



**American College of Physicians- Minnesota Chapter Annual Abstract Competition**  
**Minneapolis Convention Center**  
**October 11, 2019**  
 Abstracts Submitted for Competition

<b>Medical Students</b>	
<b>Research - Medical Students</b>	
<p> <b>Alyssa Brown</b>            Sabhya Rana            Jane Yap            Amanda Heino            Matthew Fogarty            Carlos Mantilla            Gary Sieck         </p>	<p> <i>Effect of TNF<math>\alpha</math> on Mitochondrial Function in Motor Neurons</i>            Background: Inflammation underlies a variety of neuronal diseases, including those that affect motor neurons. Mitochondrial dysfunction is also implicated in the etiology of neuronal diseases. The effects of inflammation are mediated via pro-inflammatory cytokines, such as TNF<math>\alpha</math>. The present study examined the effect of TNF<math>\alpha</math> on mitochondrial volume density and O<sub>2</sub> consumption in a model motor neuron cell line (NSC-34). Mitochondria were labeled using MitoTracker green and visualized in 3D using a confocal microscope (60x, NA 1.4 objective). Mitochondrial volume density was determined as the percent cell volume occupied by labeled mitochondria and distinguished from nuclear volume. As a fraction of both total cell volume and cytoplasmic volume, mitochondrial volume density increased by 40% in cells were exposed to TNF<math>\alpha</math> (20 ng/ml) for 24 h. Using a Seahorse XF Bioscience instrument, O<sub>2</sub> consumption was measured in response to a stress test. In addition, we used a quantitative histochemical technique to measure the maximum velocity of succinate dehydrogenase (SDHmax) activity, which is a key enzyme of the tricarboxylic acid (TCA) cycle, as well as the electron transport chain (ETC), in mitochondria. For SDHmax measurements, cells were incubated in a solution containing 80 mM succinate and 1.5 mM nitro blue tetrazolium (NBT), the reaction indicator. As the SDH reaction proceeded, images were acquired every 15 s using a 40x objective on an Olympus IX71 inverted microscope. Linearity of the SDH reaction was confirmed across an 8-min period, and SDHmax was determined and expressed as mM fumarate/L tissue/min. We hypothesize that in motor neurons, TNF<math>\alpha</math> induces an increase in reactive oxidant species (ROS) formation leading to unfolded (damaged) proteins, which triggers an endoplasmic reticulum (ER) stress response with downstream impact on mitochondrial function, as reflected by a decrease in O<sub>2</sub> consumption rate and SDHmax activity. All experiments used NSC-34 cells that were differentiated by 24-h serum deprivation until neurite structures (&gt;50 <math>\mu</math>m) were observed.            For each experiment, differentiated cells were split into two groups: either TNF<math>\alpha</math> treated or untreated. In NSC-34 cells, we found that TNF<math>\alpha</math>: 1) increases mitochondrial volume density, 2) decreases O<sub>2</sub> consumption when normalized for mitochondrial volume density, and 3) decreases SDHmax activity when normalized for mitochondrial volume density. These results support our hypothesis that in motor neurons, inflammation and pro-inflammatory cytokine exposure leads to mitochondrial dysfunction.         </p>
<p> <b>Madeleine Hage</b>            Dr. Megan Flanagan         </p>	<p> <i>Augmented QUITLINE Intervention for Pregnant Smokers</i>            Background: Cigarette smoking and tobacco use during pregnancy have been associated with pregnancy complications, preterm delivery, stillbirth, and low birth weight. In particular, the vascular constriction caused by nicotine in cigarettes results in decreased blood flow and oxygen delivery to the fetus. The aim of this study is to         </p>

assess whether using an “Augmented” Louisiana QUITLINE program for pregnant mothers will result in increased compliance with smoking cessation. Researchers will be analyzing smoking cessation rates in patients who participate in the study intervention, which combines enrollment in the free Louisiana QUITLINE program with weekly verbal encouragement from trained personnel. The “Augmented” protocol is currently being implemented in two downtown New Orleans OBGYN clinics, which predominantly serve low-income and Medicaid patients.

This study is currently enrolling subjects, with a study population goal of 100. However, the Gynecology Oncology group at Tulane School of Medicine recently used the same “Augmented” Louisiana QUITLINE program for newly diagnosed gynecologic cancer patients. Their study demonstrated that 72.3% of patients reported not smoking at least one year after entering the program.

While results for our study are pending, we anticipate similar results. The results of this study have the potential to help determine an efficacious protocol by which physicians can encourage smoking cessation in their pregnant patients. This inherently benefits both mother and child by reducing the risk of smoking-related complications of pregnancy.

**Heather Oas**  
 Dr. Dawn Wheeler  
 Dr. Ryan Kelly

*Identification and Management of Hepatitis C at an FQHC*

Introduction: Chronic hepatitis C infection is the primary cause of end-stage liver disease and hepatocellular carcinoma in the United States. As of 2016, at least 2.4 million individuals in the United States were living with chronic hepatitis C. As hepatitis C infection goes hand in hand with injection drug use, the ongoing opioid epidemic suggests these numbers are increasing exponentially. Unfortunately, anywhere between 45-85% of individuals with chronic hepatitis C are unaware of their disease status. Despite increasing availability and efficacy of hepatitis C antiviral agents, it is estimated that only about one-third of individuals with hepatitis C are ultimately referred to a higher level of care and/or treatment. Potential barriers to hepatitis C treatment include a lack of access to specialty providers, restrictive insurance requirements, as well as a lack of education and comfort surrounding hepatitis C treatment in primary care providers (PCP). In one instance, however, it was found that proper education and support of PCPs in an FQHC setting allowed for successful hepatitis C treatment at rates comparable to those seen in a specialty setting. Our investigation aims to add to this currently limited evidence base and to establish protocols for hepatitis C testing and treatment in the primary care setting.

Methods: A retrospective chart review identified patients with a positive Hepatitis C antibody test within the last 10 years from the Community-University Health Care Center (CUHCC). Further data was gathered from these medical records, including demographics, whether any additional follow-up testing was ordered (i.e. hepatitis C viral load), if referral to a higher level of care was provided, and ultimately whether or not treatment was initiated.

Results: From May 2009 - May 2019, 3,786 patients were screened for the presence of Hepatitis C antibodies. Of these individuals, 264 (6.97%) had a positive antibody test, indicating prior exposure to the virus. Within this cohort, 215 (81.5%) had a follow-up viral load ordered, and 114 (53.0%) of these individuals had evidence of chronic hepatitis C infection. 53 patients of the 114 patients with chronic hepatitis C (53.5%) were referred for hepatitis C treatment, with just 13 patients found to have completed treatment successfully, as determined by sustained virologic response.

Next Steps: As management of hepatitis C expands into our primary care clinic, we have developed a standardized treatment protocol to ease treatment flow for providers. Structured training will be delivered to all providers to improve patients’ access to treatment. Repeat chart review and evaluation will be conducted at one year in order to determine the efficacy of this training protocol.

**Clinical Vignette - Medical Students**

<p><b>Aldo Acosta-Medina</b>          Christianne Bourlon          Alexandra Wolanskyj</p>	<p><i>Acute Lymphoblastic Leukemia Relapse Masquerading as Myelopathy with Negative Imaging</i></p> <p>Introduction: Acute Lymphoblastic Leukemia (ALL) is a life-threatening condition, presenting typically with abnormal complete blood counts (CBC) demonstrating cytososes or cytopenias. Virtually all ALL relapses include a medullary component; however, isolated extramedullary relapse can occur and should always be considered in this population.</p> <p>A 43-year-old man with a history of ALL presented to the emergency room after progressive onset of bilateral lower extremity weakness without sensory, bowel or bladder changes. He was status post-allogeneic hematopoietic stem cell transplantation (allo-HSCT) 5 months prior with minimal residual disease at day +100 suggesting a deep response. Initial blood work demonstrated a normal CBC and blood smear. Neuroimaging was negative and did not revealed findings suggestive of CNS relapse, infection, or a vascular event. A lumbar puncture was performed and revealed numerous blasts compatible with CNS relapse of his ALL. Bone marrow aspirate and biopsy including flow cytometric evaluation was negative for relapse.</p> <p>The patient received intrathecal chemotherapy and was taken off graft-versus-host disease prophylaxis to induce a graft-versus-leukemia effect until CSF clearance. Although systemic therapy was recommended, the patient declined and received whole brain radiation therapy (RT).</p> <p>His LE strength improved with stabilization of his disease. Ten months later he developed bilateral increase in testicular volume, ultrasound was suggestive of leukemic infiltration. The patient received additional radiation to the testes without complications. One month after RT, the patient developed overt systemic relapse evidenced by development of bone pain throughout the lumbosacral region with presence of multiple blastic and lytic lesions throughout the appendicular and axial skeleton on CT and mediastinal lymphadenopathy. With this rapidly progressive picture, the patient was transitioned to supportive care and died 2 months later. Evidence of bone marrow relapse was exclusively present during the 2 last follow-up months after appearance of osseous lesions.</p> <p>This case illustrates the importance of the blood-brain and blood-testis barriers in limiting chemotherapy penetrance to tissues in patients with ALL. This unusual presentation of myelopathic symptoms, with negative imaging, demonstrated the need to always consider extramedullary relapse of ALL. Lumbar puncture with CSF analysis is still a pivotal study in evaluation of patients despite availability of highly-sensitive neuroimaging modalities and hematologic assessments, and early intervention is essential to optimize survival and quality of life.</p>
<p><b>Asma Adam</b></p>	<p><i>Brugada Blues: A Case of Sudden Cardiac Arrest in a Healthy Male</i></p> <p>Introduction: Brugada Syndrome is an autosomal dominant ventricular arrhythmic disorder associated with a high incidence of sudden death in healthy individuals. Research points to the RVOT as the origin of ventricular arrhythmias with characteristic right bundle branch block and ST-segment elevation in the right precordial leads on EKG, though the exact pathophysiology remains unclear. An implantable cardioverter defibrillator (ICD) is the main treatment, preventing sudden death in patients with Brugada Syndrome.</p> <p>Presented below is a case of a previously healthy male with a sudden cardiac arrest, suspected to have Brugada Syndrome</p> <p>Case Presentation: A previously healthy 53-year-old male was in his normal state of health when he suddenly became unresponsive at his home. Family members immediately began CPR. On EMS arrival, he was found to be in ventricular fibrillation and received 4 shocks, 300 mg amiodarone, and 2 amps bicarb with return of spontaneous circulation. On hospital arrival, he was hemodynamically stable but unresponsive. He was sedated, intubated, and hypothermia protocol was initiated.</p>

	<p>Head CT was without evidence of hypoxic ischemic injury. Peak troponin was 2.756, lactic acid was 7.8, and AST/ALT were 485 and 324 and were attributed to acute injury secondary to hypoperfusion following cardiac arrest. EKG with concern for Brugada Type III pattern with no evidence of myocardial ischemia. CT PA with no pulmonary embolism or pneumothorax, coronary angiogram without evidence of coronary artery disease, cardiac MRI with normal function and structure with no evidence of myocardial scarring, ECHO significant for EF of 39% with no wall motion abnormality. Etiology of cardiac arrest felt to be due to Brugada Syndrome vs. less likely ACS given absence of ischemic symptoms prior to arrest, lack of ST changes on EKG, normal cardiac imaging, and no prior family or personal history of cardiac disease.</p> <p>A single-chamber ICD was placed and he was started on 50 mg Metoprolol until further cardiac evaluation. He was also recommended to undergo genetic testing due to the autosomal dominant inheritance of Brugada Syndrome as he has siblings and children.</p> <p>Discussion: The prevalence of Brugada Syndrome worldwide is estimated at 5 in 10,000 people (more common in men) and is thought to contribute to 4% of all sudden deaths and up to 20% of sudden deaths in those with structurally normal hearts.</p> <p>Each year, 600,000 people in the United States experience a cardiac arrest, the majority of which occur outside of the hospital. The overall survival rates following cardiac arrest are low with &lt;6% of people who experience a cardiac arrest outside the hospital and 24% of patients who experience cardiac arrest within the hospital, surviving. In sudden cardiac arrest, the timely initiation of resuscitation is critical for survival and prevention of permanent impairment.</p> <p>In this case, the patient experienced a witnessed cardiac arrest with immediate initiation of effective CPR, attributing to his favorable outcome.</p>
<p><b>Yahya Almodallal</b>  Dr. Meltiady Issa  Dr. Hasan Ahmad-  Hasan Albitar</p>	<p><i>Antiepileptic-Induced DRESS Syndrome: A life-threatening complication</i></p> <p>A 30-year-old female with a history of postpartum depression and bipolar II disorder, presented to the emergency department with a 10-day history of subjective fevers, headache, sore throat and myalgias. She was started on amoxicillin for presumed sinusitis and discharged home. One day later, she returned with new-onset eye redness and a skin rash. On examination, she had splenomegaly, as well as a diffused morbilliform skin rash.</p> <p>Her medications included lamotrigine, quetiapine, and risperidone which were started 3 weeks prior to her presentation. Further questioning revealed that the patient had exposure to a baby calf that preceded the onset of her symptoms. Initial laboratory evaluation revealed elevated CRP (184.8, <math>\leq 8</math> mg/L), alkaline phosphatase (236, 35-104 U/L), ALT, AST, creatinine in addition to eosinophilia. Given her persistent fever and headache, she was initially started on broad-spectrum antibiotics for possible meningitis, however; this was ruled out with a negative lumbar puncture. EBV/CMV heterophile testing, as well as ANA and ANCA were negative as well.</p> <p>She underwent extensive infectious work-up that revealed a weakly-positive <i>Coxiella burnetti</i> IgG titer which was confirmed on repeat testing. As a result, she was started on azithromycin for treatment of possible acute Q-fever. Her kidney function continued to worsen (with increase in creatinine from 1.6 write the units to 4.1 on the 4th day of admission). Kidney biopsy was performed and showed acute interstitial nephritis. Due to presence of acute kidney and liver injury, fever, skin rash and eosinophilia, our suspicion for DRESS (Drug Reaction with Eosinophilia and Systemic Symptoms) syndrome secondary to lamotrigine/quetiapine was high. Both lamotrigine and quetiapine were discontinued and a skin biopsy was performed which confirmed the diagnosis. She was started on 60 mg of oral prednisone with significant improvement in her skin rash, as well as kidney and liver function tests.</p>

	<p>The patient was discharged on oral prednisone with a slow taper over 5 weeks in addition to azithromycin for treatment of acute Q-fever for a total of 7 days. She had normalization of her kidney and liver function tests and full resolution of symptoms on follow-up.</p> <p>DRESS is a potentially life-threatening hypersensitivity reaction to medications. Patients present with fever, skin rash, eosinophilia and/or internal organ involvement. Antiepileptic drugs are the most common cause as seen in this case. Prompt identification and discontinuation of the offending drug is the mainstay of treatment. Patients with severe DRESS may benefit from prolonged course of systemic corticosteroids.</p>
<p><b>Rachel Anderson</b> Dr. Olga Epstein</p>	<p><i>21st Century Sailor's Handshake: Ironing Out Cubital Lymphadenopathy</i></p> <p>Case Description: 62-year-old man with no significant past medical history, who presented to the emergency department after a mechanical fall resulting in a headache and nausea. He was found to have new thrombocytopenia, anemia, protein gap, and AKI. The patient denied abnormal bleeding or bruising, but did report progressive fatigue, subjective weakness, and occasional chills at night. He also noted painless 'bumps' in his forearms that had not changed over time, but he was unsure when they appeared.</p> <p>Upon physical exam, the patient was found to have multiple, 1-1.5 cm diameter, painless, rubbery, mobile nodules in bilateral proximal forearms, consistent with lymphadenopathy. Upon further exam, he was also found to have lymphadenopathy in the popliteal fossa, anterior cervical chain, and right sided submandibular area. His abdominal exam was notable for splenomegaly, which was confirmed on ultrasound. Strength in upper and lower extremities was preserved.</p> <p>Lab workup to evaluate for infection, hemolysis, and hematologic malignancies was significant for elevated lactic acid, LDH, and beta-2 microglobulin, and low haptoglobin. Hepatitis B panel and HIV testing was negative. Peripheral blood smear showed pancytopenia, increased Rouleaux formation and platelet clumps with occasional atypical lymphocytes. Due to increasing concern for lymphoma, flow cytometry was done and showed a kappa-monotypic B-cell population. SPEP and UPEP were positive for monoclonal kappa-IgM. A bone marrow biopsy was performed and was consistent with lymphoplasmacytic lymphoma with Waldenstrom's macroglobulinemia and iron deficiency anemia. The patient was started on ferrous sulfate and ibrutinib for treatment and symptom management and is clinically improving.</p> <p>Discussion: This is a patient with no significant medical history who presented with cubital lymphadenopathy and was found to have lymphoplasmacytic lymphoma with Waldenstrom's macroglobulinemia. Cubital, or epitrochlear, lymph nodes are well known for being palpated during the two-handed or "sailor's" handshake. Historically, the sailor's handshake was used to asses for epitrochlear lymphadenopathy as a sign of secondary syphilis. More recently, HIV/AIDS and sarcoidosis have been associated with frequent epitrochlear lymph node enlargement. Other causes include systemic or local infections, metastasis of distal melanomas or carcinomas, and lymphoma/leukemia.</p> <p>During a standard physical exam, medical providers do not frequently palpate the cubital lymph nodes for enlargement, but this finding is present in 27% of patients with another lymphadenopathy. Epitrochlear lymphadenopathy is almost always pathologic, making it a more differentiating clinical finding, and should prompt further workup.</p>
<p><b>Sarah Azer</b> Dr. Shruti Patel Dr. David Brennan</p>	<p><i>An Unusual Presentation of Trimethoprim-Sulfamethoxazole Induced Sweet's Syndrome with Polymorphic Rash and no Leukocytosis</i></p>

	<p>Sweet’s Syndrome (SS) is a condition characterized by fever and dramatic eruption of painful, erythematous papules, nodules, or plaques that usually involve the face, neck, and extremities. While SS is typically idiopathic or malignancy-related, available reports have established use of multiple medications to be associated.</p> <p>We herein report the case of a previously healthy 44 year old male who initially presented with a 3 day history of a polymorphic, cephalocaudal spreading skin rash that involved his scalp, neck, back, and bilateral extremities, one week after use of trimethoprim-sulfamethoxazole (TMP-SMX) for a skin infection. His rash consisted of cutaneous features typical of drug-induced SS, including maculopapular and vesicle-like components, with the additional unusual finding of pustular lesions. Extra-cutaneous manifestations included high fever (reported as high as 39.4C), malaise, pleuritic chest pain, arthralgia, and tea-colored urine. Despite historically reported objective fevers, our patient was afebrile upon evaluation (36.9C). Furthermore, initial workup demonstrated normal leukocyte count without neutrophilia (leukocyte count: 6400 cells/mm<sup>3</sup>, neutrophil count: 5500 cells/mm<sup>3</sup>), thrombocytopenia, C-reactive protein elevated to 236, negative chest X-ray, negative blood cultures, urinalysis revealing hematuria and proteinuria, and stably elevated troponins without ischemic changes on electrocardiogram (158 ng/L baseline, 155 ng/L 2 hour). Additionally, he had mildly elevated aspartate aminotransferase (88) and alanine transaminase (92) with negative Hepatitis B, Hepatitis C, and HIV testing. An extensive rheumatologic workup was negative. Overall, since pustular lesions constitute an unusual feature of SS, their presence in setting of systemic involvement and recent new medication usage may mimic the presentation of acute generalized exanthematous pustulosis (AGEP). Nevertheless, our patient’s skin biopsy revealed findings consistent with SS. He underwent treatment with prednisone with rapid improvement.</p> <p>Review of the literature reveals 8 case reports of other patients with TMP-SMX induced SS. We critically analyzed our case in context of features from the 8 other reports, demonstrating the following take home points. SS should be considered in patients with history of high fevers and malaise in setting of skin eruption. If exam reveals pustular lesions with reported recent usage of a new medication, SS should be added to the differential along with AGEP. In order to appropriately plan management strategies, definitive distinction should be made through biopsy as both disorders differ pathologically. Additionally, not all patients with SS present with the classic triad of neutrophilia/leukocytosis, fever, and skin rash. While fever patterns of SS are not well understood, our case suggests that pyrexia is not constant throughout the disease course and patients may be afebrile upon evaluation. Thus, it is important to ask about history of objective fevers. Moreover, absence of neutrophilia/leukocytosis does not exclude the diagnosis.</p>
<p><b>R. Tayler Bockstruck</b> Lily Wood Dr. Michael Lawson</p>	<p><i>Cinchonism: Acute Blindness and Deafness from an Intentional Quinine Overdose</i></p> <p>Quinine, though rarely prescribed today, is a drug with historical significance as a first-line antimalarial therapy and a remedy for muscle cramps.</p> <p>This case report presents a 62-year-old female who attempted suicide by ingesting a number of medications, including approximately 6.5 grams of quinine sulfate, initially prescribed for leg cramps. She presented with the classic triad of cinchonism, including tinnitus, QT prolongation, and blindness. While the first two symptoms resolved, her blindness persisted. She was closely followed by ophthalmology and toxicology services and it was suspected that her ingestion resulted in posterior ischemic neuropathy (PION).</p> <p>We discuss her presentation, initial treatment with hyperbaric oxygen therapy (HBOT) and nimodipine, and subsequent follow up with ophthalmology specialists. Finally, we recommend that, although rare, quinine ingestion remain on the differential for any person presenting with acute vision loss following an unknown or suspected ingestion.</p>

<p><b>Vikram Boparai</b> Sujith Puskoor Dr. Juan Alvarez</p>	<p><i>A Potential Role for Immunotherapy and Genetics Testing in Advanced-Stage Thymoma: A Case Report and Discussion</i></p> <p>Introduction: Thymomas are rare mediastinal tumors that represent just 0.2–1.5% of all solid malignancies. Their etiology and molecular biology are poorly understood. Incidence peaks between 65 to 75 years of age. Approximately 33% of patients are asymptomatic and diagnosed incidentally, while symptomatic presentations are most often attributable to concurrent myasthenia gravis. Surgical resection is first-line and often curative in early-stage tumors. Advanced-stage tumors, however, are rarely curative and commonly carry a poor prognosis despite adjunctive chemotherapy and radiotherapy options.</p> <p>Case Presentation: A 24-year-old male with no medical history presented to the ER with acute-onset chest pain and vomiting. His symptoms arose one day prior as sharp left-sided thoracic back pain at rest, worsening with inhalation. His pain progressed overnight to a persistent, diffuse chest-pressure-like sensation. He noted multiple bouts of non-bloody vomiting during the 3-4 hours prior to arrival, which he attributed to difficulty swallowing solids and liquids. His mother had had ovarian cancer and his maternal grandmother breast cancer. Vital signs were within normal limits. Physical examination found reduced left lower lung breath sounds and faint left-sided inspiratory crackles. Chest CT angiogram confirmed a large anterior mediastinal mass, multiple left pleural masses, and a loculated left pleural effusion. Video-assisted thorascopic surgery attained biopsies and re-mobilized the severely adhered pleura. Histopathology revealed loosely clustered polygonal epithelial cells interspersed among a lymphocyte-predominant background from mediastinal and pleural tissue samples, most consistent with a type B2 thymoma. Given confirmed pleural metastasis and an unremarkable staging PET-CT scan of abdomen and pelvis, the thymoma was documented as stage IVa. The tumor was deemed non-resectable and the patient began systemic chemotherapy with cisplatin, cyclophosphamide, and doxorubicin.</p> <p>Discussion: A recent trial using the above chemotherapy regimen for non-resectable metastatic thymomas reported 50% response rates with a median survival time of 38 months. Clinical trials with immunotherapy and targeted molecular therapy provide a secondary option; however, most have demonstrated inconsistent response rates. Our incomplete understanding of thymoma molecular pathogenesis likely contributes to our clinical limitations, worsened by tumor rarity, which has limited large-scale prospective research. One possible solution is to conduct immunohistochemistry staining and genetic profiling studies using biopsy tissue in all advanced-stage cases. This proactive approach would likely stratify susceptibility to enhance patient selection and efficacy of second-line treatments. Given that first-line treatments are rarely curative, most patients would likely benefit from preemptive consideration of alternative therapies. The advanced stage in this atypically young patient with a family history of cancer also raises the possibility of potential genetic predisposition. Sharing this data with The Cancer Genome Atlas project may prove particularly useful, as revealing genetic predispositions could lead to indications for screening and intervention in earlier stages, when a cure is much likelier.</p>
<p><b>Daniel Bullard</b> Dr. Aaron Boothby Dr. Bryant Megna Dr. Abdul Haseeb</p>	<p><i>From Battlefield trauma to palliative care: hemostatic powder for a hemorrhagic gastrointestinal stromal tumor</i></p> <p>Introduction: Multiple Myeloma is a malignant neoplasm of plasma cells belonging to the family of plasma cell dyscrasias which produces a spike in monoclonal immunoglobulin or “M-protein.” While multiple myeloma has many classic clinical features, a malignant pleural effusion is a very uncommon manifestation of this illness.</p> <p>Clinical presentation: A 61-year-old man with known multiple myeloma presented with shortness of breath and fatigue. On exam, he had decreased breath sounds on the left lower and mid-lung fields. CT showed diffuse lytic and sclerotic lesions</p>

	<p>throughout the skeleton and a large left sided pleural effusion. Sampling of the pleural fluid showed very bloody fluid. Testing for infection was negative and cytology showed plasma cells and findings consistent with malignant pleural effusion.</p> <p>Discussion: This case highlights an uncommon manifestation of multiple myeloma. While malignant effusions may be seen in many cancers, in myeloma it suggest very advanced disease with an associated poor prognosis.</p>
<p><b>Beret Fitzgerald</b> Dr. Sam Ives</p>	<p><i>Take Note: An Impressive Case of Diffuse Lymphadenopathy</i></p> <p>Introduction: The differential for diffuse lymphadenopathy is broad and should include infectious, malignant, and autoimmune etiologies. The region, size, and symptoms associated with the lymphadenopathy are important considerations in differentiating between the various causes. We report a case of advanced follicular lymphoma that is complicated by obstructive lymphadenopathy and malignant transformation.</p> <p>Case Presentation: A 71-year-old man with a known recent history of leukocytosis and no other medical records presented with 2 months of fatigue and generalized weakness, a 10 lb weight loss, and several days of bilateral lower extremity swelling. The patient was seen 4 months prior in an outpatient setting and was incidentally found to have leukocytosis but declined further workup at the time in order to travel abroad for several months. At presentation he was found to have diffuse lymphadenopathy and multiple non-tender palpable abdominal masses with hepatomegaly. He denied other B symptoms or prior lymphatic swelling and would not have come in except a family member said he looked ill. An infectious workup was negative. His blood smear, excisional biopsy of an axillary lymph node, and bone marrow biopsy were all consistent with follicular lymphoma. However, a PET scan showed increased tracer uptake diffusely in lymph nodes as well as the liver. The increased hepatic uptake was concerning for malignant transformation and a needle core biopsy demonstrated Diffuse Large B Cell lymphoma. Additionally, he was found to have spontaneous tumor lysis syndrome, hypercalcemia, obstructive hydronephrosis, and acute on chronic kidney failure secondary to the follicular lymphoma and Diffuse Large B Cell lymphoma. He was initially treated with calcitonin, allopurinol, rasburicase, and IV hydration. The patient's electrolyte abnormalities from TLS were stabilized and his ureter was stented, both improving his AKI, before proceeding with further oncologic treatment. After an initial course of prednisone, the patient was treated using the R-CHOP protocol.</p> <p>Conclusions: With a known history of leukocytosis and diffuse painless lymphadenopathy, there was a high suspicion for malignancy in this patient. However, his extensive bulky lymphadenopathy causing obstructive hydronephrosis and extra nodal involvement was unusual for an indolent lymphoma. It is important to correlate clinical course and severity with the diagnosis. This patient highlights a malignant presentation of diffuse lymphadenopathy as well as late presentation of an indolent cancer with malignant transformation.</p>
<p><b>Kate Geschwind</b> Dr. Sam Ives</p>	<p><i>Getting to the Heart of the Matter: Cardiac Amyloidosis as a Uncommon Cause of Syncope</i></p> <p>Introduction: Multiple myeloma and AL amyloidosis are similar plasma cell disorders. Multiple myeloma is the malignant transformation and monoclonal proliferation of plasma cells, resulting in the overproduction of monoclonal immunoglobulin, and AL amyloidosis is the deposition of the immunoglobulin light chains produced by the malignant plasma cells within extracellular tissue and organs. Approximately 12-15% of patients with multiple myeloma develop overt amyloidosis<sup>1</sup>. Rarely, amyloidosis can affect the heart, leading to conduction system disease, syncope, and a risk of cardiomyopathy and arrhythmia.</p>



	<p>Case Presentation: A 58-year-old man with a past medical history of ischemic cardiomyopathy, heart failure with reduced ejection fraction, multiple myeloma, and COPD presented to the emergency department for recurrent syncope. He had previously had an extensive workup for presumed vasovagal syncope that was unremarkable but continued to have syncopal episodes, encouraging him to seek further care. He had four episodes of syncope within the past two months, accompanied by prodromal symptoms of intense upper abdominal pain, warmth, darkened vision, mild shortness of breath, and a sense of impending doom. His most recent syncopal episode was accompanied by a bowel movement. He denied any nausea, vomiting, or palpitations associated with the episodes. Upon workup, physical exam was unrevealing and labs were unremarkable except for a CBC consistent with multiple myeloma (leukopenia, anemia, mild thrombocytopenia). An EKG was performed and revealed sinus rhythm with a very long first degree AV block with a PR interval of 301 msec. He also had a borderline prolonged QRS duration of 120 msec with a left bundle branch block pattern. In the hospital, telemetry was consistent with first degree AV block as well as short episodes non-sustained VT. Due to the patient's history of multiple myeloma there was concern for an infiltrative process, cardiology was consulted and a cardiac MRI was performed with findings consistent with amyloidosis. There was significant concern for progression of the AV block to a higher grade due to the involvement of the conduction system and so the patient was strongly recommended to have an implantable ventricular pacer. The patient was overwhelmed by the news and asked to return home to be able to discuss the plan with his family. He was sent home with a wearable cardioverter defibrillator (WCD) in case of an arrhythmia and sudden cardiac arrest and later had an implantable cardiac defibrillator (ICD) placed.</p> <p>Discussion: Among the many causes of syncope, cardiac arrhythmias are among the most serious. Cardiac amyloidosis that affects the conduction system poses the risk for sudden cardiac arrest due to arrhythmia and requires immediate intervention via ICD placement once diagnosed. Patients who wish to delay treatment should be given a WCD in case of arrhythmia, but physicians should strongly encourage patients to have an ICD placed as soon as possible to prevent fatal arrhythmias. The serious nature of sudden cardiac death associated with cardiac amyloidosis underscores the importance of being able to recognize amyloidosis as a separate disease entity in a patient with multiple myeloma in order to properly address and treat the correct underlying pathology. While multiple myeloma can cause a wide variety of symptoms, physicians should keep amyloidosis on the list of differential diagnoses as a possible cause unexplained symptoms. It is essential to recognize amyloidosis in patients with multiple myeloma to be able to provide optimal care and prevent delayed diagnosis.</p>
<p><b>Kristin Giesen</b></p>	<p><i>Variable Migratory Neurological Symptoms in a 51-year-old</i></p> <p>Introduction: Neurological symptoms present a wide differential diagnosis, especially in an immunocompromised patient, increasing the challenge diagnosing progressive multifocal leukoencephalopathy (PML). The rarity of the condition only compounds the diagnostic difficulty, which can be further challenging when PCR testing, seen as highly sensitive and specific for other conditions, does not provide the same diagnostic security when testing for PML.</p> <p>Case Presentation: A 51-year-old man presented to the emergency department with slurred speech, right facial droop and right-handed clumsiness, and was admitted with concern for stroke. Imaging, including CT and MRI, demonstrated multifocal white matter abnormalities and a predominant area of subcortical hyperintensity in the left frontal lobe with no evidence of acute pathology. A thorough medical history revealed HIV/AIDS for which he had been off HAART for 7 months due to financial issues with a current CD 4 count of 56. This admission was complicated by septicemia. A lumbar puncture (LP) was performed to rule out CNS infection, which was negative and included PCR for JC virus, CMV, and EBV. A serum test for JC virus was also negative. He was discharged after a 6-day hospitalization on antibiotics, aspirin, and</p>

	<p>atorvastatin for suspicion of a stroke, with a plan to restart HAART.</p> <p>He was rehospitalized less than 2 weeks later with stroke-like symptoms including worsening right-sided weakness and slurred speech. Imaging and clinical exam revealed no clear indication for an acute stroke and no change to the prior white matter abnormalities. His working diagnosis was TIA. He was discharged the following day to follow up with outpatient Neurology.</p> <p>Following this hospitalization, there was a period of stability in his symptoms with intermittent periods of increasing right hand weakness. He was rehospitalized 2 months later with worsening weakness now involving his right face, arm, and leg with a recent outpatient MRI showing additional subcortical hyperintensities concerning for PML, HIV or CMV encephalitis. An LP was performed as an outpatient prior to this hospitalization and PCR for JC virus was negative. A repeat MRI demonstrated progression of the prior subcortical white matter lesions, highly suspicious for PML. Neurology and Infectious Disease consultations agreed that a brain biopsy for definitive diagnosis was warranted, which was performed on left frontal lobe lesion confirming the suspicion for PML.</p> <p>Discussion: This case illustrates the diagnostic challenges of PML in the setting of multiple hospitalizations, negative PCR testing, and a wide differential diagnosis. Although the suspicion for PML arose in the first hospitalization, the definitive, gold standard tissue biopsy did not occur until months later. Despite the delayed definitive diagnosis, the treatment for PML, reestablishing the patient's adaptive immune system by restarting HAART, was timely.</p>
<p><b>Kellen Glynn</b> Dr. Albertine Beard</p>	<p><i>Trick or Treat, What Smells so Sweet? Pretty Please don't be my Feet: Myroides Cellulitis and Bacteremia in a Diabetic Patient</i></p> <p>Case Presentation: A 65 year old man with type 2 diabetes not taking medication who developed gangrene of the right great toe which required amputation. After the amputation, the wound was slow to heal weeping for almost 2 months. One week prior to presenting to the ED for severe right lower extremity pain, the wound closed.</p> <p>Exam was notable for an afebrile man with a blood pressure of 105/ 65 and pulse of 115, bibasilar crackles, and an extremely tender, edematous, and erythematous right lower extremity. The right foot skin was peeling but not weeping. Labs were notable for WBC count of 31,210.</p> <p>Blood cultures were drawn and empiric antibiotic treatment for sepsis was begun with vancomycin and ceftriaxone with improvement. On the second day of hospitalization, ¼ blood cultures grew Myroides species sensitive only to ciprofloxacin, piperacillin-tazobactam, and imipenem. The patient's antibiotics were changed to oral ciprofloxacin for 7 days. The patient recovered well and was discharged on hospital day 5.</p> <p>Discussion: Myroides is an unusual pathogen in humans with 48 cases reported including cellulitis, urinary tract infection, and bacteremia.<sup>1</sup> Myroides species are gram negative rods that are non-motile, obligate aerobic with a yellow pigment.<sup>2</sup> Additionally, the bacteria in our case is well documented to have a particular fruity odor. Myroides infection in humans are most frequently documented to be associated with natural environmental sources such as fresh water, soil, such as in farming accidents, or, in one case, a pig bite.<sup>2</sup> These cases tend to affect immunocompromised patients including cancer patients and diabetics. This bacteria is well documented to be intrinsically highly antibiotic resistant which can complicate treatment due to the bacteria's susceptibility to standard empiric treatment regimen.</p> <p>The patient in this case was immunocompromised due to his complicated, non-compliant diabetes. He reported remaining in his home/ chair for the two months prior to presentation due to his painful and weeping foot which he described as smelly. He</p>

	<p>reported no exposure to environmental fresh water. The only potential contact he reported was his sister's labrador that frequently swam in the local lakes. We believe this to be a potential source of infection either by licking or by contact with the patient's wound.</p> <p>This case adds another gram negative rod to consider in diabetic patients with a history of ulcers or open wounds with potential environmental exposure, and highlights a setting in which blood cultures clearly provide benefit in developing a treatment plan in a patient with a septic infection. It provides an example of when a detailed history may provide reason to treat with an even broader spectrum antibiotic than traditionally considered in diabetic foot infections.</p>
<p><b>Rebecca Grove</b></p>	<p><i>Diagnosis of Mantle Cell Lymphoma Reveals Concomitant Papillary Thyroid Carcinoma</i></p> <p>Introduction: Mantle cell lymphoma (MCL) is a sub-type of Non-Hodgkin's lymphoma often associated with a poor prognosis. As treatments improve and survival rates increase, it is important to understand the possible long-term complications of MCL, including the development of second primary malignancies.</p> <p>Case Presentation: A 60-year-old man presented to the emergency department with one week of left groin pain and left upper quadrant abdominal pain. Work-up was unremarkable except for dark urine and a CT stone evaluation was ordered. The CT showed hepatosplenomegaly and multiple enlarged inguinal and mammary lymph nodes.</p> <p>The patient followed up in cancer clinic where the working differential included TB vs. fungal infection vs. lymphoproliferative disorder. Initial testing showed a small atypical B-cell population, positive for CD20 and CD5, with mild leukocytosis but a normal LDH and no cells definitive for neoplasia. A subsequent PET scan was concerning for extensive adenopathy above and below the diaphragm as well as a hypermetabolic nodule in the left thyroid. The patient underwent axillary lymph node biopsy and was found to have lymphoid cells with an (11;14) translocation, positive for CD5, CD20, and Ki67. The diagnosis of MCL was made. Two weeks later, the patient returned for a left thyroid nodule fine needle aspiration that revealed papillary thyroid carcinoma.</p> <p>At that time, the patient was started on chemotherapy with Bendamustine and Rituximab (BR), with a plan for thyroid removal following completion of three BR cycles. After his second cycle of BR, the thyroid nodule had not increased in size. Resection was postponed pending completion of chemotherapy. The final cycle of BR was given seven months after the patient's initial presentation to the emergency department. A repeat PET scan exhibited no residual hypermetabolic or pathologically enlarged lymph nodes. One prominent node remained in the left inguinal region. Shortly after, the patient underwent surgery for his thyroid cancer. Due to extensive fibrosis, only a partial thyroid resection was performed. The patient received follow-up radioactive iodine ablation.</p> <p>Nearly three months post-resection (six months post-chemotherapy), a third PET scan showed no evidence of lymphoma but residual uptake in the surgical bed of the patient's neck, possibly reactive or indicative of residual carcinoma. Maintenance treatment with Rituximab every 2 months was initiated, which the patient will take until July of 2021.</p> <p>Discussion: This case demonstrates an incidental finding of papillary thyroid carcinoma discovered while diagnosing MCL. Although limited research exists, it is believed that patients with MCL are at a significantly increased risk for second primary malignancies. Consequently, cancer-specific screening should be recommended for any patient diagnosed with MCL.</p>

<p><b>Laura Hauff</b> Dr. Matthew Glogoza Dr. Amy Holbrook</p>	<p><i>The “Stones, Bones, Moans, and Groans” of a Meandering Hypercalcemia Etiology</i></p> <p>Introduction: Hypercalcemia in asymptomatic patients is a common incidental finding for internists. The most common causes are primary hyperparathyroidism and malignancy. In cases of malignancy, PTH is often low and calcium is more severely elevated (i.e. &gt;14 mg/dL), while in primary hyperparathyroidism, PTH is often elevated with mild hypercalcemia. Many patients have a benign course, but hypercalcemic crisis can be a life-threatening emergency.</p> <p>Case Description: A 59-year-old male with history of hypertension, gastric bypass, and nephrolithiasis presented with two weeks of fatigue, slow and slurred speech, dizziness, constipation, and unintended 10 lb weight loss. History was negative for supplements, recent dietary changes, and family history of endocrine disorders or malignancy. On exam, RUQ was tender to palpation and patient exhibited profound fatigue. Initial labs significant for total serum Ca<sup>2+</sup> 19.8 mg/dL, ionized Ca<sup>2+</sup> 2.33 mmol/L, and creatinine 3.73 mg/dL (previously 1.3 mg/dL). Aggressive hydration with normal saline was started, as well as a bisphosphonate. CT was negative for occult malignancy but showed a left thyroid nodule and pancreatic edema. Subsequent ultrasound confirmed left-sided thyroid nodule and a potential parathyroid adenoma. Further workup showed extremely elevated PTH 1690 pg/mL, low vitamin D 8.7 ng/mL, and normal TSH. Cinacalcet and vitamin D supplementation were started. Spot urinary Ca<sup>2+</sup>/creatinine of 0.75 ruled out tertiary hyperparathyroidism. Patient had asymptomatic transient bouts of 2:1 AV block and advanced heart block with sinus bradycardia thought to be due to hypercalcemia. Nuclear medicine parathyroid scan demonstrated subtle diffuse residual activity with an enlarged left thyroid lobe but no parathyroid adenoma was observed. Uptake was less than expected for a parathyroid adenoma with such elevated PTH. Concern for parathyroid carcinoma. Patient underwent left hemithyroidectomy and left parathyroidectomy. PTH immediately dropped from 952 to 89 pg/mL post-removal. Ca<sup>2+</sup> trended down from 12.4 to 8.9 mg/dL and calcium carbonate was started with close monitoring for hungry bone syndrome. Pathology indicated an atypical parathyroid adenoma (20 gm) as well as an incidental 2 mm papillary thyroid microcarcinoma (margins and nodes negative). At discharge, creatinine improved to 1.74 mg/dL, calcium 8.4 mg/dL, and vitamin D 22.1 ng/mL.</p> <p>Discussion: Hypercalcemia is well known for its effects on neuromuscular, renal, skeletal, cardiovascular, and gastrointestinal systems. A well delineated flowchart exists to determine hypercalcemia etiology but a flowchart is only as good as the common presentations upon which it is based. This unusual presentation of a common condition is a reminder of the importance of correlation of lab findings, imaging, and pathology in determining a final diagnosis. Despite extremely elevated calcium and PTH concerning for more rare malignancy, this patient’s fortunate outcome demonstrates that hypercalcemia is most commonly due to primary hyperparathyroidism.</p>
<p><b>Laura Klugherz</b> Dr. Diane Ahlers</p>	<p><i>Anaphylaxis: A Shocking Encounter</i></p> <p>Introduction: Anaphylaxis is a life-threatening syndrome with many causes, variable severity, and a broad differential diagnosis. Recognizing anaphylaxis is more difficult within the healthcare setting where new medications are introduced and physiology is dynamic. This is a case of intra-operative anaphylactic shock with a challenging diagnosis and a need for team-based communication.</p> <p>Case Presentaton: A 33-year-old female with HER2 positive breast cancer treated with neoadjuvant doxorubicin presented for elective lumpectomy with sentinel lymph node biopsy. She had no other pertinent history, no allergies to medications, and recent echocardiogram showed mild unchanged mitral regurgitation. General anesthesia was induced with a laryngeal mask airway (LMA) for ventilation. Cefazolin and dexamethasone were administered for infection and nausea prophylaxis. Local anesthetic was injected, as well as isosulfan blue and technetium-</p>

	<p>99 for lymph node localization. Fifteen minutes into the procedure, the patient's pulse increased to 150 BPM while blood pressure decreased to 50/30 mm Hg. Phenylephrine was administered without improvement. Given her history of doxorubicin, SVT and cardiogenic shock were suspected. Fluid, esmolol, and ephedrine were given while sevoflurane was substituted for propofol. The vitals remained unresponsive.</p> <p>Thirty minutes after symptom onset, norepinephrine administration resulted in only slight improvement. The operation was paused by anesthesiology, arterial and internal jugular lines were placed, and an endotracheal tube was inserted due to air leak. Reliable cuff and arterial BP measurements were very difficult to obtain. Labs revealed metabolic acidosis, but end tidal CO2 and oxygen saturation remained unexpectedly normal throughout the case.</p> <p>About sixty minutes after symptom onset, angioedema was observed in the extremities and face. No wheezes or rashes were noted. Diphenhydramine and vasopressin were administered leading to increased BP and decreased pulse, and the procedure was resumed and completed successfully. A chart and literature review by a medical student revealed two findings. First, the patient had received cefazolin several years earlier without incident. Second, it was discovered that isosulfan blue and technetium-99 are associated with rare anaphylactic reactions, and both had been injected prior to the case. These findings were communicated to surgical and anesthesia staff, and epinephrine was administered before ICU transport. Anaphylaxis secondary to isosulfan blue (and/or technetium-99) was presumed while vitals continued to normalize. Vasopressors were discontinued the next day and she was safely extubated on POD5.</p> <p>Conclusion: The case highlights the diagnostic difficulty of anaphylaxis with an initially more likely alternative diagnosis, lack of clear trigger, and lack of skin or pulmonary findings. Inter-team communication between the surgeon, anesthesiologist, nurse anesthetist, and medical student was necessary to manage the problem and make the diagnosis. The case prompted work to create a policy involving verbal and written communication between the surgeon and anesthetist, respectively, for all operative injections.</p>
<p><b>Lauren Lussenhop</b>  <b>Bennett Maki</b>  Dr. Edith Nyangoma  Dr. Nikifor Konstantinov  Dr. Daniel Ries</p>	<p><i>Blastomycosis: A Defense of the Review of Systems</i></p> <p>Introduction: Blastomycosis is a systemic pyogranulomatous infection arising from inhalation of <i>Blastomyces dermatitidis</i>, a dimorphic fungus endemic to regions bordering the Great Lakes, and the Mississippi and Ohio River valleys. It causes acute and chronic pneumonias, as well as disseminated infection with cutaneous lesions as the major extrapulmonary manifestation. In endemic areas, a low threshold to broaden the differential to include blastomycosis should be considered in non-resolving pneumonias. We report a case of disseminated blastomycosis in an immunocompetent patient.</p> <p>Case Description: A 34-year-old male without significant past medical history presented with acute onset right hip pain and a five-month history of enlarging and spreading skin lesions. The lesions, now fragile and draining seropurulent discharge, appeared first on the face and were thought to be acne until they spread to his extremities and torso, and continued to grow in size.</p> <p>One month prior to admission, patient developed productive cough, shortness of breath, and mild weight loss. He was diagnosed with CAP and treated with azithromycin with mild improvement of respiratory symptoms. However, a productive cough with dark brown sputum with occasional bloody tinge persisted. Admission physical exam was notable for diffusely scattered verrucous violaceous plaques most pronounced around the nares, mouth, and scalp. Right hip demonstrated tenderness over the greater trochanter but retained full ROM with no overlying skin changes.</p>

	<p>Chest X-ray showed an airspace infiltrate of the left upper lobe. Comprehensive infectious workup was undertaken. Notably, Blastomyces urinary antigen was positive, and excisional biopsy of one of the skin lesions was also consistent with blastomycosis. No underlying immunodeficiency was found. MRIs of the pelvis and the right shoulder revealed diffuse, enhancing bone marrow lesions, with evidence of anterior distal clavicle cortical destruction. Subsequently, brain MRI was performed revealing enhancing lesions and erosions of the calvarium and skull base with underlying pachymeningeal enhancement. Right clavicular debridement was performed, and subsequent fungal culture also grew Blastomyces dermatitidis. With extensive involvement observed on limited imaging, a PET/CT was performed and showed penetrating fungal involvement of all long bones, pelvis, ribs, skull and right upper lung. Due to the evidence of CNS involvement, amphotericin was started with the intention of a six-week course, with an anticipated year-long course of voriconazole thereafter.</p> <p>Conclusion: This case is a display of disseminated blastomycosis with diffuse osteomyelitis and skin lesions, pulmonary and CNS involvement, in an immunocompetent host. The initial presentation looked much like typical CAP with cough, dyspnea, and x-ray changes. However, clinical presentation, physical exam, and manifestations of Blastomyces are often nonspecific; thus, an acute clinical awareness and complete review of systems is required for diagnosis. The recognition of cutaneous Blastomyces could have avoided a delay in diagnosis and treatment, especially in an endemic area.</p>
<p><b>James McCluskey</b></p>	<p><i>L'est Jaundice be Beyond Us</i></p> <p>Introduction: Anabolic steroids have therapeutic purpose but may also be infamously used by athletes to enhance performance. Among many side effects they can cause cholestatic liver injury. The case at hand is an example of profound jaundice due to intramuscular (IM) injection of anabolic steroids, and how careful history taking can be key to solving a clinical conundrum.</p> <p>Case Presentation: An otherwise healthy 28-year-old man presented to the clinic for follow-up after a 1-day hospitalization a week ago at an out of state facility. His complaints included 2 weeks of worsening yellow eyes and skin, brown urine, diarrhea that, "looked like ripped up paper in the toilet," decreased appetite, and generalized itching. The remainder of the review of systems was negative. Social history noted employment as a heavy equipment operator. He identified as heterosexual with 4 sexual partners in the last year, rarely used alcohol, and denied tobacco, illicit drug use, and recent international travel. On exam his vital signs were normal and he had a body mass index (BMI) of 37 kg/m<sup>2</sup>. Prominent scleral icterus and diffuse jaundice were noted, but the remainder of his physical exam was normal. Review of his recent hospitalization noted a diagnosis of hyperbilirubinemia and presumed hepatitis A. Laboratory studies noted: total bilirubin 8.8 mg/dL, direct bilirubin 5.6 mg/dL, alanine aminotransferase (ALT) 203 U/L, aspartate aminotransferase (AST) 71 U/L. Urinalysis showed protein 100, urobilinogen 4.0, large bilirubin, small blood, and 20-50 red blood cells. Computed tomography and magnetic resonance imaging of the abdomen and pelvis were normal.</p> <p>Hepatitis and human immunodeficiency virus (HIV) serologies, and gastrointestinal pathogen panel were negative upon repeat. His bilirubin trended upward to a maximum of 48.5 mg/dL while aminotransferases trended down. With no known etiology, additional questioning was perused. He then admitted to using the IM drug "tren" (formally called trenbolone, a testosterone ester) for muscle building. Gastroenterology was consulted. A liver biopsy was performed that revealed zone 3 cholestasis and canicular bile plugs without fibrosis, consistent with cholestatic liver injury. Management included laboratory monitoring and symptom management. He reported improvement of his diarrhea with use of atropine-diphenoxylate as needed</p>

	<p>and avoiding dietary fat. His pruritis was managed with ursodiol and rifampicin. It took 3 months for the patient’s symptoms to resolve.</p> <p>Discussion: Many drugs can cause cholestatic liver injury. However, until recently only oral (rather than IM) anabolic steroids were thought to be a cause. In such cases, serious sequelae including vanishing bile duct syndrome and renal failure are possible, though not seen here. Lastly, this case should also serve as a reminder that revisiting the patient’s history in a diagnostic dilemma can prove to elucidate an answer.</p>
<p><b>John McGrory</b></p>	<p><i>An Uncommon Presentation of Recurrent CLL/SLL</i></p> <p>Introduction: Chronic lymphocytic leukemia and small lymphocytic lymphoma (CLL/SLL) represent a common disease process that primarily affects either the blood and bone marrow or the lymph nodes, respectively. This typically indolent malignancy usually presents in the elderly with enlarged lymph nodes, hepatosplenomegaly, recurrent infections, or asymptomatic lymphocytosis. CLL/SLL infiltration into the gastrointestinal tract is relatively rare in the literature but can be associated with several signs and symptoms including anemia or colitis, or it may signal a transformation to a more aggressive diffuse large B cell lymphoma (Richter’s transformation). The present case demonstrates an unusual presentation of gastrointestinal involvement of CLL/SLL manifesting as acute appendicitis.</p> <p>Case Presentation: A 69-year-old woman with a history of Rai stage 0 CLL diagnosed in 2015, previously treated with obinutuzumab for six months in 2018, presented to the emergency department with 24 hours of progressively worsening diffuse abdominal pain, stabbing sensation in the right lower quadrant, and signs of peritonitis, which were confirmed by abdominal CT to be caused by acute appendicitis. After undergoing a laparoscopic appendectomy without complications, her surgical pathology report noted that the appendix was infiltrated by lymphocytes which were strongly positive for CD20 and CD5 and negative for CD3 and CD21, which, in the context of a normal lymphocyte count, is consistent with SLL involving the appendix. The abdominal CT also showed bilateral retroperitoneal and pelvic lymphadenopathy, supporting a final diagnosis of stage II SLL. The patient followed up with her oncologist and ultimately decided to forego the recommendation of restarting an obinutuzumab regimen, instead opting for continued observation.</p> <p>Discussion: CLL/SLL is a relatively common and largely indolent malignancy in the elderly which can often be managed with an “active surveillance” strategy. This surveillance typically includes monitoring of lymphocyte counts and continuous assessment for the worsening or new onset of lymphadenopathy, recurrent infections, hepatosplenomegaly, fatigue, or malignancy-associated “B symptoms”. This case demonstrates the importance of investigation and oncological follow-up for any gastrointestinal-related signs and symptoms in those with known CLL/SLL as well because, although rare, such a presentation could signal spreading disease or disease transformation, which may necessitate more active therapy with chemotherapeutic agents and/or radiation.</p>
<p><b>Kevin Miller</b> N.S. Akhiyat N.J. Vinzant G.A. Wintheiser C.V. Anstine W.J. Ward</p>	<p><i>CLLassic Case of Hyponatremia</i></p> <p>Case Presentation: A 70 year-old man was admitted to the hospital with a one month history of generalized weakness, fatigue and poor appetite.</p> <p>Two years prior, he was diagnosed with chronic lymphocytic leukemia (CLL), Rai stage IV, with no TP53 disruption. This was initially treated with bendamustine-rituximab, with a resultant partial response. Three weeks before the acute presentation, he was determined to have progression of CLL with worsening lymphocytosis (129 x 10<sup>9</sup>/L), thrombocytopenia (78 x 10<sup>9</sup>/L), night sweats and weakness. At that time, his serum sodium (Na) was 132 mmol/L. He was recommended to initiate treatment with the oral BTK inhibitor ibrutinib, but this was</p>

	<p>deferred because of cost concerns. Ten days prior to the acute presentation, he was admitted to his local hospital with generalized weakness, fatigue, diarrhea, and nausea. Na was 116 mmol/L. There was a small retrocardiac opacity on chest X-ray, so he was empirically treated for community acquired pneumonia with ceftriaxone and azithromycin. The hyponatremia was thought to be syndrome of anti-diuretic hormone secretion (SIADH) given his euvolemic status and urine osmolality of 322 mOsm/kg. He initiated fluid restriction and salt tablets. Na minimally improved to 123 mmol/L at discharge.</p> <p>He presented to our institution three days later because of worsening symptoms, as well as new mild confusion. Computed tomography of the head was unremarkable. He was afebrile with a normal blood pressure (114/72 mmHg), and clinically euvolemic. There were no signs of pneumonia. Na was 119 mmol/L; Urine osmolality was 398 mOsm/kg. Fluid restriction and urea (15 grams thrice daily) were recommended. A cosyntropin stimulation test was performed the following morning because the etiology of SIADH was unclear. In the interim while waiting for the results, he continued fluid restriction and urea, but Na only improved to 122 mmol/L. When the cortisol levels finally resulted 36 hours later, his initial morning level had been 1.9 µg/dL (normal, 7-25), rising to 7.6 µg/dL one hour after cosyntropin injection (normal, ≥18 µg/dL), signifying adrenal insufficiency. Thyroid stimulating hormone was 1.7 mIU/L. He was treated with stress-dose hydrocortisone. 48 hours later, Na corrected to 138 mmol/L. His symptoms resolved significantly, and he was discharged with a prednisone taper. One week later, he started ibrutinib. Six weeks later, he had functionally returned to baseline. Na was 142 mmol/L and his morning cortisol was 17 µg/dL, suggesting the hypothalamic-pituitary-adrenal axis recovered function. He discontinued corticosteroids thereafter.</p> <p>Discussion: Herein we describe a patient with progressive CLL who presented with symptomatic hyponatremia in the setting of adrenal insufficiency. The cosyntropin stimulation test results suggested that this was either primary or chronic secondary adrenal insufficiency, the relation of which to his CLL is curious and has not been previously described.</p>
<p><b>Marin Olson</b></p>	<p><i>A Middle-Aged Man with Weight Loss and Night Sweats</i></p> <p>Case Presentation: A 52 year old man with a history of type II diabetes, a heart murmur since childhood, and inconsistent medical care was worked up by his family medicine team for 15 pounds weight loss, night sweats, and nocturia with occasional blood in his urine. As he immigrated from Mexico 30 years ago, the team was particularly concerned for tuberculosis and obtained a quantiferon gold test which was negative. Due to his lack of medical insurance, he wished to limit labs and visits. Several weeks following his initial visit, he was seen in the emergency department with persisting symptoms, and additional dizziness, non-bloody cough, shortness of breath, and tachycardia. The emergency physicians were concerned for malignancy and obtained imaging which demonstrated a lower lobe lung cavity on the right, but no other concerning masses. He was discharged home and followed up two weeks later with urology to investigate his hematuria. Urology noted no explanations for his symptoms on US or CT, and he returned once more to his family medicine physician. At just over two months since his initial family medicine visit, he noted 25 total pounds of weight loss, a night cough, soaking night sweats, and new bloody vomiting.</p> <p>He was admitted to the hospital with a long problem list including weight loss with night sweats and hemoptysis, acute kidney injury with hyperphosphatemia and hyponatremia, thrombocytopenia and anemia, gross hematuria, and a heart murmur. A TTE showed a tricuspid mass, and blood cultures were positive for strep mitis/oralis. He was diagnosed with subacute bacterial endocarditis, with poor dentition as a likely source of infection. Though a kidney biopsy was deferred due to low platelets, he was also thought to have immune complex mediated glomerulonephropathy. His team asuspected that bone marrow suppression secondary to infection was the cause of his anemia and thrombocytopenia. He was treated with ceftriaxone, had a permanent</p>



	<p>dialysis catheter placed, and was discharged to begin outpatient dialysis and continued vancomycin treatment.</p> <p>Discussion: This case illustrates the complex constellation of symptoms possible in a patient with infective endocarditis (IE), which can mimic many other concerning disease processes. IE can present acutely, subacutely, or chronically and symptoms may be vague and nonspecific such as fever, anorexia, myalgias, dyspnea and night sweats. This patient was thoroughly worked up for malignancy and tuberculosis, yet his symptoms are surprisingly common in IE. Equally interesting in this case is that although post-streptococcal glomerulonephritis (PSGN) is often taught as following streptococcal pharyngitis, it can also be a complication of IE. This patient’s simultaneous presentation of IE and PSGN required a broad differential and thorough workup. In sum, when treating a complex patient with nonspecific symptoms, IE should not be forgotten in the differential.</p>
<p><b>Olivia Ondigi</b>  Dr. Natthappon Angsubhakorn  Dr. Bradley Bart  Dr. Stefan Bertog</p>	<p><i>Acute Pericarditis with Unexpected Cardiac Tamponade: To Tap or not to Tap?</i></p> <p>Introduction: Cardiac tamponade is a life-threatening syndrome caused by an excessive fluid buildup in the pericardial space leading to compression of cardiac chambers and ultimately cardiovascular collapse. Acute pericarditis with concomitant pericardial effusion and tamponade physiology can present as a diagnostic and therapeutic dilemma. Here, we share an unexpected discovery of cardiac tamponade associated with acute pericarditis and dive into the thought process of whether to pursue pericardiocentesis.</p> <p>Case description: A 69-year-old male with no prior cardiac history presented to the ED with a one-day history of shortness of breath and substernal pleuritic chest pain that improved with sitting up. He was afebrile and normotensive with unremarkable physical exam. Initial workup was notable for leukocytosis (17.6 k/L), elevated CRP and D-dimer, negative troponin I, and diffuse PR-segment depression with concave upward ST-segment elevation on 12-lead ECG, consistent with acute pericarditis. CT chest with contrast ruled out pulmonary embolism but revealed pericardial thickening. ANA, rheumatoid factor, ANCA, interferon-gamma release assays, and Lyme antibodies were all negative and the patients was diagnosed with acute idiopathic pericarditis. He was admitted and started on ibuprofen and colchicine. On hospital day three, his chest pain and shortness of breath completely resolved; however, transthoracic echocardiogram (TTE) demonstrated a moderate circumferential pericardial effusion with the deepest pocket of 1.51 cm, early diastolic RV collapse, hepatic venous flow reversal and dilated IVC of 2.4 cm with no respiratory collapse, suggestive of cardiac tamponade. His BP and pulse rate remained normal during entire hospital stay although he had pulsus paradoxus of 14 mmHg. In conjunction with the patient, the decision was made not to perform a pericardiocentesis given that the patient showed dramatic clinical improvement with medical therapy with no clinical evidence of cardiac tamponade. A follow up TTE two days later showed slight improvement of the effusion and resolution of diastolic RV collapse and hepatic venous flow reversal.</p> <p>Discussion: Diagnosis of acute pericarditis requires at least two of the following: typical pleuritic chest pain that gets better with sitting up and leaning forward, pericardial frictional rub, ECG changes, and new/worsening effusion. It can present concurrently with pericardial effusion which may lead to life-threatening cardiac tamponade in up to 5% of cases. As a result, TTE is highly recommended for all suspected pericardial disease to detect tamponade early, just like in our patient. Pericardiocentesis is indicated for clinical tamponade, suspected purulent or neoplastic pericarditis, or for large (&gt; 2cm), or symptomatic effusions despite medical therapy. Conservative management is appropriate for “early” tamponade with minimal or no evidence of hemodynamic instability given the risk of the procedure. Inflammatory effusions associated with early tamponade may resolve with NSAIDs and colchicine with serial echocardiograms, hemodynamic monitoring and avoiding volume depletion.</p>

<p><b>Payal Patel</b> Dr. Jason Szostek</p>	<p><i>Case: Hyponatremia Secondary to Itraconazole-Induced Primary Adrenal Insufficiency</i></p> <p>Case Presentation: An 84 year old female patient with past medical history of asthma, chronic bronchiectasis status post left lower lobectomy in 1959 and right middle lobe resection in 1996, normal pressure hydrocephalus, and recent hospitalization for hyponatremia seven months prior, presented to the Mayo Clinic emergency department (ED) with a three day history of nausea and decreased oral intake in the setting of a three month history of progressive weakness and cough with increased sputum production.</p> <p>On admission, the patient complained of generalized weakness. She had sodium of 127 in the ED. Other labs were significant for a TSH of 4.7 and CK of 198. She was initially treated for hyponatremia secondary to hypovolemia and was given 500mL normal saline bolus. Repeat sodium was 123. We noted her previous hospitalization for hyponatremia resolved with steroid treatment, so we proceeded to treat for adrenal insufficiency and performed further work-up. Her co-syntropin skin test on 7/19 was 5.6 at baseline with peak of 16, and was concluded as an "inadequate/sub-optimal" response. Her corresponding ACTH level was 111. She was started on hydrocortisone, 10mg in the AM and 5mg in the PM, later increased to 20mg in the AM and 10mg in the PM. Forty-eight hours later, her sodium normalized to 141. On the day of discharge, the patient's weakness in the arms and legs and foot cramps resolved and she felt significantly better. The patient's adrenal antibodies returned negative. The etiology for her adrenal insufficiency was then thought to be secondary to her intermittent Itraconazole use. Due to her complex respiratory history and recent development of worsening pulmonary symptoms, the patient had been on Itraconazole at the time of presentation.</p> <p>Discussion: Adrenal insufficiency is categorized as primary, secondary or tertiary. Patients with primary adrenal insufficiency are typically thought to have an autoimmune process (or tuberculosis in the developing nations) as the primary case. There are several case reports of patients experiencing anti-fungal induced adrenal insufficiency, mostly linked with usage of ketoconazole. These -azole medications inhibit the CYP3A4 pathway, interfering with the synthesis of glucocorticoids. There are currently a handful of reported cases of Itraconazole induced adrenal insufficiency; however most of these are with concurrent usage of inhaled steroids. We report this case to inform clinicians of the usage of Itraconazole in the long-term and the possible predisposition to the development of adrenal insufficiency. Physicians prescribing these medications should take caution if patients develop weight loss, hypotension, hyponatremia, and generalized weakness.</p>
<p><b>James Pathoulas</b> Dr. Walter Lizewski Dr. Ronda Farah Dr. Noah Goldfarb</p>	<p><i>Malignant Metastatic Umbilical Nodule: A Rare Paraneoplastic Dermatoses</i></p> <p>Introduction: Paraneoplastic dermatoses are cutaneous evidence of internal malignancy. Most internal malignancies do not have cutaneous paraneoplastic findings. However, cutaneous findings may be the first presentation of internal malignancies and early detection can lead to earlier diagnosis and better overall prognosis.</p> <p>Case Presentation: An 88-year-old man presented with a 3-month history of an asymptomatic, growing, friable pink tumor at the umbilicus. Since the nodule appeared, the patient noted progressive appetite and weight loss. On examination, the plaque was friable and indurated. Biopsy of the lesion was suggestive of a metastatic adenocarcinoma. A CT scan was subsequently performed which revealed a mass in the head of the pancreas. A fine needle aspiration of this mass demonstrated a pancreatic adenocarcinoma. Also known by the eponym of a Sister Mary Joseph nodule, internal malignancies can first present as a cutaneous metastasis at the umbilicus. Such presentations portend a poor prognosis. The differential diagnosis of this lesions includes a primary skin cancer, such as a squamous cell carcinoma or</p>

	<p>amelanotic melanoma. If the patient were a younger female, cutaneous endometriosis would also be in the differential for a periumbilical tumor. Ultimately, given the poor prognosis, the patient opted for palliative care.</p> <p>Conclusion: Paraneoplastic dermatoses can be the first sign of occult tumors and highlight the importance of a thorough skin examination in all patients.</p>
<p><b>Kaylin Pennington</b> Dr. Sam Ives</p>	<p><i>Lung Abscess Misdiagnosed as Pulmonary Infarction: A Cautionary Tale</i></p> <p>Introduction: Cavitory lung lesions may be due to lung abscess, tuberculosis infection or less commonly lung infarct.</p> <p>Case Presentation: This clinical vignette details a patient presenting with productive cough who was initially treated with anticoagulation for a presumed pulmonary embolism (PE) leading to pulmonary infarction. Two weeks later, the patient presented with worsening dyspnea on exertion concerning for untreated necrotizing pneumonia. Case Description: A 49-year-old man with a past medical history of alcohol use disorder and homelessness presented to the Emergency Department (ED) with a one-month history of worsening cough productive of rust-colored, foul-smelling sputum. The patient also reported recent onset of pleuritic chest pain that localized to his left anterior chest and worsened with breathing, moving and coughing. On exam, the patient was afebrile and lung exam was normal. CBC showed a mild leukocytosis and chest x-ray showed a “mass-like opacity in the left upper lobe.” Because of the abnormal x-ray, a CT chest was done. This showed a wedge-shaped region of consolidation with peripheral ground-glass with nonenhancement and mild cavitation in the left upper lobe. This was thought most compatible with a pulmonary infarction. Of note, after the initial CT was done, another CT was done to look for a PE (angiogram), which did not show a PE. A bilateral lower extremity ultrasound was also negative for DVT. At the time, it was noted that the patient did not have any clear risk factors for a PE, but due to the CT read, he was started on enoxaparin as a bridge to warfarin. On clinic follow-up, he was admitted with the cavitory lung lesion to rule out tuberculosis. Acid-fast bacilli sputum cultures were obtained and were negative times three. Of note, his anticoagulation was continued.</p> <p>Finally, two weeks after his initial ED visit, he was seen in clinic for a supratherapeutic anticoagulation (INR &gt;10) and ongoing cough and dyspnea on exertion. Upon review of his case, the initial diagnosis of pulmonary infarct was reconsidered and he was treated instead for a bacterial lung abscess. His anticoagulation was stopped and he was started on amoxicillin/clavulanate. On follow-up, his symptoms and pulmonary infiltrate resolved with antibiotics.</p> <p>Discussion: Lung abscess is defined as necrosis of the pulmonary parenchyma caused by microbial infection. Some authorities use the term “necrotizing pneumonia” or “lung gangrene” to distinguish pulmonary necrosis with multiple small abscesses from a larger cavitory lesion, but this represents a continuum of the same process. In this case, the initial CT reported a left upper lobe pulmonary infarction as the most likely diagnosis, although further work-up showed no evidence of PE on angiogram or bilateral lower extremity ultrasound and no elevation of inflammatory markers.</p> <p>Discussion: This case illustrates the importance of considering infectious etiologies for a wedge-shaped consolidation and upper lobe cavitory lesion. The case also highlights the need to consider more possibilities when new information points away from the initial diagnosis. Finally, this report could propose a low threshold for empirically treating necrotizing pneumonia after ruling out mycobacterial infection, as a typical presentation with indolent symptoms and putrid sputum is highly suggestive of anaerobic infection and cure rates with antibiotic treatment are high.</p>
<p><b>Karen Riley</b> Dr. Adinan Kanda</p>	<p><i>Mysterious Myalgia</i></p>

	<p>Introduction: Anti-synthetase syndrome is a rare disorder affecting women twice as often as males with an unknown frequency and prevalence. It is likely undiagnosed or misdiagnosed due to its rarity, making it relevant and important to discuss.</p> <p>Case Presentation: A 78-year-old woman presented to ED with 9-month history of increasing proximal muscle weakness and pain, along with a 6-month history of dyspnea. She was hospitalized at a different hospital one-week prior with chest pain and SOB, with unremarkable workup for acute coronary syndrome. Patient was subsequently started on a statin for primary prevention. CT was negative for pulmonary embolism but did incidentally identify a left inferior kidney mass. The patient had no symptoms of hematuria, flank pain or dysuria. Exam on admission was notable for profound proximal muscle weakness. Labs showed markedly elevated CK, aldolase, and CRP. ANA was notably positive. On admission, myositis panel was sent, statin was discontinued, and patient was started on physical therapy (PT). Her pain and weakness improved gradually with PT, and her CK continued to trend downwards. She was eventually discharged to an acute rehab facility, with a plan to follow up with rheumatology as an outpatient. Two weeks later, the patient was readmitted for worsening pain and weakness in her upper arms. Her CK was significantly increased compared to value at discharge. MRI showed inflammation in the hamstrings with moderate muscular edema, consistent with inflammatory myositis. A muscle biopsy was subsequently completed. Results of myositis panel returned positive for anti-synthetase antibodies (Anti – OJ tRNA synthetase); a rare variant of anti-Jo which is sensitive and specific for anti-synthetase syndrome.<sup>1</sup> She was initially started on high dose IV methylprednisolone which was later transitioned to high dose oral prednisone with a taper. She was also started on pentamidine nebulizer treatments for PJP prophylaxis. High resolution CT chest was performed to evaluate for interstitial lung disease (ILD), which showed limited ILD changes in the inferior lung fields, positive in 10% of dermatomyositis and polymyositis and more common in anti-synthetase syndrome.<sup>1</sup> Her weakness and pain improved significantly with steroids, and her CK began to trend downwards again. She was discharged back to the rehab facility for continued PT and a plan to follow up with rheumatology for initiation of cellcept, pending evaluation of her renal mass.</p> <p>Conclusion: This case illustrates the importance of getting a myositis panel early, the concern for underlying malignancy with inflammatory myositis, and the importance of stopping a statin in patients experiencing muscle pain with demonstrable weakness. Prognosis for Anti-synthetase syndrome is poor, thus early intervention is necessary to prevent rapid decline.</p>
<p><b>Londyn Robinson</b> Dr. Bukhari Burale</p>	<p><i>Immunocompromised Man with Worsening Exertional Chest Pain and Productive Cough</i></p> <p>Case Presentation: A 45-year-old male presented with sharp, sub-sternal chest pain on exertion and worsening productive cough over the past three months, particularly bad 4 days prior to admission and unrelieved by albuterol inhaler. Pertinent history: AIDS, PSUD (cocaine, heroin, ETOH), cigarette smoker, asthma, gout, pseudogout, and history of multiple gunshot wounds. He has had multiple infections from various etiologies including: PJP pneumonia, pulmonary tuberculosis, and mycobacterium scrofulaceum. Approximately three months ago he was engaging in heavy exertion when he developed severe angina and dyspnea, requiring him to stop and rest for one hour. Prior to this event, he was able to walk up to 2 miles and now he can walk less than half block. He has had night sweats, orthopnea, and PND almost nightly the past 30 days. Denies IV drug use in the past 30 days, smokes marijuana regularly. Has positive family history of coronary artery disease in mother, lung cancer in father and sister. He had 4 days of severe midsternal chest pain, non-radiating.</p> <p>Upon arrival to ED, CXR revealed mildly enlarged heart and chronic metallic fragments over right clavicle and left axillary region, unchanged from prior imaging. He was tachycardic, mildly tachypneic. EKG in ED evidenced questionable ST</p>

	<p>elevation inferiorly, with lateral, biphasic T waves, similar to prior EKGs. Single dose of nitro did not relieve the pain. After stabilization, he was admitted to the medicine floor. Pertinent labs - NT proBNP 1385; troponin slightly elevated at 0.031 then serial negative. Admission immunodeficiency panel revealed CD4 count was 7.3, absolute CD4 count 148 and HIV 1 RNA was 56 copies/mL.</p> <p>On hospital day 2, a pre-stress TT echocardiogram revealed: mild left ventricular hypertrophy, decreased left ventricular systolic function with EF 38%, restrictive left-sided filling consistent with elevated left pressures, severe posteriorly directed mitral regurgitation, and a non-specific abnormality on the anterior mitral valve leaflet, all new compared to 2016 TTE. The next day, a TEE evidenced moderate-to- severe mitral regurgitation with nonspecific thickening of the leaflet tips, flow anterior greater than posterior, with a single eccentric jet of mitral regurgitation.</p> <p>The patient, who presented with exertional angina and dyspnea was subsequently found to have HFrEF (EF 38%), severe mitral regurgitation due to single eccentric jet on mitral valve. Differential diagnosis included prior infective endocarditis and non-infective endocarditis. There was no evidence of vegetation, ruptured chord, flail segments, or prolapsed valve. Obstructive coronary artery disease could not be ruled out and patient had a coronary angiogram. Infectious disease consulted and patient remains in hospital pending mycobacterium subspecies results and new diagnosis of HFrEF due to severe mitral regurgitation probably caused by (non)- infectious etiology via endocarditis.</p>
<p><b>Joel Rosenberg</b> Dr. Kay Ingraham</p>	<p><i>Beer Goggles</i></p> <p>Introduction: Acute hyponatremia is a common finding in the hospitalized patient. Rapid correction of hyponatremia can have significant consequences including osmotic demyelination syndrome (ODM). Here we present a case of acute symptomatic hyponatremia with concern for ODM.</p> <p>Case presentation: A 42-year-old man with a past medical history of severe alcohol use disorder, depression, and morbid obesity presented to an outside emergency department by EMS after being found down with altered mental status. He was euvolemic by examination. Initial labs showed: serum sodium 117 mEq/L, potassium 5.4 mEq/L, chloride 86 mEq/L, bicarbonate 22 mEq/L, BUN 31 mg/dL, creatinine 2.1 mg/dL, glucose 128 mg/dL, with low serum osmolality 265 mosmol/kg. Urine studies revealed urine osmolality 121 mosmol/kg, and urine sodium of &lt;20 meq/L. The low urine osmolality in the setting of hyponatremia in an euvolemic patient was consistent with a low ADH state. He received 1L of iv normal saline and dexamethasone at the outside hospital. The patient was admitted to the Minneapolis VAMC ICU for further cares. Twelve hours after presenting to the outside hospital his serum sodium was 125 meq/L. In view of the rapid correction of his hyponatremia, he was switched to D5W. A history from the patient revealed he was drinking 50 beers per day to cope with his depression. A diagnosis of hyponatremia due to excessive beer consumption (“beer potomania”) was made.</p> <p>On hospital day 2 he started to have upper and lower extremity weakness in the setting of a serum sodium of 128 meq/L. This was concerning for ODM secondary to rapid correction of his hyponatremia after receiving IVF at the outside hospital. However, MRI of the brain and spinal cord showed no signs of ODM and the neurologic deficits slowly improved.</p> <p>Discussion: Hyponatremia due to excessive beer consumption, termed “beer potomania,” is unique in multiple ways. Patients with beer potomania often have a long history of significant beer drinking combined with a poor diet. Therefore, their solute intake and consequently solute excretion is very low thus decreasing the amount of water that can be excreted.</p>

	<p>The second unique pathophysiologic feature of beer potomania is the suppression of ADH release by alcohol. If ADH is suppressed, water excretion increases which can contribute to a rapid increase in the serum sodium especially when combined with solute infusion in the form of iv fluids. Rapid increase in serum sodium levels can potentially lead to osmotic demyelination syndrome.</p> <p>In conclusion, the high fluid intake and low solute load from excessive beer intake and poor diet can cause acute hyponatremia. Rapid solute infusion combined with suppressed ADH levels secondary to alcohol causing increased water diuresis can cause rapid correction of the hyponatremia and increases the risk for ODS.</p>
<p><b>Noah Sanders</b></p>	<p><i>When Horses Wear Stripes: Pneumococcal Pneumonia Masquerading as Mycobacterium Tuberculosis</i></p> <p>Introduction: Streptococcus Pneumoniae is a major cause of community-acquired pneumonia. Classically, it presents with fever, cough, and shortness of breath in a patient with lobar consolidation on chest imaging. However, atypical presentations may mimic other infectious processes and pose diagnostic challenges.</p> <p>Case Presentation: A 61 year-old woman with a medical history of schizophrenia, a positive Mantoux test, and tobacco use presented to the emergency department with hypoxemia and fever. She endorsed nasal congestion, rhinorrhea, chills, and a cough productive of green sputum. She denied hemoptysis and chest pain. Vitals were significant for tachycardia, fever, and hypoxia. Physical exam showed expiratory wheezing bilaterally. An Influenza A test was positive and a chest X-ray was negative. She was treated with Azithromycin, Duonebs, Oseltamivir, and Prednisone and admitted to work up a COPD exacerbation (in the setting of her tobacco use) triggered by viral influenza. Because her history of a positive Mantoux test raised concern for Mycobacterium Tuberculosis (TB) infection, a chest CT was ordered, which demonstrated a cavitory lung lesion in the left lower lobe. Three acid fast bacilli cultures and HIV studies were negative, but sputum cultures grew Streptococcus Pneumonia. Following a course of IV antibiotics inpatient, she was discharged on oral antibiotics with instructions to follow-up with an outpatient pulmonologist. A repeat chest CT two months after discharge showed complete resolution of her cavitory lung lesion.</p> <p>The majority of cavitory lung lesions are caused by anaerobic bacteria species such as Peptostreptococcus, Prevotella, Bacteroides, and Fusobacterium secondary to aspiration. Reactivation TB infection is a far more rare, but serious, cause of cavitory lung lesions. The diagnostic workup for a cavitory lung lesion includes chest imaging (radiograph or CT), sputum culture and gram stain, as well as evaluation of sputum for a putrid odor, which confirms the diagnosis of anaerobic infection. Of note, patients with cavitory lung lesions and an unclear diagnosis - especially those who have a history of positive Mantoux test, are immunocompromised, are from a TB-endemic area, or have other TB risk factors - must be placed on airborne precautions until the diagnosis of TB infection can be ruled out.</p> <p>Discussion: In this particular patient, the findings of a cavitory lung lesion and associated hypoxemia fever in the setting of a positive Mantoux test were highly suspicious for reactivation TB infection. Appropriately, the patient was placed on airborne precautions and given a thorough TB workup. As this case demonstrates, high suspicion is not sufficient for diagnosis and should not be used as an excuse to narrow broad-spectrum antibiotic treatment before acid fast bacilli cultures have come back. A common diagnosis presenting in an atypical manner must also be considered. If it sounds like a zebra, it might just be a horse</p>
<p><b>Rob Shaver</b> Dr. David Ewart</p>	<p><i>A Serologic Quandary: A Rare Case of RNA Polymerase III Antibody Positive Diffuse Morphea</i></p>

	<p>Case Description: A 71-year-old male presented to Dermatology clinic for rapidly progressive hardening of his skin. 6 months prior he noticed scattered “intertwining”, non-painful red patches across his arms and chest. The patches coalesced and subsequently hardened. The thickening did not involve his periarticular skin and did not impair his joint range of motion. The changes also spared his hands, wrists, and feet. His review of systems was positive for unintentional weight loss, fatigue, dysphagia, and vocal hoarseness. He denied further GI symptoms, dyspnea, lower extremity edema, and symptoms consistent with Raynaud’s phenomenon. On examination, indurated, taut, shiny skin changes involving the bilateral arms, abdomen, flanks, and lower legs were apparent. The face, distal extremities, chest, and back were spared. There were no telangiectasias, digital ulcerations, or nailfold capillary changes. Punch biopsy of right forearm revealed dense dermal and subcutaneous sclerosis with an inflammatory infiltrate consisting of lymphocytes and few eosinophils at the junction of the dermis and subcutis. Based on these findings, serologic evaluation for systemic sclerosis was performed. Antinuclear, anti-topoisomerase, and anti-centromere antibodies were negative, however anti-RNA polymerase III (RNAPol III) returned positive, prompting referral to Rheumatology. Antibodies to RNAPol III are associated with systemic sclerosis with rapidly progressive diffuse skin involvement, scleroderma renal crisis, and malignancy (1). Due to the latter association and his history, a thorough malignancy work-up was performed without any obvious signs of cancer. Chest CT and an echocardiogram showed no signs of interstitial lung disease or pulmonary arterial hypertension, respectively. The overall presentation was inconsistent with diffuse systemic sclerosis given the lack of distal skin involvement and the absence of Raynaud’s. Eosinophilic fasciitis was also on the differential, but he lacked any history of peripheral eosinophilia. Based on the clinical presentation and pathology, diffuse morphea was suspected. The patient’s skin improved dramatically on methotrexate monotherapy, with resolution over all of his body surfaces except for his shins which have not improved.</p> <p>Discussion: Morphea, or localized scleroderma, is an idiopathic inflammatory disorder that causes sclerotic changes in the skin without systemic involvement. It can present with single or multiple inflammatory and subsequently fibrotic plaques that may persist for years, depending on the severity of fibrosis. Systemic sclerosis and morphea are distinct yet pathologically-related and sometimes co-occurring diseases (2). There is a single report of a patient with longstanding circumscribed morphea developing anti-RNAPol III systemic sclerosis (3). To our knowledge, this is the first case of diffuse morphea in association with antibodies to RNAPol III. While serologic tests, such as RNAPol III antibodies, are useful in diagnosing patients with complex presentations, they should always be interpreted in the clinical context of the patient.</p>
<p><b>Nathan Stratton</b> Dr. Nacide Ercan-Fang</p>	<p><i>Management of a Diabetic Foot Ulcer with Charcot Deformity in a Patient with Cognitive Impairment</i></p> <p>Introduction: Cognitive status complicates chronic and acute disease management even without a dementia diagnosis, often over a period of many years and may make it challenging to implement best practices.</p> <p>Case Description: A 73-year-old male with a history of labile type 1 diabetes mellitus complicated by worsening mild to moderate cognitive impairment and peripheral vascular disease was admitted to the hospital for management of a presumably infected diabetic foot ulcer overlying a Charcot deformity that had worsened over 2 months after a period of protracted standing over several days. Wound cultures were positive for e. faecalis and s. haemolyticus. Foot X-ray was not suggestive of osteomyelitis or gas gangrene. The patient was started on a course of intravenous vancomycin and levofloxacin, later transitioning to oral levofloxacin.</p> <p>Management of his diabetes as well as his infected foot was confounded by worsening cognitive impairment first demonstrated 5 years prior to this presentation. He first completed a Montreal Cognitive Assessment version 7.1 (MoCA) five years prior to</p>

	<p>this presentation, with a score of 23/30 indicating mild cognitive dysfunction. Repeat MoCA in 2019 was 21/30. Over that time, he had lost the ability to adequately manage an insulin pump and was unable to effectively achieve control with an intensive regimen despite the addition of continuous glucose monitoring. At admission, the patient had a hemoglobin A1c of 10.1% and was severely hyperglycemic (Blood Glucose &gt; 500 mg/dl) documented by his continuous glucose monitoring device. His A1c had increased from an average of 8.4 in 2014 to 10.1 in 2019.</p> <p>Initial recommendations for his foot ulcer from orthopedic surgery included non-weight bearing status with regular wound checks. The patient was unable to comply with the weight limitation instead requiring placement of full contact casting, reducing wound checks to a weekly basis. These evaluations were notable for worsening of the ulcer to include a second site with tracking from the primary lesion at the first weekly change but gradual resolution with aggressive wound care at cast changes for the next three weeks.</p> <p>These concerns ultimately required a 14-day admission to a transitional care unit after hospital discharge. Ultimately the patient required casting for 4 weeks followed by transition to a customized offloading shoe. The patient's ulcer had successfully epithelialized by the time of his most recent evaluation 7 weeks after the initial casting.</p> <p>Discussion: Successful management of patients whose disease is complicated by neurocognitive impairment requires adapting treatment plans and, at times, considering nontraditional practices. In this patient, the risk of amputation was mitigated by a multidisciplinary approach from medicine and orthopedic surgery including utilization of frequent casting in wound management and close monitoring by a multidisciplinary team, ultimately led to a positive outcome.</p>
<p><b>Thomas Schmidt</b> Thomas Reimann Dr. Megan Shaughnessy</p>	<p><i>Steroids as culprit and cure: Acute Respiratory Distress Syndrome (ARDS) from Strongyloides stercoralis Hyperinfection</i></p> <p>Introduction: Strongyloides stercoralis is a soil-transmitted helminth found throughout the world in tropical and subtropical climates. Patients can remain infected for decades due to the capability of the parasite to autoinfect the human host. When patients are given immunosuppressive therapy such as corticosteroids, Strongyloides can then progress to a hyper-infection state that can disseminate to other organs and become life threatening.</p> <p>Case Description: A 59 year-old Ecuadorian man living in Minnesota for the past 20 years was hospitalized for suspected pneumonia due to E. coli and rhinovirus based on culture and PCR testing. There was also concern for acute eosinophilic pneumonia due to finding of 2% eosinophils on bronchoscopy and IgE of 3663. He was treated empirically with antibiotics and steroids, leading to resolution of symptoms.</p> <p>Two weeks after discharge, he had acute onset of hemoptysis after swimming in a Minneapolis lake. He was altered, dyspneic, and hypoxic upon arrival in the emergency department, leading to intubation and admission to the intensive care unit (ICU). CT showed diffuse pulmonary infiltrates and white blood cell count was elevated at 15.77. He was started on empiric antibiotics for possible pneumonia. Bronchoscopy showed diffuse alveolar hemorrhage (DAH), leading to reinitiation of steroids. After two days, he was extubated and transferred to the medical ward. Strongyloides IgG antibody was positive and an ova and parasite examination of sputum showed strongyloides larvae. Therefore, steroids were rapidly tapered and he was initiated on ivermectin for treatment of Strongyloides hyperinfection for a planned course to extend two weeks after documented parasite clearance from his sputum.</p> <p>Two weeks later he became dyspneic and was seen in the emergency department. He had missed three consecutive ivermectin doses due to insurance issues. He was</p>



	<p>admitted to the medical ward, but had significantly increasing oxygen needs, leading to intubation and ICU transfer. He was started on broad spectrum antibiotics for possible aspiration pneumonia. Bronchoscopy showed 12% eosinophils but no DAH. Albendazole was added to augment ivermectin. Over the course of his hospitalization, multiple sputum ova and parasite examinations were negative for Strongyloides larvae and the clinical picture became more consistent with ARDS. Due to increasing difficulty with oxygenation, steroids were given for ARDS with continued use of ivermectin. This, along with lung-protective ventilator settings and prone positioning, led to gradual improvement, although he required tracheostomy and discharge to a long-term care facility.</p> <p>Conclusions: Patients can remain chronically infected with Strongyloides stercoralis for life and may have minimal or misdiagnosed symptoms of infection. If a patient needs steroids and has lived or traveled in a Strongyloides stercoralis endemic area, check Strongyloides serologies and treat empirically with ivermectin. ARDS can be a significant complication of Strongyloides hyper-infection following initiation of immunosuppressive therapy.</p>
<p><b>Lucas Zellmer</b></p>	<p><i>Cardiac Cirrhosis Secondary to Tricuspid Regurgitation</i></p> <p>Introduction: Cirrhosis of the liver is a common manifestation of numerous clinical presentations. Of importance, the extensive differential for cirrhosis can potentially lead to diagnostic and treatment challenges both on the wards and in the clinic.</p> <p>Case Presentation: A 74-year-old man with history of COPD, atrial fibrillation, alcohol abuse, and chronic ascites presented to the emergency department complaining of shortness of breath and worsening ascites. Given his past medical and social history, cirrhosis secondary to alcohol abuse was expected and cardiac ultrasound was performed to rule out cardiac causes of volume overload. Echocardiogram showed severe right ventricular and atrial enlargement, decreased right side systolic function, and overwhelming tricuspid regurgitation. A CT-PE was also performed in the ED and significant pulmonary fibrosis was noted; a suspected result of previous smoking history and amiodarone use. Given the patient's clinical status, he was admitted and cardiology was consulted; a transthoracic echocardiogram was then performed that further highlighted the aforementioned cardiac findings. At this time, cardiac cirrhosis secondary to cor pulmonale was the suspected etiology of the patient's presenting symptoms. Pulmonology was then consulted and disagreed with the original evaluation of severe pulmonary fibrosis; they further recommended right heart catheterization to determine pulmonary pressures. Euvolemia was reached via successful diuresis and right heart catheterization was performed. Interestingly, catheterization failed to show pulmonary hypertension and suggested severe primary tricuspid regurgitation of unknown etiology. After establishing a definitive diagnosis and effectively stabilizing the patient, it was determined that he was safe to follow up with outpatient cardiology and was subsequently discharged. Currently, surgical valve repair is not being pursued due to potential right ventricular decompensation and the patient is instead being treated successfully with diuretics.</p> <p>Discussion: The case outlined above further highlights the importance of a broad differential for cirrhosis in a medically and socially complex patient. Although the initial suspected diagnosis of cardiac cirrhosis secondary to cor pulmonale lacks effective treatment, reaching the correct etiology for the presenting symptoms helped identify a treatable cause of cirrhosis for this patient. This clinical vignette also exemplifies the severe consequences of chronic tricuspid regurgitation. Thus, appreciation and recognition of the interplay between pulmonary, cardiac, and social factors in cirrhosis is of great importance for internists in any clinical setting.</p>

<b>Residents</b>	
<b>Quality Improvement - Residents</b>	
<p><b>Christopher Spoke</b> Dr. Lisa Callies</p>	<p><i>Using EMR Reporting Tools to Increase Provider Awareness of Hgb A1c Rates and Improve Diabetes Care and Follow up: A QI Project</i></p> <p>Background: The prevalence of diabetic adults in the US is estimated to be around 8%, with another 34% of the population at risk of developing diabetes. Interventions to improve glycemic control and reduce cardiovascular risk factors, such as hypertension, hyperlipidemia, and tobacco use, are crucial towards preventing long term diabetic complications. The ADA estimates that between 33-49% of patients fall short of meeting targets for glycemic control, blood pressure or cholesterol control. To address this, the ADA has recommended the Chronic Care Model to address barriers to optimal care, such as utilizing clinical information systems. Clinical information systems, such as EPIC report dashboards, can help reveal these gaps in outpatient diabetes care. EMR information systems can help create data sets such as diabetes registries, quality metrics on glycemic control, and rates of secondary cardiovascular risk modification.</p> <p>Objective: To improve comprehensive diabetes medical care within ANGMA residency clinic, electronic medical record systems were utilized to identify patients who have poorly controlled diabetes and secondary cardiovascular risk factors, so as to increase provider awareness and promote more office follow up for therapeutic adjustments.</p> <p>Methods: Using EPIC report dashboard tools, a diabetes registry of patients with a diagnosis of type 1 and type 2 diabetes was created within ANGMA resident medicine clinic population. A registry of 309 patients was created; patients who were seen in clinic within the previous 2 years were included. A recent Hgb A1c, blood pressure, statin/aspirin use, tobacco status, and use of CGM were collected. The data was organized by each resident’s clinic panel. Residents were educated on their patients’ A1c levels, as well as best practices for routine diabetes care according to the ADA guidelines. Post intervention analysis was performed from April through August 2019 to evaluate for improvement in glycemic control via A1c, use of CGM, and secondary cardiovascular risk reduction interventions.</p> <p>Results: Prior to the intervention, 69.3% of patients had an A1c in goal range of less than 8.0%. Post intervention data showed of patients with uncontrolled diabetes (A1c &gt;8%), 27.5% (14/51) saw an improvement in follow up with an A1c of less than 8, and of patients with a controlled A1C (&lt;8.0%) prior to intervention, 15.2% (16/105) were found to have uncontrolled diabetes (A1c &gt;8.0%). There was also a 117% increase in use of CGM post intervention.</p> <p>Conclusion: Data prior to the intervention showed there was room to improve glycemic control and reduce cardiovascular risk factors for many clinic patients. Post intervention, the A1c improved for a number of patients, but also worsened for others. This speaks to how glycemic control can be so variable and how important routine clinic follow up is in providing the optimal diabetic care for patients.</p>
<p><b>Christine Wagner</b> Dr. Breanna Zambinski</p>	<p><i>The Ultimate Antidote; Increasing Prescriptions Rates of Naloxone to High Risk Patients</i></p> <p>Introduction: Each year hundreds of Minnesotans lose their lives to the opioid epidemic and the number of these opioid related deaths has continued to rise. In 2017, 422 Minnesotans lost their lives to opioid overdose. Alarmingly, opioid prescription rates have decreased since 2016, yet overdose related deaths have continued to rise. Naloxone, an opioid antagonist can help restore breathing and reverse sedation &amp; unconsciousness in overdose. It is currently highly under prescribed to high risk</p>

	<p>patients.</p> <p>Objective: Identify patients of our residency clinic who are at high risk of opioid overdose, provide education to residents and improve naloxone prescription rates where appropriate.</p> <p>Methods: Through chart review, patients of the residency clinic were identified with chronic opioid prescriptions (&gt;3 months). Individuals at high risk for overdose were included or excluded based on opioid dose, choice of opioid, co-prescriptions and comorbidities. Opioid doses were converted into milligram morphine equivalents (MME) to represent the relative potency prescribed.</p> <p>Resident education was conducted 5/1/2019 describing the opioid epidemic and what we can do at a resident level to prevent harm and reduce risk. Residents were provided a list of their patients with chronic opioid prescriptions with an explanation on what characteristics make them high risk. Mechanism of action, availability, and formulations of naloxone were described and conversation starter examples to introduce Naloxone to patients were given. Additional education was provided midway through the project.</p> <p>Results: At initial chart review a total of 18 patients with chronic opioid prescriptions met criteria for being high risk for overdose based on relative dose of opioid &gt;50 MME and/or prescription for a long acting opioid. Of these, only 22.22% (4/18) had prescriptions for naloxone. Individuals were excluded from the high-risk group if they had active cancer, or were at end of life or palliative care. Post-intervention chart review showed the number of naloxone prescriptions increased dramatically, roughly 3 times the initial amount. Currently 61.11% (11/18) of patients have prescriptions for intranasal naloxone.</p> <p>Discussion: Given the unfortunate ubiquity of opioid overdoses in the population of late, naloxone and education of it is exceptionally relevant and arguably vital to the teaching of residents and other physicians. Though our Resident clinic does not have an incredibly large number of patients on chronic opioids, I believe it is noteworthy that naloxone was highly under prescribed. This is consistent with Naloxone prescriptions at a national level. With appropriate intervention, prescription rates increased 275% in our resident clinic. This is a topic and intervention that could be applied to other residencies and clinics that have been affected greatly by the opioid epidemic.</p>
<p><b>Abigail Wegehaupt</b>  Sneha Mohan  Aurora Norman  Sara Medina-Bielski  Courtney Harris  Samantha Ryan  Carina Himes  John Matulis</p>	<p><i>Improving Resident Satisfaction: Warm Handoffs in Clinic</i></p> <p>Objective/Purpose: We aimed to improve the internal medicine resident clinic rooming process. Baseline surveys revealed 30% resident dissatisfaction with the rooming process. Contrastingly, nurses and patients were largely satisfied.</p> <p>Problem statement: Rooming time on Baldwin 4 is perceived to take too long, leading to 30% resident dissatisfaction.</p> <p>Aim Statement: In an effort to improve resident satisfaction with the rooming process, we aimed to increase resident satisfaction by 20% in seven months.</p> <p>Methods/Approach: This study was conducted in the Community Internal Medicine Primary Care Clinic at Mayo Clinic in Rochester, MN. A root cause analysis suggested resident dissatisfaction was related to time wasted repeatedly checking for a notification that indicated patients were roomed and ready to be seen. This notification only appeared when the resident was on the schedule screen and was not accompanied by any alerts making it easily missed. This period of underutilized time was targeted for intervention. We implemented a new process that included a warm hand-off between nurses and residents when the patient is ready to be seen. Serial surveys were performed to measure the primary outcome of increasing resident satisfaction. Nursing staff and desk operation specialists were also surveyed to assess balancing</p>

	<p>measures.</p> <p>Results/Findings: At week 1, 40.5% were satisfied, 35.1% were neutral, and 24.3% were dissatisfied. At week 4, 51.5% were satisfied, 17% were neutral, and 31.5% were dissatisfied. At week 4, residents reported receiving a warm handoff 68.6% of the time. Over 50% of residents reported increased productivity. 85.3% of residents wanted the change to be permanent. Nursing staff did not feel that this process affected their workflow. RNs reported occasionally completing warm handoff; they reported often leaving the paper at the resident’s computer (53% of the time). 75% of RNs were in favor of making this change permanent. 48% of the DOS reported opposition to the change, many stating the previous system of leaving the patient sheet at the door allowed them to track the resident’s location.</p> <p>Conclusion/Practical Implications: At 4 weeks of intervention, resident satisfaction increased by 11%. Over half of the residents felt the intervention increased their productivity in clinic. Overall, the intervention was positively received by both residents and nurses, with the majority wanting the intervention to become permanent. While the intervention was successful among nurses, who were identified as the balancing measure prior to intervention initiation, the front desk personnel expressed dissatisfaction. Future PDSA cycles are needed to address this new balancing measure.</p>
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**Research - Residents**

<p><b>Kevin Buda</b> Hardikkumar Patel Tyler Haddad Anil Jha</p>	<p><i>In-Hospital Mortality in Patients Undergoing Transcatheter Aortic Valve Replacement in Relation to Heart Failure Status in the U</i></p> <p>Introduction: Transcatheter aortic valve replacement (TAVR) is an increasingly common method of valve replacement in patients that are not candidates for open-heart surgery. In these patients, TAVR significantly reduces the rate of cardiac death and cardiac symptoms. Although comorbid conditions are often present in these patients, there is a dearth of national studies evaluating outcomes in the presence of heart failure. We investigate whether systolic and diastolic heart failure impact hospital utilization and mortality.</p> <p>Methods: The 2016 National Inpatient Sample (NIS) was used to conduct a retrospective analysis of all patients that underwent TAVR in 2016. Subgroups for systolic and diastolic heart failure were created to perform a 1:1 comparison of outcomes in TAVR patients based on ICD-10-CM Codes. Patients with non-chronic forms of heart failure and those who received percutaneous endoscopic aortic valve replacement were excluded. STATA Version 15.1 (College Station, TX) was used for statistical analysis. A univariate screen using a p-value of &lt; 0.2 was conducted to determine which variables to include in the multivariate regression analysis. Multivariate regression was used to calculate weighted nationwide estimates of in-hospital mortality, and length of stay. We adjusted for patient (age, sex, ethnicity, and Charlson Comorbidity Index), socioeconomic (median household income, insurance provider), and hospital factors (hospital bed size, region, teaching status, day and month of admission).</p> <p>Results: An estimated 40,005 pts who underwent TAVR in 2016 were identified. Of those, 2475 had systolic heart failure and 7830 had diastolic heart failure. After adjusting for confounders, patients with systolic heart failure had a longer median length of stay compared to those with diastolic heart failure (+0.483 days, p=0.013). In contrast, no significant differences were found for in-hospital mortality (OR 1.32, p=0.565).</p> <p>Conclusion: Our study is the first to show that there is no significant difference in in-hospital mortality in systolic versus diastolic heart failure patients undergoing TAVR. Further, systolic heart failure was associated with an increased length of stay.</p>
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**Matthew Glogoza**  
Dr. Jonathan Urbach  
Dr. David Tierney

*Team Based Tablet Ultrasounds Increase POCUS Performed by Residents*

Introduction: Point-of-care ultrasound (POCUS) is becoming an integral part of internal medicine training. Learning a new skillset, requiring significant hands-on practice to achieve competency, is difficult within the constraints of residency. Barriers to performing enough repetitions clinically to become proficient frequently include insufficient time and the interruption of daily workflow. We hypothesized that beyond having station-based laptop ultrasound units with phased-array and linear probes being available throughout the hospital, the addition of team-based tablet ultrasounds that could be carried in white coat pockets, with only a phased-array probe, would further minimize barriers and increase clinical POCUS volume by residents on inpatient services.

Methods: Methods: The Abbott Northwestern Hospital Internal Medicine Medside Ultrasound (IMBUS) Program began in 2010. Prospective mobile-tracking of ultrasound performance on resident cell phones began 7/2012. Station-based laptop ultrasounds were introduced to all hospital units 9/2013. Resident team-based tablet ultrasounds were added in 8/2017. Prospectively collected exam data, excluding outpatient and staff-performed exams, from baseline months 12/2015-4/2017 (laptops only) was compared to matched months 12/2017-4/2019 (laptops+tablets) due to seasonal variation in data. Paired t-tests by month were used for comparing volumes.

Results: Total patient exams performed during the laptop-only and handheld+laptop periods were 1,386 and 1,853 respectively. The mean increase in: 1) number of patients examined (26,  $p=0.002$ ), 2) number of exam areas examined (cardiac, abdominal, pulmonary, etc.) (29,  $p=0.021$ ), and number of items evaluated (86,  $p=0.073$ ) per paired-month all increased between the laptop-only and laptop+handheld periods. Volume of exams were stable across the first and second halves of both study periods. Frequency of tablet vs. laptop use for pulmonary, abdominal, and cardiac exams was 41.2% vs. 58.9%, 30.1% vs. 69.9%, and 49.2% vs. 50.8% respectively. Recording of exams was audited during the laptop only period and found to be 87%. Given a potential for increase in reporting rate during the handheld+laptop period accounting for the increase in volume, a theoretical analysis was done assuming 85% reporting during the laptop-only period and 100% reporting during this period which still demonstrated a significant mean difference in exams performed by paired-months between the two periods (16,  $p=0.048$ ).

Conclusion: The addition of team-based tablet ultrasounds increases the volume of ultrasound exams being performed by residents, even in an inpatient setting with cart-based laptop ultrasounds within 150 ft. of any patient room. Achieving POCUS competency requires performance of a large volume of normal and abnormal clinical exams. A combined program infrastructure of station-based laptop ultrasounds and “in-their-pocket” tablet devices reduces the common barriers of inadequate time and workflow disruption for residents, resulting in a larger volume of exams being performed within a POCUS training program.

<p><b>Christine Gruessner</b></p>	<p><i>Edema on Bone Marrow Biopsies: An Early Warning Sign of Ankylosing Spondylitis?</i></p> <p>Introduction: Ankylosing spondylitis (AS) is an inflammatory autoimmune disorder that predominantly affects the spine. If untreated it causes significant morbidity. AS often presents with symptoms that are both articular and extraspinal, including enthesitis, synovitis, and dactylitis. It may also be associated with nonarticular disease, such as uveitis, psoriasis, and IBD. Patients frequently carry the gene for human leukocyte antigen HLA-B27; 90-95% of patients with AS are positive for HLA-B27. Prevalence estimates of AS vary considerably, and has been shown to be higher than the national average in Minnesota. Aggressive intervention in the early stages of AS has been shown to have better outcomes in terms of symptom management, long term pain relief, prevention of bone loss, and suppression of inflammatory effects on bone. AS is often accompanied with the finding of bone marrow edema; in a recent study, bone marrow edema was present in 70% of AS patients. Radio graphic structural changes are seen on average 7-10 years prior to the clinical diagnosis of AS.</p> <p>Methods and Conclusions: The following is a study of 10 patients who have undergone bone marrow biopsy for abnormal cell counts, who several years later were diagnosed with AS. In each of these patients, bone marrow edema (ranging from mild to marked) was seen on their biopsies. The finding of edema is not customarily reported in bone marrow biopsy pathology reports; however, reporting the finding of edema on a biopsy may have important clinical implications, and may prompt screening early signs and symptoms of AS.</p>
<p><b>Diego Suarez</b> Laura Greenlund</p>	<p><i>Comparison of Outcomes and Adverse Events for Landmark Guided vs Ultrasound Guided Corticosteroid Injection for Plantar Fasciiti</i></p> <p>Introduction: Plantar heel pain is a common complaint in primary care clinics and many cases are attributed to plantar fasciitis. Initial treatment for this condition includes exercises to stretch the plantar fascia and calf muscles, avoidance of aggravating activities, use of supportive footwear and short-term use of anti-inflammatories. However, in patients with refractory symptoms, combination glucocorticoid-anesthetic injections are an option prior to considering invasive treatments such as surgical release. Although these injections are commonly offered as second line therapy, there is limited data regarding the most effective method of injection, duration of treatment response, or potential adverse events and their respective frequencies. Moreover, few studies have compared the injection technique used in order to establish a superior method.</p> <p>Methods: We conducted a retrospective, single institution study (Mayo Clinic/Mayo Health System) using chart review. We assessed the difference in outcome of ultrasound guided injections compared to those performed using landmark guides only. Specifically, we looked at time to repeat intervention or clinic visit, adverse events and demographics associated with a negative outcome. We considered an injection to be effective if its effects lasted at least 3 months or if plantar fasciitis was no longer reported as an acute complaint in subsequent visits. Overall, 485 injections were reviewed and both landmark guided and ultrasound guided injections showed similar success (72% versus 68% respectively). Adverse events were also similar among both groups.</p>

	<p>Conclusions: Our results suggest that plantar fascia injections may provide pain relief in a large amount of patients with recalcitrant symptoms after failing conservative measures. In addition, based on our results, the addition of ultrasound guidance may not significantly affect the effectiveness of the injections or reduce the associated adverse events. As such, even smaller community practices without ultrasound equipment may have the resources necessary to provide these injections as part of routine care of plantar fasciitis. Future studies may seek to validate our findings with a prospective study design. In addition, outcomes of glucocorticoid injections should be compared to those of newer alternative therapies such as platelet rich plasma or botox injections.</p>
<p><b>Clinical Vignette- Residents</b></p>	
<p><b>Paul Abear</b> Dr. Maliha Zafari</p>	<p><i>Diminishing Venous Returns</i></p> <p>Introduction: Deep vein thrombosis (DVT) is a common diagnosis encountered by most physicians, occurring at an annual rate of 1 per 1000 people per year. Several well-known risk factors for the development of DVT exist. However, rarely, anatomical venous malformations are present which can be predispositions to DVT.</p> <p>Case Description: A 21 year old female with a past medical history of asthma who was 5 months postpartum presented to the emergency department with bilateral leg pain and swelling of her right leg. Symptoms started one week prior and progressively worsened in severity. Right lower extremity ultrasound (US) was performed and showed extensive DVT from her right common femoral vein to her mid calf. She was started on an intravenous (IV) heparin infusion and admitted. On hospital day 2, she reported increasing pain in her left leg. Left lower extremity US showed extensive clot from left common iliac vein to peroneal vein below the left knee. Vascular Medicine was consulted. Computed tomography (CT) venogram showed extension of bilateral thromboses from the superficial femoral veins through the iliac veins and into the inferior vena cava (IVC). Additionally, the IVC was severely narrowed approximately 5 centimeters below the level of the renal veins. Extensive collateral veins were seen surrounding the IVC suggesting long-standing, possibly congenital, stenosis of the IVC. She was treated with catheter-directed thrombolytics, balloon angioplasty of the stenotic infrarenal IVC and bilateral iliac veins. She was discharged with apixaban and aspirin. Approximately one month later, patient had again developed swelling of her right leg. During a follow-up appointment with Vascular medicine, CT venogram was repeated and showed a right iliac venous thrombus extending distally through the popliteal vein. She was admitted and underwent mechanical thrombectomy and stenting of the right iliac vein. She remains on apixaban for anticoagulation and has continued follow-up with Vascular Medicine.</p> <p>Discussion: Congenital venous malformations occur in approximately 1% of the general population and are a rare risk factor for the development of DVT. Cases of venous malformations leading to DVT have been seen in individuals who present with unprovoked DVT, are young in age (<math>\leq</math> 40 years) and who have bilateral DVTs in the setting of a lack of other potential DVT etiologies. Venous malformations of the IVC can vary in severity from hypoplasia/stenosis (as seen in this case) to outright IVC agenesis. Though rare, the presence of a venous malformation leading to venous stasis as a cause for DVT should be considered in patients with similar presenting features and no other risk factors for the development of DVT.</p>
<p><b>Mohamad Adada</b> Dr. Edison Cevallos Dr. Maryam Mahmood</p>	<p><i>A Novel Multimodal Approach for the Treatment of Disseminated Fungal Infections in an Immunocompromised Host</i></p> <p>Introduction: Disseminated fungal infections are rare entities that are mostly seen in immunocompromised hosts. They are associated with increased morbidity and mortality, and often pose a treatment challenge. Herein, we present a case of disseminated polymicrobial infection in a severely immunocompromised host due to</p>

	<p>invasive fungal pathogens and multidrug-resistant bacteria that was successfully treated with an aggressive multimodal approach.</p> <p>Case Presentation: An 18-year-old female with a 3-month history of profound agranulocytosis, recently initiated on steroids due to suspected hemophagocytic lymphohistiocytosis (HLH), presented with fever, chills and purulent leg cellulitis initially thought to be a spider bite. Her absolute neutrophil count (ANC) was 0. She underwent incision and drainage, and was treated with a course of amoxicillin/clavulanate as outpatient with no improvement. Around the same time, she started developing URI symptoms and facial pain suggestive of sinusitis.</p> <p>She presented to our facility for further evaluation. She was found to have profound neutropenia (ANC=0) and an extensive necrotic leg wound with an extensive necrotic hard palate lesion. She underwent extensive surgical debridement of the leg wound, on which cultures grew <i>Stenotrophomonas maltophilia</i>, <i>Enterobacter cloacae</i>, and <i>Enterococcus faecalis</i>. Histopathology of the leg wound showed invasive fungal structures consistent with <i>Fusarium</i>. CT of the sinuses was suspicious for invasive fungal sinusitis and underwent sinus debridement. Biopsy of the palatal lesion grew <i>Fusarium</i> sp., <i>Neocosmospora vasinfecta</i>, <i>E. Faecalis</i>, <i>Stenotrophomonas maltophilia</i>, <i>Abiotrophia</i> and <i>Chyseeobacterium</i>.</p> <p>The patient was treated with a combination of liposomal amphotericin B (L-AmB) and voriconazole. In addition, she was treated with doxycycline, ceftazidime-avibactam, and trimethoprim-sulfamethoxazole. Given progression of the disease while on anti-microbials, she underwent subtotal palatectomy followed by daily palatal and sinus debridement, along with irrigations with L-AmB and voriconazole solutions. Patient continued to be neutropenic; and given the severity of her infections, daily granulocyte-colony stimulating factor (G-CSF) was started, and received multiple granulocyte infusions. Bone marrow biopsy showed granulocytic maturation arrest, with no other findings concerning for HLH, infection or malignancy. Her ANC improved, however she proved to be G-CSF dependent. Further genetic workup is currently under way to determine the cause of her severe neutropenia. Palate defect started showing signs of granulation after a week, and cultures became negative. Leg wound healed with the aid of the wound VAC. Patient was discharged home on 4 weeks- course of piperacillin-tazobactam, TMP-SMX and doxycycline with an extended course of voriconazole and L-AmB.</p> <p>Discussion: Disseminated <i>Fusarium</i>, <i>Neocosmospora vasinfecta</i>, is a rare occurrence. Grave outcomes with 100% mortality have been associated with persistent neutropenia and previous steroid use, both of which were present in this patient. Our novel treatment method consisting of an aggressive surgical approach in addition to multiple antifungals, and antibacterial agents along with bone marrow support, proved to be successful.</p>
<p><b>Alexandra Alejos</b> Dr. Melfer Anderson</p>	<p><i>Rhabdomyolysis Associated with Anaplasma Phagocytophilum</i> <i>Take Me BAC to the History</i></p> <p>Introduction: For patients with a single acute medical problem, there may be dozens of illness scripts to consider. However, when there are multiple organ systems involved, clinicians must additionally determine whether one organ is affecting the others, whether a more systemic disorder is at play, or whether multiple simultaneous etiologies exist – compounding an already complex process. We present a case emphasizing the importance of weighing these permutations to arrive at a correct diagnosis.</p> <p>Case Presentation: A previously healthy 48-year-old man presented to the emergency department two weeks following a self-limited upper respiratory infection now accompanied by worsening dyspnea on exertion, fatigue, and new onset lower extremity edema. He smoked ¼ pack per day and consumed alcohol intermittently. Physical examination revealed marked hypertension, cool extremities, elevated</p>



	<p>jugular venous pressure, bibasilar rales, absent third heart sound, and pitting bilateral edema. His labs were remarkable for elevated transaminases with normal alkaline phosphatase and bilirubin, a troponin of 2.77, creatinine of 1.9, and BNP of 3430. Unexpectedly, there was proteinuria and microhematuria. Chest radiography showed patchy infiltrates and small effusions bilaterally. The EKG showed unifocal PVC's. Ultrasound revealed increased renal parenchymal echogenicity concerning for medical renal disease as well as incidental findings of cirrhosis and portal hypertension. Echocardiography showed dramatically decreased EF of 15% and global hypokinesis. Right heart catheterization showed a cardiac index of 1.9 L/min/m<sup>2</sup> and pulmonary capillary wedge pressure of 40.</p> <p>Given concomitant pulmonary, cardiac, renal, and hepatic signs/symptoms, a broad work up was initiated. Alpha-1-antitrypsin deficiency, hemochromatosis, Wilson's disease, giant cell myocarditis and sarcoidosis were considered, as well as infectious etiologies including hepatitis and HIV. Evaluation for all these diseases was unrevealing. However, a repeat social history disclosed much more significant alcohol use than initially described. This discovery permitted linking several key abnormal findings. Ultimately, the cirrhosis was determined to be related to alcohol use, exacerbated by new onset heart failure. The new onset heart failure was thought to be most consistent with viral myocarditis, exacerbated by chronic alcohol consumption. Given the abnormal urinalysis, the renal dysfunction was felt to be post-infectious, exacerbated by heart failure. The patient stabilized rapidly with preload and afterload reduction, diuresis, and abstinence from alcohol.</p> <p>Discussion: This case emphasizes the importance of obtaining an accurate and complete social history and of repeating components of the history and exam when questions linger. Further, this case illustrates how multi-organ system dysfunction can be related to multiple simultaneous etiologies and their interactions with one another.</p>
<p><b>Adham Alkurashi</b>  Dr. Vuyisile Nkomo  Dr. Hasan Albitar  Dr. MarkENZler</p>	<p><i>Babesia and Borrelia Co-Infection,</i></p> <p>Introduction: Babesia microti, and Borrelia burgdorferi are tick-borne diseases transmitted by Ixodes scapularis tick, which is common in Midwestern states[1]. The tick ability to harbor multiple pathogens at the same time has allowed both diseases to occur in patients with tick bite at the same time. A recent study has found that coinfection can occur in up to 40% of cases[2]. Failure to recognize coinfection can result in severe complications and even death.</p> <p>Case Presentation: A 92-year-old male presented to the emergency department (ED) with fever and fatigue. The patient was treated for community acquired pneumonia after chest X-ray revealed a left upper lobe infiltrate. One month following discharge, the patient presented again to the ED with similar symptoms. Lab tests showed thrombocytopenia of 43 x10<sup>9</sup>/L (135 - 317 x10<sup>9</sup>/L), abnormal liver function tests of Bilirubin total 2.1 mg/dL, (&lt;=1.2 mg/dL), ALT 24u/l (7 - 55 U/L), AST 59 u/l (8 - 48 U/L), and Alkaline Phosphatase 148 u/(40 - 129 U/L). Patient also had hemoglobin of 11.6g/dl, (13.2 - 16.6 g/dL) hyponatremia of 131 mmol/l, (135 - 145 mmol/L), high creatinine of 1.44 mg/dl, (0.74 - 1.35 mg/dL), and C-reactive protein of 149mg/l, (&lt;=8.0 mg/L). On further questioning, the patient reported multiple tick exposure. A tick-borne panel was done, Lyme disease serology was positive and confirmed with positive immunoblot testing, in addition to positive Babesia microti PCR. 2.4% of red blood cells (RBC) were infected with babesia microti on peripheral blood smear. Therefore, the patient was started on Atovaquone 750 mg and Doxycycline 100 mg both orally twice daily, as well as Azithromycin 250 mg daily through intravenous (IV) route.</p> <p>Serial peripheral smears were performed and showed increasing parasite load. RBC exchange transfusion was considered, however; ultimately this was not pursued as the patient responded to treatment as evident by reduction in the RBC parasite load on peripheral blood smears. The patient's condition markedly improved and was discharged to a skilled nursing facility.</p>

	<p>Discussion: Coinfection with Babesia and Borrelia is not uncommon and can be life threatening[1], paying attention to the possible coinfection can ensure proper treatment and lower complications as the treatment differs as opposed to treatment of either organism in isolation. Babesia parasite load should be monitored closely to ensure adequate treatment as severe babesiosis patients may need blood exchange transfusion when they have high-grade parasitemia (<math>\geq 10\%</math>), severe hemolysis, or pulmonary, liver, or renal impairment[2].</p> <p>References:  [1] P.J. Krause, et al., Disease-specific diagnosis of coinfecting tickborne zoonoses: babesiosis, human granulocytic ehrlichiosis, and Lyme disease, Clin. Infect. Dis., 34 (2002), pp. 1184-1191</p> [2] Sanchez E, Vannier E, Wormser GP, Hu LT. Diagnosis, Treatment, and Prevention of Lyme Disease, Human Granulocytic Anaplasmosis, and Babesiosis: A Review. JAMA 2016; 315:1767.
<p><b>Mauricio Alonso Torrealba</b>  Dr. Sachin Mohan</p>	<p><i>Melanoma Camouflaging as Rectal Mass</i></p> <p>Introduction: Metastatic rectal melanoma is a rare entity, around 95% of the cases are identified post-mortem. The incidence of metastatic melanoma to the colon according to the literature is around 0.3%. Here we describe a patient with a metastatic rectal melanoma with PD-L1 of 15% expression and BRAF negative.</p> <p>Case Presentation: A relatively asymptomatic, long distance 60 year old truck driver, presented to the Gastroenterology clinic, for complaint of blood in the stools for several months. Hematochezia had been progressively worse for the last 2-3 weeks, with patient noticing blood on the toilet tissue every time he wiped the area. Patient denied abdominal pain. He reported prior history of hemorrhoids and no history of prior colonoscopies or upper endoscopies. His last hemoglobin check from 2017 was 15.2. No recent cross-sectional imaging had been done. Patient had no known drug allergies, he reported minimal alcohol intake and denies any smoking. No history of colon cancer on any first degree relative, but had a history of skin melanoma in his sister. Vital signs were normal. His BMI was 27.4 and no other abnormalities noted on physical examination. Hemoglobin one year prior was 15.2 with normal MCV. No other significant labs were obtained. Colonoscopy was recommended.</p> <p>Colonoscopy showed a frond-like villous, fungating, polypoid and ulcerated non-obstructing 5cm mass in the rectum with mild oozing. Biopsies obtain and showed a rectal melanoma. Tumor cells were positive for melanoma markers S100, SOX10 and panmelanoma.</p> <p>Subsequent CT A/P showed multiple liver metastases and multiple small indeterminate lymph nodes in the right upper quadrant omentum, right mesorectal fat and left internal iliac chain.</p> <p>Patient follow up with oncology and a large pigmented lesion in right buttock was noted which likely represents its primary (pending biopsies)</p> <p>Melanoma tissue from rectal mass showed BRAF negative with PD-L1 15% expression.</p> <p>Patient currently has been started on Nivolumab therapy with option for combination therapy with anti- CTLA-4 antibody Ipilimumab.</p> <p>Discussion: This case illustrates a rare diagnosis of metastatic melanoma to the rectum. According to the literature the median survival from diagnosis of large bowel metastasis is around 32 months, and overall survival rates at 1, 2 and 5 years was 68, 46 and 25% respectively. The most common symptom is rectal bleeding but often times can be asymptomatic. It is important to recognize that radiation therapy does not improve survival regardless of stage. In other case reports treatment has been immunotherapy but the response rate is not well known.</p>

<p><b>Deema Al-Souri</b> Dr. S. Allen Luis</p>	<p><i>Pancytopenia Secondary to Zinc Induced Copper Deficiency from Transient Constrictive Pericarditis: a Potentially Reversible Process</i></p> <p>Introduction: Constrictive pericarditis (CP) is a form of diastolic heart failure that arises because an inelastic pericardium inhibits cardiac filling. While this condition is typically regarded as being secondary to a fibrotic pericardium, a subset of patients may undergo spontaneous resolution or respond to medical therapy. The incidence of transient CP is estimated to be 9-17% of all CP cases, and failure to recognize this condition can result in undertreatment or inappropriate referral for surgical pericardectomy.</p> <p>Case Presentation: A 52 year old gentleman presents to the emergency with sharp pleuritic chest pain which was worse with sitting upright and lying down, associated with night sweats, on a background history of an upper respiratory tract infection 1 month prior to presentation and a remote history of self-resolving, acute inflammatory pericarditis approximately 25 years ago. Inflammatory markers were elevated with marked elevations in his transaminases, AST 131, ALT 311, and alkaline phosphatase 243. He was diagnosed with acute pericarditis and treated with ibuprofen 600 mg three times daily for seven days. However, 13 days later, he presented with worsening of his previous symptoms. Clinical examination revealed a loud three-component pericardial friction rub, and elevated Jugular venous pressure.</p> <p>Echocardiography revealed constrictive physiology, and a small pericardial effusion. Cardiac magnetic resonance (CMR) imaging showed moderately increased pericardial thickness, constrictive physiology, and bilateral pleural effusions. Colchicine 0.6mg twice daily, and Doxycycline 100mg/ twice daily were added to his therapy in addition to Ibuprofen 400 mg/ four times daily. Infectious diseases were consulted, and doxycycline was added for suspicion of leptospira. However, leptospirosis antibody, QuantiFERON, serology for toxoplasma, babesia, anaplasma, ehrlichia, Q fever, rickettsia typhi, and rocky Mountain spotted fever were all negative. Urine antigens for histoplasma and blastomyces were also negative. His symptoms eventually resolved, and his liver function tests also completely normalized after 14 days of therapy. His echocardiogram done after 16 days of treatment, showed marked improvement in the constrictive physiology.</p> <p>Discussion: Transient CP can result from significant acute or sub-acute pericardial inflammation. In this condition, pericardial inflammation with associated edema causes a loss of pericardial compliance resulting in constrictive pericardial physiology. In contrast to patients with classical fibrotic or calcific constrictive pericarditis requiring surgical pericardiectomy, such patients respond well to aggressive anti-inflammatory therapy with subsequent resolution of pericardial inflammation and the associated constrictive physiology. However, anti-inflammatory therapy may need to be prolonged and tapering guided by normalization of inflammatory markers. Assessment of inflammatory markers and CMR imaging should be considered in CP to exclude asymptomatic acute pericardial inflammation. This case also highlights the importance of a trial of medical therapy when treating CP, as the diagnosis of transient CP can only be made in retrospective manner if the patient improves.</p>
<p><b>William Archibald</b> Dr. Emily Leasure</p>	<p><i>Passing Out from Protein Passing</i></p> <p>Introduction: According to the US Renal Data System, 21.1% of people age 60 or older have an eGFR &lt;60%. 4% of Medicare patients aged 66+ have had at least one acute kidney injury hospitalization. As a result, it is critical for the internist to know the common causes of kidney dysfunction in the older adult, as well as rare causes that necessitate specialist involvement or targeted therapies.</p> <p>Case Presentation: A 69 year-old man with a history of benign prostatic hyperplasia maintained on doxazosin therapy presented to his physician complaining of syncopal</p>

	<p>episodes in the setting of a sinus infection lasting greater than 1 month. Initial evaluation consisting of BMP, CBC, TSH, chest x-ray, and EKG, was unremarkable with the exception of an elevated creatinine of 1.74 from a baseline of 1.1. Given concern that doxazosin was contributing to his syncopal episodes, he was instructed to stop doxazosin and initiate tamsulosin therapy. Urinalysis revealed &gt;100 RBCs and proteinuria. He was instructed to avoid NSAIDs. Three weeks later, on reevaluation, repeat urinalysis demonstrated worsening proteinuria and hematuria without dysmorphic RBCs or casts. Repeat BMP demonstrated rising creatinine. Hemoglobin had decreased by 2.3 g/dL (12.0 to 9.7). He was triaged to the emergency department for further evaluation due to concern for a rapidly progressive glomerulonephritis.</p> <p>In the emergency department, retroperitoneal ultrasound demonstrated unremarkable findings. He was admitted to the hospital where a comprehensive workup of glomerulonephritis and syncope was pursued, consisting of urine studies, ANCA panel, TTE, and autoimmune studies. Kidney biopsy showed a focal segmental necrotizing and crescentic glomerulonephritis consistent with pauci-immune vasculitis. Serologies returned with positive p-ANCA and positive MPO at greater than 8. This established the diagnosis of microscopic polyangiitis.</p> <p>The patient was given 2 g of methylprednisolone and started on prednisone 60 mg daily, pneumocystis prophylaxis with sulfamethoxazole-trimethoprim, and weekly rituximab. His syncopal episodes resolved. Three months later, the patient achieved remission with stable creatinine of 2.7, decreased anti-MPO antibody, and normalization of CRP and ESR. A slow steroid taper was begun.</p> <p>Discussion: The differential of worsening renal function in the older adult most commonly includes medication, obstructive uropathy, hypertensive nephropathy, and diabetic nephropathy. Urinalysis is critical when attempting to differentiate common causes of worsening renal function. ANCA-associated vasculitis with an estimated prevalence of 0.01% should be suspected in the patient presenting with hematuria and subnephrotic proteinuria; ANCAs are positive in approximately 82-94% of patients with an ANCA-associated vasculitis. In order to establish the diagnosis, biopsy of the affected organ is required, however, in the severely ill patient, empiric treatment can be performed. The goal of treatment with immunosuppression with corticosteroids combined with either rituximab or cyclophosphamide is to induce remission and prevent relapse.</p>
<p><b>Hadiyah Audil</b> Dr. Prema Peethambaram Dr. Marcia Venegas Pont</p>	<p><i>Tendon Rupture in the Setting of Aromatase Inhibitor Use</i></p> <p>Introduction: Aromatase inhibitors (AIs) are common agents for treatment of hormone receptor-positive breast cancer, with some studies estimating half of all invasive breast cancers as being eligible for AI treatment. AIs have largely replaced tamoxifen, hitherto the cornerstone of hormone receptor-positive breast cancer treatment in post-menopausal women, as preferred adjuvant therapy. Furthermore, studies have demonstrated efficacy of AIs for primary prevention of breast cancer in high-risk post-menopausal women, rendering use of these drugs extremely prevalent. Common risks/side effects of AIs include menopausal symptoms, increased risk of osteoporosis and bone fracture, and myalgias/arthralgias. Severe tendinopathy or tendon rupture secondary to AI use is exceedingly rare, with only four cases documented in the literature to date.</p> <p>Case Description: A 59-year-old female presented with acute right groin pain beginning two weeks prior to admission. Medical comorbidities included scoliosis, osteopenia, morbid obesity (BMI 44.3), and history of left breast invasive ductal carcinoma with micrometastasis to one lymph node, diagnosed in mid-2015, in remission since late 2015; she was status-post lumpectomy with sentinel lymph node biopsy and adjuvant radiation therapy, currently on adjuvant letrozole therapy. She denied any inciting factor for her groin pain (no recent trauma or strenuous activity), reporting she was asleep when the pain began. The pain radiated down her right leg and was progressive in severity, to the point where she eventually could no longer</p>

	<p>ambulate and was in a wheelchair. Upon visiting her local ED following onset of pain, MRI was concerning for trochanteric bursitis. At outpatient post-ED follow-up, CT suggested tendinitis. At subsequent follow-up with her primary care physician, repeat CT showed femoral hematoma with possible extravasation. Her only management was conservative pain medication. She then presented to our ED, and upon admission repeat MRI showed tear of the right rectus femoris adductor aponeurotic plate consistent with an athletic pubalgia-type injury, with consequent minor and stable femoral hematoma. She was discharged with optimization of pain medications and outpatient physical therapy, to be followed by possible surgical intervention if unsuccessful.</p> <p>Discussion: Our patient did not have typical risk factors for her pubalgia-type injury: she was not an athlete and had no recent strenuous activity or even baseline exercise regimen; rather, she was obese with a sedentary lifestyle, and had no risk factors for tendon rupture other than current AI therapy. She was not on any other medication known to cause tendon rupture. Review of the literature uncovered the risk of severe tendinopathy and tendon rupture in rare cases of AI use, and she was counseled on discontinuing letrozole. Given the widespread use of AIs for breast cancer treatment and chemoprophylaxis, it is essential to consider the risk of AI-associated tendinopathy and tendon rupture after other causes have been ruled out.</p>
<p><b>Ryan Balko</b> Dr. Karen Mauck</p>	<p><i>Lost in Translation: When Cystatin C and Creatinine Tell Different Stories</i></p> <p>Introduction: Creatinine and cystatin C are surrogate markers used to estimate glomerular filtration rate. Creatinine is a freely filtered, partially secreted molecule generated by skeletal muscle cells. Creatinine is eliminated from the body as an unchanged molecule in voided urine. Cystatin C is a freely filtered protein generated at a constant rate in all nucleated cells. Following filtration, &gt;99% of all cystatin C is reabsorbed by proximal tubule cells where it undergoes degradation. Distal to the kidney, creatinine is similar in concentration to serum creatinine, but cystatin C is undetectable. Understanding the difference in metabolism between creatinine and cystatin C is important when measured values are discordant.</p> <p>Case Presentation: Mr. MJ is a 74-year-old male with past medical history of CKD stage 2 and BCG-resistant urothelial carcinoma in situ. He was admitted for elective cystoprostatectomy with ileal neobladder formation. A JP drain was left adjacent to the ileal neobladder. Immediately post-operatively, creatinine was 1.1 mg/dL which corresponded to eGFR by 2009 CKD-EPI of 65 mL/min/BSA. Daily creatinine remained unchanged until POD 4 when creatinine abruptly rose to 3.63 mg/dL (eGFR 19 mL/min/BSA). Concurrent cystatin C was 0.75 mg/L (eGFR 102 mL/min/BSA). JP drain output remained &lt; 25 mL/day. Sampled JP drain fluid showed a creatinine of 3.8 mg/dL, similar to measured serum creatinine. On POD 5, creatinine peaked at 4.67 mg/dL (eGFR &lt;15 mL/min/BSA). Cystatin C remained 0.75 mg/L (eGFR 102 mL/min/BSA). The remainder of his electrolytes remained within normal limits and he maintained a urine output of &gt; 1mL/kg/hr with adequate PO intake. On POD 6, CT cystogram showed extraluminal extravasation of contrast at the anastomotic junction of the ileal neobladder and urethra, consistent with an anastomotic leak. Nephrology was consulted and conducted a comprehensive review of medications and exposures. It was determined the elevated serum creatinine reflected systemic reabsorption of creatinine from the urinary anastomotic leak, rather than reflection of renal insufficiency. He was managed conservatively with continuous bladder irrigation and urinary catheterization. On POD 7, creatinine abruptly decreased to 1.4 mg/dL (eGFR 49 mL/min/BSA). The JP drain was subsequently removed, and the patient was discharged home.</p> <p>Discussion: Estimating renal function is done by measuring surrogate serum markers, most commonly creatinine and cystatin C. Both molecules are freely filtered at the glomerulus. However, they differ in subsequent metabolism. Cystatin C is almost completely reabsorbed and degraded by the proximal tubule cells. Creatinine remains an unchanged molecule and is eliminated from the body in voided urine. While both</p>

	<p>creatinine and cystatin C are measured to estimate glomerular filtration rate, discordant values may reflect the normal renal handling of the two molecules. In a patient who has recently undergone urologic surgery, rising creatinine with normal cystatin C should prompt evaluation for intraabdominal urinary leak.</p>
<p><b>Tess Baril</b> Dr. Oana Dickinson</p>	<p><i>MINOCA in Minnesota</i></p> <p>Introduction: 6% of patients who present with acute myocardial infarction have no evidence of coronary artery disease. These patients are often diagnosed with myocardial infarction with non-obstructive coronary arteries (MINOCA). A high level of suspicion and understanding of the correct imaging studies to obtain are critical in order to make the correct diagnosis and provide the best treatment for these patients.</p> <p>Case Presentation: A 32-year-old female with a past medical history of anxiety presents to the emergency department with acute onset severe, left-sided, stabbing chest pain. Rated 9/10, non-radiating pain. She has had two uncomplicated pregnancies, children are now 16 months and 5 years old. She is a nonsmoker, drinks one glass of wine three times a week with no illicit drug use. Denies family history of cardiac disease. She endorses recent stress with family visiting over the holiday season.</p> <p>At admission, she is in mild distress, heart rate 71 beats per minute, blood pressure 129/80 mmHg, respiratory rate 14 breaths per minute and oxygen saturation is 99% on room air. Cardiac exam reveals normal rate and rhythm, no murmurs. Troponin is elevated at 0.10 ng/mL. Basic metabolic panel and complete blood count are normal. Electrocardiogram (ECG) demonstrated ST depression in V3-V5. Computed tomography angiography chest is negative for aortic dissection or pulmonary embolism. Transthoracic echocardiogram showed an ejection fraction of 60%, normal left and right ventricular size and function, normal cardiac valves. Heparin drip, aspirin, and clopidogrel load were started. Troponin peaked at 0.30 ng/mL. Subsequent coronary angiography demonstrated spontaneous coronary artery dissection (SCAD) of the first obtuse marginal artery (OM1). Treatment continued with aspirin, clopidogrel, amlodipine daily and metoprolol tartrate twice a day.</p> <p>Discussion: This case highlights the importance of maintaining a high level of suspicion for SCAD in young women presenting with chest pain. The case reviews how to diagnose SCAD as well as approaches to management. Coronary angiography is the gold standard for diagnosing acute SCAD, however, if this is negative the clinician must consider intravascular ultrasound (IVUS) or optical coherence tomography (OCT) to increase diagnostic yield.</p> <p>Treatment of SCAD remains somewhat unclear due to the lack of randomized control trials. Studies demonstrate a SCAD will heal with conservative management and when percutaneous coronary interventions (PCI) are done, there are higher rates of complications. If the patient is unstable or SCAD affects proximal LAD, PCI should be considered. The role for antiplatelet agents in absence of stent placement is unclear. Treatment should be aimed at preventing recurrence. Beta blockers and control of hypertension have been found to reduce episodes of recurrent SCAD. Therefore, it is important to determine the etiology of chest pain in a young woman in order to direct proper treatment and improve outcomes.</p>
<p><b>Grace Braimoh</b> Dr. Cole Pueringer Dr. Jason Baker</p>	<p><i>A “Whipling” Case of Diarrhea</i></p> <p>Introduction: Whipple’s disease is a rare, slowly progressive infection caused by <i>Tropheryma whipplei</i>. Herein we present a case of a man with HIV presenting with diarrhea, abdominal pain, cardiac abnormalities, and intra-abdominal lymphadenopathy. 16S RNA sequencing of lymph node tissue revealed <i>T. whipplei</i>.</p> <p>Case Presentation: A 50-year-old male with HIV diagnosed 3 years prior, on anti-</p>

	<p>retroviral therapy (Atripla), presented to clinic with two weeks of diarrhea and foul smelling stools. He was treated empirically for Clostridioides difficile colitis despite a negative infectious workup. His CD4 count is 559 cells/mm<sup>3</sup> (nadir of 354)</p> <p>Three months later he was evaluated for abdominal pain. He had lower extremity and scrotal swelling on examination. Transthoracic echocardiogram (TTE) showed non-specific densities of the aortic and mitral valves. Abdominal CT showed intra-abdominal lymphadenopathy. His abdominal pain improved in weeks without intervention.</p> <p>Two months after, he presented with cough and dyspnea. Imaging revealed an acute pulmonary embolism. Examination was notable for tenderness to palpation of his lower abdomen and unchanged dependent edema. Labs were normal except for mild hypoalbuminemia, slightly elevated alkaline phosphatase, and leukocytosis of 9-13,000 cells/mm<sup>3</sup> with a normal differential.</p> <p>Repeat TTE and abdominal CT showed slightly larger valvular vegetations and worsening abdominal lymphadenopathy respectively (image 1). Abdominal lymph node fine needle aspiration revealed sterile non-caseating granulomas. Thereafter, laparoscopic lymph node biopsy was performed which showed questionable debris on periodic acid Schiff (PAS) staining concerning for bacilli-like structures (Image 2,3). As a result, 16s ribosomal RNA sequencing was performed which confirmed Tropheryma whipplei.</p> <p>Discussion: Tropheryma Whipplei, an intracellular gram-positive bacillus found naturally in the environment. George Hoyt Whipple first described it in 1907(a). Transmission is fecal-oral and often associated with occupational exposure to soil or animals. Manifestations begin with a prodromal stage of migratory arthralgia and nonspecific symptoms, followed by a steady-state stage characterized by malabsorption, occult GI bleeding, lymphadenopathy, neurologic signs (33% of cases) or blood culture negative endocarditis with cardiac vegetations.</p> <p>In our case, we suspect the patient's immunodeficiency predisposed him to infection and uninhibited T. Whipplei replication in the intestinal tract. Suspicion for Whipple's disease should prompt tissue biopsy or fluid analysis with PAS staining and PCR sequencing (b). A positive PAS and PCR yields a definitive diagnosis while one of two indicates possible disease. Treatment involves prolonged antibiotic therapy, typically parenteral ceftriaxone followed by 1-2 years of Trimethoprim/Sulfamethoxazole (c).</p> <p>References:  a)Whipple, G. H. (1907). "A hitherto undescribed disease characterized anatomically by deposits of fat and fatty acids in intestinal and mesenteric lymphatic tissues".Bulletin of Johns Hopkins Hospital. 18: 382–93.  b)Identification of uncultured bacillus of Whipple's disease. Relman DA, Schmidt TM, MacDermott RP, Falkow S N Engl J Med. 1992;327(5):293  c)Molecular evaluation of antibiotic susceptibility of Tropheryma whipplei in axenic medium. Boulos A,Rolain JM,Mallet MN,Raoult DJ Antimicrob Chemother. 2005;55(2):178</p>
<p><b>Bukhari Burale</b></p>	<p><i>Methamphetamine Induced Pulmonary Hypertension</i></p> <p>Case Presentation: 35 year old female with history of polysubstance abuse, alcohol dependence and homelessness presented with progressively worsening of dyspnea on exertion for almost 6 months to the point of shortness of breath with walking 10 steps. She also reports cold intolerance, fatigue, hair loss and anxiety. on physical exam: vitals signs notable for hypertension, diastolic &gt;systolic. lung and heart exam unremarkable. She has bilateral lower extremity edema. Also noted to have redness and skin changes of both legs resembling livedo reticularis and both hands from knuckles down to finger tips resembling Raynaud's picture with delayed capillary refill for 3 seconds. labs notable for slightly elevated troponin to 0.031, proBNP of</p>

	<p>3121, Lytes WNL, Creatinine 1.5 (unknown baseline), CBC pertinent for Hgb of 8.4 (unknown baseline). TSH of 160. Her urine drug screen positive for methamphetamine. TTE showing dilated right heart, severe RV systolic dysfunction, severe pulmonary hypertension with estimate pressure of 83 and dilated IVC without respiratory variation. Basically she has severe PHTN, cause is unclear. Possibly idiopathic type 1 for pulmonary hypertension. Rheumatology work up for possible vasculitis and Raynaud came back negative.</p> <p>Discussion: Methamphetamine abuse can lead to drug induced PHTN. Given increased prevalence of methamphetamine use, we may see increase prevalence of pulmonary hypertension in this patient population and I wanted to shine a light on this.</p>
<p><b>Scarlett Cao</b>  Dr. Frederique St. Pierre  Dr. Omid Salaami  Dr. Chansong Choi  Dr. Christopher Ha  Dr. John Ratelle</p>	<p><i>Dietary Supplementation; An Unusual Presentation of Hypercalcemia</i></p> <p>Introduction: Hypercalcemia is a commonly encountered disorder in the hospital setting; patients with severe hypercalcemia can often present with gastrointestinal, musculoskeletal, renal, cardiovascular, and neurologic disturbances. Primary hyperparathyroidism and malignancy account for most cases of hypercalcemia.<sup>1</sup> Vitamin D toxicity is a significantly rarer cause, but an important one that merits consideration especially in patients taking dietary supplements.</p> <p>Case Presentation: A 51-year-old female presented to the emergency department with one-week of progressive confusion and hallucinations. Past medical history was significant for lupus nephritis on low-dose prednisone. On presentation, she had an elevated total calcium of &gt;16.0 (ref. 8.6-10.0 mg/dL) and new acute kidney injury, with creatinine of 4.35 mg/dL (0.59-1.04 mg/dL). CT chest, abdomen, pelvis did not show malignancy. She was treated for severe hypercalcemia, likely causing renal impairment, with aggressive IV fluids and furosemide. Additional workup showed low PTH levels at &lt;6.0 pg/mL (15-65 pg/mL) and normal PTHrP levels at 0.7 pmol/L (≤4.2 pmol/L); serum electrophoresis did not show monoclonal gammopathy. She was noted, however, to take vitamin D3 and calcium supplementation at home for osteoporosis prophylaxis on prednisone. She was found to have vitamin D toxicity, with high 25(OH)D3 of 214 ng/mL (20-50 ng/mL) and normal 25(OH)2D at 31 pg/mL (18-78 pg/mL). Her hypercalcemia resolved with treatment, with improvement in mental status and kidney function. She was found to be taking multiple forms of over-the-counter vitamin D3 and calcium at home, totaling 35,000 IU/day of vitamin D3. Her supplements were discontinued upon discharge and subsequent follow-up demonstrated normal calcium levels.</p> <p>This case highlights an uncommon cause of hypercalcemia—vitamin D toxicity—from inadvertent excessive supplementation. Recommended daily vitamin D amounts vary with guidelines, with the Endocrine Society recommending 1500–2000 IU/daily of vitamin D for at-risk-adults over 50, with an upper-limit of intake at 10,000 IU.<sup>2</sup> For development of vitamin D toxicity, previous reports have generally shown intake of 40,000 IU/day for at least several months.</p> <p>Discussion: In clinical cases of vitamin D toxicity, 25(OH)D levels are often elevated without similar elevation in 1,25(OH)2D levels.<sup>4</sup> Vitamin D is metabolized initially to 25(OH)D in the liver, a step not tightly-regulated, and then to 1,25(OH)2D in the kidney; 1,25(OH)2D binds to vitamin D receptors to increase intestinal calcium absorption.<sup>4,5</sup> Excess 25(OH)D is hypothesized to also bind vitamin D receptors as well as elevate free 1,25(OH)2D levels, contributing to hypercalcemia.<sup>4</sup> Hypercalcemia can occur with 25(OH)D concentrations at least above 80 ng/mL.<sup>5</sup></p> <p>Recognition of patients’ often-overlooked vitamin supplement use can be crucial in preventing harmful medical complications.</p>



<p><b>Lindsay Carafone</b> Dr. Juan Bowen</p>	<p><i>Detecting Vitamin D Deficiency</i></p> <p>Introduction: Vitamin D deficiency can be asymptomatic but also can present with nonspecific symptoms. These include muscle weakness, bone pain, and fatigue. The primary source of vitamin D is cutaneous production via exposure to ultraviolet rays, and decreased sunlight is a predominant risk factor for vitamin D deficiency. Those who live in northern latitudes, where cutaneous production of vitamin D is minimal in winter, are at increased risk. There are currently no recommendations for screening for vitamin D deficiency in the general population. However, it is important to identify vitamin D deficiency because of the morbidity and mortality associated with this diagnosis.</p> <p>Case Presentation: A 46 year-old Caucasian woman from southeastern Minnesota with a history of idiopathic true vocal fold paralysis and irritable bowel syndrome presented with complaints of generalized fatigue and dyspnea on exertion. Hypocalcemia of 8.5 mg/dL had been found several months prior but was not further evaluated because it was a borderline abnormality. The physical examination was unremarkable. Several tests were performed to evaluate fatigue, including thyroid function testing, iron, TIBC, ferritin, B12 and exercise EKG, all of which were normal. Due to the previously demonstrated mild hypocalcemia, parathyroid hormone and vitamin D levels were obtained. She was found to have elevated parathyroid hormone at 112 pg/mL (normal 15-65 pg/mL), which was consistent with secondary hyperparathyroidism. She was also found to have severe vitamin D deficiency with total 25-hydroxyvitamin D level of 7.2 ng/mL. She was initiated on vitamin D supplementation with 50,000 units weekly for 8 weeks. She had subsequent normalization of vitamin D, calcium and parathyroid hormone levels. She then started daily supplementation with 1000 units of vitamin D daily. Follow-up labs at three months demonstrated normal total 25-hydroxyvitamin D level of 32 ng/mL and normal total calcium of 9.2 mg/dL. There was persistent fatigue with some improvement. It was considered that vitamin D deficiency was probably one cause for her symptom of fatigue but that other reasons for fatigue were likely present.</p> <p>Discussion: Hypocalcemia can be mild or even low normal in vitamin D deficiency, so levels in this range serve as an important clue warranting further work up. Testing parathyroid hormone is an important step in identifying the cause of hypocalcemia and elevated parathyroid hormone levels occur in vitamin D deficiency. Vitamin D insufficiency/deficiency is diagnosed when serum concentration of total 25-hydroxyvitamin D is &lt;20 ng/mL, with severe deficiency defined as levels &lt;10 ng/mL. Work up for vitamin D deficiency should especially be considered for patients living in northern latitudes or those who predominantly spend time indoors with little sun exposure, both of which characterized this patient.</p>
<p><b>Gregory Challenger</b> Dr. Laura Greenlund</p>	<p><i>An Unusual Case of Knee Pain with Gout</i></p> <p>Introduction: Acute gout typically presents as intense joint pain due to inflammatory arthritis associated with intra-articular uric acid crystals and joint effusion. In contrast, gout enthesopathy occurs when uric acid crystals are deposited in a tendon surrounding a joint; this is another possible manifestation of gout that is often overlooked and likely goes undiagnosed in many affected patients.</p> <p>Case Presentation: A 60-year-old woman with a past medical history significant for crystal-proven gout and chronic kidney disease consulted her physician because of an acute onset of left knee pain. She stated that the pain had begun after spending much of the day in a dental chair with her leg extended while undergoing a root canal procedure. She had previously experienced a gout flare of the right knee, and she felt that her symptoms were very similar to that past episode. On physical exam, her left knee was mildly swollen, erythematous, warm, and tender to palpation. Her pain was particularly extreme with knee flexion, with her pain mostly localized to the anterior knee. Given her symptoms, history of gout, and physical exam, it was considered highly likely that she was suffering a gout flare affecting the left knee joint. In the</p>

	<p>context of her kidney disease and preference to avoid systemic steroids, she was referred for an intra-articular corticosteroid injection. The following day, a limited ultrasound exam showed no intra-articular effusion to be present, and no fluid could be aspirated; however, she received a corticosteroid injection under ultrasound guidance.</p> <p>Unfortunately, she experienced no relief following that procedure, and she returned to clinic three days later complaining of continuing knee pain. Physical exam at that time demonstrated point tenderness over the superior patella and an inability to flex the knee without severe pain. She was sent to the procedure clinic the same day for an ultrasound evaluation of the knee. At the procedure clinic, ultrasound exam demonstrated irregularity along the patellar cortex at the attachment of the quadriceps tendon with uric acid depositions and hyperemia consistent with enthesitis of the quadriceps tendon. An ultrasound-guided methylprednisolone/lidocaine injection around the site of quadriceps enthesitis was performed, and the patient experienced immediate relief of her knee pain. While previously describing 10/10 pain with knee flexion, following the peri-enthesitis injection, she demonstrated 90 degree flexion of the knee with no pain. She continued to enjoy sustained relief of her knee pain and has not had a recurrence to date.</p> <p>Discussion: This vignette illustrates an unusual case of knee pain due to gout enthesopathy. This condition can be diagnosed and treated using point-of-care ultrasound. Awareness of this condition, especially in patients in whom gout flares are suspected but no intra-articular uric acid crystals are observed, can substantially improve management.</p>
<p><b>Monazza Chaudhry</b> Dr. Rawad Nasr</p>	<p><i>Progressively Aggressive: A Rare Case of Autoimmune Necrotizing Myositis</i></p> <p>Introduction: Autoimmune necrotizing myositis (AINM) is a relatively recent addition to the family of inflammatory myopathies. Below, we describe a case of a young female with a relatively unknown disorder that has more commonly been described in older individuals.</p> <p>Case description: A 26 year old female presented to the emergency department with a 3 week history of progressive proximal muscle weakness involving both her upper and lower extremities. Her current symptom onset was preceded by a 2 week history of diarrhea. Lab workup revealed extensive rhabdomyolysis with a CK of 13,000 and preserved renal function. The rest of the labs included a positive stool culture for enteroaggregative Escherichia Coli and transmaninitis. She was admitted to medicine and started on aggressive fluid rehydration. Gastroenterology and Infectious Diseases were consulted and it was determined that this strain was not the source of myositis. A rheumatology referral was made and ANA and myositis panel were sent. MRI of the lower limbs was done which revealed diffuse, symmetric inflammatory changes. Muscle biopsy of the left vastus lateralis was sent and she was started on 60mg prednisone while the rest of her auto-immune panel were sent. Once CK was stabilized, she was discharged home with early follow up with rheumatology. Her initial workup yielded a low C3, C4 which normalized on repeat evaluation, +dsDNA at 44, +SSA (kD-60), +ANA 1:2560, +TPO. HMG Co-A reductase and SRP antibodies were negative along with the rest of the autoimmune and myositis panel. Within a few days post-discharge, CK levels were seen to be elevated once again and muscle biopsy revealed a necrotizing myopathy. She was re-admitted and started on pulse steroids and IVIG which showed remarkable improvement in her symptoms.</p> <p>Conclusion: This case illustrates a rare etiology of rapidly progressive immune-mediated necrotizing myositis in a young, healthy individual. AINM remains a poorly understood disorder; however, one that is associated with high morbidity due to strong associations with malignancy and other auto-immune disorders.</p>

<p><b>Chansong Choi</b> Dr. John Ratelle</p>	<p><i>When the Rash is More than Just Contact Dermatitis</i></p> <p>Introduction: Herpes zoster (HZ) results from reactivation of varicella-zoster virus (VZV) along a ganglion to the neural tissue.<sup>1</sup> In immunocompetent hosts, HZ presents with characteristic erythematous papules along a single dermatome (i.e. shingles), making the diagnosis relatively straightforward. In immunocompromised hosts, however, diagnosing HZ can be challenging because cutaneous manifestations can vary substantially.</p> <p>Case Presentation: A 71-year-old man with a renal transplant secondary to polycystic kidney disease on tacrolimus, mycophenolate, and prednisone presented to clinic with 3-4 days of erythematous, scaly, pruritic, non-painful rash on his back, groin, buttock, and abdomen. He was diagnosed with contact dermatitis with superimposed cellulitis, and was prescribed topical bactroban. Despite treatment, his rash worsened and he presented to the Emergency Department three days later with new fever, chills and myalgia. Physical exam was notable for scaly, erythematous, blanching rash with impetiginization and purulent drainage along the right S1-S5 dermatomes. Out of concern for disseminated zoster with secondary bacterial infection, he was started intravenous (IV) acyclovir and piperacillin-tazobactam. During his hospitalization, he was noted to have fecal/urinary incontinence, confusion, and tremors concerning for CNS involvement. Lumbar puncture with CSF studies revealed 433 total nucleated cells, with 90% lymphocytes, 5% monocytes, total protein 308, 155 RBCs, and PCR studies positive for varicella zoster virus. His confusion improved and he was discharged on a 14 day course of IV acyclovir.</p> <p>Discussion: This case highlights the atypical presentation of the HZ rash in a patient with solid organ transplant. In immunocompetent host, few lesions (typically less than 20) typically arise along the affected dermatome and the diagnosis of HZ is made clinically, without need for diagnostic testing. However, in persons with immunosuppression, particularly those with hematopoietic or solid organ transplants, or HIV, extensive skin involvement including several contiguous dermatomes (known as “multi-dermatomal zoster”) or spread to even non-contiguous dermatomes known as “zoster duplex unilateralis or bilateralis” is possible<sup>3</sup>. In addition, rash may appear as hemorrhagic pustules in immunocompromised as well as patients of advanced age<sup>4</sup>. In these cases, a high index of suspicion if required and the diagnosis is often made with PCR testing of skin lesions (sensitivity &gt;95%). Disseminated zoster is defined as HZ involving &gt;2 dermatomes and/or extracutaneous manifestation (e.g. encephalitis, pneumonitis, hepatitis) and is treated with IV acyclovir, typically for 2 weeks.</p> <p>References:  [1] Dworkin R.H., Johnson, R.W., Breuer, J., et al., Recommendations for the Management of Herpes Zoster, Clinical Infectious Diseases, Volume 44, Issue Supplement_1, 1 January 2007, Pages S1–S26, <a href="https://doi.org/10.1086/510206">https://doi.org/10.1086/510206</a>  [2] Atypical disseminated herpes zoster: management guidelines in immunocompromised patients. Cutis. 2017 Nov;100(5):321;324;330.  [3] Petrun, B., Williams, V., &amp; Brice, S. (2015). Disseminated varicella-zoster virus in an immunocompetent adult. Dermatology Online Journal, 21(3). Retrieved from <a href="https://escholarship.org/uc/item/3cz2x99b">https://escholarship.org/uc/item/3cz2x99b</a>  [4] Chen, S.Y., Suaya, J.A., Li, Q. et al. Infection (2014) 42: 325. <a href="https://doi.org/10.1007/s15010-013-0550-8">https://doi.org/10.1007/s15010-013-0550-8</a></p>
<p><b>Gretchen Colbenson</b> Dr. Ariela Marshall Dr. Matthew Koster Dr. Diana Kelm Dr. Kianoush Kashani Dr. John Wilson</p>	<p><i>A Case of Macrophage Activation Syndrome Associated with Parvovirus and Rheumatoid Arthritis</i></p> <p>Introduction: Macrophage activation syndrome (MAS), a form of hemophagocytic lymphohistiocytosis (HLH) associated with autoimmune disease, is characterized by uncontrolled immune activation leading to excessive macrophage activity and cytokine release. Potential triggers include infection and/or uncontrolled inflammation from rheumatic disease. MAS is characterized by fever, hepatosplenomegaly,</p>

<p>Dr. Hilary DuBrock</p>	<p>pancytopenia and markedly increased ferritin. Known infectious triggers for secondary HLH include Epstein Barr Virus (EBV), herpes simplex virus (HSV), cytomegalovirus (CMV), and human immunodeficiency virus (HIV). Here, we present a case of MAS in the setting of rheumatoid arthritis and parvovirus infection.</p> <p>Case presentation: A 60-year-old woman with a past medical history significant for long-standing, incompletely-treated, seropositive rheumatoid arthritis, hypertension, and chronic kidney disease presented with fever of 103.9°Fahrenheit, sinus pressure, general malaise, and was found to have a hemoglobin of 4.4g/dL without active signs of bleeding. She was admitted for further workup. Early in her hospital course, she developed acute kidney injury, clinical signs of shock requiring vasopressors for support, and hypoxemic respiratory failure with diffuse interstitial opacities on chest imaging concerning for acute respiratory distress syndrome. She was intubated for respiratory support and started on broad-spectrum antibiotics. Bacterial cultures from blood, urine and bronchoalveolar lavage were negative. Laboratory evaluation was subsequently notable for hemoglobin of 6.5g/dL, platelet count 32x10<sup>9</sup>/L., leukocytes of 2.0x10<sup>9</sup>/L, ferritin of 29,300mcg/L, and a positive serum parvovirus PCR. Histoplasma and Blastomyces antibodies, HIV antigen, and HSV, CMV, EBV, and adenovirus PCRs were negative. Bone marrow biopsy was positive for Parvovirus PCR and showed hemophagocytic macrophages without evidence of malignancy, consistent with MAS in the setting of rheumatologic disease and acute infection. Her presentation was believed to be more consistent with MAS rather than HLH given her underlying rheumatologic disease and no evidence of malignancy on bone marrow biopsy. Given this, treatment for MAS was initiated with Solumedrol 250 mg IV every 6 hours for 5 days followed by a prednisone taper. She was also treated with 5 days of intravenous immune globulin dosed at 0.4 g/kg. Ferritin and C-reactive protein levels down-trended, however her clinical course was complicated by intermittent hypotension and severe acute on chronic kidney disease requiring dialysis. After a lengthy hospital course, she ultimately decided to pursue hospice.</p> <p>Discussion: Parvovirus has rarely been reported in association with secondary HLH, and when observed, it is more commonly seen in the pediatric population. MAS is associated with high morbidity and mortality, but prompt recognition and treatment of MAS and the underlying trigger can improve outcomes. Standardized treatment guidelines for MAS are currently lacking, but high dose steroids with or without cyclosporine are often used as first-line therapy. MAS/HLH can be a life-threatening condition and should be considered in patients presenting with high-grade fever, hepatosplenomegaly, and cytopenias without a clear alternative cause.</p>
<p><b>Cristina Corsini</b> Dr. Edison Cano Cevallos Dr.</p>	<p><i>A 21-Year-Old with Hemolytic Anemia, Thrombocytopenia, and Atherosclerotic Cardiovascular Disease</i></p> <p>A previously healthy 21-year-old male presented from an outside facility for evaluation after newly diagnosed cirrhosis. At the time of presentation, he complained of substernal chest pain during minimal exertion, dyspnea, jaundice, abdominal distention, and hematochezia. His family history was positive for sudden cardiac death of a paternal uncle in his 20s. Physical examination was significant for scleral icterus, petechiae, abdominal distention, and left Achilles tendon thickening. Laboratory analysis revealed hemoglobin of 8.9 g/dL, platelet count of 55,000, elevated liver function tests, with an INR of 2.7, and normal albumin. A lipid panel showed total cholesterol of 152 mg/dL, LDL-C of 36 mg/dL, and HDL-C of 88 mg/dL. An abdominal CT displayed a cirrhotic liver, splenic vein thrombosis, and extensive splenic infarction. Dobutamine stress echocardiogram was positive for ischemia with inducible wall motion abnormalities and a subsequent coronary angiogram found total occlusion of the RCA and diffuse, subtotal occlusion of the mid-LAD. Measurement of plasma sterols revealed severely elevated campesterol and sitosterol (plant-derived sterols), diagnostic of sitosterolemia. Genetic testing revealed a homozygous nonsense mutation at c.1336C&gt;T, in the ABCG5 gene on chromosome 2p21, one of two genes implicated in this disorder. The patient was started on ezetimibe, a low plant-fat diet, and was evaluated for heart, lung, and liver transplant.</p>

	<p>Sitosterolemia is a very rare, autosomal recessive disorder characterized by excessive intestinal absorption and reduced biliary excretion of plant sterols due to mutation of the sterolin transport protein. Excess plasma sterols can affect cellular membrane integrity, platelet function, and promote atherosclerotic plaque formation leading to anemia, thrombocytopenia, and premature atherosclerotic cardiovascular disease. This case highlights the protean and potentially severe manifestations of sitosterolemia, as well as the importance of early diagnosis.</p>
<p><b>Meaghan Costello</b> Dr. Zachary Yetmar Dr. Thomas Beckman</p>	<p><i>Takotsubo Cardiomyopathy Physiology in the Setting of Metastatic Pheochromocytoma: A Unique Case to Illustrate the Mechanism</i></p> <p>Introduction: The pathophysiology of takotsubo cardiomyopathy (TC) is not well understood. Given its association with physical and emotional stress, many have hypothesized that excess catecholamine release is the underlying etiology. However, coronary vascular dysfunction has also been implicated.</p> <p>Case Description: A 39 year old female with known metastatic pheochromocytoma on terazosin and bisoprolol, hypothyroidism on levothyroxine, and insulin-dependent diabetes presented with severe chest and back pain, blood pressure 200/110, and heart rate 128. Laboratory studies revealed troponin 90. EKG showed sinus tachycardia without evidence of ischemia. CT angiogram of chest/abdomen was concerning for TC (otherwise known as stress-induced cardiomyopathy) with sluggish flow in left ventricle, reduced left ventricular ejection fraction (LVEF), and normal coronary arteries; as well as widely metastatic disease to the lung, mediastinum, pericardium, liver and left upper quadrant/adrenal gland. Transthoracic echocardiogram (TTE) demonstrated LVEF 27% with regional wall motion abnormalities. Plasma free metanephrines were elevated at 102 (normal &lt; 0.9). The tumor burden and metanephrine elevation were so significant that phenoxybenzamine 40mg TID, metoprolol tartrate 200mg BID, and diltiazem 90mg QID were required to obtain blood pressure and heart rate control. With improvement in vital signs, the LVEF recovered to 56%, but regional wall motion abnormalities persisted. Subsequently, metyrosine and pazopanib were initiated as chemotherapy for medical management of the widespread metastasis. As plasma metanephrines were better controlled, the regional wall motion abnormalities resolved. Follow-up studies revealed plasma free metanephrines of 5.3, as well as TTE showing LVEF 69% and normal wall motion.</p> <p>Discussion: This case – involving massive catecholamine release in the setting of metastatic pheochromocytoma – provides an unusual and striking example of how excessive adrenergic stimulation may precipitate TC physiology. The syndrome of TC was initially described in 1990, with case series highlighting the common precipitant of extreme emotional stress, often among women. Coronary artery dysfunction has also been implicated; however, coronary artery angiography in patients with TC usually demonstrates no obstructions or evidence of vasospasm. In support of this, the Mayo Clinic Diagnostic Criteria for TC includes the absence of obstructive coronary disease or plaque rupture. In our patient, treatment for metastatic pheochromocytoma with chemotherapy resulted in drastic reductions in catecholamines, with resulting normalization of heart rate, blood pressure, and LV function. Therefore, this case provides a useful illustration of the catecholaminergic mechanism of TC.</p>
<p><b>Megan Covington</b> Dr. Brandon Huffman</p>	<p><i>Pay Varicose Attention</i></p> <p>Case Presentation: A 60-year-old male presented to the emergency department with hematemesis, bilateral upper extremity swelling, and subacute engorgement of superficial chest veins. He endorsed episodic lightheadedness, intermittent headaches, facial swelling, hoarseness, and dyspnea on exertion over the last two months. His vital signs were within normal limits. On exam, he had bilateral upper extremity pitting edema and periorbital edema. He had a right subclavian intravenous access device, which was placed four years prior due to having poor intravascular access. Labs revealed a normocytic anemia with hemoglobin of 10.0 g/dL, consistent with</p>

	<p>anemia secondary to blood loss. Esophagogastroduodenoscopy and colonoscopy completed at an outside hospital were significant for proximal and mid-esophageal varices without evidence of portal hypertension. Additionally, CT did not show liver pathology or any thoracic masses. Chest CT angiogram was negative for thoracic masses and pulmonary embolism, though it demonstrated varicosities along the chest wall. There was a right subclavian port with the tip in the right atrium. He was given 40mg of IV pantoprazole and admitted for management of his hematemesis. Upper extremity ultrasound was negative for acute thrombus, but there was decreased compressibility of the axillary veins. A chest CT venogram suggested focal narrowing of the superior vena cava (SVC) at the level of the distal port catheter at the SVC-right atrial junction due to a thrombus. He was started on 1mg/kg of enoxaparin. Interventional Radiology removed the port and confirmed an acute on chronic occlusion of the SVC with prominent mediastinal and chest wall collaterals. They performed angioplasty and recanalized the SVC via stenting. Two days post-operatively in clinic his chest veins had decreased in size and he had improvement in his hematemesis and edema.</p> <p>Discussion: This case illustrates the presentation of SVC syndrome, and highlights the venous anatomy and its correlation to the patient’s hematemesis. SVC syndrome commonly presents with face or neck swelling, upper extremity swelling, dyspnea, cough, dilated chest vein collaterals, hoarseness, and headache. The obstruction of blood flow in the SVC leads to the development of venous collaterals, which can arise from the azygos, internal mammary, lateral thoracic, paraspinous, and esophageal venous systems. The proximal and mid-esophageal veins drain into the azygos and brachiocephalic veins, which are secondarily congested in SVC syndrome. Although it is rare, these proximal or “downhill” esophageal varices can cause gastrointestinal bleeding.</p> <p>This case also emphasizes an important complication of intravascular devices to consider: thrombus. Intravascular catheter-associated thrombus is the most common etiology of bleeding downhill varices and the most common noncancerous etiology of SVC syndrome. When patients present with typical symptoms and an intravenous central catheter, it is important to evaluate for the cause of SVC syndrome with appropriate imaging studies such as CT venogram.</p>
<p><b>Zachary Davidson</b></p>	<p><i>Vertiginous Tinnitus as the Chief Presentation of Anemia</i></p> <p>Introduction: Tinnitus and dizziness with nystagmus are commonly concerning for central versus peripheral nervous system etiology of vertigo. However, it is important to keep a broad differential for disease in cases of tinnitus as other concerning diseases can present with this cluster of non-specific symptoms.</p> <p>Case Presentation: A 50 year-old female presented to the Emergency Department with a history of fatigue, vertigo, and pulsatile tinnitus for 2 months. She reported feeling faint when she stood up on the train on her way to an appointment. Patient has a remote history of heavy alcohol use, but currently reports drinking 4-5 drinks per month most recently 72 hours ago. She endorsed a diet of just one sandwich daily and frequent ice-eating, stating, “it fills me up.” Review of symptoms positive for feeling chilled for the past month, with cough and URI symptoms for 1 week, although she is afebrile on presentation. Physical exam also notable for right-sided nystagmus, pale conjunctiva, and a 3/5 holo-systolic murmur loudest at the left sternal border. Labs on admission revealed a Hb of 4, and recent iron studies were consistent with iron deficiency anemia. Stool guaiac was trace positive. MRI (ordered out-patient for ENT work-up, but not yet completed) was negative for intracerebral mass or stroke, with scattered chronic small vessel ischemia on T2. Upon admission, an EGD showed Grade C esophagitis, erosive gastropathy, hiatal hernia with a Cameron lesion, duodenitis, and H pylori biopsy was positive. A chest x-ray also demonstrated patchy right-hilar opacity and she was treated for community acquired pneumonia. She was transfused with 3 units of cross-matched RBCs and her Hb increased to 7.2, while symptoms of fatigue, dizziness, and tinnitus improved. She was started on oral iron,</p>

	<p>thiamine, and folic acid and was discharged on quadruple therapy for H pylori. A visiting patient care manager follow-up one month after discharge showed the patient was in good health and clinically sober, and she was connected to food and transportation services. Patient plans for a follow-up for a repeat EGD to assess healing.</p> <p>Learning Points: It is known that anemia can present with a broad spectrum of symptoms, and this case illustrates the importance of keeping a broad differential for dizziness and tinnitus. Thus, a CBC is an important lab test in any patient with concern for pre-syncope, regardless of ongoing work-up. This patient's pagophagia ('ice-eating') and new systolic murmur are both interesting diagnostic clues to her underlying severe iron-deficiency anemia. In regards to the underlying cause of her bleeding, this patient had a H. pylori infection which is often asymptomatic, and can have an indolent presentation. This patient's poor diet could be interpreted as food intolerance and is a notable symptom.</p>
<p><b>Alberto Del Valle De Laosa</b>  Dr. Aaron Boothby  Dr. Christine Wendt</p>	<p><i>A Case of Particularly Prolonged Paralysis</i></p> <p>Introduction: Succinylcholine is a frequently used ultra short-acting, depolarizing-type, skeletal muscle relaxant with numerous reported potential complications. Individuals on cholinesterase inhibitors or with genetic deficiency of pseudocholinesterase are at particular risk of prolonged paralysis. Herein we present the case of a gentleman who suffered a particularly prolonged period of paralysis following administration of succinylcholine.</p> <p>Case Presentation: An 87 year-old male with past medical history of mild cognitive Impairment, chronic kidney disease, gastrointestinal reflux, peripheral vascular disease, and vertebral artery stenosis was hospitalized for treatment refractory major depressive disorder with psychotic features and suffered a remarkably prolonged period of flaccid paralysis following his first episode of electroconvulsive therapy (ECT).</p> <p>The procedure was uneventful. 80 mg of methohexital and 60 mg of succinylcholine were given for sedation and paralysis. However, the patient failed to emerge from anesthesia. Peripheral nerve stimulator produced zero twitches in bilateral upper extremities and the face at 10, 20, and 30 minutes He was intubated, sedated with propofol and transferred to PACU where he remained paralyzed. Given the history of cerebrovascular disease stroke code was called to rule out intracranial pathology. Neuro exam was consistent with peripheral paralysis. CT head and CTA head/neck were negative. Following imaging sedation was turned off to assist in further neurologic exam. The patient was then transferred to the ICU. On arrival a bispectral index monitor was attached and revealed a level of 70 and sedation was immediately resumed. Labs at this time were notable for hyperkalemia and respiratory acidosis which corrected with ventilator adjustment. Train of four was serially tracked and twitches returned after a period of over 6 hours. The patient was able to be safely extubated 8 hours after the initial dose of succinylcholine and had no memory of the event. On medication review the patient was noted to be on donepezil (which can slow clearance of succinylcholine) as well as omeprazole and escitalopram which may slow clearance of donepezil via cytochrome p450 inhibition. Pseudocholinesterase level and dibucaine inhibition assay sent on day 0, 2, and 9 after ECT showed low serum pseudocholinesterase level and normal dibucaine inhibition. Consistent with either decreased production or rare genetic deficit. Genetic testing is pending at this time.</p> <p>Discussion: This case illustrates the potential for significantly prolonged paralysis secondary to succinylcholine administration in a patient on cholinesterase inhibiting medication as well as cytochrome P450 inhibitors, likely vastly exacerbated by the presence of unknown underlying genetic variant. To our knowledge this case presents one of the longest periods of prolonged paralysis from succinylcholine reported in the literature to date.</p>

<p><b>Saam Dilmaghani</b> Dr. Ali Duarte Garcia</p>	<p><i>Renal Dysfunction as a Manifestation of Sjogren's Syndrome</i></p> <p>Introduction: Sjogren's syndrome (SS) is a chronic autoimmune disorder characterized by sicca symptoms – dry eyes and dry mouth. Less commonly observed, however, SS can affect many organ systems resulting in a wide array of clinical manifestations. This report presents the case of a young woman with rapidly progressive kidney dysfunction due to SS.</p> <p>Case Presentation: A 28-year-old woman was evaluated for a four-year history of progressively worsening chronic kidney disease (stage III, eGFR 46) of unknown etiology. Her medical history was notable for depression, gastroesophageal reflux disease, eczema, allergic rhinitis, migraines and a remote history of childhood seizures. The patient had a long history of dry eyes and mouth, hoarseness, and dry cough. Along with the dry mouth, the patient had severe tooth decay requiring to pull all her teeth. The patient had symmetric arthralgias and slowly resolving morning stiffness of her hands, wrists, elbows, and knees, fatigue and recently development of dyspnea on exertion. She denied any history of photosensitivity, facial rashes, oral ulcers, or nephrolithiasis. She was a nonsmoker, non-drinker, and had no history of illicit substance use. Family history included an aunt with lupus, an uncle with granulomatosis with polyangiitis and Raynaud's syndrome, and a nephew with both Raynaud's and Sjogren's syndromes. Physical exam was remarkable for complete edentulism, mild symmetrical synovitis and tenderness to palpation of the metacarpophalangeal joints, mildly enlarged and nontender parotid glands, and mild submandibular lymphadenopathy. Her laboratory studies revealed an iron deficiency anemia, hypokalemia, metabolic acidosis, progressive elevations in creatinine, and mildly elevated alkaline phosphatase, polyclonal hypergammaglobulinemia and negative cryoglobulins. She had a positive antinuclear antibody (ANA), along with anti-Ro and anti-La antibodies. Multiple urinalyses in the past had demonstrated transient proteinuria and microscopic hematuria, however she did not have active urinary sediment. Urinary retinol binding protein was elevated. Based on the symmetric polyarthritis, dry eyes and dry mouth, and positive ANA, anti-Ro and anti-La the patient was diagnosed with SS. The hypokalemia and progressive renal dysfunction were concerning for interstitial nephritis and distal (type 1) renal tubular acidosis. A kidney biopsy was performed which confirmed acute and chronic interstitial nephritis with focal mild tubulitis in a background of tubular atrophy and interstitial fibrosis.</p> <p>Discussion: This case illustrates extraglandular manifestations of SS, particularly interstitial nephritis. The most common manifestation of interstitial nephritis include tubular dysfunction, reduced glomerular filtration rate, distal renal tubular acidosis, metabolic acidosis, and hypokalemia. Although these manifestations are less common than sicca symptoms, a lack of familiarity and clinical suspicion can result in a delay of diagnosis for many years.</p>
<p><b>Christopher Dinh</b> Dr. Frank Kennedy</p>	<p><i>A Case of a Sulfonylurea-Induced Photosensitivity Rash</i></p> <p>Case Presentation: A 75-year-old male with a past medical history significant for type 2 diabetes mellitus, Sjogren's syndrome, and treated prostate cancer was referred to the Endocrinology clinic for management of his diabetes mellitus. He was diagnosed with diabetes mellitus 5 years prior and had been well controlled on metformin for several years. Glipizide was added to his regimen two years prior, after which he developed a pruritic maculopapular rash in photosensitive areas. He was seen by multiple local dermatologists with no improvement despite topical steroids. Biopsies showed perivascular dermatitis and interface dermatitis. His glipizide was discontinued and his rash resolved. His hemoglobin A1c remained elevated so glimepiride was started and a similar appearing rash returned. In our clinic, his glimepiride was replaced with a DPP-4 inhibitor and his rash eventually resolved.</p> <p>Discussion: In this case, we present a patient with a sulfonylurea-induced rash with a</p>



	<p>clear temporal relationship. Over 20 million adults in the United States have been diagnosed with type 2 diabetes mellitus and this number is predicted to increase. Clinicians have numerous pharmacologic options for diabetes management, but typically start with metformin followed by sulfonylureas in non-insulin requiring patients.</p> <p>Allergies to sulfonamide-containing antibiotics are the second most common allergic drug reaction, estimated to occur in up to 8% of hospitalized patients. The incidence of nonantibiotic sulfonamide allergies are less well characterized but include many similar adverse reactions including dermatologic rashes and gastrointestinal irritation. FDA labeling lists hypersensitivity to sulfonamides antibiotics as a contraindication to sulfonylurea use, but recent studies do not show evidence of cross-reactivity between sulfonamide-containing antibiotic allergies and nonantibiotic sulfonamide allergies. Despite the lack of cross-reactivity, nonantibiotic sulfonamide allergies are relatively common and must remain on the differential for patients on these medications.</p> <p>In conclusion, with the increasing prevalence of type 2 diabetes mellitus, clinicians must be aware that sulfonylureas contain a separate but distinct risk of allergic reaction to patients.</p>
<p><b>Michael D’Netto</b> Dr. Meltiady Issa</p>	<p><i>A Poorly-Healing “Spider Bite”</i></p> <p>Introduction: Skin lesions commonly present to primary care physicians. In fact, skin conditions are the most frequent reason for people to consult their general practitioner with a new problem<sup>1</sup>. Given the volume of skin concerns which present initially to internists, understanding what skin concerns require further evaluation is critical to not only providing optimal patient care but also utilizing healthcare resources effectively<sup>1</sup>.</p> <p>Case Presentation: A 65 year old woman with a history of atrial fibrillation on apixaban, primary biliary cirrhosis, hypothyroidism, obesity and gastric bypass, was admitted to the hospital for large necrotic leg ulcers. She was evaluated by her primary care physician 2 years ago for a spider bite that did not heal well despite conservative management. She had multiple debridements over the past year but the lesion continued to progress to necrosis and ulceration. Initial biopsy was nonspecific. She was afebrile and hemodynamically stable during this admission. Physical examination showed multiple large necrotic ulcers of the lower extremities with no superimposed infection. Her labs showed a normal creatinine, phosphorous of 3.3, calcium of 10.1 when corrected for hypoalbuminemia (2.3), normal vitamin D, low parathyroid hormone of 7 pg/ml, normal liver function tests, and normal 24 hour urine calcium. Noninvasive vascular studies were normal bilaterally. She underwent extensive debridements in the operative room where roughly 30% of her body surface area was removed. Pathology was consistent with calciphylaxis so the patient was started on intravenous sodium thiosulfate and she underwent hyperbaric oxygen therapy. Finally, with wound care, her ulcers started to improve and she was discharged to an LTACH with a plan to consider skin transplant per plastic surgery.</p> <p>Discussion: Skin concerns thought to be secondary to spider bites are commonly seen in primary care. “Many people believe that bites from various spider species cause necrotic ulceration, despite evidence that most suspected cases of necrotic arachnidism are caused by something other than a spider bite.”<sup>2</sup> As seen with this patient, a more sinister etiology may be at play. While calciphylaxis is often associated with end stage renal disease (ESRD), non-uremic calciphylaxis (NUC) is diagnosed with cutaneous biopsy with no ESRD, after excluding mimickers such as nephrogenic systemic fibrosis, vasculitis and warfarin necrosis<sup>3</sup>. Pathophysiology is uncertain and mortality is high (52%)<sup>3</sup>. Treatment includes debridement and pain control<sup>3</sup>. Further treatments are largely based on treatment of uremic calciphylaxis, making NUC a diagnostic and therapeutic challenge<sup>3</sup>.</p>
<p><b>Michael Downey</b></p>	<p><i>A Case of Anterior Fibrosing Mediastinitis Presenting as Unstable Angina</i></p>

<p>Dr. Kellen Albrecht Dr. Mike Zhang Dr. Andrew Olson</p>	<p>Case Presentation: A 71-year-old male presented to the emergency department symptoms of acute progressive dyspnea on exertion and worsening angina. His past medical history was notable for coronary artery bypass grafting twenty years ago, hypertension, chronic obstructive pulmonary disease, and a recent hospitalization 1 month earlier for similar symptoms.</p> <p>During the patient’s previous admission, his resting electrocardiogram did not demonstrate evidence of ischemia, but his transthoracic echocardiogram revealed a left ventricular ejection fraction of 45-50% with mild global hypokinesis. Although the medical team offered a coronary angiogram to the patient, he elected to pursue medical management instead and started on an antianginal regimen with appropriate resolution of his symptoms at discharge.</p> <p>In the subsequent weeks, the patient’s dyspnea continued to worsen; he could no longer walk 100 feet without needing to stop to rest. He also developed pitting left arm edema and left facial plethora. On admission, his vitals signs were stable, his baseline EKG was unchanged, and a chest x-ray did not identify focal infiltrates. Laboratory results included an undetectable cardiac troponin at 0 and 3 hours, and a complete blood count that was within normal limits. Because the patient’s symptoms had clearly worsened, he underwent an exercise stress echocardiogram which demonstrated anterolateral wall motion abnormalities. This prompted a diagnostic cardiac catheterization, but no culprit stenosis or bypass occlusion was found.</p> <p>Separately, a left upper extremity venous duplex ultrasound was performed to investigate the patient’s left arm and facial edema, revealing an occlusive thrombus of his left subclavian vein. Subsequent chest CT demonstrated an anterior mediastinal mass externally compressing the vein and also encasing the patient’s left internal mammary artery to left anterior descending artery bypass graft. Cardiothoracic surgery, interventional radiology and pulmonary medicine were consulted to assist with obtaining a biopsy of this mass for diagnosis. Biopsies were obtained, and a diagnosis of anterior fibrosing mediastinitis was made.</p> <p>Learning Points:</p> <ol style="list-style-type: none"> <li>1. Base Factual Knowledge: Common etiologies of mass lesions in the inferior mediastinum vary based on the anatomic subdivision: Anterior (Terrible Ts) Middle (Lymphadenopathy) Posterior (neurogenic tumors)</li> <li>2. Specific Pathophysiologic Knowledge: Anterior Fibrosing Mediastinitis is most commonly thought to be triggered by immune responses to endemic fungi (i.e. Histoplasmosis) yet anti-fungal therapy and immunosuppressive therapies are ineffective therapies. Most treatments are supportive and palliative via structural supports/stents in response to compression of local anatomic structures (e.g. great veins, coronary arteries, and mainstem bronchi).</li> <li>3. Clinical Reasoning Pearl: Be cautious of “Definitional Fallacies” when faced with diagnostic dilemmas. Decreased coronary artery perfusion leads to myocardial ischemia and can impeded myocardial contractility. Progressive ASCVD leads to decreased coronary artery perfusion and can have subsequent myocardial ischemia. BUT decreased coronary artery perfusion is not de facto ASCVD.</li> </ol>
<p><b>Anthony Fragola</b> Dr. Sami Ryan</p>	<p><i>A Case of Diffuse Alveolar Hemorrhage Hiding in a Heart Failure Diagnosis: Where’s DAH Hemorrhage?</i></p> <p>Introduction: Diffuse alveolar hemorrhage (DAH) is a known complication of systemic vasculitis. When the presenting complaint is hemoptysis, the diagnosis is often straightforward. Other times, though, DAH can be easy to overlook. In rare cases, DAH may be subacute or chronic and, if symptoms are non-specific, it may</p>

	<p>hide under the guise of other diagnoses entirely.</p> <p>Case Description: A 76-year-old female with known microscopic polyangiitis, chronic kidney disease stage III and atrial fibrillation presented to the emergency department (ED) for dyspnea on exertion, noted to be progressively worse over the week prior to admission, as well as an acute 8-pound weight gain and new bilateral lower extremity swelling. Initial lab-work revealed an elevated NT-Pro BNP of 2622 (normal range 5 – 230 pg/mL), new iron deficiency anemia with a hemoglobin of 7.1 (normal range 11.6 - 15.0 g/dL) and a serum creatinine of 2.08 (normal range 0.59 - 1.04 mg/dL), elevated from her baseline of 1.4. Chest X-ray showed cardiomegaly, bilateral pulmonary edema and patchy airspace infiltrates. A transthoracic echocardiogram (TTE) showed normal left ventricular size and function with an ejection fraction of 59%, mild right ventricular enlargement and a right ventricular systolic pressure of 42 (normal range 15 – 25 mmHg). She was diuresed for acute congestive heart failure. An esophagogastroduodenoscopy (EGD) was performed to evaluate for a source of bleeding, during which blood was seen exuding from the trachea. A chest CT scan showed bilateral ground glass opacities compatible with DAH and bronchoalveolar lavage confirmed DAH. She was treated with steroids and Rituximab and her clinical status improved.</p> <p>Discussion: The classic presentation of DAH is abrupt, often including hemoptysis. However, in up to 33% of cases, hemoptysis may be absent, even when hemorrhage is severe. The patient’s primary complaint may be dyspnea. Chest radiograph findings are often nonspecific, including patchy or diffuse opacities. Classic CT findings include diffuse and bilateral ground glass opacities. Bronchoscopy with bronchoalveolar lavage (BAL) with hemorrhagic aliquots of fluid is diagnostic. Pulmonary function testing (PFTs) is most often notable for increased diffusing capacity of carbon monoxide (DLCO) due to the presence of blood in the airspace.</p> <p>Teaching Points: 1) Hemoptysis is absent in up to 33% of cases of DAH. 2) In the absence of hemoptysis, DAH should be suspected in patients with new respiratory symptoms, ground glass or consolidative opacities on imaging studies (especially in a patient with new anemia and/or known vasculitis), and PFTs with increased DLCO. 3) BAL with hemorrhagic return or &gt;20% hemosiderin-laden macrophages is diagnostic of DAH.</p>
<p><b>Max Fuller</b></p>	<p><i>Neisseria Meningitidis Infection in Common Variable Immunodeficiency</i></p> <p>Introduction: 33 year old female with PMH significant for HIV (CD4 count of 250), Burkitt’s Lymphoma, Common Variable Immunodeficiency (CVID) and Bipolar Disorder admitted to Intensive Care with sepsis secondary to meningococemia and meningococcal meningitis.</p> <p>Case Presentation: Presenting with 1-2 weeks of general unwell feeling. On arrival to ED, found to be febrile to 39.1 C, GCS of 11, tachycardic and hypotensive. Due to concerns regarding ability to protect airway, patient was intubated. Started on broad spectrum antibiotics including Vancomycin, Meropenem and Acyclovir given concern for CNS infection. Lumbar puncture done with 8,915 nucleated cells and 98% neutrophils. Blood cultures grew <i>Neisseria meningitidis</i>. CSF culture without growth likely due to delay in obtaining lumbar puncture. Patient had previously regularly received IVIg infusions as outpatient but this had been stopped due to adverse side effects. Due to patient’s history of CVID, she was treated with one infusion of IVIg in addition to antimicrobial therapy. Care was taken to find preparation of IVIg that contained the least amount of IgA due to concerns for anaphylaxis given patient’s known IgA deficiency. Able to narrow antibiotics to ceftriaxone and completed one week of treatment. Continued to clinically improve without any apparent adverse neurological effects.</p> <p>Discussion: CVID is a primary B cell disorder characterized by low levels of immunoglobulins and decreased antibody response to both vaccinations and</p>

	<p>infections. Patients often present with recurrent sinopulmonary infections but are also at increased risk for autoimmune disorders. There is also an increased risk for malignant disease particularly Non Hodgkin’s Lymphoma, which this patient previously had. Management of CVID includes regular immunoglobulin treatments usually every 3-4 weeks. The use of IVIg in patients with CVID has significantly reduced infections and their complications. The use of IVIg is not standard for acute infections in patients with CVID but has been used with success in Meningococcal meningitis. Although CVID is a relatively rare disease, it is important for the general internist to be able to recognize patients with immunodeficiencies and be aware of the specific treatments these patients need.</p>
<p><b>Abdulrahman Gamam</b> Dr. Jessica Padniewski Dr. Rawad Nasr</p>	<p><i>Hip Septic Arthritis Presenting as Knee Pain Leads to the Diagnosis of Rectal Adenocarcinoma</i></p> <p>Introduction: The association between invasive streptococcal infections and gastrointestinal malignancies has been well established.<sup>1</sup> Little has been published on the role of Streptococcus dysgalactiae subspecies equisimilis (SDSE). We report a case of a patient with knee pain due to hip SDSE septic arthritis leading to the diagnosis of rectal cancer.</p> <p>Case Presentation: A 51-year-old male presented to the urgent care clinic with 1 month of right knee pain and unintentional weight loss. A right knee arthrocentesis was performed which revealed a bland aspirate and a right knee steroid injection was given without clinical improvement. Rheumatology was consulted. Meticulous Physical Exam showed pain with passive flexion of the right hip. MRI of right hip was obtained which showed findings highly concerning for septic arthritis involving the right hip joint, right psoas abscess, discitis and osteomyelitis at L3-L4, and a rectal mass. CT of chest, abdomen, and pelvis showed intraluminal rectal lesion, focal rim-enhancing lesions in the liver concerning for metastatic rectal cancer as well as a psoas abscess. He was admitted, started on empiric antibiotics, and an emergent excisional irrigation and debridement of the right septic hip and the right psoas abscess was performed which revealed SDSE. Colonoscopy showed a fungating mass of distal rectum which was biopsied and confirmed to be adenocarcinoma.</p> <p>Discussion: This case highlights the following: 1. Knee pain could be referred pain from hip arthritis, 2. Septic arthritis could be due to SDSE and 3. SDSE could be associated with GI malignancy. There are few reported cases of invasive SDSE infections in association of GI malignancy, and only one reported case from 1987 of septic arthritis secondary to “group G streptococcus” in a patient with colon cancer.<sup>2–4</sup></p>
<p><b>Alexandra Hall</b></p>	<p><i>Multidisciplinary Case Review of an Anterior Mediastinal Mass: Beyond the ‘Terrible T’s’</i></p> <p>Introduction: Clinicians rely on first impressions and heuristics to guide all aspects of patient care. However, focalism can be counterproductive and delay accurate diagnosis and treatment. Here we present a case in which multidisciplinary discussion successfully broke anchoring bias and drastically affected a patient’s prognosis and management.</p> <p>Case Presentation: A 71 year old male with a past medical history significant for hypertension, coronary artery disease (CABG in 1999), HFpEF, and COPD presented to the emergency department with worsening dyspnea, exertional chest pain and 1 week progressive swelling of his left arm and face which had become painful.</p> <p>Ultrasound noted sub occlusive clot in the proximal left subclavian vein with no visualization of the left innominate vein. Subsequent CT scan demonstrated an occluded innominate vein along with infiltrative soft tissue that encased the left innominate vein, LIMA, and proximal common carotid artery. Borderline enlarged lymph nodes were noted. Multiple pulmonary nodules were also visualized. Overall, findings were highly concerning for malignancy. FNA of an enlarged lymph node was</p>

	<p>attempted but pathology returned nondiagnostic. Subsequent PET scan revealed intense FDG uptake associated with the anterior mediastinal mass with adjacent lymph nodes—a pattern suspicious for malignancy with nodal metastases. Favored differentials radiologically included thymic carcinoma and lymphoma. Parasternal needle core biopsy was performed; tissue obtained was identified as normal thymus. A repeat biopsy was recommended. Given the location of the mass and its proximity to the carotid and LIMA (which stress testing revealed was the primary supplier of his coronary circulation), the case was presented at our institution’s chest conference to discuss possible approaches. Consensus emerged from interventional radiology, pulmonary medicine, and thoracic surgery that the most likely diagnosis was fibrosing mediastinitis. This was supported by the presence of extensive collateral circulation and negative biopsies.</p> <p>Discussion: Fibrosing mediastinitis is a rare, insidious, progressive disease characterized by an excessive fibrotic reaction in the mediastinum which can result in the compromise of airways, great vessels, and other mediastinal structures. Signs and symptoms largely depend on what structures are affected. On CT, fibrosing mediastinitis is strongly suggested by the presence of paratracheal, hilar, and subcarinal lymphadenopathy and circumferential encasement of mediastinal structures. It can also demonstrate high FDG uptake on PET scan, mimicking malignancy. As in this case, it can be difficult to make a definitive histopathological diagnosis using minimally invasive methods. Many patients require surgical biopsy in order to confirm the diagnosis and rule out neoplasm. Treatment is challenging, as no therapy has proven effective.</p> <p>This case illustrates the importance of a multidisciplinary review of apparently elusive cases. What initially appeared to be an aggressive malignancy was ultimately diagnosed as fibrosing mediastinitis with the help of an interdisciplinary conference.</p>
<p><b>Hiba Hashmi</b></p>	<p><i>Hyperaldosteronism – A Family Matter</i></p> <p>Introduction: Primary hyperaldosteronism (PH) presents with recalcitrant hypertension, hypokalemia and an elevated aldosterone: renin ratio. It is commonly attributed to adrenal adenomas or hyperplasia with familial hyperaldosteronism (FH) remaining a rare etiology. We report the case of a young African woman diagnosed with familial hyperaldosteronism and end stage renal disease (ESRD) during pregnancy.</p> <p>Case Presentation: A 21-year-old female with a five-year history of hypertension presented to medicine clinic with headache, chest pain, fatigue, anorexia and heat intolerance for two weeks. She was hypertensive to 163/113 mmHg and tachycardic to 113 bpm. She had moved to Minneapolis from Ethiopia and had no prior medical records or testing available. She had been taking an unknown antihypertensive until three weeks prior. She was found to have a creatinine of 3.49 corresponding with a GFR of 18. Renal ultrasound revealed bilateral, small echogenic kidneys without any evidence of renal artery stenosis. An intrauterine pregnancy was also incidentally discovered. Her aldosterone level was elevated to 486 ng/dL and her renin activity was 1.3 ng/ml/hr, with a ratio of 373, diagnostic of primary hyperaldosteronism. Due to the markedly high ratio, a saline suppression test was deemed unnecessary for confirmation. Her serum potassium was low at 3.6, likely due to poor renal clearance. Given renal failure, a CT non-contrast of the adrenal glands was performed with normal findings.</p> <p>Based upon her young age at presentation, family history of early onset hypertension in her father, grossly elevated aldosterone: renin ratio and unrevealing workup for a primary tumor or hyperplastic adrenals, a diagnosis of familial hyperaldosteronism was made. Genetic testing was submitted. She failed a month-long trial of dexamethasone therapy, therefore glucocorticoid remediable aldosteronism was excluded. She was subsequently started on spironolactone with good response. Adrenal vein sampling was considered to find a surgical target for adrenalectomy but</p>

	<p>could not be performed given worsening kidney function. After discussion with Nephrology she opted for pre-emptive renal transplant, rather than pursuing dialysis and is undergoing pre-transplant evaluation. Results of genetic testing for sub-classification are pending.</p> <p>Discussion: FH is a rare condition occurring in approximately 6% of hypertensive cases at referral centers. It is further sub-divided into glucocorticoid remediable, type I, and non- glucocorticoid remediable, types II – IV. The initial diagnosis of such a condition during pregnancy and in the setting of worsening kidney disease presents a diagnostic and management challenge as this precludes adrenal vein sampling and contrast imaging. This makes identification of a potential surgical target difficult. Our case illustrates the need for updated guidelines on hyperaldosteronism workup in the setting of ESRD and highlights the importance of early screening for PH.</p>
<p><b>Daniel Halichek</b> Dr. Mitchell Pitlick Dr. Christopher Stephenson</p>	<p><i>Lower Extremity Purpura in an Adult: A Case of Cutaneous Limited IgA Vasculitis</i></p> <p>Introduction: IgA vasculitis, formerly known as hench-schonlein purpura (HSP), is the most common systemic vasculitis in the pediatric age group. However, it remains less described in the adult population. We present a case of adult onset cutaneous limited IgA vasculitis diagnosed by the characteristic lower extremity purpura and a positive skin biopsy.</p> <p>Case Presentation : A 42 year old male without significant medical history presented with a two week history of a gradually worsening rash on the bilateral lower extremities, trunk, and right upper extremity. The initial symptom was a nonpruritic rash on the right shin, noted at the time to be like pinpoint macules. These areas enlarged over the course of 2-3 days prompting him to seek care. Physical exam revealed scattered 1cm erythematous macules with necrotic centers on the right leg. He was diagnosed with dermatitis of uncertain etiology and prescribed a 10 day course of Bactrim and a Medrol dose pack. Despite this treatment the rash continued to worsen so approximately 1.5 weeks later he presented to the ED.</p> <p>On presentation vital signs were unremarkable. Workup was remarkable for a leukocytosis of 14 with a neutrophilic predominance. Electrolytes, inflammatory markers, MPO/PR3 antibodies, and a basic infectious workup were all either negative or unremarkable. Culture of the lesions and blood cultures did not demonstrate growth. The lesions were biopsied revealing weak granular deposition of IgA within the walls of the superficial dermal vessels. He was diagnosed with adult onset cutaneous limited IgA vasculitis and was managed conservatively with vanicream dressings and given close PCP and dermatologic follow-up.</p> <p>Discussion: IgA vasculitis is the most common form of systemic vasculitis in children. However, it is much rarer in adults, who contribute only approximately 10% of IgA vasculitis cases. The classically taught symptoms include palpable purpura without thrombocytopenia or coagulopathy, arthritis/arthralgia, abdominal pain, and renal disease. Adult patients, however, appear less likely to experience the non-rash symptoms of IgA vasculitis. This is consistent with our patient who denied any abdominal pain or arthritis. Of note, adults are more likely to develop renal disease, which can develop after the acute episode. Treatment is largely supportive as studies have not shown convincing long-term benefit with steroids. Given the higher chance of renal involvement in adults, we referred the patient for weekly follow-up with his PCP for continued renal/electrolyte monitoring.</p>
<p><b>Antionius Hazim</b> Dr. Gordon Ruan Dr. Diego Suarez Dr. Meltiady Issa</p>	<p><i>Light Chain Cast Nephropathy: A Race Against Time</i></p> <p>Case Presentation: A 57-year-old woman, with a past medical history of systolic heart failure (ejection fraction of 37%), presented to the emergency room for progressive fatigue, oliguria, and dyspnea of 2 weeks duration. Laboratory evaluation showed a creatinine of 9.22 mg/dL (up from 0.54 mg/dL five months prior), bicarbonate of 17 with an anion gap of 21, and albumin of 3.3 g/dL. Hemoglobin, platelets, potassium,</p>

	<p>and calcium were all normal. Urinalysis revealed a protein to osmolality ratio of 4.88 with predicted 24-hour urine proteinuria of 2819 mg/day, 4-10 white blood cells, and no red blood cells. Computed tomography imaging of the abdomen and pelvis showed no hydronephrosis. She was admitted to the hospital and, given the concern for glomerulonephritis, underwent a renal biopsy, which revealed lambda light chain cast nephropathy. Serum protein electrophoresis showed a significantly elevated lambda free light chain of 446 mg/dL. Bone marrow biopsy revealed 30% lambda light chain-restricted plasma cells, confirming the diagnosis of multiple myeloma. Skeletal survey showed lytic lesions in the C7 and T1 vertebral bodies. The patient was promptly started on chemotherapy in addition to emergent plasmapheresis, which resulted in improvement in her renal function with a new baseline creatinine at 5.27.</p> <p>Of note, the patient's kidney biopsy revealed a section of the interstitial compartment that stained positive for Congo red. Systemic amyloidosis was later confirmed with fat pad aspiration. Suspicion for cardiac amyloid was high due to a low-voltage electrocardiogram and a repeat transthoracic echocardiogram that revealed concentric left ventricular hypertrophy with elevated filling pressure, abnormal strain with apical sparing, and a small pericardial effusion.</p> <p>Discussion: Acute renal failure from light chain cast nephropathy is a myeloma defining event that requires immediate diagnosis and early institution of therapy to prevent irreversible kidney damage and restore renal function. It is important to keep multiple myeloma on the differential, especially in renal failure, as it could be the main presenting manifestation as seen in this patient. Similarly, this case emphasizes the benefit of obtaining a renal biopsy when faced with a rapidly progressive acute renal failure of unclear etiology. Finally, AL/AHL amyloidosis should also be ruled out if there are clinical signs suggestive of amyloid involvement as up to 30% of patients with multiple myeloma may also have amyloidosis at presentation.</p>
<p><b>Meghan Hill</b> Dr. Adam Sawatsky</p>	<p><i>Hypersensitivity Pneumonitis Presenting as an Acute Febrile Illness</i></p> <p>Introduction: Hypersensitivity pneumonitis (HP) is a syndrome characterized by small antigens in the alveoli that provoke an immune response of the small airways and lung parenchyma. The inciting antigens include agricultural dusts, fungi, bacterial and animal proteins, and low molecular weight chemical compounds. HP is classified into acute, subacute, and chronic, but there is variability and overlap in the presentation.</p> <p>Case: A previously healthy, unvaccinated 20-year-old man presented with new onset headache and altered mental status. He lived on a farm with multiple barn animals and worked at a sawmill. He was shoveling moldy corn, and a few hours later developed fevers and a diffuse headache. Other symptoms included non-bloody emesis, arthralgia, myalgia, weakness, shortness of breath, nonproductive cough, chest pain, confusion, and lethargy. He was evaluated in the emergency department the following day due to worsening symptoms.</p> <p>On arrival, his temperature was 39.4C, heart rate 108, blood pressure 150/74, respiratory rate 33, and oxygen saturation was 99% on room air. On exam, he was toxic-appearing with neck rigidity. His lungs were clear to auscultation bilaterally. Laboratory evaluation showed a WBC count of 11.6 (3.4 – 9.6). His chest x-ray was normal. A lumbar puncture was performed which was unremarkable, except for a protein of 41mg/dl (0– 35 mg/dl). He was given vancomycin and ceftriaxone for possible meningitis and intravenous fluids. About 4 hours after admission, he developed worsening dyspnea and required 5 liters of oxygen via nasal cannula. Repeat chest x- ray exhibited new bilateral lower lung infiltrates with increased pulmonary vascularity. A chest CT demonstrated bilateral ground glass densities with prominence of the interlobular septa involving the bilateral lower lobes. Further workup, including blood cultures, urine analysis, HIV, CSF studies, Q Fever, Lyme disease, brain MRI, and transthoracic echo, were negative.</p> <p>He showed significant improvement on his second day of admission with resolution</p>

	<p>of fevers and his oxygen requirement. Given his negative infectious workup, clinical improvement, and relation of his symptoms to his exposure to the moldy corn, he was diagnosed with acute hypersensitivity pneumonitis. He was discharged on his third day of admission in stable condition with counselling to avoid exposure to moldy corn.</p> <p>Discussion: This case highlights the importance of considering hypersensitivity pneumonitis in patients presenting with a non-specific febrile illness. HP patients can present acutely and be misdiagnosed as having a serious infection such as meningitis. Lung exam and imaging are often normal at initial presentation, so a high index of suspicion is necessary for making the diagnosis. The treatment of acute hypersensitivity pneumonitis is primarily supportive and requires avoidance of the instigating antigen exposure.</p>
<p><b>Rachel Husmann</b> Dr. Evan Beacom</p>	<p><i>The Lump in My Throat: A Rare Presentation of Mediastinal Tuberculous Lymphadenitis in an Immunocompetent Adult</i></p> <p>Introduction: One quarter of the world population is thought to be infected with Mycobacterium tuberculosis. Tuberculous lymphadenitis is the most common form of extrapulmonary tuberculosis, and typically develops from latent disease reactivation in lymph nodes seeded during a primary infection. Cervical lymph node involvement is most common, however, any group of lymph nodes may be affected. Mediastinal nodal disease most often accompanies pulmonary infection and is more often seen in immunocompromised hosts.</p> <p>Case Description: An 18 year old woman who immigrated from East Africa at age 12 and had no past medical history presented with three weeks' sensation of a lump in her throat that moved up and down with swallowing. This progressed to dysphagia with solids, then to severe odynophagia even when swallowing liquids. There was accompanying burning epigastric pain radiating to the throat, vocal hoarseness, chills, and night sweats. Rapid strep antigen, TSH, heterophile antibodies, and thyroid ultrasound had been performed and were normal. An esophagram was obtained which showed an esophageal filling defect at the level of the aortic arch. A subsequent chest CT showed severe narrowing of the mid-esophagus and a large heterogeneously enhancing posterior mediastinal mass. There were several foci of air in the mass and associated mediastinal and lower cervical lymphadenopathy.</p> <p>The patient was admitted to the hospital for worsening symptoms and inability to take food or drink by mouth. Screening for HIV, histoplasma, and blastomyces was negative. An IGRA was positive. Initial cultures from EUS FNA of the mass grew GI commensals but failed to yield tissue diagnosis. Repeat biopsy showed necrotizing granulomatous inflammation but was negative for tuberculosis by probe and smear. Flow cytometry showed no lymphocyte clonality. Empiric treatment for tuberculosis was started, and cultures later grew M. tuberculosis consistent with a diagnosis of mediastinal tuberculous lymphadenitis. Isolates were pan-sensitive and the patient continued four-drug therapy.</p> <p>Discussion: Mediastinal tuberculous lymphadenitis is an uncommon presentation of extrapulmonary tuberculosis, and is most often a complication of primary tuberculosis infection rather than latent tuberculosis reactivation. Mediastinal tuberculous lymphadenitis may present with dysphagia, odynophagia, or chest discomfort due to compression of the esophagus and trachea. Complications may include vocal cord paralysis due to compression of the recurrent laryngeal nerve, esophageal perforation, or pulmonary artery occlusion. It is unusual to see such manifestations of this common disease in the immunocompetent host. This presentation was thus unlikely in our patient, a young immunocompetent woman with no evidence of active pulmonary disease. Globus sensation as a primary presenting complaint is a yet rarer experience of the disease, and is added here to many known and unusual presentations of mediastinal tuberculous lymphadenitis, while broadening the differential for "a lump in my throat."</p>



<p><b>Steven Hwang</b> Dr. George Wang Dr. Erika Weil Dr. Rajiv Pruthi</p>	<p><i>Spontaneous Heparin-Induced-Thrombocytopenia Presenting as Cerebral Venous Sinus Thrombosis</i></p> <p>Introduction: Heparin-induced thrombocytopenia (HIT) is a disorder with autoimmune-like features that causes thrombocytopenia and promotes thrombosis, typically following exposure to heparin. However, there have been reports in the literature of clinically similar syndromes without identifiable proximate heparin exposure. This disease entity has been termed “spontaneous HIT” and appears to have a strong predilection for patients who have undergone orthopedic surgery, specifically total knee arthroplasty (TKA). Although the pathophysiology of this association remains unclear, platelet activation triggered by the formation of immunoglobulin (IgG)-platelet factor 4 (PF4) antibody complexes with specific polyanions found in cartilage has been proposed as a possible mechanism.</p> <p>Case Presentation: We present a case of a 56-year-old female who underwent TKA without proximate exposure to heparin, and on postoperative day (POD) 11 presented to a local emergency department for evaluation of acute-onset bi-frontal headache and aphasia. Non-contrast head CT showed a small intraparenchymal hemorrhage involving the posterior left temporoparietal region. CT venogram revealed thrombosis of the left transverse and sigmoid venous sinuses and upper left internal jugular vein. Her platelet count on presentation was 43 x 10<sup>3</sup> per ul, as compared to a preoperative baseline of 360 x 10<sup>3</sup> per ul. The patient’s serum tested positive for anti-PF4/heparin antibodies by enzyme-linked immunosorbent assay (ELISA) and by serotonin release assay (SRA). The patient was treated with the direct thrombin inhibitor bivalirudin and also received two doses of IVIG. Her platelet count increased to 148 x 10<sup>3</sup> per ul on POD 25. She was then transitioned to warfarin for long-term anticoagulation therapy in the outpatient setting.</p> <p>This case represents the 20th reported case of spontaneous HIT, of which 12 (60%) occurred shortly after knee replacement surgery. To our knowledge, our patient is the first case of cerebral venous sinus thrombosis resulting from a complication of spontaneous HIT. Of the 11 previous post-TKA cases, initial presentations included: 4 adrenal hemorrhages, 3 pulmonary embolisms (PEs) and/or deep vein thromboses (DVTs), 3 PEs with adrenal hemorrhages, and 1 thrombotic vertebral artery stroke. On review of the literature, there have been 6 cases of treatment failure (defined as symptomatic thrombosis or major bleed while on therapeutic levels) with argatroban. Given this, we decided to treat our patient with bivalirudin and her thrombocytopenia resolved 14 days after initial presentation.</p> <p>Discussion: In summary, we report the novel complication of CVST in a case of spontaneous HIT after knee replacement surgery effectively treated with bivalirudin and IVIG. Further investigation of risk factors, potential pathophysiology, clinical presentation, and effective therapies is warranted as this rare subtype of heparin becomes more widely recognized in the literature. This case ultimately highlights the importance of considering spontaneous HIT when presented with thrombocytopenia and thrombotic complications after orthopedic surgery, especially knee arthroplasty.</p>
<p><b>Candace Jackson</b> Dr. Alice Gallo De Moraes</p>	<p><i>Severe Babesiosis with Lyme Co-infection Requiring Exchange Transfusion</i></p> <p>Background: Babesiosis is a rare protozoan infection transmitted by the Ixodes scapularis tick. Babesia microti is the most prevalent species in the U.S. and endemic to the Northeast and upper Midwest. Infection can be asymptomatic or cause severe life-threatening disease, particularly in older and immunocompromised patients. Potential complications include hemolytic anemia, thrombocytopenia, disseminated intravascular coagulation (DIC), acute respiratory distress syndrome (ARDS), and acute renal failure.</p> <p>Case Presentation: A 77-year-old man presented to a hospital in Wyoming during the summer with rigors, weakness, and lightheadedness after flying in from Florida. He was febrile, hypoxic, and hypotensive, with acute kidney injury and a normal CBC.</p>

	<p>Vancomycin and ceftriaxone were started, but urinalysis, chest x-ray, blood cultures were ultimately unremarkable. Additional history revealed that he was recently outdoors in Pennsylvania. He was started on empiric doxycycline and Lyme serologies were sent. However, a couple days later, he developed anemia (hemoglobin 11.3 g/dL) and thrombocytopenia (platelets 39x109/L). Liver tests showed total bilirubin 1.8 mg/dL, AST 182 U/L, and ALT 61 U/L. Haptoglobin was low and LDH was significantly elevated (&gt;900 U/L). Blood smear showed intraerythrocytic inclusions concerning for babesiosis, with parasitemia estimated at 10-20%. Babesia microti serology and PCR were positive. Quinine and clindamycin were started, but the patient developed septic shock, oliguria, and ARDS. He was volume resuscitated, intubated, and initiated on vasopressors. Red blood cell exchange transfusion was ultimately performed, with parasitemia decreasing from 3.9% to 0.59%. Continuous renal replacement therapy was initiated, and he was subsequently extubated. After stabilization, antibiotics were changed to azithromycin and atovaquone to complete a 14-day course. Lyme serology and immunoblot returned positive, and the patient completed a 21-day course of doxycycline for Lyme co-infection.</p> <p>Discussion: We describe a case of severe babesiosis in a patient who presented to a community emergency department where Babesia microti is not endemic. This case highlights the importance of obtaining a thorough travel history and maintaining a high index of suspicion for tick-borne diseases in patients presenting with flu-like illnesses during the summer. While babesiosis infections can be asymptomatic, some patients present with severe clinical manifestations. Severe disease is often associated with parasitemia of 4% or greater and occurs in patients who are older or immunocompromised and in those with asplenia. Antimicrobial therapy includes azithromycin plus atovaquone or clindamycin plus quinine. Usual duration is 7-10 days in immunocompetent patients. If there is significant parasitemia or severe multi-organ dysfunction, transfer to a center capable of performing red blood cell exchange transfusion should be considered. Lastly, an important consideration is the potential for co-infection with other tick-borne illnesses, as Borrelia burgdorferi and Anaplasma phagocytophilum are also transmitted by the Ixodes scapularis tick.</p>
<p><b>Sana Khan</b> Dr. Haruka Torok</p>	<p><i>Right Pleural Effusion and Left Neck Swelling: Are They Connected?</i></p> <p>Case Presentation: A 19-year old African American female presented to the outside ED with a 2-day history of a lump in the left side of her neck, associated with pain, chest tightness and shortness of breath. CT of the neck and chest demonstrated retropharyngeal edema, fluid and inflammatory changes in the left neck, as well as moderate right-sided pleural effusion. She was started on empirical antibiotics and transferred to our hospital due to the concern for retropharyngeal abscess. On admission, she described episodes of nausea with dry heaving with subsequent odynophagia and dysphagia. She reported subjective fever, but denies any URI symptoms, dental problems or any major trauma. Her past medical history was significant for polycystic ovarian syndrome and attention deficit hyperactivity disorder. She was not on any medication. Family history was unremarkable. She was working as a nursing aid in a nursing home. She was smoking marijuana on most days for the past six months. On physical exam, she was afebrile with normal vital signs. Left neck appeared full, tender to palpation without areas of fluctuance or induration. Oropharynx was clear. No palpable cervical or supraclavicular lymphadenopathy. Breath sounds were diminished at the right lower lung field. The remainder of her exam was unremarkable. Her labs were significant for WBCs of 12,100/mm3. ESR was 5mm and C-reactive protein was negative. Thoracentesis was performed and yielded 700 ml of pink milky effusion. Fluid analysis showed WBCs of 591/micro l (neutrophils 24%, lymphocytes 8%), protein 3.5 g/dl, LDH 227 u/l and triglycerides were markedly elevated at 1,939 mg/dl (serum triglyceride of 82 mg/dl) suggestive of chylothorax. Pleural fluid cytology, gram stain, bacterial and fungal cultures were all negative. Antibiotics were discontinued. She was initially made NPO and transitioned to no-fat diet. Further testing revealed nonreactive HIV, negative ANA and TB Gold quantiferon tests, normal complement C3 and C4, and IGG subclasses. Esophagogram was performed to rule out esophageal injury leading to thoracic duct irritation, which</p>

	<p>was unremarkable. After the thoracentesis, her chest symptoms improved, and her neck symptoms gradually resolved with supportive care. Repeat neck and chest CT on hospital day 5 revealed resolution of right sided pleural effusion and near complete resolution of retropharyngeal edema. Lymphangiogram and lymphoscintigraphy were deferred as there was no re-accumulation of fluid. <b>DISCUSSION:</b> A pleural fluid triglyceride concentration greater than 110 mg/dl strongly supports the diagnosis of chylothorax.</p> <p>Major causes of chylothorax include traumatic and non-traumatic. Traumatic could be iatrogenic and non-iatrogenic. Non-traumatic causes are divided into malignant and non-malignant. Non-malignant causes can be idiopathic, congenital and miscellaneous. To evaluate the cause of chylothorax, CT of the thorax and abdomen is indicated to identify the presence of lymphadenopathy or masses and the site of thoracic duct rupture. If the site of leak remains uncertain, lymphangiography or lymphoscintigraphy may be helpful. Lymphangiography can identify areas of chyle leakage in the majority of patients. However, it is a technically difficult procedure and presents risks.</p> <p>Discussion: Although we were unable to confirm, the possible etiology in our patient is thoracic duct disruption from repetitive dry heaving and retching spells. The thoracic duct leak closes spontaneously in nearly 50% of patients, and thus, conservative management is the first step. Chyle production can be reduced by instituting TPN or a fat-restricted oral diet supplemented with medium chain triglycerides. Other treatment options include octreotide, which reduces the splanchnic blood flow and the absorption of triglycerides, decreases gall bladder contractility and bile flow, and has been used for chylothorax with great success. For patients not responding to conservative therapy, definitive intervention is warranted. Options include pleurodesis, thoracic duct embolization or disruption (TDE/TDD), thoracic duct ligation (TDL), or combinations of these.</p>
<p><b>Brittany Kimball</b> Dr. Hannah Lichtsinn</p>	<p><i>Sickle Cell <math>\gamma\delta\beta</math>-zero : A Rare Phenotype with the Same Risk for Pain and Stigma</i></p> <p>Case Presentation: A 21-year-old woman with a history of sickle cell disease (SCD) with Hgb S/Black (<math>A\gamma\delta\beta</math>)<sup>0</sup>-thalassemia was admitted for vaso-occlusive crisis with pain in her right arm. Her disease course had been complicated by avascular necrosis of the right shoulder, pulmonary necrosis of the right lung, and repeated vaso-occlusive crises of the right humerus. She had been taking NSAIDs and oral oxycodone at home without relief. On admission, her hemoglobin was 12.5 mg/dL and she was noted to have evidence of hemolysis. Her baseline Hgb F was 20-25%, not on hydroxyurea. She was treated with IV fluids and fentanyl for a sickle cell crisis. When planning for discharge, the attending physician documented his reluctance to provide outpatient pain treatment, saying: "The question is, how much pain should a patient like this [Hgb S heterozygote] have?... I do know we have a national epidemic with opiates and we should be cautious in giving opiates at discharge."</p> <p>Discussion: People with SCD often face stigma related to treatment with opioids for pain. This stigma has anecdotally increased with the increased attention to the opioid epidemic in the United States. However, opioid-related death and by extension, opioid abuse, are rare among patients with SCD. Further, it is important to recognize and understand the myriad of SCD phenotypes. This patient has a very rare phenotype, Hgb S/Black (<math>A\gamma\delta\beta</math>)<sup>0</sup>-thalassemia. Despite relatively mild anemia and high hemoglobin F values characteristic of this variant, patients develop classic complications of SCD, including pain crises. Not recognizing the significance of this variant can lead to under treatment of SCD complications. Through a social lens, this case provides an opportunity to explore the intersections between bias in medicine and the ways in which reactions to the opioid crisis have affected people with SCD.</p>
<p><b>Bradly Kimbrough</b> Dr. Vuyisile Nkomo</p>	<p><i>A Case of Thyrotoxic Periodic Paralysis in a 40-Year-Old Male</i></p>

	<p>Introduction: Periodic paralysis is a rare manifestation of hyperthyroidism that predominately affects males and those of Asian ancestry. Though uncommon, practitioners should be aware of this symptom and its connection to hyperthyroidism as well as key points in management.</p> <p>Case Presentation: A 40-year-old Asian male presented to the emergency department (ED) with significant generalized weakness. For the previous 3 months, he experienced episodic paralysis typically affecting his lower extremities and occurring after intense exercise, alcohol, or development of an upper respiratory tract infection. Each episode would last a couple of hours. He had no significant past medical history other than a recent diagnosis of hyperthyroidism three days prior when he was found to have an undetectable thyroid stimulating hormone and a free thyroxine (T4) of &gt;7.7 ng/dL (0.9-1.7 ng/dL). He was subsequently started on methimazole.</p> <p>In the ED he was found to have significant weakness of both his upper and lower extremities. Laboratory analysis was significant for severe hypokalemia to 2.3 mmol/L (3.6-5.2 mmol/L) and minimal hypercalcemia to 10.1 mg/dL (8.6-10.0 mg/dL). He was administered 20 mEq of intravenous and 60 mEq of oral potassium supplementation and admitted to the cardiology service. When he arrived to the floor he had already regained his strength. Endocrinology was consulted and recommended continuation of methimazole and initiation of propranolol. He was discharged with endocrinology follow-up and instructions to take 20 mEq of oral potassium if symptoms of paralysis resumed.</p> <p>Discussion: About 95% of cases of thyrotoxic period paralysis (TPP) occur in males, and among Asian men with hyperthyroidism, about 10% will experience TPP. Episodes tend to occur after intense exercise, large meals, alcohol intake, infection, or albuterol use. Though not fully understood, it is thought that increased levels of thyroid hormone leads to increased beta adrenergic receptor responsiveness and thus sodium-potassium ATPase pump activity. This causes hypokalemia leading to hyperpolarization of the muscle membrane. Clinicians should also be aware that of patients who develop TPP, 15% will exhibit paralysis prior to other symptoms of hyperthyroidism and 50% will exhibit paralysis at the same time as other symptoms of hyperthyroidism.</p> <p>Effective long-term treatment of TPP is centered on correction of the underlying thyrotoxicosis. In the acute setting, however, gentle replacement of potassium generally results in symptomatic improvement. Clinicians should be aware that hypokalemia does not reflect a deficiency in total body potassium, and over-administration of potassium may result in subsequent hyperkalemia. Therefore, it is not recommended to give more than 90 mEq of potassium in the first 24 hours.</p> <p>Additionally, oral propranolol has been shown to reduce frequency and severity of TPP. In cases refractory to potassium supplementation, intravenous propranolol may be administered which blocks excessive beta adrenergic activity thereby reducing further intracellular shifting of potassium.</p>
<p><b>Vinayak Kumar</b> Dr. Catalina Sanchez Dr. Uma Thanarajasingam</p>	<p><i>A Paralyzing, Nerve-Wracking Ordeal</i></p> <p>Introduction: Systemic Lupus Erythematosus (SLE) is a disease associated with malar rash, joint disease, serositis, oral ulcers, hematologic cytopenias, renal dysfunction, and neurologic dysfunction. Neuroimmunologic disorders such as multiple sclerosis and Neuromyelitis Optica Spectrum Disorder (NMOSD) cause neurologic deficits in the central nervous system, most commonly in a relapsing-remitting clinical course. Transverse myelitis is a manifestation of all three diseases.</p> <p>Case Presentation: KG is a 32 y.o. Native American female with multiple miscarriages and recent uncomplicated pregnancy (G4P1) in August. She subsequently had a 1-month history of arthralgias and myalgias that led to a very recent diagnosis of SLE, and was initiated on prednisone and Plaquenil with unclear</p>

	<p>medication adherence. One month later, she presented to an outside hospital with flaccid paralysis and loss of all sensation in the bilateral lower extremities, symmetric joint pains throughout the upper body and extremities, and a heliotrope rash. Initial labs were notable for anemia (Hgb 8), transaminitis, and autoimmune serologies consistent with lupus (elevated ESR/CRP, low C3/C4, and antibodies to ANA, dsDNA, U1RNP and Smith). MRI of the spine demonstrated longitudinally extensive transverse myelitis. She was initiated on solumedrol 1 g daily for 8 days and received 3 treatments of PLEX before being transferred to Mayo for further care.</p> <p>At Mayo Clinic, lumbar puncture indicated elevated protein, low glucose, and lymphocytic predominance with elevated IgG without oligoclonal bands. CSF was negative for infection and NMO/AQP4-IgG. Subsequent serologic workup confirmed lupus serologies (low C3/C4, with antibodies to ANA, dsDNA, and Ribosome P). Bloodwork indicated normal vitamin levels, low copper/ceruloplasmin with otherwise normal metal levels, and no infectious causes. A neuroimmunology panel demonstrated a positive MOG antibody, confirming the diagnosis of NMOSD as a driver for the longitudinally extensive transverse myelitis, rather than SLE or copper deficiency. The patient was continued on prednisone 60 mg daily with taper over 3-6 months, started on azathioprine after TMPT test indicated normal metabolism of the medication, and underwent PLEX for a total of 7 sessions. Repeat MRI indicated improvement of spinal inflammation, though there is no clinical improvement. There is ongoing discussion regarding switching medications to Cytoxan with appropriate prenatal/postnatal counseling.</p> <p>Discussion: Transverse myelitis and Guillain Barre should be considered when a patient presents with bilateral lower extremity flaccid paralysis. The differential for transverse myelitis includes vascular myelopathies, metabolic and nutritional myelopathies, CNS neoplasms, autoimmune disease, infection, paraneoplastic processes, and multifocal neurologic disorders. It is important to characterize the extent of the transverse myelitis on MRI to narrow the differential. Lastly, this disease takes 1-3 months for partial improvement, may take years for full recovery, and has a 70% recurrence rate in patients with systemic autoimmune disease.</p>
<p><b>Rebecca Kummer</b></p>	<p><i>The Case of the Positive Pulmonary O&amp;P</i></p> <p>Introduction: Strongyloides stercoralis is an intestinal helminth endemic to the tropics and subtropics that is unique for its ability to re-infect its host by reentering the blood stream from the GI tract or perianal skin in a process called autoinfection. This allows for the infection to persist for decades in the host, often without producing symptoms. Strongyloides hyperinfection syndrome occurs when patients chronically infected with S. stercoralis become immunosuppressed, most commonly by administration of corticosteroids, allowing the larvae proliferate uncontrollably and potentially disseminate to end-organs. Mortality rates of greater than eighty percent have been reported in hyperinfection syndrome with disseminated disease.</p> <p>Case Presentation: Here we present a case of a 59-year-old Ecuadorian man who originally presented with cough, dyspnea and hypoxia and was treated with antibiotics and corticosteroids. He was discharged in good health but presented 2 weeks later with hemoptysis and respiratory distress. He was found to have strongyloides hyperinfection syndrome, diagnosed by larvae present in his sputum. His course was complicated by acute respiratory distress syndrome and prolonged hypoxic respiratory failure leading to tracheostomy.</p> <p>Discussion: It is important to consider strongyloides infection in all patients who are from or who have traveled to endemic regions, and to test for and treat Strongyloides prior to administering corticosteroids in order to prevent strongyloides hyperinfection syndrome and its complications.</p>
<p><b>Jeffrey Larson</b></p>	<p><i>Back to the 80s: Disseminated Histoplasmosis, CNS Toxoplasmosis, and Fulminant Pneumonia as an Initial Presentation of HIV/AIDs</i></p>

	<p>Case Presentation: A 23 year old male with unknown past medical history presents with fevers, shortness of breath, and altered mental status. He was promptly intubated for acute hypoxic respiratory failure with initial chest imaging consistent with ARDS.</p> <p>Admission labs notable for positive HIV-1 antibody, HIV-1 viral load &gt; 2 million, low CD4 count suggestive of AIDS, severe anemia, renal failure requiring CRRT, and hepatitis. CT head notable for multifocal hypo-intense areas of edema and follow-up MRI was notable for multifocal ring-enhancing lesions. Peripheral smear was negative for hematologic malignancy, but notable for neutrophils and monocytes containing small oval intracytoplasmic inclusions suspicious for histoplasmosis. His urine, serum, and CSF samples had positive histoplasmosis antigens as well, overall consistent with disseminated histoplasmosis s/p initiation of CNS-dose liposomal amphotericin B. His quantitative CMV levels were elevated s/p treatment with ganciclovir in the setting of progressively worsening transaminitis and hyperbilirubinemia. His serum toxoplasmosis IgG and CSF toxoplasmosis PCR both returned positive s/p treatment with bactrim for CNS toxoplasmosis (significant financial barriers to first-line therapy with pyrimethamine and sulfadiazine). He was also empirically treated with IVIG in the setting of positive parvovirus PCR given the severity of his illness.</p> <p>On hospital day 14 antiretroviral therapy was initiated as the risks of ongoing immunosuppression outweighed the risks of immune-reconstitution inflammatory syndrome (IRIS). Patient was extubated on hospital day 16 and endorsed knowledge of his HIV status, previous intermittent use of ART (citing cost and lack of health insurance as barriers), and cited MSM as his source of transmission. He eventually developed IRIS on hospital day 24 as depicted by significant fever and acutely worsening mental status requiring re-intubation and a course of steroids. Fortunately, the patient was able to be extubated, his myriad of infections gradually improved, and he was eventually discharged after ~2 months of hospitalization to a rehabilitation facility given the presence of persistent neurologic deficits.</p> <p>Discussion: Despite significant advancements in HIV treatment since the US AIDS epidemic in the 1980s, barriers to healthcare and poverty continue to propagate HIV/AIDS presentations today. Furthermore, the most striking-feature of his initial presentation was ARDS secondary to fulminant bilateral pneumonia. A case-report published by clinicians at HCMC in 2017 (Prekker et. al) provides a concise guideline for empiric treatment of fulminant bilateral pneumonia in addition to a list of offending agents to consider which is a helpful resource for all internal medicine providers. Additionally, first-line therapy for CNS toxoplasmosis was unavailable to the patient due to high costs of said medications in the inpatient setting; fortunately, medical providers and social workers are able to circumnavigate these financial constraints in some uninsured patients that enroll in emergency medical assistance and medicaid during their acute illness.</p>
<p><b>Christina Lee</b> Dr. Erik Engebretson</p>	<p><i>Liver Infarction and Rupture Secondary to Left Segmental Portal Vein Thrombosis</i></p> <p>Introduction: Portal vein thrombosis is the most common cause of extrahepatic portal vein obstruction, having a prevalence of 1% in autopsy studies. Patients typically present with abdominal pain, diarrhea, bloody stools, and fever. Diagnosis is made with CT with contrast and/or ultrasound, and management consists of anti-coagulation.</p> <p>Case Presentation: This case begins with a 73-year-old man, with a past medical history notable for HTN, and celiac trunk and hepatic artery aneurysms, for which he underwent open bypass surgery 1 month prior to presentation. He presented to the hospital with one day of sudden onset of severe, constant, and sharp right upper quadrant pain that radiated to the back. The pain was associated with nausea, non-bloody emesis x1, and 4-6 loose, brown stools daily for the past 6 weeks, with no hematochezia or melena. Enteric panel was negative at the time. Patient denied fevers, chills, recent travel, diet changes, and alcohol use. On physical exam, his abdomen</p>

	<p>was distended, tense, and exhibited tenderness to light touch in the RUQ, and positive Murphy’s sign, but no rebound or guarding. Labs were notable for the following: WBC 13 K/cmmm (4-11), CRP 4.9 mg/L (0-3), lactic acid 2.5 mmol/L (0.4-2), lipase 5831 U/L (77-393), AST 198 U/L (15-37), ALT 93 U/L (13-61), Alk phos 144 U/L (45-117), Total bilirubin 1.4 mg/dL (0.2-1), direct bilirubin 0.9 mg/dL (0.1-0.2). Due to concerns for acute cholecystitis, an ultrasound was performed, which was unremarkable. Patient became increasingly febrile and hypoxic, prompting a CT abdomen/pelvis w/o contrast, which showed left liver lobe infarction. Due to concerns for stenosis of the recently repaired aneurysms, angiogram was performed, which showed widely patent hepatic artery bypass, with patent spleen and mesenteric arteries. A subsequent CT a/p w/o contrast showed free abdominal air consistent with liver capsule rupture. Shortly thereafter, he developed E.coli sepsis and required a long convalescent period.</p> <p>Upon second review of the CT a/p by radiology, there were multiple areas of subsegmental thrombi in the left portal vein. Further discussion with the vascular surgeon revealed that the left hepatic artery was lost at the time of surgery. Thus, the patient lost both arterial and portal blood flow to the left lobe of the liver, resulting in ischemia, necrosis, abscess formation, and capsular rupture.</p> <p>Discussion: The liver has a unique dual blood supply designed to protect against ischemic damage and hepatic infarction requires a dual hit to the blood supply. Once the hepatic tissue is necrotic, intestinal bacteria can travel via the portal system into the dead tissue and form abscesses. With gas forming microorganisms, this can result in capsular rupture and systemic infection.</p> <p>This case highlights the importance of having a high suspicion for liver infarction, especially in patients with potentially impaired blood supply.</p>
<p><b>Allison Levy</b></p>	<p><i>Collateral Damage</i></p> <p>Introduction: Esophageal varices are the most common manifestation of portal hypertension, and ruptured varices are the deadliest complication. Cirrhosis is the most common cause of portal hypertension and varices in the United States. Varices can arise in the absence of cirrhosis or even portal hypertension as in the case of systemic hypertension secondary to an Inferior Vena Cava thrombosis.</p> <p>Case Presentation: A 62 year old man with history of right lower extremity deep vein thrombosis on Xarelto, chronic bilateral lower extremity edema presented to the emergency department with acute episode of coffee ground emesis and presyncope. He was found to have a hemoglobin of 7.9. Overnight, his hemoglobin dropped to 6.5 and he received 3 units of packed red blood cells and underwent urgent upper endoscopy where he was found to have two &gt;5 mm esophageal varices. Two bands were placed with complete eradication. With no prior history of underlying liver disease, he underwent further evaluation which was significant for normal liver function tests, abdominal ultrasound, and CT chest abdomen pelvis with no evidence of cirrhosis and patent portal vein. ANA, AMA, ASMA, hepatitis panel, Factor V Leiden, and Factor II gene mutation all negative. Iron studies were unremarkable and A1AT was within normal limits. Interventional radiology was consulted to perform a liver biopsy and upon review of CT, found evidence of a chronic IVC clot and apparent communication with portal venous system with recanalized paraumbilical veins seen. This was thought to be the etiology of the varices and patient underwent recanalization of the bilateral iliac veins and IVC and angioplasty and stenting of the infrarenal IVC, left and right common iliac veins, external iliac, and common femoral veins. He was continued on life-long anticoagulation and discharged in stable condition without recurrence of bleed.</p> <p>Discussion: This case illustrates a late consequence of DVT with extension into the IVC resulting in post-thrombotic syndrome, development of collaterals, and esophageal varices secondary to venous systemic hypertension. Although IVC clot</p>

	<p>with resultant esophageal varices is rare, post-thrombotic syndrome is not. Additionally, it is highly morbid. Therefore, recognition of this condition is critical to ensure appropriate therapy and early recanalization of the occluded vasculature.</p>
<p><b>Marissa Li</b> Dr. Mrinal Patnaik</p>	<p><i>A Rare Case of Cutaneous Blastic Plasmacytoid Dendritic Cell Neoplasm Arising from Clonal Cytopenia of Unknown Significance</i></p> <p>Introduction: Clonal cytopenia of unknown significance (CCUS) is a premalignant disorder characterized by the presence of expanded somatic mutant clones and peripheral blood cytopenias, in the absence of a defined hematological malignancy.<sup>2</sup> The most common mutations encountered in CCUS are DNMT3A, TET2, and ASXL1, which are typically associated with subsequent development into MDS and AML.</p> <p>Case Presentation: In our case study, a 68 year old gentleman with asymptomatic pancytopenia consistent with CCUS developed acute transformation into cutaneous blastic plasmacytoid dendritic cell neoplasm (BPDCN). Next generation sequencing was done on bone marrow and was positive for TET2 and ZRSR2 mutations. Sequencing done on his cutaneous lesions were similarly positive for TET2 and ZRSR2, but also included copy number deletions of CDKN2A, CDKN2B, and MTAP. He underwent 3 cycles tagraxofusp (anti- CD123 conjugated monoclonal antibody) and an allogenic stem cell transplant. After the 2nd cycle of tagraxofusp, he had near complete remission of his skin lesions. On our last follow up, he is 60 days post-transplant and doing very well. Blastic plasmacytoid dendritic cell neoplasm (BPDCN) is a rare and clinically aggressive hematologic malignancy. About 10-20% of patients who develop BPDCN have a history of MDS, AML or CML,<sup>4</sup> but the prevalence rates of antecedent CCUS remain unknown.</p> <p>Discussion: Our case illustrates the potential of CCUS to develop into not just MDS or AML, but also into BPDCN. We also demonstrate the correspondence of the two mutations (TET2, ZRSR2) found in the bone marrow of CCUS with the skin lesions of his subsequent BPDCN. The presence of CDKN2A/B and MTAP deletions in the BPDCN skin lesions suggest copy number variations as a likely methodology of clonal evolution from CCUS to BPDNC. Contrary to the thought that CCUS represents a cytopenia of unknown significance, this case indicates that CCUS does in fact have significant oncogenic potential for a variety of hematologic neoplasms - with subsequent genetic and epigenetic events determining progression to said variety of hematologic neoplasms.</p>
<p><b>Salman Mahmood</b> Dr. Parastoo Fazeli</p>	<p><i>Acute Onset Chest Pain: An Unusual Presentation of Giant Cell Arteritis</i></p> <p>Case Presentation: A 70-year-old Caucasian female presented with sudden-onset, central, non-radiating chest pain. She denied any associated symptoms including shortness of breath, nausea, vomiting, joint pain and swelling, fevers, headaches, jaw claudication and vision changes. Physical examination revealed normal heart sounds and there was no tenderness over the temporal arteries. There was no evidence of proximal muscle weakness and skin and joint exam was normal as well. She received a GI cocktail in the emergency department and within a few hours her chest pain resolved. Imaging revealed continuous thickening of the aorta extending from the ascending to the infra-renal portion, with adjacent soft tissue stranding. The ascending portion was also mildly aneurysmal at 4.2 cm but no dissection was seen. ESR and CRP were both significantly elevated. Temporal artery ultrasound did not reveal any areas of inflammation. A rheumatoid factor as well as immunoglobulin levels including IgG4 were normal. ANA, anti-dsDNA, MPO and PR3 were all negative. Workup for infection including syphilis, HIV and tuberculosis was also negative. During her admission, she developed intermittent fevers and empiric broad spectrum antibiotics were initiated. Blood cultures returned negative.</p> <p>The patient continued to have recurrent fevers despite antibiotic therapy and was subsequently started on high-dose prednisone in consultation with the rheumatology</p>



	<p>service. After a few days of receiving steroids the patient’s inflammatory markers started to improve and her fevers stopped. A temporal artery biopsy was later pursued which showed segmental transmural thickening of the vessel walls suggestive of healed arteritis. The patient was discharged and instructed to continue high-dose prednisone.</p> <p>At follow-up her inflammatory markers continued to improve and she was ultimately switched from prednisone to tocilizumab for maintenance. The patient stated that in retrospect she may have had pain and stiffness in both shoulders for a few months prior to presentation which she had attributed to shoveling the snow during winter.</p> <p>Discussion: This represents a case of giant cell arteritis with isolated aortitis at the time of presentation. The patient never developed any of the usual symptoms such as headache, jaw claudication, scalp tenderness or vision changes. She did endorse bilateral shoulder stiffness and pain suggestive of polymyalgia rheumatica which may have predisposed her towards developing giant cell arteritis. Even though the patient did not have any tenderness in her temporal arteries and her ultrasound was unrevealing, her biopsy revealed areas of trans-mural thickening suggestive of healed arteritis confirming the diagnosis. She was successfully treated initially with high dose steroids and later switched to tocilizumab with sustained benefit.</p>
<p><b>Jack McHugh</b> Dr. C.E.Harris</p>	<p><i>Acting Fast in Fasciitis</i></p> <p>Introduction: Necrotizing soft tissue infections (NSTIs) are among the most challenging diagnoses encountered by the internist. The initial presentation often resembles a relatively benign superficial skin infection. Meanwhile, the disease progresses rapidly below the surface, often with devastating consequences. Here, we describe a case that demonstrates the deceptive nature of this disease process, and discuss strategies to assist with early identification.</p> <p>Case Presentaton: A 63 year-old female reported to the emergency room with an 8-day history of general malaise. Medical history was significant for end-stage renal disease (ESRD) secondary to Type II diabetes mellitus, chronic anemia, and distant history of colorectal cancer status-post abdominoperineal resection with ileostomy formation. Three days prior to presentation she had an episode of peri-stomal leak, after which she developed progressive abdominal tenderness.</p> <p>On admission to the floor patient was afebrile and hemodynamically stable. Examination was notable for peri-stomal warmth, erythema, and tenderness. Laboratory testing showed WCC 8.4, CRP 310, ESR 134 and Glucose 534. Blood and urine cultures did not grow any organisms. Patient was started on IV Cefazolin and Metronidazole for presumed abdominal wall cellulitis. 24-hours post-admission the patient became hypotensive and hypothermic. Re-examination of the abdomen revealed spreading erythema with formation of hematoma and hemorrhagic bullae. Antibiotics were converted to IV Vancomycin, Cefepime, Metronidazole, and Clindamycin, and general surgery was consulted.</p> <p>Exploration and debridement of abdominal wall tissue was undertaken revealing dirty dishwater fluid and full-thickness necrotic subcutaneous tissue encircling the stoma to the level of the fascia. Patient required transfer to the ICU for vasopressor support and CRRT. Cultures of fluid revealed growth of Klebsiella oxytoca, Enterococcus faecalis, Streptococcus anginosus, and Staphylococcus epidermidis. Following two further resections of necrotic abdominal wall tissue, patient was discharged to a rehab facility two weeks post-admission.</p> <p>Discussion: NSTIs are among the most devastating infectious processes, with observed mortality rates of up to 21% for Type I polymicrobial necrotizing fasciitis, as seen in the this case<sup>1</sup>. Delays in surgery of greater than 24 hours are associated with increased mortality<sup>2</sup>; early diagnosis is therefore crucial. Here, the patient was hemodynamically stable at presentation, cutaneous findings were benign, and use of a</p>

	<p>scoring system such as LRINEC for NSTI identification was complicated by underlying co-morbidities (anemia, ESRD).</p> <p>In such cases clinical suspicion should be raised by the following: pain out of proportion to symptoms, hypotension, hemorrhagic bullae, elevated inflammatory markers, or hyperglycemia. Additional laboratory tests of benefit are CK and AST, which may be elevated in muscle necrosis. Finally, CT or MRI imaging should be undertaken which may identify soft-tissue swelling or gas.</p> <p>Conclusion: Internists should have a high index of suspicion for NSTIs when laboratory findings are discordant with cutaneous findings early in the disease course.</p>
<p><b>Charles Meade</b> Dr. Mark Enzler</p>	<p><i>An Unusual Cause of B Symptoms in a Cardiac Surgery Patient</i></p> <p>Case Presentation: A 60 year old man was admitted to the hospital for further evaluation of 6 month history of fatigue, arthralgias, night sweats, cognitive complaints, intermittent fever, and 20 pounds of unintentional weight loss. Medical history notable for hypertension, hyperlipidemia, type 2 diabetes mellitus controlled on metformin, and bicuspid aortic valve replacement.</p> <p>Physical exam was notable for scattered indurated violaceous papules on patient's abdomen and flank. Patient notes that skin rashes had started approximately 5 months ago and that new lesions have gradually appeared over time. Areas of rash are nonpruritic and nonpainful.</p> <p>Before presenting for evaluation, patient had undergone extensive evaluation in setting of B symptoms and skin changes. Negative TEE, cross sectional imaging of chest and abdomen, MRI of brain and spine, colonoscopy, CSF studies. Further negative serologic evaluation for evidence of HIV, syphilis, parvovirus, West Nile, CMV, Lyme, EBV, endemic fungal infections. No significant findings on evaluation of rheumatologic causes with ANA, ANCA, RF. Leukemia and lymphoma phenotyping on peripheral blood negative.</p> <p>Ultimately, he underwent broad further evaluation by infectious disease, neurology, hematology, and dermatology consult services. Further testing was pursued with bone marrow biopsy with bacterial, viral, and fungal staining, heavy metal screening, Q fever serologies, TB serology, cryoglobulins, and paraneoplastic antibody testing which were unrevealing. PET scan showed small pulmonary nodule, and uptake from areas of skin changes but was generally interpreted as unrevealing.</p> <p>Dermatologic assessment was suspicious for possible granulomatous process and proceeded with biopsy of representative lesion. Biopsy results revealed caseating and non-caseating granulomas as well as rare acid fast organisms on AFB and Fite staining. Mycobacterial cultures speciated <i>Mycobacterium chimaera</i>.</p> <p>Discussion: <i>Mycobacterium chimaera</i> is a non-tuberculous mycobacterium that has been linked to an outbreak of prosthetic valve infection and disseminated mycobacterial infection. Extensive epidemiologic investigation into the source of <i>M. chimaera</i> ultimately identified a contaminated manufacturing facility for heater-cooler units used for cardiac bypass procedures. Stockert 3T devices manufactured prior to September of 2014 have subsequently been removed from use. Health care professionals caring for patients who have previously undergone surgery should recognize this rare cause of disseminated mycobacterial disease in patients presenting with an appropriate clinical presentation. The initial manifestation of symptoms may occur up to years following cardiac surgery, as in our patient. Disseminated <i>M. chimaera</i> associated with prosthetic valve endocarditis can be difficult to treat and generally requires management in subspecialty non-tuberculous mycobacterial clinics.</p> <p>Our patient ultimately underwent aortic valve replacement and prolonged therapy with multiagent regimen of azithromycin, clofazimine, rifampin, and ethambutol. He</p>

	<p>continued to suffer from metastatic spread of his infection and recently was found to have biopsy-proven <i>M. chimaera</i> abscess at S1 level.</p>
<p><b>Thomas Meehan, Jr.</b>  Dr. Aaron Boothby  Dr. Rachel Heuer  Dr. Michael Newman</p>	<p><i>An Accelerated Workup of Pancytopenia at the Minneapolis VA</i></p> <p>Introduction: Pancytopenia is defined as concurrent anemia, leukopenia, and thrombocytopenia. It is not a disease itself, but a manifestation of an underlying disorder. Herein, we examine our diagnostic workup to determine the mechanism and cause of pancytopenia in a veteran in the inpatient setting.</p> <p>Case: A 77 year old man with metastatic castration-resistant prostate adenocarcinoma, atrial fibrillation on anti-coagulation, EtOH abuse, and 2 weeks s/p left ureter stent removal was admitted from the oncology clinic with a CBC showing WBC: 3.5, Hgb: 9.5, and Plt: 26, and a 4-month elevation in his PSA from 1.6 to 63. He endorsed malaise, a 20-pound weight loss over 2 months, and recent hematuria, hemoptysis, and epistaxis. He was last binge-drinking 1 month ago and his medication list included Propan with Iron (a non-FDA approved Filipino appetite stimulant). His wife endorsed ongoing upper respiratory symptoms since her recent return from the Philippines. On admission he was afebrile, breathing comfortably, frail appearing, and had ecchymoses on his face and forearm. Admission labs showed normocytic anemia, normal transferrin and elevated ferritin, nucleated RBC's, hypo-proliferative reticulocyte index, normal Haptoglobin, negative DAT, elevated LDH and fibrinogen, normal copper, zinc, B9, and B12 levels, low albumin, and a normal coagulation panel. A CT A/P was negative for a retroperitoneal bleed and a CXR was negative for an infectious process. A peripheral smear showed no biochemical evidence of hemolysis and a bone marrow biopsy showed 10% marrow cellularity and abundant metastatic carcinoma cells, involving &lt;5% of the sampled marrow space. Dexamethasone was started for possible concomitant ITP, but platelets were refractory to treatment. The veteran was resistant to palliative care and requested further cancer treatment. He was started on weekly Docetaxel and remained inpatient for 4 weeks for as needed transfusion therapy, in total receiving 12 units of platelets and 2 units of PRBC's.</p> <p>Conclusion: Although prostate cancer most frequently metastasizes to the bone, marrow invasion with resultant cytopenias is rare. We hypothesize that this patient's cell lines were low for a myriad of reasons including impaired production secondary to metastatic marrow infiltration, nutritional deficiency, and ongoing alcohol abuse, further exacerbated by post-surgical blood loss. It is paramount to obtain a complete history in working up an abnormal CBC when such a broad differential exists. Peripheral smear and bone marrow biopsy are safe and efficient diagnostic modalities with much utility in this setting.</p>
<p><b>Bryant Megna</b>  Dr. Aaron Boothby</p>	<p><i>Filled with Eosinophils: A Case of New-Onset Eosinophilic Ascites</i></p> <p>Introduction: Eosinophilic ascites is an uncommon cause of ascites that usually presents as a manifestation of gastrointestinal or peritoneal infection. The differential of eosinophilic ascites includes chronic pancreatitis, trans-serosal Crohn's disease, abdominal lymphoma, vasculitides, abdominal tuberculosis, various parasitic infections, hypereosinophilic syndromes, and eosinophilic gastroenteritis. Herein we present a case of new-onset eosinophilic ascites secondary to longstanding occult gastrointestinal toxocarasis.</p> <p>Case Description: A 33 year-old Vietnamese female (G2P2) with a past medical history significant for diet-controlled gestational diabetes presented with abdominal distension, abdominal pain, diarrhea, nausea and headaches. On presentation, her vitals were all within normal limits and exam was notable for moderate distress with diffusely distended and tender abdomen. Her admission labs were notable for a WBC of 27.4k (11.6k/uL eosinophils). Her CMP, lipase, electrolytes, lactic acid, C.difficile/stool pathogen PCR, hepatitis serologies, C4, C1-esterase, T-spot, gonococcal/chlamydial swab and a urinalysis were all within normal limits. A CT</p>

	<p>abdomen/pelvis identified marked bowel wall thickening from the distal esophagus to the jejunum and large volume-low attenuation ascites. IV ceftriaxone and IV methylprednisolone were administered and the patient was admitted. Paracentesis was completed and fluid demonstrated 6.1k nucleated cells (93% eosinophils) and 15k RBCs. Biopsies obtained from upper endoscopy illustrated dense intraepithelial eosinophilic infiltrate in the esophagus, antrum, and small bowel. A diagnosis of eosinophilic gastroenteritis was entertained and further steroid therapy was proposed. Final laboratory work up found an elevated IgE level (244) and a positive toxocara antibody. The patient was initiated on ivermectin and albendazole therapy for presumed <i>Strongyloides stercoralis</i> vs <i>Toxocara canis</i> infections. Peripheral eosinophilia, ascites, and all gastrointestinal symptoms resolved over the following four weeks.</p> <p>Discussion: The differential for new-onset eosinophilic ascites is broad and contains disparate pathologies. Our case holds true to many paradigms associated with parasitic/eosinophilic ascites. For instance, many cases resemble serosal eosinophilic gastroenteritis radiographically, endoscopically, and clinically. Physicians must use serologies, risk factors, and a degree of suspicion to diagnosis toxocariasis. This distinction is especially important as treating an occult parasitic infection with corticosteroids can have serious implications. Interestingly, our patient had not been to Vietnam for over six years and she was unable to identify any other risk factors for transmission since living in the United States. This may represent so-called covert toxocariasis. This syndrome is characterized by abdominal pain, gastrointestinal symptoms, headaches, malaise, significant peripheral eosinophilia and toxocara antibody positivity. Another unique feature of the covert form of this illness is that temporality with exposure is often difficult to establish and these patients almost never manifest visceral larva migrans or ocular toxocariasis. Appreciation for the broad differential of new-onset eosinophilic ascites is important. Even more, implementing the appropriate diagnostic schema to cases such as ours will likely lead to prompt and appropriate treatment.</p>
<p><b>Ryan Mello</b> Dr. James Gitter Dr. Anya Jamrozy</p>	<p><i>A Six-Pack of Paralysis: A Case of a Paralysis in a Previously Healthy 27-Year-Old</i></p> <p>Introduction: Hypokalemic periodic paralysis is a rare disorder with autosomal dominant inheritance. A mutation in skeletal muscle calcium channels is the most common genetic abnormality. Attacks generally begin the first or second decades of life and present as sudden-onset painless weakness/paralysis. Symptoms may be precipitated by heavy exercise, fasting, or a high-carbohydrate meal. If hypokalemic periodic paralysis is suspected, workup should be focused on ruling out secondary causes of hypokalemia and weakness. Herein we present a case that demonstrates the importance of this workup in an atypical presentation of this rare condition.</p> <p>Case Description: A previously healthy 27-year-old male presented to the emergency room with severe weakness. Symptoms started two days prior to arrival with mild pain of the arms and legs and subjective weakness. No recent changes in physical activity or oral intake. The morning of admission, patient was unable to get out of bed. On admission, exam was notable for profound extremity weakness. Initial workup demonstrated K 1.7, Mg 2.1, Phos 1.0, Cr 0.95, CK 506, TSH 0.41, and QTc 622. Muscle weakness improved with potassium replacement. Subsequent urine studies did not demonstrate excess renal potassium loss and patient had no history of extra renal losses. Serum renin and aldosterone levels were within normal limits. CNS studies were unremarkable. Upon further questioning, patient reported drinking a six-pack of soda the day prior to admission (equivalent carbohydrate load to 8 pieces of a large pizza).</p> <p>Discussion: Despite an atypical presentation of hypokalemic periodic paralysis (first episode in third decade of life, no family history, and the presence of pain), the workup did not reveal an alternative diagnosis. There were no alternative causes of hypokalemia identified, such as diarrhea, thyrotoxicosis, diuretic use, renal tubular</p>

	<p>acidosis, hypomagnesemia, or increased mineralocorticoid activity. Similarly, no other etiologies of weakness, such as a myositis (only mild elevation in CK and no myoglobinuria) or myasthenia gravis. QT was prolonged, but there were no other features of Anderson Syndrome, such as short stature, hypertelorism, clinodactyly, micrognathia, or ventricular dysrhythmias. This case demonstrates the importance of having a high suspicion for hyperkalemic periodic paralysis in the setting of hypokalemia and weakness, even if some aspects of the presentation are not typical for the diagnosis. Also, the systematic approach for evaluating alternative diagnoses enabled prompt treatment and education regarding preventative measures.</p>
<p><b>William Minter</b> Dr. Dr. Rafid Mustafa Dr. Michel Toledano</p>	<p><i>Tick-Tock! Time for a New Pathogen in Minnesota</i></p> <p>Case Presentation: A 28 year-old female with hypothyroidism presented to the Emergency Department with fevers (Tmax 40C), rigors, diaphoresis, photophobia, headache, vomiting, and neck stiffness over a 24 hour period. She was otherwise healthy, working as a Neonatal ICU nurse. She had travelled to northern Minnesota two weeks prior to presentation for lake activities, but denied entering forested areas or insect bites. On presentation, the patient was febrile, tachycardic to the 130s, tachypneic, but normotensive. Physical examination was remarkable only for diaphoresis and rigors. Laboratory studies were notable for thrombocytopenia. CSF was clear with 2 nucleated cells, total protein 41 mg/dL, and glucose 48 mg/dL. Various serum and CSF tests for viral and tick-borne diseases were negative. She was discharged on hospital day 2 following clinical improvement after receiving doses of ceftriaxone, doxycycline, and acyclovir. After discharge, serum PCR returned positive for <i>Borrelia mayonii</i>. She was prescribed doxycycline. Her platelet count normalized within one week. She remained asymptomatic and had attended a wedding.</p> <p>Our patient presented with headache, neck pain, vomiting, and high fever. Serum PCR was positive for a recently recognized tick-borne pathogen, <i>Borrelia mayonii</i>. Although the patient had some clinical features suggestive of meningitis, CSF was not supportive, and diagnostic criteria for neuroborreliosis were not met. <i>B. mayonii</i> was first documented in 2016 and is endemic to Minnesota and Wisconsin. The first publication, which contained only 6 patients, suggested that the clinical presentation of this new pathogen may be different than the typical <i>B. burgdorferi</i> infection and is associated with high fever, nausea, and vomiting—symptoms not usually reported with Lyme borreliosis. When present, a diffuse macular rash, not typical of erythema migrans, was reported. Due to the known clinical heterogeneity of Lyme borreliosis and the low number of patients confirmed to have been infected with <i>B. mayonii</i>, more robust datasets are required to further support these observations. Nonetheless, our patient’s high fever, as well as prominent nausea and vomiting, were consistent with the previously reported cases and suggests that <i>B. mayonii</i> should be considered in the differential diagnosis of patients presenting with these clinical features and supportive exposure history.</p> <p>Discussion: Currently available information suggests that patients with <i>B. mayonii</i> infection develop a serologic response similar to that of patients infected with <i>B. burgdorferi</i>. However, tests for Lyme disease in the acute setting are flawed due to the poor sensitivity of the traditional two-tier testing and CSF antibody index. Authors from the publication note higher levels of <i>B. mayonii</i> are detectable in the blood compared to typical levels of <i>B. burgdorferi</i>, which may allow increased sensitivity of serum PCR testing. Here, we review testing modalities for confirmation of borreliosis and additional considerations with the emergence of the Mid-West pathogen <i>B. mayonii</i>.</p>
<p><b>Leah Mische</b> Dr. Antonious Hazim Dr. Sae Jang Dr. Mark Enzler</p>	<p><i>Solid Hepatic Abscess Masking as a Neoplasm: Imaging and Tissue Culture as Aids to the Diagnosis</i></p> <p>Introduction: The differential for a solid, hepatic mass is broad. We describe a case of a solid liver abscess, ultimately characterized by MRI as an organizing, septated abscess with many small, cystic collections. Blood cultures suggest <i>Streptococcus</i></p>

	<p>intermedius as the pathogen.</p> <p>Case Description: A 59-year-old woman with a history of steatohepatitis and cholecystectomy was brought into the ED for erratic driving and altered mental status. She reported four days of arthralgias, myalgias, and shortness of breath with two days of subjective fevers, severe diarrhea and rare emesis. Infection exposure included recent consumption of raw fish, months of intermittent exposure to freshwater lakes, and a brief trip to the Dominican Republic six months prior.</p> <p>On arrival to the emergency department, she was afebrile, heart rate was 141 bpm and respiratory rate was 39. Laboratory evaluation was notable for: ALT 348, AST 426 and lactate 4.6. CT abdomen with contrast demonstrated a 9cm heterogeneous hepatic mass. She was given three liters of normal saline, started on vancomycin/piperacillin-tazobactam and was admitted to the MICU for presumed sepsis. She required one dose of phenylephrine 0.2mg for pressure support and was transferred to the general medical floor. Abdominal ultrasound demonstrated a solid mass without internal cystic components. For this reason, a neoplastic process was strongly suspected. CEA, AFP and CA19-9 were obtained and were negative. Subsequent biopsy of the mass was strongly suggestive of an intrahepatic abscess. Blood cultures grew pan-sensitive <i>Streptococcus intermedius</i> in two draws after 10 hours. Other infectious work-up, including tick-borne panel, fecal parasite analysis, and TEE for endocarditis were negative. Despite continuation of broad-spectrum antibiotics with vancomycin, cefepime, and metronidazole, and clearance of blood cultures, she developed intermittent fevers up to 39.4 degrees Celsius and remained septic.</p> <p>In evaluating for intervention feasibility, abdominal MRI demonstrated growth of the liver process to 13cm with the presence of many small, connected fluid-filled pockets. Due to the involvement of several major hepatic vessels, surgical resection was deferred. She underwent attempted percutaneous drainage with an output of 13cc of fluid and analysis demonstrated many gram positive cocci on Gram stain.</p> <p>Most recently, her vital signs have not significantly changed despite continuation of broad-spectrum antibiotics. Current plans include periodic IR drainage with possible complex endoscopic intervention.</p> <p>Discussion: <i>Streptococcus intermedius</i> is the most pathogenic species of the <i>Streptococcus anginosus</i> bacterial group. It has a proclivity for solid organ abscess formation, including the brain, liver, and lung. Although liver abscesses typically present as fluid-filled structures, they may appear solid early in the process. MRI can be a useful tool in differentiating malignant and infectious etiologies of an ultrasound-characterized solid liver mass. When biopsying these lesions, tissue culture evaluation should be considered.</p>
<p><b>Nathan Nesbitt</b>  Dr. Qian Ye  Dr. Vuyisile Nkomo</p>	<p><i>When Acute Stroke Crosses Hemispheres, Follow Your Heart</i></p> <p>Introduction: Mitral annular calcification (MAC) is a degenerative process of the mitral valve that has been shown to have a strong association with cardiovascular risk factors, as well as an increased risk of all cause death. However, findings of MAC related to risk of clinical stroke have been inconsistent, and patients with MAC who have strokes often have concomitant atherosclerotic disease that can better explain the event. However, we present a case in which acute stroke can be confidently linked to MAC. This case illustrates one mechanism by which MAC can precipitate embolic stroke.</p> <p>Case Presentation: A 74 year-old gentleman with medical comorbidities including type 2 diabetes, hypertension, hyperlipidemia, smoking, who presented with two days of right arm numbness, tingling, and discoordination. Upon arrival to the emergency department, the patient had normal vitals and a laboratory evaluation only significant for a mild leukocytosis. CT head without contrast was unremarkable, and CT head and neck angiogram with IV contrast showed plaque build up in bilateral internal</p>

	<p>carotid arteries. However, brain MRI revealed small multifocal acute infarcts to the right cerebellum, right posterior temporal lobe, left posterior lentiform nucleus, and left posterior frontal periventricular white matter. Further evaluation with a trans-esophageal echocardiography revealed a moderately calcified mitral annulus, and a highly mobile globular mass measuring 1.4 by 1.3cm below the mitral valve. Neurology was consulted and they stated that anticoagulation was acceptable if necessary. The primary team balanced the risk of bleeding or hemorrhagic stroke conversion with repeat embolism and decided to forgo anticoagulation except for aspirin prior to surgery. Two peripheral blood cultures were negative, and a PET CT scan revealed no FDG avid infection. The patient was then transferred to the cardiology service for cardiovascular surgery evaluation.</p> <p>After an unremarkable coronary angiogram, the patient underwent open heart surgery in which a mass with two separate segments arising from the posteromedial commissure of the mitral valve was resected in its entirety. Pathology revealed a mass-like calcification with adherent thrombus most consistent with extruded material from mitral annular calcification. The patient recovered well, although he maintained an altered sensorium and dysmetria in his right upper extremity. He was discharged to rehabilitation within a week of surgery.</p> <p>Conclusion: While MAC has been associated with increased risk of cardiovascular death in the past, there have been few direct links between MAC and embolic stroke. The case presented here, involving acute, multi-focal, bi-hemispheric stroke, is almost certainly due to embolic origin, with intra cardiac mass as the most likely source. Based on pathological findings, this mass appears to be directly generated from MAC, and represents a clear and simple mechanism by which MAC could generate multifocal embolic stroke.</p>
<p><b>Michelle Nguyen</b></p>	<p><i>Chronic Migraine Associated with Brain Arteriovenous Malformation</i></p> <p>Introduction: Brain arteriovenous malformations (bAVM) are rare cerebrovascular lesions, in which there are abnormal direct connections between cerebral arteries and veins. bAVMs can present as spontaneous intracranial hemorrhage (ICH), seizures or headaches. Patients may additionally have focal neurologic deficits independent of presence of ICH. We report a case of 59 year old man who presented with chronic migraines and syncope in setting of undiagnosed bAVM.</p> <p>Case Presentation: A 59 year old man with history of hypertension and chronic migraines was transferred to the Hennepin County Emergency Department for syncope with possible intracranial hemorrhage vs mass seen at an outside hospital CT Head non-contrast. In the ED, the patient was vitally stable, alert and oriented with no neurologic deficits. He had a very minimal headache without external signs of trauma except for small cut above lateral right eyebrow. The repeat CT head with angiogram demonstrated a large left occipital dural Grade 3 malformation with no signs of active bleeding. The patient had been coming to the ED for the past several years for left sided throbbing headaches with photophobia with increasing frequency, no pathology noted on CT head in the past. The patient then underwent urgent curative endovascular embolization with neuro-interventional radiology.</p> <p>Discussion: Brain arteriovenous malformations have low incidence, occurring in about 0.1 percent of the population [1]. The most concerning risk in patients with bAVMs is intracranial hemorrhage, which can be life threatening. The clinical presentation of AVMs includes intracranial hemorrhage (40-60 percent), seizure (10-30 percent), incidental finding on imaging (10-20 percent), focal neurologic deficit and headache [2]. In one study, 0.2 percent of patients presenting with headache in setting of normal neurologic exam were found to have bAVM [3]. In this case, the patient had presented several times to the emergency department and primary care clinic for ongoing non-intractable unilateral migraine with photophobia, which improved with olanzapine and sumatriptan. Though it is possible that the patient had both bAVM and chronic migraines, it is difficult to differentiate between a primary</p>

	<p>migraine and secondary headache to the bAVM. In one case report, the patient with both headache and bAVM had symptomatic improvement after treatment [4]. Diagnosis is made by CT or MR angiography [6]. Treatment is stratified by the Spetzler–Martin grading scale, created to predict outcomes of microsurgical and radiosurgical treatment [6]. Though incidence is rare, it is important to look for other causes of chronic, unilateral headaches , such as bAVM as the consequences can be severe.</p> <p>References</p>
<p><b>Edith Nyangoma</b> Dr. Joel Fuchs Dr. Howard Fink</p>	<p><i>"What A pain in the Neck!"</i></p> <p>Case Presentation: An 84-year old male with a history of peripheral vascular disease presented with two weeks of worsening neck pain. The pain was intermittent, achy, severe, located diffusely over the posterior neck, and associated with fatigue and confusion. He denied recent fevers, photophobia, headache, cough, rashes, or arthralgia, and reported no recent trauma, sick contacts or recent travel. On examination, he was in no distress although oriented only to name.</p> <p>Exam showed decreased neck range of motion laterally, and to anterior flexion and rotation, as well as slight tenderness with palpation over the cervical and lumbar paraspinal muscles. Labs revealed 14,500 white blood cells/<math>\mu</math>L, C-reactive protein of 190mg/L, and an erythrocyte sedimentation rate of 80 mm/hr. Computed tomography (CT) of the head and cervical spine with angiography showed no acute intracranial process or significant vascular stenosis. Following a negative infectious disease work up and no clinical improvement in neck pain after 48 hours, rheumatology was consulted who retrospectively found significant crown-shaped calcium deposition surrounding the odontoid process on CT cervical spine imaging consistent with Crowned Dens Syndrome. The patient was started on 40mg of prednisone daily and symptoms rapidly improved. The patient was discharged home with a prednisone taper.</p> <p>Discussion: Crowned Dens Syndrome (CDS) is a rare manifestation of pseudogout or calcium pyrophosphate deposition affecting the articular structures of the atlantoaxial junction surrounding the odontoid process, or dens process. This syndrome often presents with neck pain, rigidity and sequelae of severe inflammation such as fever and fatigue and is diagnosed by clinical and radiographic evidence. CDS creates a diagnostic dilemma as the clinician is faced with a large differential diagnosis for neck pain; proper diagnosis and management is key in the face of more dangerous processes such as meningitis. Pseudogout more commonly presents as oligoarticular disease with acute pain, swelling, and tenderness of large joints such as knee, wrist, or shoulder affecting older adults. Our patient presented as is typical of CDS with neck pain, rigidity, fatigue, and elevated inflammatory markers. Notably, he was afebrile, though he had altered mental status, which could be attributed to acute inflammation in the setting of older age and mild cognitive impairment. The patient underwent infectious disease workup and several radiographic studies. The CT cervical spine imaging retrospectively revealed the diagnosis. Clinicians should be aware of CDS as a cause of neck pain, particularly in elderly patients. While proper workup to rule out an infectious etiology is important, a diagnosis of CDS can be made on history and CT cervical spine imaging alone, avoiding the need for invasive procedures or expensive repeat imaging.</p>
<p><b>Pat O'Connor</b></p>	<p><i>Knowing is Half the Battle</i></p> <p>Case Presentation: A 61 year old man presented to the emergency department by ambulance for worsening dyspnea. He said that over the preceding month he gradually developed worsening dyspnea, intermittent chest pain, and cough that was becoming more frequent and was newly productive. He had a history of chronic kidney disease from focal segmental glomerulosclerosis and was on high dose steroids daily as well as prophylactic trimethoprim/sulfamethoxazole (TMP/SMX) for the prior four months. The patient was hypoxic and started on oxygen. He was found</p>



	<p>to have a dense right upper lobe opacity on chest x-ray. Broad spectrum antibiotics were given for suspected pneumonia. A few hours after admission he developed respiratory distress, was started on BiPAP to maintain his oxygen saturations, and was transferred to the medical intensive care unit. A sputum sample was collected and the gram stain showed many beaded branching filamentous gram positive bacilli; acid fast staining was performed and the organism weakly stained acid fast. The infectious diseases team was consulted and started the patient on intravenous TMP/SMX for suspected pulmonary Nocardiosis.</p> <p>Even though the patient did not have any neurologic symptoms an MRI was ordered which was negative. 16S PCR of the sputum was added on and eventually identified the organism as Nocardia nova. The sputum culture was also sent for antibiotic susceptibility testing to a specialized outside laboratory. After a few days without clinical improvement, intravenous linezolid was added. Over the following days the patient began to recover and the linezolid was switched to imipenem. Two weeks later the patient was breathing comfortably, off of oxygen, and was discharged on imipenem and TMP/SMX. Outpatient infectious disease will follow and decide duration of the imipenem, and at this point the TMP/SMX has a tentatively planned duration of one year. His chronic prednisone is currently being tapered off as during his hospitalization his kidney disease progressed to end stage renal disease and he was started on hemodialysis.</p> <p>Discussion: Nocardiosis is a rare disease with worldwide distribution and the general internist needs to know when to suspect it as delay in diagnosis or premature discontinuation of therapy is associated with poorer outcomes. Nocardia most commonly infects the lungs but can disseminate to any organ and especially the central nervous system and for this reason most patients should have brain imaging. Two thirds of patients with Nocardia are immunocompromised and infections can occur even when patients are taking TMP/SMX prophylactically as was the case for this patient. Molecular and antibiotic susceptibility testing is indicated to guide long term treatment, and Nocardiosis has a tendency to relapse or progress despite appropriate antibiotics.</p>
<p><b>Timothy O'Dowd</b> Dr. Mitchell Padkins Dr. Melitady Issa</p>	<p><i>Non-Traumatic, Malignancy-Associated Hemoperitoneum</i></p> <p>Case Presentation: A 74 year-old man, with cirrhosis secondary to a recently diagnosed liver metastasis from an unknown primary cancer, was admitted to the hospital with progressively worsening shortness of breath and abdominal fullness. Physical exam revealed significant abdominal distention with shifting dullness and no rebound tenderness or rigidity. Laboratory evaluation showed hemoglobin of 7.4 g/dL, down from 10.3 g/dL two weeks prior. Computed Tomography (CT) scan of the abdomen and pelvis with contrast demonstrated the known hepatic masses in the left lobe with extracapsular extension. Ascites was also present but no obvious blood products or active extravasation was detected (Figure 1A). We performed an ultrasound-guided diagnostic and therapeutic paracentesis which yielded 2.9 liters of grossly bloody peritoneal fluid (Figure 1B). Given these findings, we consulted Interventional Radiology, and they performed a left hepatic artery embolization with excellent angiographic results. The patient felt slightly better, but given the patient's overall poor prognosis, he elected to focus on his quality of life and was subsequently discharged home with hospice care.</p> <p>Discussion: Non-traumatic hemoperitoneum poses a diagnostic challenge; it is a rare entity with a paucity of literature. If this diagnosis is missed however, it can be life-threatening. The most common cause is rupture of a hypervascular neoplasm. This includes both primary malignancies, such as hepatocellular carcinoma, and metastatic malignancies, such as lung carcinoma, renal cell carcinoma, and melanoma. Many factors are thought to contribute to bleeding from these tumors such as rapid growth with necrosis, erosion of vasculature, and increased intra-abdominal pressure. Peripherally-located tumors are more likely to bleed into the peritoneum as they have no normal surrounding hepatic parenchyma.</p>

	<p>CT plays an integral role in the diagnosis of many acute abdominal pathologies. It can usually detect hemoperitoneum given the higher attenuation of blood compared to other body fluids. Multiple variables however can lower that attenuation such as low peritoneal fluid hematocrit levels and hemorrhage older than 48 hours. In these cases, CT might be negative, and paracentesis might be the first clue as demonstrated in this case. Management includes resuscitation of hypovolemic shock, achieving hemostasis, and treatment of the underlying tumor.</p> <p>In summary, non-traumatic hemoperitoneum is a fatal condition with high morbidity and mortality. Prompt diagnosis and management is crucial, and a negative CT does not completely rule it out. Paracentesis can help when there is clinical uncertainty such as in this case.</p>
<p><b>Fredrick Ogugua</b></p>	<p><i>The Complex Patient: A Complicated Case of SBO</i></p> <p>Introduction: The ‘complex patient’ is a word that has been used widely among multidisciplinary healthcare teams. But what does it really mean and what are its practice implications?</p> <p>Case Presentation: A 65-year-old male with past medical history of ESRD, right-sided weakness and expressive aphasia due to CVA, hypertension was brought to ED by family with complaint of 1-day history of altered mental status. On physical exam, patient was hypothermic, hypotensive and bradycardic. Abdomen was distended and tender to palpation. Bedside abdominal ultrasound showed apparent diffuse shock bowel. Labs were significant for hyponatremia, lactic acidosis, leukocytosis with neutrophilic dominance. CT-abdomen and pelvis showed internal small bowel hernia with closed-loop configuration. Extensive mesenteric edema, fat stranding, and ascites in the associated bowel concerning for bowel ischemia. He was resuscitated, received broad antibiotic coverage for septic shock picture and General surgery was consulted. General surgery attempted conservative management, but patient’s bowel obstruction didn’t improve. Necessitating abdominal exploration which revealed several adhesions, on further exploration, it was determined that a porcelain gallbladder was the sentinel lesion causing an internal hernia and multiple adhesions. Patient underwent adhesiolysis and hernia detorsion and was placed in temporary closure, eventually closed 2-days later. Patients dialysis line clotted, attempt to declot was unsuccessful necessitating femoral permacath placement by the Vascular team. Upon extubated patient had non-reassuring neuro exam. CT-head showed new cerebellar infarcts most likely embolic from incidental 10mm aortic valve mass found on TTE concerning for infective versus non-infective endocarditis. He subsequently developed NSTEMI suspected to be cardioembolic in origin and was started on NSTEMI protocol. Due to cerebral infarction, aortic valve mass and risk of anticoagulation; coronary angiography was contraindicated. In this patient with poor baseline functioning, extremely complex comorbidities with marked worsening in clinical status, Palliative care was consulted; after which the family elected to discharge home on hospice with comfort cares.</p> <p>While hospitalized the patient was cared for by the critical care, surgery, vascular, interventional, anesthesia, nephrology, neurology, cardiology, podiatry, infectious-disease, palliative, nutrition, social-work, and nursing staff.</p> <p>Conclusion: In defining the ‘complex patient’, healthcare professionals usually consider words like comorbidities, poly-pathology, dual-diagnosis, disability. To accurately define the complex patient, the scope must be widened to encompass issues beyond the patient’s pathophysiology like internal and external social determinants of health. An accurate definition is vital to advancing diagnosis, treatment and delivering optimum patient care. To define a complex patient; he/she is one with multiple comorbidities and disability, living with unstable internal and external social circumstances worsened by the limitations of his/her immediate clinical context and</p>

	<p>their physicians' characteristics and ability. In dealing with complicated patients it is important to have a multidisciplinary healthcare team working in tandem.</p>
<p><b>Emily Olson</b> Dr. Michael Wilson</p>	<p><i>When the Tick Bites: Shock in the setting of Human Monocytic Ehrlichiosis</i></p> <p>Background: Tick borne diseases should be considered in patients presenting with toxic shock. Common laboratory abnormalities for human monocytic ehrlichiosis (HME) and human granulocytic anaplasmosis (GHA) include thrombocytopenia, leukopenia and elevated liver transaminases.</p> <p>Case Presentation: A 74 year old man presented to the emergency room with new visual hallucinations after 10 days of general malaise, increased disorientation, neck stiffness, lower extremity weakness, diminished appetite with nausea, vomiting, and jaundice. Past medical history was significant for a clonal gammopathy of renal significance now with end stage renal disease on dialysis, atrial flutter on warfarin, ischemic cardiomyopathy, and anemia of chronic disease.</p> <p>On exam, he was afebrile, had a blood pressure of 97/62, heart rate of 77, was oriented to name and place only, and did not have active hallucinations. A petechial rash covered his right calf. Labs were significant for platelets 124, AST 363, ALT 142, direct bilirubin 3.2, albumin 2.0, alkaline phosphatase 304, INR 6.0, NT-pro BNP 52014, lactate 2.7, and high-sensitivity troponin 193. Recheck of the troponin was 2 hours later was unchanged. Urinalysis had moderate bilirubin, 11-20 red blood cells without dysmorphism, and no white cells or bacteria. He quickly required IV vasoactive medications to maintain blood pressures. Thus, he was initiated on vancomycin and piperacillin/tazobactam for septic shock in addition to the plan for dialysis for treating decompensated heart failure causing a potential congestive hepatitis. A broad laboratory work-up for liver failure was initiated. Throughout the following day he had increasing vasopressor requirements.</p> <p>The tick borne panel resulted 24 hours after admission identifying acute Ehrlichia Chaffeensis and Anaplasma phagocytophilum (IgG titers both 1:1024). Repeated blood smears did not identify morulae. Further discussion with his wife revealed there was a tick in their bedroom approximately 3 weeks prior. He was initiated on doxycycline with significant improvement in his hemodynamic and mental status. No lumbar puncture occurred to assess for central nervous system (CNS) disease since it would not have altered therapy. With concern for possible underlying Ehrlichia induced myocarditis, he underwent transthoracic echocardiography which revealed unchanged ejection fraction from three months prior.</p> <p>Discussion: Immunosuppressed individuals, including those with gammopathies, are at increased risk for tick-borne illnesses. While oftentimes HGA and HME infections can be subclinical in immunocompetent patients, it is frequently fatal in those who are immunocompromised, oftentimes resulting in multi-organ failure resembling toxic or septic shock. Ehrlichiosis CNS infection may explain this patient's new hallucinations; if undertaken, a lumbar puncture would most likely have a lymphocytic predominance. Comparatively, HGA rarely involves the CNS. While co-infection with both Ehrlichia and Anaplasma is possible, there are also reports of cross-reactivity on serology. High clinical suspicion and early treatment influences morbidity and mortality associated with these emerging tick-borne illnesses.</p>
<p><b>Luke O'Neil</b></p>	<p><i>Disseminated Nocardiosis Presenting as Metastatic Lung Cancer; the Answer is in the Barium Swallow</i></p> <p>Case Presentation: This is a 61 year old man with history of essential hypertension and chronic migraines who presents to the emergency department with one month ataxia, diplopia and sixty pounds of weight loss. While in the emergency department, CT and subsequent MRI of the brain illustrated mixed solid and cystic lesions in the brain, brainstem and cerebellum concerning for malignancy or infection.</p>

	<p>Of note, this ED presentation follows recent outpatient workup for chronic cough that revealed a new hilar lung mass on chest CT. Initial bronchoscopy completed in the outpatient setting was negative for malignancy yielding findings consistent with chronic inflammation and no obvious organisms. Repeat endobronchial ultrasound (EBUS) and fine needle aspiration (FNA) during this admission had rapid pathology interpretation concerning for malignancy, which was especially convincing given the patient's sixty pack-year history of smoking. Given active neurologic symptoms and the brain imaging findings, empiric steroids and whole brain radiation were completed. On hospital day three, a barium swallow study to workup dysphagia illustrated paravertebral soft tissue thickening that was further characterized as rim-enhancing retropharyngeal fluid collections on CT. These fluid collections had gram stain showing beaded and branching gram positive bacilli that speciated to Nocardia. ID consultation was obtained and antibiotic therapy with bactrim, meropenem and linezolid was initiated. Following treatment with antibiotics, the patient had radiographic improvement in the lung, neck and brain lesions, all of which were presumed to be dissemination of Nocardia.</p> <p>Discussion: Disseminated nocardiosis is more likely to occur in those who are immunosuppressed with some studies citing &gt;60% of individuals having cancer, HIV, chronic steroid use or malignancy. This patient had no clear immunodeficiencies. Regardless of the host's immune status, Nocardia can affect nearly any organ with brain involvement being common. Unfortunately for this patient the initial rapid pathology from EBUS/FNA of the hilar mass illustrated findings concerning for malignancy and whole brain radiation therapy was employed. Given the morbidities associated with brain metastasis, whole brain radiation therapy given his neurologic symptoms and preliminary pathology was appropriate at the time. It was only until a more accessible target for tissue acquisition was incidentally discovered - fluid collections in paravertebral tissues by barium swallow study - did FNA yield a causative organism and allow for appropriate therapy. In this example of an elderly man with extensive tobacco use and significant weight loss, nocardia mimics lung cancer in that both can manifest in the lung and brain and cause B-symptoms. This case further highlights the importance of the tissue sampling for proper diagnosis of nocardiosis.</p>
<p><b>Mitchel Padkins</b> Dr. Meltiady Issa</p>	<p><i>Troponin-Positive Chest Pain with Non-Obstructive Coronary Arteries</i></p> <p>Case Presentation: A 29 year old man presented to the Emergency Department (ED) with crushing sub-sternal chest pain that radiated to his jaw and was associated with diaphoresis, dyspnea, nausea, and vomiting. He had a history of hypertension, hypertriglyceridemia, and tobacco use. In the ED, the patient was hemodynamically stable. EKG was unremarkable. Labs were notable for initial high-sensitivity troponin of 1950 ng/L (reference: &lt;15 ng/L), 2-hour troponin 2165 ng/L, and 6-hour troponin 2413 ng/L. NT-pro-BNP was elevated at 1692 pg/mL (reference: &lt;51 pg/mL). C-reactive protein was 50.9 mg/L (reference: &lt;8 mg/L). His presentation was initially concerning for acute coronary syndrome (ACS), so he was started on aspirin, heparin, and clopidogrel. Transthoracic echocardiogram (TTE) demonstrated an ejection fraction (EF) of 30% with severe generalized left ventricular hypokinesis. Cardiology was consulted and recommended cardiac catheterization which revealed normal coronary arteries without obstructive lesions. Given these findings, cardiac magnetic resonance (CMR) imaging was performed and findings were consistent with acute myocarditis. With aggressive diuresis and preload reduction, the patient's chest pain improved and he was started on anti-inflammatory therapy. TTE was repeated after one month of medical therapy, and his left ventricular function improved with an EF of 63%.</p> <p>Myocarditis is a common myocardial inflammatory process that can mimic ACS. The etiology is usually viral in origin; however, bacterial pathogens have been identified along with a host of medications such as antipsychotics. Clinical presentation may range from mild symptoms to severe heart failure. The diagnostic gold standard is an endomyocardial biopsy, but the advent of CMR has helped clarify this diagnosis</p>

	<p>without pursuing invasive measures. CMR enables detection of various features of myocarditis, including inflammatory hyperemia, edema, and myocyte necrosis, which makes it useful to support the diagnosis and monitor disease progression.</p> <p>Discussion: In summary, the evaluation of patients with ACS and non-obstructing coronary arteries should be complemented by CMR if available. Establishing a correct diagnosis is important for such patients, both from a prognostic and therapeutic point of view.</p>
<p><b>Jessica Padniewski</b> Dr. Lin Ngo</p>	<p><i>Persistent Asthma; A Case of Eosinophilic Granulomatosis with Polyangiitis</i></p> <p>Introduction: Eosinophilic granulomatosis with polyangiitis (eGPA) is an extremely rare anti-neutrophilic cytoplasmic antibody (ANCA) associated vasculitis with an incidence in the U.S. from 1-3 cases per 100,0001. eGPA is a vasculitis of small and medium sized arteries with vasculitic symptoms often not initially apparent. In fact, respiratory involvement of eGPA commonly causes chronic rhinosinusitis, asthma, and prominent eosinophilia. More than 90% of patients present with asthma requiring repeated corticosteroids bursts2,3. Many patients do not develop the classic pulmonary or renal hemorrhage, palpable purpura or mononeuritis multiplex. We present a case of a patient diagnosed with steroid dependent, uncontrolled asthma, COPD, vocal cord paralysis, and upper airway inflammation requiring tracheostomy who was ultimately found to have eGPA.</p> <p>Case Presentation: A 56 y.o. African-American woman with multiple admissions for COPD and asthma exacerbations since 2013 presented with shortness of breath. She was maintained on systemic prednisone for respiratory symptoms since 2015. Multiple tapering attempts resulted in flares often requiring intubation and use of high dose intravenous corticosteroids. Chronic corticosteroid use caused Cushing Syndrome and multiple soft tissue infections. Chronic airway inflammation and recurrent intubations ultimately required tracheostomy tube placement. Multiple laryngoscopic evaluations revealed severe vocal cord inflammation with stenosis, upper airway inflammation and septal perforation with no improvement over time. She was noted to have a peak absolute eosinophil count of 9.8% coinciding with the onset of COPD and asthma. ANCA and myeloperoxidase(MPO)-antibodies were significantly elevated. Diagnosis of ANCA-positive eGPA was suspected and rituximab was started. One month after 2 rituximab infusions, repeat laryngoscopy showed full resolution of visual inflammation. Prednisone was tapered successfully without relapse. Tracheostomy tube removal is being discussed. Laboratory studies revealed improvement of eosinophilia, MPO-antibody levels and inflammatory markers.</p> <p>Discussion: eGPA has 3 stages; prodromal phase of atopy, allergic rhinitis and asthma in adults; eosinophilic phase with peripheral eosinophilia and infiltration of organs; and vasculitic phase with development of small-medium vessel vasculitis which carries a poor clinical prognosis4. Respiratory symptoms commonly precede vasculitic complications by 8-10 years5,6. Only 30-60% patients have ANCA-antibodies6. Of those that are ANCA-antibody positive, 70-75% of patients have MPO-antibodies7. Our patient possessed both ANCA and MPO-antibodies, significant eosinophilia and steroid dependant respiratory inflammation. Delayed diagnosis can lead to undesirable side effects of long term corticosteroid exposure such as increased risk of infection, Cushing Syndrome, osteoporosis, and type II diabetes. In conclusion, eGPA should be suspected in patients with adult onset asthma requiring chronic corticosteroid therapy. Earlier diagnosis could lead to the prevention of more severe manifestations of vasculitis and avoidance of long term corticosteroid use. Optimal care for these complex patients requires a multidisciplinary team approach and use of steroid sparing immunomodulators.</p>
<p><b>Paul Park</b></p>	<p><i>When it is Not All About the Liver: A Case of Idiopathic Hyperammonemia</i></p>

	<p>Introduction: Hyperammonemia is a metabolic condition characterized by elevated levels of ammonia in the blood, leading to various symptoms including confusion and, in severe case, coma. Even though the most common causes of hyperammonemia in adults are liver cirrhosis and medication side effect, clinicians should always consider other etiologies (late-onset inborn error of metabolism, infection with urea producing organism, idiopathic, etc.), which causes a diagnostic and therapeutic challenge.</p> <p>Case Presentation: A 60-year-old woman with history of type 2 diabetes, chronic kidney disease, and chronic normocytic anemia was transferred from outside hospital (OSH) with ongoing acute metabolic encephalopathy secondary to hyperammonemia of unknown etiology. She was admitted to OSH twice prior to transfer with ammonia levels in the 600s, requiring continuous renal replacement therapy (CRRT) and close monitoring. Upon transfer, physical exam was remarkable for orientation only to self and absence of asterix. Labs were significant for hyperammonemia to 102 and normocytic anemia, but otherwise normal. Genetics was consulted due to concern for late-onset inborn error of metabolism and ultimately recommended amino acid supplements while waiting for additional labs. However, she was transferred to MICU twice during the hospitalization due to worsening ammonia level, encephalopathy, and concern for airway protection. She required a brief run of dialysis but quickly stopped because of no improvement in mentation. She was started on high-protein formula via nasojejun tube with concern for starvation potentially causing catabolic state as well as lactulose to remove ammonia and azithromycin to empirically treat possible mycoplasma/ureaplasma infection. For additional work-ups, ultrasound of the abdomen with doppler showed normal vasculature, which was confirmed through MRI of the abdomen. Head CT was normal and EEG results were consistent with diffuse encephalopathy. Interventional Radiology (IR) guided biopsy did not reveal any evidence of cirrhosis. Additional testing for urea cycle defect was unremarkable, therefore, amino acid supplements were stopped. Her ammonia level slowly improved through a combination of high-protein formula and lactulose, which was optimized over several days of adjustment. With extensive diagnostic tests for hyperammonemia returning negative, she was diagnosed with idiopathic hyperammonemia and eventually discharged to a rehab facility on regular diet and lactulose four times daily.</p> <p>Discussion: This case demonstrates the danger of anchoring on common causes of hyperammonemia and emphasizes clinicians to consider non-cirrhotic causes of hyperammonemia in differentials, such as late-onset inborn error of metabolism and mycoplasma/ureaplasma induced hyperammonemia. It also emphasizes the often frustrating but humble aspect of medicine: physicians do not know all the answers, but are constantly evolving and learning.</p>
<p><b>Abigail Peterson</b> Dr. Christina Fanola</p>	<p><i>A Real Pain in the Eyes: An Uncommon Cause of Syncope</i></p> <p>Introduction: Syncope is a common presentation to emergency departments and many of these patients ultimately require hospitalization. Etiologies for syncope include neurocardiogenic, orthostasis, and medication induced, although there are numerous rare causes. Initial evaluation should include history, orthostatic vitals, physical examination and electrocardiogram. Patient's history may dictate further evaluation for neurological causes including head imaging or cardiac causes including telemetry monitoring and echocardiogram. Routine laboratory evaluation is not supported by evidence.</p> <p>Case Presentation: A 39 year old African American male presents to the emergency room after an episode of syncope. Patient denies significant past medical history. He describes an episode of severe left eye pain, lacrimation, blurred vision, and syncope. The patient's significant other reports that the patient slumped down while driving and was unarousable for approximately 30 seconds before he awoke again. Physical exam is remarkable for a normal fundoscopic exam, cardiac exam without murmur, and non-focal neurological examination. Laboratory evaluation was remarkable for normal TSH, negative lyme serology, C-reactive protein and sedimentation rate were</p>

	<p>normal. While in the emergency department had a recurrent episode of eye pain and subsequent syncope. During this period telemetry was remarkable for six second sinus pause. Throughout hospital stay patient had numerous episodes of eye pain followed by sinus pauses with associated hypotension, received multiple doses of atropine, and ultimately requiring temporary pacer placement. He reported dizziness and near syncope with these episodes which resolved with temporary pacer placement. The patient was evaluated by electrophysiology, ophthalmology and neurology during his hospital stay. Patient was ultimately diagnosed with sinus pauses secondary to cluster headaches. His cluster headaches were treated with prednisone taper and his symptoms resolved. He was offered permanent pacemaker placement and patient declined.</p> <p>Discussion: Cluster headaches are uncommon, and characterized by unilateral pain, typically orbital or temporal. Pain is accompanied by autonomic symptoms including ptosis, miosis, conjunctival injection, rhinorrhea, and lacrimation. Autonomic symptoms are ipsilateral to location of pain and subside as the pain subsides. Syncope in association with cluster headache is exceedingly rare, but case reports have noted its association. This is thought to be related to vagal stimulation and resultant bradycardia due to AV nodal dysfunction. In this case, syncope occurred in the setting of sinus pauses rather than heart block which is typically seen. In these reports syncope has been aborted by management of pain attacks. Typical treatment of cluster headache includes 100 percent oxygen as first line or subcutaneous triptan. Preventative therapy includes verapamil, glucocorticoids, and ergotamine. In our patient oxygen alone was effective in aborting acute attacks. For further prevention verapamil was not preferred given bradycardia, so the patient was discharged on a glucocorticoid taper without recurrence known of syncope or headache.</p> <ul style="list-style-type: none"> <li>- Formulate appropriate plan for initial evaluation of syncope</li> <li>- Understand pathophysiology of vagal mediated syncope</li> <li>- Define treatment options for both acute cluster headache attack and prevention of further attacks</li> </ul>
<p><b>Shrikar Rajagopal</b> Dr. Joe Lau Dr. Lisa Fish</p>	<p><i>Euglycemic Diabetic Ketoacidosis Induced by SGLT-2 Inhibitor Use</i></p> <p>Introduction: Euglycemic diabetic ketoacidosis (DKA) is rare DKA subtype defined by the American Diabetes Association as blood glucose &lt; 250 mg/dL, with metabolic acidosis (arterial pH &lt; 7.3 with serum bicarbonate &lt; 18 mEq/L) and ketosis. Historically associated with decreased caloric intake, heavy alcohol consumption, chronic liver disease, pregnancy, and glycogen storage disorders, the incidence of euglycemic DKA may now be rising with the increased use of sodium-glucose cotransporter-2 (SGLT-2) inhibitors, as recent clinical trials have highlighted the cardiovascular benefits of SGLT-2 inhibitor use in patients with atherosclerotic cardiovascular disease (ASCVD) and diabetes mellitus type 2 (DM2) not at goal with metformin monotherapy. These benefits include reduced major adverse cardiovascular events<sup>1,2</sup>, hospitalizations for heart failure<sup>1,2</sup>, and cardiovascular death<sup>2</sup>.</p> <p>Case Description: A 47-year-old female with a known history of DM2 and cesarean-section with tubal ligation presented to the ED with 1-day history of nausea, vomiting, and diffuse abdominal pain. On presentation, she was well-oriented and endorsed irregular bowel movements with ability to pass flatulence. Also endorsing long-standing polydipsia and polyuria. Vital signs positive for tachycardia, otherwise within normal limits. Physical examination with dry mucous membranes, mild tenderness to palpation in the right lower quadrant without guarding or rebound tenderness. Medications included metformin, empagliflozin, ibuprofen and acetaminophen. Routine point of care blood glucose was 239 mg/dL. The blood work showed the following: WBC 11.02 K/cmm, Na+ 143 mEq/L, K+ 3.8 mEq/L, Cl-103 mEq/L, bicarb 12 mEq/L. CT findings were concerning for partial small bowel obstruction. Patient was kept NPO with gastric suction in preparation for likely surgical intervention, and started on 0.9% saline infusion, anti-emetics, and pain</p>

	<p>control medications. Patient noted to have dyspnea with subsequent monitoring, VBG showed pH of 7.03, PCO<sub>2</sub> of 55 mmHg, and bicarb of 10 mEq/L. Beta-hydroxybutyrate was elevated to 3.43 mmol/L, lactate at 1.5 mmol/L, repeat blood glucose at 218 mg/dL. She was admitted to the hospital and treated for DKA. Subsequent testing showed HbA<sub>1c</sub> of 10.2%. GAD65 and Islet cell antibody testing negative. Metabolic acidosis and bowel function improved on day two of hospitalization. She was discharged in stable condition with all prior to arrival medications with exception of empagliflozin.</p> <p>Discussion: The incidence of euglycemic DKA is likely to increase as the prevalence of SGLT-2 inhibitor use in patients with DM2 and ASCVD rises. Despite euglycemia, ketoacidosis remains a medical emergency and necessitates urgent treatment following standard DKA protocols. Contraindications to future SGLT-2 inhibitor use in affected patients remains unknown as the mechanism leading to euglycemic DKA remains unclear.</p> <p>References:</p> <ol style="list-style-type: none"> <li>1. Canagliflozin and Cardiovascular and Renal Events in Type 2 Diabetes. <i>New England Journal of Medicine</i> 2017;377(21):2097–9.</li> <li>2. Empagliflozin, Cardiovascular Outcomes, and Mortality in Type 2 Diabetes. <i>New England Journal of Medicine</i> 2016;374(11):1092–4.</li> </ol>
<p><b>Jorge Reyes Castro</b></p>	<p><i>Anasarca: Unusual Presentation of Cardiac Sarcoidosis</i></p> <p>Introduction: Anasarca is the presenting sign of volume overload for a wide spectrum of differential diagnoses. This clinical vignette details a 53 year-old male with PMHx of pulmonary sarcoidosis presenting to the emergency department with worsening SOB and anasarca.</p> <p>Case Description: 53 y.o. male with significant PMHx significant for pulmonary sarcoidosis and 1st degree AV block presented to the emergency department with worsening SOB and anasarca for two weeks. Per patient report, the edema started on his thighs and progressed up to his abdomen, and further down his legs. On arrival, physical exam was remarkable for presence of JVD, bilateral leg edema + 3 and clear breath sounds. Initial work up showed elevated BNP, EKG with newly discovered atrial flutter with 4:1 and 5:1 conduction, and a chest x-ray showed right pleural effusion. He was started on IV diuresis. On the first day of admission, TTE reported; right ventricular enlargement with decreased right ventricular systolic performance associated to severe tricuspid regurgitation, estimated PASP was 22mmHg. Due to the combination of atrial flutter with significant AV conduction disease, manifested by a very slow ventricular response, the possibility of infiltrative disease/cardiac sarcoidosis was considered. A cardiac MRI was ordered, unfortunately, the patient was not able to tolerate the MRI and refused endomyocardial biopsy. On the 3rd day of admission, right heart catheterization demonstrated mild pulmonary HTN (mean PA 29mmHg), and elevated RV filling pressure with RVEDP consistent with right ventricular failure. On the 8th day of admission, PET CT reported infiltrative pattern especially in the right ventricular free wall, as well as the septum, interpreted as being diagnostic for cardiac sarcoidosis. The decision was made to begin steroids. During the hospital course, he had received aggressive diuresis during 9 days, with IV bumex (net negative 22L). On discharge, his symptoms and pedal edema had improved dramatically.</p> <p>Discussion: Sarcoidosis is a multisystemic disorder of unknown cause, characterized by the formation of granulomas in the organs involved. The lungs and the lymphatic system are predominantly affected, but virtually every organ may be involved[1]. Cardiac involvement occurs in perhaps 5% of patients with sarcoidosis[2]. Anasarca as severe manifestation of right side heart failure in a patient with pulmonary sarcoidosis is usually suspected to be secondary to pulmonary hypertension. However, conduction system abnormalities in EKG as our patient developed, should prompt further investigation for sarcoid involvement in the heart. In addition, our case</p>



	gives us a unique opportunity to learn more about how to work up and initially treat cardiac sarcoidosis.
<b>Michael Ritchie</b> Dr. Dr. Amy Holbrook	<p><i>Not Just the Munchies: A Case of Squamous Cell Carcinoma of the Neck Initially Presenting as Iron Deficiency Anemia and Atypical</i></p> <p>Pica is classified by the DSM 5 as a psychiatric disorder whose diagnosis requires non-nutritive ingestion for 1 month that is not associated with cultural practices and inappropriate for the patients level of development. In clinical practice it is often associated with mineral deficiencies, such as iron or magnesium, often it is sometimes the only clue to the underlying mineral deficiency. Our case is of a 74 year old male with a history of diabetes, chronic mild iron deficiency anemia due to intestinal arterio-venous malformations and a severe marijuana use disorder, using up to twenty times per day for over 50 years. He initially presented for routine diabetes management follow-up when he noted an odd new practice he had started the month before, an intense urge to “huff” dirt. He reported placing dirt in a bag then agitating and inhaling the contents. Initial evaluation revealed a profound iron deficiency anemia and he was admitted for further evaluation. While his GI history was the initial focus he related that he huffed the dirt due to difficulty swallowing, which given his tobacco and marijuana use raised concern for a neck mass. Computed Tomography (CT) of the neck showed a large mass and biopsy revealed squamous cell carcinoma. He was subsequently treated with chemotherapy and radiation without resolution and was transferred to comfort care and passed away within 6 months of his diagnosis. This case highlights both an atypical manifestation of Pica that led to a much more dire diagnosis and the importance of considering novel patient concerns as they can be the initial harbinger of more life-threatening disease.</p>
<b>Michael Richter</b> Dr. Hannah Nordhues	<p><i>A Warning Against Medication Non-Adherence in Antiphospholipid Syndrome</i></p> <p>Case Description: A 39 year old woman was hospitalized with acute-on-chronic dyspnea. She had a history of an undifferentiated connective tissue disease manifesting with a livedoid rash, Raynaud’s phenomenon, and arthralgias. Her dyspnea began two months prior but progressed rapidly over the previous few days and was associated with orthopnea and lower extremity pitting edema. Initial laboratory evaluation revealed an elevated d-dimer, elevated creatinine of 1.5 mg/dL, and mild normocytic anemia. CT chest was negative for pulmonary embolism but showed extensive alveolar opacities and bilateral pleural effusions. Subsequent echocardiogram revealed severe mitral regurgitation with mitral valve lesions consistent with Libman-Sacks endocarditis. Further laboratory testing showed a positive ANA, anti-dsDNA, anti-phospholipid IgG, anti-beta 2 glycoprotein, and anti-cardiolipin antibodies. She was started on enoxaparin and aspirin for presumed antiphospholipid syndrome along with intravenous diuresis for congestive heart failure thought to be secondary to her valvular disease. She responded well, her respiratory status returned to baseline, and she was discharged on warfarin.</p> <p>Two weeks after discharge, the patient returned to the ED with encephalopathy, fever, and acute renal failure. Family members reported a three day history of somnolence, hallucinations, and abnormal language. Brain MRI showed extensive cerebral and cerebellar infarcts secondary to microembolization. Laboratory evaluation revealed a hemoglobin of 6.9, platelets of 56,000, creatinine of 3.6 mg/dL, elevated fibrinogen, and elevated LDH. An ADAMTS13 assay was within normal limits. She was started on an unfractionated heparin drip, broad-spectrum antibiotics, and pulsed IV methylprednisolone for catastrophic antiphospholipid syndrome. Her renal function and respiratory status continued to worsen over the first 48 hours and she was started on hemodialysis and plasma exchange. Bronchoscopy also revealed diffuse alveolar hemorrhage. Her hospital course was further complicated by severe pseudomembranous colitis and a hemodialysis line infection. It was later discovered that three days after her first hospitalization, the patient stopped taking warfarin as prescribed due to doubts about its necessity. At the time of dismissal, the patient was on a steroid taper and was advised to continue anticoagulation indefinitely as</p>

	<p>discontinuation of anticoagulation was thought to incite catastrophic antiphospholipid syndrome (CAPS).</p> <p>Discussion: Clinicians should be aware of antiphospholipid syndrome (APS) and the critical importance of anticoagulation. In rare cases, APS progresses to CAPS causing vascular occlusion of multiple organs. This condition can be difficult to recognize and is associated with a mortality rate as high as 50%. There have been several case reports of APS progressing to CAPS upon discontinuation of anticoagulation, which was thought to be the inciting event in this case.</p>
<p><b>Parrisha Roane</b>  Dr. Karim Thomas  Sadak  Melissa Claar  Dr. Cuong Pham</p>	<p><i>The Internist and the Transitioning Sickle Cell Disease Patient</i></p> <p>Case Presentation: A 20-year-old male with a history of anxiety and HbSS disease complicated by acute chest syndrome (ACS), vaso-occlusive crisis, avascular necrosis of the femur, mild mixed obstructive/restrictive pulmonary disease, and unprovoked LLE DVT, presented to Pediatric Hematology clinic for his final appointment prior to transitioning to adult care. At the time of presentation, he was asymptomatic. His last transcranial doppler (TCD) screen in 2014 was normal and his ophthalmologic screen two years ago showed no retinopathy. His microalbuminuria screen six months ago showed a mildly elevated urinary protein. Vaccinations were up to date. Labs obtained at the visit were consistent with his baseline.</p> <p>Self-management skills were discussed, including strategies to remember his appointments and for taking his medications daily. Socioeconomic barriers to care were re-visited, including inability to obtain mental health support due to financial difficulties and working full time. He was provided with a healthcare summary and advised to follow-up with his internist.</p> <p>Conclusion: There is an insufficient number of sickle cell disease providers and a lack of providers who feel comfortable caring for sickle cell patients. However, transitioning patients may present to the ambulatory internist in hopes of establishing care. This case highlights the need for internists to be familiar with the unique healthcare maintenance of sickle cell patients undergoing transition. The 2014 National Heart and Lung Blood Institute (NHLBI) Evidence-Based Management of Sickle Cell Disease, states that:</p> <ol style="list-style-type: none"> <li>1. All patients with SCD should have an annual dilated retinal examination beginning at 10 years of age due to risk of retinopathy.</li> <li>2. All patients with SCD should be screened for nephropathy with an annual microalbuminuria screen.</li> <li>3. All patients should be vaccinated against Streptococcus pneumoniae.</li> <li>4. All adult patients should be screened for hypertension and treated to a goal blood pressure of <math>\leq 140/\leq 90</math>.</li> <li>5. All patients should be assessed for respiratory problems (such as asthma, COPD, restrictive lung disease, or obstructive sleep apnea).</li> <li>6. While patients with HbSS or HbSB0 should be screened annually from ages 2-16 years old for stroke prevention, adult patients were not recommended to be screened.</li> <li>7. Hydroxyurea therapy should be initiated in adults with sickle cell anemia if patients have three or more moderate to severe pain crises in a 12-month period, daily sickle cell pain that affects quality of life, severe or recurrent ACS, and/or severe chronic anemia.</li> </ol> <p>In addition to health care maintenance considerations, this case also highlights the necessity of personal skills development, addressing socioeconomic barriers to care, and medical summaries in the transition process. The American Society of Hematology spearheaded the development of a sickle cell readiness assessment and clinical summary that are available to the internist to assist in the transitions process.</p>
<p><b>Alexander Sahakian</b>  Dr. Gordon Ruan  Dr. Luke Hafdahl</p>	<p><i>Chronic Diarrhea: Grow the Differential</i></p> <p>Introduction: Small intestinal bowel overgrowth (SIBO) is an increasingly recognized cause of diarrhea due to the pathologic proliferation of colonic-type bacteria in the</p>

	<p>small intestine. Its diagnosis can be challenging as patients often present with nonspecific gastrointestinal (GI) symptoms, however patients will typically have identifiable predisposing risk factors.</p> <p>Case Presentation: A 61 year old homeless male presented with generalized weakness in the setting of acute on chronic diarrhea. His past medical history includes type 2 diabetes mellitus (DM2), hypertension and chronic normocytic anemia. He was previously lost to follow up for 13 years shortly after being diagnosed with DM2 with a HbA1c of 10.2%. He declined treatment at that time and did not reestablish care until he began suffering vision loss, severe neuropathic pain and manifestations of diabetic nephropathy. In the interim, he unintentionally lost 100lbs and repeat HbA1c was 6.2%.</p> <p>On admission, the patient reported 15-20 liquid, often explosive, bowel movements per day. He endorsed experiencing mild diarrhea for the past 5 years, but noted a marked increase in frequency and severity over 6 months prior to admission. His symptoms briefly improved after beginning loperamide 4mg four times daily, but the diarrhea acutely worsened after one week. He denied fever, chills, sick contacts, recent travel, antibiotic use, melena, hematochezia, steatorrhea or any obvious dietary contributors, including artificial sweeteners.</p> <p>Complete blood count showed hemoglobin 9.9 g/dL, but no leukocytosis. Electrolytes, liver function tests and thyroid stimulating hormone were within normal limits. C-reactive protein was elevated at 21 mg/L. Stool pathogen panel, including <i>Clostridium difficile</i>, was negative. Celiac disease serology was unremarkable. Colonoscopy with random biopsies was negative for microscopic colitis. Duodenal biopsies obtained via esophagogastroduodenoscopy (EGD) showed normal mucosa without evidence of Whipple’s disease, celiac sprue, <i>Giardia</i> or amyloid deposition. However, small bowel aspirate was notable for a total intestinal flora greater than 100,000 CFU/mL consistent with a diagnosis of SIBO. His symptoms dramatically improved with a 7 day course of metronidazole.</p> <p>Discussion: This case illustrates the importance of including SIBO in the differential diagnosis of diarrhea. The diagnosis can be challenging as the clinical presentation is often fairly nonspecific and patients may not have the classic symptom of steatorrhea. This is especially true for patients with risk factors such as surgical anatomical alterations, GI dysmotility, immunodeficiency and proton pump inhibitor use. In this patient’s case, years of uncontrolled diabetes likely led to impaired GI motility and gastroparesis, as evidenced by residual food visualized on EGD despite adequate fasting.</p> <p>For patients with predisposing conditions, testing for SIBO, whether through breath testing, empiric treatment trials or small bowel aspirate, can be of great value as patients typically improve with antibiotics.</p>
<p><b>Tyler Schmidt</b></p>	<p><i>Right on "Target"; Diagnosing Intussusception in a Healthy Adult Female</i></p> <p>Case Presentation: A 41 year old female with peptic ulcer disease and history of Roux-en-Y gastric bypass presented to the Emergency Department with left lower quadrant abdominal pain, nausea and vomiting. The patient noted that her abdominal pain started to increase for one week especially in the left lower quadrant but the day before presentation became acutely worse with radiation to the back and associated nausea and vomiting. Due to the worsening pain not responding to oral pain medications she subsequently presented to the local Emergency Department for further workup and management. In the Emergency Department, the patient was hemodynamically stable. Initial labs revealed negative CBC, BMP, lactate, and urine b-hCG. CT Abdomen/Pelvis with contrast revealed a segment of bowel intussusception at her jejunojunal anastomosis. Surgery was consulted and, due to history of abdominal surgeries and concern for hostile abdomen, she was recommended conservative management with bowel rest, pain control, and fluid</p>

	<p>rehydration. Her pain progressively improved over several days and she rapidly demonstrated regular bowel movements. She was ultimately discharged with plans to follow up with bariatric surgery regarding possible surgical options for her anastomosis.</p> <p>Discussion: Intussusception is a telescoping of a proximal segment of gastrointestinal tract within the lumen of an adjacent tract. This disorder involves disruption of normal peristalsis. Intussusception is rare in adults, with only 5% of all intussusception cases in the adult population. It is also a rare form of bowel obstruction, compromising 1-5% of all bowel obstructions in adults. Unlike in children, intussusception in adults is usually secondary to a pathologically point, such as polyps, colonic diverticula, or strictures. The clinical presentation of intussusception is nonspecific, unlike the classical triad in children with colicky abdominal pain, hematochezia, and palpable abdominal mass. Often, adults with intussusception will present with symptoms suggestive of small-bowel obstruction, such as nausea, vomiting, and abdominal pain. Diagnosis of intussusception is most effectively achieved with CT scan, with sensitivity in some studies up to 100%. This imaging modality is also recommended due to ability to further image the abdomen for causes of the intussusception, such as polyps, diverticula, and neoplasms. Due to the elevated risk of malignancy associated with intussusception in adults, the most definitive treatment is surgical reduction/resection based on the viability of tissue.</p>
<p><b>Brent Schotl</b> Dr. David Tierney</p>	<p><i>The Dirt on Infectious Horner's Syndrome</i></p> <p>Introduction: Blastomyces is a pathogen primarily reported around North America's Ohio and Mississippi River valleys and Great Lakes. It has multiple infectious phenotypes, from asymptomatic to severe disseminated disease, with case reports of nearly every organ system being affected. Blastomyces most often causes pneumonia and can progress to ARDS if not treated promptly.</p> <p>Case Presentation: An 81-year-old man with a history of coronary artery disease, atrial fibrillation, heart failure with reduced ejection fraction, chronic kidney disease, and type II diabetes initially presented to urgent care with URI-like symptoms. He was empirically treated with azithromycin for bronchitis. His symptoms progressed and he presented to his PCP where a chest X-ray demonstrated a right upper lobe pneumonia and was treated with oral levofloxacin. After 3 days without improvement he presented to his local hospital where he was treated with piperacillin-tazobactam for presumed aspiration pneumonia. Recent history was remarkable for staying at his cabin in Northern Minnesota but reported being mostly indoors. Legionella, Histoplasma, and Blastomyces antigens were negative on admission. The patient developed respiratory failure requiring intubation. Bronchoscopy was performed and lavage sample grew Blastomyces dermatitidis. Initial treatment was liposomal Amphotericin B for severe ARDS. Several days after starting antifungal therapy the patient developed ptosis, anisocoria with left pupil larger than right, worsened in a dark environment, and generalized weakness. In the setting of therapeutic heparin for atrial fibrillation, concern for intracranial bleed resulted in an emergent head CT and subsequent MRI, both normal.</p> <p>With a normal brain MRI, his anisocoria and slight ptosis was consistent with a right Horner's syndrome due to progressive infiltrative disease in the right pulmonary apex. The patient improved with prolonged vasopressor and ventilator support and was discharged to an LTACH for ongoing medical care and rehabilitation. The patient was weaned from the ventilator and his Horner's syndrome resolved within 3-months and was treated with 1-year of oral itraconazole. The day prior to discharge, his wife told us about him picking blueberries in Northern Minnesota one month prior to admission which was the only known soil exposure in recent patient history.</p> <p>Discussion: Infectious causes of Horner's syndrome are rare, but Dr. Henry Pancoast's original 1924 paper included a series of patients with Mycobacterium tuberculosis as a cause of Horner's syndrome. Subsequently, multiple other case</p>

	<p>reports have shown various infectious pathogens causing Horner’s syndrome, but to our knowledge Blastomyces dermatitidis has not been reported as a cause in humans. Blastomycosis pneumonia may go undiagnosed for several weeks and may present as a chronic pneumonia or progress to ARDS. Due to the prolonged disease course treatment is often with itraconazole for 6-12 months.</p>
<p><b>Richard Silbert</b> Dr. Adam Sawatsky</p>	<p><i>Sarcoidosis Presenting as Vomiting and Weight Loss</i></p> <p>Introduction: Sarcoidosis is a multisystemic disorder which rarely presents without pulmonary involvement. Diagnosis of extrapulmonary sarcoidosis requires a high degree of suspicion, compatible biopsy demonstrating non-caseating granulomas, and thorough exclusion of alternative causes.</p> <p>Case Presentation: A 55 year old African-American female presented to the hospital with 7 months of epigastric pain, nausea, and vomiting. Additional symptoms included diffuse musculoskeletal pain, arthralgias, and 70 pound unintentional weight loss.</p> <p>Upon admission, lab work was remarkable for hypokalemia, normocytic anemia, and elevated C-reactive protein. A thorough infectious disease workup including HIV, hepatitis B and C, tuberculosis, cytomegalovirus and Epstein Barr virus was negative. Autoimmune serologies including an extractable nuclear antigen panel, anti-nuclear antibodies, anti-neutrophil cytoplasmic antibodies, anti-cyclic citrullinated peptide, and antiphospholipid antibodies were also negative. AM cortisol and thyroid function testing was normal.</p> <p>Chest CT showed no hilar lymphadenopathy or lung parenchymal involvement. Abdominal CT showed mild retroperitoneal and inguinal lymphadenopathy. Due to persistent vomiting and poor oral intake, an Esophagealgastroduodenoscopy was performed which demonstrated erythematous gastric mucosa and duodenitis with non-specific inflammation on duodenal biopsy.</p> <p>Later during hospitalization the patient developed pruritic lichenoid plaques on her lower extremities. Biopsy revealed non-caseating granulomas. Persistent hypercalcemia was noted on lab work and treated with fluids and diuretics. Angiotensin converting enzyme (ACE) levels were elevated. Subsequently, she began having recurrent episodes of fever, hypotension and tachycardia. Broad spectrum antibiotics were started for sepsis without clinical improvement. An FDG-PET scan was performed to assess for vasculitis or occult malignancy. This showed avid FDG uptake in the spleen as well as inguinal and popliteal lymph nodes. Right inguinal lymph node biopsy demonstrated non-caseating granulomas.</p> <p>A diagnosis of extra-pulmonary sarcoidosis was made. The patient was started on methotrexate and high dose steroids with near resolution of her abdominal pain and vomiting. Lab work including hemoglobin, calcium, and inflammatory markers had normalized at 2 month follow up.</p> <p>Discussion: This case represents a rare instance of sarcoidosis presenting with primarily gastrointestinal (GI) manifestations. Clinically evident sarcoid involvement of the GI tract is present in less than 1% of cases of sarcoidosis and is even less frequently the presenting complaint. The stomach is the most commonly involved site but any portion of the GI tract can be affected. Our patient’s epigastric pain and vomiting were likely due to gastric involvement, however no gastric biopsy was obtained for confirmation. A diagnosis of sarcoidosis requires evidence of non-caseating granulomas, exclusion of alternative causes of granulomas, and a compatible clinical syndrome. The rarity of GI involved sarcoidosis makes diagnosis based on symptoms difficult. In this case, it was only through an extensive workup for alternative causes and the development of more classic sarcoid skin involvement and hypercalcemia that allowed a diagnosis to be made.</p>
<p><b>Caleb Smith</b> Dr. Elie Berbari</p>	<p><i>Red Blood Cell Exchange for Severe Babesiosis</i></p> <p>Introduction: Babesiosis is caused by intraerythrocytic protozoa of the genus Babesia. Clinical manifestations of infection range from asymptomatic illness to fulminant</p>

	<p>disease resulting in multi-organ failure. While most patients with mild to moderate disease are successfully managed with antibiotics alone, those suffering severe disease (clinically significant anemia, or renal, hepatic, or pulmonary compromise) with heavy parasite burden (&gt;10%) may benefit from red blood cell exchange.</p> <p>Case Description: A 63 year-old male with a medical history significant for chronic hepatitis C presents to his primary care provider with fever, headache, and hematuria. Physical examination revealed left upper quadrant tenderness. Laboratory evaluation was significant for a hemoglobin of 16.2 g/dL, platelet count <math>36 \times 10^9/L</math>, WBC <math>7.4 \times 10^9/L</math>, creatinine 0.75 mg/dL (baseline 0.70 mg/dL), total bilirubin 2.2 mg/dL, and AST 52 U/L. Urinalysis demonstrated pyuria (WBC 21-30 units/hpf) and urine culture grew Escherichia coli. Cefdinir was prescribed for his presumed urinary tract infection. Four days later the patient presented to the emergency department with persistent fevers, night sweats, left upper quadrant pain, and worsening weakness. On physical examination he was confused and sluggish to respond to questioning. Sclera icterus was present and his spleen was palpable at the left costal margin and exquisitely tender. Laboratory studies were notable for a hemoglobin of 12.4 g/dL, platelet count <math>40 \times 10^9/L</math>, WBC <math>10.3 \times 10^9/L</math>, Creatinine 2.69 mg/dL, total bilirubin 6.6 mg/dL, AST 146 U/L, Haptoglobin &lt;14 mg/dL, LDH 1892 U/L, PTT 31 sec, INR 1.1, and fibrinogen 778 mg/dL. Computed tomography scan of the patient's abdomen demonstrated splenomegaly with a splenic infarct. Serologic testing for infections secondary to Anaplasma, Ehrlichia, and Borrelia species was negative. Peripheral blood smear revealed intraerythrocytic parasites in 10.2% of red blood cells. Babesia microti IgG and PCR were positive. Therapy was initiated with azithromycin 500mg daily and atovaquone 750mg twice daily. Due to the patient's parasite burden greater than 10% and significant renal injury, the patient underwent one exchange transfusion session. Following exchange transfusion the patient's parasite burden decreased to 0.9%. His clinical status improved and he was discharged on hospital day 6 to complete a course of oral antimicrobial therapy.</p> <p>Discussion: This case illustrates a presentation of severe babesiosis with rapid improvement following red blood cell exchange therapy. Though antimicrobial therapy remains the cornerstone of treatment in babesiosis, patient's presenting with severe disease may benefit from additional supportive interventions. Familiarity with indications for utilizing red cell exchange may improve outcomes in patients presenting with severe babesiosis.</p>
<p><b>Joseph Steffens</b>  Dr. Michael Megaly  Dr. Bradley Bart  Dr. Louis Kohl</p>	<p><i>Left Coronary-Cameral Fistula Contributing to Myocardial Infarction, or Red Herring?</i></p> <p>Introduction: Coronary-cameral fistulas are rare malformations that are predominantly congenital and asymptomatic. They are connections between the coronary vasculature and one or more chambers of the heart - most commonly from the right coronary artery to the right ventricle.</p> <p>As congenital malformations, coronary-cameral fistulas seldom produce hemodynamic consequences. In some cases, shunting caused by these fistulas can lead to pathophysiology. Left to right shunting that occurs with fistulas that drain to the right ventricle can result in pulmonary hypertension and right-sided heart failure symptoms. Shunts to the left ventricle can result in heart failure, ischemia, arrhythmia, and infarction. We present a case of a 57-year-old man with shortness of breath, chest tightness, and elevated troponin in the setting of systolic hypertension who was found to have left coronary artery to left ventricle coronary-cameral fistula and evidence of possible apical hypokinesis.</p> <p>Case Description: A 57-year-old man presented with shortness of breath and chest tightness of two days duration. He was found to have elevated serum troponin levels (0.151 mcg/L, followed by 0.16 mcg/L, 0.141 mcg/L, and 0.133 mcg/L), dynamic EKG changes including new T wave inversions in leads V3 and V4 on his electrocardiogram, chest radiography consistent with pulmonary edema, and</p>

	<p>echocardiogram which showed a possible apical wall motion abnormality and ejection fraction of 43%. He underwent coronary angiography which showed a fistula between OM2, OM3, D3, and the left ventricle. No severe stenoses were present. The left ventricular end-diastolic pressure (LVEDP) was elevated at 38-40 mmHg, in the setting of systemic hypertension (~160/100).</p> <p>The patient was diagnosed with heart failure with reduced ejection fraction, non-ST-elevation myocardial infarction, and mixed ischemic and hypertensive cardiomyopathy. He was initiated on optimal medical therapy for heart failure and coronary artery disease with carvedilol, aspirin, lisinopril, furosemide, and atorvastatin, and was discharged.</p> <p>On subsequent encounters in the clinic, his blood pressure and symptoms improved with medical therapy. A repeat echocardiogram showed resolution of his apical wall motion abnormality and an ejection fraction of 55%. Closure of the fistula was deferred since the patient's symptoms and possible apical wall motion abnormality resolved with medical therapy.</p> <p>Discussion: Our case illustrates the rare entity of left coronary-cameral fistula presenting with heart failure and myocardial infarction. It is likely that heart failure resulted from uncontrolled hypertension and possible that myocardial infarction occurred from the coronary-cameral fistula causing hemodynamically significant shunting, or "coronary steal."</p> <p>Most patients with coronary-cameral fistulas are asymptomatic. However, among patients with coronary-cameral fistulas who are symptomatic, closure is the mainstay of therapy. Our patient's coronary artery fistula was likely congenital in origin and his symptoms and heart failure resolved with medical therapy. Therefore fistula closure was not indicated.</p>
<p><b>Frederique St-Pierre</b>  Dr. Sayed Obaidulah Aseem  Dr. Will Schouten</p>	<p><i>Burned by the History: Life-Threatening Consequences of a Blistering Eruption</i></p> <p>Introduction: Blistering skin disorders are characterized by the presence of fluid-filled lesions on the skin. The differential diagnosis for this finding is broad, including autoimmune disease, infection, allergy, trauma, burns, and systemic illness. The disease course ranges from indolent to life-threatening. Making this distinction on history and physical exam alone can be challenging, and it is imperative to maintain adequate suspicion for potentially dangerous and unexpected causes of cutaneous blisters.</p> <p>Case Presentation: An 82-year-old woman with a medical history significant for hypertension and hyperlipidemia presented to the Emergency Department for a one-week history of progressive bilateral lower extremity weakness and difficulty ambulating. Physical exam was remarkable for a painful, erythematous, blistering eruption on her left face and ear. The patient stated she had suffered a burn from a heating pad. Initial investigations were significant for serum sodium of 125 mEq/L, and random glucose of 333 mg/dL. Hemoglobin A1c was 11%, and she was diagnosed with uncontrolled diabetes mellitus type 2. After IV fluid administration and initiation of insulin therapy, she quickly improved and her gait and serum sodium normalized. Her presumed facial burn was treated with bacitracin ointment, and swab of an unroofed blister was sent for herpes simplex and varicella zoster PCR testing as a precaution.</p> <p>The following night, she was noted to have progressively worsening neck swelling and labored breathing with inspiratory stridor. Neck X-ray showed thickening of the submandibular soft tissues with narrowing of the subglottic trachea. She was transferred to the ICU, and required emergency intubation. Treatment with broad-spectrum IV antibiotics and dexamethasone was initiated. Testing performed on the facial blister returned positive for varicella zoster virus. IV acyclovir was added to her treatment regimen. Bacterial culture of a second skin swab grew MRSA. It became</p>

	<p>evident that her facial rash was in fact a herpes zoster infection of the left cervical dermatomes with superimposed MRSA infection, subsequently resulting in surrounding soft tissue swelling and severe upper airway obstruction. Within 24 hours of steroid and antimicrobial treatment, she had markedly improved and was successfully extubated.</p> <p>Discussion: Bacterial superinfection is a well-established complication of herpes zoster. In patients hospitalized with shingles, this may occur in as many as 16% of cases. In turn, airway compromise is a feared complication of facial cellulitis and deep neck space infections. However, airway obstruction from herpes zoster infection has rarely been reported in the literature. This case highlights the fact that airway compromise, although rare, is a potential life-threatening complication of zoster infections involving the head and neck, specifically when superinfection occurs. It is imperative to maintain suspicion for alternate diagnoses when a patient presents with cutaneous blisters, as timely diagnosis and treatment could potentially prevent the development of life-threatening complications.</p>
<p><b>Kevin Sullivan</b> Dr. Anahita Alvanpour</p>	<p><i>Inhibiting the Inhibitor; A Case of Lupus-Induced Severe Angioedema</i></p> <p>Introduction: Angioedema is an uncommon but severe symptom of systemic lupus erythematosus, which can be deadly if not treated properly. This is a case of a new diagnosis of lupus presenting as recurrent severe angioedema requiring emergent cricothyrotomy with subsequent tracheostomy, in the setting of recurrent pneumococcal disease.</p> <p>Case presentation: A 45 year-old Latina female with a past medical history of facial swelling thought to be 2/2 pneumococcal sepsis eight months prior to presentation, on no current medications, presented to the emergency room with facial swelling causing airway obstruction. She received diphenhydramine, dexamethasone, and intramuscular epinephrine, but her symptoms did not improve and she required emergent cricothyrotomy in the ED, which was immediately converted to a tracheostomy in the OR. She was admitted to the medical ICU and ultimately found to have pneumonia and bacteremia, again from streptococcus pneumoniae. Other initial labs were significant for leukocytosis but severe absolute lymphopenia (<math>0.0 \times 10^9/L</math>), severe anemia (6.6 g/dL), acute kidney injury (creatinine=1.4 mg/dL), elevated ESR=82mm/hr, low C3=43mg/dL, and low C4=3.7mg/dL. Upon further review, the patient had previously had a positive ANA obtained for workup of anemia, and further workup had found a positive anti-dsDNA, low C3, low C4, and low CH50, but she had been lost to follow up before being able to discuss these results further. With her laboratory findings and clinical symptoms, the patient was diagnosed with systemic lupus erythematosus with concern for lupus nephritis and started on methylprednisolone 1000mg daily. Given her angioedema, C1-esterase inhibitor function and levels were obtained; the function was normal (70%), but the level was low (16). C3 and C4 remained persistently low, total complement (CH50) was low, and C1q was undetectable. A kidney biopsy was obtained, which showed mild focal global and focal segmental glomerulosclerosis consistent with type V (membranous) lupus nephritis. The patient's symptoms quickly improved on IV steroids, her acute kidney injury resolved, and she was transitioned to oral prednisone and plaquenil.</p> <p>Discussion: This is a dramatic case of recurrent angioedema due to C1-esterase inhibitor deficiency, and general complement deficiency leading to recurrent pneumococcal disease. Acquired angioedema (AAE) can be caused by a lymphoproliferative disorder or rarely an autoimmune disease. In the case of autoimmune disease such as SLE, there can be increased complement consumption or development of antibodies against C1-esterase inhibitor, either leading to kinin-mediated angioedema. Our patient had a normal C1-esterase inhibitor activity but a low serum level of the protein, consistent with this pathophysiology. AAE due to lupus can have a delay in diagnosis and be difficult to reverse, often leading to significant morbidity and death; swift diagnosis and effective treatment of the underlying lupus is extremely important.</p>



<p><b>Alex Tarabochia</b> Dr. Brianna Vaa</p>	<p><i>Stress Induced Cardiomyopathy: More than emotion?</i></p> <p>Introduction: Stress cardiomyopathy (SC), aka takotsubo cardiomyopathy or apical ballooning syndrome, is a rapidly reversible form of acute heart failure reported to be triggered by emotionally and physically stressful events.<sup>1</sup> The clinical presentation often mimics acute myocardial infarction (AMI) with cardiac symptoms (predominantly chest pain and dyspnea), ST changes (typically ST elevation), troponin elevation, and global ventricular hypo/akinesis without evidence of a perfusion defect.<sup>2</sup> We present a case of SC in a patient with no prior cardiac disease who was hospitalized for rhabdomyolysis in the setting of a mechanical fall.</p> <p>Case Description: The patient is a 76-year-old woman with past medical history of hemorrhagic stroke with residual ataxia and dysarthria who was brought to the emergency department after a fall with prolonged immobilization (&gt;two hours). Physical exam and imaging including knee x-ray and CT head were all unremarkable. Labs were remarkable for creatinine kinase (CK) of 9,139 consistent with rhabdomyolysis. This was managed with aggressive IV fluid resuscitation without a rise in serum Cr.</p> <p>However, on hospital day two she developed dyspnea and a new oxygen requirement of two liters. She denied chest pain/pressure, or numbness and tingling of the arm or neck. A stat electrocardiogram showed diffuse ST-elevation in the anterior leads with a troponin of 680. Her coronary angiogram revealed mild diffuse atherosclerosis without evidence of an obstructing lesion. Transthoracic echocardiogram showed an ejection fraction of 39% and left ventriculography demonstrated global akinesis of the anteroapical wall, consistent with SC. Her acute systolic heart failure was managed with metoprolol succinate and furosemide. Within a few days she clinically improved, and was discharged to a skilled nursing facility for ongoing rehab.</p> <p>Discussion: SC occurs in as many as 1-2% of patients presenting with suspected ACS.<sup>3</sup> The most likely underlying mechanism is a heightened sympathetic response, as catecholamine levels are 2-3x's as high as their ACS counterparts.<sup>4</sup> The most classic finding is characteristic hypo/akinesis of the mid and apical segments of the left ventricle with sparing of the basal systolic function without obstructive coronary lesions.<sup>3</sup> Optimal management includes <math>\beta</math>-blockade, diuretics, and anticoagulation in patients with severely compromised EF or evidence of thrombus on echo, with the vast majority of patients achieving recovery within eight weeks of receiving medical management. While the "typical" patient is an elderly female with significant recent emotional stress, physical stressors such as rhabdomyolysis as seen in our patient, malnutrition, and recent exposure to <math>\beta</math>-agonist and catecholamine medications can all be causes of SC and should be considered in any patient with new ACS symptoms.<sup>5</sup></p>
<p><b>Lauren Thornton</b></p>	<p><i>An Unexpected Diagnosis: Sarcoidosis</i></p> <p>Case Presentation: A 41-year old woman with history of asthma presented to the emergency department with a two-hour history of shortness of breath and chest pain, as well as chronic non-productive cough. Chest x-ray revealed a large right-sided pneumothorax, and a chest tube was placed with immediate improvement in symptoms. Chest x-ray also revealed multiple masses in the left lung, and non-contrast chest CT revealed bilateral upper lobe cavitory versus cystic lesions. She was also presumed to have an aspergilloma. She was admitted for further workup.</p> <p>A broad infectious workup was pursued and negative including AFB/fungal cultures, HIV, histoplasma, blastomyces and aspergillus antigens, and 1,3 beta-d-glucan. She was initially treated with voriconazole, however this was discontinued with negative workup. Autoimmune workup was also pursued including negative ANCA, sweat chloride, and alpha-1-antitrypsin. Her rheumatoid factor was noted to be mildly elevated and ACE level elevated as well.</p> <p>The pneumothorax was felt to be spontaneous secondary to bleb rupture in setting of</p>

	<p>cystic lung disease. During admission, she had persistent air leak despite an additional chest tube. Cardiothoracic surgery was consulted for excision of the leaking bullae which was performed, and her pneumothorax resolved. Ultimately, lung biopsy tissue obtained during bullae excision revealed non-caseous epithelioid cell granulomas with focal necrosis. She also had a crusted lip lesion which was punch biopsied, consistent with mixed tuberculoid and sarcoidal granulomatous dermatitis. The consensus diagnosis was consistent with necrotizing sarcoidosis with an atypical presentation.</p> <p>At time of discharge, she continued to experience a non-productive cough, but shortness of breath had resolved. Outpatient, she was closely followed by pulmonology, ENT, infectious disease, and cardiothoracic surgery. She was initiated on prednisone therapy, and restarted on voriconazole for anti-fungal prophylaxis given presumed aspergilloma.</p> <p>Discussion: This case ultimately represents an atypical presentation of sarcoidosis. The steps to arrive at this diagnosis highlight the importance of a broad differential and workup of cystic lung disease. This case highlights management of and testing for presumed fungal infections including challenges with culturing and management of asymptomatic aspergillomas. Finally, it illustrates that the management of pneumothorax in the setting of cystic lung disease is challenging and must be multidisciplinary.</p>
<p><b>Siri Urquhart</b> Dr. Randall Pearson</p>	<p><i>Testosterone Injections – A Cause for Mesenteric Venous Thrombosis?</i></p> <p>Introduction: Mesenteric venous thrombosis (MVT) may be an incidental finding on abdominal imaging or a cause of abdominal pain. Prothrombotic states, malignancy, and local abdominal inflammatory disorders are common predisposing conditions. Many medications have been implicated in the development of prothrombotic states, including hormonal therapies. Use of exogenous testosterone as a cause of MVT has rarely been described in the literature. Therefore, it is important to recognize commonly prescribed medications as an etiology of MVT.</p> <p>Case Presentation: A 44 year-old man with a past medical history of hypertension presented with postprandial abdominal discomfort with associated nausea and constipation. On presentation, he was hypertensive, but otherwise hemodynamically stable. He had leukocytosis (14.7x10<sup>9</sup>/L) with neutrophilic predominance and mildly elevated ALT (73 U/L). His hemoglobin, electrolytes, kidney function, lipase, and remaining liver studies were within normal limits. Gallbladder ultrasound was unremarkable. CT scan of the abdomen/pelvis with IV contrast was notable for complete thrombosis of the superior mesenteric vein and its branches extending to the portal vein confluence with mild surrounding mesenteric edema. There was complete thrombosis of the intrahepatic right portal vein. The distal left intrahepatic portal vein branch was also occluded. He was initiated on IV heparin. After further review of his history, he reported receiving weekly testosterone injections prior to onset of symptoms. He was adopted, and therefore did not know of any family history of thrombophilia disorders or malignancies. Thrombophilia work-up was notable for negative antiphospholipid, beta-2 glycoprotein, and cardiolipin antibodies. He was transitioned to enoxaparin and warfarin on hospital dismissal with outpatient follow-up in vascular medicine and repeat imaging in three months. Additional evaluation for etiologies of MVT including infection or malignancy was planned for the outpatient setting. JAK2 analysis and Factor V Leiden testing were pending on hospital dismissal.</p> <p>Discussion: MVT is a rare condition accounting for 1 in 5,000 to 15,000 inpatient admissions and 6% to 9% of all cases of acute mesenteric ischemia. The presentation of MVT can be acute with sudden onset of symptoms, subacute with presentation over days to weeks, or chronic. The mean age at presentation is 40 to 60 years and is more common in males. Abdominal pain is the predominant symptom; however nausea, vomiting, diarrhea, or gastrointestinal bleeding may also occur. CT imaging diagnoses more than 90% of cases of MVT. The diagnostic finding for MVT is the</p>

	<p>presence of thrombus within the vein seen as focal translucency. Medical management includes anticoagulation with unfractionated heparin followed by transition to low-molecular-weight heparin and warfarin therapy. Exogenous testosterone as a cause of MVT is rarely described in the literature, and this novel case highlights the importance of considering this in the differential for MVT.</p>
<p><b>Elida Voth</b> Dr. Maryam Mahmood</p>	<p><i>Lung Abscess: A Rare Presentation of Legionnaires' Disease</i></p> <p>Introduction: Legionnaires' disease is often classified as an "atypical pneumonia"; however, it clinically presents similar to other forms of community-acquired pneumonia. Radiographically, the most common presentation is a patchy infiltrative process in the lower lobes. There are 14 serogroups of Legionella pneumophila which account for nearly 90% of cases, with serogroup 1 accounting for approximately 80%. We describe the case of a middle-aged immunocompromised man with lung abscess complicated by persistent fevers despite several days of broad spectrum antibiotics.</p> <p>Case Presentation: A 52-year-old man presented to the emergency department with 2 weeks of fevers and altered mental status. He had a past medical history notable for recurrent peripheral T cell lymphoma status post recent cycle of chemotherapy, Raynaud's phenomenon, eosinophilic fasciitis on prednisone and mycophenolate, and hypertension. Upon admission, physical exam was notable for bibasilar crackles, supraclavicular lymphadenopathy, and diffuse abdominal tenderness without guarding. Review of systems was notable for dry cough, dysuria, abdominal pain, and loose stools. Labs were significant for normocytic anemia, leukocytosis with neutrophilic shift, mild hyponatremia, significantly elevated C-reactive protein, and elevated lactate. Initial infectious work-up, including blood cultures, Legionella urine antigen, Streptococcus pneumoniae urine antigen, and respiratory pathogen panel was negative. Urinalysis was negative. Chest x-ray demonstrated a new retrocardiac opacity localized to the left lower lobe. Computed tomography (CT) scan of the chest showed necrotizing pneumonia with an associated non-cavitary abscess. The patient was initiated on meropenem and vancomycin. The following day, the patient became hypotensive, tachycardic, and continued to have high fevers. His hemodynamics responded appropriately to fluid resuscitation; however, he continued to have persistent fevers for several days.</p> <p>Six days after admission, a repeat CT scan showed worsening of the necrotizing pneumonia and pulmonary abscess with multiple locules of gas. A bronchoscopy was performed with results notable for positive Legionella PCR on bronchoalveolar lavage. The patient recalled frequent use of a hot tub and a humidifier with tap water at his home. He was initiated on levofloxacin and a percutaneous drain was placed into the abscess for source control. He continued to improve from an infection standpoint; but unfortunately, expired several weeks later due to complications of his underlying malignancy.</p> <p>Discussion: We report the case of a lung abscess in an immunocompromised patient as a rare presentation of Legionella pneumonia. This case highlights the limitations of the Legionella urine antigen with an estimated sensitivity of about 70-80%, as it only detects Legionella pneumophila serogroup 1. Lastly, lung abscess has been reported as a rare presentation of Legionella pneumonia, particularly in hosts with impaired cell mediated immunity and corticosteroid use.</p>
<p><b>Victoria Walston</b> Dr. David Brennan</p>	<p><i>Not Your Typical Reaction: Anaphylaxis in the Office</i></p> <p>Case Presentation: A 68-year-old man was evaluated in the Primary Care Clinic for knee pain, suspicious for a gout flare. The knee was prepped with chlorhexidine and joint aspiration was performed, yielding 35 cc of cloudy straw-colored fluid. After confirming acute gout under polarized light microscopy, the knee was injected with lidocaine (5 ml of 1%) and methylprednisolone (40 mg). Within minutes, the patient complained of a mild itch and asked to lay down. Physical exam revealed normal</p>

	<p>pulse rate, acrocyanosis and bilateral breath sounds with mild wheezing. He gradually became obtunded and developed hypotension and hypoxia. He had no rash or angioedema on exam but was noted to have urinary incontinence. EMS was called, oxygen applied, and nebulized albuterol ordered. The patient was given intramuscular epinephrine, bolused with IV fluids, intubated and taken to the Emergency Department. Tryptase level was elevated at 93.4 ng/ml confirming suspected diagnosis of anaphylaxis.</p> <p>He required mechanical ventilation and pressor support for several hours. He quickly improved and was discharged from the hospital after 3 days. He was later seen in Allergy Clinic, where he declined formal skin testing. Lidocaine is the presumed allergen.</p> <p>Discussion: Anaphylaxis to local anesthetics is extremely rare with case reports describing this phenomenon. Presenting symptoms are highly variable, posing a diagnostic challenge, but prompt pattern recognition and empiric treatment with intramuscular epinephrine are required. Most episodes are triggered through an immunologic mechanism involving IgE leading to mast cell and basophil activation with subsequent release of inflammatory mediators causing rapid respiratory obstruction and cardiovascular collapse. While signs and symptoms of anaphylaxis are unpredictable and variable, they often involve findings from multiple organ systems including skin, respiratory, cardiovascular, gastrointestinal, neurologic, oropharyngeal (table 1). Clinical diagnosis during an acute episode is primarily based on signs and symptoms, and confirmation of diagnosis is supported by skin testing and/or in vitro IgE tests, elevated concentrations of mast cell and basophil mediators such histamine or tryptase after onset of symptoms. Acute treatment begins with rapid assessment of breathing and circulation, followed by immediate administration of intramuscular epinephrine and emergent transport to the hospital for evaluation and further supportive care.</p> <p>Conclusions: Systemic anaphylaxis to local anesthetics is a rare yet potentially fatal phenomenon. Prompt recognition of symptoms involving multiple organ systems and rapid empiric treatment with intramuscular epinephrine are critical to prevent mortality.</p>
<p><b>Mason Webb</b> Dr. Mitchell Pitlick Dr. Christopher Stephenson</p>	<p><i>It AIN't AIN: A Case of Infection-Related Glomerulonephritis</i></p> <p>Introduction: Steroids are a powerful intervention that are critical for managing allergic and autoimmune diseases. However, diagnostic certainty is necessary in patients with high infection risk, as treatment may cause significant harm. We present a case of infection associated glomerulonephritis with initial signs concerning for allergic interstitial nephritis (AIN). Consideration of the patient's clinical picture is important before initiating empiric treatment, as treatment with high-dose steroids in a patient with concurrent infection can lead to significantly worsened morbidity and mortality.</p> <p>Case Presentation: A 61-year old man with recent MSSA epidural and bilateral psoas abscesses status post evacuation and IV cefazolin for one month presented to the emergency department with oliguria and hematuria for the past 24 hours. Lab evaluation showed a creatinine of 3.81 mg/dL, up from 0.56. Bilateral renal ultrasound revealed no signs of obstruction or hydronephrosis. A urinalysis revealed gross non-dysmorphic hematuria, pyuria, and renal epithelial cells. A presumptive diagnosis of allergic interstitial nephritis was made.</p> <p>Antibiotics were discontinued and renal biopsy was ordered to confirm diagnosis. High dose steroids were held due to concern for residual infection and pending renal biopsy. Renal biopsy returned positive for infection associated glomerulonephritis and acute tubular necrosis with red cell casts. The patient was started on hemodialysis and steroids were not initiated. At this time, the patient's mental status deteriorated and a MRI of the brain and lumbar spine revealed ongoing discitis osteomyelitis. Treatment with cefazolin was reinitiated and continued for another 6</p>

	<p>weeks. Discussion: AIN classically presents with acute kidney injury and urinary findings of hematuria, pyuria, and white cell casts. The most common causes of AIN are drugs, with the most common drug being beta-lactamase antibiotics, including cephalosporins. Current medical management recommends concurrent renal biopsy and initiation of high dose steroids without delay. However, due to concern for concurrent infection, we did not initiate treatment with high-dose steroids until renal biopsy returned.</p> <p>Discussion: Additional retrospective studies have suggested steroids may not improve renal recovery. Further, high dose steroids may lead to lymphopenia and significantly increased risk of infection, which could lead to increased morbidity and mortality in a patient with a latent CNS infection. As such, it is critical to assess the patient and weigh risks and benefits of high dose steroids in patients where there is diagnostic uncertainty.</p>
<p><b>Garrett Welle</b> Dr. Ravinda Ganesh</p>	<p><i>A Rare Presentation of Progressive Acral Vascular Necrosis in a Patient with Hepatocellular Carcinoma</i></p> <p>Case Presentation: A 62-year-old male with hepatitis C complicated by hepatocellular carcinoma (HCC) s/p transarterial chemoembolization (TACE), peripheral vascular disease s/p below-knee amputation, heart failure with preserved ejection fraction (LVEF 55%), and end-stage renal disease (ESRD) on peritoneal dialysis presented to our clinic for progressive bilateral acral vascular necrosis (AVN).</p> <p>Three months prior, he underwent TACE treatment for HCC which was complicated by the development of cold, painful, and discolored fingers on post-treatment day one. A large mobile mitral valve vegetation was visualized on TTE in the absence of positive blood cultures, leading to a diagnosis of AVN secondary to septic emboli from culture-negative infective endocarditis. He was treated with 6 weeks of IV antibiotics and repeat TEE demonstrated severe mitral annular calcification without vegetations, so he was discharged home on warfarin.</p> <p>He presented to our clinic a month after discharge for a second opinion. At this time, he was afebrile and hemodynamically stable but had progressive necrosis involving the distal right 2nd and 4th digits with left-hand involvement of 1st, 2nd, 4th, 5th digits with amputation of the distal 2nd and 3rd phalanges. He was then directly admitted for evaluation of his AVN. Warfarin was discontinued and he was placed on a heparin drip with final transition to Apixaban.</p> <ul style="list-style-type: none"> <li>• Dermatology was consulted to evaluate for atypical calciphylaxis due to elevated calcium-phosphate products and biopsy notable for vascular calcification, vessel thrombosis, and epidermal necrosis. After careful consideration at Dermatology Grand Rounds, calciphylaxis was felt to be unlikely.</li> <li>• Rheumatology was consulted to evaluate for autoimmune and small-vessel vasculitis. Rheumatology ruled out vasculitis, antiphospholipid antibody, and cryoglobulinemia, and concluded it was likely an arterial thrombotic disorder secondary to his hepatocellular carcinoma.</li> <li>• Infectious Disease was consulted for concern of marantic endocarditis given his history, and new <i>Morganella morganii</i> bacteremia, thought to be secondary from his necrotic fingers. He was then started on cefepime.</li> <li>• The patient went for upper extremity angiography that showed arterial occlusive disease of the hand consistent with atherosclerosis seen in ESRD.</li> <li>• The discovery of a splenic infarct, portal vein thrombosis, and penile necrosis led to a goals of care discussion with subsequent transition to comfort cares and hospice.</li> </ul> <p>Discussion: AVN is most commonly associated with smoking, autoimmune connective tissue diseases, vasculitis, and local injuries. Malignancy is a far less common cause of AVN with a prevalence of 2.2%-8% of cases and predisposes patients towards a multifactorial-induced hypercoagulable state. The low incidence of malignancy-associated AVN often presents a diagnostic challenge which can delay appropriate management and thus warrants an early multidisciplinary approach. This patient was ultimately found to have malignancy associated AVN secondary to his hepatocellular carcinoma which is one of the rarer potential differential diagnoses.</p>

<p><b>Emily Westergard</b> Dr. Amy Holbrook</p>	<p><i>The ABVD's of Drug Induced Pulmonary Toxicity</i></p> <p>Introduction: In the treatment of Hodgkin's Lymphoma, ABVD therapy has long been considered the gold standard of therapy. Though effective, this regimen does not come without risk.</p> <p>Case Presentation: This poster presents the case of a seventy-eight year old male with history of diffuse large B cell lymphoma, ischemic cardiomyopathy, hypertension, chronic lymphocytic leukemia, hyperlipidemia, chronic atrial fibrillation, newly diagnosed Hodgkin's Lymphoma, and history of tobacco use who was admitted with weakness and shortness of breath. Patient had recently been diagnosed with stage 4B Hodgkin's Lymphoma in April 2018. He was started on adjuvant chemotherapy with ABVD (adriamycin, bleomycin, vinblastine, dacarbazine) therapy minus adriamycin (held due to systolic cardiomyopathy). He had completed 3.5 cycles of BVD therapy at time of hospital admission, with last chemotherapy dose held due to neutropenia, for which he received G-CSF. Throughout hospital admission, respiratory status continued to decline despite steroids and diuresis. Differential initially included pneumonia, PE, acute CHF exacerbation, and drug induced toxicity. Ultimately, with assistance of pulmonology and oncology, he was diagnosed with drug induced pulmonary toxicity secondary to bleomycin, which unfortunately led to his demise.</p> <p>Discussion: Bleomycin induced pulmonary toxicity is a rare, but often fatal complication of a frequently used chemotherapy agent. Patients should be screened for risk factors prior to initiation. Additionally, clinicians should watch closely for evidence of this disease process, as treatment for bleomycin induced drug toxicity has several nuances which are further discussed within this presentation.</p>
<p><b>Thomas Williams</b> Dr. Peter Lund</p>	<p><i>AL Amyloidosis Presenting as Cardioembolic Stroke</i></p> <p>Case Presentation: A 49-year-old male presented with acute left hemiparesis, dizziness, and speech difficulty. Though he was healthy years ago, this symptom cluster represented his 6th presentation with a new medical issue over the past year, each requiring a new specialist, and each seemingly unrelated.</p> <p>One year prior he developed bilateral median neuropathy secondary to carpal tunnel syndrome, initially attributed to repetitive motions as a pilot. Orthopedics performed bilateral carpal tunnel release surgery following failed conservative therapy. Subsequently he developed oropharyngeal dysphagia evaluated by gastroenterology with negative esophagram, EGD, manometry and limited improvement with empiric therapy for GERD. He then had dysphonia, recurrent "pneumonia", and associated weight loss. Over the course of a year of declining health he was evaluated by multiple subspecialists for discrete localized symptoms, but a more global diagnosis remained elusive.</p> <p>Following presentation for hemiparesis, brain MRI confirmed new right-sided frontoparietal infarct. Though arrhythmia evaluation and cerebrovascular imaging was unremarkable, follow-up transthoracic and transesophageal ECHO showed 47% EF with severely increased wall thickness suggesting infiltrative cardiomyopathy. Cardiac MRI revealed extensive cardiac amyloidosis. Bone marrow biopsy demonstrated 30-40% monotypic plasma cell population and urine and serum free light chain analyses demonstrated overproduction of lambda light chains, serum kappa lambda ratio of 0.01. Fat pad biopsy showed amyloidosis, confirmed with Congo Red stains. Further evaluation demonstrated hepatomegaly, and elevated troponin, BNP, beta-2 microglobulin, and urinary protein. Creatinine, hemoglobin, and calcium were all relatively unremarkable. The patient was diagnosed with extensive systemic AL amyloidosis secondary to lambda light chain multiple myeloma.</p> <p>Discussion: This case demonstrates how easily AL amyloidosis can be overlooked, and illustrates import concepts that make the internist well-suited to make this diagnosis. In the appropriate clinical setting, a high index of suspicion and systematic</p>

	<p>approach is needed in order to arrive at timely diagnosis. This requires a refined understanding of its many possible manifestations, of which carpal tunnel syndrome is not uncommon. Though attributable to plasma cell dyscrasias, only 10% of AL amyloidosis cases concomitantly meet diagnostic criteria for multiple myeloma with myeloma related events, making CRAB criteria insensitive for diagnosis. A less well-known fact of amyloid cardiomyopathy is its propensity for cardiac thromboembolism with up to 50% of AL amyloid hearts harboring intracardiac thrombus on autopsy studies, 27% on transesophageal ECHO. Anticoagulation is appropriate presumptive therapy for embolic ischemic stroke of undetermined etiology in amyloid cardiomyopathy. Finally, cardiac involvement in AL amyloid is a strong prognostic factor, with several prognostic algorithms involving BNP and troponin levels as central measures to predict survival. This study will review these salient details to better equip us for this elusive diagnosis.</p>
<p><b>Katrina Williamson</b> Dr. Denise Dupras</p>	<p><i>Renal Infarction and Bilateral Limb Ischemia: A Unique Presentation of Essential Thrombocythemia</i></p> <p>Introduction: Essential thrombocythemia (ET) is an important diagnosis to consider in patients presenting with venous or arterial thrombosis. Thrombocytosis may be missed in the setting of platelet consumption or mistakenly attributed to a reactive state. ET patients with history of thrombosis are at high risk of recurrent clotting events and may require a combination of anticoagulation, antiplatelet therapy, and cytoreductive therapy.</p> <p>Case Presentation: A 48 year-old man with active tobacco and alcohol abuse presented to the emergency department for two days of severe left side abdominal pain and bilateral leg paresthesias. Vitals were normal aside from systolic hypertension. Exam demonstrated abdominal tenderness, diminished femoral and absent pedal pulses, and decreased sensation below the knees bilaterally. Notable labs included leukocytes 14.5 x 10<sup>9</sup> cells/Liter, platelets 334 x 10<sup>9</sup> cells/Liter and creatinine 1.43. CT abdomen/pelvis demonstrated hypoenhancement of the left renal parenchyma consistent with infarction, severe narrowing of the abdominal aorta, and severe stenoses of the common femoral arteries. Treatment was initiated with intravenous heparin for acute limb ischemia. Subsequent CT angiogram showed mural thrombi of the ascending aorta and occlusion of bilateral tibial vessels. The patient underwent bilateral lower extremity thromboembolectomy and endovascular aortic and bi-iliac artery stenting and was subsequently placed on warfarin and antiplatelets. The patient had persistent thrombocytosis, for which JAK2 mutation testing and bone marrow biopsy were completed. These showed presence of JAK2 V617F mutation and increased megakaryocytes with occasional large hyperlobulated forms, consistent with a diagnosis of essential thrombocythemia.</p> <p>Discussion: Essential thrombocythemia is a chronic myeloproliferative neoplasm defined by clonal platelet production. Most often, this is driven by mutations in JAK2, CALR, or MPL. It is often diagnosed incidentally. However, symptomatic patients may present with vasomotor symptoms (headache, dizziness, vision changes), thrombosis, hemorrhage, or even pregnancy loss. Those with thrombosis may present with stroke, MI, DVT/PE, or, as in this case, have multiple arterial thrombi with organ infarction and critical limb ischemia. ET patients with leukocytosis and JAK2 V617F mutation are at greater risk of arterial clots, as are those with active smoking, hypertension, and diabetes. Given the overlap with atherosclerotic risk factors, providers must be careful to consider underlying prothrombotic pathology such as ET before attributing arterial events to atheroembolic disease. It is important that ET be recognized since reducing risk of recurrent clots requires appropriate anticoagulation and antiplatelets (if arterial thrombosis is present) as well as hydroxyurea to reduce the platelet count. Managing modifiable cardiovascular risk factors also becomes tremendously important.</p>
<p><b>Mung Ting Yung</b> Dr. Laura Katkish</p>	<p><i>If at First Biopsy You don't Succeed, Try, Try Again: A Case of Skull Base Osteomyelitis</i></p> <p>Introduction: Skull base osteomyelitis (SBO) is a rare, potentially fatal disease. Typical cases of SBO (lateral SBO) are due to <i>Pseudomonas aeruginosa</i>, involve the temporal bone, and are a complication of otitis externa in immunocompromised patients. Central skull base osteomyelitis (cSBO) is even rarer and involves the sphenoid or occipital bone, often the clivus, without preceding otitis externa. cSBO</p>

	<p>can present with only headache; facial pain and cranial nerve deficits can also occur, but fever is uncommon. Classic MRI findings involve clival bone marrow T1 hypointensity and preclival soft tissue infiltration. Even with treatment, mortality rate is 10-20%, with long-term neurologic sequelae in up to 31% of patients.</p> <p>Case Presentation: A 68 year old man with history of polyarteritis nodosa on azathioprine/prednisone presented with four months of headache, hearing loss, and encephalopathy. Work-up included CT and MRI head/neck which revealed an ill-defined soft tissue mass within the posterior nasopharynx, extending into adjacent bony structures with areas of focal lytic destruction, concerning for malignancy. Besides elevated CRP/ESR, blood and CSF studies were negative. Biopsy of the mass preliminarily suggested lymphoma and patient was discharged with oncology follow up; however, final results showed only chronic inflammation without malignancy. Progressive symptoms prompted readmission, again with elevated CRP/ESR but otherwise negative work-up. A second biopsy of the mass showed only focal fibrosis suggestive of prior biopsy. Subsequent studies included WBC scan, which showed no abnormal uptake to suggest infection, and PET/CT scan which showed marked FDG activity associated with the mass, "consistent with neoplasm" per report.</p> <p>After interdisciplinary team discussions, ENT performed a third biopsy of the mass, this time also obtaining a sample of the clivus. Bone culture ultimately grew <i>Pseudomonas aeruginosa</i>, <i>Kocuria kristinae</i>, and MRSE, confirming diagnosis of osteomyelitis. IV ceftazidime was started, then broadened to daptomycin and meropenem. Repeat MRI brain showed mild improvement in bony involvement and stable to slightly improved abnormal soft tissue. He completed 6 weeks of IV antibiotics with improvement in cognition, downtrending CRP/ESR, and resolution of headache. He remains on suppressive therapy with PO levofloxacin per Infectious Disease.</p> <p>Discussion: cSBO is a difficult diagnosis to make, as biopsies may show only non-diagnostic inflammation, and lytic bone destruction and soft tissue involvement on imaging may lead to incorrect diagnosis of malignancy. In this case, inability to confirm cSBO by obtaining a bone sample delayed treatment by several weeks. Treatment is with antibiotics for 1-6 months; in some cases, adjuvant hyperbaric oxygen or surgical debridement is used. Obtaining appropriate tissue samples is critical for correct diagnosis of cSBO and it should remain on the differential for immunosuppressed patients presenting with headache and suggestive MRI/CT findings, even in the absence of sepsis.</p>
<p><b>Margot Zarin-Pass</b></p>	<p><i>Look in the Mirror: It's Never Carcinoid Syndrome</i></p> <p>Introduction: We are biased toward developing unifying diagnoses, as we are trained that these are the most likely and simplest explanation. However, in a well appearing ambulatory patient, it is more likely that a patient has multiple causes of common symptoms than one rare unifying cause of several common symptoms.</p> <p>Case Presentation: A 44 year old woman with history of ADHD, Irritable Bowel Syndrome with Diarrhea (IBSD) and hypothyroidism presented with 3 weeks of skin flushing, profuse watery diarrhea, and extreme fatigue. Prior to this episode her chronic conditions had been stable for many months, and at baseline she had 2 loose stools per day. The episodes were debilitating – she went from being active and employed mom of three to being nearly bedridden because of fatigue and frequent bathroom visits. The flushing was profound and she showed videos of herself turning bright red in the face and then back to normal skin tone within minutes.</p> <p>Urine 5-HIAA to creatinine ratio was normal, making carcinoid syndrome extremely unlikely. Thyroid studies were notable for a TSH of 7.44 (3 months prior it had been 1.92) with a normal T4 despite no recent change in levothyroxine dosing. On chart review, the patient had recently started colestipol therapy for her IBS-D. She reported recently switching her schedule to taking both medications at the same time. Colestipol is a binding agent and can interfere with levothyroxine absorption, which is our theory in this case.</p>



	<p>The patient started an increased dose of levothyroxine and staggered it with her colestipol. Her fatigue and thyroid labs improved. She began a FODMAP limiting diet and her diarrhea improved. The flushing resolved during this time as well.</p> <p>Discussion: This case highlights some common themes in primary care medicine – rare diagnoses are rare, medication interactions are common, and sometimes symptoms resolve on their own without explanation.</p>
<p><b>Diana Zychowski</b> Dr. Ryan Kelly</p>	<p><i>Ocular Syphilis, Often Overlooked</i></p> <p>Introduction: The incidence of primary and secondary syphilis is on the rise. Neurological and ocular manifestations can occur at any stage, however these symptoms are not reliably reported in national surveillance systems. While uveitis is the predominant manifestation of ocular syphilitic disease, <i>Treponema pallidum</i> can also present with keratitis, retinitis, or vasculitis.</p> <p>Case: A 75 year-old female with a history of allergic rhinitis presented to primary care clinic with bilateral ophthalmalgia and conjunctival hyperemia without change in visual acuity. Her symptoms previously waxed and waned for three years but worsened acutely over the past two weeks. A presumptive diagnosis of acute bacterial conjunctivitis was made and she was prescribed ofloxacin drops. Her symptoms persisted and she was seen three days later by ophthalmology. Slit lamp examination showed severe bilateral conjunctival injection and bilateral corneal degeneration with peripheral keratinization. Intraocular pressure was 14mm Hg OU (ref: 10-21mm Hg). Visual acuity OD: 20/200, OS: 20/40. Funduscopic exam was normal. Diagnosis at that time was concerning for peripheral corneal degeneration versus ocular rosacea. She was prescribed artificial tears, ophthalmic cyclosporine, and prednisolone drops. Serial exams over several weeks noted only mild improvement, and the patient was referred to outpatient rheumatology for autoimmune causes of scleritis. ROS was negative for fevers, fatigue, dry mucous membranes, joint pains, skin rash or ulcers. Outside of ocular findings, physical exam was unremarkable. CBC and CMP were normal. <i>Treponema</i> antibodies were positive, but rapid plasma reagin (RPR) returned negative. A confirmatory test, <i>Treponema pallidum</i> particle agglutination assay, was positive, establishing the diagnosis of ocular syphilis. Of note, she had no prior treatment, history of genital lesions, or known exposure to a partner with syphilis. She was treated with a 14 day course of IV Penicillin G.</p> <p>Discussion: Ocular syphilis continues to pose a challenge due to the disease's ability to mimic a wide spectrum of illness leading to misdiagnosis and delay in treatment. Further complicating a diagnosis is the nuanced testing. <i>T. pallidum</i> cannot be cultured and must be identified by direct visualization or serological detection. The traditional screening algorithm starts with a quantitative nontreponemal test such as RPR, and is followed by a specific treponemal test for confirmation. Many high-throughput labs are now using reverse testing. Nontreponemal tests have reduced sensitivity in late latent syphilis and can also yield a false-negative result when high concentrations of antibodies interfere with target antibodies. This phenomenon, known as a prozone reaction, can be mitigated by dilution. Traditional testing in our case would have stopped at the negative RPR and thus missed the diagnosis. The keen clinician must therefore be mindful of not only the diagnosis but also the method of detection.</p>