

**American College of Physicians- Minnesota Chapter Annual Abstract Competition**  
**Poster Session**  
**November 1, 2024**  
 Abstracts Submitted for Competition

<b>Medical Students</b>	
<b>Research - Medical Students</b>	
<p><b>Elena Cattaneo</b>            Dr. Ariella Bar-Gil            Shitrit            Dr. Eugenia Shmidt            Dr. Sunanda Kane</p>	<p><i>Outcomes of Multiple Gestation Pregnancies in Inflammatory Bowel Disease</i></p> <p>Background: Given the increasing use and success rates of assisted reproductive technology (ART) in Inflammatory Bowel Disease (IBD), it stands to reason that more multiple gestations will occur. To date there are no data about the outcomes of multiple gestation pregnancies in IBD in terms of maternal or neonatal health in the literature as these are excluded from analyses. We aimed to examine maternal and fetal outcomes in multiple gestation pregnancies from IBD patients and compare them to population-based outcomes in healthy women.</p> <p>Methods: Retrospective chart review of multiple gestation pregnancies in women with IBD who delivered at one of the three participating sites. Medical records were reviewed to identify patients with a history of IBD who had multiple gestation pregnancies. Variables collected included maternal demographics, need for ART, disease-related characteristics and disease activity, maternal complications and neonatal outcomes.</p> <p>Results: Thirty-three multiple gestation pregnancies were identified (mean age 31, range 20-38 yo). Twenty-one women had a history of Crohn’s disease, 11 had Ulcerative Colitis and one had indeterminate colitis. Twenty-one pregnancies occurred naturally and ten occurred with ART. Ten women were on Tumor Necrosis Factor-alpha inhibitor therapy. Of the four women who took steroids during pregnancy, three had active disease with one experiencing fetal distress; one woman on prednisone had maternal hypertension. There were three spontaneous abortions, two with loss of both twins at 10 and 20 weeks and one with demise of one triplet at seven weeks followed by delivery of the remaining two at 28 weeks. Outcomes were compared to population-based data.</p> <p>Conclusions: Among women with IBD with multiple gestation pregnancies, rates of preterm delivery and low birth weight may be higher compared to those reported in literature for non-IBD women. Prospective studies are needed to further characterize the maternal and fetal outcomes in this understudied population.</p>
<p><b>Hyo Bin You</b>            William Chow</p>	<p><i>Predicting 90-day Mortality Following Gamma Knife Procedures: A Data Driven Approach Using Patient-Driven Factors</i></p>

<p>Bobby Do Jenna Schwartz Andrew Pumford Conan Zhao Dr. Ali Gharibi Loron Dr. Michael Keough Dr. Ian Parney</p>	<p>Objective: Brain metastases are a significant cause of morbidity and mortality for one-fifth of cancer patients. Poorer survival outcomes for patients with brain metastases are associated with older age (except in breast cancer), male sex, and low KPS scores. Stereotactic Radiosurgery (SRS) as a safe and effective treatment, with 80% reporting local tumor control. SRS is recommended practice for managing brain metastases in neurosurgical societies consensus guidelines owing to no compromise in survival outcomes as well as no notable increase in neurocognitive toxicities. Although, some studies have investigated patterns of failure in brain metastasis treated with SRS; a practical criterion for selecting patients who are more likely to benefit significantly before mortality remain elusive. This study aims to develop a predictive model to identify patients with an anticipated life expectancy of more than three months, thereby determining those who would benefit from active SRS for brain metastases.</p> <p>Methods: From May 2015 to October 2023, 1546 patients underwent SRS for brain metastases in our institute. 171 patients who survived less than 91 days were identified, the rest of patients were screened considering their age at 1st SRS, gender, and histological diagnosis using the Jaccard distance metric. Therefore, a total of 344 patients were retrospectively analyzed with 171 matched controls. Pre-SRS data included surgical history, neurological deficits, and comorbidities. Procedure data included number of lesions targeted, location of tumors, and margin radiation dose. Post-SRS data included later procedures for the intracranial metastases, cause of death, and post-operative Karnofsky score. These variables were then analyzed through XGBoost (Extreme Gradient Boosting) to predict 90-day mortality.</p> <p>Results: 5-fold cross validation resulted in average accuracy of 81.60% (F1 score = 82.08, MSE = 0.1469). Based on feature importance, the two most significant factors were Karnofsky and frailty scores while the least significant factor was whether &gt;50% of metastases by volume were infratentorial or supratentorial.</p> <p>Conclusions: The machine learning technique employed in this study is an ensemble of weak learners (simple decision trees). Despite such an architecture and with minimum adjustment of default hyperparameters such as maximum depth and L2 regularization, the model was able to achieve high accuracy with low MSE. Analysis of individual features can allow development of a succinct screen panel that could predict mortality. While the performance could be improved, the findings suggest that patient data could be helpful in determining whether stereotactic radiosurgery is the right procedure for a patient with brain metastases.</p>
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**Clinical Vignette - Medical Students**

<p><b>Ashley Battenberg</b> Dr. Allan S. Jaffe Dr. Marysia Tweet  Finalist</p>	<p><i>Challenges with MI in the Elderly – Anemia Driven Ischemia and Role of Dual Antiplatelet Therapy</i></p> <p>Background: The management of antiplatelet therapy in patients with myocardial infarction (MI) and anemia due to gastrointestinal bleeding can be complex.</p> <p>Case Summary: A 90-year-old man with a history of heart failure and occult</p>
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	<p>gastrointestinal bleeding due to diffuse angiodysplasia was hospitalized with chest pain and acute-on-chronic anemia with a hemoglobin of 6.2 g/dL. He was diagnosed with non-ST-elevation MI in the setting of serial increasing troponin T elevations of 168, 224, and peak 353 ng/L (normal &lt;15 ng/L) and new T-wave inversions in the lateral leads on his electrocardiogram. His echocardiogram showed a left ventricular ejection fraction of 54% with anterior left ventricular regional wall motion abnormalities. MI etiology was thought possibly secondary to oxygen supply/demand mismatch and his symptoms improved after transfusion of 3 units of packed red blood cells. However, type 1 MI could not be definitively ruled out. Prior to invasive coronary angiography, he underwent colonoscopy, which showed angiodysplasias that were treated with argon beam coagulation and clipping. Afterwards, given that these lesions can be diffuse, he was challenged with dual antiplatelet therapy (DAPT) and maintained a stable hemoglobin. Subsequent coronary angiography revealed significant coronary artery disease with a focal LAD aneurysm amenable to percutaneous coronary intervention (PCI). Due to the concern of occult bleeding, he was continued on the DAPT challenge and scheduled for outpatient PCI. However, his hemoglobin decreased from 9.8 to 6.6 g/dL over the course of 1 week prompting cessation of clopidogrel. He had further decline of 1 g/dL in the following week which prompted cessation of aspirin. This led to a decision against PCI. Since DAPT cessation, his hemoglobin has remained stable in 3 months follow-up, and his symptoms have responded to uptitration of anti-anginal therapy.</p> <p>Discussion: In this case, treating the primary cause of myocardial injury, the patient’s anemia, was crucial. PCI could have reasonably reduced ischemic risk in this patient with an LAD lesion corresponding to the regional wall motion abnormalities, and regardless of PCI, DAPT therapy is typically recommended for one year after myocardial infarction. However, the danger of a subsequent ischemic event seemed most related to the possibility of future bleeding in this elderly patient. The risks of future gastrointestinal bleeding outweighed the anticipated benefit of PCI and prolonged DAPT. In addition, the patient preferred to remain off antiplatelet therapy. If antiplatelet monotherapy is considered in the future, clopidogrel monotherapy would be preferred due to recent data indicating that P2Y12 inhibitors are associated with less bleeding and similar efficacy when compared to aspirin.</p>
<p><b>Hamza Hai</b> Dr. Christopher Kraemer Dr. Mithun Suresh</p>	<p><i>Refractory Cryptococcal Meningitis in Pregnant Patient</i></p> <p>Case Summary: A 28-year-old female, G3P2 at 18w3d gestation initially presented with headache, nausea, and vomiting. Cerebral spinal fluid (CSF) analysis was positive for cryptococcus antigen, and she was diagnosed with cryptococcal meningitis.</p> <p>Methods: Autoimmune work-up, including HIV testing was all negative. Accordingly, the only risk factor that precipitated this infection was felt to be related to pregnancy. Due to being pregnant, treatment options were limited, and she was started on amphotericin B. Fetal MRI showed multiple abnormalities in the fetus, possibly due to the cryptococcus infection itself, and after consultation with specialists, the patient decided to terminate the</p>

	<p>pregnancy. Following termination of pregnancy, fluconazole and flucytosine, which could not be used during pregnancy, were added to amphotericin B. Anti-fungal agents were then consolidated to itraconazole based on minimum inhibitory concentration data, and she was ultimately discharged on itraconazole. Throughout this hospitalization, serial lumbar punctures were performed for management of elevated intracranial pressure due to the infection. Unfortunately, she had a recurrence of her symptoms approximately 4 months later. Lumbar puncture was performed again, and CSF analysis was positive for cryptococcal antigen, and she was diagnosed with cryptococcal meningitis once again. Etiology was felt to be related to the original cryptococcus infection that had not been fully treated. She was reinitiated on flucytosine and amphotericin B, and then transitioned to isavuconazole. Similar to her previous hospitalization, serial lumbar punctures were performed for management of elevated intracranial pressure. Following discharge, she was followed closely as an outpatient by infectious diseases. With infectious diseases, the original plan was to treat with isavuconazole for 6 months, but because the serum cryptococcus antigen titers continued to remain positive even after 6 months, plans were made to continue treatment with isavuconazole for another 6 months. Finally, after completing treatment with isavuconazole for 1 year, her serum titers were negative.</p> <p>Case Summary: This case highlights the complexity of managing cryptococcal meningitis. The patient's recurrent and refractory meningitis, initially occurring during pregnancy, posed unique challenges for treatment. Aggressive antifungal therapy, typically involving flucytosine and fluconazole in addition to amphotericin B, could not be pursued initially due to the patient's pregnancy. Then, persistently positive antigen titers warranted prolonged treatment with antifungal medications.</p> <p>Conclusion: Future research should focus on improving diagnostic tools, including rapid detection and biomarker identification for early relapse. Treatment innovations are needed, such as less toxic antifungals and combination therapies, alongside immunotherapy to enhance treatment efficacy. Better strategies for managing increased intracranial pressure and preventing relapses should be prioritized. Some of the facts related to this case may have been changed to protect the identity of the patient and per institutional guidance and policy.</p>
<p><b>Kiyan Heybati</b>  Dr. Eric Zuberi  Keshav Poudel  Domenic Ochal  Dr. Elizabeth Dineen  Dr. Bryan Taylor  Dr. Sam Farres  Dr. Kevin Landolfo  Dr. Brian Shapiro</p>	<p><i>Re-heart-ening Exercise: Post Cardiac Rehabilitation Following Acute Thoracic Aortic Dissection with Residual</i></p> <p>Background: Exercise-based cardiac rehabilitation (CR) has been associated with lower incidence of major cardiac events and higher health-related quality of life (HRQoL). The 2022 ACC/AHA guidelines recommended an exercise intensity of 3-5 metabolic equivalents of task (METs) for the post-operative management of patients with thoracic aortic dissection. However, many patients do not engage in exercise due to provider and/or patient concerns about elevated blood pressure, especially in those with residual</p>

<p>Finalist</p>	<p>dissections, despite no clear association with recurrence or progression of dissection.</p> <p>Case Presentation: A 41-year-old male with a history of hypertension (managed with a beta-blocker and calcium channel blocker), coronary artery disease, and class III obesity presented to the emergency department with sharp, non-radiating substernal chest pain, 2/6 diastolic murmur best heard at the right upper sternal border, and new T-wave inversions on baseline electrocardiogram. Initial and repeat troponin levels were 8 and 8, respectively. Imaging revealed an acute thoracic aortic dissection extending from the sinotubular ridge to the bilateral iliac arteries, involving the left kidney, mesentery, and lower extremities. He required replacement of the ascending aorta and hemiarch, along with right lower extremity fasciotomy. Post-operative transthoracic echocardiogram revealed an ejection fraction of 52% with mild aortic regurgitation, and CT imaging demonstrated residual dissection of the descending thoracic aorta.</p> <p>After discharge, he was referred to cardiac rehabilitation, enrolling 36 days following surgery. His rehabilitation program included three 35-minute in-person sessions and three additional home sessions, totaling six active days per week. During rehabilitation, he reported perceived exertion levels of 10-14 (Borg scale 6-20), achieved peak exertion of 4.7 METs, and had a maximum exercise heart rate of 142 bpm (79.2% predicted) and blood pressure of 142/70 mmHg. He completed 32 sessions and reported only mild symptoms, specifically intermittent aches in his right lower extremity both at rest and with exertion. Compared to pre-rehabilitation assessments, he showed considerable improvements: his 6-minute walk distance increased from 230.1 to 487.7 meters, depressive symptoms (PHQ-9 score) decreased from 4 to 0, HRQoL (Dartmouth index) improved from 25 to 14, body mass reduced from 125.1 to 108.9 kilograms, and resting blood pressure remained stable (100/66 to 112/58 mmHg). At 24 months of follow-up, there were no documented adverse events, with no recurrence or progression of dissection noted on imaging.</p> <p>Key Takeaways: Following thoracic aortic dissection repair, despite limited exercise intensity, CR improved HRQoL and functional capacity. While the patient had a residual dissection following initial surgery, there were no adverse events at 24 months of follow up. In addition to routine imaging and close specialist follow up, internists caring for patients with prior thoracic aortic dissections should engage in discussions about the risks and benefits and subsequently consider referrals for cardiac rehabilitation. Further research is needed to better understand patient outcomes and establish evidence-based safety margins for exercise in this population.</p>
<p><b>Catherine Knier</b>  Dr. Kiyam Heybati  Dr. Ben Luek  Dr. Joey Maiocco  Dr. Victoria Kalinoski-Dubose  Finalist</p>	<p><i>When Things Don't Add Up, Start Subtracting: A Case of Hyponatremia and Azotemia</i></p> <p>Introduction: Hyponatremia in the setting of high levels of serum urea (azotemia) presents with elevated serum osmolality (S<sub>osm</sub>). Unlike other causes of hypertonic hyponatremia, such as elevated blood glucose, urea is not osmotically active. Urea raises the measured S<sub>osm</sub> resembling a hypertonic state, but the effective S<sub>osm</sub> is actually reduced. Corrected S<sub>osm</sub> can be calculated as [measured S<sub>osm</sub>-BUN/2.8]. If the corrected S<sub>osm</sub> is</p>

	<p>hypotonic (&lt;275), the next best step to identify the underlying etiology is a thorough history and physical exam to assess volume status.</p> <p>Case Presentation: A 78-year-old male presented to the ED after outpatient labs demonstrated hyponatremia and acute kidney injury on top of pre-existing chronic kidney disease. Pertinent medical comorbidities included heart failure with mid-range ejection fraction of 46% on torsemide, spironolactone, and carvedilol, persistent atrial fibrillation on amiodarone, and hypothyroidism. He was seen in clinic the day before for hypotension (88/49) with lightheadedness on standing. He described a month of progressive generalized weakness, decreased oral intake, and less frequent urination, but no change in mental status. He shared he had stopped taking his levothyroxine a few weeks prior. Admission weight was 3kg below his reported dry weight and diagnostics including ECG were unchanged from prior. In addition to electrolyte abnormalities, labs revealed a S<sub>osm</sub> of 311mOsm/kg. BUN was 131mg/dL and urine osmolality was 313mOsm/kg. TSH was elevated though downtrending at 72.1mIU/L, but T4 was within normal limits. On admission, levothyroxine was restarted. His guideline-directed medical therapy was put on hold while he recovered from the AKI. He responded well to initial management with fluid resuscitation including 50mL of 3% saline and 1L normal saline. He received a short course of sodium chloride tablets while encouraging PO intake and advancing nutrition. Blood pressure and heart rate remained stable. He was asymptomatic and euvolemic for the remainder of the hospitalization. Electrolytes and renal function slowly trended towards normal and he was discharged to a skilled nursing facility for continued rehabilitation 4 days after admission.</p> <p>Discussion: In patients presenting with both azotemia and hyponatremia, high urea in the serum masks the expected drop in osmolality seen in true (hypotonic) hyponatremia. Since urea is an ineffective osmole, S<sub>osm</sub> must be corrected to account for azotemia. After correction, this patient’s serum was indeed hypotonic (311-131/2.8=264). Determining the volume status using history and physical exam revealed a month of decreased intake/output and orthostatic symptoms on diuretic therapy. High urine osmolality in the absence of weight gain, JVP elevation, or edema was consistent with a hypovolemic state. The mainstay of treatment is volume resuscitation. Interestingly, in the treatment of hyponatremic patients with azotemia, azotemia has been found to be protective against osmotic demyelination syndrome (ODS) and dialyzing can actually re-introduce the risk of ODS.</p>
<p><b>Christopher Kraemer</b>  Dr. Mithun Suresh  Hamza Hai</p>	<p><i>An Innovative Strategy for Providing Timely Interventional Cardiology Care Via a “Round Trip” in a Patient with Acute Coronary Syndrome</i></p> <p>Introduction: During the COVID-19 pandemic, many hospitals frequently lacked bed capacity to accept patients due to being full or not having enough staff to care for patients. This was particularly true in rural areas of Minnesota. Accordingly, many rural health systems developed innovative strategies to ensure that timely care was being delivered, particularly for time sensitive conditions. One of these strategies developed by our health system was a “round-trip” service for critical access hospitals in Minnesota where patients hospitalized at another facility could send their patients to our hospital to have certain select procedures, and then return to the</p>

	<p>transferring hospital for ongoing cares. This service could be utilized when our hospital lacked capacity to accept new patients for direct admission. This service was utilized by many procedural subspecialties, including interventional cardiology, and the following case describes the use of the round-trip service in a patient needing a coronary angiogram.</p> <p>Description of Case: 78-year-old female with no known past medical history developed sudden onset, crushing, substernal, and left-sided chest pain that radiated to her jaw in the evening while hospitalized in the medical floor at a critical access hospital for dehydration. ECG demonstrated normal sinus rhythm with no acute ST-T wave changes. Troponin I was elevated at 0.98 ng/mL (normal is less than 0.04 ng/mL). Transfer was sought to a facility with a cardiac catheterization laboratory due to concerns for acute coronary syndrome, but no hospitals with vacant beds could be found. Nitroglycerin and heparin infusions were started and Troponin I peaked at 1.39 ng/mL overnight. In the morning, a hospital with a cardiac catheterization laboratory and a vacant bed still could not be found, but chest pain continued to be present. In addition, the repeat ECG demonstrated new T wave inversions in the precordial leads concerning for active ischemia. So, arrangements were made for a round-trip coronary angiogram at our hospital. After arrival at our hospital, a coronary angiogram followed by percutaneous coronary intervention was performed with drug-eluting stent placement to the left anterior descending and right coronary arteries. Following the procedure, the patient returned to the transferring facility for ongoing care, with cardiology post-procedure rounding via telemedicine.</p> <p>Discussion: The COVID-19 pandemic forced health systems to develop innovative strategies to care for patients. This case highlights the use of one of these strategies, a round-trip service, to perform a time sensitive intervention, a coronary angiogram, in a patient with acute coronary syndrome. Our health system has subsequently used this care strategy to help patients hospitalized at rural and critical access hospitals and needing other time sensitive procedures, including endoscopy, cystoscopy, bronchoscopy, and procedures with radiology.</p> <p>Note: Some of the facts related to this case may have been changed to protect the identity of the patient and per institutional guidance and policy.</p>
<p><b>Caryn Libbert</b> Dr. Erica Levine</p>	<p><i>Tissue is the Issue: A Rare Diagnosis of Diffuse Large B Cell Lymphoma in a Patient with Behcet's Disease</i></p> <p>Introduction: Behcet's disease is a rare systemic vasculitis most often presenting with recurrent oral ulcers, genital ulcers, and ocular lesions, as well as skin manifestations. While exact pathogenesis is unknown, it involves immune system dysregulation and has been linked to an increased risk of malignancy including diffuse large B cell lymphoma (DLBCL).</p> <p>Case Presentation: A 71-year-old male presented with six weeks of fatigue, weight loss, and intermittent fever. Medical history was significant for Behcet's disease, well controlled on immunosuppressants. He had two recent hospitalizations in the month prior. Initial imaging revealed large pulmonary nodules. Treatment included antibiotics and antifungals. Immunosuppressants were held. Work up was unrevealing, including</p>

	<p>extensive infectious work up, core needle biopsy, and endobronchial biopsy consistent with necrosis but negative for malignancy. He was discharged on a rapid steroid taper but represented later that week with weakness. Steroids were increased and he was discharged on a prolonged taper. Despite this, he represented two and a half weeks later and was readmitted. Labs revealed elevated inflammatory markers and imaging redemonstrated bilateral pulmonary nodules, necrotic mediastinal lymph nodes, and an abdominal aortic thrombus. High dose steroids were continued and heparin was started. Another episode of fever and altered mental status again raised concern for infection. Broad spectrum antibiotics were reinitiated and steroids were de-escalated. A third tissue biopsy was obtained via fine needle aspiration which revealed candida and actinomyces. Bronchoalveolar lavage was positive for aspergillus. Antifungal coverage was added. Pathology revealed necrosis with no evidence of malignancy.</p> <p>PET was obtained given ongoing consideration for malignancy. This revealed hypermetabolic pulmonary masses and lymph nodes. A fourth tissue specimen was obtained via a second core needle biopsy. While awaiting pathology results, he continued to decline with new onset atrial fibrillation and worsening respiratory status prompting transfer to the Intensive Care Unit. Echocardiogram revealed a pericardial effusion which progressed to tamponade. He also developed a large pleural effusion. Pathology results returned concerning for B cell malignancy. Definitive diagnosis could not be made due to tissue necrosis. Pericardial and pleural fluid pathology had no evidence of malignancy although repeat thoracentesis had atypical lymphocytes favoring a neoplastic process. In light of the new results, wedge resection or excisional lymph node biopsy were considered. However, given his critical illness, additional tissue was obtained with a third endobronchial biopsy, which again revealed only necrotic tissue. Ultimately, an excisional lymph node biopsy was pursued which revealed a definitive diagnosis of EBV-positive DLBCL. He unfortunately had continued clinical deterioration and passed away shortly after.</p> <p>Discussion: This case illustrates the rare diagnosis of DLBCL in a patient with Behcet’s disease. The initial presentation warranted consideration of multiple etiologies to prevent premature closure and a missed diagnosis. It is important to recognize the increased risk of hematologic malignancies, including DLBCL, in multiple autoimmune disorders. In addition, obtaining a definitive diagnosis was complicated by multiple negative biopsies. Clinicians should consider escalation to an excisional lymph node biopsy when there is high concern for malignancy and negative specimens from less invasive methods.</p>
<p><b>Julia Meyer</b> Dr. Sharon Li Dr. David Perlman</p>	<p><i>Pheochromocytoma Presenting with Cardiogenic Shock and Acute Decompensated Heart Failure</i></p> <p>Introduction: Pheochromocytomas are rare adrenal tumors that secrete catecholamines, typically presenting with episodes of hypertension, headaches, sweating, palpitations, and hypermetabolism. We present a case of a previously healthy woman with a large adrenal mass who presented with cardiogenic shock.</p> <p>Case Presentation: A 43-year-old female with a medical history significant</p>



	<p>for hypertension, prolonged QT syndrome, and stress cardiomyopathy presented to the emergency department (ED) with chest pain, abdominal pain, nausea, episodic tachycardia, and hypertension. She was found to have hypokalemia (potassium 2.8 mmol/L), elevated troponin (1.16 ng/mL, reference range 0.00-0.03), and lactic acidosis (12.8 mmol/L). Electrocardiogram showed no ST segment changes but revealed new T wave inversions. Computed tomography angiogram of the chest, abdomen, and pelvis was obtained and showed a 9 centimeter right adrenal mass abutting the right adrenal gland, liver, and right kidney concerning for neoplasm. Laboratory workup was sent to assess for catecholamine-secreting tumor.</p> <p>The patient was admitted to the intensive care unit for management of shock. Further workup included a transthoracic echocardiogram (TTE), which showed a severely depressed left ventricular ejection fraction (LVEF) of 15% in a pattern consistent with stress-induced cardiomyopathy. The patient underwent an emergent right heart catheterization on hospital day 1, which revealed elevated pulmonary capillary wedge pressure of 20 mm Hg and diminished cardiac index of 1.2 L/min, findings consistent with cardiogenic shock with biventricular dysfunction. Clinical suspicion was high for catecholamine-driven stress cardiomyopathy in the setting of a pheochromocytoma. Her plasma metanephrines and normetanephrines were found to be elevated, but these results were difficult to interpret because she was receiving exogenous epinephrine and dobutamine for hemodynamic support. Supportive cares were initiated with inotropes, vasopressors, and aggressive volume resuscitation. Her clinical picture and hemodynamics slowly improved over the next 2 days, and repeat TTE on hospital day 3 showed a recovered LVEF of 55-60%. She was discharged on hospital day 6 with cardiology and endocrinology follow-up.</p> <p>Three days after hospital discharge, the patient again presented to the ED with chest pain, headache, palpitations, nausea, and weakness. She was found to be severely hypertensive, initially 172/116, and bradycardic. Plasma metanephrines and normetanephrines levels were re-sent prior to initiation of vasopressors and found to be elevated, confirming the diagnosis of pheochromocytoma. Alpha blockade was initiated with doxazosin and metyrosine, and carvedilol was used for perioperative beta blockade. Surgical resection of the pheochromocytoma via right adrenalectomy was successful, and she was discharged on postoperative day 4 in stable clinical condition.</p> <p>Discussion: This case highlights an atypical presentation of pheochromocytoma, in which recurrent catecholamine surge resulted in stress cardiomyopathy that progressed to cardiogenic shock and acute decompensated heart failure. Symptoms associated with pheochromocytomas are usually intermittent and chronic over time but can develop into life-threatening crises in rare cases such as this. For these patients, lifelong follow up with laboratory testing and imaging is required for both hereditary and sporadic variants of the disease due to ongoing, significant risk of tumor recurrence following resection.</p>
<p><b>Abdullah Nacer</b> Ahmad Zaro</p>	<p><i>Unique Complication of Kyphoplasties: A Pulmonary Emboli Made of Cement</i></p>

<p>Dr. Michael Schnaus Dr. Maria Teresa Gonzalez-Bolanos Dr. Natalie Wilson</p>	<p>Background: Kyphoplasties are a common neurosurgical procedure done to relieve compression fractures of the spinal vertebrae. There have been reported cases in which cement injected into the bones leaked into the pulmonary circulation, forming a pulmonary cement embolism.</p> <p>Case Presentation: Our patient is a 77-year-old female with a past medical history significant for osteoporosis and stage IV colon cancer. A routine CT done to monitor her chemotherapy progress showed compression fractures at T11, L4, and L5. She was sent to the Emergency Department and subsequently admitted. Neurosurgery performed 3 image-guided kyphoplasties at the respective locations. Excellent fill was noted at L4 and L5, while good fill was noted at T11. Post-op Day 1 and 2, the patient was doing well and vital signs were stable. Post-op Night 2, the patient developed acute respiratory failure and was placed on 10+ liters of supplemental O2. A thoracic X-Ray showed a new opacity in the right lung base, suspicious for post-op pneumonia. Bilateral lower extremity dopplers were obtained and ruled out DVT. A CT pulmonary angiogram was then obtained to evaluate for a pulmonary embolism. This showed evidence of a linear pulmonary cement embolism in the right pulmonary artery extending into the lobal, segmental, and subsegmental pulmonary artery branches of the right upper lobe, as well as the lobal and segmental pulmonary branches of the right lower lobe, along with a mixed attenuation consolidation in the right lower lobe as seen on her thoracic x-ray. The patient's right ventricle did not demonstrate evidence of strain on CT imaging. Thrombectomy was discussed with the pulmonology team, but was ultimately decided against due to the patient's hemodynamic stability. Anticoagulation with heparin was initiated in the hospital given the patient's elevated bleeding risk with advanced colon cancer, and after proving stability in blood counts, the patient was sent home with Eliquis for 6 months.</p> <p>Conclusion: As demonstrated in this case, post-kyphoplasty patients are at risk for cement leakage into their pulmonary circulation. As such, any patients presenting with acute respiratory symptoms following a kyphoplasty should be worked up for pulmonary cement embolism. Existing literature shows a PCE incidence rate of up to 23% following a kyphoplasty<sup>1</sup>, and patients may even be asymptomatic for up to 5 years before presenting with pulmonary symptoms, so a kyphoplasty at any point in the patient's past should be cause for suspicion if presenting with unexplained shortness of breath or hypoxemia, or unexplained hyperdensities seen on chest radiographs. Thorough chest X-Ray readings should be sufficient to identify a PCE<sup>3</sup>, but a pulmonary CT angiogram with contrast may be helpful if X-Ray comes back inconclusive or definitive angiographic imaging is needed. Anticoagulation therapy should be considered for PCEs presenting with pulmonary symptoms, but current literature does not show strong evidence for anticoagulation treatment for asymptomatic PCEs.</p>
<p><b>Aishwarya Pradeep</b> Dr. Eric Zuberi Dr. Jennifer Cowart</p>	<p><i>Chronic, Nonspecific, Abdominal Symptoms: A Rare Case of Difuse Lymphangiomas Causing Transient Mesenteric Ischemia in a 65-Year-Old Woman</i></p> <p>Introduction: Abdominal lymphangiomas are benign cystic malformations of lymphatic vessels. Patient presentation ranges from incidental findings noted on imaging to gradual onset of symptoms such as abdominal pain,</p>

back pain, nausea, vomiting, palpable abdominal masses, ascites, change in bowel habits, or fever. Approximately 65% of abdominal lymphangiomas are congenital and rarely occur in adulthood. Early detection is helpful as abdominal lymphangiomas can exert a mass effect on surrounding structures, however management can be challenging in cases of diffuse spread with vascular compression.

Case Presentation: We present the case of a 65-year-old woman who arrived to the emergency department with severe abdominal pain, cramping, transient episodes of orthostatic hypotension, constipation, and diarrhea. She was hemodynamically stable, and her physical exam was remarkable for moderate, diffuse abdominal tenderness. Computed tomography (CT) of abdomen and pelvis with contrast revealed wall hypoenhancement of the rectosigmoid colon which was further evaluated with CT angiography (CTA) with contrast to assess for chronic mesenteric ischemia. The CTA revealed a long segment of hypoenhancing sigmoid colon with a soft tissue density surrounding the inferior mesenteric artery (IMA) and its branches supplying the hypoenhanced bowel. Her lactate was 1.3 mmol/L.

Vascular surgery and gastroenterology teams were consulted. Since the patient was hemodynamically stable with a normal lactate, no immediate surgical intervention was recommended. Flexible sigmoidoscopy revealed patches of erythematous mucosa with no ulcerations, bleeding, or masses, and normal biopsies. All vasculatures were patent. We suspected ongoing intermittent ischemia causing transient orthostatic hypotension secondary to compression of mesenteric vasculature by the soft tissue density. She was discharged after three days of hospitalization with planned outpatient magnetic resonance imaging (MRI) of the abdomen to evaluate the tissue density. This revealed diffuse lymphangiomatosis involving the retroperitoneum, retrocrural space, and abdominopelvic mesenteric vessels. The patient was evaluated by gastroenterology, who recommended symptomatic management given involvement of abdominal vasculature and diffuse spread of lymphangiomas.

Discussion: Abdominal lymphangiomas are exceedingly rare in adults. While resectable, surgical excision is challenging when they are present near mesenteric vasculature such as IMA branches as seen in this patient, which can increase risk of hemorrhage. The diffuse nature in this patient made her a poor candidate for surgery and other evidence-based interventions such as sclerotherapy. Sirolimus has proved successful in newborns, however her history of hyperlipidemia prevented use given its adverse effect of dyslipidemia. Abdominal lymphangiomas do not have metastatic potential but rather a mass effect.

This unusual case demonstrates the importance of considering abdominal lymphangiomatosis when evaluating a patient with persistent episodes of orthostatic hypotension, chronic abdominal pain, constipation and diarrhea refractory to evidence-based management. Patients may present with a wide range of non-specific symptoms and a low threshold must be maintained to obtain immediate imaging. CT and MRI of the abdomen and pelvis, especially angiography, can help identify masses proximal to vasculature. This unique presentation warrants multidisciplinary

	<p>involvement of hospitalists, gastroenterologists, and vascular surgeons. Management differs on a case-by-case basis given currently limited guidelines and depends on the nature and characteristics associated with the condition.</p>
<p><b>Paige Stueve</b> Dr. John Meisenheimer Dr. Noah Goldfarb</p>	<p><i>Super Vena Cava (SVC)</i></p> <p>Learning Objectives: Recognize SVC syndrome as a potential complication of pacemaker lead occlusion. Understanding complexities of managing SVC syndrome in older adults with multiple comorbidities.</p> <p>Case Presentation: An 88-year-old male with a history of second-degree AV block (post dual chamber permanent-pace maker placed 11 years prior; abandoned and replaced 6 months prior due to malfunctioning), atrial fibrillation, mitral insufficiency (post-Mitraclip 6 years prior), and hypertension presented with a one-month history of slowly enlarging, asymptomatic neck swelling and infra-orbital edema. Initial examination and CT with contrast revealed nonspecific, uniform thickening of the skin and subcutaneous fat of the neck, with no definitive mass or lymphadenopathy. The patient was referred to otolaryngology, and a laryngoscopy was performed without abnormality. CT neck with contrast indicated worsening soft tissue edema, and also noted suspected severe chronic stenosis of the bilateral brachiocephalic veins and upper portion of the SVC (superior vena cava) leading to SVC syndrome. After consulting cardiology, it was concluded that the occlusion was likely caused by pacemaker lead placement through the right brachiocephalic vein, as the left brachiocephalic was already occluded from a previous lead placement 11 years ago. An angioplasty was under consideration with vascular surgery, but 4 months after initial evaluation, the patient had a stroke. Conservative management and monitoring were chosen due to the high risk of surgical complications and the low likelihood of a lasting treatment response.</p> <p>Discussion: SVC syndrome results from occlusion of the SVC or bilateral brachiocephalic veins, most commonly due to thrombosis or tumor infiltration.<sup>1</sup> Around 70% of SVC syndrome is due to malignancy, but the rate of iatrogenic SVC syndrome is rising.<sup>1</sup> SVC syndrome is a rare complication of cardiac pacemaker leads, occurring in about 1 in 1000 cases.<sup>2</sup> The mean time to develop SVC syndrome is 15 months after pacemaker placement.<sup>2</sup> Development of SVC syndrome in these cases is often gradual, so it is rare for patients to present symptomatically due to the development of collateral vasculature.<sup>2</sup> The most common presenting sign of SVC syndrome is facial edema, as observed in this case. However, other signs include non-pulsatile distended neck and chest veins and upper extremity edema.<sup>1</sup> Other symptoms are rare in pacemaker-associated SVC syndrome, but may include dyspnea, cough, hoarseness, stridor, syncope, headaches, and confusion.<sup>1</sup> SVC syndrome may be life-threatening due to a sudden increase in intracranial pressure leading to cerebral edema.<sup>1</sup> Endovascular therapy is frequently used in the treatment of SVC syndrome, although the presence of pacemaker leads may limit effective treatment options.</p> <p>Conclusion: In patients with pacemakers presenting with facial edema and neck swelling, SVC syndrome should be considered. SVC syndrome can be</p>

	<p>life-threatening, but when associated with pacemaker leads, it is often asymptomatic due to the gradual progression and development of collateral vasculature.</p> <p>References:</p> <ol style="list-style-type: none"> <li>1. Azizi AH, Shafi I, Shah N, et al. Superior Vena Cava Syndrome. JACC: Cardiovascular Interventions. 2020;13(24):2896-2910.</li> <li>2. Bolad I, Karanam S, Mathew D, John R, Piemonte T, Martin D. Percutaneous treatment of superior vena cava obstruction following transvenous device implantation. Catheterization and Cardiovascular Interventions. 2005;65(1):54-59.</li> </ol>
<p><b>Shannon Zhou</b>  Dr. Matthew Young  Dr. James  Leatherman</p>	<p><i>Thoracic Endometriosis: A Case Study of Recurrent Hemopneumothorax</i></p> <p>Introduction: Thoracic endometriosis refers to the ectopic growth of endometrial tissue in the lung parenchyma or on the pleura. The most common manifestation is catamenial pneumothorax, though other presentations may include catamenial hemothorax, hemoptysis, chest pain, lung nodules, and pleural effusion. While there are several reports of catamenial pneumothorax or hemothorax, catamenial hemopneumothorax is a rare manifestation with very few cases reported in the literature. We present a case of recurrent right-sided catamenial hemopneumothorax.</p> <p>Case Presentation: A 44-year-old woman with a history of heavy menses, frequent travel to an African country, and several years of recurrent chest pain with pleural effusions on imaging presented to the emergency department with four days of right-sided, tight, burning central chest pain with radiation to the right shoulder, back, and upper abdomen. She denied hemoptysis. Previous effusions either resolved spontaneously or required symptomatic relief via thoracentesis, occasionally producing bloody aspirate. Tuberculous pleural effusion was initially considered due to her travel history and close-contact exposure, and she was treated empirically despite a negative Quantiferon test. Treatment was prematurely discontinued due to intolerable peripheral neuropathy, and subsequent thoracentesis cultures were negative, making tuberculosis less likely. The effusion eventually resolved spontaneously before further workup. Further history revealed that episodes of hemothorax coincided with menses, raising suspicion for thoracic endometriosis. As such, the patient was started on Lupron for approximately one year, as well as Norethindrone to manage Lupron-induced hot flashes and breakthrough bleeding. However, work-related travel requiring extended stays in Africa interfered with her ability to receive Lupron injections consistently. A missed injection subsequently led to the patient’s presentation to the emergency department with the aforementioned symptoms.</p> <p>On examination, she had decreased breath sounds and dullness to percussion of the right lower lung lobe. Laboratory workup was largely unremarkable with a normal complete blood count and chemistry panel. Chest x-ray and CT with subsequent thoracentesis revealed a large hemopneumothorax. Despite repeated thoracentesis, the hemopneumothorax persisted intermittently over the next three months. She ultimately underwent Video-Assisted Thoracoscopic Surgery (VATS) pleurodesis. Lupron was discontinued because it was ineffective in reducing</p>

further episodes and logistical challenges. Norethindrone, which managed Lupron side effects, was also discontinued. To maintain hormonal suppression and prevent further episodes, she was started on a combined oral contraceptive (norethindrone acetate-ethinyl estradiol-ferrous fumarate). Six months postoperatively, the patient remained free of hemopneumothorax, chest pain, and dyspnea. Follow-up chest x-ray showed postsurgical changes consistent with pleurodesis.

Discussion: We present a rare manifestation of thoracic endometriosis: recurrent catamenial hemopneumothorax. In addition to bringing greater awareness to its clinical presentation, we highlight the importance of obtaining a thorough history, particularly regarding the timing of symptoms in relation to menses. For patients with barriers to medication adherence and persistent symptoms, VATS pleurodesis may be considered earlier to prevent further complications. This case underscores the importance of understanding a patient’s social circumstances and the critical need to include menstrual history when evaluating patients with recurrent hemothorax and/or pneumothorax.

**Quality Improvement - Medical Students**

**Basra Osman**  
Dr. Dame Idossa

*Lost in Translation: The Lack of Validated Translation Tools in Non-English Speaking Research Participants*

Background: Non-English-speaking individuals are underrepresented in clinical trials, in part due to limited availability of translated research documents. Currently, there are no validated tools to assess the accuracy and reliability of translated materials. Inclusion of non-English speaking individuals in research studies requires a concerted effort to create an inclusive environment in clinical research.

Methods: A literature search was conducted to evaluate for validated language tools that can be utilized to conduct forwards and backward translations from English into Somali. Despite searches in databases such as National Institute of Health (NIH), Pubmed and Google Scholar, studies specifically focusing on validated language tools were not identified. Available research documents translated from English to Somali by certified professional translators were then inputted into four different publicly available translation tools (Quillbot, Paperpal, Google Translate and ChatGPT). These four translation tools were chosen from a simple google search of easily accessible language tools available online. The backward translated materials were then analyzed for accuracy by native Somali speaking medical student.

Results: No validated tools examining the accuracy or reliability of translated materials into Somali currently exist. Quillbot and Paperpal translation tools were not able to translate into Somali. The accuracy of the forward and backward translated materials were deemed to be 70% accurate with Google Translate, and 90% accurate with ChatGPT. Google Translate produced some translations that may obscure the intended meaning. For example it translated the original phrase “if you cancel your permission, you will no longer be in the research study” into “if you delete the permission, you will not be there anymore research study.” ChatGPT produced an

	<p>accurate translation that conveyed the same message as the original document, except for some minor alterations in wording. For example, the original research documents used the word “give” while ChatGPT used “obtain.”</p> <p>Conclusion: Minnesota has the largest Somali population in the United States, with a large subset of this population being non-English speaking. The development of validated language tools is urgently needed to ensure that non-English speaking individuals have resources they need to participate in clinical trials.</p>
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**Transitional Medical Graduates**

**Research - Transitional Medical Graduates**

<p><b>Parul Berry</b>  Dr. Kanika Sehgal  Dr. Raseen Tariq  Dr. Darrell Pardi  Dr. Sahil Khanna</p>	<p><i>Real World Experience of Bezlotoxumab for the Prevention of Recurrent Clostridioides Difficile Infection: A Systematic Review and Meta-Analysis</i></p> <p>Objective: Clostridioides difficile infection (CDI) is a leading cause of healthcare-associated diarrhea, and recurrent CDI (rCDI) is a major challenge in treatment. Bezlotoxumab (BEZ), a monoclonal antibody against C. difficile toxin B, has shown efficacy in reducing rCDI in clinical trials. This study aimed to evaluate the real-world effectiveness of BEZ for preventing rCDI through a retrospective analysis of patients treated at our center and a systematic review and meta-analysis of published data.</p> <p>Methods: The study included two parts: a retrospective analysis of patients treated with BEZ at our center between 2017 and 2021, and a systematic review of literature with a meta-analysis. In the retrospective study, patient demographics, CDI risk factors, and outcomes were collected. CDI recurrence was defined as an episode occurring within 8 weeks of the prior infection. Statistical analysis included Kaplan-Meier survival curves and Cox regression models.</p> <p>For the systematic review, databases such as Cochrane Central, Embase, Medline, Scopus, and Web of Science were searched for studies evaluating BEZ in rCDI. Studies were included if they reported on CDI resolution rates and followed patients for at least 8 weeks. Data from selected studies were pooled using a random-effects model, and the weighted pooled resolution (WPR) rates were calculated. Heterogeneity was assessed using the I2 statistic, and publication bias was evaluated through funnel plots and Egger’s test.</p> <p>Results: The retrospective study at our center included 47 patients treated with BEZ, of which 34 (72.3%) achieved CDI resolution, while 13 (27.6%) had recurrent infections. The median time to recurrence was 95 days (IQR 52-216). Patients with more risk factors had a higher recurrence rate; 53% of those with three risk factors experienced recurrence. The systematic review included 28 studies involving 2639 patients, 1786 of whom received BEZ. The pooled analysis showed a CDI resolution rate of 81.6% (95% CI 77.2-85.6%), with significant heterogeneity (I2 = 77.3%). Subgroup analysis comparing BEZ with standard-of-care (SOC) antibiotics alone revealed a pooled relative risk of recurrence of 0.56 (95% CI 0.36-0.88, p&lt;0.01).</p>
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	<p>Patients receiving BEZ with SOC had a higher resolution rate (83.3%, 95% CI 75.5-91.1%) compared to those receiving SOC alone (70.8%, 95% CI 62.7-78.8%).</p> <p>Conclusion: Bezlotoxumab is effective in reducing the recurrence of CDI in real-world settings, with resolution rates comparable to those reported in clinical trials. The meta-analysis supports the use of BEZ in conjunction with SOC antibiotics, but further prospective studies are needed to better understand its role in specific patient populations and its long-term effectiveness.</p>
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**Clinical Vignette - Transitional Medical Graduates**

<p><b>Mohamed Eldesouki</b> Dr. Hazem Abosheaishaa</p>	<p><i>Altered Mental Status on Top of Anaplasmosis-Induced Severe Rhabdomyolysis: A Rare Clinical Presentation</i></p> <p>Introduction: Human granulocytic anaplasmosis (HGA) is a disease caused by tick-borne infection of <i>Anaplasma phagocytophilum</i>. The typical symptoms are fever, malaise, and body aches accompanied by abnormal blood tests such as leukopenia, thrombocytopenia, and transaminitis. Some rare complications may occur, especially in patients living in heavily wooded areas, with a mean age of 70 years.</p> <p>Case Presentation: We present a case of a 67-year-old male who was admitted for lower abdominal pain, fever, and diarrhea with derangement of his blood tests. Despite treatment, his condition deteriorated and complicated rhabdomyolysis and acute kidney dysfunction.</p> <p>Discussion: Empiric treatment including doxycycline was initiated while waiting for the infection blood work results. PCR came back positive for HGA. Empiric therapy was narrowed down to doxycycline for 14 days, and the patient's condition began to improve gradually and steadily. Aggressive hydration markedly improved rhabdomyolysis and, in turn, kidney function.</p> <p>Conclusion: Our case underscores the importance of considering HGA in ambiguous clinical scenarios and highlights the value of early diagnosis, empiric treatment, and intravenous hydration, especially in the presence of rhabdomyolysis.</p>
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<p><b>Damni Advani</b> Dr. Filip Jovanovic Dr. Madhuri Chengappa Dr. Hind Alameddine Dr. Thejaswi K Poonacha</p>	<p><i>A Hypocalcemic Puzzle</i></p> <p>Introduction: We present a case of acute severe and persistent hypocalcemia in the setting of treatment for metastatic prostate cancer. The persistent nature of hypocalcemia despite vigorous replacement points to a unique cause of hypocalcemia. While the osteoblastic nature of metastatic prostate cancer and the calcium chelating dye (EDTA) received can cause hypocalcemia, a less known cause of severe persistent hypocalcemia is use of denosumab, a bone anti-resorptive drug, if given to a patient with low serum calcium level.</p> <p>Case Summary: An 82-year-old male with a history of hypertension, chronic kidney disease, and seizure disorder was admitted to the hospital in July 2024 for severe hypocalcemia. In June of 2024, he was diagnosed with</p>
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	<p>metastatic prostate cancer with extensive skeletal metastases and hydronephrosis of left kidney. Baseline labs on 7/1/24 showed low calcium at 8.2mg/dl (albumin level at 4.1g/dl). The patient received chemotherapy, degarelix along with denosumab on 7/10/24. For hydronephrosis, he underwent an unsuccessful attempt at left ureteral stenting followed by failure of internalization of the percutaneous nephrostomy tube on 7/22/24. On that day, his labs revealed calcium of 5.5 mg/dl.</p> <p>On 7/24/24, calcium was down to 4.6, with ionized calcium at 2.7. The patient was started on IV calcium gluconate, followed by calcium gluconate infusion at a rate of 1 gm/hr., with calcium levels being checked every 4 hours. By the evening of 7/25/24, 11 grams of IV calcium gluconate was infused. Despite this, the patient had a moderate rise in serum calcium (6.6 mg/dl). The patient received 5 grams of IV calcium gluconate on 7/26/24 and 3 grams on 7/27/24, with additional oral supplementation of calcium carbonate 1.5 grams 4 times daily and calcitriol 0.25 mcg twice daily. A 24 hrs. urinary calcium was &lt; 0.8 mg/dl. By 7/29/24, the patient had received a total of 23 grams of IV calcium gluconate. The patient was discharged on 30 July with oral calcium supplements once his calcium levels had stabilized with a corrected calcium level of 8.5 mg/dl. He had another admission for severe hypocalcemia a week later and currently remains on high-dose oral calcium supplementation along with calcitriol.</p> <p>Discussion: While the differential is broad, the development of acute severe hypocalcemia after chemotherapy administration and interventional radiology procedure with EDTA, either the dose of denosumab or EDTA on a background of osteoblastic metastasis are possible causes. With the amount of calcium received, one would have expected the levels to normalize, if the cause was entirely EDTA, whose half-life is 20-60 minutes. With osteoblastic metastasis, a patient already had a baseline low calcium of 8.2 mg/dl, leaving denosumab as a possible cause for persistent, severe hypercalcemia. Denosumab inhibits osteoclast activity, leading to decreased bone resorption and reduction in calcium release into the bloodstream. This mechanism, combined with the effect of denosumab on PTH and vitamin D can disrupt the delicate balance of calcium homeostasis resulting in hypocalcemia. Denosumab can impair the renal synthesis of calcitriol, an active form of vitamin D, which further contributes to the development of hypocalcemia.</p>
<p><b>Ali Babar</b> Dr. Hafsa Shahwaiz Dr. Babar Babak Haddadian</p>	<p><i>Recurrent Effusive Pericarditis After COVID-19 mRNA Vaccination</i></p> <p>Background: With the recent breakthrough novel mRNA vaccines against COVID-19, possible adverse side effects and treatments must be highlighted. One such complication that if not addressed appropriately can have a lasting effect on patient health outcomes and quality of life is the development of acute pericarditis. Acute pericarditis following live virus vaccinations such as influenza, smallpox, and Hepatitis B has been a well-known occurrence. It is now also being observed with the use of COVID-19 vaccinations especially those developed with mRNA. These types of vaccines give your cells instructions for how to make the S protein found on the surface of the COVID-19 virus. CDC's vaccine safety task force has published their safety data that now recognizes the risk of developing pericarditis and myocarditis as a direct result of vaccination. Therefore, an internist needs to have this</p>

	<p>rare complication in their differentials to be able to identify these complications. As the population continues to get vaccinated with COVID-19 vaccinations, we are seeing more and rare side effects with these vaccinations.</p> <p>Case Presentation: Our case highlights a scarce complication of recurrent effusive pericarditis following COVID-19 vaccination. Early detection and management of such similar cases is of imperative importance to prevent any serious health complications. This case also highlights the use of riloncept a novel treatment for recurrent pericarditis in the treatment for recurrent pericarditis after COVID-19 vaccination.</p>
<p><b>Erica Lara Ovaes</b> Dr. Kiril Dimitrov</p> <p>Finalist</p>	<p><i>When the Sun Hurts and the Liver Fails: A Rare Case of Late-Onset XLP</i></p> <p>Introduction: X-linked protoporphyria (XLP) is a rare genetic disorder of the heme biosynthetic pathway, accounting for approximately 10% of cases in the U.S. It results in overproduction and systemic accumulation of protoporphyrin IX (PPIX) in the skin, liver, and red blood cells. Clinically, XLP presents with severe phototoxic reactions similar to erythropoietic protoporphyria (EPP). However, XLP carries a higher risk of protoporphyric hepatopathy, leading to progressive liver disease and, in rare cases, end stage liver disease. Late-onset XLP, particularly in elderly patients, is extremely rare and often under-recognized, with only a few reported cases, all in association with hematologic malignancies. This case describes an elderly female patient with late-onset XLP complicated by acute liver failure.</p> <p>Case Description: A 70-year-old female with a history of invasive ductal carcinoma (in remission post-mastectomy and chemotherapy) and type 2 diabetes, was diagnosed with XLP three years prior. The diagnosis followed the onset of painful photosensitivity and blistering of her hands. Symptoms were managed conservatively with sun protection measures. Genetic testing revealed a previously unreported heterozygous nonsense mutation in the ALAS2 gene, confirming XLP. A year prior to her presenting to our service, she had started workup as an outpatient for progressively worsening nausea and vomiting associated with chronic abdominal pain.</p> <p>She presented to the emergency department with progressive abdominal pain, jaundice, and significant transaminitis (ALP 1069, ALT 230, AST 417, TBili 6.4, DBili 5.09). Elevated lactate levels (11.8) and moderate anemia (Hgb 9.8, MCV 96) with a normal iron panel were also noted. Free erythrocyte protoporphyrin (FEP) levels 12,020 µg/dl. Workup excluded viral hepatitis, and imaging (CT abdomen, RUQ ultrasound) revealed no acute findings. A recent liver biopsy showed severe canalicular cholangiolar and hepatocellular cholestasis with hepatocellular injury, mild fibrosis and characteristic Maltese cross features of protoporphyric hepatopathy on polarizing microscopy. Despite hemin infusions and supportive care, she deteriorated, developing acute hypoxic respiratory failure requiring intubation.</p> <p>Management included red blood cell exchange (RBCE), therapeutic plasma exchange (TPE), and hemin infusions. With this multi-modality approach, her condition stabilized, and she was extubated by day 3 in the ICU. Long-term management included outpatient TPE, RBCE, and a combination of</p>

	<p>cholestyramine, ursodiol, and vitamin E to enhance PPIX excretion with frequency and dosage adjustments as needed. Her symptoms have fluctuated since and have been reflective of her FEP levels.</p> <p>Discussion: Acute liver failure is a rare but life-threatening complication of XLP. This case highlights the importance of early recognition of protoporphyric hepatopathy in XLP patients, especially those with late-onset presentations. The treatment approach for XLP-related liver failure is not standardized, but this case suggests that combined interventions aimed at reducing PPIX levels (e.g. RBCE, TPE, hemin infusions) along with those that increase excretion (e.g.cholestyramine, ursodiol) and reduce ALA synthase activity (vitamin E) can stabilize liver function and improve outcomes. Close monitoring of FEP levels is essential in guiding therapy, and patients with rapidly progressing liver disease should be considered for sequential liver and bone marrow transplant evaluation.</p>
<p><b>Lakshiya Ramamoorthy</b>  Dr. Saman Rashid  Dr. Bharat Duraisamy  Swami Kanna</p>	<p><i>NELL-1 Associated Membranous Nephropathy: A Case Report of Lipoic Acid-Induced Nephrotic Syndrome</i></p> <p>Background: Membranous nephropathy (MN) often arises from secondary causes such as hepatitis, HIV, or heavy metal poisoning, but occasionally occurs without any identifiable underlying cause. Typically, more than 70% of cases are positive for phospholipase A2 receptor 1 (PLA2R1) and thromboplastin type 1 domain-containing protein 7 A (THSD7A). However, emerging evidence suggests associations with novel biomarkers such as NELL-1, NCAM-1, and EXT1/2. Furthermore, lipoic acid, an over-the-counter supplement commonly used in diabetics, has been identified as a potential cause of NELL-1-positive MN, further stressing the need to consider all possible etiological factors.</p> <p>Case Report: Our case report highlights the rare association of membranous nephropathy with the novel biomarker NELL-1, possibly due to lipoic acid as a potential contributing factor and highlights the necessity of a complete diagnostic review for managing nephrotic syndrome for timely initiation of appropriate treatment and to mitigate the progression of this disease.</p>
<p><b>Varshini Srinivasan</b>  Dr. Abhishek Chandra  Dr. Prajith Ramesh  Finalist</p>	<p><i>Vasopressin Withdrawal Induced Diabetes Insipidus: A Case Report of a Rare Complication in Septic Shock Management</i></p> <p>Introduction: Vasopressin has emerged as a standard therapy for hemodynamic support in the management of septic shock. Given its ubiquitous use, it is important to be aware of the potential side-effects and complications of its use. Withdrawal from vasopressin can lead to diabetes insipidus (DI). However, there are few published reports of this complication. We present a case of the rapid development of this complication and detail its pathophysiology to contribute to the growing body of literature.</p> <p>Case Description: A 50-year-old male, with no significant past medical history and taking no home medications, presented to the emergency department with a six hour history of severe right-sided lower-quadrant abdominal pain, localized in the iliac region. On admission his white-blood cell count was 14,000. Ultrasound examination identified a mass in the right</p>

	<p>iliac fossa. A computed-tomography scan of his abdomen revealed an enhancing appendiceal wall with a focal defect, as well as extraluminal gas suggestive of appendicular perforation. He was emergently taken to the operating room. Abdominal exploration confirmed a perforated appendix and an appendectomy was performed.</p> <p>In the immediate postoperative period the patient became altered and was found to be hypotensive with a critical blood pressure of 80/50. With a qSOFA score of 2 he was at high-risk for in-hospital mortality. Appropriate treatment was started, including both noradrenaline and vasopressin. The initial infusion rate for vasopressin was 1 unit/hour and was increased to a peak of 4 units/hour. His sodium on postoperative day 1 (POD 1) was 128 mEq/L. His blood pressure began to improve and the decision was made to taper him off vasopressin on POD 2. Shortly following a six-hour taper, the patient developed polyuria with a urine output of 500-600 ml/hour. At this time his sodium had risen to 140 mEq/L, his urine sodium was 20 mEq/L, and by the end of POD 2 he had fourteen liters of urine output in forty-eight hours. DI was suspected and desmopressin 0.1 mg twice daily was started the same day. On POD 3 his morning sodium was 145 mEq/L, but later in the evening had improved to 138 mEq/L. He was weaned off of desmopressin over the next three days with resolution of his polyuria and with subsequent measurements of serum sodium and urine osmolality normalizing (Figure 1). One month of follow-up in the clinic found no signs of DI.</p> <p>Discussion: This case highlights a rare and serious adverse effect of vasopressin use in the management of septic shock. Endogenous vasopressin levels are markedly decreased in severe septic shock, explaining its utility in hemodynamic support alongside norepinephrine<sup>1</sup>. It is important to monitor for signs of DI following withdrawal of vasopressin. If DI is recognized, prompt treatment with oral desmopressin is recommended.</p>
<b>Residents</b>	
<b>Quality Improvement - Residents</b>	
<p><b>Tessa Herman</b>  Dr. Kawthar Mohamed  Dr. Daniela Guerrero Vinsard  Dr. Morgan Freeman  Amy Gravely  Anders Westanmo  Dr. Lisa Smith  Dr. Mohammad Bilal  Dr. Brian Hanson</p>	<p><i>Time and Experience do not Lead to Improved Adenoma Detection Rate with Artificial Intelligence: An 11-Month Implementation Trial</i></p> <p>Introduction: Artificial intelligence-assisted colonoscopy (AIAC) with computer-aided detection (CADe) technology is designed to improve colonoscopy quality. While randomized controlled trial data is largely positive with improved adenoma detection rate (ADR) using AIAC, real-world implementation trials demonstrate mixed results, including a six-month pre-post implementation trial we previously reported on using two AIAC technologies which showed lower ADR with AIAC. However, current implementation trials are limited by short study durations and thus the impact of AIAC on ADR over time is unknown.</p> <p>Methods: We extended the duration of our single-center, quasi-experimental implementation study to 11 months using one AIAC technology with the aim to evaluate if the ADR improves over time with continued AIAC use and experience. We compared the ADR at</p>

	<p>approximately 6-month intervals after AIAC implementation. Colonoscopies performed for screening, surveillance, or positive fecal immunochemical test indications were included. Secondary endpoints included sessile serrated lesion (SSL), hyperplastic polyp, and benign, non-adenomatous, non-hyperplastic (BNANH) lesion (e.g. lymphoid aggregate, normal mucosa) detection rates. Statistical analysis was performed using Pearson’s chi-square or ANOVA Type III F-test, as applicable.</p> <p>Results: We evaluated 295 AIAC colonoscopies in the first 6-month interval (May-October 2023) and 419 AIAC colonoscopies in the second 5-month interval (November 2023-March 2024). Groups were balanced without differences in patient demographics (age, gender, body mass index), endoscopists involved, or polyps per colonoscopy. ADR did not improve between AIAC cohorts (74.2% versus 74.7%, <math>p=0.89</math>). Similarly, we did not see improvement in other polyp detection rates with continued AIAC use (Table 1), although there was a near-significant trend towards decreased resection of BNANH lesions (36.3% versus 29.4%, <math>p=0.05</math>) and rebounding ADR over time (Figure 1).</p> <p>Discussion: Our real-world implementation study failed to show improvement in ADR with continued use and experience with AIAC at nearly 1 year after its implementation. This is unexpected as we hoped that over time we would have more success with ADR as endoscopists become more familiar and comfortable with the technology. Thus, this finding may reflect a deficit of the CADe technology in detecting adenomatous lesions in our cohort rather than a product of the endoscopist-AI interaction.</p>
<p><b>Cassandra Roeder</b>  Dr. Peter Lund  Jacob Langness  Jihan Eli</p> <p>Finalist</p>	<p><i>Hospital Hypoglycemia: The Sweet Slip of Sulfonylureas</i></p> <p>Background: There is an established risk of hypoglycemia caused by sulfonylureas (SU’s) [1,2]. Professional endocrine organizations recommend against continuing SU’s in the hospital [3]. Though new and better agents are available, patients remain on SU’s in the outpatient setting [4]. An admitted patient at Abbott Northwestern Hospital experienced a hypoglycemia-associated adverse event while on their home sulfonylurea.</p> <p>Aim: Quantify inpatient sulfonylurea use and associated hypoglycemia across Allina hospitals.</p> <p>Methods: A retrospective chart review was done for the dates 8/1/2022 - 7/30/2023 of patients admitted to any Allina hospital. The patients included had a type 2 diabetes mellitus diagnosis and were administered a sulfonylurea (glyburide, glimepiride, glipizide) on the medication administration report (MAR). Patients admitted to rehabilitation units and mental health units were excluded. Patients who had blood glucose between 1-70 mg/dL were manually reviewed for further data collection to review potential patterns of hypoglycemia.</p> <p>Results: In total, 1319 admitted patients received a sulfonylurea at Allina hospitals over a one-year period. Ten percent of these patients (N = 133) experienced a hypoglycemic event. Of those who had hypoglycemia, 103 patients (74%) had documented kidney disease, 130 patients (94%) were primarily managed by internal medicine, 44 patients (33%) were on SU</p>

	<p>monotherapy or on SU with sliding scale insulin, 89 patients (67%) received another oral diabetic medication or long-acting insulin in addition to SU, 121 patients (91%) had the Allina hypoglycemia treatment protocol ordered, 128 patients (96%) had recurrent glucose checks ordered, and 7 patients (5%) were given a SU while NPO status was ordered. In response to hypoglycemic events, the SU was discontinued in only 30% of cases. There was a dose reduction of the SU in an additional 6% of cases.</p> <p>Intervention: These researchers met with multiple Allina interprofessional committees to present results and discuss possible interventions. Options included provider education, education/alerts in the electronic medical record (EMR), removal of SU's from formulary, and order restriction to the Endocrine service. The approved action from these discussions was an alert in the electronic medical record (Epic) when providers order SU's. Results of this study were presented to hospitalist groups for provider education.</p> <p>Future Steps: Once the alert is active in the EMR, we will collect new data for a 6-12 month period. If there is no reduction in sulfonylurea-associated hypoglycemic events, we would propose removal of sulfonylureas from hospital formulary.</p>
<p><b>Zachary Scharf</b>  Dr. Isaac Burrig  Dr. Paulina Marell  Dr. Melissa Bogin</p>	<p><i>Lending a Helping Hand – Increasing Resident Awareness of Patient Social Determinants of Health</i></p> <p>Background: Much literature has been published on social determinants of health (SDOH) and their importance in affecting patient care and outcomes. The proliferation of electronic health records has provided a tool by which physicians can monitor SDOH and address them. However, for meaningful improvements in patient care to result, the tools and services available must be known and utilized. To this end, we undertook an initiative to measure how many residents were using the SDOH features in Epic and then work to improve awareness and utilization of these resources.</p> <p>Methods: A preliminary survey was constructed for Internal Medicine residents to assess how many were using the SDOH features in Epic. A chart review was conducted for one full clinic day to validate the survey results. The percentage of patients with documented SDOH concerns and the percentage of these that had their concerns addressed during the clinic visit were determined. Narrative interviews were done to identify roadblocks to utilization. Using this information, various interventions were created within the resident continuity clinic from July through September 2024. Initial interventions were a script to be read before clinic (8/19/2024-8/30/2024) and an infographic sheet with social work's pager and information for FindHelp.org. A QR code linking to the infographic and an Epic dot-phrase for documentation were created. The number of users that took ownership of the dot-phrase and/or scanned the QR code was tracked. A subsequent Epic chart review was undertaken to determine if SDOH were addressed for patients who had them noted on Epic after the introduction of these interventions.</p> <p>Results: The preliminary survey was answered by 24 residents out of a total of about 150. If extrapolated, this revealed only about 12 residents in the program (8%) were consistently using the SDOH features in Epic. Initial chart</p>

	<p>review results revealed that 13 patients out of 83 appointments (16%) on the day reviewed had documented SDOH concerns. Among patients with SDOH concerns identified, 2 (15%) had their needs addressed during the visit. Narrative comments from residents revealed uncertainty about resources and “notification fatigue” as contributing factors to not addressing SDOH. The script that was created was recalled by 64% of residents present in clinic. The QR code was scanned a total of 9 times and the Epic dot-phrase was added by 10 users. The subsequent chart review of four clinic half-days (n=92 appointments) showed that 19.5% of patients had SDOH concerns flagged on Epic but 0 of these encounters used the SDOH dot-phrase.</p> <p>Conclusion: These results were enlightening and showed the difficulty of implementing a new care priority into busy resident workflows. It shows the limitations of human-focused quality improvement interventions, as only about 60% of participants recalled hearing the script announcement and fewer still acted by scanning the QR code or adding the new dot-phrase. Next steps in the project will focus on moving up the hierarchy of effectiveness into standardization protocols and the adoption of forcing functions, such as adding a SDOH section to the standard resident clinic note template.</p>
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**Research - Residents**

<p><b>Nadhem Abdallah</b>  Dr. Mohammed Samra  Dr. Ammar Aladaileh</p>	<p><i>Impact of Systemic Lupus Erythematosus on Outcomes in Patients Hospitalized for Aortic Stenosis: A Retrospective Analysis</i></p> <p>Background: Individuals diagnosed with Systemic Lupus Erythematosus (SLE) are at an elevated risk for cardiovascular complications and tend to experience unfavorable outcomes during various medical emergencies. Nonetheless, there is limited research focusing on the connection between SLE and Aortic Stenosis.</p> <p>Methods: Hospitalizations for Aortic Stenosis were extracted from the National Inpatient Sample dataset, covering the period from 2016 to 2019. The study compared in-hospital outcomes between patients with and without a diagnosis of SLE. The main outcome measured was overall mortality, with secondary outcomes being the use of vasopressors, the average length of hospital stay (LOS), and total hospitalization costs (THC). Confounding variables were controlled using multivariable regression models.</p> <p>Results: Of the 94,359 Aortic Stenosis-related hospital admissions, 1,625 (0.3%) also had a diagnosis of SLE. Those with SLE had significantly increased odds of death (adjusted odds ratio [aOR] 5.4, 95% confidence interval [CI] 2.1-13.6, p&lt;0.001) compared to patients without SLE. Additionally, patients with polycystic kidney disease (PCKD) were more likely to require vasopressors (aOR 2.7, 95% CI 1.01-7.4, p=0.049), had longer hospital stays (mean LOS 4.45 days vs. 3.95 days, p&lt;0.001), and incurred higher hospitalization costs (\$207,368 vs. \$199,052, p&lt;0.001).</p> <p>Conclusion: Patients with SLE hospitalized for Aortic Stenosis experience more severe adverse outcomes, including higher mortality and increased</p>
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	healthcare resource use, in comparison to those without SLE.
<p><b>Hassan Akram</b>  Dr. Katy Lehenbauer  Kate Knowles  David Van Riper  Dr. Jeremy Van'tHof</p>	<p><i>Examining Urban and Rural Pharmacy Availability</i></p> <p>Background: Cardiovascular disease (CVD) remains the leading cause of death in the United States, underscoring the critical importance of managing risk factors such as diabetes and hypertension. Effective treatment and prevention strategies hinge on medication adherence, which can be significantly influenced by pharmacy access. This access varies across geographic areas, potentially impacting patient outcomes.</p> <p>Objective: The objective of this study is to characterize pharmacy access in Minnesota by comparing data from 2009 and 2020.</p> <p>Methods: Data were obtained from the Minnesota Board of Pharmacy, encompassing a comprehensive list of pharmacies from both 2009 and 2020. The pharmacies were geocoded and categorized based on census tracts designated by Rural-Urban Community Area (RUCA) codes. Three geographic RUCA divisions were established: urban, large rural city/town, and small/isolated rural town. Each pharmacy was classified as a chain, supermarket-based, community, or affiliated with a health system. We assessed pharmacy type, operational hours, total count, and density across each geographic area.</p> <p>Results: Chain pharmacies represented nearly half of all pharmacies in both 2009 and 2020. Notably, community pharmacies experienced a decline across all regions in Minnesota. In areas outside urban centers, the presence of supermarket and health system pharmacies remained relatively stable. Supermarket pharmacies exhibited the best access regarding extended and weekend hours, particularly in non-urban areas. While chain pharmacies offered high access levels in urban regions, their availability decreased significantly in large rural cities and small towns. Independent pharmacies were more likely to be open on weekends but generally lacked extended hours. Access to pharmacies beyond regular business hours was notably higher in urban settings, with over 70% of pharmacies offering extended hours and more than 82% open on weekends. In contrast, access in small rural areas was substantially limited, with fewer than 40% providing extended hours and under 75% open on weekends.</p> <p>Conclusion: Pharmacy access in Minnesota demonstrates significant geographic disparities, particularly affecting residents in less populated areas who have fewer pharmacy options and limited accessibility outside standard business hours. This disparity may hinder medication adherence and, consequently, effective disease management. Addressing these access issues is essential for improving healthcare outcomes and controlling CVD risk factors in rural populations.</p>
<p><b>Ammar Aladaileh</b>  Dr. Nadhem Abdallah  Dr. Mohammed Samra</p>	<p><i>Polycystic Kidney Disease and ST-Elevation Myocardial Infarction: Impact on 30-Day Readmissions and Hospitalization Outcomes</i></p> <p>Background: Individuals with Polycystic Kidney Disease (PCKD) are known to have an elevated risk for cardiovascular diseases and tend to experience adverse outcomes during various medical emergencies. Despite this, data</p>



regarding the interaction between PCKD and ST-Elevation Myocardial Infarction (STEMI) remains limited.

Methods: This study analyzed hospital admissions for STEMI using data from the 2016-2020 Nationwide Readmissions Database. Patients with and without PCKD were compared in terms of in-hospital outcomes. The primary outcome was 30-day readmission for any cause, while secondary outcomes included the average length of hospital stay (LOS) and total hospitalization costs (THC). Multivariable regression models were applied to adjust for potential confounding factors.

Results: Out of 182,308 STEMI admissions, 204 patients (0.1%) had a concurrent diagnosis of PCKD. When compared to patients without PCKD, those with PCKD had higher odds of 30-day readmissions (adjusted odds ratio [aOR] 1.2, 95% confidence interval [CI] 1.04-1.4). Furthermore, PCKD patients had a longer hospital stay (mean LOS 5.9 days vs. 4.6 days,  $p < 0.001$ ) and incurred higher hospitalization costs (\$153,497 vs. \$135,888,  $p < 0.001$ ).

Conclusion: Patients with PCKD who were hospitalized for STEMI exhibited higher rates of 30-day readmissions, extended hospital stays, and greater healthcare costs compared to those without PCKD. These findings underscore the need for further research to confirm these observations and to explore strategies that could improve outcomes in this high-risk population.

**Jackie Blomker**

*Rates and Characteristics of Post-polypectomy Interval Colorectal Cancer: A Single-Center Retrospective Cohort Study*

Introduction: Post-colonoscopy colorectal cancer (PCCRC) can occur due to missed polyps or incomplete polyp resection on initial colonoscopy. High incidence of PCCRC in the proximal colon is thought to be secondary to missed lesions and therefore a second look in the right side of colon is recommended. Polyps in the rectum are thought to be less likely to be missed due to less surface area of the rectum. Thus, we hypothesized that PCCRC in the rectum is more likely to occur due to incomplete polyp resection. We aimed to assess the risk and characteristics of post-polypectomy colorectal cancer.

Methods: We included all patients with colorectal cancer (CRC) who underwent a colonoscopy within 5 years prior to diagnosis of CRC at our institution from 2004-2023. PCCRC was defined as CRC (colorectal cancer) within 5 years of a colonoscopy. Patients who had CRC on initial colonoscopy or those with history of inflammatory bowel disease and familial polyposis syndromes were excluded from the analysis. Post-polypectomy colorectal cancer (PPCRC) was defined as CRC at the exact site of previous polypectomy within 5 years of a colonoscopy. Site of the previous polypectomy was confirmed by reviewing endoscopic images. Demographic, clinical, and adenoma characteristics as well as endoscopy details were analyzed and extracted. Our primary outcome was the rate of PPCRC. The secondary outcome was proportion of PPCRC stratified by location.

Results: Out of 414 cases of CRC diagnosed in the study period, 57 were

PCCRC (43 with colon cancer and 14 with rectal cancer). 36 out of 57 had PPCRC (63.1%), with 80% (29/36) being colon cancer and 19.4% (7/36) rectal cancer. Table 1 shows demographics, colonoscopy, and pathology details of PPCRC. There was no difference in rates of PCCRC based on location (rectum 11.6% vs colon 13.7%, p=0.45). There was also no difference in proportion of PPCRC as stratified by location (rectum 50% (7/14) vs colon 67.4% (29/43), p:0.245). 23 out of 36 cases of PPCRC were at site of a prior advanced adenoma, with 82.6% (19/23) in the colon and 17.4% (4/23) in the rectum.

Discussion: Our study highlights that the majority of interval PCCRCs occur at a site of previous polypectomy. In addition, there was no difference in proportions of PPCRC when stratified by location of cancer. These findings reinforce the need for quality metrics focusing on polypectomy technique and careful inspection of polypectomy sites to assess for any residual polypoid tissue.

**Tracy Bui**  
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 Eugenia Raichlin  
 Takushi Kohmoto  
 Lyle Joyce  
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 Matt Cooper  
 Stephanie Zanolowski  
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*Racial Disparities in Access to Heart Transplant Among Advanced Heart Failure Patients Evaluated for Advanced Therapies*

Purpose: Evaluation of advanced heart failure patients for Mechanical Circulatory Support (MCS) and/or Heart Transplantation (HT) requires a multi-disciplinary approach. We examined whether race is independently associated with the MDT committee’s decision for durable left ventricular assist device (LVAD) and HT above and beyond clinical variables.

Methods: Advanced heart failure patients referred for Destination Therapy (DT), Bridge To Transplant (BTT), and transplant only evaluations from 1/2018 to 6/2021 were included. The primary outcome was treatment designation by committee (none, DT, BTT/transplant). A priori defined clinical and psychosocial variables were systematically compared by referral groups and by race (collapsed into White and non-White). Multiple logistic regression analyses were independently conducted for each referral group to elucidate associations between predictor variables, notably race, and the likelihood of each treatment determination. A backward selection approach, guided by the Akaike Information Criterion (AIC), was employed to select predictors for inclusion. All tests were two-sided, with statistical significance at p<0.05.

Results: Patients referred for DT (n=88) were older (p=0.03) and with more advanced NYHA class (p=0.001) relative to those referred for BTT (n=117) and transplant only (n=36). Forty-four percent of those referred for DT had died by 10/2023 compared to 28% in BTT and transplant referral groups. Table 1 depicts consideration of all a priori clinical and psychosocial variables prior to backward selection for the BTT referral group. Non-White patients were 35 times more likely to be designated for DT treatment and nearly 7 times more likely to be designated for no treatment compared to White patients. Increased NYHA class, increased BMI, presence of diabetes and dyslipidemia, and higher psychosocial risk (as reflected by SIPAT score) were also associated with increased odds for DT decision. Lower albumin, no diabetes, and higher SIPAT were associated with no treatment.

<p><b>Kari Falaas</b>  Dr. Michael Schnaus  Dr. Margaret Singer  Dr. Allison Hochstetler  Pang Nhia Khang  Katherine Schmiechen  Dr. Elie Gertner</p>	<p><i>CASE SERIES: Devastating Morbidity and Mortality Rates in the Hmong Population with a Diagnosis of Gout who had a COVID-19 Infection</i></p> <p>Background: Hyperuricemia is associated with an elevated risk of developing cardiovascular diseases, diabetes, chronic kidney disease, and metabolic syndromes. The Hmong population, in particular, has a higher predisposition to hyperuricemia/gout and associated comorbid conditions. More recently, studies have also shown that all individuals with gout have higher risk of morbidity and mortality with COVID-19 infection. We examined outcomes of COVID-19 infection in the Hmong population with gout to determine whether these mortality and morbidity risks were compounded and of even greater import in this population.</p> <p>Methods: Through retrospective chart review, 21 Hmong patients with COVID 19 and a clinical diagnosis of gout were identified. These patients were all <math>\geq 18</math> years old, had documented COVID-19 infection, required oxygen, and presented to two tertiary care centers between March 1, 2020-December 31, 2021. Descriptive statistics were used for analysis.</p> <p>Results: The average age of the 21 patients with severe COVID-19 identifying as Hmong and with a comorbidity of gout was 62.9 years . Fourteen (63.6%) of these patients identified as male, with an average BMI of 28.9. The average maximum uric acid level prior to admission was 8.02. Six had a history of tophaceous gout, 3 had a history of uric acid nephrolithiasis, and 14 were being treated with allopurinol prior to admission. Other underlying comorbidities for this cohort included chronic lung disease, chronic heart failure, renal disease, and diabetes.</p> <p>The average length of stay was 12.1 days. Seven patients required low flow oxygen via nasal cannula, 3 patients required high flow oxygen, 1 patient required noninvasive ventilation, and 10 of the patients (48%) required mechanical ventilation. The average number of ventilator days was 15.5 days. Seventeen of the patients received steroids, 6 received remdesivir, and 1 received tocilizumab. Eleven of the twenty-one patients ultimately transitioned to comfort care. The 30 day mortality for Hmong patients with a prior comorbidity of gout from COVID-19 was 57% (n=12).</p> <p>Conclusion- Our case series describes markedly severe COVID-19 outcomes in the Hmong population with gout with a 30-day mortality rate of 57%. The combination of genetic predisposition to hyperuricemia, the comorbid conditions associated with hyperuricemia, and the presence of COVID-19 infection was devastating in the Hmong population. These findings again highlight the comprehensive efforts needed in healthcare systems in approaching hyperuricemia and gout in all populations. Furthermore, in order to prevent future morbidity and mortality, there is a critical need for outreach and education to diverse ethnic and racial groups with gout due to the serious nature of the underlying disease process.</p>
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<p><b>Ciana Keller</b>  Dr. Lorraine Mascarenhas  Dr. Sue Duval  Dr. David Benditt</p>	<p><i>Quantitative Testing Reveals Severity of Autonomic Dysfunction after Acute COVID-19 Infection: A Comparison with Controls and Autonomic Failure</i></p> <p>Background: COVID-19 infections have been associated with cardiovascular autonomic dysfunction (AD). Clinical findings include fatigue, cognitive impairment, and postural intolerance. However, quantitative post-COVID AD assessments are lacking.</p> <p>Objective: Compare autonomic testing measures of post-COVID-19 subjects to controls and those with pure autonomic failure (PAF).</p> <p>Methods: Autonomic testing included 1) change in heart rate (HR) and blood pressure (BP) with active standing (AS) and tilt table testing (TT), 2) time to BP nadir and recovery during AS and TT, 3) Valsalva ratio (VR), and 4) respiratory sinus arrhythmia (RSA). Comparisons between two groups were made using t-tests, Kruskal-Wallis, or chi-square tests. Multivariable linear regression was used to adjust findings for age and sex. A p-value of &lt;0.05 was considered significant.</p> <p>Results: Control subjects (n=25, median 32 [21-39] years, 76% female) and post-COVID patients (n=91, median 36 [25-50] years, 84% female, mean 11.96 [±9.14] months from infection) were similar in age and sex, while the PAF group (n=38, median 64 [56-74] years, 53% female) was older and comprised of more males (Table 1). The VR and RSA of the post-COVID group was significantly lower than controls, but higher than the PAF group (Table 2). The change in BP with AS and TT for post-COVID subjects was significantly higher than controls and lower than the PAF group. The post-COVID group also had a significantly increased change in HR with TT compared to both the control and PAF groups. Across all four tests, the post-COVID group had a higher proportion of abnormal results compared to controls. After adjusting for age and sex, post-COVID subjects continued to have a greater change in HR and BP with AS and TT and a lower RSA compared to controls. However, with this adjustment several differences compared to the PAF group were attenuated (Table 3).</p> <p>Conclusion: Post-COVID subjects were observed to have multiple quantitative autonomic testing abnormalities. Once corrected for sex and age, AD severity in post-COVID-19 patients is comparable to that seen in neurodegenerative PAF.</p>
<p><b>Madison Okuno</b>  Dr. Thomas Schmidt  Dr. Molly Lindstrom  Dr. Rima Gurung  Dr. Javad Parvizi  Dr. Don Bambino  Dr. Geno Tai</p>	<p><i>Assessing the Responses of Artificial Intelligence Versus Human Experts in Answering Clinical Practice Guideline Questions Related to Periprosthetic Joint Infections</i></p> <p>Background. Traditional guidelines are essential for clinicians, offering consolidated insights from experts on current evidence in specific topics. However, assembling an expert panel and formulating these guidelines is an extensive and time-consuming task. Our goal is to explore the potential of generative artificial intelligence (AI) in developing 'living' clinical practice guidelines.</p> <p>Materials. We selected 13 guideline questions on antimicrobials from the Proceedings of the Second International Consensus Meeting on</p>

	<p>Musculoskeletal Infection (ICM). We used the articles cited by the human authors and asked ChatGPT 4 to answer identical questions using the references. We used prompts that mirror the instructions given to expert panels, including answering the questions from the perspective of an expert panel. We performed a deductive thematic analysis to compare its responses to human authors. We analyzed the responses using the themes prescriptiveness and general guidance in relation to the strength of the evidence presented.</p> <p>Results. Our analysis of responses to guideline questions revealed notable differences in their responses. While both AI and human authors acknowledged that there was limited evidence to answer the questions definitively, AI was less prescriptive. It showed hesitancy to recommend specific treatments without strong supporting evidence. AI tends to interpret questions literally and respond more directly, resulting in answers that are less nuanced compared to those provided by human authors. This difference becomes more noticeable in cases where the questions are ambiguous.</p> <p>A key limitation identified in this study is the AI's dependency on the structure and clarity of the prompts provided. The AI's performance in providing relevant and accurate information was significantly influenced by how questions were posed, underscoring the importance of precise and unambiguous prompting when seeking information from AI sources.</p> <p>Conclusions. AI's objective approach to evidence synthesis and drafting documents could improve our ability to craft living clinical practice guidelines. Further research is necessary to identify additional areas where AI can automate the process of updating guidelines.</p>
<p><b>Daniel Pollmann</b>  Jayne Parry  Gretchen Benson  Ellen Cravero  Susan White  Dr. Larissa Stanberry  Carolyn Wambach  Dr. Michael Miedema</p> <p>Finalist</p>	<p><i>In with the New: A GLP-1 Cardiology/Pharmacy Integrated Program</i></p> <p>Background: Glucagon-like peptide-1 receptor agonist (GLP-1) medications have been utilized to improve cardiovascular risk in overweight patients with or without diabetes. Practical barriers to use these medications often exist, some of which include medication shortages, prior authorizations, and frequent dose titrations to mitigate side effects and achieve maximum effect. An interdisciplinary GLP-1 program with pharmacist support was devised to remedy these challenges.</p> <p>Methods: This retrospective cohort study assessed the prescribing success and health outcomes among 385 patients referred to the cardiology/pharmacy GLP-1 program from June 2023 to March 2024. Patients were referred to the program by cardiologists and deemed eligible if they had a BMI &gt;30 and established cardiovascular disease (CVD), diabetes, or multiple CVD risk factors. A pharmacist would then review the patient's chart to confirm eligibility, prescribe the appropriate GLP-1 medication, and address any prior authorization or appeal letters needed. A pharmacist would call the patient every 2 weeks to address dosing changes and possible side effects. Outcomes evaluated include rates of initiation and continuation, and changes in CVD risk factors.</p> <p>Results: Of the 385 patients referred to the Allina/MHI GLP-1 program, 259 (67%) patients were initiated on GLP-1 therapy. Of the 235 initiated patients</p>

with >1 month follow-up, 198 (84%) patients continued GLP-1 therapy. Among initiated patients, weight changed by a median of -8 kg (median follow-up 155 days), total cholesterol by -16 mg/dL (119 days), blood glucose by -13 mg/dL (119 days), and A1C by -0.45% (114 days). Systolic blood pressure and diastolic blood pressure changed by a median of -7 and -2 mmHg (145 days).

Conclusions: Implementation of an integrated GLP-1 program showed high rates of initiation and continuation in patients appropriate for GLP-1 use with subsequent improvement in important cardiovascular risk factors. Efforts to utilize all available resources to optimize use of this important class of medications are warranted. The approach presented here would benefit appropriately selected patients in a primary care population.

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*Cardiovascular Outcomes in Heatstroke: A Systematic Review and Meta-Analysis*

Background: Rising global temperatures and an increase in heat-related deaths underscore the growing significance of heat stroke. While effects on other systems have been studied, there is a notable lack of research on the cardiovascular manifestations and complications during heat stroke.

Methods: For this systematic review and meta-analysis, MEDLINE, Embase, and Scopus were searched for articles published from inception till August 2023. Studies were included if they reported clinical outcomes in heat stroke patients. Studies only reporting heat exhaustion and other heat-related illnesses were excluded. The extracted data underwent arcsine transformation, was pooled using a random effects model, and analyzed using OpenMetaAnalyst. Our outcomes of interest were hypotension (including circulatory failure), tachycardia, electrocardiogram (ECG) abnormalities, elevated cardiac enzymes, and in-hospital mortality. Secondary outcomes included specific ECG abnormalities.

Results: Our systemic review pooled 38 studies and a total of 5,777 heat stroke patients. Pooled meta-analysis demonstrates that tachycardia (72.9%; 95% CI, 62.4% - 83.3%) was the most prevalent cardiovascular outcome in heat stroke patients, followed by ECG abnormalities (63.6%; 95% CI, 35.2% - 92.0%), elevated cardiac enzymes (41.1%; 95% CI, 22.9% - 59.3%) and hypotension (33.6%; 95 CI, 27.0% - 40.1%). For the secondary outcomes, data on specific ECG abnormalities were analyzed. Sinus tachycardia (70.1%; 95% Ci, 49.7% - 90.6%) was the most prevalent ECG abnormality, followed by ischemic changes (25.0%; 95% CI 19.3% - 30.7%), arrhythmias (22.4%; 95% CI, 10.5% - 34.4%), atrial fibrillation (10.6%; 95% CI, 6.4% - 14.8%) and STEMI (4.0%; 95% CI 19.3% - 30.7%). The prevalence of in-hospital mortality among patients with heat stroke was 13.3% (95% CI, 10.3%-16.4%).

Conclusion: Our systematic review and meta-analysis conclude that there is a high incidence of adverse cardiovascular outcomes in patients with heat stroke. Therefore, these patients need to be monitored rigorously and have timely interventions done if needed.

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 Anders Westanmo  
 Amy Gravely  
 Dr. Mohammad  
 Bilal  
 Dr. Aasma Shaukat

*Root Cause Analysis of Post-Colonoscopy Colorectal Cancer in FIT-Positive Individuals: Results from a Nationwide Database*

Introduction: Post-colonoscopy colorectal cancer (PCCRC) is defined as CRC diagnosed six months or later after a colonoscopy in which no cancer is identified. The World Endoscopy Organization (WEO) has proposed guidelines for categorization of PCCRCs. The aim of this study is to apply the WEO categorization and perform a root cause analysis (RCA) of PCCRCs in fecal immunochemical (FIT)-positive individuals using a nationwide cohort.

Methods: Data was obtained from the Veterans Health Administration Corporate Data Warehouse. Individuals who underwent their first colonoscopy after a positive FIT between January 2015 and August 2023 were included. PCCRCs were identified using the VA Central Cancer Registry and Systematized Nomenclature of Medicine codes. PCCRC was defined per WEO guidelines as CRC diagnosed 6 to 48 months after colonoscopy where no CRC was identified.

All available records were reviewed for each potential PCCRC. PCCRCs were then categorized per the WEO system as interval (diagnosed before the recommended surveillance interval) or non-interval type A (diagnosed at the recommended surveillance interval), type B (diagnosed after recommended surveillance interval), or type C (diagnosed when no surveillance interval was recommended). The appropriateness of recommended surveillance intervals based on guideline recommendations was also evaluated. An RCA was then performed to determine the most plausible etiology of PCCRC.

Results: A total of 52,167 colonoscopies were performed after a positive FIT [Table 1]. 100 cases of PCCRC were identified. The median time to PCCRC diagnosis was 23 months. 75 PCCRCs were diagnosed within 3 years, resulting in a PCCRC 3-year rate of 5.9% (95% CI: 4.6%–7.2%). Subcategories of PCCRCs were as follows: interval (32%), non-interval type A (31%), type B (35%), type C (2%). Inappropriate surveillance interval was recommended in 12% of cases and accounted for 11 interval PCCRCs. The most common plausible etiology for PCCRC was possible missed lesion with adequate exam (38%) followed by incompletely resected lesion (25%), detected lesion that was not resected (24%), and missed lesion with inadequate exam (13%). PCCRC-related mortality rate was 14% [Table 2].

Conclusion: Missed lesions and incomplete lesion resection were the most common plausible causes of PCCRC. This reinforces the importance of second look in the proximal colon and development of quality metrics focusing on polypectomy technique. In addition, medicine providers should be aware of and adhere to colonoscopy surveillance guidelines.

**Clinical Vignette - Residents**

**Jesse Abelson**  
 Dr. Anya Jamrozy

*Out of Breath and Out of Nowhere: A Sudden Case of Explosive Pleuritis*

Introduction: Community-acquired pneumonia is one of the leading causes of death from infectious diseases, causing approximately 2.5 million annual deaths globally. Parapneumonic effusions are among the most common complications of pneumonia. In extremely rare cases, parapneumonic

	<p>effusions can rapidly progress, causing a condition termed “explosive pleuritis “. Explosive pleuritis is a rare medical emergency defined by the rapid development of a pleural effusion involving over 90% of the hemithorax over a 24-hour period. This causes compression of the lungs and mediastinal shift, which can rapidly lead to hemodynamic collapse and death if not promptly treated.</p> <p>Case Presentation: A 37-year-old male with no pertinent cardiopulmonary history presented to the ED with shortness of breath and left shoulder pain which was exacerbated with deep breathing. Initial vitals were notable for tachycardia to 115 and SpO2 at 93%. A familial history of blood clots was noted, and a CT PE showed patchy lingular infiltrate without pleural effusion or pulmonary embolism. He was able to ambulate on room air and was discharged with oral antibiotics. The following day, he presented again to the ED with worsening shortness of breath. Chest X-ray was notable for left basilar opacity, concerning for pneumonia versus edema. He was tachypneic and hypoxic to 86-91%, requiring supplemental O2. Bedside ultrasound of left lung without pleural effusion. He was admitted and started on IV antibiotics.</p> <p>Hospital Day (HD) 1: At 0900, the patient developed increased oxygen requirements, eventually requiring BiPAP. At 1000, a bedside ultrasound showed a new loculated pleural effusion in the left lung. A STAT CT Chest was ordered. Prior to completion, a behavioral emergency was called for confusion and agitation. At 1426, a CT Chest showed a newly massive pleural effusion causing compression of the left lung with tracheal deviation. At 1510, he underwent emergent chest tube placement with 1.3L output within the first 20 minutes.</p> <p>HD 2-13: Pleural cultures grew Strep Intermedius and Capnocytophaga spp. Thoracic surgery attempted enzymatic debridement of the empyema with tPA administered via chest tube. Despite this, the patient had continued fevers, tachycardia, and leukocytosis. Repeat imaging showed loculated empyema. The patient underwent a VATS procedure on HD 5 and was intubated post-op. He was extubated on HD 6 and his chest tubes were removed on HD 8 and HD 10. He was discharged on HD 13 with planned antibiotic therapy for 4 weeks post-VATS procedure.</p> <p>Discussion: Explosive pleuritis is a rare medical emergency that occurs most often with group A streptococcus species, though can occur with any infection as seen in this case. It is theorized that explosive pleuritis occurs due to blockage of the peribronchial and subpleural lymphatics with cellular and necrotic debris. Explosive pleuritis should be considered in patients with pneumonia and acutely worsening respiratory status. Bedside ultrasound can expedite the identification of explosive pleuritis as rapid development of pleural effusion should be easily visualized with this modality. Treatment with an emergent chest tube is lifesaving and should be done rapidly to prevent hemodynamic collapse and hypoxemia.</p>
<p><b>Khalid Abu-Zeinah</b> Dr. Neel Shah</p>	<p><i>Polymicrobial Pyogenic Liver Abscess in a Patient with Familial Adenomatous Polyposis and Hepaticoduodenostomy</i></p>



Finalist	<p>Introduction: Pyogenic liver abscesses are associated with significant morbidity and mortality, most commonly due to complications such as sepsis and/or septic shock. Liver abscesses usually develop via four main routes: hematogenous seeding (bacteremia), direct spread from ascending biliary infection, extension from intra-abdominal infection, or abdominal trauma.</p> <p>Case Description: We describe a case of a polymicrobial hepatic abscess in a 47-year-old female with a history of Roux-en-Y reconstruction, common bile duct resection, and hepaticoduodenostomy for a suspected type 1 choledochal cyst. These procedures were performed 5 years prior to her presentation. The patient also had a clinical diagnosis of familial adenomatous polyposis (FAP) and had undergone total colectomy and total abdominal hysterectomy in 2006. She reported first-degree relatives apparently affected with FAP, but no APC gene testing had been performed on her. She initially presented to the emergency department with left upper quadrant abdominal pain and nausea. Initial investigations revealed elevated leukocytes (<math>18.2 \times 10^9 /L</math>), but no acute CT abnormalities, and she was discharged. Persistent leukocytosis (<math>22.8 \times 10^9 /L</math>) was noted on follow-up with her primary care provider. Four weeks later, she returned to the emergency department with worsening of her initial symptoms, increasing leukocytosis (<math>36.7 \times 10^9 /L</math>), and a CT scan revealing a 10 cm gas-containing abscess in the left hepatic lobe, with pneumobilia. Treatment included empiric antibiotics and percutaneous drainage of the abscess. Cultures from the abscess grew <i>Streptococcus anginosus</i> and <i>Escherichia coli</i>, but peripheral blood cultures were negative. A repeat CT 3 weeks after treatment showed resolution of the abscess.</p> <p>Discussion: <i>Streptococcus anginosus</i> and <i>Escherichia coli</i> are part of the normal gut flora but have the potential to cause liver abscesses. The patient had no history of trauma or recent gastrointestinal procedures, nor evidence of bacteremia or active infection in the biliary tree. It is likely that her altered gastrointestinal (GI) anatomy, due to her hepaticoduodenostomy 5 years prior, facilitated direct translocation of gut flora to the liver, resulting in an abscess. Liver abscesses are rare in immunocompetent patients without active bacteremia or biliary tract infection, but patients with hepaticoduodenostomy may be at an elevated risk. Patients with prior hepatobiliary surgery are also more likely to develop polymicrobial infections and have a higher prevalence of <i>Escherichia coli</i>, as observed in our case (Lin et al. 2024).</p> <p>Additionally, patients with pyogenic liver abscesses were shown in one study to have a 4-fold increased risk of developing GI cancers, including small intestine, pancreatic, and colorectal cancers (Lai et al. 2014). A pyogenic liver abscess may, therefore, be an indicator of the onset of GI cancer (Kao et al. 2012). For a patient with FAP, who already faces an increased risk of malignancies of the GI tract, pancreas, and hepatoblastoma (Karstensen et al. 2023, Trobaugh-Lotrario et al. 2018), surveillance for these types of cancers may be especially pertinent following the presentation of a pyogenic liver abscess.</p>
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<p><b>Hassan Akram</b> Dr. Aisha Shabbir Dr. Mateen Ahmad Khadija Alam Muhammad Uzair Khan Fatima Kaleem Ahmed Laibah Arshad Khan</p>	<p><i>Non-Vitamin K Antagonist Oral Anticoagulants for Patients with Hypertrophic Cardiomyopathy and Atrial Fibrillation: A Systematic Review and Meta-Analysis</i></p> <p>Background: Hypertrophic cardiomyopathy (HCM) is a hereditary cardiovascular disorder, often complicated by atrial fibrillation (AF). The efficacy and safety of Non-vitamin K antagonist oral anticoagulants (NOACs) versus vitamin K antagonists (VKAs) in HCM patients with AF remain untested.</p> <p>Methods: PubMed and Scopus databases were searched up to September 2024 to identify studies comparing the effects of NOACs and VKAs in patients with HCM and AF. The outcomes of interest included efficacy measures such as systemic stroke/embolism, ischemic stroke, and all-cause death, as well as safety outcomes like major or clinically relevant bleeding, intracranial hemorrhage, and gastrointestinal bleeding. Risk ratios (RR) with 95% confidence intervals were pooled using a random-effects model.</p> <p>Results: Five studies involving 6,848 patients were analyzed. NOACs significantly reduced the risk of ischemic stroke (RR: 0.50, 95% CI: 0.35-0.71) and all-cause mortality (RR: 0.44, 95% CI: 0.35-0.55), with comparable risks for systemic stroke/embolism. NOACs also lowered the risk of intracranial hemorrhage (RR: 0.42, 95% CI: 0.24-0.74, P=0.002), while other safety outcomes showed no significant differences.</p> <p>Conclusion: NOACs offer a favorable safety and efficacy profile compared to VKAs in both patients with HCM and AF. Future research should focus on long-term outcomes and specific patient subgroups to further refine anticoagulant therapy in these patients.</p>
<p><b>Yasmin Ali</b></p>	<p><i>A Rare Case of Superior Mesenteric Venous and Portal Vein Thrombosis as a Complication of Appendicitis</i></p> <p>Introduction: Superior mesenteric thrombosis and portal vein thrombosis are rare but can lead to life-threatening complications, including ischemic hepatitis, liver failure and bowel ischemia. The aim of this report is to highlight the diagnostic and management challenges encountered in this case.</p> <p>Case description: 25-year-old male previously healthy presented with a 5-day history of epigastric pain radiating to the right upper quadrant, mainly triggered by eating. Associated with nausea, vomiting, fever and chills. Pain medications and antacid provided no relief, prompting his visit to the emergency department. Upon arrival, he was febrile at 39.3°C and tachycardic to 105. Examination was notable for guarding and tenderness in the epigastric and right upper quadrant regions.</p> <p>Complete blood count showed thrombocytopenia of 85 k/cmm, otherwise within normal limits. Liver function tests (LFT) revealed elevated alkaline phosphatase of 133 IU/L, alanine aminotransferase of 60 IU/L, and total and direct bilirubin to 4.3 mg/dL and 3.8 mg/dL, respectively. Gallbladder US showed no evidence of cholecystitis or duct dilatation. CT scan abdomen showed a dilated appendix suspicious for appendicitis without rupture, in</p>

	<p>addition to hypo-enhancement of the superior mesenteric vein and left portal vein, indicating non-occlusive thrombosis of the superior mesenteric vein and occlusive thrombosis of the left portal vein. Consequently, a heparin drip was initiated. Doppler US demonstrated patency of the portal venous system but was suspicious of intrahepatic biliary dilatation. Gastroenterology was consulted, recommended discontinuing the heparin drip and obtaining an MRCP, which ruled out biliary dilatation but revealed hyperintensity in the anterior left lobe of the liver, suggestive of acute hepatitis. MRI abdomen with venous phase confirmed the initial concerns for superior mesenteric vein thrombosis and intrahepatic peripheral portal vein thrombosis. Surgical consultation thought the appendiceal dilatation was likely due to reactive ischemia or vascular congestion from the clots rather than inflammation, with no initial surgical intervention. He was restarted on a heparin drip. An extensive workup was negative for viral, metabolic, and autoimmune etiologies for hepatitis. However, hypercoagulable workup revealed a positive lupus anticoagulant (LA), the rest were negative.</p> <p>On day 5 of hospitalization, he developed acute localized RUQ pain, with interval changes in his abdominal examination. Additionally, his white blood cell count trended to 16k/cmm. Given concerns for progression of appendicitis, a repeat CT scan demonstrated uncomplicated acute appendicitis. The decision was made to proceed with surgery, which ultimately revealed an appendiceal rupture. Appendectomy and liver biopsy were performed during the same procedure.</p> <p>Postoperatively, his symptoms resolved, and he was discharged on oral anticoagulants. Biopsy results were negative for malignancy or infiltrative disease. He followed up with He had outpatient follow ups. LFTs normalized, and LA 3-months later was negative. Follow-up CT scan at 6-months showed resolution of the thromboses. He completed a 6-month course of anticoagulation. The etiology of the thrombosis was thought to be a complication of severe appendiceal inflammation.</p> <p>Conclusion: In cases of acute appendicitis complicated by liver dysfunction and blood vessel thrombosis, prompt and effective management of the source of inflammation is essential to avoid life-threatening complications.</p>
<p><b>Alexandra Allman</b> Dr. Joe Orndorff</p>	<p><i>Don't Forget the Bug Spray!</i></p> <p>Case description: A previously healthy 62-year-old gentleman presented with fevers, fatigue, and dizziness which progressed over a week to include vomiting, headache, and confusion. After he fell while walking, he was brought to an outside emergency department.</p> <p>Laboratory workup revealed mild leukocytosis peaking at 13,400 u/L on hospital day 3, hemoglobin 15.0 gm/dL, platelets 90 x10<sup>9</sup> /L, serum creatinine 1.39 mg/dL, creatinine kinase (CK) 1,990 U/L. A CTA chest demonstrated an 8.5cm anterior mediastinal mass however an MRI further characterized the mass as likely a thymoma. Neurology was consulted, and a lumbar puncture revealed elevated protein levels (132 mg/dL), and an increased total nucleated cell count of 255 cu mm with 173 polymorphonuclear cells and a negative gram stain. Myasthenia gravis</p>

	<p>markers and meningitis/encephalitis panel PCR from the CSF were negative. Neurology concluded this was unlikely to be myasthenia gravis, Guillain Barre Syndrome, or meningitis.</p> <p>The patient had extensive exposure to ticks and mosquitos at his home in North Dakota and cabin in northern Minnesota; however tick panel (Babesia, Anaplasma, and Ehrlichia PCR) from the blood was negative. He was started on empiric antibiotics; however he developed progressive ascending flaccid paralysis requiring intubation, and he was transferred to Mayo Clinic.</p> <p>Infectious disease was consulted. Given the high suspicion of tick or mosquito born illness, antibodies were sent for Borrelia, Anaplasma, Ehrlichia, Babesia, West Nile virus, LaCrosse encephalitis, East and West Equine Encephalitis, and St. Louis Encephalitis. Powassan virus PCR from the blood was also sent, despite previous negative tick panel PCRs. West Nile virus IgM and IgG were positive, confirming a diagnosis of West Nile meningoencephalomyelitis. The patient improved with supportive care and was discharged to inpatient physical rehabilitation.</p> <p>Discussion: West Nile virus, transmitted by infected mosquitos, can present with a range of symptoms, from asymptomatic, to flu-like symptoms with fevers, headache, and myalgias, and in severe cases, encephalitis, meningitis or acute flaccid paralysis. Despite the potential severity of these presentations, death is rare. Incidence is highest in the Midwest from mid-July to early September, and treatment is primarily supportive. Diagnosis of West Nile encephalitis relies on detecting IgM and IgG antibodies in the serum and CSF. IgM positivity in the CSF is diagnostic of neuroinvasive disease as IgM does not cross the blood-brain barrier. IgM positivity indicates recent infection and can be positive after viral clearance with negative PCR testing.</p> <p>“Tick/Mosquito Panels” are common; however it is essential to understand what these panels include and the appropriate use of serology versus PCR testing. These panels typically consist of PCRs for common bacteria, detecting actively circulating DNA of the pathogen. PCR testing is most useful during the acute phase of illnesses, typically within the first few days to a week of symptoms before the viral DNA is cleared. In contrast, serology is most useful after the initial acute phase and detects antibodies (IgM and IgG) to pathogens that take time to develop. Clinicians should select appropriate tests based on duration of symptoms and the expected likelihood of positive results.</p>
<p><b>Suchapa Arayakarnkul</b></p>	<p><i>Outcomes of Interval Cholecystectomy Following Endoscopic Ultrasound-Guided Gallbladder Drainage: A Systematic Review and Meta-Analysis</i></p> <p>Introduction: Cholecystectomy is the gold standard for management of acute cholecystitis (AC). However, AC patients who are medically unfit for surgery at index presentation can undergo endoscopic ultrasound-guided gallbladder drainage (EUS-GBD) for treatment of AC. EUS-GBD is typically limited to patients who will not be candidates for interval cholecystectomy as the formation of cholecystoenteric fistula can make future surgery challenging. Recent studies have suggested feasibility of EUS-GB as a bridge</p>

	<p>to subsequent surgery. We aim to conduct a systematic review and meta-analysis to determine the outcomes of interval cholecystectomy in patients with previous EUS-GBD.</p> <p>Case Presentation: We conducted a literature search of multiple electronic databases (inception-June 2024) for studies evaluating outcomes of interval cholecystectomy following EUS-GBD. Primary outcomes were pooled proportion of patients undergoing cholecystectomy, conversion rate of laparoscopic-to-open cholecystectomy, rates of open cholecystectomy and rate of adverse events (AEs). Secondary outcomes were procedural time, length of stay (LOS), and mortality. A meta-analysis of proportion was performed using the random-effects model. I<sup>2</sup> statistic was used to assess heterogeneity.</p> <p>Out of 1001 citations, 16 studies including 716 patients (51.8% male, mean age 71.7±14.8, mean Charlson Comorbidity Index 5.93±3.5) who had previously undergone EUS-GBD [stent site: duodenum 47.5%; stomach 27.7%; jejunum 0.7%] were analyzed. Pooled proportion of successful interval cholecystectomy was 0.37 (CI: 0.16-0.57; I<sup>2</sup> 99%) [laparoscopic: 0.74 (CI: 0.6-0.88; I<sup>2</sup> 82%); open: 0.13 (CI: 0.04-0.22; I<sup>2</sup> 80%); laparoscopic converted to open: 0.16 (CI: 0.06-0.26; I<sup>2</sup> 77%)]. None of the studies reported aborted surgery. Interval cholecystectomy was performed after 52.8±65.5 days from the initial EUS-GBD. Pooled proportion of overall AEs was 0.16 (CI: 0.06-0.25; I<sup>2</sup> 65%), including postoperative infection: 0.1 (CI: 0.02-0.18; I<sup>2</sup> 70%). Mean procedural time was 120±67.3 minutes, with mean LOS 5.8±3.6 days. There was no procedure-related mortality.</p> <p>Conclusion: Our study demonstrates that interval cholecystectomy is technically safe and feasible after EUS-GBD. Nearly 75% of patients underwent successful laparoscopic cholecystectomy, with conversion to open reported in only 16%. However, these results could have a selection bias due to candidate selection for interval cholecystectomy after EUS-GBD.</p>
<p><b>Amro Awad</b></p>	<p><i>CNS Lymphoma Mimicking a Tick-Borne Illness: A Case Report</i></p> <p>Introduction: CNS lymphomas have very variable first presentations. This clinical vignette is a case of a forty-seven-year-old patient with B Cell CNS lymphoma who presented with progressive weakness and paresthesia that was thought to be neuroborreliosis.</p> <p>Case Description: A 47-year-old male was admitted after presenting with headache, dizziness, and numbness in his right face, arm, and leg after being bitten by a tick on his waist eight weeks prior. Physical exam showed a rash in the pattern of erythema migrans on the right waist. Neurological exam showed numbness in the right lower extremity and around the bite site, otherwise, range of motion and muscular strength from head to toe were intact. The patient was admitted for further work-up and started on Ceftriaxone. WBCs were within normal limits. Lumbar puncture revealed protein of 151, glucose of 58, lymphocytes of 69 and culture with no growth. Lumbar MRI results were unremarkable. HSV PCR, total Lyme Ig, West-Nile Ig came back negative.</p> <p>Afterwards, an MRI of the brain was done that showed multiple enhancing</p>

	<p>nodules/mass lesions involving both cerebral hemispheres, largest in the left cerebral hemisphere with significant surrounding vasogenic edema. After consulting with the infectious disease and neurology teams, Ceftriaxone was stopped, and the patient was started on Prednisone 60mg daily for edema. The patient’s symptoms improved on steroids, and he was discharged with follow-ups to infectious disease and neurology clinics. At the time of discharge T. solium antibodies; Anti-Ganglioside panel (includes Anti- GM1 / GM2 / GD1A / GD1B, GQ1B), quantifereon-gold, cytology, and toxoplasma gondii CSF results were still pending.</p> <p>Ten days later, the patient presented with worsening symptoms. The numbness over the right side of the body worsened along with new-onset right sided extremity weakness, headache and dizziness. Repeat brain MRI showed a slight increase in the size of the multiple nodules/masses, with worsening of midline shift, now up to 6 mm. Further consultation with neurology, neurosurgery, and infectious diseases teams prompted tissue biopsy given the rest of his infectious work-up came back negative. Biopsy showed Primary CNS B-Cell Lymphoma, and the patient was started on chemotherapy of Methotrexate, Temozolomide and Rituximab.</p> <p>Discussion: Primary CNS lymphoma (PCNSL) is a rare form of extranodal non-Hodgkin lymphoma that is typically confined to the brain, eyes, and cerebrospinal fluid without evidence of systemic spread [1]. Patients usually develop progressive neurological signs over weeks or months. Given the wide range of possible presenting symptoms, and non-specific imaging findings, alternative diagnoses might come to mind at first. Especially in the case presented where the patient came with a history of tick bite, which shifted the differential into something more likely to be infectious as neuroborreliosis. Stereotactic brain lesion biopsy remains the gold standard for the diagnosis of PCNSL [2], but because it is an invasive procedure that carries risks of complications and may not always provide the required results [3-4], confirming the diagnosis remains challenging given the high similarity in its presentation compared to other disease processes such as tick-borne illnesses with CNS involvement.</p>
<p><b>Jacob Beery</b> Dr. Rachel Hansen Dr. Patrick Arndt</p>	<p><i>The New Hot Joints in Town: A Case Report of Polyarticular Septic Arthritis</i></p> <p>Background: Septic arthritis (SA) is a rare but serious condition, often caused by bacterial infection, which can rapidly lead to joint destruction or death if untreated. The diagnosis can be complex, especially in atypical cases with multi-joint involvement which rarely can present as septic shock. In this clinical vignette, we present a case of polyarticular septic arthritis followed by an acute gout flare. A 67-year-old male with a PMHx of interstitial lung disease, pulmonary hypertension, and gout, initially presented with fever, hypotension, oliguria, and joint pain in both knees and ankles. Initial labs and imaging were suggestive of infection, ultimately requiring broad spectrum antibiotics and vasopressors. Synovial fluid analysis showed no crystals, and an elevated WBC with a neutrophilic predominance, though no bacteria were identified. The patient ultimately was treated with antibiotics and later steroids and anakinra for a subsequent gout flare identified on imaging.</p> <p>Case Presentation: This case highlights three important learning points: the</p>

	<p>unusual presentation of septic arthritis mimicking septic shock, the application of the Modified Newman’s Criteria for diagnosis, and the importance of avoiding premature diagnostic closure. In this case, the patient was treated for septic shock of initially concerning for pulmonary or urologic origin. Despite the absence of pathogenic organisms in synovial or blood cultures, the clinical features and joint fluid findings met the Modified Newman’s Criteria, affirming a diagnosis of SA. Additionally, while the patient showed no improvement with antibiotics alone, a shift to therapeutic interventions for a secondary gout flare, led to significant clinical improvement, emphasizing the need for continuous reassessment in complex cases of systemic infection and inflammatory arthritis. In total, this case underscores the diagnostic and therapeutic challenges of polyarticular septic arthritis in patients with multiple comorbidities.</p>
<p><b>Christopher Behrend</b></p>	<p><i>Diagnosis of Type II Refractory Celiac Disease</i></p> <p>Introduction: This clinical vignette describes a patient who was diagnosed at a later age with what was presumed to be mixed picture lymphocytic colitis and celiac disease (CD), however, underwent further workup with diagnosis of refractory CD, type II. Such cases carry poor prognosis with increased likelihood of progression to lymphoma.</p> <p>Case Description: A 61-year-old man with previously diagnosed mixed picture of lymphocytic colitis and CD was admitted with ongoing non-bloody diarrhea, abdominal pain, and hypokalemia. Colonoscopy 4 years prior for frequent diarrhea and bright red blood per rectum revealed normal appearing colon and terminal ileum, biopsies consistent with microscopic colitis. EGD revealed benign duodenum with total loss of villi and markedly increased intraepithelial lymphocytes (IELs), consistent with CD. He was started on a gluten-free diet (GFD) and short steroid course. Symptoms improved for years until returning with frequent diarrhea. Differential included infectious gastroenteritis, microscopic colitis flare, CD flare, and inflammatory bowel disease (IBD). Stool panel was negative, and tissue transglutaminase was within normal limits. EGD and colonoscopy demonstrated gastric mucosal atrophy, non-bleeding duodenal ulcers, and segmental severe inflammation, concerning for a mixed picture of CD and IBD. Eventual pathology was more consistent with mixed celiac and microscopic colitis picture; however, T cell derangement was seen, concerning for T-cell lymphoma.</p> <p>Despite continued adherence to a GFD and newly initiated steroid therapy, the patient was re-admitted with continued abdominal pain and watery diarrhea 3 weeks later. ESR was mildly elevated at 13, but CRP was within normal limits. On repeat EGD and colonoscopy, pathology revealed evidence of refractory celiac disease (RCD), type II, which carries a poor prognosis and increased risk of progression for enteropathy associated T-cell lymphoma (EATL). Flow cytometry yielded abnormal T cell population (CD3+, CD8/4/5/30-), however, not conclusive for lymphoma. PET scan with no evidence of mass. He was continued on long-term steroids and GFD, however, had continued worsening of symptoms over the following months. The patient continued to lose weight in the setting of his abdominal symptoms and underwent multiple re-admissions with 4 EGD and 2 colonoscopy procedures within a 9-month period. He was started on</p>

	<p>additional TPN without improvement and unfortunately passed due to complications from RFD.</p> <p>Discussion: CD is one of the most common autoimmune diseases and is prevalent in approximately 1% of the Western population. Complications include neurological symptoms, cutaneous lesions, osteopenia, as well as increased risk for gastrointestinal malignancies. While treatment of CD involves strict adherence to a lifelong GFD, there remain rare instances of patients who do not respond to such modifications. Patients who do not respond to 1 year of diet modification are classified as RCD. This encompasses 2 classes, RCD type I, characterized by normal intraepithelial lymphocytes (IELs), and RCD type II, characterized by abnormal IELs. This case highlights the diagnosis of RCD, type II, which is considered an in situ aggressive T-cell lymphoma with high rates of progression to EATL, which makes its diagnosis important to consider when treating patients with CD.</p>
<p><b>Dina Belhasan</b> Dr. Thomas Leventhal  Finalist</p>	<p><i>Now You See Me, Now You Don't: A Case of Liver Injury that Wasn't</i></p> <p>Introduction: Metastatic prostate cancer, and its directed treatments, can be associated with elevated liver function tests (LFTs) – raising concerns for liver injury. This case presents a patient with metastatic prostate cancer who exhibited sustained AST and ALT elevations without any signs of hepatocellular injury, inflammation, or dysfunction.</p> <p>Case Presentation: A 75-year-old male with metastatic prostate cancer was referred to hepatology after routine labs revealed a serum AST of 230 and ALT of 747. The patient exhibited no symptoms or exam findings suggestive of hepatic dysfunction, aside from mild fatigue over the previous month. His chemotherapy included denosumab, leuprolide, and enzalutamide, which he had been taking for several months without changes. The patient had no risk factors for liver disease; he reported infrequent alcohol use, no use of dietary supplements, and no known family history of liver disease. A thorough medication review indicated no drugs associated with significant risk of liver injury. His chemotherapy medications were classified as "Category E," indicating a very low risk of causing acute liver injury.</p> <p>Laboratory results showed normal INR, TSH, and iron studies. He underwent a comprehensive lab evaluation to assess for genetic causes of chronic liver disease, potential muscle breakdown, autoimmune liver disease, deposition disorders, metabolic causes, and various infectious diseases affecting the liver. All tests returned negative including: infectious hepatitis, EBV, CMV, HSV, serum IgG, anti-smooth muscle IgG, IgA, CK, ceruloplasmin levels, alpha-1 antitrypsin, tissue transglutaminase IgA, and paraneoplastic antibodies (ANNA-1, ANNA-2, PCCA-1, and PCCA-Tr [DNER]). ANA was borderline positive at 1:40, but negative three months later. An abdominal ultrasound revealed normal-appearing liver parenchyma with borderline enlargement. The patient underwent percutaneous liver biopsies that sampled both the right and left liver lobes at three and five months after his initial visit; both showed absence of any degree of hepatitis or ongoing liver injury, without evidence of fibrosis.</p> <p>Given negative workup but possibility of a drug induced injury not captured on biopsy, he was trialed on a prednisone taper starting at 40 mg daily;</p>



	<p>given no improvement in transaminases, therapy was discontinued. Throughout the several months his evaluation took place, AST/ALT remained persistently elevated, ranging from ~300 to 1300. Approximately seven months after the initial elevation, the patient’s LFTs improved to below 300 but did not fully normalize following the cessation of his initial chemotherapy and anti-hormone therapy.</p> <p>Discussion: Cases of elevated LFTs observed in metastatic prostate cancer most often indicate liver injury and dysfunction. This case presents a unique instance of elevated AST/ALT levels without any signs of liver injury, inflammation, or dysfunction - in the context of an extensive, negative evaluation. The etiology of these elevations remains unclear; however, we hypothesize an as-yet unspecified paraneoplastic process may account for the elevations. Alternatively, another plausible hypothesis is that this may be a drug-induced phenomenon impairing clearance of ALT and AST enzymes secondary to this patient’s chemotherapy regimen. This case also highlights the importance of maintaining a broad differential and performing a diligent evaluation in cases of abnormal LFTs.</p> <p>Diagnosis was confirmed when the CSF and serum returned positive for West Nile virus IgM. Acyclovir was discontinued after CSF VZV/HSV PCR returned negative. The patient was treated with supportive cares, and her mental status improved over the next 48 hours allowing a more comprehensive motor assessment. Her exam showed moderately severe patchy flaccid paralysis in her limbs consistent with multifocal anterior horn cell injury. The patient was counseled that despite improvement in her encephalopathy, she will likely experience some permanent motor impairment as the viral damage to the anterior horn cells is largely non-reversible. She continued to work with PT and OT, and placement was arranged at a SNF for ongoing therapy.</p> <p>Discussion: This case demonstrates typical risk factors for West Nile virus infection: occurrence in late summer in a patient who is frequently outdoors and from the Midwest. It also illustrates that attention to the neurological exam provides clues to nervous system injury that could otherwise be masked by metabolic/infectious encephalopathy. Given the variety of syndromes West Nile virus can produce and its increasing incidence, providers should have a low threshold for including this virus on the differential for any case with a presentation in this realm.</p>
<p><b>Nicholas Bergeron</b></p> <p><b>Finalist</b></p>	<p><i>Rocky Mountain Spotted Fev-Ear: A Case of Rickettsial Induced Hearing Loss</i></p> <p>Case Presentation: A 21-year-old male with medical comorbidities significant for obstructive sleep apnea and a prior traumatic brain injury presented to the hospital with hearing loss, fever, and severe headache. He also had a history of ongoing vague symptomatology since being stationed in Africa 1 year prior to presentation including significant weight gain, personality changes, and fatigue. One week prior to presentation, he started to notice worsening fatigue, periodic headaches, tachycardia and nausea. Two days later, his headache intensified in severity and was associated with almost complete hearing loss on the right side and a fever to 39oC.</p> <p>He presented to the emergency department locally. The patient was</p>

	<p>originally from suburban Oklahoma. He was previously a part of the military and was deployed to Eastern Africa for 6 months. He traveled to rural areas frequently and spent significant time around cattle and horses. He reported frequent tick bites. He was not sexually active, and adamantly denied any sexual activity during his deployment. A CT of the head was performed and was without acute intracranial abnormality. A lumbar puncture did not demonstrate evidence of meningitis. Serologic testing including for <i>Rickettsia rickettsii</i>, <i>Francisella tularemia</i>, Lyme Disease, and autoimmune testing were performed and negative. Microbial cell-free DNA testing (Karius Test™) was ordered.</p> <p>Three days prior to presentation, he began to develop a nonpruritic, nonblanchable rash which spared the face but was present on the palms and soles. He had an allergy to penicillin and was empirically started on ceftriaxone and doxycycline due to concerns for syphilis, though RPR was negative. He was started on high dose methylprednisone due to hearing loss. He presented to this hospital for a second opinion. His headache had improved and the rash had resolved from on the soles of his feet. An MRI of his brain demonstrated punctate foci in the inferior right parietal lobe and right centrum semiovale representing tiny acute infarcts. Microbial cell free DNA testing returned positive for <i>Rickettsia rickettsii</i>. Serologic testing was positive for Spotted Fever Group IgG at a titer of 1:512. Ceftriaxone was discontinued and he was treated for 10 days with doxycycline. He had significant improvement in his acute symptoms, though his hearing has not returned to baseline.</p> <p>Conclusion: Rocky Mountain Spotted Fever (RMSF), caused by <i>Rickettsia Rickettsii</i>, should be a suspected cause in presumed infectious hearing loss. Typically, <i>Rickettsial</i> induced hearing loss is typically seen in fulminant cases and is thought to be secondary to vasculitis. Meningitis is an important diagnosis to exclude and empiric antibiotics can be considered given the morbidity of disease. When an infectious cause is suspected, and meningitis has been effectively ruled out, it is important to consider spirochete diseases such as syphilis and RMSF as an acute cause of hearing loss. The sensitivity and specificity of serologic testing vary widely depending on the assay. Often, when rash presents in RMSF, it is often later in the disease course. If RMSF is suspected, empiric doxycycline should be initiated.</p>
<p><b>Carla Borre</b> Dr. Ronald Go</p>	<p><i>Kitchen and Laundry Cravings: Curb Your Enthusiasm with Iron</i></p> <p>Case Presentation: A 38-year-old female with no significant comorbidities was referred to Hematology following the evaluation for sclerotic bone lesions in the shoulders, hips, knee joints, and calvarium. These lesions were first identified on an MRI of her left knee performed after a mechanical fall. During her workup, she was incidentally found to have anemia. Although initially concerned about a potential histiocytic neoplasm like Erdheim-Chester disease, her evaluation revealed concurrent iron deficiency anemia, which became a key focus of her clinical management.</p> <p>For several years, she had been having fatigue and brain fog. Interestingly, she described an intense craving for the smell of laundry detergents, particularly Gain®. She also reported an obsession with chewing pickles,</p>

	<p>specifically craving the crunchy feeling. She did not have pica. No pallor, hepatosplenomegaly or lymphadenopathy were identified on the physical exam. Cardiovascular and neurologic exam were unremarkable. Laboratory findings at the time of visit revealed a ferritin of 24 ng/mL (reference range: 11–307 ng/mL). However, this was 12 days after an intravenous iron infusion given by her primary care physician. Other tests were also consistent with iron deficiency anemia, including a hemoglobin of 10.8 g/dL (reference range: 11.6–15.0 g/dL), mean corpuscular volume (MCV) of 76.8 fL (reference range: 78.2–97.9 fL), and red cell distribution width (RDW) of 30.0% (reference range: 12.2–16.1%). She also had deficiencies in vitamin D (8.5 ng/mL; &lt;10 ng/mL severe deficiency) and vitamin B12 (133 ng/L; reference range: 180–914 ng/L). Testing for intrinsic factor blocking antibodies was negative, ruling out pernicious anemia. Six years prior, she had undergone Roux-en-Y gastric bypass surgery, likely the underlying cause of her malabsorption of iron and other nutrients, leading to recurrent iron deficiency anemia, as well as vitamin D and B12 deficiencies.</p> <p>Conclusion: This case highlights a classic example of desiderosmia and hapticophagia. Desiderosmia refers to an excessive craving for specific smells, often associated with iron deficiency. This condition was described in a series of cases published in 2017, where symptoms resolved with iron supplementation. Hapticophagia, on the other hand, involves tactile cravings related to chewing and was documented in a 2020 case series, where most patients reported resolution of chew cravings after iron repletion. Both symptoms are distinct from pica, as they do not involve a desire to ingest the craved substance. To date, no published cases have documented a patient presenting with both desiderosmia and hapticophagia simultaneously, making this case particularly noteworthy.</p> <p>Remarkably, her desiderosmia and hapticophagia resolved within two weeks of receiving an additional intravenous iron infusion, underscoring an association between iron repletion and the resolution of these cravings. After intravenous iron replacement, the patient was recommended regular monitoring of her red blood cell indices and ferritin levels. Further evaluation, including tibial biopsies, was negative for hematologic malignancies or other significant pathology. Molecular testing for the BRAF V600E mutation, commonly associated with Erdheim-Chester disease, was negative.</p> <p>In summary, this case emphasizes the importance of recognizing atypical symptoms of iron deficiency and considering the broader implications of a patient's surgical history in managing chronic nutrient deficiencies.</p>
<p><b>Abhishek Chandra</b> Dr. Yahye Samatar Dr. Amy Holbrook</p>	<p><i>Back to Basics: The Pharmacokinetics of Direct Oral Anticoagulants in Short-Gut Syndrome</i></p> <p>Introduction: Direct-acting oral anticoagulants (DOACs) are a staple alternative to warfarin for stroke prevention and thromboembolism treatment and prevention. DOACs are absorbed in the upper gastrointestinal tract, but patients with altered gastrointestinal anatomy or physiology were not included in the Phase II and III investigations that assessed their efficacy and safety. This report details a patient with altered gastrointestinal anatomy and physiology who developed splenic infarcts</p>

	<p>while on oral anticoagulation.</p> <p>Case Description: A 37-year-old male with Pelizaeus-Merzbacher Disease (an inherited CNS hypomyelination leukodystrophy) and a JAK2 mutation with recurrent DVTs on rivaroxaban presented to the emergency room with abdominal pain. History revealed that he had been taking half-doses of his recommended rivaroxaban. Computed tomography (CT) imaging revealed a superior mesenteric vein thrombus that extended to his portal vein. The patient was started on catheter-directed thrombolytics. A transhepatic intrajugular portosystemic shunt (TIPS) was attempted to address the thrombus, but was complicated by large volume hematochezia necessitating emergent exploratory laparotomy with resection of approximately 120 cm of necrotic small bowel. In the coming days, he underwent a second TIPS procedure that was successful and a second small bowel resection leaving approximately 150 cm of small bowel intact. Postoperatively, he required vasopressor support, total parenteral nutrition, and intravenous heparin.</p> <p>Ten days after admission, he was transferred out of the intensive care unit. He began to show signs of worsening mentation and was found to have hepatic encephalopathy. A percutaneous endoscopic gastrostomy tube was placed and he was started on lactulose therapy which was effective. His mentation was sensitive to any discontinuation of lactulose and so the taper was slowed. He was transitioned to tube feeds and from heparin to 5 mg of apixaban twice-a-day. Thirteen days later, he developed worsening abdominal pain with distension, decreased responsiveness, and had a large-volume emesis. His tube-feeds were held and his lactulose was increased. CT imaging revealed multiple splenic infarcts and he was transitioned back to heparin. Once able to tolerate tube-feeds, he was transitioned back to apixaban.</p> <p>He continued to have high stool output requiring large amounts of fluids and free water boluses. With concern for short-gut syndrome, he was started on octreotide and was transitioned to rifaximin from lactulose for his mentation. There was also concern that with the large volume of diarrhea, apixaban absorption would not be dependable and the patient was transitioned to therapeutic heparin.</p> <p>The patient remains hospitalized with ongoing effort to balance diet advancement, diarrhea mitigation, and prevention of recurrent encephalopathy. He remains on therapeutic intravenous anticoagulation.</p> <p>Discussion: Patients with Janus Kinase 2 (JAK2) mutations are at greater risk for thrombus development and complications and may require lifelong anticoagulation. For many patients, oral anticoagulation is sufficient and appropriate management for thromboembolism prevention. DOACS, however, are absorbed largely in the upper gastrointestinal tract. In patients with altered gastrointestinal anatomy or physiology (short-gut syndrome, lactulose, bacterial overgrowth) it is critical to be cognizant of the pharmacokinetics of enterally absorbed medications as their efficacy may be limited or function entirely impaired.</p>
<p><b>Ellenor Chi</b> Dr. Ganiru Mathis</p>	<p><i>A Fowl Case of Diarrhea</i></p>

<p>Dr. David Phelan</p>	<p>Case Presentation: A 66-year-old woman with a history of Crohn’s disease, previously stable on infliximab, presented to urgent care with multiple episodes of non-bloody diarrhea and fever. Vitals were notable for a blood pressure of 151/81, heart rate of 123, and temperature of 102.5F. Laboratory testing revealed negative respiratory viral panel, negative GI pathogen panel, normal lactate (0.9 mmol/L), elevated CRP (37.4 mg/L), and elevated fecal calprotectin (121 mcg/g). She was prescribed levofloxacin and instructed to defer her upcoming infliximab infusion in the setting of suspected infection versus a Crohn’s disease flare. Following her release from urgent care, the patient was informed that her blood culture was positive for <i>Listeria monocytogenes</i> in one of two bottles at twenty-one hours, and she presented for hospitalization.</p> <p>The patient recalled that a few days prior to the onset of her symptoms, she had consumed a few bites of turkey deli meat before disposing of it. On admission, she was neurointact but reported recent onset of headaches and memory issues, in addition to gastrointestinal distress and malaise. As this was concerning for potential CNS infection, she was initiated on both ampicillin and gentamicin. Her physical exam was positive only for abdominal tenderness and was without nuchal rigidity or other focal findings. Cerebrospinal fluid analysis was notable only for elevated protein (70 mg/dL) with a negative culture, gram stain, and meningitis-encephalitis panel. Consequently, as there was no sign of intracranial involvement, gentamicin was discontinued. The patient clinically improved on ampicillin. Following negative repeat blood cultures, she was discharged on a four-week course of antibiotic therapy with infliximab infusion held until after completion.</p> <p>Discussion: <i>Listeria monocytogenes</i> is an intracellular, gram-positive bacillus which can cause invasive disease in neonates, elderly adults, pregnant women and immunosuppressed patients, such as the patient on infliximab above. <i>L. monocytogenes</i> is typically transmitted as a food-borne illness, with multiple outbreaks occurring in 2024. Incidence of laboratory confirmed cases is low but should be reported to the CDC. At risk patients should be counseled on the risks of consuming unpasteurized foods, as well as to thoroughly cook their meals. Invasive infection typically manifests as central nervous system infection (meningitis or meningoencephalitis), febrile or nonfebrile gastroenteritis, or bloodstream infection with variable symptoms. Our patient presented with febrile gastroenteritis but had few classic symptoms of meningitis such as nuchal rigidity and photophobia. In fact, she was without chills or rigors on admission despite known bacteremia.</p> <p>Diagnosis of gastroenteritis is not always feasible due to low sensitivity of stool testing; however, blood cultures and cerebrospinal fluid analysis can establish a diagnosis of bacteremia and CNS infection respectively. Treatment should be initiated promptly, given high risk of mortality. Preferred antibiotic therapy is ampicillin or penicillin with a short course of gentamicin if there is concern for CNS involvement or severe disease. Alternative regimens can include TMP-SMX or carbapenems. Therapy duration varies based on extent of infection and whether the host is immunocompromised but is typically between two to six weeks.</p>
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**Sung Jun Cho**  
Dr. Hussein Magale  
Dr. Kiril Dimitrov

*Cemented in Place: Kyphoplasty-Associated Pulmonary Cement Embolism: A Case Report*

Background: Kyphoplasty-associated cement extravasation into surrounding tissue and vasculature can lead to life-threatening complications. We present a rare case of significant inferior vena cava cement burden that resulted in pulmonary embolism.

Case presentation: A 74-year-old Caucasian woman with a history of severe osteoporosis, recurrent falls, end-stage renal disease on dialysis, and spinal compression fracture status-post kyphoplasty of the L4–L5 vertebrae, presented to the emergency department 2 days post-vertebral kyphoplasty due to chest pain, back pain, and dyspnea.

Lab findings were notable for elevated serum creatinine of 5.27 mg/dL and BUN of 38.3 mg/dL. Additional findings on metabolic panel showed hypocalcemia of 7.7 mg/dL and hyperphosphatemia of 6.2 mg/dL. Liver function tests showed elevated alkaline phosphatase at 360 U/L, while ALT, AST, and total bilirubin were within normal limits, 12 U/L, 21 U/L, and 0.6 mg/dL respectively. Complete blood count was notable for mild anemia with hemoglobin of 11.2 g/dL, white count of 10.2 10<sup>3</sup>/uL, and platelets of 164 10<sup>3</sup>/uL. Computed tomography of the chest and abdomen showed a metallic density within the inferior vena cava extending superiorly approximately 10 cm from the vertebral L5 level. She was also found to have right lower lobe pneumonia. The patient finished a 10-day course of antibiotics and was discharged home with a 1-month long course of anticoagulation with apixaban per recommendations of a multidisciplinary team consisting of Hematology/Oncology, Interventional Radiology, Vascular Surgery, and Orthopedic Surgery.

Unfortunately, the patient was readmitted a month later with shortness of breath. Work up was notable for an influenza type A infection and computed tomography findings of pulmonary cement embolism. The respiratory distress was resolved with supportive care. Despite pulmonary cement burden, the multidisciplinary care team recommended no further anticoagulation. Patient was discharged home with close clinical follow-up and 6 months has since passed at the time of this report without reported complications.

Discussion: This is a rare case of a large cement burden in the inferior vena cava after a kyphoplasty procedure leading to pulmonary cement embolism. However, extravasation of cement material into surrounding tissues and vasculature may be underreported. This case asks the question of whether postoperative imaging should be standard of care following kyphoplasty, especially in elderly patients with a history of falls. It is unclear if anticoagulation would have prevented worsening of her symptoms and whether the benefits outweigh potential bleeding. Lastly, this case underscores a continual need to examine and improve our understanding of percutaneous methods of treating vertebral fractures, both in whether one method (kyphoplasty versus vertebroplasty) is superior and whether better materials exist to meet the increasing burden of osteoporosis induced compression vertebral fractures in our aging population.

**Brett Cornforth**  
Dr. Charlie  
Reznikoff

*Prosthetic Valve Endocarditis in a Patient with Fabry Disease and Complex Cardiac Comorbidities*

Introduction: Infective endocarditis of a bioprosthetic valve requires surgical replacement to cure the infection. There are many complications that can arise during the surgical pre-operative phase that impact the plan of care. This clinical vignette explores multiple such complications in a patient with Fabry disease, a rare lysosomal storage disease which caused multiple cardiac manifestations in this patient.

Case Description: A 67-year-old female presented with shortness of breath and was found to be in atrial flutter and had clinical signs of new heart failure. She had a significant cardiac history including Fabry disease causing infiltrative cardiomyopathy with basal inferolateral scarring, ascending aortic aneurysm requiring repair, bicuspid aortic valve requiring bioprosthetic valve replacement, and coronary artery disease with stenting of the second diagonal branch of her left anterior descending coronary artery (LAD). The etiology of her acute heart failure was multifactorial due to Fabry disease, tachyarrhythmia-induced from atrial flutter, and ischemic cardiomyopathy. She was successfully cardioverted into sinus rhythm after loading with amiodarone and started on a heparin drip. Transthoracic echocardiogram revealed a mass on the aortic valve. Follow-up transesophageal echocardiogram confirmed a mass on the aortic valve, as well as a mass on the native mitral valve, and a perforated mitral leaflet causing moderate posterior regurgitation. Infectious disease was consulted for antibiotic management of bioprosthetic aortic valve and native mitral valve endocarditis. With blood cultures showing no growth, she was empirically treated with vancomycin and ceftriaxone, which were continued until her valves were replaced.

Cardiothoracic surgery was consulted for evaluation for combined valve replacements. Surgical planning was complicated due to findings of scattered acute embolic infarcts on her brain MRI. Neurology concluded that the emboli were sterile, likely cardio-embolic from when she was in atrial flutter. If the emboli had been septic, this would have caused a delay in her surgery, as septic emboli are at a higher risk for hemorrhagic conversion. Coronary angiogram found that she had total occlusive disease in her mid-LAD at the level of her previously stented second diagonal, with 99% in-stent re-stenosis. Cardiothoracic surgery recommended single-vessel coronary artery bypass grafting (CABG) to address the obstructive coronary artery disease (CAD). The patient successfully had open-heart surgery with aortic valve replacement, mitral valve replacement, and single-vessel CABG to the LAD.

Discussion: This is a case of a patient with many cardiac manifestations of Fabry disease, and infective endocarditis affecting both native and bioprosthetic heart valves. Prosthetic valve endocarditis is a well described complication of aortic valve replacement. Studies have found that bioprosthetic valves, like the one this patient had, are more likely to become infected than mechanical valves. Individuals with prosthetic valves are also far more likely to get endocarditis than individuals with native valves. Fabry disease has multiple cardiac manifestations including cardiomyopathy

	<p>(typically left ventricular hypertrophy but can also cause scarring as in this case), aortic aneurysms, valvular disease, and coronary artery disease, and should be considered when etiology of cardiac disease is unknown, especially when infiltrative disease is suspected.</p>
<p><b>Margaret Crosby</b>  Dr. Anna Cox  Dr. Peter Hannon  Dr. David Flemig  Dr. Charles Reznikoff</p>	<p><i>The Role of Addiction Medicine Collaboration During Cancer Treatment in Patients with Head and Neck Cancer: A Case Report</i></p> <p>Introduction: Patients with head and neck cancer (HNC) have higher rates of severe cancer pain and many continue to experience pain for years following the completion of cancer treatment. This, combined with higher rates of tobacco use, alcohol use, existing opioid use and other substance use disorders among HNC patients, increases the risk of long-term opioid therapy (LTOT) and opioid use disorder (OUD). As cancer survival rates improve, this clinical situation is increasingly common and there is sparse evidence to direct clinical care. Thus, the development of safe and effective regimens for pain management is needed in these complex situations.</p> <p>Case Presentation: We present a case of a 70-year-old man with a history of opioid use disorder and ongoing heroin use who underwent concurrent chemoradiotherapy for squamous cell carcinoma of the vocal cords and larynx. His treatment course was complicated by psychosocial barriers, missed appointments, prior adverse reaction to buprenorphine/naloxone, and self-treatment of his cancer-related pain with heroin. He was referred to Palliative Care for management of his cancer-related pain. Medications for opioid use disorder were recommended prior to use of full opioid analgesics for cancer pain. However, the patient had adverse effects from buprenorphine/naloxone and his cancer treatment schedule conflicted with the methadone clinic schedule.</p> <p>Given the complexity of this patient’s pain requirements, Addiction Medicine was consulted, and they collaborated on initiation of buprenorphine patches, which led to improvement in cancer pain, decreased symptom burden, and better treatment adherence.</p> <p>Conclusion: One existing case reports the benefit of Addiction Medicine consultation for opioid de-escalation support in a HNC survivor with long-term opioid use. Here we document the role of collaboration between Palliative Care and Addiction Medicine during active cancer treatment to support a HNC patient with significant OUD to develop safe pain management strategies.</p>
<p><b>Tyler Crowe</b>   Finalist</p>	<p><i>Empyema Necessitans: A Rare Complication Despite Clinical Improvement</i></p> <p>Introduction: Empyema necessitans is a rare complication of empyema where pleural fluid spreads through the chest wall into the subcutaneous thoracic tissues. Its delayed presentation despite otherwise overall clinical improvement presents a diagnostic challenge.</p> <p>Case Presentation: A 68-year-old woman was admitted to the medical ICU for altered mental status and shock. Of note, she had recently been hospitalized for treatment of a community-acquired pneumonia complicated by a left-sided parapneumonic effusion requiring chest tube</p>



	<p>placement and removal later that hospitalization 3 weeks prior. Though thorough workup on admission did not demonstrate a clear infectious cause, pulmonary edema and a left-sided pleural effusion were present. Her respiratory clinical picture was clouded by comorbid COPD requiring oxygen at baseline and heart failure with preserved ejection fraction. She responded well to empiric treatment with high-dose steroids, broad-spectrum antibiotics, and aggressive diuresis (net negative 20 liters), gradually improving to her home oxygen requirement in 10 days. She was afebrile throughout. During this time she complained of acute-on-chronic back pain, especially over her left thoracic region. She had fallen several times in the week prior to admission, and took pain medication daily. Serial examination initially showed diffuse tenderness without other abnormalities progressing to profound tenderness and induration in 48 hours. Subsequent CT imaging showed a left-sided, loculated pleural effusion with significant extra-thoracic extension consistent with empyema necessitans. This was successfully drained by Interventional Radiology, who placed two drains via CT-guidance. Initial output was noted to be purulent, though cultures had no bacterial or fungal growth and acid-fast staining was negative. Drain output quickly slowed. Her pain was improved, and she remained otherwise both hemodynamically stable and afebrile as before. She was transferred successfully from the ICU, but unfortunately her respiratory status later worsened. In the setting of these setbacks, anticipating the complex cardiothoracic procedures to completely treat her empyema necessitans, and her overall declining health, the patient elected for comfort care. She passed away peacefully with her family present at bedside.</p> <p>Discussion: This case shows the importance of thorough physical examination and reassessment of the complete clinical picture. Because of the rarity of this complication and insidious onset, empyema necessitans can be a challenge to many clinicians. Given the significant morbidity and complexity of management, recognition of this rare complication without obvious clinical presentation is important for all clinicians.</p>
<p><b>Timothy Davie</b>  Dr. Samarth Goyal  Dr. Deniz Aslan  Dr. Daniela Guerrero Vinsard</p>	<p><i>Rectosigmoid Involvement of Mpox in Immunodeficiency</i></p> <p>Introduction: Mpox is a zoonotic virus more commonly found in endemic regions with a notable outbreak in the U.S. in 2022. One of its defining features is a papular rash found in oral, genital, and perianal regions, but in rare cases can involve rectosigmoid mucosa and cause proctocolitis.</p> <p>Case Presentation: A 50-year-old male with a history of HIV, not on antiretroviral therapy (ART) for the past 3 years due to medication intolerance, presented to the emergency department after one week of malaise, abdominal cramps, headaches, nausea and vomiting, and bright red blood per rectum with associated pain. He also developed a pruritic papular and pustular rash found on his back, abdomen, forearms, scalp, tongue, and perianal region (Figure 1). CD4 count was found to be 172 cells/mm<sup>3</sup>, consistent with AIDS. A CT of the abdomen and pelvis showed diffuse wall thickening of the rectosigmoid colon with inflammatory stranding and adenopathy (Figure 2). A flexible sigmoidoscopy was performed, showing significant non-bleeding rectal ulceration and erythematous mucosa with friability (Figure 3). There were also smaller erosions present in the sigmoid colon. Histopathology of the rectal ulcer biopsy demonstrated inclusion-like</p>

	<p>crypt epithelial nuclei with multinucleation with surrounding suppurative inflammation and necrosis (Figure 4). CMV, HSV type 1 and 2, VZV, spirochete immunohistochemistry testing of the rectal ulcer tissue was negative. Acid-fast and fungal staining of the tissue found no organisms. A skin and rectal lesion was swabbed and PCR-based DNA assay for viral testing was performed. The Minnesota Department of Health confirmed a positive mpox (orthopoxvirus) test. The patient was restarted on ART and on tecovirimat via clinical trial.</p> <p>Conclusion: This case demonstrates the rare presentation of proctocolitis in an immunodeficient patient with Mpox. Identifying Mpox infection can be challenging as patient symptoms and appearance of the rash may be non-specific. Utilizing endoscopy and mucosal biopsy may be beneficial in patients with symptoms concerning for Mpox who present with rectal bleeding or rectosigmoid wall thickening identified on imaging.</p>
<p><b>Madeline DeAngelo</b>  Dr. Griffin Reed  Dr. John Davis</p>	<p><i>A 45-year-old man with Joint Pain and Polyneuropathy</i></p> <p>Case Presentation: A 45-year-old man presented to the emergency department for chronic abdominal pain. His medical comorbidities included longstanding tobacco use with 30 pack-years, prior alcohol use, and esophagitis with gastric and duodenal ulcers. Recent past medical history included new diagnosis of rheumatoid arthritis, necrotizing pancreatitis, and progressive polyneuropathy including a foot drop.</p> <p>Five months prior to admission, he developed bilateral hand and foot pain. His primary care provider referred him to a local rheumatologist. Three months prior to admission, he received a diagnosis of seropositive rheumatoid arthritis with elevated rheumatoid factor and anti-cyclic citrullinated peptide (anti-CCP) antibodies. He was treated and maintained on low-dose prednisone due to intolerance of methotrexate. Two months prior to admission, he developed shooting pains in his bilateral hands and feet followed by ulcerative lesions on the distal bilateral hands and feet. These lesions became necrotic, and he subsequently developed paresthesias with predominantly distal weakness and difficulty with ambulation.</p> <p>Upon admission his vitals demonstrated mild hypertension and tachycardia. Exam was notable for a chronically ill-appearing man with bilateral hyperreflexia, red-brown macules on the digits of both hands, multiple &lt;1 mm purpura of the periungual area and digital pulp, a circular necrotic ulceration on the distal right great toe, and an ulceration of the distal left great toe. He had marked weakness of his distal extremities.</p> <p>Laboratory evaluation revealed significantly elevated rheumatoid factor and anti-CCP antibodies, decreased complement levels, and negative hepatitis serologies. Imaging demonstrated peripheral wedge-shaped defects in both kidneys and multiple small aneurysms of the vessels within the kidneys and mesenteric distribution. Sural nerve biopsy showed epineural perivascular inflammatory cell collections consistent with rheumatoid vasculitis. High-dose intravenous steroids and rituximab were initiated.</p> <p>Conclusion: Rheumatoid vasculitis is an extra-articular manifestation of rheumatoid arthritis. It typically presents in patients with longstanding</p>

	<p>rheumatoid arthritis but can less commonly present early in disease course. The clinical features of rheumatoid vasculitis are diverse, with some individuals affected by only localized or self-limiting disease, and some affected by life-threatening end-organ ischemia. Clinical features often include peripheral neuropathy, necrotic ulcers, hemorrhagic blisters, palpable purpura, and constitutional symptoms, but can progress to involve multiple end organs manifesting as bowel ischemia or myocardial infarction.</p> <p>Rheumatoid vasculitis is primarily a clinical diagnosis but does involve serological markers and often histopathology. Serological markers of rheumatoid factor and anti-CCP are typically strongly positive. Histopathology may show evidence of an inflammatory infiltrate, fibrinoid necrosis, perivascular cuffing, axonal degeneration, and immune complexes. The treatment for rheumatoid vasculitis involves immunosuppression, with the cornerstone of initial management being high-dose glucocorticoids. Glucocorticoids in combination with either rituximab or cyclophosphamide are used to control disease, with use of rituximab often being the clinician’s choice given the more favorable safety profile. The prognosis of rheumatoid vasculitis remains quite poor despite improvements in immunosuppressive therapies.</p>
<p><b>Joshua Elmer</b>  Dr. Claudia Gyimah  Dr. Gustavo Cortes  Puentes</p>	<p><i>A Classic Presentation of Cefepime Neurotoxicity</i></p> <p>Case presentation: A 68-year-old female with history significant for skull base osteomyelitis on 6-week course of cefepime presented from home with 1 day of altered mental status, nausea, and vomiting. On day of presentation, she woke up confused appearing and nonverbal in response to her spouse’s questions. Her husband also noted new occasional jerking movements in the upper extremities. He denied any known toxic ingestions and reported good compliance with her home medications.</p> <p>Regarding her osteomyelitis, cefepime was initiated 3 weeks prior to presentation and was self-administered at home through a PICC line at a dose of 2 grams every 12 hours. Two days prior to presentation, labs revealed newly elevated creatinine at 3.63. Patient was contacted and cefepime dose was decreased to 2 grams every 24 hours. Vitals on admission to the ICU were HR 96, RR 20, BP 158/86, 94% on room air. On exam, she was obtunded and aphasic but with no focal weakness and normal cranial nerve exam. She was unable to follow commands and was minimally verbal, occasionally saying the words “what?” and “no.” Asterixis was noted in the left upper extremity. She had 5 beats of bilateral ankle clonus. Laboratory studies were remarkable for WBC 11.9, potassium 5.6, creatinine 6.45 (baseline 1.1-1.3), BUN 94, anion gap 18, bicarbonate 13, pH 7.25 on VBG, normal liver function tests. Urinalysis revealed proteinuria with predicted 24-hour protein of 2,791 mg/24 hour. Urine drug screen and ethanol level were unremarkable. CT head and MRI brain both redemonstrated findings consistent with known skull base osteomyelitis, but no hemorrhage, ischemia, or masses.</p> <p>The decision was made to change antibiotics to levofloxacin and vancomycin. Cefepime level from the day of admission returned on hospital day 2 and was very elevated at 84.1 mg/L. Due to consistent laboratory and exam findings, she was diagnosed with cefepime toxicity in setting of non-</p>

	<p>oliguric acute kidney injury. Over the following days, she made gradual improvement despite unimproved creatinine and BUN, initially with increasing alertness and speaking in 2–3 word phrases, and later conversing appropriately with full sentences by hospital day 4. Ankle clonus and asterixis improved around the same time and the patient was cleared for discharge home. At post hospital follow up 1 week after discharge, creatinine had improved to 3.74 and the patient was noted to have good mentation without relapse of symptoms.</p> <p>Conclusion: Cefepime neurotoxicity is a phenomenon in which high concentrations of the antibiotic cross the blood brain barrier and lead to encephalopathy via what is hypothesized to be GABA antagonism. Symptoms include a decreased level of consciousness, disorientation, and myoclonus. Less commonly, convulsive seizures and non-convulsive status epilepticus are possible. Clinicians should maintain a high index of suspicion for cefepime neurotoxicity in patients who develop new onset confusion, aphasia, or seizure with myoclonus in the setting of poor renal function, especially if dose adjustments have not been made. Prognosis for neurologic recovery is good and is reported as a median of 2 days following cessation of the drug.</p>
<p><b>Erik Engelsgerd</b> Dr. Breanna Zambinski</p>	<p><i>A World Turned Sideways. An Atypical Presentation of Dizziness Resulting from Pneumocephalus</i></p> <p>Introduction: Dizziness is a nonspecific symptom that is one of the most common complaints presented by patients. The differential is broad; common etiologies include peripheral vestibular dysfunction, central brainstem vestibular lesion, psychiatric disorder, as well as presyncope or disequilibrium [2]. A thorough history and physical exam are paramount to revealing CNS abnormalities necessary to detect during the first evaluation of the patient [3].</p> <p>Case Presentation: A 57-year-old female with a history of type II diabetes, hypertension, paroxysmal supraventricular tachycardia, pulmonary embolism (on warfarin), hypothyroidism, childhood ear infections, and obstructive sleep apnea presented to the emergency department due to dizziness and weakness. The patient endorsed ongoing intermittent dizziness for several months. On the day of arrival to ED, the patient underwent a hysteroscopy for an endometrial polyp. After the procedure, she reported feeling ill and fatigued. Every time she got up from a resting position, she developed dizziness and loss of balance. She also endorsed intermittent “bubbling” sounds in her left ear starting about 1 week prior to the ED visit. She noticed it most with changes in positions or differences in pressure. She also endorsed left ear fullness and muffled hearing. She denied ear discharge, ear fullness, fever, chills, trauma, recent airplane flights, and recent scuba diving. Per the instruction of her ENT specialist, patient had been washing her ears after showering with white vinegar and alcohol to keep them dry leading up to the ED visit. –On arrival, she was hemodynamically stable. No laboratory abnormalities noted. CT Head demonstrated a focal area of pneumocephalus in a non-vascular pattern and CT temporal showed dehiscence of the left tegmen tympani with circumscribed gas collection arising cephalad from the middle ear into the left middle cranial fossa. Subsequent MRI/MRV was without signs of</p>

	<p>infection and further CT head imaging showed stability in pneumocephalus. Neurosurgery was consulted and noted superior tegmen dehiscence. Patient was discharged and readmitted 1 week later for left middle fossa craniotomy for repair of tegmen dehiscence. Lumbar drain was placed without complication. Drain was eventually able to be removed and patient discharged. Follow-up CT head showed resolution of pneumocephalus.</p> <p>Conclusion: This case illustrates the importance of a complete evaluation for a patient presenting with dizziness. Even in a patient with a history of vertigo, such as this case, it is important to fully delineate the cause of new onset dizziness. It is crucial to rule out patients with a CNS etiology. A thorough neurologic exam is very important for these patients with extra attention being placed on eye movements, vestibular function, and hearing. [1] With the vestibular system being a brainstem function, your physical exam should test for cerebellar and brainstem dysfunction such as dysarthria, diplopia, facial weakness, ataxia, incoordination, dysconjugate gaze, etc [3] Overall, dizziness is a symptom of many different disease processes, and it is crucial to utilize a history and physical exam to find the “must not miss” etiologies that can save your patients from permanent damage.</p>
<p><b>Stefan Farrugia</b>  Jade Ho  Agata Sularz  Dr. Grace Ibitamuno  Dr. Sam Kosydar  Dr. Meltiady Issa</p>	<p><i>Unmasking the Hidden Culprit: A Case of Back Pain Leading to Osteomyelitis Diagnosis</i></p> <p>Introduction: Back pain is a common clinical complaint that can stem from a variety of causes, ranging from benign musculoskeletal issues to more serious underlying conditions. Vertebral osteomyelitis, a rare but significant infection, often presents with persistent back pain and can be challenging to diagnose due to its nonspecific symptoms. This case highlights the diagnostic journey of a patient presenting with back pain, ultimately found to have vertebral osteomyelitis.</p> <p>Case Presentation: An 87-year-old man, with past medical history of chronic low back pain, was admitted from his independent living to the hospital with progressive deterioration of low back pain to the point of being unable to walk. No trauma or precipitating event was known. His family reported an episode of urinary incontinence. Pain was localized to the lumbar area which was tender to palpation. No numbness or saddle anesthesia was noted, but lower extremity muscle strength was challenging to assess given the severe pain. Lumbar spine MRI showed progressed multilevel spondylotic changes, most pronounced at L3-4, with advanced spinal canal stenosis. Also noted was advanced active-on-chronic degenerative changes centered at the L3 interspace, but it was difficult to exclude infection. Conservative management and physical therapy were implemented with no success. A few days later, he developed leukocytosis of 11.4 with left shift and elevated CRP of 230, although he had no new localizing signs or symptoms. PET CT discovered active osteomyelitis in the L4 vertebral body. Blood cultures returned positive for Streptococcus bovis. Transesophageal echocardiography was negative for endocarditis. He started a 6-week course of IV ceftriaxone. His clinical condition stabilized, and he was discharged to acute rehabilitation with outpatient follow-up and colonoscopy to look for the source of Streptococcus bovis.</p>

	<p>Discussion: Vertebral osteomyelitis is a frequently missed diagnosis, especially in the elderly who may not mount the usual immune response. Prompt recognition is essential for improving outcomes. This complex case highlights the importance of clinical suspicion in working up osteomyelitis which was not seen clearly on MRI. Understanding the clinical presentation, diagnostic challenges, and management strategies for vertebral osteomyelitis is crucial for timely and effective treatment, potentially preventing severe complications.</p>
<p><b>Madeline Franke</b> Dr. Can Sungur</p>	<p><i>Hiding in Plain Sight: A Case of Severe Granulomatosis with Polyangiitis</i></p> <p>Case presentation: A 44-year-old woman with a medical history significant for hypothyroidism, tobacco use, and allergic rhinitis presented to the hospital with fatigue, dry cough, and poor oral intake.</p> <p>For three months prior to admission, she was seen by multiple providers for various evolving complaints including cough, shortness of breath, fevers, and pleuritic chest pain. She presented to the emergency room (ER) with these symptoms and chest x-ray revealed bilateral multifocal opacities, so she was diagnosed with pneumonia and discharged on antibiotics. She returned to the ER multiple times for worsening symptoms, each time being sent home with antibiotics. She was referred to pulmonology, but workup was delayed due to a rising creatinine. She was instructed to report to the hospital for urgent treatment of acute kidney injury and ongoing infectious symptoms.</p> <p>On admission, initial workup was notable for a creatinine of 1.96, along with acidemia, anemia, and elevated inflammatory markers: CRP significantly elevated at 244.8 mg/L and ESR &gt; 140 mm/1hr. There was increased concern for an underlying systemic inflammatory process and serologies returned with positive c-ANCA (1:1024) and PR3 antibodies (&gt; 8.0 U). A broad infectious workup was performed and was negative. She underwent bronchoscopy which demonstrated diffuse alveolar hemorrhage (DAH). Treatment was quickly initiated out of concern for progressing ANCA-associated vasculitis with intravenous methylprednisolone, rituximab, and avacopan, a complement 5a receptor antagonist. Confirmatory kidney biopsy was pursued due to her worsening kidney function. This biopsy revealed diffuse necrotizing and crescentic glomerulonephritis involving approximately 80% of glomeruli (c-ANCA-PR3 positive). Her renal function continued to decline requiring initiation of continuous renal replacement therapy. Additionally, she became hypoxic with episodic hemoptysis, requiring admission to the ICU for further management of her respiratory and renal function. Eventually, lower extremity doppler and chest angiogram revealed bilateral acute DVTs and acute subsegmental pulmonary emboli, respectively. High intensity heparin was initiated after being off anticoagulation because of her ongoing bleeding. Although initially tenuous, she continued to improve and was stabilized from a respiratory, renal, and hematologic standpoint. Her hemoptysis resolved and she was eventually discharged on Warfarin after discussions with vascular medicine. She continues to be managed by rheumatology and nephrology for vasculitis and hemodialysis, respectively.</p> <p>Conclusion: This case discusses ANCA-associated vasculitis complicated by</p>

	<p>severe DAH and renal involvement. It emphasizes the importance of early recognition of vasculitis and prompt initiation of treatment. Our patient had a total of 5 chest CTs and 2 chest x-rays before her hospitalization, all revealing evidence of worsening DAH. Although difficult to differentiate from infection, further investigation was warranted after her failure to respond to multiple rounds of different antibiotics. Finally, this case highlights the careful balance of bleeding and clotting in these patients. She was not receiving prophylactic anticoagulation for clots due to her bleeding risk in the setting of her DAH. She ended up with multiple acute DVTs and pulmonary emboli that complicated her hospitalization. Risks and benefits of anticoagulation (prophylactic and therapeutic) should be heavily weighed and individualized to the patient and their specific needs.</p>
<p><b>Gunjan Gaur</b> Dr. Nadir Bhuiyan</p>	<p><i>Chronic Kidney Disease from Renal Thrombotic Microangiopathy Following Autologous Hematopoietic Stem Cell Transplantation for Multiple Sclerosis</i></p> <p>Background: While hematopoietic stem cell transplantation (HSCT) is known to cause transplant-associated thrombotic microangiopathy (TA-TMA), autologous hematopoietic stem cell transplantation (auto-HSCT) is much less frequently implicated in TA-TMA than allogeneic HSCT and may go unrecognized, and the mechanism of pathology is unclear. To our knowledge, this is the first report of renal TMA (also called atypical hemolytic uremic syndrome) following auto-HSCT for treatment of multiple sclerosis.</p> <p>Case presentation: A 56-year-old female with a history of congenital solitary kidney and auto-HSCT for multiple sclerosis presented for evaluation of progressive chronic kidney disease (CKD) since auto-HSCT 13 months ago. She received cyclophosphamide/fluudarabine before transplantation, and immediate post-transplant course was complicated by febrile neutropenia from pyelonephritis treated with amoxicillin-clavulanate, new pancytopenia (Hgb 8.3 mg/dL, Plt 72,000/uL, WBC 0.04 x 10<sup>3</sup>/uL), bilateral hydrothorax, and bilateral lower extremity pitting edema. Urinalysis and serum creatinine (Cr) were reported to be normal, and she was discharged on an oral prednisolone taper. Over the following year, she had multiple hospitalizations for hypertensive urgency with systolic blood pressures over 200 mmHg and moderate-large pleural effusions, undergoing 8 thoracenteses yielding transudative fluid. Workup revealed progressive proteinuria (peak of 500 mg/dL), moderate red and white blood cells, and increasing serum Cr (peak of 6.98 mg/dL). Her symptoms were thought secondary to chronic glomerulonephritis, though renal biopsy was deferred due to risk with solitary kidney. C3 and C4 levels; ANCA antibodies; and serology for HIV-1/2, EBV, HBV, HCV, and HSV-1/2 were normal. Renal biopsy 10 months after HSCT ultimately showed chronic thrombotic microangiopathy (TMA), with moderate arteriosclerosis, mesangial lysis, and glomerular basement membrane duplication. Staining was negative for IgA, IgG, IgM, C1q, C3, and fibrinogen.</p> <p>Subsequent evaluation found no evidence of systemic TMA such as hemolysis or platelet clumping. Sequencing of ADAMTS13, C3, C5, CD46, CFB, CFH, CFHR1-4, genes implicated in complement-mediated TMA, found no known mutations. She was briefly treated with eculizumab, a terminal complement inhibitor, without improvement, and finally began regular</p>

	<p>hemodialysis for symptomatic uremia with vomiting, hypervolemia, oliguria, and mild confusion. She is now awaiting renal transplantation. Hypertension has been treated with the initiation and uptitration of four anti-hypertensives.</p> <p>Discussion: Broadly, TMA results from endothelial injury mediated by complement, certain infections, or auto-antibodies. TA-TMA typically presents within 6-12 months of HSCT with hemolysis and renal or other end-organ ischemic injury, though effects may be limited to the kidney. Our case demonstrates that a high index of suspicion among internists is needed to diagnose renal-limited TMA following auto-HSCT. Hypertension and proteinuria may be better markers than serum creatinine, and cyclophosphamide exposure is a risk factor. Lack of hemolysis does not rule out TMA, and renal biopsy is invaluable when the diagnosis is unclear. Further investigation of pathogenesis and treatment of TA-TMA is needed. While allo-HSCT-associated-TMA is thought to involve graft-versus-host disease, this cannot be the case in auto-HSCT, and our case found no complement abnormalities or instigating infections. Possibly, given our patient’s history of autoimmune disease, yet-unidentified auto-antibodies are responsible for TMA and may be therapeutic targets.</p>
<p><b>Cameron Gmehlin</b>  Dr. Regina Koch  Dr. John Bundrick</p> <p>Finalist</p>	<p><i>The Great Mimicker: A Case of Axial Gout Presenting as Osteomyelitis and Epidural Phlegmon</i></p> <p>Introduction: Gout is an inflammatory arthropathy caused by the deposition of monosodium urate (MSU) crystals into periarticular and extraarticular tissues. While it is traditionally thought to affect peripheral joints, it can be a “great mimicker” and deposit in any location in the body. Here we present a case of axial gout mimicking a spinal infection.</p> <p>Case Presentation: A 49-year-old male presented for evaluation of lumbar back pain with radiation into his lower extremities. His medical history included hypertension on amlodipine and isosorbide mononitrate, hyperlipidemia, primary hyperparathyroidism status post parathyroidectomy, and chronic kidney disease stage 4 (creatinine of 3.02). He had immigrated from Liberia two months prior and reported 4 month of night sweats and episodes of swelling in his right foot. He had been seen by his primary care provider and treated with antibiotics with no improvement in his symptoms. In the Emergency Department, he was hypertensive to 150/110 mmHg, tachycardic to the 130s, but otherwise afebrile and saturating well on room air. His physical exam was notable for diffuse soft tissue swelling of the left ankle and foot, focal tenderness of the 2nd and 3rd metatarsophalangeal joints, and right paraspinal tenderness. There were no strength or sensory deficits noted in his lower extremities. Initial laboratory analysis was notable for an elevated C-reactive protein, leukocytosis with left shift, and an acute elevation in creatinine to 3.31. CT lumbar spine was obtained and suggestive of L5-S1 discitis/osteomyelitis with a phlegmonous mass on the right anterolateral aspect of L5. He was started on Ceftriaxone and Vancomycin and admitted to the floor.</p> <p>Upon admission, antibiotics were held, and an MRI was obtained redemonstrating CT findings. Vertebral biopsy was pursued which showed</p>



no signs of infection. Given his history of relapsing right foot pain, gout was considered and a serum uric acid level was found to be elevated to 12.4. Aspiration of the left ankle joint was attempted but revealed no MSU crystals. He subsequently underwent DECT of his feet and lumbar spine which showed osseous erosive changes of his MTP/IP joints and T11-S1 endplates with green pixelation, consistent with MSU deposition. He was started on renal dose colchicine and low-dose prednisone with complete resolution of his symptoms.

Discussion: Axial gout is an under-recognized clinical entity often misdiagnosed as osteomyelitis, tumor, or spinal stenosis. It can present as localizing back pain with associated radiculopathy. Studies suggest that the prevalence of axial gout may approach 34%. Diagnosis can be challenging and is often made after biopsy or during surgery. DECT can be a useful tool to diagnose spinal gout with a high sensitivity and specificity, although it might not detect MSU crystals during flares. While there are no current guidelines regarding management of spinal gout, case reports suggest that medical management is equally efficacious when compared to surgical interventions.

In summary, clinicians should maintain a high degree of suspicion for axial gout in patients presenting with back pain, especially if they have concurrent arthralgias in the setting of either metabolic syndrome or chronic kidney disease.

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 Dr. Carla Borre  
 Elizabeth Farkouh  
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*Turning Red: A Case of Vancomycin Induced DRESS after Total Knee Replacement*

Case presentation: A 58-year-old man was admitted for continued intermittent fevers and a progressive rash after starting a 6 week course of vancomycin for a recent left total knee arthroplasty complicated by MRSA bacteremia. He developed intermittent fevers, chills, flushing, and a red, itchy rash that began on his upper torso and back and spread down to his legs. He tried treating this with diphenhydramine, loratadine and topical emollients with no benefit. He went to the emergency department for further evaluation. In the emergency department, he was febrile (38.6°C) and tachycardic. His knee pain was improving even with weight bearing, and his knee erythema and swelling were decreased from previously. Left knee arthrocentesis demonstrated a total neutrophil count of 1144 with 52% neutrophils. He was admitted to hospital medicine for further management. Initial labs revealed peripheral eosinophilia (0.54 x 10<sup>9</sup>/L ) and an increase in his creatinine (1.43 mg/dL with baseline of 1.08 mg/dL). His white blood count and C-reactive protein had decreased significantly since his surgery. Despite switching vancomycin to daptomycin, his fevers persisted, and his diffuse erythematous rash extended to his limbs. His labs showed increasing eosinophilia (0.71 x 10<sup>9</sup>/L), creatinine (1.54 mg/dL) and liver enzymes (AST 72 U/L, ALT 97 U/L).

We suspected the diagnosis of drug reaction with eosinophilia and systemic symptoms (DRESS) secondary to vancomycin. We used the RegiSCAR score to determine the probability of DRESS. RegiSCAR considers symptoms, physical exam, labs, skin biopsy and duration to determine the likelihood of DRESS. His RegiSCAR score was 5, indicating a probable case. His skin biopsy

	<p>pathology was consistent with DRESS. The patient started on prednisone 80 mg daily. He was given calcium and vitamin D supplementation, a proton-pump inhibitor, and Pneumocystis prophylaxis. His liver and kidney function improved, and he was discharged on an 8-week steroid taper with dermatology follow-up.</p> <p>Discussion: DRESS is a severe systemic drug reaction that is responsible for 10-20% of all cutaneous adverse drug reactions in the hospital. It is triggered by allopurinol, antiepileptics (phenytoin), sulfa drugs (Trimethoprim-Sulfamethoxazole), vancomycin and certain anti-tuberculosis antibiotics. Classic skin findings include an erythematous maculopapular rash presenting 2-8 weeks after inciting medication. Systemic symptoms include fevers, lymphadenopathy, eosinophilia/atypical lymphocytes, and organ involvement including the liver, kidneys, lungs or heart. DRESS can be differentiated from other maculopapular drug rashes (i.e. Steven-Johnson Syndrome) through both time course and systemic involvement. DRESS tends to present 2-8 weeks after inciting agent while other drug rashes present within a week of taking the offending agent. Additionally, DRESS is characterized by systemic involvement, not seen in other drug rashes. Eosinophilia is classic and absent in other drug rashes. DRESS is a clinical diagnosis, supported by the RegiSCAR scoring system and skin biopsy. Treatment includes discontinuation of the offending medication and initiating high-dose steroids for at least 8 weeks. Criteria for initiating steroids include severe or persistent systemic involvement, severe cutaneous manifestations and persistent hematologic abnormalities.</p>
<p><b>Grant Goss</b></p>	<p><i>Coronary Vasospasm as a Cause of Sudden Cardiac Death</i></p> <p>Introduction: POEMS syndrome is a known rare disorder named as an acronym for its presentation: polyneuropathy, organomegaly, endocrinopathy, monoclonal gammopathy, and skin changes.</p> <p>Case Presentation: Here we present a case of a patient with known POEMS syndrome who presented to the hospital with acute liver failure secondary to anaplastic myeloma infiltration diagnosed by core biopsy. Presentation: A 66 year old woman with a history of POEMS syndrome (severe pulm HTN 2020. Lambda FLC 2.7g, splenomegaly on CT, VEGF 369 in 2022), and followed closely by Hematology/Oncology previously on lenalidomide, CKD3, chronic HFpEF, who was admitted for rapidly progressing liver injury and non oliguric acute kidney injury. She progressed to acute liver failure with hepatic encephalopathy and coagulopathy while liver core biopsy was pending from a prior admission for similar issues. Her liver biopsy demonstrated infiltration of sinusoids with anaplastic myeloma and she was treated with steroids, cyclophosphamide, and bortezomib. However, she did not improve and ultimately transitioned to comfort cares.</p> <p>Conclusion: This case of liver failure due to anaplastic myeloma infiltration in the setting of known POEMS syndrome is not previously described in literature and may represent a dramatic and difficult to treat complication of POEMS syndrome.</p>
<p><b>Samarth Goyal</b> Dr. Timothy Davie</p>	<p><i>Gastric Bloom: Flower-Like Lesions of Langerhans Cell Histiocytosis in an Adult Stomach</i></p>

<p>Dr. Adina Cioc Dr. Daniela Guerrero Vinsard</p>	<p>Introduction: Langerhans cell histiocytosis (LHC) is a proliferative disorder characterised by abnormal accumulation of antigen presenting cells known as Langerhans cell. Though they are vital immune system components, the proliferation of these cells can lead to a wide variety of symptoms ranging from very benign forms to disseminated, aggressive diseases. The BRAF V600E mutation is present in more than half of the cases, and activation of mitogen-activated protein kinase(MAPK) pathway is a key driver of this neoplastic disorder. LHC most commonly affects the skin and bones but can also involve the pituitary gland/central nervous system, liver, spleen, lungs and very rarely GI tract.</p> <p>Case Presentation: Our case is of a 64-year old man with history of melanoma who presented to the primary care clinic for annual follow-up with no major complaints and a benign physical exam. A low dose CT chest was ordered as a part of routine lung cancer screening which showed normal lung findings but mild diffuse oesophageal thickening. He had no GI symptoms except occasional heartburn. Upper endoscopy was done which revealed a few dispersed flower-like diminutive lesions in the gastric body and fundus. Biopsies were consistent with LHC with BRAF V600E+. The CD1a and S100 are the most characteristic combination of immunohistochemical stains for Langerhans cells which were also positive. The patient was referred to oncology for further management. A PET/CT was done to analyse the extent of disease which showed the tumour burden being restricted only to the stomach. Considering asymptomatic presentation and limited anatomical location, it was decided to follow the patient every 6 months with PET/CT for disease progression with no active intervention.</p> <p>Discussion: LHC is an extremely rare disease of childhood with an incidence of 2-5/million children per year which is even more rare in adults. Of all adults LHC cases ( approx 0.0001%-0.00015% per year) the percentage of cases seen in subgroups are 1) single-site (unifocal), disease(15-20%); 2) single-system multifocal disease (&gt;1 disease site) (5%); 3) single-system pulmonary LCH (40-50%); and 4) multisystem disease (35-50%). Out of these subgroups, isolated GI involvement is very rare with only 17 cases reported in literature since 1983. As most reviews on LHC have been in the pediatric age group it has been found that of cases that were diagnosed via endoscopy, the most common involvement was duodenal followed by rectum, sigmoid, and colon. In the limited literature that is reported on adult LHC, the mean age of presentation is found to be 58y/o with 75% of cases reported in women out of which half were asymptomatic. Most of these cases were reported with a solitary, small intramucosal colorectal polyp.</p>
<p><b>Adam Greer</b> Dr. Amy Holbrook</p>	<p><i>Tough to Swallow: Primary Achalasia and Untreated Helicobacter Pylori Infection</i></p> <p>Introduction: Helicobacter pylori (H. pylori) is the most common chronic bacterial infection in humans affecting up to 50% of the global population. Testing is widely available however treatment is complex and burdensome due to high innate anti-infective resistance. Untreated infection has several well established complications including peptic ulcer disease and gastric malignancy. While H. pylori is not thought to directly affect the</p>

gastroesophageal sphincter a 2023 case-control series found a significant relationship between H. pylori infection and achalasia suggesting indirect autoimmune processes could contribute to development of primary achalasia.

Case Presentation: An 18-year-old woman who had immigrated from Kenya 4 years earlier presented to her PCP for evaluation of headaches and dyspepsia. On exam she was noted to have a BMI of 16 and had a distended abdomen. Her H. Pylori stool antigen test returned positive and she was treated with clarithromycin triple therapy but was not tested for cure. The patient was seen sporadically over the subsequent 2 years for ongoing dyspepsia, chronic cough, and headaches. She had a brief hospital admission after presenting with these symptoms and new dysphagia. EGD done during this admission was unremarkable and biopsies for celiac disease were negative. The patient did follow up with gastroenterology but did not tolerate attempted esophageal manometry. She continued to have intermittent primary care follow-up for chronic abdominal pain, nausea, and headaches. She underwent urea breath testing about 2 years after hospitalization which was negative. She was treated again empirically for H. pylori with clarithromycin triple therapy without test for cure. She was then essentially lost to primary care follow-up for the next 7 years during which time she had 4 children. She presented to the emergency department about 1 month after her most recent delivery for nausea, vomiting, and fever and was admitted for severe hypernatremia, acute renal failure, and CT chest with suspected achalasia and signs of acute on chronic aspiration. She underwent EGD with findings consistent with achalasia and esophageal candidiasis. Repeat EGD with balloon manometry confirmed achalasia and she received botulinum toxin injection. Despite the toxin injection the patient was unable to advance her diet and ultimately required Heller myotomy of the gastroesophageal junction. Before she was discharged EGD biopsy results confirmed H. pylori infection. She was started on bismuth quadruple therapy and discharged. Since discharge she was seen for outpatient surgical follow-up but has not established with a primary care provider and no test for cure is available.

Conclusion: H. pylori is the most common chronic bacterial infection in humans. All patients with confirmed H. pylori should be treated due to known sequela associated with chronic infection. This case emphasizes the importance of close follow-up when treating H. pylori infection and the need for test for cure. It also demonstrates the relatively high rate of clarithromycin-based therapy failure and need for appropriate salvage therapy. Finally, there is some data to suggest an indirect relationship between H. pylori infection and primary achalasia of which this case may be an example.

Discussion: Fungal endocarditis carries mortality rates approaching 50% and as seen in our patient, history of open heart surgery and residence in an endemic area are important risk factors. Our case offers an example of how Histoplasma endocarditis can present, and the importance of considering an echocardiogram in every patient with disseminated histoplasmosis particularly with prosthetic valves and organomegaly, irrespective of reassuring cardiac exam and initial blood culture results, as this can be key

	<p>in determining prognosis and governs the duration and type of treatment in such cases.</p>
<p><b>Grace Hagan</b>  Dr. Michael Cullen  Dr. Frank Brozovich</p>	<p><i>Tying the Clot: A Case of Recurrent Mitral Valve Annuloplasty Thrombus</i></p> <p>Background: Annuloplasty thrombus is a rare complication following mitral valve (MV) repair. It may present with MV obstruction or embolic phenomena. Standard treatment is 3 months of warfarin therapy. Previous cases demonstrate resolution with anticoagulation ± repeat MV repair. Here we present a case of recurrent annuloplasty thrombus despite adequate anticoagulation, exclusion of infection, and repeat repair.</p> <p>Case Presentation: A healthy 61-year-old male was diagnosed with severe MV regurgitation secondary to a flail scallop of the posterior leaflet. After successful MV repair with annuloplasty ring, he initiated 6 weeks of apixaban. Two months later, he developed diplopia. Head computed tomography (CT) identified embolic cerebral infarcts. Transesophageal echocardiogram (TEE) revealed a 1 cm echodensity on the MV annuloplasty. Infectious evaluation was negative. INR goal increased to 2-3. After 1 month, TEE revealed lesion enlargement. Investigations for infectious, inflammatory, malignant, allergic and pro-thrombotic etiologies, including cell-free DNA testing and cancer screening, were negative. He underwent aspiration thrombectomy. Pathology revealed thrombus. INR goal increased to 3-4.</p> <p>TEE 1 month later revealed new thrombus. Annuloplasty band was removed and MV re-repaired. Tissue cultures were negative. Repeat TEE after 1 month showed recurrent thrombus despite the MV re-repair. He was transitioned to enoxaparin and clopidogrel. Just 1 week later, he developed dysarthria. CT showed new infarct with MV thrombus on TEE. He continued enoxaparin targeting suprathreshold anti-Xa levels. TEE 1 month later revealed mild improvement in thrombus size. Decision-making: Thorough evaluation for source of recurrent thrombus formation including infection, malignancy and hypercoagulable states was unrevealing. The predisposition to thrombus formation remains unclear. Decision was made for surveillance TEE at 6 months from previous, unless symptomatic.</p> <p>Conclusion: This unusual case of recurrent MV annuloplasty thrombus despite adequate anticoagulation, exclusion of infection and other secondary causes, and MV re-repair demonstrates a rare complication of surgical MV repair.</p>
<p><b>Rachel Hansen</b></p>	<p><i>Climbing to the Top: A Rare Case of Culture Negative Meningitis from Asymptomatic Ventriculoperitoneal Shunt Bowel Perforation</i></p> <p>Introduction: Meningitis is a life-threatening condition with a high mortality rate if not diagnosed promptly. Furthermore, meningitis symptoms can often be masked by other generalized complaints including nausea, vomiting, malaise, and fatigue that often mimic more common pathologies like viral infections leading to a delay in diagnosis. Here, we present a case of spontaneous ventriculoperitoneal shunt migration causing asymptomatic bowel perforation leading to meningitis.</p>

	<p>Case Description: A 64-year-old man with a past medical history of normal pressure hydrocephalus with ventriculoperitoneal shunt placement in 2017 presented with a fever and a severe headache. A lumbar puncture was performed to evaluate for meningitis. While the initial results of the cerebrospinal fluid were concerning for meningitis given the elevated white blood cell count with neutrophilic predominance and elevated protein levels, the BioFire panel for bacterial pathogens and additional cultures (bacterial, viral, fungal) remained negative. As the patient did not have any other localizing symptoms of infection, further evaluation of the ventriculoperitoneal shunt was pursued per infectious disease recommendations. A CT scan of the abdomen and pelvis was performed and showed perforation of the hepatic flexure of the colon. The ventriculoperitoneal shunt remained fixed in the intraluminal space, serving as a reservoir for gastrointestinal pathogens to migrate to the cerebrum. A multidisciplinary discussion was held to discuss removal of the shunt. The neurosurgery team removed the cephalic portion of the shunt to just below the clavicle, and subsequently the gastroenterology team performed a colonoscopy and removed the remaining 40 cm of abdominal shunt material using a forceps. The colonic defect was then closed using a combination of argon plasma coagulation and over the scope clip placement. The patient then completed a 2-week course of antibiotics from the removal date of the ventriculoperitoneal shunt. He is currently doing well on 12 weeks of follow up without evidence of residual neurological defects or infections since removal of the shunt.</p> <p>Discussion: Our case highlights the importance of having a high index of suspicion for underlying infection, including meningitis, in patients with a ventriculoperitoneal shunt who present with vague symptoms. Additionally, these patients will often have culture negative CSF results from a lumbar puncture, prompting further investigation. This patient had a unique etiology of meningitis resulting from ventriculoperitoneal shunt migration into the colon necessitating a combined approach for neurosurgical and endoscopic intervention for ventriculoperitoneal shunt removal and in turn improvement in patient's meningitis.</p>
<p><b>Cambria Heuston</b>  Dr. Josh Trujeque  Dr. Nicole Zantek  Dr. Joan Beckman  Dr. Gregory Vercellotti</p> <p>Finalist</p>	<p><i>Bloodletting Reimagined: Manual Blood Exchange for Protoporphyrin Crisis in the Intensive Care Unit</i></p> <p>Introduction: X-linked protoporphyria (XLP) is a rare metabolic disorder characterized by a disruption in the heme synthesis pathway, leading to the accumulation of heme precursors. Therapeutic plasma exchange (TPE) and red blood cell (RBC) exchange are potential interventions for managing a protoporphyric crisis. However, the optimal schedule and combination of TPE and RBC exchange remain unclear. This report details the management of a protoporphyric crisis in an elderly female with XLP, manifesting as severe lactic acidosis in the intensive care unit (ICU).</p> <p>Case Description: A 65-year-old woman with XLP, initially diagnosed after developing a blistering rash on her hands following sun exposure and a history of stage IIIB breast cancer, presented to care with acute abdominal pain. She was found to have acute liver injury, severe lactic acidosis, and diffuse bilateral ground glass opacities. She was admitted for an acute porphyria flare and started on heme infusions. Three days later, she</p>

	<p>developed acute hypoxic respiratory failure associated with worsening lactic acidosis peaking at 25 mmol/L, which required ICU transfer and intubation. A bronchoalveolar lavage was performed and findings were suggestive of diffuse alveolar hemorrhage (DAH). Despite intensive supportive measures and treatment with hemin, vitamin E, and ursodiol, her condition deteriorated, prompting the use of TPE and RBC exchange as salvage therapies. Manual RBC exchange was performed twice at the bedside, leading to near-immediate resolution of her metabolic derangements. She then underwent alternating TPE and RBC exchanges, with the first four performed daily, resulting in an improvement in her protoporphyrin levels. She was discharged home and continues on hemin, hydroxyurea, and TPE/RBC exchange maintenance therapy.</p> <p>Discussion: Acute porphyria flares can have life-threatening implications; it is critical that they are recognized so appropriate treatment can be initiated. XLP is caused by an X-linked ALAS2 mutation, leading to ALAS2 synthase overactivity and excessive protoporphyrin production. This case is unusual due to the patient’s age of onset, suggesting a possible acquired etiology, and the severity of her crises. Complications include phototoxic reactions, gallstones, cholestasis, and liver failure. Treatment for acute porphyria flares in XLP includes ursodiol and cholestyramine to increase protoporphyrin excretion, hydroxyurea to decrease erythrocyte production, hemin to suppress ALA synthase, and TPE/RBC exchange to remove excess protoporphyrins. Manual whole blood exchange, such as in our case, can provide an alternative effective treatment when hemin or apheresis are not available.</p>
<p><b>Mason Hinke</b></p>	<p><i>Ascariasis Lumbricoides-Induced Ascending Cholangitis: A Case Report of Parasitic Complications</i></p> <p>Introduction: Ascariasis lumbricoides is one of the most common human helminth infections in the world. Transmission occurs through ingestion of either contaminated water or food. Patients can have a variety of symptoms, ranging from either asymptomatic to respiratory symptoms to severe intestinal obstruction.</p> <p>Case Presentation: The patient in this case is a 34-year-old female with a past medical history of a recent admission for choledocholithiasis status post cholecystectomy one month ago, who was admitted with acute onset of abdominal pain. Following her recent procedure, she previously was doing well aside from minor postoperative pain but then woke up on the evening of admission with severe epigastric pain, emesis, fevers and chills. Upon arrival to the ED, she was hypotensive into the 90s/30s, heart rate was in the 90s and she was febrile to 38.8 celsius. Her initial labs were notable for a white blood cell count of 12.5, hemoglobin of 10.6 and an absolute eosinophil count of 1.9. Her liver function tests were abnormal with an ALT of 331, AST 198 and alkaline phosphatase of 223. Social history was acquired and she recently traveled from Ecuador to the US via the Darien gap and had been consuming water from deep in the amazon jungle. CT of the abdomen was ordered which revealed extensive intrahepatic biliary dilatation as well as a centrally hypoattenuating collection which was suspicious for an intrahepatic abscess. Her clinical picture was overall concerning for ascending cholangitis so she was started on IV Zosyn, given IVF and</p>

	<p>admitted for further evaluation.</p> <p>GI was consulted the following morning and she underwent an MRCP. During the procedure, the major papilla orifice was found to be bulging due to sludge and foreign bodies were seen that were suspicious for parasites. This was removed and immediately brought to the lab which identified the parasites as <i>Ascariasis Lumbricoides</i>. She was given one dose of Ivermectin in addition to her antibiotics and she significantly improved within twenty-four hours. She was ultimately discharged on levofloxacin 750 mg daily x 4+ weeks with follow up with infectious disease for close monitoring of her hepatic abscess.</p> <p>Conclusion: This case highlights a severe case of ascariasis. A majority of infections occur in Asia, Africa and South America but should be considered in patients with recent travel history. Symptoms depend on where in the life cycle it is. Since ascariasis migrates initially through the lungs, early presentations will include respiratory symptoms such as eosinophilic pneumonitis and pneumonia. Later presentations include intestinal obstruction, hepatobiliary involvement and pancreatic involvement. Treatment includes a onetime dose of albendazole or ivermectin as an alternative agent.</p>
<p><b>Hannah Irwin</b></p>	<p><i>A Devastating Outcome of Provoked Atrial Fibrillation</i></p> <p>Introduction: Patients with atrial fibrillation who meet criteria for long term anticoagulation can pursue left atrial appendage occlusion if anticoagulation is otherwise contraindicated. This can be done by percutaneous placement of a WATCHMAN device, obstructing the left atrial appendage and significantly reducing future chances of stroke or systemic embolism caused by atrial fibrillation. While patients do require short term anticoagulation for the procedure, they are able to avoid long-term anticoagulation.</p> <p>Case Presentation: The patient is a 66-year-old man who had one recorded episode of atrial fibrillation while admitted to the hospital with an acute gastrointestinal bleed due to arteriovenous malformation. Due to his CHA2DS2-VASc score of 3 (4.6% annual risk of stroke), the patient was encouraged to initiate apixaban, which he agreed to. He was also started on sotalol. No outpatient extended cardiac monitoring was performed. Notably, the patient was on apixaban for over a month without adverse effects. Later, he was referred for discussion of implantation of a WATCHMAN device to avoid long-term anticoagulation. He ultimately elected to pursue the left atrial appendage closure and device placement.</p> <p>During the procedure, the patient became pale and unresponsive. He was noted to have ST elevation on telemetry, was intubated for airway protection, and the procedure was converted to an emergent angiogram. The patient was found to have an air embolism of the right coronary artery and was transferred to our institution for hyperbaric oxygen treatment. Subsequent transthoracic echocardiogram showed no wall motion abnormality or signs of ischemia. However, while in the ICU, the patient was found to have left sided deficits. Brain imaging showed multifocal right MCA territory infarcts, related to the air embolism. Patient was eventually extubated and had persistent dense left hemiparesis. He was also found to</p>



	<p>have urinary retention, with neurogenic bladder, requiring chronic indwelling foley catheter.</p> <p>Ultimately, the left atrial appendage was too small to place a Watchman device, and the patient was discharged from the hospital to a transitional care facility, still requiring apixaban. Fortunately, the patient has not had subsequent bleeding complications. On follow-up with Cardiology, sotalol was discontinued, as the patient has had only one known atrial fibrillation event. He has been prescribed an outpatient, 14-day cardiac monitor, the results of which are pending.</p> <p>Discussion: While the WATCHMAN device has been found to be noninferior to anticoagulation for long term outcomes in patients with atrial fibrillation and indications for long term anticoagulation, this case details one unfortunate potential outcome in a patient undergoing placement of a WATCHMAN device. This case highlights how every procedure carries with it some risk. When pursuing novel therapies, it is important to continue to engage in a thorough investigation of symptoms and disease patterns before initiating invasive modalities. This case of provoked atrial fibrillation would have certainly benefited from further work-up and more time to determine burden of disease before pursuing a seemingly low-risk procedure that had devastating outcomes for the patient.</p>
<p><b>Guneet Janda</b></p>	<p><i>A Challenging Case of Multifactorial Anemia</i></p> <p>Case Presentation: A 69-year-old female with a history of type 2 diabetes mellitus and chronic iron deficiency anemia (IDA) presented with acute on chronic profound fatigue and weakness. Duration of her symptoms was 10 years and worsened to the point she was forced to leave her nursing job. Two weeks prior to her presentation, her fatigue had progressed to the point where she was largely bedridden.</p> <p>She had IDA dating back to her teenage years. Her hemoglobin (Hb) had dropped to as low as 5 mg/dL while pregnant. She had intermittent anemia over the past few decades for which she received intramuscular injections of iron. Also reported a history of heavy menstrual bleeding but was post-menopausal a decade prior to her current presentation. She was G3P2, with one spontaneous miscarriage and two live births. Both of her sons were in their middle-30's without known health issues. Her social history was notable for no drug or alcohol use and adhering to a vegetarian diet. Laboratory current studies were notable for Hb 10.5 g/dL, mean corpuscular volume (MCV) 66.4 fL, RBC 5.07 million, RDW 27, and ferritin 10 ng/mL. Blood smear demonstrated hypochromic-microcytic erythrocytes, suggestive of iron deficiency, thalassemia, or hemoglobinopathy. Esophagogastroduodenoscopy and colonoscopy were both normal. Small bowel biopsies negative for malabsorptive syndrome. The microcytic, hypochromic anemia with a normal RBC count suggested thalassemia, however, Hb electrophoresis and alpha-globin gene analysis were both normal. Lead, mercury, arsenic, cadmium, and zinc levels all normal. Copper level mildly elevated, consistent with over-supplementation.</p> <p>Given her persistent anemia while on iron supplementation, profound fatigue, and vegetarian diet, additional testing revealed a serum vitamin B12</p>

	<p>level of 231 ng/dL (reference range, RR: 200 – 650 ng/dL), homocysteine of 14 umol/L (RR: &lt;13 umol/L) and methylmalonic acid (MMA) of 1.09 umol/L (RR: &lt;0.4 umol/L). A diagnosis of B12 deficiency anemia with superimposed iron deficiency anemia was made. Within a few weeks of beginning parenteral B12 and continued oral iron supplementation the patient’s energy levels improved and her hemoglobin levels normalized.</p> <p>Take home messages: 1) Most cases of anemia are multifactorial, thus work up can be challenging. 2) Careful review of CBC components (MCV, RDW and RBC count) and peripheral blood smear point to the etiology and aid in the diagnostic work up in the majority of cases, but are not absolute. 3) Taking into account patient dietary habits and co-morbidities is important in the guiding diagnostic work up. 4) B12 deficiency as a cause of anemia can be a particular diagnostic challenge: serum B12 may be normal or low normal as in this case. 5) For a high clinical suspicion, testing for MMA provides high specificity of B12 deficiency.</p>
<p><b>Emma Johns</b> Dr. Nadia Akhiyat Dr. Amir Lerman</p>	<p><i>Double Trouble: Diagnostic Challenge in Sudden Circulatory Arrest</i></p> <p>Case Presentation: A 62-year-old man developed acute unprovoked substernal chest pressure, falling unconscious en route to the hospital. His past medical history included atrial fibrillation without anticoagulation, ascending aortic aneurysm, hyperlipidemia, bipolar disorder and previous gastric bypass surgery. His wife called 911, and EMS arrived in two minutes with initiation of CPR. Rhythm analysis showed ventricular fibrillation. Spontaneous circulation was reestablished after seven episodes of defibrillation and 100 mg dose of lidocaine.</p> <p>On ED arrival, his Glasgow Coma Scale was 3 with vitals including blood pressure 121/83 mmHg, respiratory rate 13, pulse rate 65, O2 96% through iGel airway device. Physical exam was significant for 2+ femoral pulses, irregular heart rhythm, clear bilateral breath sounds, and no abdominal tenderness. Pupils were equal and reactive. ECG showed ventricular rate 67 bpm, QTC interval 521 mmsec with significant changes including atrial fibrillation, PVCs, and right bundle branch block with secondary ST-T segment abnormalities. Emergent transesophageal echocardiogram performed in resuscitation bay showed no aortic dissection but active clot passage from right atrium, through right ventricle, and into pulmonary artery. Point of care labs included Troponin 23, Lactate 5.18, and Venous pH 7.25. His blood pressure decreased to 80/68 mmHg with 48 bpm and remained hypotensive after 2 liters of IV fluids. Repeat ECG showed evolving ST elevations in leads II, III, and aVF. He was intubated and sedated. Medical response teams including cardiology, intensive care, and emergency medicine discussed the decision of systemic thrombolytics for massive PE or percutaneous coronary intervention for right ventricle STEMI. Subsequently, he was loaded with aspirin, clopidogrel and heparin, and transferred to catheterization lab. Angiogram found a distal RCA thrombosis, causing right ventricle STEMI. He underwent successful RCA thrombectomy and drug-eluting stent placement to the distal RCA. After the procedure, blood pressures remained low at 85/57 mmHg. Post angiogram CT triple rule out was negative for pulmonary embolus, despite visualization of thrombus in transit. Extremity ultrasounds were negative for DVT. Repeat TEE showed no remaining intracardiac thrombus but moderate-severely enlarged right</p>

	<p>ventricular size with moderate-severely reduced systolic function. Upon extubation, he sustained mild short-term memory loss and continued mild hypotension. He was discharged home on clopidogrel for 12 months and apixaban indefinitely.</p> <p>Discussion: Differentiating between right ventricular MI and massive PE is a challenging clinical scenario. This case illustrates the importance of rhythm pathophysiology in circulatory arrest as a guide towards etiology and management. Pulseless electrical activity is the presenting rhythm in 65% of PE-induced arrests, whereas ventricular fibrillation is strongly linked to acute coronary syndrome and ischemia. ECG can show right bundle branch block and ST-T segment changes in the early stages of both conditions. Echocardiogram can show right heart systolic dysfunction and interventricular septum motion defects in both conditions. Intracardiac thrombus can be seen in both conditions due to stasis of the right ventricle. If concerned for STEMI, PCI should be obtained as quickly as possible. Factors that directly influence out-of-hospital arrest survival include early high-quality cardiopulmonary resuscitation, the initial rhythm of cardiac arrest, and early defibrillation.</p>
<p><b>Whitney Johnson</b>  Dr. Quan Phung  Dr. Meghan Lindstrom  Dr. Heather Beckwith</p>	<p><i>Intraosseous Xanthoma and Pulmonary Anthracosis Mimicking Metastasis in a Patient with Breast Cancer</i></p> <p>Introduction: Patients with cancer frequently undergo imaging studies for diagnosis, staging, and surveillance. Incidental findings can be difficult for clinicians to interpret and often lead to further imaging and other evaluations. We present a patient with breast cancer who presented with multifocal bone pain in whom imaging to rule out bone metastases showed lymphadenopathy and a lytic rib lesion. Biopsy of separate lesions showed intraosseous xanthoma and anthracosis, respectively.</p> <p>Case Description: A 74-year-old female with a past medical history of hyperlipidemia, osteoporosis, and grade 3, ER/PR positive, HER2 negative, T1bN0M0 invasive ductal carcinoma of the right breast presented to her oncologist with new, diffuse bony pain. She was diagnosed with breast cancer on a screening mammogram performed eleven months prior. She underwent a right breast lumpectomy and right axillary sentinel lymph node biopsy with negative surgical margins. Adjuvant chemotherapy was recommended; however, the patient declined and was started on tamoxifen. Her family history was unremarkable, but she did have a CHEK2 mutation. A nuclear medicine bone scan demonstrated an area of increased uptake in the left fifth rib and multiple solid pulmonary nodules measuring up to one centimeter. Further evaluation with a PET scan noted, "findings of metastatic disease with hypermetabolic mediastinal, hilar, and bilateral cervical level 2A lymph nodes." Serum protein electrophoresis (SPEP), light chains, and immunoglobulin levels were normal. Biopsies of multiple mediastinal lymph nodes demonstrated "lymphohistiocytic aggregates with anthracotic pigment" and a rib lesion biopsy revealed an intraosseous xanthoma.</p> <p>The patient reported a personal history of hyperlipidemia, but a cholesterol panel was normal. She did not smoke, lived in a large city for a short period, and traveled frequently to the Middle East, but without contact with</p>

	<p>minerals or mining. A follow-up CT scan 6 months later demonstrated stable mediastinal lymph nodes and rib lesion with no new findings.</p> <p>Discussion: In this patient, despite initial imaging findings that were highly suggestive of metastatic malignancy, on further workup, this patient was only found to have two benign but uncommon diagnoses. Xanthomas are deposits of lipid-laden macrophages usually found in soft tissues. Intraosseous xanthomas are infrequent, especially in the ribs, but when present are lytic lesions that can interrupt the cortex and mimic osseous malignancy or metastasis. Importantly, all patients with xanthomas should have their lipid levels checked. Our patient's lipid levels were surprisingly normal despite not being on any lipid-lowering therapy. In addition, hypermetabolic lymph nodes were biopsied, consistent with anthracosis. Anthracosis is carbon buildup in the airways that can cause obstructive airway or fibrotic lung disease, but in many patients is asymptomatic. Risk factors include coal dust, biomass smoke, or significant air pollution exposure. Interestingly, our patient did not have any of these risk factors.</p> <p>While the diagnoses in this patient are rare, "incidentalomas" are common, especially for patients with malignancies given frequent imaging. This patient's case details the challenges of incidental findings on imaging and the importance of further workup in patients with a history of malignancy.</p>
<p><b>Vijayvardhan Kamalumpundi</b> Dr. Cadman Leggett</p>	<p><i>Unmasking Secondary Achalasia: A Critical Diagnosis Not to Miss</i></p> <p>Introduction: Secondary achalasia is a rare condition that mimics primary achalasia and can pose a significant diagnostic challenge due to its similar clinical presentation. We present a patient with dysphagia initially diagnosed with achalasia, later revealed to be secondary to an underlying malignancy, highlighting the importance of considering secondary achalasia in the differential diagnosis.</p> <p>Case Presentation: A 71-year-old male with a history of gastroesophageal reflux disease presented to the emergency department with a 3-month history of unintentional 30-pound weight loss over two months, regurgitation of food, progressive dysphagia, and early satiety. Computed tomography of the chest revealed a diffusely distended esophagus with abrupt tapering at the gastroesophageal junction, suggestive of achalasia. Esophagogastroduodenoscopy (EGD) with biopsy demonstrated ulceration and severe stenosis of the gastroesophageal junction, with pathology confirming moderately differentiated gastric adenocarcinoma. Due to severe dysphagia, a jejunostomy was placed for nutrition. Endoscopic ultrasound (EUS) identified a hypoechoic, partially circumferential mass at the gastroesophageal junction, and malignant-appearing paraoesophageal lymph nodes. The patient underwent chemoradiation, followed by Ivor-Lewis esophagectomy. Frozen biopsies demonstrated moderately differentiated muscle-invasive adenocarcinoma. Unfortunately, the patient developed a small bowel volvulus around the jejunostomy site and died shortly after surgery.</p> <p>Conclusion: Secondary achalasia, a condition that mimics primary achalasia, is usually caused by malignant tumors of the gastroesophageal junction. In fact, malignant neoplasms comprise 4% of achalasia-like syndromes.</p>

	<p>Differentiating secondary achalasia from idiopathic/primary achalasia poses a significant diagnostic challenge. Standard treatments for achalasia are ineffective—and potentially harmful—in cases of secondary achalasia, delaying the diagnosis of underlying malignancies. EGD with biopsy is recommended in all patients with suspected achalasia to exclude malignancy. A short symptom duration (&lt;5 months), and difficulty passing the endoscope through the LES should raise suspicion for secondary achalasia. Additional evaluations, including abdominal CT for regional lymph nodes and EUS for submucosal and infiltrative lesions, are valuable in informing the diagnosis.</p>
<p><b>Muneeb Khan</b></p>	<p><i>Alveolar Hemorrhage from Severe Mitral Regurgitation, Uncommon Complication of a Common Abnormality</i></p> <p>Introduction: Presenting a case of alveolar hemorrhage in a patient with a history of system lupus erythematosus (SLE) and mitral valve disease.</p> <p>Case Description: A 68-year-old male with a history of SLE with lupus nephritis on chronic immunosuppression, CML on dasatinib, and chronic mitral regurgitation was admitted with acute hypoxic respiratory failure requiring intubation and severe hypertension. Initial lab work showed pancytopenia; notably a hemoglobin of 7.4, and a platelet count of 22. Bronchoscopy was performed for full infectious workup, which did not show infection but revealed alveolar hemorrhage diffusely without evidence of serially bloody aliquots. The initial assumption for etiology was lupus pneumonitis vs antiphospholipid antibody syndrome, however, disease markers were stable. A large focus was still held on an immunologic cause, and treatment continued with pulse dose steroids. However, patient had persistent respiratory failure and intermittent need for blood transfusions with ongoing bloody secretions. Thus, further work-up with a right heart catheter (RHC) was pursued.</p> <p>RHC demonstrated pressure in the pulmonary artery at 36/14 (n25/10), RV 35/9 (n25/0-5) PCWP of 17 (n&lt;12). Trans-thoracic echocardiogram showed moderate to severe MR and LVEF of 45%. With these findings, it was concluded that the patient’s acute hypertension led to a dynamic worsening of the patient’s chronic mitral regurgitation, elevating pulmonary venous pressure, causing flash pulmonary edema and for blood to diffuse into the alveolar space. Treatment was directed towards afterload reduction, with a target MAP of 65. The patient was started on a nicardipine drip, and scheduled administration of hydralazine and valsartan. With targeted afterload reduction the patient improved and was extubated to room air. On discharge, cardiology follow-up was scheduled for evaluation of a mitral clip.</p> <p>Discussion: This case highlights the importance of maintaining a broad differential. Placing the right heart catheter to evaluate filling pressures represented a shift in focus from autoimmune pneumonitis. Instead, we were able to uncover an uncommon presentation (alveolar hemorrhage) of something we commonly see (mitral valve disease). It is rare for diffuse alveolar hemorrhage to require multiple transfusions, this type of bleeding is typically milder, and is self-resolving. Transfusions were required in this case due to the patient’s thrombocytopenia, believed to have been caused</p>

	<p>by dasatinib, a known complication of tyrosine kinase inhibitors. The patient’s mitral regurgitation was a long-standing issue and not one that required any intervention. Surprisingly, echo during admission did not show rupture of the chordae tendineae or papillary muscles, meaning the decompensation was not from acute mitral valve failure. Instead, the backflow through the intact valve worsened acutely in the setting of severe hypertension. Because the patient’s chronic mitral regurgitation was mild, the left atrium hadn’t adapted to the sudden increase in volume, thus causing a flash pulmonary edema, respiratory failure, and most shockingly, alveolar hemorrhage.</p>
<p><b>Ameya Kumar</b></p>	<p><i>Plant sterols are a type of dietary fats which are found in vegetable oils, nuts, seeds and legumes. They have been associated with a cholesterol-lowering effect and improved cardiovascular health.</i></p> <p>Introduction: Absorption of plant sterols is mediated through ATP-binding cassette transporter (ABC transporter) proteins, which are coded by ABCG5 and ABCG8. A mutation of the ABCG5 &amp; ABCG8 genes results in disruption of this regulation and in turn leads to excessive, unchecked absorption of plant sterols from the gut.</p> <p>Case Presentation: We present a case of A 25-year old woman presented to the clinic and was incidentally noted to have elevated total and HDL cholesterol levels. She also reports a history of premature coronary disease in her family. Physical exam was not remarkable for any evidence of Xanthomas/corneal arcus or xanthelasma. Given concern for familial dyslipidemia, we sent a genetic panel, which was positive for ABCG8 homozygous mutation with DH19 variant. With the homozygous mutation, a diagnosis of sitosterolemia was made and plant sterols levels were obtained which were also found to be high. She was initiated on Ezetimibe, given dietary education and referred to preventative Cardiology.</p> <p>Discussion: Sitosterolemia is a rare inherited disorder of lipid metabolism characterized by excessive accumulation of plant sterols due to mutations in ABCG5/ABCG8 genes. Fewer than 100 cases have been reported worldwide. Individuals affected by this condition can present with xanthomas, xanthelasmas, corneal arcus and may be predisposed to premature atherosclerotic disease. Plant sterols are a type of dietary fats which are found in vegetable oils, nuts, seeds and legumes. They have been associated with a cholesterol-lowering effect and improved cardiovascular health. Absorption of plant sterols is mediated through ATP-binding cassette transporter (ABC transporter) proteins, which are coded by ABCG5 and ABCG8. A mutation of the ABCG5 &amp; ABCG8 genes results in disruption of this regulation and in turn leads to excessive, unchecked absorption of plant sterols from the gut. Individuals affected by this condition can present with xanthomas, xanthelasmas, corneal arcus and may be predisposed to premature atherosclerotic disease.</p>
<p><b>Caroline Ledet</b> Dr. Matthew Schnorenberg</p>	<p><i>Blood Letting, Blood Clots, and Bewilderment: Unraveling Shock using Right Heart Catheterization</i></p> <p>Introduction: Shock is classified into 4 distinct categories: distributive, cardiogenic, hypovolemic, and obstructive. Treatment is cause-specific. The</p>

etiology of shock can be diagnosed at bedside, especially with the incorporation of point-of-care imaging. In the case of undifferentiated shock, advanced diagnostics may be required to guide diagnosis.

Case Presentation: A 72-year-old male was diagnosed with spinal cord infarct, as well as right main pulmonary artery (PA) embolism by CT angiography. Hypercoagulability due to polycythemia was identified as the provoking risk factor; hematocrit was elevated to 60.6% (normal: 38.3-48.6%). 1 L of blood was removed by therapeutic phlebotomy to target a hematocrit of less than 45%. 1.25 L crystalloid fluid was administered, and a heparin drip was initiated.

One hour following phlebotomy, the patient developed hypotension requiring vasopressor administration. He was tachycardic but non-hypoxic. Lactate was elevated to 2.6 mmol/L. Shock was diagnosed. The differential diagnosis of his shock included obstructive shock due to pulmonary embolism and hypovolemic shock due to phlebotomy-induced blood loss. Bedside transthoracic echocardiogram showed mildly enlarged right ventricle (RV) chamber size, mildly reduced RV function, and normal inferior vena cava (IVC) size and inspiratory collapse. Given concern for obstructive shock due to pulmonary embolism (PE) and contraindication to thrombolysis due to spinal cord infarction, thrombectomy was considered. Emergent right heart catheterization (RHC) was performed with the following results: PA pressure (PAP) of 24/13 mmHg (normal: 15-25/8-15 mmHg), RV pressure of 24/4 mmHg (normal: 15-25/8-15 mmHg), right atrial pressure (RAP) of 4 mmHg (normal: 0-6 mmHg), pulmonary capillary wedge pressure (PCWP) of 11 mmHg (normal: 6-12 mmHg), cardiac output (CO) by Fick method of 2.4 L/min (normal: 4-8 L/min), and cardiac index (CI) of 1.5 L/min/m<sup>2</sup> (normal: 2.5-4 L/min/m<sup>2</sup>). Hypovolemic shock due to inadequate volume resuscitation following phlebotomy was diagnosed given normal PAP, low-normal CI, and normal IVC size and inspiratory collapse. He received an additional 2.5 L of crystalloid fluid, and his mean arterial pressure improved without further vasopressor requirements.

Discussion: Bedside evaluation with point-of-care imaging is an important first step for differentiating the etiology of shock. RHC is a useful diagnostic test when the etiology of shock remains elusive and when invasive interventions, such as thrombectomy in this case, are considered. Therapeutic phlebotomy removes whole blood, and that intravascular volume must be replaced to maintain hemodynamic stability. It was previously thought that 3-4 times the loss blood volume needed to be administered with crystalloid fluid due to only a fraction of the administered crystalloid remaining in the intravascular space. When this theoretical framework was empirically tested, a more optimal ratio appeared to be 1.5:1. A lower ratio may be appropriate in patients at risk for complications of hypervolemia. However, this patient, who had RV pre-load dependence in the setting of PE, experienced hypovolemic shock and benefitted from a higher ratio of crystalloid fluid resuscitation.

**Emma Leirdahl**  
Dr. Anya Jamrozy

*Beyond Myalgias: A Rare Case of Statin-Induced Immune-Mediated Necrotizing Myopathy*

	<p>Introduction: Myalgias are a frequently reported complaint in primary care, and often attributed to statins. While true statin-related myalgias are infrequent overall; in rare cases they can result in severe complications including myonecrosis and rhabdomyolysis that can be life threatening.</p> <p>Case Presentation: A 48-year-old male with a history of type 2 diabetes, hypertension, and previous CVA with residual L sided weakness presented with 10 days of generalized weakness and exertional chest pain. Labs were notable for moderately elevated transaminases and a HS troponin T of 1200. Initially, a cardiac etiology seemed the most likely cause of his symptoms. However, serial HS troponin T measurements were stable, there were no notable EKG changes, and he remained symptom free. Further workup revealed a severely elevated CK to 17,000. Serial dilutions of HS troponin I were negative suggesting the markedly high troponin T represented fetal troponin from skeletal muscle ischemia. Cardiac MRI returned negative for myocarditis, TTE was unremarkable, and ANA returned positive. Muscle biopsy revealed an active severe necrotizing myopathy. IgG 3-hydroxy-3-methylglutaryl-coenzyme A reductase (HMGCR) was positive, confirming the diagnosis of necrotizing autoimmune myopathy (NAM), which has been described in a subset of statin-treated patients. He improved with steroids and was subsequently discharged to inpatient rehab. On follow up with outpatient rheumatology, he completed IVIG therapy and remains on high dose steroids and methotrexate with ongoing improvement. Lifelong statin avoidance was recommended.</p> <p>Conclusion: This case illustrates a potential severe complication of statin therapy, necrotizing autoimmune myopathy. Although rare, this complication cannot be missed and requires urgent rheumatologic evaluation and lifelong avoidance of statins.</p>
<p><b>Leeore Levinstein</b>  Catherine Halley  Dr. Kanchan Hulasare  Dr. Elizabeth Schroer    Finalist</p>	<p><i>Atypical Presentation of Leukemia as Post-Vaccination Rash</i></p> <p>Background: Leukemia cutis is a well-documented cutaneous manifestation of both acute and chronic leukemia. It is a relatively rare finding with a frequency of 2-30%, and is most commonly observed in children. The majority of cases occur when leukemia is already diagnosed, and its presence is usually associated with advanced disease.</p> <p>Case Presentation: A previously healthy 74-year-old man developed a painless rash after receiving pneumococcal and influenza vaccines. The following week, he received the COVID-19 and RSV vaccines, then subsequently developed fever and fatigue. This prompted him to take a home COVID-19 test, which was positive. Fevers improved, over a few days, but he had ongoing fatigue. Four weeks after the initial onset of rash, he presented to the ED due to persistent rash and fatigue. In the ED, he had a diffuse rash of flat papules on the anterior and posterior torso and scattered on the arms and legs. The rash spared the face, hands, feet, and mucosal membranes. He was vitally normal. Complete blood count showed white blood cell count of 101.1, hemoglobin of 10.7, and platelet count of 38,000. He was admitted to the hospital. Peripheral blood flow cytometry and bone marrow biopsy confirmed diagnosis of acute myeloid leukemia with monocytic features (NPM1, KRAS, FLT3 TKD, PTPN11). Skin punch biopsy from a torso lesion showed diffuse dermal and subcutaneous involvement</p>



	<p>by atypical lymphoid infiltrate with myeloid features, consistent with leukemia cutis. He was started on appropriate therapy.</p> <p>Discussion: Vaccinations are known to cause a variety of cutaneous reactions. These reactions are generally benign, have complete spontaneous resolution, and require no additional management. However, new onset dermatologic disease can be mistaken for an adverse vaccine reaction, causing a delay in diagnosis or even misdiagnosis. In this case, a rash manifesting shortly after vaccination was found to be leukemia cutis. This case highlights that with new onset rash maintaining a broad differential diagnosis is critical, especially when the rash is unresolving or worsening. In this setting, pursuing further evaluation such as skin biopsy can be a crucial diagnostic step in determining the etiology of a rash. Consideration of cutaneous diseases, such as leukemia cutis, is important for timely diagnosis and treatment of new onset rash.</p>
<p><b>Miranda Lin</b> Dr. Thanh Ho</p>	<p><i>A Young Patient with Blood Loss Anemia</i></p> <p>Introduction: Anemia is a common symptom of a broad list of etiologies. Here, we present a rare cause of acute blood loss anemia in a young adult patient.</p> <p>Case Presentation: A 45-year-old healthy male presented to his primary care physician with a three-week history of fatigue, headaches, diffuse myalgias, pallor, and exercise intolerance. He recently started taking Ibuprofen for pain and had two episodes of melena. Physical exam was remarkable for pallor. Pertinent laboratory studies revealed hemoglobin 6.2, mean corpuscular volume 65.7, leukocytes 5.7, platelets 350, creatine kinase 129. Lyme, Anaplasma, Erlichia, Babesia, and Epstein-Barr Virus studies were negative. Additional labs included low serum iron (11), high transferrin (380), high total iron binding capacity (475), and low transferrin saturation (2), consistent with iron deficiency anemia. He was admitted for further work up and received red blood cell transfusions and iron infusions. He underwent upper endoscopy, which revealed Barrett’s esophagus, erosive gastropathy, and a 4.9 cm submucosal, ulcerated mass in the second portion of the duodenum. Endoscopic ultrasound with fine needle biopsy of the duodenal mass was performed, which revealed gastrointestinal stromal tumor (GIST). CT abdomen/pelvis showed the large duodenal mass with no evidence of metastatic disease. The patient was referred to general surgery and medical oncology for management of GIST with resection and perioperative imatinib, an oral tyrosine kinase inhibitor.</p> <p>Discussion: Anemia in an otherwise healthy patient is a symptom of an underlying medical condition. The differential is broad and includes: blood loss, malabsorption of nutrients for blood cell production, medications, inherited mutations, infections, and malignancies. Mean corpuscular volume helps distinguish microcytic, normocytic, and macrocytic anemia. Microcytic anemia is caused by defective hemoglobin synthesis from deficiency in iron (ie. Nutritional, blood loss), globin chains (ie. Thalassemia), or heme (ie. Sideroblastic anemia, lead poisoning). Macrocytic anemia is caused by megaloblastosis (ie. Folate deficiency, B12 deficiency, myelodysplastic syndrome, aplastic anemia), red cell membrane changes (ie. Liver disease, excess alcohol, hypothyroidism), or reticulocytosis. Normocytic anemia is</p>

	<p>typically secondary to chronic inflammation, kidney disease, heart failure, adrenal insufficiency, or cancer. A low reticulocyte count may indicate impaired bone marrow function. If the reticulocyte count is high, then hemolysis labs (haptoglobin, lactate dehydrogenase, and bilirubin) can help detect presence of red cell destruction. For patients with microcytic anemia, iron studies can help identify the cause. Low serum ferritin, an indicator of the body's iron stores, is suggestive of iron deficiency. Genetic disorders of iron metabolism (ie. Hemochromatosis, thalassemia) or heme synthesis may have elevated ferritin and transferrin saturation. Family history, persistent anemia despite iron supplementation, or signs of iron overload such as neurological symptoms, liver dysfunction, and endocrinopathies, may aid diagnosis of a genetic disorder.</p> <p>GIST is a rare sarcoma that often presents with anemia due to bleeding from ulcerated tumor mucosa. Our case demonstrates initial presentation and work up of microcytic anemia from gastrointestinal bleeding in a young patient, leading to diagnosis of GIST. Immediate management includes treatment with RBC transfusion and work up includes identifying source of bleeding via endoscopy and imaging.</p>
<p><b>Erica Loon</b> Dr. Joanne Billings Dr. Nicholas Lim</p>	<p><i>Resolution of Portal Hypertension in Cystic Fibrosis Liver Disease After Start of CFTR Modulator: A Case Report</i></p> <p>Introduction: Portal hypertension (PH) secondary to cystic fibrosis liver disease (CFLD) is the third most common cause for mortality (after lung failure and lung transplantation) in adults with cystic fibrosis (CF). Notably, PH in CFLD more often occurs in absence of cirrhosis (i.e. non-cirrhotic PH, NCPH). Here, we describe a patient with NCPH secondary to CFLD, with resolution of PH after starting a cystic fibrosis transmembrane conductance regulator (CFTR) modulator.</p> <p>Case Description: A 73-year-old male diagnosed in 1964 with CF, homozygous F508del, characterized by 'severe' pulmonary obstruction and CF-related diabetes mellitus underwent lung transplant evaluation in 2015. Abdominal ultrasound revealed a nodular liver and splenomegaly (14.1 cm). Transjugular liver biopsy was non-diagnostic due to insufficient sampling, however, portosystemic gradient was elevated to 28 mmHg. Upper endoscopy (EGD) showed a single, grade I esophageal varix. Follow-up EGDs showed esophageal varices, including a grade III varix requiring band ligation in 2018, and PH gastropathy. Repeat biopsy confirmed portal and hepatic venous abnormalities and 3 mmHg portosystemic gradient, but no cirrhosis. The patient was declined listing for lung-liver transplant due to his advanced age and presence of PH.</p> <p>In 2019, he was started on CFTR modulator tezacaftor/ivacaftor and ivacaftor. Six months later, the patient was switched to the newer elexacaftor/tezacaftor/ivacaftor (ETI). Eight weeks after starting ETI, he was off oxygen at rest. By month 10, he was off all oxygen and short-acting insulin, and swimming 1,000 feet daily. In 2021, 18 months after starting ETI, EGD revealed a normal esophagus and stomach with resolution of esophageal varices and PH gastropathy. Ultrasound in 2022 again demonstrated a nodular liver but now with a normal-sized spleen (12.8 cm). EGD in 2023 confirmed absence of varices and gastropathy. In December</p>

	<p>2023, the patient was downgraded from ‘severe’ to ‘moderate’ pulmonary obstruction and achieving 20,000 steps per day.</p> <p>Discussion: Information on the efficacy of CFTR modulators in patients with CFLD is limited. As demonstrated in this patient, CFTR modulators may provide extra-pulmonary benefits including reversal of NCPH. Long-term use of CFTR modulators could potentially result in reductions in mortality from PH and need for future liver or combined lung-liver transplantation in patients with CFLD.</p>
<p><b>Isabel Lopera</b> Dr. Sarah Chalmers</p>	<p><i>Prescription Perils: A Case of Respiratory Failure Induced by Polypharmacy in a Patient with Multimorbidity</i></p> <p>Introduction: Polypharmacy is described as the use of multiple medications in one patient at a given time. It is highly associated with adverse drug events, including drug-drug and disease-drug interactions, and lower level of adherence. For adults in the US with multiple chronic conditions, or multimorbidity, polypharmacy can be inevitable. Age is often viewed as a major risk factor for polypharmacy; however, multimorbidity, cognitive impairment, residing in long term care facility, and frailty are also primary characteristics of individuals impacted by this phenomenon.</p> <p>Case Description: A 49-year-old female presented to the ED from her nursing home with increased somnolence and slurred speech. Her medical history is significant for traumatic brain injury (TBI) secondary to motor vehicle accident, complicated by quadriplegia, dysphagia with recurrent aspiration events, contractures requiring Botox, neurogenic bladder, bladder cancer status post radical cystectomy and urostomy with ileal conduit, pectus excavatum, obstructive sleep apnea (OSA), and chronic left loculated pleural effusion. Two days prior to her admission, she reported feeling more fatigued to her family. Her speech was noted to be progressively slurred, and she became increasingly somnolent. She denied fevers, chills, diarrhea, chest pain, shortness of breath, and recent sick contacts. The only notable change in her routine was the addition of buspirone to her medication regimen four days prior to presentation. Home medications included quetiapine, baclofen, aripiprazole, sertraline, and pregabalin.</p> <p>Upon arrival to the ED, she was noted to have a venous blood gas (VBG) showing pH 7.23, pCO<sub>2</sub> 96 mmHg. She was initiated on BiPAP without improvement on repeat blood gas and was subsequently intubated for increased work of breathing and uptrending CO<sub>2</sub>. Chest imaging revealed a stable left loculated effusion. She had no leukocytosis, fever, and her urinalysis was unremarkable. She admitted to the MICU for acute on chronic hypercapnic respiratory failure. Repeat VBG on mechanical ventilation was improved pH to 7.44 and PCO<sub>2</sub> 48 mmHg. She was successfully extubated with improvements in her mental status. Infectious workup continued to be negative, and antibiotics were discontinued. Her home medications were restarted with clinical improvement; buspirone was discontinued indefinitely, and she was successfully transferred to the floors.</p> <p>Discussion: Buspirone on its own carries a low sedation risk; however, in conjunction with other medications such as sertraline, aripiprazole, pregabalin or baclofen, it can precipitate drowsiness and even sedation as</p>

	<p>seen in this case. In patients with baseline risk factors for comprised respiratory status such as neuromuscular weakness, chest wall deformities, OSA, and chronic pleural effusion, careful consideration is warranted when managing their medication regimen. If changes are necessary, assessing the effects of not only the singular drug in question, but also potential side effects from its combined use with other medications is needed.</p>
<p><b>Giuseppe Maiocco</b>  Dr. Emma Johns  Dr. Rafid Mustafa</p>	<p><i>A Pressure Problem: Diagnostic Complexity in a Patient with Metastatic Lung Adenocarcinoma</i></p> <p>Case Presentation: A 65-year-old woman was admitted with altered mental status, lethargy, and generalized weakness. Her medical history includes stage IIIA lung adenocarcinoma diagnosed 4 months prior, status post radiotherapy on active carboplatin/paclitaxel. Leading to admission, she reported 2-day history of coughing fits with frontal headache and 1-day history of lower back pain. Initial work-up included CT head, revealing 4 new hemorrhagic lobar lesions likely intracranial metastases, the largest in the right temporal lobe. Local mass effect was present without midline shift, ventriculomegaly or hydrocephalus. CT spine demonstrated no spinal metastases. She received intravenous dexamethasone. On admission at 7:00pm, vitals were stable with largely normal neurologic examination, with plan for overnight hydrocephalus watch with morning MRI, oncology, and neurosurgical evaluation.</p> <p>Overnight, her lower back pain steadily worsened, with vague localization and episodic lower extremity radiation. Three times, her pain was punctuated by brief, self-resolving episodes of posturing without alteration in consciousness or positional association. Between episodes, she returned to her neurologic baseline. Pain was minimally controlled by medical management. Scheduled 6 hour CT showed stable hemorrhagic lesions without hydrocephalus. The following morning, she experienced two focal neurologic episodes. Initially, she developed brief, resolving unilateral left lower extremity weakness with unchanged stat CT head. During return transport, she became unresponsive with upper extremity flexion, worsening right-sided ptosis and right-sided pupillary dilatation. Repeat CT showed no interval changes. Dexamethasone and labetalol were administered, with neurosciences ICU transfer. Seizure was suspected, particularly given right temporal lobe lesion. She received levetiracetam load with subsequent mental status improvement. EEG demonstrated global alteration of cerebral functioning without epileptiform discharges. Neurosurgery was consulted, and with high clinical suspicion of seizure secondary to hemorrhagic metastasis, did not recommend emergent CSF diversion. She was stable until evening. She underwent MRI at 6:00pm. At 7:30, she became somnolent, with right-sided ptosis and pupillary dilatation. EEG demonstrated changes concerning for herniation (burst suppression pattern). MRI resulted, with newly identified leptomeningeal enhancement concerning for meningeal carcinomatosis. She was intubated, hyperventilated and administered mannitol with aim to medically reduce intracranial pressure (ICP). Further intervention including external ventricular drain placement was discussed with patient’s family. After thoughtful discussion, she was transitioned to comfort care.</p> <p>Conclusion: Here, we present a case of plateau waves of increased ICP as a</p>

presenting symptom for leptomenigeal carcinomatosis. Leptomenigeal carcinomatosis is a rare metastatic site for lung adenocarcinoma, occurring in 3-5% of non-small cell lung cancer patients. Plateau waves are an underrecognized consequence, resulting from short ICP spikes, with symptoms including altered mental status, headache, ataxia, tonic posturing, and visual changes. In acute settings, diagnosis is complicated as episodes can mimic seizures, or even result from seizure, and be overshadowed by significant comorbid disease. ICP spikes result from impaired CSF resorption, however, concomitant ventricular stiffening may hide radiographic evidence including ventriculomegaly and hydrocephalus. CT has poor sensitivity for leptomenigeal disease, while MRI is most sensitive (76-87%). Leptomenigeal carcinomatosis represents a poor prognosis, with elevated ICP necessitating pressure stabilization via CSF diversion, such as lumbar puncture or ventricular drain placement.

**Danielle Mangine**  
Dr. Amy Holbrook

*Mad Tau Disease*

Introduction: Prion diseases are rare neurodegenerative diseases that lead to rapidly progressive dementia and other devastating neurologic decline. While there are different variants and kinds of prion diseases, sporadic Creutzfeldt-Jacob disease (CJD) is the most common human form. In the United States, roughly 350 cases of CJD are diagnosed each year. The diagnosis of CJD remains challenging due to its rarity and similarity to other diseases presenting with rapidly progressive dementia.

Case Presentation: A 69-year-old male with a past medical history of hypertension, hyperlipidemia, and type II diabetes presented with two months of rapidly progressive memory loss, impaired executive functioning, speech impairment, increased confusion, and gait abnormality. On ED presentation, he was vitally stable. CBC, BMP, LFTs, TSH, and ammonia were within normal limits except for mild hyperglycemia. An MRI of the brain showed abnormal diffusion restriction of right greater than left caudate/putaminal nuclei and right hemispheric cerebral cortices. The radiologist opined that MRI imaging was possibly consistent with Creutzfeldt-Jacob disease (CJD). Neurology was consulted to assist in diagnosis of rapidly progressive neurologic symptoms. Video electroencephalogram (vEEG) was initiated and showed generalized abnormalities that suggested diffuse bilateral cortical dysfunction and encephalopathy with generalized periodic discharges throughout. Additionally, the patient underwent a lumbar puncture for CSF analysis which only showed two nucleated cells. Meningitis/encephalitis multiplex PCR was negative, making CNS infection unlikely. Additional CSF studies were sent out to test for 14-3-3 protein and T tau protein. CT chest/abdomen/pelvis was negative for malignancy lowering suspicion for a paraneoplastic process. The patient's course was complicated by a rapid response called for seizure-like activity. Review of vEEG demonstrated progressive bradycardia to a total of eight seconds of asystole followed by tachycardia. Ultimately, the combination of presenting symptoms, MRI, CSF, and vEEG findings were most consistent with CJD. Palliative care was consulted for assistance with goals of care given poor and rapid prognosis with CJD. The patient was transitioned to comfort care and discharged to home with home hospice. The patient died within weeks of discharge. Send out CSF studies returned: 14-3-3 protein positive and T tau protein > 20,000,

	<p>confirming the diagnosis of CJD.</p> <p>Conclusion: This case illustrates the diagnostic challenges that accompany prion diseases. Diagnosis of these rare diseases require maintaining a broad differential and workup to rule out other causes, as the prognosis for prion diseases are universally fatal. Uniquely in this case, the constellation of rapidly progressive neurologic symptoms, MR brain with abnormal diffusion restriction in the caudate/putaminal nuclei, and EEG with generalized periodic discharges were hallmark for CJD although all three findings are not universally present in patients with CJD. As such, completing a thorough work up aimed at identifying other potential reversible causes remains imperative to ensure accurate diagnosis given the danger and thus rarity of formal tissue sampling for diagnosis confirmation.</p>
<p><b>Oscar Monterrosa</b> Dr. Aditya Chauhan Dr. Kristina Krohn</p>	<p><i>Rapid Explosive Streptococcus anginosus Necrotizing Pneumonia After Suspected Seizure Aspiration</i></p> <p>Introduction: Seizures are a well-known risk factor for the development of aspiration pneumonia given the lack of protective reflexes during seizure activity. Typical causative organisms are polymicrobial, however one particular organism that is commonly associated with dental caries and abscesses is the Streptococcus anginosus group (SAG) species. These groups of organisms are known to lead to the development of invasive necrotizing infections, likely due to the organism’s ability to cross fascial planes. Extensive and fulminant infections, however rare in immunocompetent young adults, is seen in this case that resulted in lung decortication and resection.</p> <p>Case Description: A young male adult was admitted to the emergency department following a vehicle accident, where he sustained minor injuries. During triage, he experienced a seizure. He subsequently had multiple seizures while undergoing a CT scan and upon admission to the emergency department, necessitating intubation for airway protection.</p> <p>During his ICU stay, he developed a fever (101.3F) and leukocytosis (WBC count of 15), but initial cultures were negative for infection. The patient reported diffuse abdominal pain, and a CT of the abdomen and pelvis suggested aspiration pneumonia, leading to treatment with ceftriaxone. Sputum cultures later identified Haemophilus influenzae, prompting a switch to cefuroxime prior to discharge.</p> <p>Five days post-discharge, the patient presented via ambulance with palpitations, shortness of breath, seizure, and cough productive of brown purulence and bowel incontinence. Chest X-ray revealed a significant new lucency over the right lung with mediastinal shift. A subsequent CT scan showed a large multiloculated empyema in the left hemithorax with near complete collapse of the left lung and signs of tension. It was initially thought that there was bowel in the thoracic cavity due to diaphragmatic injury given the extensive septations on imaging. A chest tube was placed, draining 750 mL of foul-smelling purulent fluid, with cultures positive for Streptococcus anginosus and Bacteroides fragilis. The patient underwent video-assisted thoracoscopic surgery (VATS) and total decortication with left upper lobe wedge resection. Postoperatively, a chest X-ray indicated a left</p>

	<p>apical/lateral hemothorax, necessitating reoperation to remove a retained clot. During this procedure, a ruptured abscess in the left lower lung was discovered, resulting in a left lower lobe resection.</p> <p>Discussion: This case highlights the rapid progression of a small left pleural effusion with consolidation into a massive multiloculated empyema with near complete collapse of the left lung over the span of 5 days. SAG organisms are commonly encountered in oral flora, particularly prevalent in individuals with dental caries or abscesses. In patients who develop suspected aspiration pneumonia or radiographic evidence of pneumonia in the form of consolidations or pleural effusions, extra considerations must be taken to ensure these cases do not progress into life-threatening, necrotizing infections. As seen with this case, the surgical management with extensive lung parenchymal involvement often requires VATS decortication or lung resection in complicated cases.</p>
<p><b>Jordan Nunnelee</b> Dr. James Utz</p>	<p><i>Think Twice About Asthma</i></p> <p>Case Description: A 67-year-old female patient with history of osteopenia and asthma in childhood and early adulthood presented with progressive dyspnea and fatigue. She was seen in her local urgent care two months prior to hospitalization with dyspnea and wheezing. Chest x-ray reportedly demonstrated a right lower lobe pneumonia. She was prescribed antibiotics without clinical improvement. She was treated repeatedly for asthma exacerbations. Subsequent chest CT showed near collapse of the right lower lobe with high-density material filling the bronchi, and left-lower lobe central bronchiectasis. Due to progressive symptoms, she presented to the emergency department. She arrived normotensive, with normal oxygen saturations on room air. There were decreased breath sounds in the right lower lung fields, though without abnormal breath sounds or increased work of breathing at rest. She was previously very active, but recently dyspneic after 300 steps. She lived in the same home for decades, adjacent to farms. She had not been on any inhalers for asthma in decades until her pneumonia was diagnosed two months earlier, without improvement.</p> <p>Labs demonstrated a mild leukocytosis to <math>12.8 \times 10^9</math> (ref: <math>3.4-9.6 \times 10^9</math>) with peripheral hypereosinophilia to <math>1.92 \times 10^9</math> (ref: <math>0.03-0.48 \times 10^9</math>). Histoplasma and Blastomyces urine and serum antigens were negative. She underwent bronchoscopy, which demonstrated significant mucous impaction of the right lung below the right upper lobe takeoff. Copious rubbery mucous was aspirated, with some in cast formation. Her anti-aspergillus IgE and IgG titers were elevated, with an isolated total IgE elevation. Fungal cultures from bronchial washings grew <i>Aspergillus fumigatus</i> and <i>Aspergillus niger</i> complex.</p> <p>She was diagnosed with allergic bronchopulmonary aspergillosis and discharged with prednisone and posaconazole due to high fungal burden and severity of symptoms. After bronchoscopy, she had significant improvement in exercise capacity.</p> <p>Discussion: This case demonstrates a classic case of allergic bronchopulmonary aspergillosis (ABPA), though without a characteristic lifelong chronic, refractory asthma history. However, she had recent</p>

	<p>recurrent asthma symptoms. Our patient met diagnostic criteria for ABPA through her predisposing asthma, positive serum anti-Aspergillus fumigatus IgE and elevated total IgE &gt;1000 IU/mL (both required), hypereosinophilia, and elevated serum anti-Aspergillus IgG. While CT imaging did not demonstrate typical bilateral upper lobe predominant pulmonary opacities, there was evidence of mucoid impaction leading to bronchial obstruction and central bronchiectasis. Bronchoscopy confirmed stiff mucoid bronchial material characteristic of ABPA. The IDSA recommends antifungal treatment upfront to reduce long-term steroid use. Our patient was started on an upfront antifungal due to osteopenia and significant disease burden. ABPA is not an invasive Aspergillus infection, but rather a reactive process thought to be a Th2-mediated response to inhaled Aspergillus antigen. A delay in diagnosis can lead to irreversible bronchiectasis due to recurrent inflammatory response.</p> <p>It is important to consider ABPA in a patient with history of refractory asthma, even if asthma symptoms are only recently recurrent. ABPA should be suspected when acute infection has been ruled out. Bronchoscopy can be helpful to identify mucoid bronchial material, which is helpful in diagnosis and therapeutic in cases of bronchial obstruction.</p>
<p><b>Anusha Parisapogu</b> Dr. Renee Donahue Carlson Dr. Mary Kasten</p>	<p><i>Disseminated Mycobacterium hassiacum in a Woman with Adult-Onset Anti-IFN-γ Auto-antibody Immunodeficiency</i></p> <p>Background: Mycobacterium hassiacum (M. hassiacum) is a rapidly growing non-tuberculous mycobacterium (NTM), that was originally described in 1997 as a species not believed to cause disease in humans. We found only five reported cases of M. hassiacum disease. Disseminated NTM infection can pose significant diagnostic and therapeutic challenges, particularly in patients with underlying immunodeficiencies. We present a complex case of disseminated M. hassiacum in a patient with adult-onset anti-interferon-gamma (IFN-γ) autoantibody immunodeficiency, highlighting the diagnostic and therapeutic complexities involved.</p> <p>Case Presentation: A 42-year-old Laotian woman, living in the U.S. for 10 years, presented with several months of unexplained weight loss, arthralgias, leg pain, chronic cough, and a new pruritic rash over her trunk and thighs. Physical examination revealed tender hepatosplenomegaly and diffuse lymphadenopathy. Initial laboratory workup showed elevated inflammatory markers, leukocytosis with mild eosinophilia, mild anemia, and polyclonal hypergammaglobulinemia. Rheumatologic testing was largely unremarkable, except for a positive rheumatoid factor and ANA at 1:80. Imaging studies revealed extensive lymphadenopathy, hepatosplenomegaly, and pulmonary nodules. Biopsies of her cervical lymph nodes and bone marrow were initially inconclusive, leading to a differential diagnosis that included atypical rheumatoid arthritis or adult-onset Still's disease. She was treated empirically with ivermectin, prednisone 40 mg daily, methotrexate 12.5 mg weekly, and TMP/SMX for prophylaxis.</p> <p>Ten days after discharge, mycobacterial cultures from her bone marrow and blood returned positive. Further immunologic testing revealed a negative HIV result, normal CD4 counts, and the presence of anti-IFN-γ antibodies, confirming her immunodeficiency. The rapidly growing mycobacterium was</p>



	<p>identified as <i>M. hassiacum</i>. Antimicrobial susceptibility testing guided the initiation of a multidrug regimen, including ceftaroline, azithromycin, imipenem, and moxifloxacin. She responded well, but her clinical course was complicated by macrophage activation syndrome, requiring treatment with anakinra, intravenous immunoglobulin (IVIG), and rituximab. Drug-related side effects complicated her management, and she was transitioned to an oral regimen of omadacycline, tedizolid, azithromycin, and moxifloxacin. Her symptoms resolved over several months, with negative blood cultures at two months and one-year post-treatment. Due to recurrent anti-IFN-<math>\gamma</math> antibodies, she received additional rituximab one year later. Repeat imaging showed resolution of her lymphadenopathy and hepatosplenomegaly, with stable or resolved pulmonary nodules. She was successfully taken off <i>M. hassiacum</i> prophylaxis and remains asymptomatic.</p> <p>Discussion: Anti-IFN-<math>\gamma</math> antibodies are increasingly recognized as a cause of susceptibility to opportunistic infections, particularly NTM, non-typhoid <i>Salmonella</i>, <i>Cryptococcus</i>, and varicella zoster virus. This immunodeficiency predominantly affects women of Asian descent [1]. This case underscores the importance of recognizing anti-IFN-<math>\gamma</math> autoantibodies early in patients presenting with recurrent or unusual infections. Mismanagement with immunosuppressive therapies can worsen the patient’s condition. Monitoring anti-IFN-<math>\gamma</math> antibody levels is critical for preventing relapse, and targeted therapies such as rituximab appear beneficial in reducing antibody production and improving outcomes.</p> <p>References: Hong GH, Ortega-Villa AM, Hunsberger S, et al. Natural History and Evolution of Anti-Interferon-<math>\gamma</math> Autoantibody-Associated Immunodeficiency Syndrome in Thailand and the United States. <i>Clin Infect Dis.</i> 2020;71(1):53-62. doi:10.1093/cid/ciz786</p>
<p><b>Dil Patel</b> Dr. Bradley Bart</p>	<p><i>Silent Myocardial Infarction: Downstream Consequences of Failing to Recognize Infarct Pattern Unmasked by Premature Ventricular Contractions</i></p> <p>Introduction: Myocardial infarctions (MIs) are commonly suspected in patients with chest discomfort or other cardiac symptoms in the setting of known coronary artery disease (CAD) or established cardiac risk factors. However, “Silent MIs” tend to present without cardinal symptoms and often go unrecognized in the outpatient setting. Consequently, people with unrecognized MIs are left untreated, which puts them at risk for further complications.</p> <p>Case Presentation: A 76-year-old male, with no prior CAD, was seen in the primary care clinic for his annual exam. He had no complaints or symptoms concerning for ischemia or heart failure. The provider obtained a 12-lead electrocardiogram (ECG), but they did not recognize new bradycardia and a set of premature ventricular contractions (PVCs) revealing a new infarct pattern in the anterior precordial leads.</p> <p>Twelve days later, he came to the emergency department (ED) for nausea, vomiting, and diarrhea. He was discharged home with a presumptive diagnosis of gastroenteritis. Four days thereafter, his symptoms progressed along with new onset dyspnea and excessive fatigue. Upon returning to the ED, the patient was hypotensive and in significant respiratory distress</p>

	<p>requiring 6-10 L of oxygen. A new 12-lead ECG showed inferolateral ST depression and T wave inversions, which were new compared to prior ECGs. High-sensitivity troponin was greater than 30,000, and a stat echocardiogram (ECHO) showed an ejection fraction (EF) of 35% with a large wall motion abnormality involving the left anterior descending (LAD) territory and a large left ventricular apical thrombus. The patient's condition deteriorated, and he was ultimately intubated. With a diagnosis of cardiogenic shock, he was sent to the cardiac catheterization laboratory where he was found to have severe three-vessel disease with chronic total occlusions of the LAD, second diagonal, posterior descending, posterior atrioventricular, and posterolateral arteries. Additionally, there was a 95% occlusion in the second obtuse marginal suggestive of a culprit lesion. Due to a low cardiac index and elevated filling pressures, an intra-aortic balloon pump was placed, and pressors were started. Twelve days later, the patient was stable enough to be taken to the operating room for a coronary artery bypass graft x 4.</p> <p>He was discharged three weeks after admission, and he was independent with his activities of daily living. His prior-to-discharge ECHO demonstrated an EF of 25% with mild to moderate mitral regurgitation.</p> <p>Discussion: This case highlights the importance of being aware of the entity of “Silent MIs” and being able to recognize clinically silent infarct patterns on ECGs. Approximately 20-30% of acute MIs are either asymptomatic or associated with non-specific symptoms. In this case, the fortuitous occurrence of PVCs helped identify the infarct. If the ECG finding had been recognized at that clinic visit, then potentially, the nearly fatal cardiogenic shock episode, that occurred two weeks later, could have been avoided. It is important for all providers, especially primary care, to identify these infarct patterns and seek an expert’s opinion if they are uncertain. Early recognition of “Silent MIs” allows for early intervention and lowers the risk of subsequent catastrophic events.</p>
<p><b>Parth Patel</b>  Dr. Richa Shah  Dr. Kevin Stanko</p>	<p><i>Loss of Consciousness: A Multifactorial Diagnosis in the Setting of Iron Deficiency Anemia</i></p> <p>Introduction: Fainting and lightheadedness can arise from various etiologies; it requires a systematic evaluation for accurate diagnosis.</p> <p>Case Presentation: We present a 44-year-old female with medical comorbidities notable for prior sleeve gastrectomy with subsequent 25-kg weight loss who presented with recurrent fainting and loss of consciousness (LOC) over the past few months. These episodes initially occurred once per week but increased in frequency to 3-4 times weekly. They last 10-15 seconds to 1-2 minutes and generally occur while standing without specific triggers; they are preceded by nausea, lightheadedness, and double vision. Per her husband, episodes are characterized by complete LOC without seizure-like activity. While she endorsed immediate return to her cognitive baseline upon regaining consciousness, palpitations and fatigue would persist for several hours thereafter. Between episodes, she is asymptomatic. She consumes small meals with minimal fluid intake (2-3 glasses of water daily). On presentation, she was hemodynamically stable, with no current symptoms and negative orthostatic vitals. Labs revealed severe microcytic</p>

	<p>anemia with hemoglobin of 7.5 g/dL and ferritin of 3 ng/mL, indicating iron deficiency anemia (IDA). Folate, vitamin B12, and vitamin D levels were normal. Cardiac workup, including ECG, echocardiogram, cardiopulmonary stress test, and Holter monitoring, was negative, lowering suspicion of cardiogenic syncope. She denied blood loss including heavy menstrual and gastrointestinal bleeding. Nonetheless, concerns remained about occult bleeding, malabsorption, and dysautonomia. EGD excluded upper gastrointestinal bleeding. Colonoscopy was declined due to prep intolerance. Given her low ferritin levels, she was started on intravenous iron repletion. H. Pylori breath testing also returned positive, and she completed treatment with confirmed eradication. At follow-up, hemoglobin improved to 10.2 g/dL and ferritin to 158 mcg/L. She reported increased energy and significantly fewer syncopal episodes.</p> <p>Conclusion: This case emphasizes the importance of maintaining a broad differential when evaluating LOC to include both syncopal and non-syncopal causes. Syncopal etiologies for LOC included cardiogenic (arrhythmias, structural heart disease), reflex (vasovagal, situational, carotid hypersensitivity), or orthostatic (autonomic failure, postural orthostatic tachycardia syndrome). Non-syncopal etiologies included metabolic derangements (hypoglycemia) and neurologic/psychiatric manifestations (seizures, transient ischemic attacks, strokes, panic attacks). Given the substantial improvement following iron repletion, we concluded IDA was a major contributor to her presentation. We suspected her underlying etiology to be orthostatic hypotension based on her position-dependent and prodromal symptoms. Her low fluid intake and iron deficiency likely exacerbated her episodes through reductions in intravascular volume and hemoglobin levels, respectively. Although orthostatic testing was nonconfirmatory, this one-time finding cannot be used as a definitive rule-out, especially when accounting for her symptoms and presentation. Our case further highlights how multiple underlying conditions like nutritional deficiencies and infection can coalesce to present as LOC. We contemplated if autonomic dysfunction and/or vasovagal syncope could also contribute to her symptoms. Tilt-table testing was initially recommended but declined due to symptom improvement after iron repletion. Ultimately, her improvement with iron supplementation and H. pylori treatment demonstrated the importance of addressing even subtle contributors, illustrating the necessity of a comprehensive and stepwise approach when evaluating complex presentations like LOC.</p>
<p><b>Carol Pengshung</b> Dr. Alexander Khoruts</p>	<p><i>Fecal Microbiota Transplant: Curative Treatment for Fulminant Clostridium Difficile Colitis</i></p> <p>Introduction: Fulminant Clostridium difficile colitis (FCDC) is a life-threatening infection characterized by hypotension, shock, or toxic megacolon. It is rare and occurs in 1-3% of all C. difficile infections (CDI) with an in-hospital mortality rate of 35%. Urgent abdominal surgery is indicated for patients with FCDC when medical treatments fail. However, surgery carries a mortality of 35% to 80%. Fecal microbiota transplant (FMT) is a promising alternative treatment to surgery.</p> <p>Case Presentation: A 63-year-old man with a history of chronic lymphocytic leukemia, myocardial infarction in 2018, possible Kawasaki's disease, and</p>

	<p>recent West Nile encephalitis was admitted for fever and lethargy. Chest X-ray demonstrated bibasilar opacities for which he received Zosyn for aspiration pneumonia. He then developed diarrhea and tested positive for CDI. He started PO vancomycin while systemic antibiotics were continued. He subsequently became hypotensive requiring stress dose steroids and vasopressors. CT chest abdomen-pelvis showed new airspace consolidation concerning for pneumonia, diffuse severe colitis, and diffuse anasarca. He was ultimately intubated. He started acetylcysteine with improvement and was extubated with decreasing vasopressor requirements. However, the following day, he became febrile and more hypotensive. Repeat CT showed severe pancolonic wall thickening and enhancement as well as transverse colon distention measuring up to 10 cm. His CDI progressed to FCDC with a toxic megacolon.</p> <p>Colorectal surgery was consulted, but the patient’s family declined surgical intervention based on his previously expressed wishes. He transferred to the ICU, requiring ventilator and vasopressor support. His colonoscopy showed diffuse pseudomembranes consistent with late stage FCDI. He ultimately required four courses of FMT prior to transfer to a long-term acute care hospital for rehab with a prolonged course of fidaxomicin (and later vancomycin). After four months in the hospital, he was finally discharged home. A consolidation FMT using oral capsules was administered in the outpatient clinic.</p> <p>Conclusion: This case highlights the treatments available for FCDC when medications fail as well as the importance of shared decision-making and involving the appropriate specialties early. For FCDC, surgical interventions include colectomy and alternatively diverting loop ileostomy. More recently, FMT has emerged as a less invasive option to surgery without the associated high mortality. The hospital’s FMT protocol for FCDC consists of three phases. Initially, infusion of fecal microbiota achieves dampening of the systemic inflammatory response, which results in improvement in hemodynamic parameters. Antibiotics against C. difficile are continued during phase two. However, patients who have suffered severe/fulminant CDI remain at high risk for CDI relapse. Therefore, consolidation FMT is performed to repair the antibiotic-induced dysbiosis.</p>
<p><b>Lauren Pomerantz</b> Dr. Henry Schultz</p>	<p><i>A Master of Disguise: A Case Series of Transthyretin Cardiac Amyloidosis Masquerading as Heart Failure</i></p> <p>Introduction: Transthyretin cardiac amyloidosis (ATTR-CM) is a common but underrecognized systemic disorder caused by tissue accumulation of a misfolded transthyretin (TTR) protein produced by the liver. ATTR-CM can mimic hypertensive heart disease, hypertrophic cardiomyopathy, concentric hypertrophy from aortic stenosis, and idiopathic HFpEF. ATTR amyloid is distinct from AL amyloidosis, a monoclonal plasma cell disorder.</p> <p>There are two types of ATTR amyloidosis: wild type (wt) and hereditary (v). Specifically, ATTRwt tends to be seen in older Caucasian men, and is responsible for 75% of ATTR-CM in the US. Over 130 mutations can lead to hereditary TTR amyloid (hATTR or ATTRv), each with a different phenotype.</p> <p>Here we present three cases of patients with ATTRwt cardiac amyloidosis.</p>

	<p>An 83-year-old male presented with biventricular heart failure, bilateral carpal tunnel syndrome, spinal stenosis, and atrial fibrillation (A-fib). He had a suspicious TTE, equivocal cardiac PYP scan, negative fat aspirate, and an endomyocardial biopsy positive for TTR amyloid by mass spec. Gene sequencing revealed no mutations.</p> <p>An 84-year-old male with a history of severe spinal stenosis, HFpEF, HTN, A-fib, and bilateral carpal tunnel syndrome. Cardiac PYP scan was suspicious for amyloidosis. ATTRwt was confirmed by Congo red staining and mass spec of lumbar spine resection tissue.</p> <p>An 87-year-old male patient with HFpEF, severe AS, CAD, Pulmonary Hypertension, CKD stage IV, and HTN who had a suspicious TTE, grade 3 uptake on PYP scanning, and Cardiac MRI findings consistent with cardiac amyloidosis. Endomyocardial biopsy and mass spec confirmed ATTRwt.</p> <p>All three patients are receiving therapy with the TTR stabilizer Tafamadis, with demonstrated clinical improvement. Concern for cardiac amyloidosis is often raised in idiopathic heart failure patients with suspicious echo findings. Next, workup must exclude AL amyloidosis, by obtaining serum immunoglobulin free light chains and immunofixation. If monoclonal protein testing is positive, further workup for AL amyloidosis is indicated. Using circulating transthyretin as a diagnostic marker has recently been investigated, though its utilization remains limited to clinical research. A positive biopsy with supportive imaging findings is diagnostically sufficient. Of note, fat aspirate is more sensitive for AL amyloidosis and is often negative in patients with ATTR-CM. If monoclonal proteins aren't present, perform cardiac nuclear scintigraphy. A positive scintigraphy scan should prompt TTR genetic testing. Positive genetic sequencing indicates hereditary TTR. If negative, the patient has wild-type TTR. If nuclear scintigraphy is negative, but clinical suspicion remains, obtain a myocardial biopsy. If carpal tunnel release tissue or spinal stenosis decompression tissue is available, stain with Congo red and if positive, endomyocardial biopsy can be avoided.</p> <p>PYP scan findings alone can be diagnostically sufficient for ATTR-CM if there is grade 2/3 uptake and no evidence of a monoclonal protein. Mass spectrometry determines involvement by TTR vs AL. There are two mainstays of treatment for hATTR: TTR knockdown agents or silencers (e.g., Patisiran) specifically approved for the polyneuropathy phenotype, and TTR stabilizers (e.g., Tafamadis), approved for cardiac phenotypes. TTR stabilizers are approved only for ATTR-CM, although a recent study of the silencer Vutrisiran revealed its high effectiveness.</p>
<p><b>Adam Poquette</b> Dr. Anya Jamrozy</p>	<p><i>A Rare Case of Pancreatitis-Induced Intramural Gastric Hematoma</i></p> <p>Introduction: Pancreatic pseudocysts are a well-known complication of chronic pancreatitis, occurring in ~10% cases. These thin-walled structures are typically filled with pancreatic enzymes and can grow to impressive sizes leading to a variety of consequences including infection, mass effect, and enzymatic degradation of adjacent tissues.</p> <p>Case Presentation: A 35-year-old woman with a history of asthma and remote post-partum hemorrhage presented to the Emergency Room</p>

	<p>following a near syncopal event while at work. She also disclosed 1-2 months of early satiety and abdominal bloating. Initial vitals notable for tachycardia to 115 bpm though she was otherwise hemodynamically stable. Physical exam revealed low muscle mass with protuberant abdomen-though soft and nontender to palpation. Laboratory evaluation was significant for profound macrocytic anemia with hemoglobin of 6.3 and MCV of 103, potassium 2.9, magnesium 1.1, AST 174, ALT 61, bilirubin of 2.4 and normal lipase. FAST exam was without hemorrhage and rectal exam hemoccult negative. Patient transfused 1 unit pRBC, started on BID PPI, and admitted for further workup.</p> <p>Initially, the etiology of the patient's anemia was theorized to be multifactorial: potential gastritis or ulcer given history of NSAID and possible alcohol overuse, menstrual blood loss and folate deficiency hypoproliferation. However, despite blood transfusion, the patient's hemoglobin declined further to 6.1 on hospital day 1. CT abdomen pelvis obtained given concern for unidentified bleeding which revealed multiple pancreatic pseudocysts as well as 8.2 x 8.3 x 8.7 cm heterogeneous fluid collection arising from the greater curvature of the stomach suggestive of intramural gastric hematoma. A small amount of hemoperitoneum was also noted. Urgent endoscopy revealed an intramural bulge as well as mucosal erosions without stigmata of bleeding. Patient transfused an additional 2u pRBC and transferred to the ICU for close observation. Follow up triple-phase angiogram was without extravasation of contrast. General surgery opted to monitor with conservative management. Patient's blood pressure remained normal, and her hemoglobin rose appropriately with transfusion and remained stable at 8-9. Patient tolerated a regular diet by hospital day 2. She transferred out of the ICU later that day and discharged home on hospital day 4 with close outpatient follow-up. Phosphatidyl ethanol later returned notably elevated at 249.</p> <p>Discussion: This case represents a rare consequence of a common condition. Intramural hematoma is classically caused by trauma, though there have been cases described as a potential consequence of acute or chronic pancreatitis. Despite the concerning presentation, most patients do well with conservative management. This case was unique in that the patient presented without abdominal pain, had no subjective or recorded history of pancreatitis, and reported mild alcohol use. Intramural hematoma should remain on the differential in the correct clinical context if workup for worsening anemia returns otherwise unrevealing.</p>
<p><b>Yogitha Posani</b></p>	<p><i>Acute Psychosis Caused by Thyrotoxicosis in a Patient with Untreated Graves' Disease</i></p> <p>Introduction: Neuroendocrine small cell type carcinomas are aggressive cancers that are commonly found in the lung but very rarely found in extrapulmonary sites. Given the rarity of this cancer, there are no randomized clinical trials or prospective clinical studies to guide management. Current treatments are based on the site of cancer with information from case reports and personal experience. This clinical vignette focuses on a case of primary small cell carcinoma of the thyroid managed with multimodal therapy.</p>

	<p>Case Description: A 59-year-old woman was hospitalized with worsening anterior neck pain and difficulty swallowing. Initial imaging revealed a hypo-enhancing mass in the inferior left thyroid lobe, compressing the trachea suggestive of a malignant thyroid neoplasm. Fine needle aspiration of the mass confirmed high-grade neuroendocrine carcinoma, small cell type. Subsequent PET/CT scans showed extensive disease, including cervical node involvement and hypermetabolic lesions in the lungs and pancreas. She began treatment of the thyroid mass following guidelines for limited-stage small cell lung cancer (SCLC) with cisplatin and etoposide, followed by radiation of the thyroid. Follow-up PET scans showed varied responses, including stable and progressive disease in different areas. After 6 cycles of treatment, new masses were identified in the pancreas that were later biopsied and found to be high-grade neuroendocrine small cell type as well.</p> <p>Afterward, the patient was started on immunotherapy with Ipilimumab and nivolumab with initial stabilization of the pancreatic masses. However, follow-up PET imaging showed progression with new lesions in the cervical and abdominal region.</p> <p>Further discussion with an outside institution prompted the patient to be started on Docetaxel, given the progression while on immunotherapy. Three months later, the patient’s condition deteriorated, with new brain metastases identified on MRI. She was initiated on 4th line FOLFIRI treatment and underwent brain radiation, leading to some improvements in metastatic lesions. However, by the end of January, her condition worsened again, prompting further radiation therapy to a new left upper quadrant mass and initiation of treatment with Temodar. Despite treatment efforts, her cancer progressed, and she transitioned to comfort cares in April. She ended up passing away, a little more than 1.5 years after diagnosis.</p> <p>Discussion: Small cell type carcinomas of the lung are rare and aggressive cancers with a tendency to reoccur and spread to distant sites [1]. Furthermore, extrapulmonary small cell type neuroendocrine tumors are even rarer with an overall incidence of approximately 0.1% to 0.4% [2]. The rarity of this subset of cancer makes studying it and forming guidelines for management difficult. Through this case, we were able to see the entire course of this cancer from its first detection to its response to multimodal treatments including chemotherapy, radiation therapy, and immunotherapy. In this case, the cancer had progressed despite aggressive management. More information is needed about this subtype of cancer and how it behaves to guide appropriate therapy.</p> <p>References  1. <a href="https://doi.org/10.1038/s41572-020-00235-0">https://doi.org/10.1038/s41572-020-00235-0</a>  2. <a href="https://doi.org/10.1002/cncr.22887">https://doi.org/10.1002/cncr.22887</a></p>
<p><b>Maximilian Redemann</b>  Dr. Mohammed Khalid</p>	<p><i>Inferior Wall Inferno: Recurrent Stent Thrombosis</i></p> <p>Introduction: Rasmussen aneurysms are rare, potentially fatal vascular complications of cavitory pneumonia, occurring in 5-8% of such cases and carrying mortality rates as high as 50-100% when ruptured. This report describes a case of a 43-year-old male who experienced multiple episodes of new-onset hemoptysis due to ruptured Rasmussen aneurysms secondary to</p>

cavitary necrotizing pneumonia. The patient underwent multiple interventional radiology (IR) coil embolization procedures and, despite several recurrences of massive hemoptysis, was successfully treated. This case emphasizes the challenges of early identification and management of this rare condition and the need for further research into improved diagnostic and treatment strategies.

**Case Presentation:** The patient, a 43-year-old male with a past medical history of Type 1 Diabetes, initially presented to the emergency department (ED) with pleuritic chest pain, cough, and shortness of breath following recent treatment for influenza B. During the evaluation, he was found to be in acute hypoxic respiratory failure, secondary to superimposed polymicrobial bacterial pneumonia. Cultures revealed the presence of methicillin-resistant *Staphylococcus aureus* (MRSA) and *Streptococcus pneumoniae*. Despite aggressive management, the patient's condition deteriorated rapidly, progressing to septic shock and acute respiratory distress syndrome (ARDS). Due to the severity of his condition, the patient was emergently intubated and placed on veno-venous extracorporeal membrane oxygenation (VV-ECMO) on 5/6/2024.

The clinical course was further complicated by the development of necrotizing pneumonia and pneumothorax, necessitating chest tube placement. Additionally, a *Klebsiella* ventilator-associated pneumonia (VAP) was identified, further exacerbating the patient's respiratory condition. On 5/25, the patient experienced sudden massive hemoptysis, prompting an exploratory bronchoscopy and a computed tomography (CT) pulmonary angiogram. Although the initial imaging was negative for pseudoaneurysm, due to ongoing bleeding, the right bronchial artery was embolized.

Recurrent hemoptysis episodes occurred on 6/4, 6/7, and 6/8, with subsequent imaging on 6/4 revealing a 4mm pseudoaneurysm in the cavitary lesion. Additional coil embolization procedures were performed on 6/7; however, new pseudoaneurysms were identified on 6/11, necessitating further IR interventions on both 6/11 and 6/12. Despite these procedures, massive hemoptysis continued until a final coiling procedure on 6/19 successfully stabilized the patient. After this procedure, the patient's condition improved significantly, allowing for decannulation from ECMO and mechanical ventilation. The patient was eventually discharged to a rehabilitation center on 8/13/2024, with planned discharge home on 9/3/2024.

**Discussion:** This case emphasizes the severity of Rasmussen aneurysms as a rare but dangerous complication of cavitary pneumonia. Despite the critical role of CT imaging in diagnosis, negative results do not rule-out their presence, especially in patients with hemoptysis. Recurrent bleeding is common even after successful initial embolization due to the formation of new aneurysms, underscoring the need for heightened clinical suspicion and repeat imaging in such cases. Prompt management through embolization or surgical resection is essential to prevent life-threatening hemorrhage. This case also highlights the need for further research into early recognition, improved diagnostic techniques, and more effective treatment options to reduce mortality and improve outcomes for affected patients.



<p><b>Maria Santiago Estevez</b>  Dr. Rebecca Brooks  Dr. Leticia Rolon</p>	<p><i>A Tale of MPGN</i></p> <p>Introduction: MPGN is glomerular injury characterized by mesangial deposition and endocapillary proliferation. MPGN can present as a diverse spectrum of syndromes, including nephrotic syndrome, nephritic syndrome, rapidly progressive glomerulonephritis, or even asymptomatic proteinuria and hematuria. There are both idiopathic and secondary causes of MPGN that also differ in treatment. In this case report, we investigate the unique presentation of idiopathic MPGN in a transplant kidney recipient.</p> <p>Case Presentation: A 23-year-old Hispanic female presented with a stage 3 acute kidney injury (AKI) given a creatinine of 6.2 mg/dL in 2002. Renal biopsy showed MPGN of unclear etiology. She was diagnosed with immune-complex mediated MPGN and was treated with steroids and cyclophosphamide. Despite treatment, her creatinine increased to 7.1 mg/dL and she was initiated on hemodialysis. Fortunately, she received a living related renal transplant in 2002 and immunosuppression was transitioned to cyclosporine and mycophenolate. Over the following years, the patient's medical history was complicated by bilateral sensorineural hearing loss, uveitis, celiac disease, and renal cell carcinoma requiring a bilateral native kidney nephrectomy. Additionally, she had multiple UTIs and admissions for pyelonephritis that led to temporary elevations in creatinine but with return to her baseline of 1.0 mg/dL following treatment.</p> <p>During an evaluation for a UTI in early 2024, she was noted to have hypertension. She was treated with cefdinir and initiated on nifedipine and carvedilol. However, both were discontinued due to side effects. Following over six months without treatment, she was able to tolerate amlodipine. In July 2024, she presented with tenderness at the site of kidney transplant and lower extremity edema. Evaluation showed a creatinine of 2.60 mg/dL elevated from a recent baseline of 1.3-1.4 mg/dL and a urinalysis showing 180 WBC/HPF, positive leukocyte esterase and nitrites. She received a dose of ceftriaxone and was discharged with outpatient follow up. Over the following days, her symptoms worsened, and she was admitted to the hospital. Initial evaluation was notable for a creatinine of 3.0 mg/dL and 24-hour urine protein of over 13 grams. Evaluation of autoimmune, infectious, and malignant causes was unrevealing. Due to continued rise in creatinine, a renal allograft biopsy was pursued revealing recurrent MPGN with acute tubular injury. Immunofluorescence studies showed IgG, IgM, C1q, C3, kappa and lambda chain deposits suggestive of autoimmune disease. She was initiated on Solumedrol for 3 days, followed by prednisone along with an increased dose of mycophenolate. Her blood pressure was managed with torsemide, nifedipine, and carvedilol. Upon discharge, her creatinine had stabilized to 4.12 mg/dL. At follow-up, her creatinine had continued to improve to 2.39 mg/dL.</p> <p>Conclusion: Recurrence of MPGN following transplant can be masked by frequent UTIs in transplant recipients due to the variety of presentations that include hematuria and pyuria on urinalysis, thus confounding the clinical picture. This case demonstrates the importance of evaluating rises in creatinine and the development of resistant hypertension in recipients of kidney transplants aggressively in close collaboration with transplant nephrologists. Early recognition of re-occurrence of MPGN could have</p>
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	prevented the worsening of the patient’s kidney function and multiple hospitalizations.
<p><b>Zachary Scharf</b>  Dr. Maryam Own  Dr. Elena Joerns</p> <p>Finalist</p>	<p><i>Diagnostic Dilemma: An Atypical Case of Macrophage Activation Syndrome</i></p> <p>Case Presentation: A 69-year-old woman presented with undifferentiated connective tissue disease characterized by recurrent anasarca and pleural effusions with ANA+ serologies. She was on hydroxychloroquine, mycophenolate, and prednisone. Her course was complicated by painful lower extremity ulceration with pseudomonas sepsis for which she was admitted. After the patient was admitted and initiated on antibiotics, she was noted to have worsening anasarca with bilateral pleural effusions on imaging, necessitating bilateral chest tube placement. Despite antibiotics, the patient had profound hypotension and decompensated into shock with biventricular failure (LVEF dropped to 25% from a previously recorded 67%). She was transferred to the ICU and required profound inotropic and vasoconstrictor support with veno-arterial ECMO cannulation. Her workup revealed hypoalbuminemia and a positive MGUS. Given the patient’s profound anasarca in addition to hypotension with intact mentation, MGUS, and hypoalbuminemia, a preliminary diagnosis of Clarkson’s disease (idiopathic systemic capillary leak syndrome) was made, and she was treated with IVIG infusions 2 mg/kg over two days. Later, her blood cultures grew pseudomonas. She had initial improvement; however, she developed pancytopenia with platelet levels that were refractory to transfusions. PLEX was initiated with some improvement in her platelets. Given her persistent pancytopenia, a bone marrow biopsy was obtained and revealed increased histiocytes and hemophagocytic cells consistent with hemophagocytic lymphohistiocytosis (or potentially macrophage activation syndrome given underlying rheumatic condition and elevated H Score). For this, she was initiated on IV Anakinra 100 mg every 6 hours. Bronchoscopy with bronchoalveolar lavage was performed prior to an unsuccessful extubation attempt and returned positive for Aspergillus antigen and a few Mucor species. The patient never grew Aspergillus but was empirically treated with posaconazole and amphotericin B.</p> <p>In the days following initiation of this therapy, the patient’s thrombocytopenia resolved. She was successfully decannulated from ECMO and had decreasing output from her bilateral chest tubes, which were soon removed. Her biventricular dysfunction resolved, and she underwent a successful tracheostomy procedure. Her mental status normalized, and she was transferred from the ICU to the general medicine floor. She completed a 10-day course of Anakinra and was continued on hydroxychloroquine with outpatient Rheumatology follow up.</p> <p>Conclusion: Hemophagocytic lymphohistiocytosis is a life-threatening syndrome involving excessive immune activation. The hyperinflammatory immune state that results is believed to be caused by the absence of normal downregulation by macrophages and lymphocytes. While it can have many possible triggers, infection is a common cause. Macrophage activation syndrome refers to a form of secondary hemophagocytic lymphohistiocytosis that occurs in patients with rheumatological disease. It is unclear if infection, underlying rheumatic condition, or both were the precipitating factor in this case. However, treatment with Anakinra quickly</p>

	<p>improved this patient’s clinical status. Therefore, this case displays the importance of considering macrophage activation syndrome in a patient with underlying rheumatologic disease who is acutely ill. If recognized, appropriate treatment with a disease-modifying therapy, such as an IL-1R antagonist, could prevent a life-threatening syndrome from progressing. For long-term therapy, continuation of Anakinra for what appears to be serositis can be considered.</p>
<p><b>Reid Schlesinger</b>  Dr. Nathaniel Davis  Dr. Justin Carrington  Dr. Jay Roth  Dr. Lindsey Wanberg  Dr. James Gregoire</p>	<p><i>A Rare Presentation of Scleroderma Renal Crisis</i></p> <p>Case Presentation: An 81-year-old woman with long-standing limited cutaneous systemic sclerosis overlap syndrome (lcSSc-OS) presented with shortness of breath after months of treatment-resistant hypertension and progressive kidney dysfunction. Her lcSSc-OS symptoms had been stable for decades on low-dose prednisone, with clinical manifestations including sclerodactyly, telangiectasias, heartburn, arthritis, and sicca symptoms. Her antibody profile showed a high-titer nucleolar anti-nuclear antibody (1:5120) and positive SSA/Ro, SSB/La, and anti-PM/Scl-100 antibodies; anti-centromere, anti-RNA polymerase III, and anti-Scl-70 were negative. Eight months before admission, she developed new-onset severe hypertension (systolic &gt;200 mmHg), with a rise in her creatinine from baseline 0.65 mg/dL to 0.90 mg/dL. Treatment with lisinopril and furosemide partially reduced her systolic blood pressure to 140 mmHg.</p> <p>Over the ensuing months, she was hospitalized three times for hypertensive urgency and worsening kidney function; creatinine rose to 2.25 mg/dL. Despite aggressive management with numerous antihypertensives, her blood pressure remained elevated. On admission, blood pressure was 170/60 mm Hg, and she showed signs of volume overload, including bilateral pleural effusions. Labs showed creatinine 2.5 mg/dL, BUN 70 mg/dL, and elevated cystatin C 4.54 mg/L, indicating significant kidney impairment disproportionate to her creatinine given low muscle mass. Urinalysis revealed sub-nephrotic proteinuria with bland sediment. Comprehensive evaluation for secondary hypertension was unremarkable. A kidney biopsy revealed severe vascular microangiopathy with subacute and chronic glomerular changes consistent with scleroderma renal crisis (SRC). She was managed with maximized doses of lisinopril and other antihypertensives. Despite treatment, kidney function continued to decline, necessitating hemodialysis. She was discharged home on dialysis, with plans to monitor for potential kidney recovery.</p> <p>Conclusion: SRC is a rare but life-threatening complication of systemic sclerosis, occurring in about 5% of patients. It is most commonly associated with diffuse cutaneous SSc (dcSSc) and typically presents within five years of diagnosis. Risk factors include anti-RNA polymerase III antibodies, recent high-dose corticosteroid use, and rapidly progressing skin involvement. The pathogenesis involves obliterative vasculopathy of renal arterioles, leading to ischemia and acute kidney injury.</p> <p>The SRC presentation in this case was atypical due to her long-standing lcSSc-OS, negative anti-RNA polymerase III antibodies, and subacute progression over months. She did not exhibit worsening sclerodactyly but developed interstitial lung disease, indicating disease progression. The bland</p>

urinalysis was consistent with SRC, and the kidney biopsy was crucial in establishing the diagnosis. The subacute and chronic findings on kidney biopsy suggests damage occurred over time, limiting reversibility, possibly explaining her lack of response despite appropriate ACE inhibitor therapy. Early recognition is critical for improving outcomes in SRC; however, in subacute cases, irreversible damage may have already occurred at diagnosis.

This case underscores that SRC can occur in lcSSc patients after decades of stable disease without typical risk factors. Clinicians should maintain a high index of suspicion for SRC in any SSc patient with new-onset hypertension and kidney dysfunction. Recognizing atypical presentations is essential for timely intervention, which may improve prognosis and preserve kidney function.

**Jessica Seledotis**

*Case of Metastatic Hepatocellular Carcinoma to the Brain*

Introduction: Hepatocellular carcinoma (HCC) accounts for 75% of primary liver cancer in the world, and primary liver cancer is the second most common cause of cancer mortality (1). Risk factors for HCC commonly include viral hepatitis and alcohol-related liver disease, and now metabolic dysfunction-associated liver disease is increasingly being recognized as a major risk factor for HCC (2). Because of the broad risk factors and growing screening efforts, patients with HCC are surviving longer, and therefore sequelae, including rare sites of metastatic disease, are increasingly important to recognize.

Case Presentation: A 63-year-old male with a past medical history of HCC due to chronic hepatitis B infection and later development of metastases to the lungs, MGUS, serrated adenoma treated with right hemicolectomy, and end stage renal disease on dialysis, was admitted for new onset generalized tonic-clonic seizure. The seizure occurred after patient returned home from dialysis, and patient’s family note that he had been having some behavioral changes and leaning more to his left side in the few weeks prior.

Methods: Patient was intubated for airway protection and underwent head CT demonstrating a right frontal brain mass with internal hemorrhage and vasogenic edema. He was admitted to the ICU, started on corticosteroids and levetiracetam. Oncology and Neurosurgery were consulted. Given patient had an ECOG score of 1, with previous excellent response to therapy for HCC, as well as a single intracranial lesion, the patient underwent right frontal craniotomy and tumor resection on hospital day 2 without post-operative complications. Pathology results were consistent with metastatic HCC to the brain. He underwent stereotactic radiation surgery and was restarted on bevacizumab/atezolizumab. Patient has not had any recurrence of brain metastatic disease and continues on immunotherapy for HCC.

Conclusion: Brain metastases from HCC are rare, portend a poor prognosis, and have a high risk of severe complications including intracranial hemorrhage. Surgical resection is considered in patients with a single or few lesions when systemic disease is well controlled and with good performance status, as in the case of this patient. Surgical management has been shown to provide a significant survival benefit when compared to non-surgical treatments or supportive care (3). This case demonstrates an example of a

	rare sequela of HCC which is clinically relevant as both the risk factors and survival of HCC are increasing.
<p><b>Matthew Semler</b> Dr. Mary Fredrickson</p>	<p><i>A Rare Case of Dermatomyositis</i></p> <p>Introduction: Dermatomyositis is an uncommon cause of muscle inflammation typically presenting with a rash involving the periorbital region, shoulders, chest, and hands. While commonly associated with malignancy, we present a case of dermatomyositis with proximal muscle weakness and atypical cutaneous findings without an associated malignancy or autoimmune condition.</p> <p>Case Presentation: A 29-year-old previously healthy male was admitted with one month of progressive, bilateral proximal muscle weakness and multiple pruritic, erythematous patches. He endorsed fever and fatigue two days prior to developing weakness with night sweats, abdominal cramping and a 20-pound unintentional weight loss. Physical exam was notable for 3/5 strength in the bilateral proximal upper extremities, 2/5 strength in the bilateral proximal lower extremities, and erythematous patches on the abdomen, left deltoid, and right medial thigh. No rashes were present in the periorbital region, neck, shoulders or hands. Initial labs were notable for a CK 10,592, ESR 23, CRP 0.8, and a troponin of 0.19 with no EKG changes. ANA, C4 and Anti-cardiolipin IgM were elevated, but his myositis antibody panel was negative. CT chest, abdomen and pelvis was negative for malignancy but demonstrated colitis and a developing colovesicular fistula. Follow up colonoscopy showed diffuse inflammation in the sigmoid colon and a sigmoid stricture. Colon biopsies showed focal active colitis negative for Crohn’s disease. MRI of the bilateral femurs showed intramuscular edema that was concerning for myositis. Left thigh muscle biopsy showed increased dermal mucin and mild perivascular lymphocytic inflammation consistent with dermatomyositis. He was started on high dose steroids and received one dose of IVIg. Despite this, his CK remained elevated to 5,000. Serum and urine electrophoresis were normal and PET scan demonstrated diffuse musculature uptake consistent with myositis, but again no malignancy. He was ultimately discharged on prednisone and azathioprine. Follow up colonoscopy 2 years later showed chronic colitis, but again no Crohn’s disease or evidence of malignancy. Currently, he is controlled on azathioprine, methotrexate and IVIg every two weeks with plan for sigmoidectomy to address his sigmoid stricture.</p> <p>Conclusion: Our patient represents an atypical case of idiopathic dermatomyositis. Pathognomonic cutaneous findings such as Gottrons papules and a heliotrope rash were absent. Additionally, less common findings like dystrophic cuticles, Holster sign, poikiloderma of the hips, and scalp involvement were also not present. Instead, he presented with only muscle weakness and asymmetric erythematous patches. Although commonly associated with malignancy such as lung, stomach, colorectal and pancreatic cancers in males, dermatomyositis may be idiopathic and not associated as a paraneoplastic syndrome. Our patient is over 2 years from his diagnosis with no known malignancy or diagnosis of associated inflammatory bowel disease. Finally, our patient had a negative myositis antibody panel which does not rule out dermatomyositis. The combined antibodies have an overall sensitivity of 68% and specificity of 94%. This case</p>

	<p>highlights the value of MRI and muscle biopsy to obtain an accurate diagnosis.</p>
<p><b>Danuzia Silva</b> Dr. Sarah Chalmers</p>	<p><i>Hyperbaric Oxygen Therapy: A Salvage Treatment for SSTIs Secondary to DRESS Syndrome</i></p> <p>Introduction: The promotion of wound healing via Hyperbaric Oxygen Therapy (HBOT) is multifactorial<sup>1</sup> and encompasses enhancing angiogenesis, exerting antimicrobial effects and promoting immunomodulatory effects that decreased inflammation to tissues. The evidence for the efficacy of HBOT for the management of Dress Syndrome is scant and comprised mostly of case reports<sup>2</sup>. However, this therapy is commonly used as a standard of care for the treatment<sup>1</sup> of Clostridium myositis/myonecrosis, refractory osteomyelitis, Fournier’ gangrene and other necrotizing soft tissue infections.</p> <p>Case Description: 23 years-old male with Common Variable Immunodeficiency Syndrome on chronic IVIG with extensive enteropathy and malnutrition undergoing treatment for septic shock from primarily MSSA bacteremia. Hospital course was complicated by COVID 19 infection with superimposed pneumonia requiring mechanical ventilation, central line-associated Pseudomonas aeruginosa bacteremia, multiple skin soft tissue infections (SSTI) with necrosis positive for Vancomycin-Resistant Enterococci requiring surgical debridements, acute cholecystitis with bile culture positive for Enterococcus faecium resistant to Daptomycin status post cholecystostomy tube placement, peritonitis secondary to Candida parapsilosis infection, acute renal failure requiring intermittent HD, anemia status post multiple packed RBC transfusions, hepatocellular lesions concerning for adenoma versus carcinoma, and diffuse whole body morbilliform desquamative rash and erythroderma status post skin biopsy consistent with DRESS Syndrome considered to be secondary to Beta-Lactam (Zosyn, Cefazolin, Cefepime and Nafcillin) and Glycopeptide (Vancomycin) antibiotic therapy.</p> <p>Patient was admitted to the ICU upon hospital presentation for the treatment of septic shock with Norepinephrine, Vasopressin, Angiotensin II, Stress Dose Steroids, Acetaminophen, Meropenem, Levofloxacin, Vancomycin and Micafungin as cultures were positive for MSSA, Pseudomonas aeruginosa and Candida parapsilosis. Based on patient’s clinical improvement, he transitioned to Cefazolin followed by Cefepime with Micafungin and transferred out of the ICU. There was concern for recurrence of septic shock and patient received Zosyn, Vancomycin, Meropenem, and Micafungin before readmission to the ICU. At the ICU, he continued therapy with Micafungin, started on Metronidazole, Daptomycin and Ciprofloxacin. He developed a diffuse morbilliform desquamative rash deemed to be secondary to Vancomycin and Beta-Lactam antibiotics by Dermatology and Infectious Diseases specialists given eosinophilia and skin biopsy results. Thus, patient transitioned to Linezolid and Micafungin. He transiently improved, but had recurrence of Pseudomonas bacteremia, high fevers despite antipyretic therapy, persistent tachycardia and worsening of desquamative rash, leading to initiation of Meropenem and Fluconazole. Hyperbaric Oxygen Therapy was initiated to aid in the management of SSTIs and promote resolution of desquamative rash, given limited antibiotic</p>

	<p>options, recurrence of bacteremia and concern with evolution to septic shock.</p> <p>Discussion: As the occurrence of multiple antibiotic allergies becomes more prevalent and resistance to multiple antibiotic therapies continues to rise, the utilization of hyperbaric oxygen therapy for the management of SSTIs in immunocompromised patients may offer an alternative therapy for the management of these patients.</p> <p>References</p> <p>1: Ortega MA, Fraile-Martinez O, et al. A General Overview on the Hyperbaric Oxygen Therapy: Applications, Mechanisms and Translational Opportunities. <i>Medicina (Kaunas)</i>. 2021 Aug 24;57(9):864. doi: 10.3390/medicina57090864.</p> <p>2: Alkhateeb H, Said S, et al. DRESS syndrome following ciprofloxacin exposure: An unusual association. <i>Am J Case Rep</i>. 2013 Dec 4;14:526-8. doi: 10.12659/AJCR.889703.</p>
<p><b>Grant Simonson</b> Dr. Jeremy Taylor</p>	<p><i>False Sense of Sensitivity: An Initially Reassuring Presentation of Acute on Chronic Mesenteric Ischemia</i></p> <p>Case Presentation: A 74-year-old man with a medical history including hypertension, type II diabetes mellitus, multivessel coronary artery disease, ischemic cardiomyopathy, atrial fibrillation on long-term anticoagulation, and peripheral arterial disease presented to the emergency department of a community hospital with acute postprandial lower abdominal pain and nausea. He described the pain as severe and unlike anything he had experienced before.</p> <p>Vital signs on arrival were normal, and physical examination demonstrated a comfortable appearing man with mild lower abdominal tenderness on deep palpation. Laboratory findings were notable only for leukocytosis (14,300/mm<sup>3</sup>) and mildly elevated erythrocyte sedimentation rate (57 mm/hr). There were no elevations in lactic acid (&lt;1.6 mmol/L), lactate dehydrogenase (218 IU/L), C-reactive protein (5.7 mg/L), transaminases, lipase, or creatinine. Initial CT of the abdomen &amp; pelvis with contrast showed no abnormality to explain his pain. There was low suspicion for bowel ischemia given these results, and the patient was treated with IV analgesia with the intent to discharge home. His pain did not improve however, and he was admitted for observation. Lactic acid remained normal on two further law draws 6 hours apart (1.2 and 1.6 mmol/L). After several hours of ongoing pain despite high-dose multimodal pain control, CT angiography of the abdomen was obtained revealing severe stenosis of the celiac, superior mesenteric, and inferior mesenteric arteries along with pneumatosis of the small bowel wall. The patient was taken urgently to the operating room for vascular stenting and exploratory laparotomy with small bowel resection, during which a roughly 25 cm segment of frankly necrotic ileum was observed.</p> <p>Conclusion: This case highlights the imperfect sensitivity of various common lab markers and imaging modalities for mesenteric ischemia. A high index of suspicion for acute on chronic mesenteric ischemia is required in similar</p>

	<p>patients with documented arterial disease even if an initial workup and exam are reassuring.</p>
<p><b>Alexandra Skovran</b></p>	<p><i>Unveiling the Dark Side of Statins: Immune-Mediated Necrotizing Myopathy</i></p> <p>Introduction: Statins are a main class of medications used to decrease cholesterol for the prevention of cardiovascular disease. They are typically well tolerated as a class but a rare side effect, seen in 2-3 per 100,000 patients, is statin-induced immune-mediated necrotizing myopathy (IMNM). 1.</p> <p>Case presentation: A 48-year-old male with past medical history of diabetes mellitus, hypertension, hyperlipidemia, and previous stroke with residual left sided weakness presented to outside facility with fatigue, weakness and decreased appetite. On exam, he was noted to have bilateral weakness, unable to lift left arm or right leg against gravity. Other notable labs were troponin elevation to 1200, later found to be troponin T elevation, creatinine kinase (CK) elevated to 17K and transaminitis. His statin medication was stopped on admission but his CK continued to rise, peaking at 18K. He was then noted to have ANA 1:320 and was transferred to a facility with rheumatology. He then underwent an MRI of the right thigh that showed diffuse intramuscular edema and prompted a muscle biopsy. Unfortunately, he developed severe sinus bradycardia that delayed his procedure so the decision was made to start him on methylprednisolone empirically. With this, his CK began to downtrend and he was switched to oral prednisone 5 days later. His muscle biopsy eventually came back with findings of “numerous myofibers are seen undergoing necrosis, without additional myopathic features. There are no inflammatory infiltrates. Phosphorylase A and Adenosine Monophosphate Deaminase (AMPDA) are present. There is no increase in intramuscular lipids.” A myositis panel resulted with autoantibodies against 3-hydroxy-3-methylglutaryl-coenzyme A (HMG-CoA) reductase thus confirming the diagnosis of statin-induced immune-mediated necrotizing myopathy. His CK downtrended as did his transaminases and he was discharged to an inpatient rehab facility.</p> <p>Discussion: Statin-induced immune-mediated necrotizing myopathy is a rare side effect of statin medications. It is characterized by severe proximal muscle weakness, myofibril necrosis, elevated CK and autoantibodies against anti-HMG-CoA reductase. The exact mechanism is unknown but there is thought to be a genetic component. Typical treatment includes immediate discontinuation of statin, immunosuppression with glucocorticoids and intravenous immunoglobulin (IVIG) with therapies typically continuing for at least 1-2 years.</p> <p>Dixit A, Abrudescu A.. A case of atorvastatin-associated necrotizing autoimmune myopathy, mimicking idiopathic polymyositis. Case Rep Rheumatol. 2018;2018:5931046. doi: 10.1155/2018/5931046.</p>
<p><b>Dakota Snustad</b> Dr. Cutler Lewandowski</p>	<p><i>Left Ventricular Pseudoaneurysm Rupture Presenting in a Stable Patient with a Two-Day History of Chest Pain</i></p> <p>Case Presentation: We present the case of a 64-year-old male with a past medical history of abdominal aortic aneurysm status post endovascular</p>



	<p>aortic repair, carotid dissection, ischemic stroke, patent foramen ovale with hypoxia status post closure, coronary artery bypass grafting x3 in 2021, COPD and a neurocognitive disorder with memory deficits who presented to the Emergency Department (ED) with a two-day history of progressive left-sided chest pain and was found to have a left ventricular pseudoaneurysm rupture with hemopericardium which was subsequently surgically repaired. The patient presented to the ED following two days of a worsening, paroxysmal, squeezing left-sided chest pain that radiated down his left arm. Upon initial presentation, the patient’s vital signs were stable, and no abnormal findings were present on physical exam. Labs were significant for elevated high sensitivity troponin of 6,530ng/L, and his ECG revealed normal sinus rhythm with minor ST depressions in leads V2-V4 and a right superior axis deviation. A CTA chest was ordered in the ED and revealed a perforation with a large defect in the inferolateral wall of the left ventricle with associated extraluminal contrast extravasation and moderate hemopericardium. The patient underwent a coronary angiogram that showed patent bypass grafts, followed by a re-do sternotomy with left ventricular pseudoaneurysm repair using a double patch technique with bovine pericardium. Following a post-operative hospital stay complicated by hypervolemia, E. coli ventilator-associated pneumonia, and atrial fibrillation with rapid ventricular response, the patient was discharged home following a 17-day hospital course.</p> <p>Conclusion: Left ventricular pseudoaneurysms can present with nonspecific symptoms, and up to 10% of cases may be asymptomatic. They typically occur as complications 3-14 days after an acute myocardial infarction, cardiac surgery, or endocarditis, but they can also be seen up to 12 months post-myocardial injury. They form when necrotic myocardium is remodeled to thin fibrous scar tissue by macrophage activity in the necrotic tissue. In this case, it is possible that the patient had a recent myocardial infarction that resulted in the pseudoaneurysm formation, but it seems more likely the patient developed the pseudoaneurysm and rupture in response to his prior cardiac insults and procedures. Because the formation of a pseudoaneurysm is an inflammatory and pro-thrombotic process, potential wall ruptures can be initially contained by clotting and adhesions, which is likely what occurred in this case, allowing the patient to remain vitally stable long enough to receive definitive surgical treatment. Patients who develop left ventricular pseudoaneurysms are at risk for arrhythmia, clot formation, heart failure, and life-threatening left ventricular free wall rupture and strokes. This case highlights the nonspecific symptoms with which patients with a left ventricular pseudoaneurysm can present, the myocardial remodeling that occurs following a myocardial injury, and the importance of regular follow up of patients following myocardial injury.</p>
<p><b>Paul Strain</b> Dr. Evelyn Shen</p>	<p><i>Lights, Camera, Actinomyces: An IUD Associated Infection!</i></p> <p>Introduction: Actinomyces infections can be severe with abscess formation, fistulae, and draining sinus tracts. However, Actinomyces is also a part of normal gastrointestinal flora; asymptomatic female genital colonization is not uncommon. Actinomyces may be an incidental finding on Pap smear in women with intrauterine devices (IUDs). However, active infection must be considered if the patient is symptomatic. This clinical vignette describes an unexpected Pap smear finding of Actinomyces in a symptomatic patient.</p>

	<p>Case Presentation: 60-year-old woman with a history of type 2 diabetes, hypertension, depression, moderate persistent asthma, pruritus nodularis, multinodular thyroid s/p thyroidectomy who presented for pelvic pain. She was postmenopausal with last menstrual period at age 45. Upon chart review, it was determined that an IUD was placed prior to beginning therapy with dupilumab for pruritus nodularis 15 years ago. The IUD was removed in clinic and cervical cytology and HPV testing was performed for routine cervical cancer screening. The pathologist noted bacteria consistent with actinomyces on HPV cytology. The IUD was discarded prior to this result and IUD culture was not obtained. The patient was called at home to disclose results and described experiencing fevers, chills, and body aches for the past few days. She was instructed to go to the ER for further evaluation.</p> <p>On initial presentation she was afebrile, hemodynamically stable and nontoxic appearing. Exam was notable for lower quadrant abdominal pain and cervical motion tenderness. Pelvic ultrasound and CT abdomen pelvis were unremarkable. WBC was 6.7. Vaginal swab grew gram positive bacilli on gram stain and culture was positive for 1+ Morganella morganii. Actinomyces specific culture was sent on the same sample but was negative. Infectious Disease was consulted; given the vaginal culture and previous cytology results they recommended treatment for presumed mild cervical/vaginal Actinomycosis for a minimum duration of 2 weeks. The patient was admitted for observation and remained afebrile with improvement in symptoms. She was discharged home on oral amoxicillin 1g q8h with ID follow up.</p> <p>Discussion: In the absence of pelvic inflammatory disease (PID) symptoms, IUD removal and Actinomyces treatment are typically not necessary. However, in a patient with symptoms concerning for PID and an incidental finding of Actinomyces on Pap smear, the IUD should be removed. In this case, the IUD was removed prior to the Pap smear finding as the patient had no indication for contraception but was symptomatic. A cervical culture should be done for confirmation and imaging should be obtained to evaluate the extent of disease. If imaging is suggestive of invasive disease, then targeted therapy consists of intravenous penicillin followed by oral therapy until 1-2 months after radiographic resolution of disease. If imaging is benign, as in this case, then it is likely an early stage IUD-associated infection which warrants a shorter duration of directed treatment.</p> <p>Given that Actinomyces is known to readily spread across anatomic tissue planes, there should be a low threshold for further workup with incidental findings, particularly with symptomatic patients.</p>
<p><b>Agata Sularz</b>  Dr. Stefan Farrugia  Dr. Richard Elias  Dr. Meltiady Issa</p>	<p><i>Recurrent Metastatic Breast Cancer Mimicking Acute Decompensated Cirrhosis</i></p> <p>Case Presentation: An 83-year-old woman presented to the Emergency Department with three weeks of lower back and abdominal pain, lower extremity swelling, and shortness of breath on exertion. Her medical history included bilateral estrogen-positive invasive ductal breast cancer, treated with bilateral lumpectomy and radiation a year prior, along with mild right-sided heart failure, hypertension, hyperlipidemia, and obesity. She denied</p>

any history of liver disease or significant alcohol consumption.

On admission, the patient was hypoxic, requiring 3L of oxygen. Physical examination demonstrated jaundice, limb ecchymosis, abdominal distension with shifting dullness and positive fluid wave, and peripheral edema extending to the abdominal wall. Chest X-ray showed bilateral pleural effusions. Laboratory results revealed thrombocytopenia (Plt  $72 \times 10^9/L$ ), elevated liver enzymes (AST 145 U/L, ALT 72 U/L), direct hyperbilirubinemia (total bilirubin 2.5 mg/dL), elevated creatinine (1.47 mg/dL; baseline 1 mg/dL), and INR of 1.6. CT imaging demonstrated moderate ascites, coarse liver echotexture, and diffuse retroperitoneal lymphadenopathy. Transthoracic echocardiogram showed a preserved ejection fraction (65%), moderate right ventricular impairment, and diastolic dysfunction. A paracentesis removed 1.5L of fluid consistent with portal hypertension (SAAG 2.0 g/dL, protein 1.7 g/dL, PMN 16/mcL). Abdominal ultrasound confirmed coarse liver echotexture with small hypoechoic foci, and patent portal and hepatic veins with reversed portal flow. Autoimmune and infectious hepatitis serologies were negative.

Initially, she responded to IV furosemide, but developed encephalopathy, which improved with lactulose. Her renal function worsened, raising concerns about hepatorenal syndrome. IV albumin was administered, leading to renal recovery with subsequent resumption of diuresis. The patient's liver function continued to decline, with INR 2.0 and rising bilirubin. Worsening thrombocytopenia (Plt  $35 \times 10^9/L$ ) prompted a disseminated intravascular coagulation (DIC) evaluation, which was positive, with low fibrinogen, elevated D-dimer, and prolonged aPTT. Cytology from peritoneal and pleural fluid demonstrated metastatic breast adenocarcinoma, raising concerns for a visceral crisis with pseudocirrhosis. A PET-CT scan revealed diffuse metastatic uptake in the bone marrow, lymph nodes, and liver. A bone marrow biopsy confirmed metastatic carcinoma with hypocellularity. Due to coagulopathy, a liver biopsy was not performed. After discussion with the patient and her family, the patient was transitioned to hospice care.

Case Discussion: Pseudocirrhosis is a cirrhosis mimic, characterized by multifocal scarring, stromal retraction, and compensatory hyperplasia of interval parenchyma, most commonly in the context of metastatic breast cancer. The resultant changes in hepatic contour and nodularity resemble cirrhosis radiographically. Further confounding the diagnosis is that it can present with ascites and hepatic dysfunction, similar to acute decompensated cirrhosis. Typical histological findings on liver biopsy are diffuse metastatic infiltration of the liver and desmoplastic reaction leading to the distortion of hepatic architecture but with otherwise normal liver parenchyma. Here the diagnosis was suggested by imaging findings, malignant cells in peritoneal fluid, liver avidity on PET-CT, and worsening liver dysfunction with DIC. The diagnosis of pseudocirrhosis should be considered in patients with a history of treated locoregional breast cancer presenting with findings resembling acute decompensated cirrhosis. Timely diagnosis is critical because sustained clinical improvement relies on effectively treating the underlying malignancy.

<p><b>Lyubov Tiegs</b>  Dr. Taylor Bowler  Dr. Bryant Megna  Dr. Mauricio  Torrealba Alonso</p>	<p><i>Sapphire Shadows: A Case Series Describing Unusual Ileal Discoloration in a Veteran Population</i></p> <p>Introduction: Existing literature has described the presence of black granular pigment deposits in the base of Peyer patches of the terminal ileum, presumed to be secondary to macrophage uptake of microparticles. However, large spots of pigmentation grossly visible on routine colonoscopy with ileal intubation have not been reported.</p> <p>Herein, we present four cases of visible blue pigmentation in the ileum of curious etiology. Between January and April 2024, we encountered four cases of macroscopically evident pigmentation of the ileum. In all cases, the patients underwent colonoscopies with ileal inspection during which multiple patches with a blue hue were identified in the terminal ileum. In all cases, histopathology described dark clusters of pigmented granules deep in the Peyer’s patches. The iron staining was negative, and the pigment was inert, and therefore unlikely to be of clinical significance. The patients were subsequently interviewed, and their records examined revealing the following commonalities. All patients were veterans, their service times including the Vietnam and post-Vietnam era. Although they served in different areas, on interview they endorsed exposure to lead paint during their service time, as well as exposure to guns, grenades and other weapons during basic training. There was no obvious overlap of dietary trends. The patients denied the use of non-prescribed medicines or supplements and denied ingestion of dark-dyed foods. In all four cases, histopathology identified pigment granules in the deep portions of the Peyer’s patches. Pathology reports described that the pigment accumulated within macrophages and has been shown by x-ray spectroscopy to contain mineral composition that includes silicates, aluminum, and titanium, with the most plausible explanation being of atmospheric or dietary source.</p> <p>Conclusion: Literature review revealed that titanium dioxide serves as an opacifier and stabilizer in pharmaceuticals and is a major component of training grenades. Aluminosilicates are used in the pharmaceuticals as adsorbents and demulcents and in military operations. Ultimately, the primary cause of blue patches in the ileum remains unclear, but given the military background of the patients, a training or war-related exposure could be presumed. While unlikely clinically harmful, it is important to know that these findings can be present in the veteran population during endoscopic evaluation.</p>
<p><b>Lane Uhing</b></p>	<p><i>A Less-Than-Crystal-Clear Cause of Acute Renal Failure in a Patient with Acute Diarrhea and Previous Bariatric Surgery</i></p> <p>Case Presentation: A 64-year-old man with stage 3b chronic kidney disease, ischemic cardiomyopathy, and previous bariatric surgery with biliopancreatic diversion with duodenal switch (BPD/DS) presented for orthostasis and reduced urine output; he was admitted to the hospital with acute renal failure. A coronary angiogram without intervention had been performed for a non-ST segment elevation myocardial infarction two weeks prior to presentation. He had developed acute diarrhea from enterotoxigenic Escherichia coli one week before admission. He continued to take antihypertensive and diuretic medications and also used NSAIDs</p>

	<p>during the diarrheal illness. Initial vital signs and physical exam were unremarkable. Serum creatinine was 7.79 mg/dL, elevated from a baseline of 1.3 – 1.6 mg/dL. Urinalysis showed calcium oxalate crystals with 4-10 white blood cells (1-5% eosinophils) and 1-3 renal epithelial cells per high power field. Renal ultrasound and a noncontrast CT scan revealed nonobstructing nephrolithiasis, normal renal arteries, and no hydronephrosis. Renal failure progressed; creatinine rose to 9.96 mg/dL while worsening metabolic acidosis and hyperphosphatemia ensued. Hemodialysis was initiated.</p> <p>Several differential diagnoses were entertained, including acute tubular necrosis from renal hypoperfusion, cholesterol embolization and contrast-induced nephropathy from the coronary angiogram, medication-related acute interstitial nephritis, and oxalate nephropathy. A plasma oxalate level was obtained and returned markedly elevated at 37.5 mcmol/L. A kidney biopsy specimen demonstrated an abundant number of intratubular calcium oxalate crystals associated with acute tubular injury, and a diagnosis of oxalate nephropathy was made. He was treated with a low oxalate diet, calcium acetate, and several sessions of hemodialysis. Renal function recovered and he discharged home off hemodialysis. A plasma oxalate level was 15.8 mcmol/L three days after hospitalization, and serum creatinine was 1.86 mg/dL three months after hospitalization.</p> <p>Discussion: Acute renal failure is one of the most common problems faced by internists. Oxalate nephropathy is an uncommon and underrecognized cause of acute renal failure. Because of its implications for patient outcomes, the internist should consider this condition in a compatible clinical scenario. Malabsorptive conditions, including BPD/DS bariatric surgery, are associated with enteric hyperoxaluria. High dietary oxalate consumption also contributes to hyperoxaluria; this patient later endorsed intake of the oxalate-rich foods cocoa and rhubarb. Infectious diarrhea may increase the risk of urinary calcium oxalate precipitation via volume depletion.</p> <p>Conclusion: Presentations of oxalate nephropathy include acute renal failure, progressive chronic kidney disease, and nephrolithiasis. A diagnosis is supported by an elevated plasma oxalate level, increased urinary oxalate excretion, calcium oxalate crystals on urinalysis, or pure calcium oxalate kidney stones. Definitive diagnosis requires renal biopsy, which typically demonstrates tubular injury and abundant tubular deposits of calcium oxalate crystals. The management of oxalate nephropathy focuses on reversing hyperoxaluria to optimize the chance for renal recovery. Strategies include a low oxalate diet, calcium supplements to bind enteric oxalate, and correction of hypovolemia to reduce urinary supersaturation. Hemodialysis may be utilized for clearance of systemic oxalosis, or as required in cases of advanced renal failure.</p>
<p><b>Mara Wilson</b> Dr. Tyler Bridge Dr. Michael Schnaus</p>	<p><i>A Chilling Mystery: A Case of Spontaneous Periodic Hypothermia</i></p> <p>Case Presentation: An 83-year-old man with CKD stage 3, permanent atrial fibrillation and DVT on apixaban, BPH with chronic Foley, and hypothyroidism on thyroid hormone replacement was admitted with</p>

unexplained recurrent shock, hypothermia to 31.8°C, bradycardia, pancytopenia, and encephalopathy.

Over four months, the patient had six admissions for recurrent hypothermia, encephalopathy, and dysarthria. Despite treatment for presumed UTI and sepsis, no infectious source was identified, and no environmental cold exposure preceded the episodes. Causes of hypothermia including medication-induced, hypoglycemia, malnutrition, dermal dysfunction, metabolic, and endocrine disorders were ruled out. Brain MRI showed generalized atrophy without hypothalamic or pituitary lesion, agenesis of the corpus callosum, or other abnormalities. EMG, Titin antibody, striated muscle IgG antibodies and AchR binding/blocking antibodies were also unremarkable.

During this admission, broad-spectrum antibiotics were initiated for presumed pneumonia and shock. However, the patient declined and was transferred to the ICU for vasopressors and stress-dose steroids. Evaluation for adrenal insufficiency including ACTH and cortisol was normal. Despite further workups—including head imaging, thoracentesis, EEG, serum autoimmune encephalopathy panels, and a bone marrow biopsy—no cause for pancytopenia or shock was found. Vitamin B1, TSH, and free T4 levels were normal, and PEth testing confirmed alcohol abstinence. While on antibiotics, the patient remained minimally responsive for 10 days with temperatures of 32-34°C, requiring NG tube feedings. He intermittently followed simple commands and notably had no hyperhidrosis. After reviewing the literature and without another cause of hypothermia identified, a diagnosis of Spontaneous Periodic Hypothermia (SPH) was considered. On day 10 his family consented to a trial of Clomipramine 25 mg daily via NG tube. Temperatures were checked via bladder probe. Within 4 hours of dosing, his temperature increased from 33.3°C to 36.3°C and stabilized near 35.0°C. After three days of stable temperatures, the dose was increased to 25 mg BID. Within hours his temperature rose again, remaining between 35.8°C and 36.8°C until discharge. As his temperature normalized, he showed significant improvement in mental status, removed his NG tube, began feeding himself, and ambulated with therapy. He was discharged on Clomipramine 25 mg BID.

Discussion: Spontaneous Periodic Hypothermia (SPH) is a rare neurological syndrome involving recurrent hypothermia (<35.0°C) without environmental exposure or a systemic cause. Hypothermia itself can manifest as encephalopathy, dysarthria, cytopenias, hypotension, and bradycardia.

This case describes an 83-year-old man with recurrent hypothermia and encephalopathy who improved dramatically on Clomipramine. SPH has been linked to Shapiro syndrome (paroxysmal hypothermia, hyperhidrosis, agenesis of the corpus callosum), but its exact pathogenesis remains unclear. Disordered neurotransmitter function in thermoregulation is suspected, as successful treatments with clonidine, clomipramine, and cyproheptadine have been reported. This case highlights the importance of recognizing the manifestations of hypothermia, considering SPH in the differential diagnosis of unexplained hypothermia, and demonstrates the effective use of Clomipramine in its treatment.

<p><b>Kevin Wolfe</b></p>	<p><i>Iron Pill Aspiration: A Bronchoscopic Emergency</i></p> <p>Introductio: Iron pill aspiration syndrome is a rare but potentially life-threatening event that can lead to severe and often long-term complications. Typical symptoms include cough, wheezing, dyspnea, and hemoptysis. This report presents a case of an iron pill aspiration and discusses key points for clinicians to consider.</p> <p>Case Presentation: A 58-year-old female with a history of asthma, diabetes, and cirrhosis with portal hypertension was in her usual state of health before experiencing acute chest pain, dyspnea, and coughing after taking her pills. Chest CT revealed a 1 cm density in the right lower lobe of the lung, concerning for aspiration of her oral iron tablet. The patient was intubated, sedated, and transported to the ICU for bronchoscopy, which showed significant mucosal burn injuries and friable tissue. The pill had already partially dissolved, however it was successfully cleared with multiple lavages. Follow-up bronchoscopy one day later was revealing for persistent mucus but no structural airway obstruction. She was discharged in stable condition with antibiotics and steroids.</p> <p>Conclusion: this clinical case highlights the fact that iron pill aspiration requires prompt recognition and intervention to prevent complications such as airway stenosis or hemorrhage. Clinicians should be mindful of this syndrome, and should be cautious when prescribing iron pills to patients with dysphagia.</p>
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**Early Career Physician**

**Clinical Vignette – Early Career Physician**

<p><b>Dhairya Jarsania</b> Dr. Stephanie Grach</p> <p>Finalist</p>	<p><i>A Perplexing Case of Neurologic Decline</i></p> <p>Introduction: Myalgic encephalomyelitis, also known as chronic fatigue syndrome (ME/CFS), is a debilitating multisystem disease that is often misdiagnosed, resulting in excess health care utilization and disease progression from mismanagement. ME/CFS can be challenging to recognize in the inpatient setting due to its complex chronic nature; however, doing so can help facilitate an appropriate transition to outpatient management and improved outcomes.</p> <p>Case Presentation: A 38-year-old female presented to the ED for a myriad of progressive neurologic symptoms including slurred speech, bilateral posterior headaches, intermittent forgetfulness, and intermittent difficulty with ambulation over the past 6 months. Her comorbidities included pseudoseizures x20 years, postconcussive syndrome, recent diagnosis of functional neurologic disorder, and cervical radiculopathy. Six months prior, she experienced two tonic-clonic seizures, witnessed by EMS, which differed from her usual episodes. An MRI and EEG performed at that time were negative. Since that episode, she experienced slowed speech (not improving with speech therapy), varying degrees of difficulty concentrating, notable fatigue from activities that were previously easy for her, and unrefreshing sleep. She then presented to our ED for worsening of the above symptoms.</p>
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Further review of systems was negative. She was afebrile and hemodynamically stable in the ED and physical exam was overall unrevealing. CBC was notable for new leukocytosis of 19.2. The remainder of her labs including a CMP and TSH were normal. CT head was negative for acute findings. Additional studies including urinalysis, urine culture, COVID/flu swab, and chest x-ray were normal or negative for acute changes. She was admitted for ongoing toxic/metabolic workup of potential encephalopathy.

Fortunately, her leukocytosis resolved on recheck after a 1-liter fluid bolus and the remainder of her lab work including blood cultures returned normal. Inpatient neurology consultation provided no additional recommendations. A thorough history obtained by her inpatient medical team raised the possibility of ME/CFS, noting the CDC recommended Institute of Medicine 2015 Diagnostic Criteria: (1) significant new-found fatigue and inability to perform basic activities compared to prior for at least 6 months, (2) post-exertional malaise (notable worsening of her symptoms after previously tolerated activities needing hours to days to recover), and (3) unrefreshing sleep with either brain fog or orthostatic intolerance. Further inpatient testing was deferred, and she was discharged with outpatient autonomic testing and consultation with the General Internal Medicine department for further evaluation. Diagnosis of ME/CFS was confirmed and she was started on low dose naltrexone and a pacing activity management strategy with improvement in her symptoms and activity tolerance noted on follow-up 6 months later.

Conclusion: This case highlights the diagnosis of an emerging complex, chronic disease which affects millions of people across the nation. They are often misdiagnosed with conditions such as functional neurologic disorder and thus not appropriately treated. With viral triggers including SARS-CoV-2, these numbers are expected to exponentially increase making it vital for providers in all medical settings to be aware of patients suffering from ME/CFS.