperative evaluation for spinal fusion surgery. A newly detected holosystolic apical murmur prompted an outpatient TTE, which revealed a flail posterior mitral valve leaflet (PMVL) with severe regurgitation but no vegetations. A computed tomography scan also revealed new splenic infarcts. She was scheduled for outpatient transesophageal echocardiography (TEE), but before this could be completed, she presented to the emergency department with severe substernal chest pain and was diagnosed with an acute anterior STEMI. Coronary angiography revealed complete mid–left anterior descending (LAD) artery occlusion without disease in other vessels, suggesting an embolic etiology. She underwent successful primary percutaneous coronary intervention; however, within 24 hours, she developed in-stent thrombosis versus recurrent embolization, requiring aspiration thrombectomy. Her course was complicated by cardiogenic shock, necessitating mechanical circulatory support—initially intra-aortic balloon pump (IABP) followed by Impella—and inotropic therapy. Blood cultures

	grew polymicrobial bacteremia from periodontal origin. TEE revealed multiple mobile vegetations on the PMVL, and she underwent surgical mitral valve replacement, which confirmed leaflet destruction secondary to IE. She was successfully weaned off Impella but remained inotrope-dependent and is currently undergoing heart transplant evaluation while completing a prolonged course of intravenous antibiotics. Discussion: This case underscores the diagnostic challenge of recognizing IE in patients presenting with nonspecific symptoms and lacking traditional risk factors. In this patient, a flail PMVL on TTE, combined with systemic symptoms and splenic infarcts, should have raised earlier concern for IE. Delayed recognition led to septic coronary embolism, cardiogenic shock requiring both pharmacologic and mechanical support, and irreversible myocardial injury necessitating heart transplant evaluation. Early utilization of TEE and heightened clinical suspicion are crucial for preventing catastrophic complications of unrecognized IE.
Kiran Ponduru Dr. Sreekanth Avula Dr. Samuel Ives	*Cancer isn't Cancer: Nodular Regenerative Hyperplasia Mimicking Malignant Biliary Obstruction* Introduction: Obstructive jaundice with weight loss and a hilar mass in older patients often raises strong suspicion for malignancy, especially cholangiocarcinoma or pancreatic cancer. However, hepatovascular disease and parasitic infections, such as Fasciola hepatica, may closely mimic malignancy both clinically and radiographically. These mimics complicate diagnosis and can lead to invasive procedures or empiric therapies before clarification. We describe a case where suspected cholangiocarcinoma was ultimately revealed to be nodular regenerative hyperplasia (NRH), an uncommon cause of non-cirrhotic portal hypertension and biliary obstruction. Case Presentation: A 63-year-old man with alcohol and tobacco use presented with four months of jaundice, pruritus, anorexia, and weight loss. Having lived in Colombia and Mexico, he was at risk for parasitic exposure. Labs showed hyperbilirubinemia, thrombocytopenia, and hypoalbuminemia; carcinoembryonic antigen (CEA) was mildly elevated. Imaging demonstrated intrahepatic and common bile duct dilatation with a hilar mass encasing the hepatic artery and portal vein, suggestive of cholangiocarcinoma. Peritoneal nodularity raised concern for advanced disease. MRI/MRCP confirmed long-segment strictures. ERCP and EUS-FNA were performed, but multiple brushings and biopsies were negative for malignancy. Stenting improved symptoms. Given his background, parasitic workup was pursued; though serologies and stool studies were negative, empiric antiparasitic therapy was given. Because of suspected non-cirrhotic portal hypertension, a liver biopsy was obtained and revealed nodular regenerative hyperplasia without

	fibrosis, accounting for portal hypertension and mimicking malignancy. He was treated with non-selective beta-blockers, continued biliary stenting, and surveillance. Bilirubin improved, and no malignancy was found on follow-up. Discussion: This case underscores how benign hepatic vascular disease can convincingly imitate cancer. The combination of obstructive jaundice and hilar mass effect often leads to an oncologic pathway, yet portal hypertension-related biliary changes (portal hypertensive biliopathy) may produce strictures, dilation, and mass-like lesions. Tumor markers such as CEA are unreliable and potentially misleading in this context. NRH is an under-recognized cause of non-cirrhotic portal hypertension, typically linked to altered hepatic blood flow, chronic portal vein thrombosis, or prothrombotic states. Diagnosis requires histopathology, as imaging cannot reliably distinguish NRH from neoplastic or cholestatic disease. Awareness is critical to avoid unnecessary surgery or chemotherapy. This case also highlights the importance of improved diagnostic access for parasitic infections like Fasciola hepatica in U.S. populations with epidemiologic risk. Conclusion: Obstructive jaundice with hilar mass usually suggests malignancy, but absence of pathologic confirmation should prompt consideration of vascular or infectious mimics. In this case, biopsy clarified the diagnosis as nodular regenerative hyperplasia with portal hypertension. Broader awareness can prevent misclassification as cancer and guide appropriate management.
Aishwarya Pradeep Dr. Gerald Volcheck Dr. Amjad Kanj	*When HAM doesn't Slam: A Case of "Severe" ABPA with Minimal Symptoms* Introduction: Allergic bronchopulmonary aspergillosis (ABPA) is a complex hypersensitivity reaction of the airways to Aspergillus in patients with asthma or cystic fibrosis. It usually presents with difficult-to-control airway disease and impaired mucus clearance leading to chronic pulmonary infiltrates and bronchiectasis. Diagnosis requires a compatible clinical or radiological presentation, Aspergillus sensitization with total serum IgE ≥500 IU/mL, Aspergillus-specific IgE ≥0.35 kUA/L, plus at least two of the following: peripheral eosinophilia, positive Aspergillus-specific IgG, or characteristic imaging. High-attenuation mucus (HAM), defined as mucus plugs denser than skeletal muscle (≥70 Hounsfield units [HU]), is pathognomonic for ABPA, seen in 15-20% of cases, and is associated with severe disease and long-term corticosteroid dependence. Current guidelines recognize that HAM confirms the diagnosis even if other criteria are not met. We present an unusual case of a 69-year-old-female who met criteria for severe ABPA with HAM but was asymptomatic apart from an occasional cough and review management approaches.

	Case Presentation: A 69-year-old female had incidental abnormal lung findings on magnetic resonance imaging obtained for evaluation of cervical radiculopathy. Her past medical history was pertinent for eosinophilic esophagitis, seasonal allergies, and asthma (not requiring maintenance therapies). A dedicated chest CT revealed right upper lobe consolidations with mild bronchiectatic changes in the mid lung fields and a right perihilar consolidation. She did not report any significant respiratory symptoms except for a mild, intermittent cough that was occasionally productive of brown mucus plugs. She denied dyspnea, wheezing, fevers, chills, and chest pain. Chest CT was concerning for tubular/nodular structures with HU > 80-110 consistent with impacted HAM, raising strong concern for ABPA. Workup revealed elevated total serum IgE (4807 U/L), Aspergillus-specific IgE (59 kU/L), and positive skin testing for Aspergillus fumigatus. Blood eosinophils were 420/μL and oral exhaled nitric oxide was 62 ppb. Cystic fibrosis was excluded with a full CFTR gene analysis. PFTs revealed borderline obstruction (baseline FEV1/FVC 62%, FEV1 85% predicted) with a significant bronchodilator response (+14% from predicted). Given her mild presentation, she was managed with a mucociliary clearance regimen consisting of hypertonic saline and oscillating positive expiratory pressure therapy, in addition to an inhaled corticosteroid/long-acting beta-agonist (ICS/LABA). Systemic corticosteroids and anti-fungal therapy were deferred considering the relative lack of symptoms. She continues to do well 2 months since follow-up with no complaints. Conclusion: Patients with ABPA typically have very poor lung function, often with reduced FEV1, vital capacity, and gas transfer, with more severe impairment than seen in asthma alone and frequently requiring systemic corticosteroids. Although HAM is pathognomonic for ABPA and associated with severe disease, some patients may demonstrate relatively preserved pulmonary function and only mild clinical symptoms. While systemic corticosteroids remain the mainstay of therapy to control the immune response and anti-fungal agents can be used to reduce fungal burden, management should be individualized mainly based on symptom burden. Current guidelines do not recommend treating asymptomatic ABPA patients with systemic therapy. This is important for hospitalists and primary care providers when determining whether to initiate treatment independently or refer to a pulmonologist.
Matthew Record Dr. Claire Embree Dr. Jairo Hernandez Dr. Reid Schlesinger Dr. David Phelan	*The Dysautonomic Dilemma: Unusual Presentation of Guillain-Barré Syndrome in a 90-Year-Old with Cardiac Risk Factors* Background: Dysautonomia encompasses a wide differential diagnosis, especially in individuals with underlying coronary artery disease (CAD) and heart failure, where autonomic dysfunction may be multifactorial and life threatening. Recognizing dysautonomia as a potential initial manifestation of Guillain-Barré Syndrome (GBS) is

	critical, as it may precede or overshadow motor symptoms and is associated with increased morbidity and mortality. This case describes an unusual presentation of dysautonomia and nonspecific neurologic symptoms in a patient with CAD but without known infectious trigger or ascending paralysis who was found to have GBS. Case Presentation: A 90-year-old male with chronic bilateral lower extremity neuropathy, CAD, atrial fibrillation, and pituitary macroadenoma presented with new-onset neurologic symptoms, including bilateral hand numbness, gait instability with recent fall, dull headache, and double vision. He denied recent infection, vaccinations, or changes in medication or medical history. Vital signs revealed significant bradycardia (heart rate 20 BPM), irregularly irregular rhythm, and elevated blood pressure (SBP 180s). Physical examination revealed right eye exotropia, full strength in both upper and lower extremities; reflexes: 1+ lower extremities, 2+ upper extremities. Sensory exam revealed bilateral stocking and glove deficits in upper and lower extremities. Hip imaging was unremarkable. Neurology was consulted for the patient's evolving neurologic exam; CT Brain, CT venogram, and MRI Brain were negative. Dedicated pituitary MRI revealed slight compression of the third cranial nerve nucleus but no pituitary apoplexy or acute macroadenoma progression. Cardiology and heart failure services were also consulted; pacemaker placement was deferred for patient’s asymptomatic bradycardia, and fat pad aspiration was negative for systemic amyloidosis. During the hospitalization, the patient’s motor weakness worsened without reflex changes or new respiratory or bulbar symptoms. He also developed worsening urinary retention without saddle anesthesia. Upper and lower extremity EMG and NCS revealed forearm conduction blocks, conduction velocity slowing, prolonged distal motor latencies, and prolonged blink reflexes, suggesting an acquired demyelinating process. Lumbar puncture was deferred due to advanced age and chronic anticoagulation; West Nile, Lyme, and syphilis serologies were negative. Based on the presentation including sudden onset sensory deficits, dysautonomia, and worsening urinary retention, a diagnosis of acute inflammatory demyelinating polyradiculoneuropathy (e.g., GBS) was made. An empiric five-day course of IVIG was initiated, and the patient’s symptoms subsequently improved, prompting discharge to a skilled nursing facility. Discussion: Infectious or immunologic triggers that often precede GBS were absent in this case. Autonomic involvement is common and potentially life threatening; here, bradycardia and urinary retention were key features. Interpreting dysautonomia was complicated by the patient’s pre-existing cardiac comorbidities, where arrhythmias and blood pressure variability may have multifactorial origins. Sensory findings were confounded by a pre-existing length-dependent neuropathy.

	Conclusion: This case highlights the complexity of GBS in patients with chronic neuropathy and cardiovascular disease, which underscores the importance of recognizing atypical presentations, especially when dysautonomia presents and may rapidly become life threatening.
Zachary Scharf Dr. Marius Stan	*The Anomalous Adrenals: A Challenging Case of Ectopic Cushing's Syndrome and Its Considerations* Case: A 45-year-old male farmer presented to the emergency department with subjective weakness, dyspnea on exertion, and abdominal pain over the preceding three months. On arrival, the patient was unable to sit up or ambulate. His comorbidities included atrial fibrillation on warfarin, heart failure with reduced ejection fraction of 35-40%, hypertension, obstructive sleep apnea, and new-onset diabetes. Furthermore, he struggled with lower extremity edema over the preceding several years with non-healing, draining ulcers. A lung mass identified two years earlier was noted to be enlarging. Prior to this presentation, it was diagnosed as aspergillosis via bronchoscopy. Despite voriconazole, he was now deteriorating and was admitted for further evaluation. Given his dyspnea on exertion and abdominal pain, CT chest, abdomen, and pelvis imaging was obtained. He was subsequently found to have bilateral adrenal thickening, a pneumoperitoneum, and complicated sigmoid diverticulitis with a 10 cm abscess. General surgery was consulted. Given his presentation with plethora, large body habitus, and abdominal striae, an AM cortisol was obtained and was elevated to 46 mcg/dL with an ACTH of 257 pg/mL. The endocrinology-guided workup included 24-hour urine cortisol testing elevated to 3199 mcg/24 hours (REF 3.5-45 mcg/24 hours) and dexamethasone suppression testing elevated to 5150 ng/dL. A pituitary MRI was obtained and was negative for a pituitary adenoma. Ultimately, a diagnosis of ACTH-dependent Cushing's syndrome was made due to suspected ectopic disease, for which no obvious source was identified despite an extensive evaluation. Under the direction of the surgery service, he underwent a sigmoid colectomy with end colostomy for abscess treatment. During the case, he required dual vasopressor therapy. Given his instability, the initial plan for bilateral adrenalectomy was deferred. He was initiated on metyrapone to inhibit cortisol production with serial cortisol levels monitored. In the setting of this therapy, he developed orthostatic hypotension and was subsequently started on hydrocortisone therapy for metyrapone-induced adrenal insufficiency. With this therapy, he progressed appropriately. He was discharged with endocrinology follow up.

	Post-hospitalization, he underwent a successful bilateral adrenal cryoablation. He was then transitioned to adrenal hormone replacement therapy with prednisone and fludrocortisone. In the following five months, the patient lost over fifty pounds. He was no longer dyspneic and returned to his farmwork. His aspergillosis resolved after a twelve-week course of voriconazole. His lower extremity wounds healed, and he transitioned from near constant use of compression stockings to regular socks. Conclusions: This case demonstrates multiple learning points. Firstly, it presents a case of ectopic Cushing's syndrome, a challenging diagnosis, and elucidates the diagnostic process. Workup to identify the primary tumor responsible for excess ACTH is essential but, unfortunately, not always successful. Secondly, it demonstrates significant immunosuppression as a complication of Cushing's syndrome. It is profound immunosuppression that likely resulted in aspergillosis, abscess formation, and chronic non-healing wounds for this patient. Thirdly, this case demonstrates how to manage ectopic Cushing's syndrome, even when surgery is not an immediate option. Lastly, the case touches upon the importance of balancing therapy with the side effects of medication or treatment-related adrenal insufficiency.
Hannah Schull	*A Broad-Based Blindspot: A Diagnostic Dilemma of CNS Blastomycosis* Case Presentation: A 36-year-old male with a past medical history of GERD, tobacco use and an occupational history of construction work presented to the emergency room after having two seizure-like episodes at home. Prior to this, he was feeling well with no symptoms. MRI head obtained on presentation demonstrated a new left frontal lobe mass measuring 16 x 14 x 14 mm with associated vasogenic edema, concerning for an underlying neoplasm, favored to represent a primary glioma in the absence of a known alternative primary. The patient was started on dexamethasone, levetiracetam and admitted to the hospital. He was evaluated by neurosurgery who also expressed concern for a primary brain neoplasm with plans for tissue biopsy. A CT chest abdomen pelvis was obtained to evaluate for alternative primary neoplasm and demonstrated innumerable pulmonary nodules. The patient was ultimately discharged home on high dose dexamethasone and antiepileptics with plans for surgical biopsy of brain mass in the coming weeks. An fMRI was obtained one week after discharge and showed interval decrease in the size of his brain mass, which would be atypical in the setting of a glial tumor. Roughly two weeks after his discharge from the hospital, the patient returned to the ED with chest pain, body aches, and palpitations. In the emergency department, he was hypoxic to the 60%s requiring BiPAP support and tachycardic up to a heart rate of 180. CT chest imaging at that time

	showed bilateral, multilobular diffuse ground-glass opacities and coalescent consolidations. He was admitted to the intensive care unit. His serum histoplasma antigen returned positive, raising the concern for fungal infection that had worsened from high dose dexamethasone. He was treated with amphotericin. Two days later, broad-based budding yeast was identified on his sputum culture consistent with blastomycosis. Repeat imaging of the head was obtained which showed a subtle ring enhancing lesion in the left frontal lobe, raising the concern for fungal abscess. Ultimately, the patient's respiratory status decompensated further into fulminant ARDS. He was cannulated for VV-ECMO and remained on this for twenty days. Unfortunately, he succumbed to his illnesses in the setting of overwhelming sepsis from gram-negative bacteremia and gut ischemia only six weeks after his initial presentation. Discussion: Solitary brain lesions often prompt concern for primary CNS tumors, but infectious etiologies - especially endemic mycoses - must remain in the differential. Blastomycosis, caused by Blastomyces dermatitidis, is a dimorphic fungus endemic to regions overlapping with Histoplasma capsulatum. These pathogens share geographic distribution, environmental exposures, pulmonary manifestations and their antigen tests may cross-react, complicating diagnosis. Both can disseminate into the CNS. Early diagnosis is challenging due to nonspecific imaging findings and overlap with neoplastic processes. This case highlights the diagnostic pitfalls and severe outcomes associated with CNS blastomycosis presenting as an isolated brain mass.
Joshua Schwanke Dr. Courtney Burnett	*The Pan Positive Patient: A Case of Necrotizing Pneumonia Secondary to Presumptive MSSA Endocarditis Complicated by Hepatic Micro-Emboli, Blastomycosis, Coccidioides and New HIV and Hepatitis C Infections.* Background: Every new medical student is taught the importance of gathering a detailed history, building a broad differential, and ordering appropriate tests. We present a case in which a broad differential of possible diagnoses led to an appropriate work-up in a patient whose testing revealed multiple new infections. Many of the infections were discovered after a convincing diagnosis had been made. Case Presentation: A 31/yo male with no known PMH presented with one week of cough, fever, fatigue, night sweats, shortness of breath and chest, shoulder, and back pain. He was initially hypotensive, with a lactate of 3.4. Chest x-ray noted cavitary appearing nodular opacities of the upper lungs and lower left lung prompting CT scan with contrast of his chest which found diffuse bilateral nodular cavitary and non-cavitary lesions, CT head had no evidence of pathology. CMP was notable for creatinine 1.20 AST 1,181 and ALT 251. CBC with differential found leukocytosis with neutrophilic predominance. Blood

	cultures were drawn and he was started on empiric broad spectrum antibiotics. Fungal urine antigen panel, atypical bacterial panel, viral respiratory panel, viral hepatitis panel, and HIV were ordered, resulting in positive HIV, Hepatitis C, and MSSA blood culture results. Echocardiogram revealed a probable 2 cm vegetation on his tricuspid valve, and eventually his urine blastomycosis and coccioides came back positive. He was treated with cefazolin and itraconazole. Given his shoulder, back, and ankle pain he received MRI of those sites which revealed no infectious process. This patient worked for a carnival game company based in South Dakota. He had been in Texas, Oklahoma, and Wisconsin before presenting to the hospital in Minnesota. Upon expanded social history, he reported a distant history of injection drug use and incarceration as well as more recent safe sex with one partner and negative STD testing at an unknown medical facility. His history hinted at the possibility of other potential causes for his presentation even with blood cultures positive for MSSA. Initial differential diagnosis included community acquired pneumonia, fungal pneumonia, tuberculosis, and pulmonary embolism. Given the high rates of mortality for MSSA bacteremic pneumonia (21.3%) with rates even higher for patients admitted to the ICU (36.5%) managing potential complications was important. Broadening his work-up allowed for the prompt addition of anti-fungal coverage. Discussion: This patient had a clear diagnosis that explained his symptoms, labs, and imaging (necrotizing pneumonia and hepatic micro-emboli secondary to MSSA endocarditis). However, without a thorough history, testing for other possible causes of his presentation (particularly coccidioides, HIV, and hepatitis C) may have been deferred, potentially complicating his clinical course. Conclusion: It is always important to limit unnecessary testing to try and limit costs, decrease chances of negative effects, and prevent health anxiety in patients. However, potential diagnoses in critically ill patients should never be ignored, even if a most likely diagnosis has been made, as a missed diagnosis could have catastrophic effects for the patient.
Zeeshan Shahid Dr. Casandra Ladas Dr. David Flemig	*Erector Spinae Plane Block, an Under-Utilized Tool for Pain Relief During Hospice* Introduction: Bladder Cancer is a highly aggressive malignancy with a high rate of recurrence and metastatic potential [1]. One of the most common regions for metastasis is the spine leading to severe cancer-related pain refractory to systemic opioids reducing the quality of life and limiting the ability to undergo definitive treatment such as radiation [2, 3].

	Case Presentation: We report a case of a 39-year-old gentleman presenting with metastatic bladder cancer, CKD and HTN. Nine years after his initial diagnosis, the disease had spread to his spine. Going through severe intractable pain despite high dose IV hydromorphone (80mg). The pain prevented him from maintaining the optimum position necessary for receiving radiotherapy. As a result, our patient would have remained in the hospital for extreme pain management along with the risks and mortality that it entails. Treatment and Outcome: Through palliative medicine, a decision was made to initiate Erector Spinae Plane Block (ESPB) with continuous catheter infusion. The ESPB catheter was maintained for three weeks with the patient experiencing a significant improvement, thus allowing him to tolerate radiotherapy. This allowed us to reduce the dosage of hydromorphone successfully from 80mg to 18mg. Upon removal of the catheter, the patient continued to experience sustained pain relief and improved functional status. With a reduced need for analgesia, our patient was able to be discharged home and spend his remaining days, of the hospice period, in the comfort and company of those that he loved. Conclusion: This case demonstrated the utility of ESPB as a safe and effective opioid sparing modality that can be used in management of chronic pain in metastatic cancer [4]. In addition to reducing pain, it facilitates intervention such as radiotherapy that would be limited in patients with uncontrolled pain. This is a vastly underutilized procedure; a modality that can free patients from high dosage of opiates, improving their quality of life during hospice. References: Dobruch J, Oszczudłowski M. Bladder cancer: current challenges and future directions. Medicina. 2021 Jul 24;57(8):749. Ahmadi H, Duddalwar V, Daneshmand S. Diagnosis and staging of bladder cancer. Hematology/Oncology Clinics. 2021 Jun 1;35(3):531-41. Takei D, Tagami K. Management of cancer pain due to bone metastasis. Journal of Bone and Mineral Metabolism. 2023 May;41(3):327-36. Capuano P, Alongi A, Burgio G, Martucci G, Arcadipane A, Cortegiani A. Erector spinae plane block for cancer pain relief: a systematic review. Journal of Anesthesia, Analgesia and Critical Care. 2024 Nov 15;4(1):76.
Lauren Slarks	*More Than Chronic Pelvic Pain* Background: Women with chronic pain syndromes may experience delayed recognition of underlying physical conditions, either missed during initial evaluation or arising subsequently. This diagnostic challenge is compounded by the frequency of such presentations and

	by the well-documented tendency for women to receive later diagnoses than men across many conditions. These factors highlight important diagnostic considerations necessary to ensure equitable care. Case Presentation: A 32-year-old, G2P2 black woman presented to the emergency department for abdominal pain, nausea and vomiting, weight loss and anemia. The pain started one year prior as bothersome bloating and nausea that occurred a few days around her menstrual cycle, progressing to debilitating left, mid and right abdominal pain that radiated to her back and groin. She also had emesis every 2-3 days, and 50 lb weight loss during that time related to food avoidance due to pain. Prior to presentation, workup included laparoscopy 6 months prior which was negative for endometriosis, and CT showing mild ileitis, which was deemed to be secondary to NSAID use. At that time, she started on hydrocodone for management of the pain, however despite discontinuing NSAIDs and starting opioids, her pain progressed, ADLs were impacted, and she became bedbound. In the emergency department, abdominal CT showed marked mural edema, hyperenhancement and perienteric stranding about the terminal and distal ileum (approximately 25 cm long segment), along with stricture and enteroenteric fistula between two loops of terminal/distal ileum and tip of appendix and multiple focal ulcerations visible in the wall of the terminal ileum. In the hospital, she was diagnosed with Crohn ileitis and treated with IV methylprednisolone, then was transitioned to 40 mg PO prednisone. She was able to begin ambulating, tolerate oral intake, and discontinue hydrocodone prior to discharge. Following up with GI outpatient, she was started on infliximab and azathioprine as she tapered off of prednisone. She did start dicyclomine for IBD symptoms of cramping, bloating, pain and diarrhea. Conclusion: This case underscores the risk of missed diagnoses when pain is managed as chronic without appropriate reevaluation. It also highlights the well-documented disparities in pain management, with Black patients less likely to receive adequate treatment, further compromising quality of life. Timely recognition of conditions such as Crohn's disease is essential to reduce morbidity, particularly in young women with aggressive presentations.
Dakota Snustad Dr. Milos Brancovic Dr. Sasha Prisco	*Iron Overload Cardiomyopathy in an HFE C282Y Heterozygote with End-Stage Liver Disease due to Alcohol-Associated Cirrhosis* Background: Iron homeostasis is a complex process that, when disrupted, can present with iron overload. Iron overload cardiomyopathy (IOC) is a rare yet relevant infiltrative cardiomyopathy that should be included in the differential diagnosis of non-ischemic cardiomyopathy. Case Presentation: A 54-year-old man with alcohol-associated cirrhosis presented with shortness of breath with exertion, nonproductive cough, abdominal distension, and unintentional 25lb

	weight gain over three weeks. Cardiac magnetic resonance imaging revealed severe biventricular dilatation, with late gadolinium enhancement imaging demonstrating findings consistent with myocardial iron overload, characterized by very low T1 map and T2 map values, and a T2* myocardial value of 10 ms. Subsequent iron studies demonstrated elevated ferritin of 922 ng/mL, iron-binding capacity below the level of detection, low transferrin of 83 mg/dL, and normal serum iron of 92 ug/dL. Genetic testing revealed a C282Y heterozygous genotype in the HFE gene. The patient was started on iron chelation therapy with deferasirox and vitamin E while awaiting evaluation for a combined heart-liver transplant. Discussion: In this case, the onset of heart failure closely mirrored the progression of the patient's cirrhosis. The patient did take a daily multivitamin containing 27mg of iron, but his ferritin and serum iron levels were normal three years before presentation, he had no recent blood transfusions, and he had a coronary angiogram completed seven months prior to presentation, which revealed mild non-obstructive coronary artery disease, making pre-existing iron overload and ischemic cardiomyopathy less likely. Rather, the dysregulated iron metabolism caused by his liver dysfunction and his HFE mutation may have contributed to the development of severe heart failure in less than one year. Conclusion: IOC is a rare cause of infiltrative cardiomyopathy that should be included in the differential diagnosis of non-ischemic cardiomyopathy, and cardiac MRI is an excellent modality to evaluate for the presence and extent of IOC. Generally, HFE gene heterozygosity is clinically insignificant; however, in rare cases of concomitant severe liver disease, the iron overload phenotype may emerge.
Mary Soukup	*Severe Acute Interstitial Nephritis on Re-exposure to a Proton Pump Inhibitor* Introduction: The most common cause of unexplained acute kidney injury (AKI) is drug-induced interstitial nephritis (AIN), accounting for approximately 20% of all unexplained AKIs that lead to biopsy (1). Most cases of AIN are mild, do not require biopsy, and resolve with discontinuation of the drug. This clinical vignette describes a particularly severe case of AIN following re-exposure to a proton pump inhibitor. It highlights how the cell mediated mechanism of injury can cause unusually severe cases, the role of early steroid administration, and the importance of a thorough medication review in patients with unexplained AKI. Case Presentation: A 78-year-old male presented to the emergency room with a chief complaint of epistaxis and was found to have a serum creatinine of 8.7 mg/dL, a rapid increase from 1.67 mg/dL at a clinic visit 7 days prior. Urinalysis showed a few leukocytes but no hematuria. Urine protein-creatinine ratio was elevated at 1.18 mg/mg.

	Bladder ultrasound showed no sign of urinary retention. His only recent medication change was starting high dose omeprazole 15 days prior to his emergency room presentation, which was prescribed for erythematous duodenopathy seen on esophagogastroduodenoscopy. Omeprazole was held on admission to the hospital, but his creatinine continued to rise to 11.52 mg/dL. The patient underwent a kidney biopsy on hospital day three that revealed a high degree of tubulointerstitial inflammation, which included eosinophils. Given his clinical picture, these findings were most consistent with AIN due to omeprazole. Of note, the patient had previously started omeprazole approximately 8 months prior to this presentation and continued taking it for three months before discontinuing. Given the lack of improvement in creatinine with discontinuation of the offending agent and biopsy findings consistent with AIN, the patient received 500 mg IV methylprednisolone daily for three days followed by a ten-week prednisone taper. Fortunately, his kidney function recovered without kidney replacement therapy. After completion of the steroid taper, his creatinine was 1.39 mg/dL (previous baseline was 1.2 mg/dL) and urine protein-creatinine ratio was 0.12 mg/mg. Discussion: This case highlights the immunologic mechanism by which AIN develops. This patient's case was likely more severe and rapidly progressive because of previous exposure to omeprazole. His T cells had already been sensitized to the antigen, allowing for a more significant inflammatory response on re-exposure. This case also illustrates the importance of early administration of steroids in severe AIN when discontinuation of the suspected offending agent alone does not result in improvement in kidney function. Rapid treatment with corticosteroids likely prevented early fibrotic changes, which can develop as early as 7-10 days after the start of the inflammatory process (2). Finally, this case demonstrates the importance of a thorough medication review for patients with an unexplained AKI. References: Moledina DG, Perazella MA. Drug-Induced Acute Interstitial Nephritis. Clin J Am Soc Nephrol. 2017 Dec 7;12(12):2046-2049. doi: 10.2215/CJN.07630717. Epub 2017 Sep 11. PMID: 28893923; PMCID: PMC5718279. Praga M, González E. Acute interstitial nephritis. Kidney Int. 2010 Jun;77(11):956-61. doi: 10.1038/ki.2010.89. Epub 2010 Mar 24. PMID: 20336051.
Rachel Sweet Dr. Anya Jamrozy	*Beyond the PRESsure Paradigm: Identifying Risk Factors for Development of PRES* Background: Posterior reversible encephalopathy syndrome (PRES) is a neurologic condition characterized by headache, decreased level of consciousness, visual changes and seizures and is commonly associated with severely high blood pressure (systolic measurements

	above 160mmHg). Risk factors related to endothelial dysfunction have also been described as an etiology of PRES. These include systemic infection, severe uremia, and accelerated escalation of mean arterial pressure. These risk factors have been associated with PRES in the absence of severe hypertension in nearly 25% of cases. Case Presentation: A 28-year-old male with a history of opioid use disorder on methadone and nontraumatic rhabdomyolysis presented to the emergency department with 48 hours of flank pain, nausea, and vomiting after repetitive physical labor. Blood pressure (BP) on presentation was 105/67. Review of systems was notable for dark urine and poor oral intake. Creatinine kinase was found to be > 32K, creatinine 7.53 from recent baseline of ~1, BUN 49, urinalysis with brown urine, 4+ blood, but only 2 - 8 red blood cells. He was initially admitted for further treatment of acute renal failure due to rhabdomyolysis but was transferred to a higher level of care the following day for possible dialysis due to reduced urine output. Creatinine after arrival to the quaternary center was 9.76, BP 139/84. Methadone dose was renally adjusted to 75% of prior daily dose. Nephrology was consulted and recommended IV fluids with aggressive diuresis. Urine output (UOP) subsequently improved, although creatinine rose to 10.8 the following day. Dialysis was deferred given continued adequate UOP and downtrending rate of rise in creatinine. Overnight the patient developed chest pain, and chest XR revealed mild pulmonary edema. There were difficulties quantifying UOP as the patient was frequently voiding independently, it was later determined UOP had likely dropped. On AM evaluation patient reported headache and that he “could not see”, torsional nystagmus was present on exam, BP 162/102. STAT head CT was normal. PRES was suspected, Keppra load and continuous video EEG were ordered, but the patient sustained a 30 second witnessed seizure prior to receiving this dose. Eventual MRA/MRV revealed multiple irregular T2 FLAIR lesions involving the subcortical white matter and gray-white junctions, consistent with PRES. He was emergently dialyzed with improvement in blood pressures and eventual full recovery in neurological and renal function. Conclusion: This case illustrates known alternative etiologies of PRES including volume overload and renal failure. Conceptualizing PRES more broadly as an endovascular disorder may help clinicians consider this diagnosis in patients with systolic blood pressures < 160 and could lead to earlier support and intervention to improve outcomes.
Jacqueline Turner Heather Montane Dr. Svetomir Markovic	*Liver-Directed Therapy in Metastatic Uveal Melanoma: A Case Report Evaluation of Overcoming Therapeutic Resistance and Metastatic Organotropism* Background: Uveal melanoma is a rare subtype of melanoma that occurs within the orbit and has a unique propensity to metastasize to

	the liver. Hepatic involvement occurs in up to 95% of patients with stage IV disease, and these metastases are strongly associated with poor prognosis and mortality. Despite advances in systemic immunotherapies, uveal melanoma remains largely refractory to traditional and novel interventions, highlighting the need for effective liver-directed therapies. Case Presentation: We report the case of a 56-year-old man with uveal melanoma involving the choroid and ciliary body who developed diffuse hepatic metastases. He was ineligible for tebentafusp due to HLA-A*02:01 negativity and was treated sequentially with combination ipilimumab/nivolumab, single-agent nivolumab, and nivolumab-relatlimab, but progressed with persistent hepatic tumor burden. The patient subsequently underwent three cycles of percutaneous isolated hepatic perfusion with melphalan (HEPZATO), achieving significant radiographic improvement with >30% reduction in hepatic tumor volume on MRI. Suspected extrahepatic disease, including lung and peritoneal nodules, remained stable during treatment. While he developed immune related-adverse events from the prior immune checkpoint blockade, the HEPZATO treatment was well tolerated without significant toxicity. Conclusion: This case highlights HEPZATO as a promising liver-directed therapy for refractory uveal melanoma, capable of achieving meaningful hepatic disease control and contributing to prolonged survival. HEPZATO is a promising therapeutic option for patients with refractory uveal melanoma and illustrates the importance of liver-directed interventions.
Sai Sudha Valisekka Lauren Fontana Poorva Singh Dr. Mehrnoosh Tashakori Dr. Joan Beckman	*Hidden in the Marrow: Diagnostic and Therapeutic Pitfalls of Bone Marrow-Confined Parvovirus B19-Associated Pure Red Cell Aplasia* Introduction: Pure red cell aplasia (PRCA) is a rare syndrome characterized by severe anemia, reticulocytopenia, and a marked reduction of erythroid precursors in the bone marrow. Parvovirus B19 can cause PRCA in immunocompromised hosts, and diagnosis and treatment are complicated by the effects of immunosuppression on viral persistence and reactivation. Case Presentation: A 55-year-old female with Crohn's disease and breast cancer (both under surveillance) presented with progressive macrocytic anemia (hemoglobin 8 g/dl) and reticulocytopenia. Extensive evaluation, including nutritional studies, autoimmune serologies, viral studies, T-cell receptor gamma/delta studies, specialised testing (paroxysmal nocturnal hemoglobinuria, antiphospholipid syndrome, interleukin-6, vascular endothelial growth factor, serum/urine protein electrophoresis), was unremarkable. Bone marrow biopsy revealed erythroid hypoplasia with left shift and mild dyserythropoiesis. Parvovirus B19 DNA was detected by qualitative Polymerase Chain Reaction (PCR) in bone marrow, but was negative in peripheral blood, confirming the diagnosis of PRCA. Initial management with daily IVIG (400 mg/kg/day) for 5 days yielded only

	a transient increase in hemoglobin (to 10.4 g/dL), with subsequent relapse into transfusion dependence. Three months later, repeat bone marrow biopsy and qualitative PCR were negative for parvovirus B19, prompting a shift to immunosuppressive treatment strategies including Cyclosporine and Sirolimus for presumed refractory PRCA. Hemoglobin stabilization (10.0 -10.8 g/dl) was achieved on immunosuppression for 2 years, but it was then dropped to 9.0 g/dl. Subsequently, a repeat bone marrow biopsy had a positive parvovirus B19 qualitative PCR, with a negative qualitative peripheral blood PCR, indicating bone marrow-confined viral reactivation. The patient was restarted on IVIG (0.4 mg/kg/day) for 5 days, followed by monthly IVIG maintenance (0.4mg/kg/day), resulting in significant symptomatic and hemoglobin improvement (11.1-12.0 g/dl). Discussion: Diagnosis of parvovirus B19 PRCA relies on a combination of molecular and serologic assays. Quantitative and qualitative PCR are highly sensitive for detecting parvovirus B19 DNA in blood and bone marrow, with bone marrow PCR often more sensitive than peripheral blood PCR in immunocompromised patients. Serologic testing for parvovirus B19 IgM and IgG is useful in immunocompetent hosts, but may be negative in immunosuppressed individuals due to impaired antibody production. Bone marrow stains, including immunohistochemistry and in situ hybridization, can identify viral inclusions and confirm infection, especially when PCR and serology are inconclusive. Immunosuppression can prolong viral DNA positivity, complicate the interpretation of test results, and extend illness duration. Reactivation may occur during periods of increased immunosuppression, highlighting the importance of ongoing surveillance. Monthly IVIG maintenance, extrapolated from transplant literature, can be effective in preventing relapse in patients with ongoing immunosuppression or recurrent disease. Conclusion: This case highlights the importance of a multi-modal diagnostic approach for parvovirus B19 PRCA in immunocompromised hosts, the utility of bone marrow PCR and stains, and the potential need for repeated or maintenance IVIG in chronic or relapsing disease. Immunosuppression can prolong viral persistence and complicate management and diagnosis; careful medication trials, multidisciplinary collaboration, and ongoing surveillance are essential to optimize outcomes.
Brianne Vanderheyden Dr. Daniel Pollman	*A "Hole" Lot Sooner Than Expected* Case Presentation: A 71-year-old woman with no prior cardiac history presented to the Emergency Department with sudden-onset substernal chest pressure while getting out of the shower. On initial evaluation, high-sensitivity troponin T was elevated to 1609. Her ECG showed ST elevations in leads V2 and V3 and ST depressions in V5 and V6, concerning for anterior wall ST elevation myocardial infarction (STEMI). Nitroglycerin provided partial relief, and she received

	aspirin, ticagrelor, and heparin while awaiting coronary angiography. Coronary angiography on initial read showed no obstructive lesions. Transthoracic echocardiogram (TTE) showed hypokinesis of the basal to mid-anteroseptum and anterior wall, concerning for stress cardiomyopathy. Cardiac MRI showed an acute-appearing mid-apical septal transmural infarction and a 19 x 7 mm ventricular septal defect (VSD) with significant left-to-right shunting (Qp:Qs 1.7) and surrounding edema suggesting an acute process. On secondary review of the previously completed coronary angiogram, a possible small second diagonal (D2) branch occlusion was noted. The patient required an intra-aortic balloon pump with transition to Impella to allow for scarification of the new VSD for 13 days prior to undergoing patch repair complicated by inferior wall laceration. The postoperative course was complicated by arrhythmia requiring amiodarone and pericardial effusion. The patient gradually stabilized and was weaned from mechanical support, with postoperative TTE eight days after VSD showing no residual VSD but a new wall motion abnormality in the left anterior descending artery distribution with left ventricular ejection fraction 30–35%. Guideline-directed medical therapy was initiated prior to discharge, and she was recommended a repeat cardiac MRI but declined. Discussion: This case highlights the interesting temporal relationship between this patient's acute onset of chest pain and rapid development of VSD. Initially, these findings were attributed to myocardial infarction with non-obstructed coronary arteries (MINOCA) due to stress cardiomyopathy leading to the life-threatening mechanical complication of VSD. However, her possible D2 branch occlusion raises the possibility of a type I MI as the underlying etiology of her VSD. Mechanical complications of acute MI are increasingly rare but remain possible in patients with transmural infarction [4–6]. VSD occurs in <1% of acute MI patients and is commonly seen within the first week, with a mean time interval of 3–5 days after onset of symptoms [7]. Notable in this case is how quickly her VSD seemed to develop after symptom onset, less than the mean interval time commonly seen, with evidence of this even on initial TTE (not identified in the formal read) and surrounding edema on MRI confirming its acuity. Notable as well are the risk factors for post-MI VSD, including advanced age, female sex, absence of smoking history, hypertension, right ventricular infarction, and extensive MI, with this patient having three of these risk factors [7]. Conclusion: This case emphasizes the importance of close hemodynamic monitoring in MINOCA patients, consideration of stress-related mechanisms in MINOCA evaluation, typical onset of VSD, risk factors for VSD development, and early multidisciplinary management including Cardiology, Interventional Cardiology, and Cardiac Surgery for mechanical complications of MI including VSD.
Alison Vecellio	*From Delivery to Diagnosis: An Elusive Endocrine Emergency*

Dr. Breanna Zarmbinski	Background: Postpartum hypopituitarism also known as Sheehan Syndrome is secondary to necrosis of the pituitary gland, often due to ischemia in the setting of massive postpartum hemorrhage (1). It is more common in less developed countries, but remains present in developed countries even with improvement in obstetric care and notably, history of significant postpartum bleeding is not always present (2). The main clinical hormones affected are growth hormone and prolactin, but may also include FSH, LH, TSH and adrenocorticotropic hormone (1). Sheehan Syndrome symptoms can be nonspecific which often leads to delayed diagnosis (2). Case Presentation: This is a case of a 38-year-old multiparous woman with gestational diabetes and eclampsia admitted to the ICU in multiorgan failure after post-partum hemorrhage of about 1.5 L. In the initial days after delivery the patient was noted to have no breast milk production. She remained intermittently hypotensive and a few days after delivery additionally developed hypothermia and hypoglycemia. Further lab work-up in the acute period revealed low IGF-I, inadequately elevated prolactin, inappropriately normal total cortisol, and undetectable ACTH. Her blood pressure, temperature and glycemic control improved with starting hydrocortisone. Further monitoring of labs moving forward revealed initially normal TSH with downward trend into hypothyroidism as well. Her FSH and LH remained within the normal range on subsequent follow up. MRI Brain WO obtained on postpartum day 7 showed nonspecific fullness of the pituitary gland with heterogenous T1 hyperintense signal within the superior gland potentially representative of subacute pituitary apoplexy. On Endocrinology follow up 7 months after delivery she remained amenorrheic. She is maintained on hydrocortisone for adrenal insufficiency, levothyroxine for hypothyroidism and desmopressin for diabetes insipidus. Hormone replacement therapy is being considered for ongoing hypogonadism. Conclusion: This case is a good example of Sheehan syndrome diagnosed in the acute setting. However, given many cases present and are diagnosed later on it is important for the internist to keep panhypopituitarism on the differential for patients presenting with nonspecific symptoms outside of the immediate postpartum period including decades later. Symptoms may include amenorrhea or other reproductive issues, fatigue, appetite or weight changes, depression, hypotension, hypoglycemia and hyponatremia. Diagnosis is important for initiating hormone replacement and for monitoring and treatment for additional risk factors associated with hypopituitarism including increased cardiovascular risk factors, osteoporosis and depression (2, 3, 4).
Kyle Wansing	*A Silent Threat Revealed: Pulmonary Artery Mycotic Aneurysm Secondary to Endocarditis*

	Background: Mycotic aneurysms are a rare but dangerous form of vascular lesion caused by infection of the arterial wall. Despite the name, they are usually bacterial rather than fungal. They account for less than 1% of all aneurysms, yet they are clinically significant due to their high rate of rupture. Pulmonary artery aneurysms rupture in roughly 42% of reported cases, while aortic mycotic aneurysms rupture in 50–85%, underscoring the need for rapid recognition and intervention. Case Presentation: We present the case of a 26-year-old woman with a history of MRSA tricuspid valve endocarditis, polysubstance use disorder, and bipolar disorder. This case illustrates how careful bedside evaluation, early recognition, and collaboration across specialties can change the trajectory of care, even in a complex patient. The patient came to our emergency department after leaving another hospital AMA. She was hemodynamically stable but had known Enterococcus faecalis bacteremia with tricuspid valve vegetations. She was restarted on culture-directed antibiotics, with a planned six-week course. Several days before discharge, she reported "feeling off." On exam, she had worsening CV waves, hepatomegaly, an accentuated P2, and new egophony. Coupled with fever, these findings raised concern for septic emboli, prompting a CTA. Imaging revealed a large lobular aneurysm of the left lower segmental pulmonary artery, measuring 3.2 × 3.0 × 3.9 cm, consistent with a mycotic aneurysm. Given the rupture risk, a multidisciplinary team was quickly consulted. Based on the patient's risk factors and deteriorating clinical status, an endovascular approach with pulmonary artery embolization by interventional radiology was performed. This approach, while new, has demonstrated high technical success rates and favorable outcomes in recent meta-analyses and case studies. During the procedure, the patient developed hemoptysis, and contrast extravasation was noted into the left mainstem bronchus. An endobronchial blocker was placed by thoracic surgery to control bleeding, and embolization was successfully completed. The blocker was left overnight and removed the next day. She was extubated without recurrence of hemoptysis. The patient ultimately recovered, though she left AMA later in her hospitalization. She was discharged on antibiotics to complete her course and scheduled for cardiology follow-up for future valve replacement. Discussion: This case emphasizes the need to consider mycotic aneurysms in patients with bacteremia and endocarditis who develop new focal symptoms. It also highlights the continued importance of physical exam in directing imaging, and how crucial a multidisciplinary team can be in managing these high-risk lesions.

	Finally, it underscores the expanding role of endovascular techniques as effective alternatives to open surgery in selected patients. Conclusion: In summary, though rare, pulmonary artery mycotic aneurysms carry substantial mortality risk. Early recognition can be lifesaving, and leaning on the experts around you can ensure positive outcomes for our patients.
Rebecca Windschitl Dr. Anya Jamrozy	*Confused but Not Cirrhotic: Uncovering an Unusual Cause of Hyperammonemia* Introduction: Altered mental status is a frequent complaint that brings patients to the emergency department. Evaluation often includes checking an ammonia level. When this is elevated, cirrhosis is the underlying etiology in 90% of cases. This case details an unusual cause of elevated ammonia. Case Presentation: A 58-year-old male with end stage renal disease on hemodialysis after a failed deceased donor kidney transplant, type 1 diabetes with successful pancreas transplant, and coronary artery disease with coronary artery bypass presented to the emergency department from his dialysis unit for altered mental status. Initial vitals were overall normal. On exam he was awake but confused and not answering questions appropriately. An ammonia of 148 in the setting of otherwise normal liver function was noted. A urea cycle disorder was considered but thought less likely with the patient's age and normal glutamine, glycine, and carnitine. Urine orotic acid and organic acids were unable to be collected due to anuria. Treatment at that time included continuous renal replacement therapy, in addition to lactulose and rifaximin for cryptogenic hyperammonemia. Unfortunately, after this he had numerous life-threatening episodes of encephalopathy from hyperammonemia. His presentations were thought to be from bacterial overgrowth of urease-producing organisms residing within the patient's pancreaticoduodenal pouch, with acute exacerbations caused by upper gastrointestinal bleeding increasing protein delivery to the causative organisms; he was empirically given doxycycline to treat bacterial overgrowth without recurrence of events. Subsequently a liver biopsy showed nodular regenerative hyperplasia, a non-cirrhotic cause of portal hypertension associated with solid organ transplant patients that, in advanced stages, can cause portosystemic shunting and hyperammonemia as a result. Discussion: Four known sources of ammonia metabolism defects include infection with urease-producing organism, undiagnosed inborn errors of metabolism, acquired urea cycle dysfunction caused by clinical exposure (valproic acid, 5-FU containing chemotherapy), and otherwise unexplained urea cycle dysfunction. Many patients initially present with altered mental status, seizure, or coma. Risk factors

	include severe protein malnourishment, post-transplant or gastric bypass patients, and valproate use. Helpful blood tests include zinc, amino acids, acylcarnitine profile, free and total carnitine, and creatinine kinase. Urine studies including orotic, organic, and amino acids are also helpful. Treatment is not well understood but includes methods to remove ammonia from the blood (renal replacement therapy, nitrogen scavenger therapy), reduce ammonia gut recirculation (rifaximin or lactulose), reduce exogenous protein catabolism (IV dextrose and intralipids), support urea cycle function (amino acids), and empiric treatment of urease-producing organisms. Conclusion: Recognizing noncirrhotic hyperammonemia and conducting an appropriate work up could help expedite treatment and reduce complications including cerebral edema and death.
Stefanie Worwa Dr. Sahar Koubar	*From Renal Decline to Delivery: A Case of High-Intensity Dialysis in a Pregnant Woman with End-Stage Diabetic Nephropathy* Introduction: Pregnancy in women with any stage of chronic kidney disease (CKD) poses significant challenges due to increased maternal and fetal risks, including higher rates of pre-eclampsia, cesarean delivery, preterm birth, intrauterine growth restriction, and neonatal ICU admission. These risks are proportional to the stage of CKD, the degree of proteinuria, and the level of blood pressure control. Case Presentation: We present the case of a 29-year-old G3P0020 woman with hypertension, hypothyroidism, congenital left ureteropelvic junction obstruction post-pyeloplasty, and longstanding uncontrolled type 1 diabetes mellitus (A1C > 10% 2013-2020) complicated by diabetic nephropathy and CKD stage 3B (pre-pregnancy baseline creatinine 1.7-1.9 mg/dl). At 22 weeks of gestation, she was admitted with worsening hypertension, severe nephrotic-range proteinuria (24-hour urine collection 21 grams), and rising serum creatinine (3.1 mg/dl). She underwent a kidney biopsy, which showed severe diabetic nodular sclerosis and significant scarring with no signs of preeclampsia. Her escalating blood urea nitrogen (BUN) levels raised concerns about fetal well-being, prompting initiation of intensive hemodialysis (6 days per week, 4 hours each session) targeting a mid-week pre-dialysis BUN level around 40 mg/dl. Her pregnancy progressed uneventfully after dialysis initiation, with good metabolic and blood pressure control. She was induced to deliver at 34 weeks as her diabetic retinopathy made it difficult for her to drive to dialysis. Ultimately, labor induction failed, and she underwent a Cesarean section. She delivered a healthy male infant with appropriate weight for gestational age. She remained off dialysis postpartum with stable biochemical parameters and absence of uremic symptoms, although her CKD persisted at stage V, prompting her placement on the kidney-pancreas transplant waiting list, where she remains today, 2 years after delivery.

	Discussion: An increasing number of babies are now being born to women with kidney disease, thanks to advances in dialysis techniques, medical management, and neonatal care. This case demonstrates that with intensive dialysis protocols and targeted BUN levels, successful fetal outcomes are achievable even in women with advanced CKD. It also highlights the importance of early intervention and individualized dialysis regimens dependent on residual renal function. Several studies have shown that BUN levels between 35-48 mg/dL are optimal for improved fetal and maternal outcomes. BUN acts as a surrogate marker for other uremic toxins such as hippuric acid, indoxyl sulfate, and kynurenic acid. These nitrogenous waste products can cross the placenta and adversely affect the placental function and the intrauterine environment, resulting in worse pregnancy outcomes. BUN can also act as an osmotic molecule, leading to increased fetal urine production, resulting in polyhydramnios and preterm labor. Therefore, maintaining lower BUN levels through intensified dialysis is associated with better metabolic control, prolonged gestation, and improved fetal outcomes. Given the rising prevalence of CKD among women of reproductive age driven by obesity, hypertension, and diabetes, proactive management strategies during pregnancy are essential. This case advocates for early referral, preconception counseling, and tailored dialysis approaches to optimize both maternal and fetal health in pregnancies complicated by CKD. Further research is needed to refine dialysis targets and protocols to enhance outcomes in this high-risk population. Conclusion: Successful pregnancies in women with advanced CKD are possible with careful planning and meticulous multidisciplinary management The most common trigger for dialysis initiation in pregnant women is a high BUN. It is recommended to keep mid-week pre-dialysis BUN level < 35-50 mg/dL. The standard dialysis regimen (3x/week) is insufficient in pregnancy because it cannot adequately control BUN. Intensive dialysis (6x/week) can mimic normal renal physiology, optimizing fetal growth and intrauterine stability.
Kevin Wolfe	*Fat Embolism Syndrome: A Rare but Severe Complication of Sickle Cell Crisis* Introduction: Fat embolism syndrome is a rare complication of sickle cell disease associated with significant morbidity and mortality. While hemoglobin SC disease is typically associated with a more indolent disease course than hemoglobin SS disease, it is more likely to precipitate avascular necrosis in sickle cell crisis. 1 This can lead to fat embolism syndrome, a life-threatening complication of bone marrow necrosis.

	Case Presentation: This case describes a patient with HbSC disease who developed fat embolism syndrome requiring veno-venous extracorporeal membrane oxygenation (VV ECMO) support. An otherwise healthy 27-year-old female with HbSC disease presented to the emergency department with dyspnea and poorly localized chest and abdominal pain. Initially she was vitally stable and her hemoglobin was near her baseline of 11.2, however, on the second day of admission she developed hemodynamic instability and her hemoglobin notably dropeed to 6.7. Chest CT revealed new lung opacities consistent with acute chest syndrome, and additional labs were notable for hemolysis. She was emergently transferred to the intensive care unit to facilitate exchange transfusion and received 8 units of pRBCs with subsequent improvement of her Hgb S from 42% to 6% and Hgb improved to 10.3. Despite this, the patient continued to clinically deteriorate and developed acute respiratory distress syndrome, acute renal failure, and multifactorial shock. She subsequently suffered a PEA cardiac arrest necessitating cannulation for VV ECMO. While the etiology of her decompensation was initially unclear, fundoscopic eye exam in the intensive care unit revealed cholesterol deposits consistent with fat emboli. Her trajectory improved and she was able to be decannulated from ECMO after 12 days, however, disseminated intravascular coagulation also developed and the patient developed necrosis of the distal extremities due to microvascular occlusion. Once medically stable, amputations of the bilateral upper and lower extremities were required. Despite imaging showing signs of hypoxic brain injury, she recovered to her neurological baseline, and she was discharged with robust home support and outpatient follow up to be fitted for prosthetics after a 52 day hospitalization. Discussion: This case highlights the profound morbidity and mortality associated with fat embolism syndrome, as well as the diagnostic challenge it presents due to the lack of confirmatory testing and its clinical presentation mimicking other more common pathologies. Conclusion: Clinical decompensation can occur rapidly, and while fat emboli were not immediately recognized in this case, prompt exchange transfusion may have prevented death. Clinicians should be mindful of the possibility of avascular necrosis as a complication of sickle cell crisis, particularly in HbSC disease.