



**American College of Physicians- Minnesota Chapter Annual Abstract Competition**  
**Virtual Poster Session**  
**November 7, 2020**  
 Abstracts Submitted for Competition

<b>Medical Students</b>	
<b>Quality Improvement - Medical Students</b>	
<b>Austin Pickup</b> Dr. Smarika Sapkota Emily Stupca Baila Elkin	<p><i>Reducing Readmission by Improving Post-Discharge Follow Up</i></p> <p>Introduction: A multi-step quality improvement project was conducted to evaluate a cohort of patients who were discharged to home from the University of Minnesota Medical Center (UMMC) unit 5A and 5B and were readmitted within 30 days to identify gaps in care leading to readmission and formulate an intervention. The overall purpose was to reduce the readmission rate for patients with a discharge diagnosis of severe sepsis.</p> <p>Methods: Patients who were discharged from unit 5A/5B and readmitted within 30 days from January 2018 to February 2019 were identified. Information on index admission was ascertained from the electronic medical record. Information was gathered on primary care provider (PCP) follow up recommendation, appointment scheduling, and appointment attendance. Our intervention included incorporating questions about PCP follow-up in the post-discharge call and connecting with primary care to ensure PCP follow-up. Plan, do, study, and act (PDSA) improvement method was used for this quality improvement project and post-intervention data on readmission and post-discharge PCP follow-up were calculated.</p> <p>Results: 30-day readmission rate from January 2018 to February 2019 for 5A/5B unit discharges was 23.0% for patients who were discharged to home. Multiple causes for readmission were found based on chart review. The discharge diagnoses of severe sepsis had the highest rate of readmission when compared to other discharge diagnoses, with 24.8% of patients readmitted within 30 days. When these patients were evaluated, the PCP follow-up process was found as the main focus for intervention since this was one of the avoidable causes of readmission. 81% of the patients had PCP follow-up recommended on discharge, 50% had PCP follow-up scheduled within 14 days and only 44% had followed up with PCP within 14 days of discharge. Intervention to incorporate questions regarding PCP follow up and to coordinate PCP follow up with the clinic if not scheduled was conducted during August-November 2019. Overall, there was a reduction in readmission rate to 12.5% (7/56) after the intervention from 24.8% which was statistically significant with a p-value of 0.029. There was also an increase in PCP follow-up from 44% to 48.2% (27/56) which was not statistically significant with a p-value of 0.9.</p>

	<p>Conclusion: Interventions to improve post-discharge follow-up with PCP have been utilized to reduce readmission rate for high-risk patients. In this project, although there was a reduction in overall readmission, there was no difference in PCP follow-up after the intervention. This indicates there are other factors also impacting readmission rate. Although the process measure was not statistically different after the intervention, this project revealed discharge location, discharge diagnoses, and multiple causes that have an impact on readmission rate.</p>
<p><b>Paul Strain</b> Regina Martinez Lorenzo</p>	<p><i>Impact of Student Deployment to MDH for Case Investigation and Contact Tracing During COVID-19 Pandemic</i></p> <p>Background: The COVID-19 pandemic altered medical training in March of 2020—remote coursework and virtual rotations were quickly created and administered to ensure students could make adequate progress towards graduation and complete career discernment. Initially, the American Medical Association suggested trainees could be used on a volunteer basis to help efforts in pandemic management including: phone triage, child care assistance, indirect patient outreach, research, and test center contact. However, months into the pandemic, it became clear that the nation’s public health capacity to respond to COVID-19 was taxed. Public health departments continue to face increasing cases to track as stay-at-home orders are lifted. Additionally, increasing cases were disproportionately affecting hospitalists and intensivists caring for hospitalized COVID-19 patients.</p> <p>We sought to explore ways to expand and formalize medical student and graduate public health student collaborations with the healthcare system and state public health efforts. Therefore, in collaboration with faculty at the University of Minnesota and staff at the Medical Reserve Corps (MRC), we designed a deployment for medical students and graduate public health students with the Minnesota Department of Health (MDH) on COVID-19 case investigation and contact tracing efforts.</p> <p>Objective: To create a model for deploying medical students and graduate public health students into case investigation and contact tracing efforts for COVID-19 at the Minnesota Department of Health and evaluating the impact of that deployment.</p> <p>Methods: With indirect supervision, third- and fourth-year medical students and graduate public health students assisted in 4 6-hour case investigation and contact tracing shifts per week for four weeks to assist in the MDH efforts to investigate COVID-19 cases throughout the state in order to prevent the ongoing spread of disease. To ensure data privacy is followed and that students are protected, students were required to complete a brief orientation with MDH prior to the start of the deployment and were asked to register with the University of Minnesota MRC. We will evaluate the impact of the deployment from both the student and MDH staff perspective using two Qualtrics survey instruments to determine the strengths, weaknesses, and opportunities for future deployments and partnerships with MDH public health efforts.</p> <p>Results: This study has received Institutional Review Board approval from the University of Minnesota in September 2020 to conduct evaluation surveys of students and MDH staff. We are currently in the process of collecting data. Preliminary data should be available by the time of the ACP Minnesota Internal Medicine: 2020 - Virtual Meeting.</p>

	<p>Conclusions: It is hypothesized that this deployment will positively impact both medical student and graduate public health student education and the MDH case investigation and contact tracing efforts during the COVID-19 pandemic. We also anticipate that this deployment will serve as a model for future partnership with MDH during pandemics or other public health crises.</p>
<p><b>Research - Medical Students</b></p>	
<p><b>Ainslee Crose</b> Stephanie Singroy, Qi Wang, Alison Alvear</p>	<p><i>Quality of Life Measures in Time-Restricted Eating</i></p> <p>Introduction: Time restricted eating (TRE) reduces weight in humans, yet its effects on quality of life have not been well characterized. Using a randomized clinical trial, we examined the effects of TRE (12 week intervention, 8 hour eating window) vs non-TRE (unrestricted eating) on quality of life measures in overweight adults without diabetes. We hypothesized that TRE improves quality of life (QoL) measures more than non-TRE.</p> <p>Case Presentation: 20 subjects with overweight [17F/3M, mean (SD), age: 45.5 years (12.1)], BMI [34.1 kg/m<sup>2</sup> (7.5)], and prolonged eating window [15.4 hours (0.9)] were randomized to either 12 weeks of TRE [8 hour eating window: n=11] or non-TRE [n=9]. QoL data was collected with the 36-item Short Form Survey (SF-36) pre and post intervention.</p> <p>TRE group improved several SF-36 derived QoL measures relative to baseline and to the non-TRE group. The TRE group improved limitations due to emotional health from an average score of 66.7 to an average score of 97.0 and perceived change in health over the last year from an average of 52.3 to 68.2 (all p &lt; 0.05). In contrast, in the non-TRE group the changes from baseline were not significant. Limitations due to emotional health changed from 63.0 to 55.6 and perceived change in health from 41.7 to 44.4. The improvements in limitations due to emotional health and perceived change in health post-intervention were significant when comparing the TRE group to the non-TRE group (p &lt; .05). There were no significant differences between the non-TRE and TRE groups among the other domains of QoL including physical functioning, role limitations due to physical health, energy/fatigue, emotional well-being, social functioning, pain, and general health; these measure also did not significantly change relative to baseline. Within the TRE group, there was no correlation between change in the domains of QoL, eating window restriction, or extent of weight loss.</p> <p>Results: Among these study participants, TRE improved several SF-36 derived QoL measures; these changes were significant relative to baseline and non-TRE. Further investigation is needed to determine the durability of these QoL changes, whether this translates into sustainability of TRE beyond 12 weeks, and comparison relative to standard-of-care caloric restriction.</p>
<p><b>Laura Maciejko</b> Dr. Jean Fox Michelle Steffens Paul Decker Dr. Young Juhn Dr. Chung Wi Dr. Christi Patten Dr. Pamela</p>	<p><i>Unmasking motivators and barriers to wearing a mask to prevent the spread of COVID-19 in Southeastern Minnesota</i></p> <p>Introduction: The COVID-19 pandemic drastically changed U.S. preventative health measures, introducing the novel behavior recommendation to wear face masks in public. Experts suggest community-wide mask wearing minimizes droplet transmission, thereby potentially reducing deaths and economic losses. Mask wearing is an inexpensive and low-risk measure, but its efficacy in</p>

<p>Sinicrope</p>	<p>disease prevention requires community cooperation, buy in, and/or political mandates, all of which have become controversial within the U.S. Our goal is to identify critical gaps in understanding of motivators and barriers to wearing a mask to prevent the spread of COVID-19, and identify opportunities for education, awareness, and intervention.</p> <p>Methods: A community-based cross-sectional, anonymous survey of social cognitive theory-based motivators and barriers to wearing a mask. The study was reviewed by the Mayo Clinic IRB and deemed exempt. The brief (about 15 min.) survey was offered in English, Spanish, or Somali to adults aged 18+ residing in Southeastern Minnesota from August 4-September 4, 2020. The survey was administered while there was a state mask mandate. The primary outcome measure was: “When out in public, how willing or not willing are you to wear a mask to stop the spread of Coronavirus?” with a 4-point response options from ‘not at all willing’ to ‘very willing.’ Additional variables included: sociodemographics (e.g. age, gender, rural/urban), perceived sense of community, COVID-19: knowledge, stress, experience and perceived severity, and mask-wearing: outcome expectations, barriers, and self-efficacy. Comparisons across groups were made using chi-square tests (Fisher’s exact) and two-sample t-tests/analysis of variance (Kruskal-Wallis) as appropriate.</p> <p>Results: The sample consisted of 7793 respondents [78% women, 51% rural location with age groups: 18-29 (14%), 30-39 (26%), 40-49 (23%), 50-59 (18%), and 60+ years old (19%)]. Among all, 9% reported being ‘not at all willing’ to wear a mask, 28% reported being ‘somewhat willing/willing’ and 64% reported being ‘very willing’ Men, rural residents, and those under age 59 were less willing to wear a mask (<math>p &lt; 0.001</math> in each cases). Those less willing to wear a mask reported a lower sense of community belonging, lower levels of COVID-19 knowledge, experience (personal or through friend/family), and perceived severity and distress. They also reported lower self-efficacy for wearing a mask, and more negative outcome expectations for wearing a mask. Concerns and barriers to wearing a mask included: foggy glasses, too hot, and trouble understanding what people are saying.</p> <p>Conclusion: Despite a statewide mask mandate, there are concerns and barriers to masking. Intervention opportunities are apparent using our social cognitive framework. There is a need to focus on men, rural residents, and those younger than 59 in developing mask wearing interventions. Additional indications include decreasing practical barriers to wearing a mask, improving masking self-efficacy, and cultivating a greater sense of community belonging.</p>
<p><b>James Markos</b>  Dr. Robin Molella  M. Hassan Murad  Dr. Shari Bornstein</p>	<p><i>Collaboration of Medicine and Public Health: An Assessment of Clinicians’ Public Health Knowledge, Education, and Engagement</i></p> <p>Background: For decades, medicine and public health have operated in separate silos with less than ideal collaboration. Many organizations have called for closer partnership between the two fields, but progress towards integration has been slow. Despite the importance of physicians understanding key public health structures and functions, the baseline public health knowledge of physicians has not been evaluated, and there exists no established instrument to do so. To address this gap, we surveyed clinicians regarding knowledge of public health fundamentals, functions, and federal health programs, as well their level of engagement with local public health entities and prior public health education.</p>

	<p>Methods: Four key domains were surveyed: (1) knowledge of public health concepts and functions, (2) knowledge of health systems and policy, (3) personal engagement with public health, and (4) prior public health education and interest in CME. Questions for the knowledge domains were based on accepted public health competencies. A pilot survey was trialed on a group of internal medicine and preventative medicine attendings, and final survey questions were finalized after feedback. The paper survey was administered to attendees of two general medical conferences in 2019, prior to the COVID-19 pandemic.</p> <p>Results: Responses were received from 102 of 402 attendees (response rate = 25.4%). The majority were male (56%), over the age of 50 (51%), and physicians (86%). Respondents demonstrated good knowledge of the Affordable Care Act (84% correct) and Medicare (74%) but poorer regarding Medicaid (45%). Knowledge of public health concepts was good, including social determinants of health (88%), the CDC (80%), and surveillance (91%). State and local health department activities were each correctly identified by 84%. Less than half (45%) had a personal interaction with their local health department in the last two years, and 46% were aware of opportunities to work with public health organizations in their community. Very few respondents rated their public health education during primary degree training or graduate medical education as good or excellent, 7% and 15%, respectively. Most (75%) were interested in public health and health policy CME.</p> <p>Conclusion: We discovered good overall knowledge of federal health programs, policy, and public health concepts among clinicians, with some gaps identified. Clinicians overwhelmingly perceive their prior public health education as poor, and many express interest in CME on these topics. Due to the COVID-19 pandemic, current engagement with public health structures is likely much higher than when our survey was administered, which presents an opportunity to improve future collaboration.</p>
<p><b>James Markos</b> Bradley Alexander Alexandra Arguello Blake Sowers Gerald McGwin Aaron Owen Brent Ponce Norman Turner</p>	<p><i>Characterizing COVID-19 Disruptions in the 2020-2021 Residency Application Cycle: A Survey of Fourth-year Medical Students</i></p> <p>Background: The COVID-19 pandemic has disrupted medical education at all stages. Fourth-year medical students face particular uncertainty as they navigate a 2020-2021 residency application cycle complicated by novel pandemic-related obstacles. Our study sought to characterize the COVID-19 related challenges facing U.S. senior medical students applying to residency in the 2020-2021 cycle.</p> <p>Methods: A survey was created by medical students and program directors regarding key aspects of the ERAS application form and residency matching process affected by the COVID-19 pandemic. After securing Mayo Clinic IRB approval, the anonymous online survey was administered to fourth-year medical students via student affairs contacts at United States medical schools. Students not applying to residency during fall 2020 were excluded. Data were collected from May 15th to June 7th, 2020.</p> <p>Results: Responses were received from 749 medical students applying to all major specialties. Of those surveyed, 31% will have one or more incomplete core clerkships when applying, and 60% saw their USMLE Step 2 CK exam canceled or rescheduled. Fifty-seven percent of participants had accepted or</p>

applied to an away rotation prior to rotations being canceled. The inability to interview in-person was ranked as the most negative pandemic-related change and viewed as a major (64%) or moderate (28%) inhibition to determining overall fit with programs. Nearly all respondents (98%) expressed concern regarding the pandemic's impact on their application. As a result, 79% of respondents plan to increase the number of residency programs to which they apply and 70% to rank more programs. Additionally, 10% of respondents are considering changing their desired specialty altogether due to COVID-19. Uniformly, osteopathic students experienced greater disruptions than allopathic students.

Conclusion: We identified substantial pandemic-related disruptions in the 2020-2021 residency application cycle. Recently-issued guidelines promote flexibility of application requirements, but applicants still express a high level of concern due to incomplete application components, the absence of away rotations, and transition to virtual interviews. Despite calls for application and/or interview limits, students plan to apply to and rank a higher number of programs. Our findings could inform the creation of subsequent guidelines.

**Clinical Vignette - Medical Students**

**Kristin Chu**  
Dr. Ryan Kelly

*Beethoven's Unusual Symptom-onies: A Case of Ludwig's Angina*

Introduction: Ludwig's angina is a severe, diffuse cellulitis with an acute, rapid onset that can affect the submandibular, sublingual, and submental spaces—submandibular being the primary site of infection (1). Most cases are odontogenic in etiology. Prior to the age of accessible antibiotics, mortality was greater than 50%; though improved, this diagnosis still portends a mortality rate around 8% (2-3). Serious complications can result from inadequately managed symptoms including airway obstruction, mediastinitis, pneumonia, and cervicofacial necrotizing cellulitis.

Case Presentation: A previously healthy 23 year old female presented to an outpatient dental clinic with 5 days of facial swelling and pain. She was diagnosed with a left lower tooth abscess and prescribed 10 day of Augmentin. Two days later, she returned to the dental clinic due to progressive swelling, erythema, dysphonia, and dysphagia. Suspecting a worsened soft tissue infection, the dental team referred her to be seen in primary care clinic the same day. She was given a dose of Augmentin and immediately transferred to the ED for further evaluation.

On arrival to the ED, the patient was protecting her airway and saturating appropriately on room air. However, a fiberoptic laryngoscopy was performed urgently and revealed that the patient's airway was significantly edematous and narrowed. Due to concern for respiratory decompensation, the patient was intubated with some visualization difficulty due to significant edema of the posterior pharyngeal structures. CT soft tissue neck scan with contrast showed a 1.5 x 1.6 x 3.5 cm peripherally enhancing hypodense lesion at the floor of the mouth bilaterally consistent with Ludwig's angina. She was started on ampicillin-sulbactam and taken for I&D of the abscess, where two drains were placed for continuous drainage.

She remained intubated in the surgical ICU due to persistent airway swelling and placed on broad spectrum antibiotics (vancomycin / zosyn) for MRSA coverage and steroids for airway edema. Patient remained intubated until post-op day 4. One week after surgery, her swelling improved and drainage

	<p>abated, so the remaining drainage tube and submental sutures were removed. Cultures of the fluid obtained from incision and drainage eventually grew <i>Parvimonas micra</i> and <i>Prevotella buccae</i>. The patient was discharged to complete a 9 day course of Augmentin with close follow-up with her dental and primary care provider.</p> <p>Results: This case highlights how common presentations such as dental abscesses, can result in rare but deadly complications like Ludwig’s angina. A keen eye can prevent a delayed diagnosis so that providers can initiate life-saving treatment early on, thus the importance of spreading awareness and educating medical professionals to be vigilant about including rare conditions on their differentials, even when caring for young patients. Other key mentions are the importance of timely airway protection, surgical intervention and antibiotic administration which can significantly reduce mortality rate (4). *Of note, unable to attach references with abstract due to word count restriction.</p>
<p><b>Michelle Kihara</b> Dr. Sharon Li Dr. Melissa Plesac</p>	<p><i>Nutritional Deficiency: The case for patient follow-up after Roux-en-y Gastric Bypass</i></p> <p>Introduction: Roux-en-y gastric bypass (RYGB) is a procedure that alters the anatomy of the gastrointestinal system in order to induce nutrient malabsorption. While malabsorption facilitates the goal of weight loss, it places patients at high risk of developing nutritional deficiencies. Despite the importance of continued nutritional supplementation after the procedure, patients are often lost to follow up. A study comparing nutrient supplementation at two years and 10 years post-RYGB found that reduced time since most recent bariatric surgery clinic follow-up was an independent predictor of improved multivitamin use.</p> <p>Case Presentation: The patient is a 36 year old female with past medical history of RYGB, pulmonary embolism, CKD III, and non-alcoholic steatohepatitis (NASH), who was admitted to an outside hospital for evaluation of one week progressive abdominal pain, vomiting, and a diffuse rash. The patient was noted to have a painful rash that had two components: scaly and erythematous on the perioral and extensor surfaces, and small bullae on the bilateral distal lower extremities. CT of the abdomen showed diffuse colonic wall thickening and hepatic steatosis. Colonoscopy with biopsy showed terminal ileum atrophy with increased intraepithelial lymphocytes and plasma cells. These findings in addition to a mildly elevated anti-nuclear antibody (ANA) were concerning for an autoimmune process, prompting transfer to our hospital for further evaluation by rheumatology.</p> <p>On admission, the exam was significant for hair loss, angular cheilitis, bilateral conjunctival injection, glossitis, livedo reticularis of the bilateral upper extremities, the rash as above, and bilateral lower extremity edema. The workup for systemic lupus erythematosus, antiphospholipid antibody syndrome and glucagonoma as the cause of suspected necrolytic migratory erythematosus was negative. Rheumatology was consulted and determined that the distribution of the rash, glossitis, anasarca with low albumin, and poor oral intake were more consistent with nutritional deficiencies than autoimmune disease. Dermatology determined that in light of the patient’s history of RYGB and likely malabsorption, the presentation was most consistent with acquired acrodermatitis enteropathica (zinc deficiency), vitamin A deficiency, multiple B vitamin deficiency, or essential fatty acid deficiency; punch biopsy confirmed that the dermatitis was secondary to</p>

	<p>nutritional deficiency. Ophthalmologic evaluation showed bilateral epithelial defects of the cornea and optic neuropathy consistent with A and B vitamin deficiencies. Labs were consistent with deficiencies in zinc, calcium, copper, vitamins A, B6, D, E, and hypoalbuminemia secondary to liver disease. Dietitian started the patient on mineral and multivitamin supplementation.</p> <p>Results: This case illustrates the complex clinical presentation that is consistent with ongoing malnutrition following RYGB. It is an example of the multidisciplinary approach required to promptly diagnose and treat nutritional deficiencies in the setting of other chronic diseases. Areas of ongoing inquiry include efforts to identify and reduce barriers to short term and long term follow up post RYGB procedure.</p>
<p><b>Keegan McCabe</b> Hannah Thiry</p>	<p><i>Interesting Case of Vasculitis</i></p> <p>Background: Vasculitis is a rare autoimmune process, where the immune system attacks vasculature. It is highly associated with anti-neutrophil antibodies. The cause of the autoimmune process is often unknown, but can happen because of an infection, medication, or another disease. A necrotizing vasculitis results and affects arteries, veins and capillaries, virtually throughout all major organ systems, and most prominently presents in the respiratory track, renal system, and digestive system.</p> <p>Case Presentation: A 71-year-old female with a history of superior mesenteric artery stenosis (stent placement 1 month prior), hypertension, atrial fibrillation, major depressive disorder, chronic abdominal pain w/ irritable bowel syndrome presented to the emergency department with a rash on bilateral lower extremities. The rash had been present for the last week and got progressively more swollen and painful. Patient also had diffuse abdominal pain and chronically dry eyes; both have been present for years. Physical exam was significant for non-palpable purpuric rash extending from the feet to upper thigh and 2+ left lower extremity edema. Epistaxis noted in nasopharynx. Left lower quadrant abdominal tenderness. Family history was significant for multiple siblings and mother who suffer from irritable bowel syndrome.</p> <p>Initial labs revealed microcytic anemia, elevated alkaline phosphatase, elevated ESR and elevated creatinine. Urinalysis revealed proteinuria and hematuria. Clinical presentation was suspicious for a vasculitis and biopsy was taken along with additional immunological studies. Elevated ANA. Negative C-ANCA, P-ANCA, anti-Rho, anti-La, chronic hepatitis, HIV, C3, C4. Biopsy showed a leukocytoclastic vasculitis with negative IgA immunofluorescence. Patient was started on low dose methylprednisolone prior to biopsy results and showed significant symptomatic improvement in rash, abdominal pain and overall constitution. Based on the negative serologic and immunologic studies and non-confirmatory biopsy, top on the differential is Polyarteritis Nodosa or seronegative granulomatosis with polyangiitis. Patient is being actively managed for their condition and will likely undergo renal biopsy for definitive diagnosis.</p> <p>Conclusion: Vasculitis can be a deadly disease if not treated within 1 year of symptom onset, and it can be challenging to make a diagnosis due to the vast possibility of clinical presentation. Because of the significant clinical improvement of patients' experience once starting glucocorticoids, it is advantageous to not delay treatment prior to confirmed diagnosis. As per vasculitis-treatment guidelines, we started the patient on glucocorticoids</p>



	<p>immediately, despite not having a confirmed diagnosis and the potential side effects associated with steroids. Per literature, there have been several cases of vasculitis with delayed treatment due to diagnostic difficulties. In our case, it was in the patient's best interest to begin treatment. Her improvement also allowed her treatment team to further support a diagnosis of vasculitis versus other etiologies.</p>
<p><b>Kayla Murphy</b> Baila Elkin</p>	<p><i>A Rare Case of Dementia in a Galaxy Fahr, Fahr Away</i></p> <p>Introduction: Fahr syndrome, or familial idiopathic basal ganglia calcification, is a rare neurodegenerative disorder presenting with neuropsychiatric features. We present a case that highlights the importance of keeping a broad differential diagnosis in the context of worsening or new clinical findings, despite a known comorbidity that can explain them.</p> <p>Case Presentation: A 64-year-old Veteran with a history of asthma, antisocial personality traits, polysubstance use, and recent homelessness was evaluated for cognitive decline and increasingly erratic behavior that started after his wife's death. His former diagnosis was an unspecified neurocognitive disorder possibly related to his history of substance use. His family was concerned by his continuing decline, urging him to have a thorough evaluation at the VA's geriatric research center (GRECC). Clinically, the patient exhibited slight right greater than left upper extremity asterixis, a fine postural tremor in the bilateral upper extremities, and slightly brisker reflexes on the right upper and lower extremities. MoCA was 17/30, previously 19/30 a year before, with low fluency and delayed recall. As part of initial and subsequent work-ups, B1, B9, B12, Hep B and C, HIV, RPR, MMA, arsenic, lead, mercury, copper, calcium, phosphorous, and TSH and were all within normal limits or negative. As part of the GRECC work-up, re-evaluation of previous imaging found bilateral thalamic and left basal ganglia calcifications. Despite the minimal movement symptoms, the characteristic imaging along with his cognitive decline with behavioral impairments led to a consensus diagnosis of Fahr Syndrome with polysubstance abuse as a secondary cause. Per chart review, management included helping the Veteran find a supportive, stable, and safe living environment in a long-term care facility, but no genetic counseling or medications were recommended due to the advanced stage of his disease.</p> <p>Discussion: The classic presentation of Fahr syndrome features movement disorders and neuropsychiatric symptoms, likely due to intracranial calcifications in the vasculature of the basal ganglia. Like other progressive, incurable neurodegenerative conditions, treatment is supportive and symptom based. This case demonstrates that Fahr syndrome should be considered in a patient presenting with neuropsychiatric symptoms, particularly when metabolic and infectious work-up are negative, and imaging does not support cerebrovascular disease or dementia. Substance use should not be regarded as the primary cause of neurological decline unless a thorough work-up and re-evaluation of previous imaging yield no diagnosis.</p>
<p><b>Simisola Odusanya</b></p>	<p><i>Leptospirosis: A Case of Sudden Multi Organ Failure in a Previously Healthy Male</i></p> <p>Introduction: Leptospirosis is a zoonotic blood infection caused by the bacteria <i>Leptospira Interrogans</i>. The organism infects a variety of animals, especially rodents and animals associated with farming. Humans represent incidental infection usually through work-related contact through skin or mucous membranes. The main occupational groups at risk are farm workers,</p>

	<p>field agricultural workers, plumbers, sewer workers, sanitation workers and military troops. Hawaii reports the highest number of cases in the United States, the WHO estimated that there are about 873,000 cases worldwide annually with 48,600 deaths</p> <p>Case presentation: A previously healthy 49-year-old male with no PMH, presented from OSH in septic shock and multi organ failure. Patient’s wife reported that he developed a headache, diffuse muscle aches and a fever to 102F a week before presentation, and subsequently SOB, hemoptysis, diarrhea, nausea, vomiting, conjunctival suffusion, and decreased urine output one day later. Labs were significant for elevated inflammatory markers and elevated creatinine. Social history significant for hobby farming. Patient was started on ceftriaxone and azithromycin. Emergent bronchoscopy at OSH showed diffuse alveolar damage. Respiratory status worsened, patient was intubated, started on CRRT for AKI and airlifted to UMMC.</p> <p>On arrival at UMMC physical exam was significant for profound jaundice, scleral icterus, and diffuse crackles, vital signs notable for hypotension, tachycardia, oxygen saturation of 82%, and fever at 102F. Inflammatory markers were elevated ESR 38, CRP 328.4, and lactate 0f 3.9. Initial laboratory evaluation revealed marked elevations in his transaminases, ALT 200, AST 500, anemia (Hgb 9), thrombocytopenia (platelet 18), leukocytosis (WBC 29K), elevated D-dimer, elevated creatinine 5, elevated CK of 10K and INR of 1.26. CXR showed diffuse interstitial opacities, bilateral effusions, and Echo showed LVEF of 35-40%, and diffuse hypokinesis. Antibiotics were broadened to vancomycin, fluconazole, doxycycline, zosyn, and patient was started on pressors for distributive shock. A comprehensive infectious workup was negative. Further investigation with Karius assay returned positive for <i>Leptospira Interrogans</i>. Patient’s clinical condition subsequently improved 10 days after admission, CRRT was stopped and he was started on hemodialysis. The patient had significant improvement in his kidney and liver function tests and was discharged with continuation on outpatient dialysis until complete renal recovery</p> <p>Discussion: This case illustrates the importance of physical exam findings in evaluation of a clinical presentation. Conjunctival suffusion is an important but frequently overlooked sign in leptospirosis. It occurs in about 55% of patients, and is not a common finding in other infectious diseases. Its presence in a patient with a nonspecific febrile illness should raise the possibility of leptospirosis. This case also highlights the importance of a broad differential, and social history when evaluating a patient with acute decompensation, and suspected infectious etiology. Finally, it demonstrates the importance of highly specific and sensitive diagnostic modalities such as the Karius assay, especially when the illness is characterized by nonspecific findings and a wide differential diagnosis with inconclusive preliminary results</p>
<p><b>Sujaytha Paknikar</b>  Dr. Nirosha Perera  Dr. Melanie White  Dr. Matthew Samec  Dr. Kerry Wright</p>	<p><i>Granulomatosis with Polyangiitis and Celiac Disease, an Unexpected Co-presentation</i></p> <p>Introduction: Granulomatosis with Polyangiitis (GPA) is a rare condition that typically causes immune mediated inflammation in the small vessels of the upper-airway, lungs, and kidneys. The majority of patients diagnosed with GPA have documented elevated Proteinase 3 (PR3) antibodies and positive c-ANCA. Patients typically present with a combination of B-symptoms, epistaxis, dyspnea, cough, hemoptysis, and hematuria. Abdominal involvement, bullous rash, and eosinophilia are uncommon in GPA, but we</p>

	<p>describe one such atypical presentation.</p> <p>Case Presentation: A 23 year old previously healthy female presented with 10 days of abdominal pain, nausea, and vomiting, with recent development of a purpuric and vesiculo-bullous rash involving the lips, feet, hands, mouth, buttock, and extensor surfaces. Abdominal CT revealed a segment of jejunal wall thickening. Labs were remarkable for WBC 13.8X10<sup>9</sup>/L, eosinophils 1.33X10<sup>9</sup>/L, CRP 89.6 mg/L, and D-dimer of 11,903 ng/mL. Creatinine was at baseline and urinalysis was unremarkable. Autoimmune workup was positive for PR3 antibody, c-ANCA, and tissue transglutaminase antibody IgA (11.7 U/mL). It was negative for ANA, myeloperoxidase antibodies, p-ANCA and cryoglobulins. Chest CT revealed bilateral, peribronchial, vascular, ground-glass opacities with surrounding consolidation. The patient developed a nonproductive cough and fever, so a bronchoscopy was performed which revealed progressive bloody aliquots of fluid and 64% hemosiderin laden macrophages, confirming diffuse alveolar hemorrhage. Dermatopathology of the left finger, hand, and arm biopsies returned with findings of neutrophilic papillitis and IgA stippling at the basement membrane on immunofluorescence microscopy, consistent with dermatitis herpetiformis secondary to Celiac disease, as well as leukocytoclastic vasculitis without evidence of eosinophil involvement, consistent with GPA. Due to continued abdominal pain and the CT finding of jejunal thickening, an extended endoscopy was performed which showed jejunal inflammation; biopsies were obtained revealing pathology consistent with dermatitis herpetiformis versus another autoimmune process. Celiac gene pair testing was performed and HLA-DQA1 and HLA-DQB1 variants associated with Celiac disease were present.</p> <p>In light of PR3/c-ANCA positivity and the above multi-organ findings, the patient was diagnosed with an atypical co-presentation of GPA and celiac disease. As such, the patient was treated with IV methylprednisolone 1g daily for three days, during which time she had rapid clinical improvement. The patient was started on rituximab induction therapy after a negative infectious workup and advised to maintain a gluten-free diet on hospital discharge.</p> <p>Discussion: Without intervention, the natural course of GPA portends a high degree of mortality; early diagnosis and treatment is crucial even with atypical presentations of the disease. In this case, unusual symptoms and co-occurrence of Celiac disease added to the diagnostic complexity. The goal of sharing this case is to provide a reference for other similar presentations to facilitate earlier diagnosis and treatment of a condition with potential for multi-organ involvement and high morbidity.</p>
<p><b>Chintan Patel</b> Dr. Paul Rajan</p>	<p><i>Pneumomediastinum in an IPF Patient in the Setting of Non-invasive Positive Pressure Ventilation</i></p> <p>Introduction: Pneumomediastinum is a rare pulmonary complication in those afflicted by idiopathic pulmonary fibrosis (IPF). This complication can be attributed to the rupture of honeycombing cysts in subpleural areas. In this Clinical Vignette, we discuss the role that non-invasive positive pressure ventilation, specifically a newly prescribed Trilogy machine, may have played in the development of pneumomediastinum and subcutaneous emphysema in an IPF patient.</p> <p>Case Presentation: A sixty-five-year-old female with a past medical history of IPF on 6L of home Oxygen, who recently started wearing a trilogy machine at home, presented with a two-day history of shortness of breath, neck pain,</p>

	<p>cough, and generalized weakness. The neck pain was described as a dull, non-radiating ache at the base of the neck that did not change with movement or palpation. On examination the patient had swelling on her face, neck and upper extremity with palpable crepitus. Fine crackles were heard diffusely on inspiration during auscultation. CT chest demonstrated large pneumomediastinum extending up into the neck and honeycombing suggesting interstitial fibrosis.</p> <p>Results: Pneumomediastinum occurs in about 5% of patients with IPF. It has been stated in the literature that pneumomediastinum is a potential predictor of mortality in IPF patients. It has been shown that most patients die shortly after the occurrence of pneumomediastinum in IPF patients. Importance of this rare case can be attributed to the fact that non-invasive positive pressure ventilation such as Trilogy should be avoided in patients with IPF, or patients should be warned to look for symptoms of pneumomediastinum when using non-invasive positive pressure ventilation to decrease the risk of mortality in such patients.</p>
<p><b>Archna Patel</b> Dr. Jaime de la Fuente Dr. Robert Kraichely</p>	<p><i>Melena, Dyspnea, and Dysphagia in a Cancer Survivor</i></p> <p>Case Presentation: A 68-year-old woman with medical comorbidities including coronary artery disease, hypertension, hyperlipidemia, right breast cancer s/p resection and radiation, double mastectomy, and remote history of lymphoma in her teens was admitted to the hospital after presenting with melena, progressive dyspnea on exertion, and weakness. The patient has been on the antineoplastic anastrozole for 5 years and denied NSAID use outside of her daily 325 mg aspirin. She quit smoking 35 years ago and does not drink alcohol.</p> <p>In addition, over the past month, she has had progressive dysphagia with solids greater than liquids with associated 13 pounds of unintentional weight loss due to decreased oral intake. She denied abdominal pain, emesis, fevers, night sweats, orthopnea, paroxysmal nocturnal dyspnea, and chest pain.</p> <p>Initial laboratory evaluation (reference ranges in parenthesis) was significant for a hemoglobin of 8.6 g/dL (11.6-15.0 g/dL), a leukocytosis of <math>12.0 \times 10^9/L</math> (<math>3.4-9.6 \times 10^9/L</math>), a mild thrombocytosis of <math>378 \times 10^9/L</math> (<math>157-371 \times 10^9/L</math>), an elevated BUN/creatinine ratio of 34, and elevated inflammatory markers. CT chest showed a large, mass-like lesion filling the majority of the esophagus, extending from the thoracic inlet to the gastroesophageal junction over a craniocaudal length of 15.7 cm. CT abdomen and pelvis were unremarkable.</p> <p>A large pedunculated mass was found initiating at the level of the hypopharynx off the right arytenoid, extending through the entire length of the esophagus, and protruding through the gastroesophageal junction. The lesion was causing partial obstruction of the esophagus. There was no active bleeding, but stigmata of recent bleeding was seen at the gastric portion of the lesion, likely from the mass prolapsing through the esophageal hiatus. Otolaryngology resected the mass through a trans-cervical approach and histology revealed fibroadipose tissue with atypical, hyperchromatic stromal cells scattered throughout that were positive for smooth muscle actin. There was also amplification of the MDM2 gene region, frequently identified in well-differentiated liposarcoma.</p> <p>Conclusion: Primary esophageal liposarcomas are rare, pedunculated,</p>

	<p>intraluminal lesions that usually arise from the cervical esophagus and account for 0.5% of all esophageal malignancies. Well-differentiated type is most prevalent and patients are often middle-aged males. As the tumor is pulled down the esophageal lumen with swallowing and peristalsis, it grows in length and most patients develop dysphagia. Diagnosis is difficult by biopsy, as it often requires surgical specimens to rule out liposarcoma. Diagnosis can be made by observing MDM2 amplification, which has frequently been identified in atypical lipomatous tumor/well-differentiated liposarcoma and dedifferentiated liposarcoma. These masses can be life threatening due to risk of asphyxiation by it refluxing from the esophagus into the trachea; therefore, removal should be performed in all cases. Although endoscopic approaches may result in lower morbidity, surgical approaches are more common due to the usually highly vascular nature of the tumor. Patients should be monitored long-term as there is potential for recurrence.</p>
<p><b>Archna Patel</b>  Dr. Chineze Akusoba  Dr. Zachary Yetmar  Dr. Hussam Tabaja  Dr. Audrey Schuetz  Dr. Michael Camilleri  Dr. Omar Abu Saleh</p>	<p><i>Mycobacterium marinum</i> presenting as progressive left arm skin lesions</p> <p>Case Presentation A 61-year-old woman from Texas presented with 5 months of a progressive, ascending, nodular left upper extremity rash following a knife injury to her left thumb while cutting fish. She subsequently developed a pustule at that site which she self-drained using a ‘flame-sterilized’ needle. Additional lesions ascended up her arm to the level of her elbow with associated erythema and serous drainage despite treatment with trimethoprim-sulfamethoxazole.</p> <p>Initial biopsy revealed neutrophilic dermatosis and despite steroid treatment, her disease progressed. Repeat biopsy 13 weeks later revealed neutrophilic microabscesses throughout the dermis and acid-fast bacilli. Acid-fast bacilli were recovered in the culture after 13 days. Colonies produced yellow pigment after exposure to light consistent with a photochromogen. Matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF MS; Bruker Biotyper) identified the isolate as <i>Mycobacterium marinum</i>. Laboratory evaluation was unremarkable. MRI of the left hand and forearm revealed soft tissue infection and tenosynovitis with no involvement of joints or bones. She was started on oral clarithromycin 500 mg twice daily, ethambutol 1200 mg daily, and rifampin 600 mg daily.</p> <p>Conclusion: <i>Mycobacterium marinum</i> is a slow-growing nontuberculous mycobacterium. It is a rare but important cause of skin and soft tissue infection. It is typically caused by exposure to saltwater aquariums or crustacean puncture during seafood preparation. The lesions appear as papules or nodules on an extremity and ascend in a sporotrichoid manner. There is no standard therapy; however, <i>M. marinum</i> is generally susceptible to macrolides and combination therapy with ethambutol and rifampin is often utilized for complicated infections. Mean duration of treatment is 3.5 months and surgical debridement is warranted for deep tissue involvement.</p>
<p><b>Vikram Puram</b></p>	<p><i>Mepolizumab-Induced Posterior Reversible Encephalopathy Syndrome: A Previously Unreported Side Effect</i></p> <p>Introduction: Posterior Reversible Encephalopathy Syndrome (PRES) is a neurotoxic state characterized by seizures, headache, vision change, paresis, nausea, and altered mental status. PRES has an important place in internal medicine due to the wide variety of causative diseases, infections, and medications that precipitate its mysterious onset. Although exposure to medications, particularly immunosuppressants, cancer chemotherapy, and</p>

	<p>biologic drugs is a common occurrence in patients who develop PRES, Mepolizumab has never before been associated. The following report outlines the first reported case of Mepolizumab-included PRES in the literature.</p> <p>Case Presentation: A 67-year old male with a significant history of chronic asthmatic bronchitis, obstructive sleep apnea, hypertension, and type II diabetes presents to the ED with an acute event of altered awareness. The patient was watching TV with his family when suddenly he had an unusual sensation of sand in his eyes. The next thing the patient remembered was being in an ambulance. During this episode, the patient’s family reported that he was having a “blank stare into space” and thus called 911 as the patient was then verbally unresponsive for 15 minutes. The patient was back to baseline when he was evaluated in the ED where he was subsequently started on the Kepra (Levetiracetam) for suspected complex partial seizure. With initial CT findings concerning for infection and malignancy, a follow-up MRI was ordered and showed nodular meningeal enhancement in the temporal, occipital, and anterior frontal lobes as well as significant white matter edema. ESR/CRP, CBC, BMP, lumbar puncture, as well as abdominal and chest imaging for underlying malignancy were unremarkable. Due to a lack of any other explainable etiology for this atypical PRES presentation, neurology requested discontinuation of Mepolizumab based solely on the temporal relationship between Mepolizumab initiation and symptom onset. After discontinuation of Mepolizumab, imaging showed marked improvement in 1 month and a complete resolution in 6 months. Our patient has been slowly titrated off Kepra and has been symptom-free since his initial episode.</p> <p>Conclusions: This report illustrates the first reported case of Mepolizumab-induced PRES and is highly relevant to pulmonologists, immunologists, neurologists as well as general hospitalists who will most likely have the first and earliest opportunity to identify the causative agent upon patient admission to the hospital. Treatment of severe asthma, asthma-exacerbations as well as diseases such as eosinophilic granulomatosis with polyangiitis (formerly Churg-Strauss) with mepolizumab is rapidly gaining popularity ever since the drug’s recent FDA-approval. This case report hopes to raise awareness of this potentially life-threatening and previously unreported side effect of Mepolizumab since early identification of the causative agent is the key to preventing the severe neurologic disability and possible death that may occur from the delayed treatment of PRES.</p>
<p><b>Jenna Ruggiero</b> Dr. Sara Hylwa Dr. Matthew Mansh</p>	<p><i>Golfer's Vasculitis: Exercise-Induced Vasculitis on the Feet of a Male Golfer</i></p> <p>Case Presentation: A 51-year-old with no underlying health issues presented to the outpatient dermatology clinic with an intermittent rash on his feet. The rash occurred on his feet every several months, always after physical exertion, such as long walks or playing golf. The rash eruption would be associated with skin sensitivity, warmth, and pain. There was no associated fevers, arthralgias, gastrointestinal complaints, and the skin was without pruritus or bullae formation. One of his worst eruptions occurred was after walking extensively around Washington D.C. Of note, he could run triathlons and play hockey without any issue. He treated the rash with ibuprofen and topical application of triamcinolone 0.1% cream, and the rash resolved after approximately five days with these measures. Cutaneous examination revealed scattered deeply pink macules, small patches, and thin papules across the dorsal aspect of his feet and extending onto the ankles bilaterally. A diagnosis of exercise-induced vasculitis was made.</p>

	<p>Discussion: This case illustrates an uncommon presentation of a small vessel vasculitis, exercise-induced vasculitis (EIV), sometimes called Disney rash or golfer’s rash/vasculitis. EIV commonly presents on the lower extremities of patients over 50 years of age after lengthy walking. The typical cutaneous presentation is on the lower legs, sparing areas compressed by socks. Our patient case is interesting as the vasculitis appeared after activities involving lengthy walking (golfing, long walks), but not running or skating (triathlons, hockey). Therefore, specific physical activities, shoe support, or terrains may trigger EIV, and the triggers are likely patient specific. EIV spontaneous resolves, typically within 10 days. Cutaneous biopsies show leukocytoclastic vasculitis. Blood investigations are negative. Many patients have resolution of the skin eruption with supportive treatment alone, but topical corticosteroids may reduce symptoms. For more extensive cases, short courses of oral steroids can be administered; for patients wanting a more preventative approach, oral hydroxychloroquine, colchicine, or dapsone can be tried.</p>
<p><b>Mudassar Sandozi</b> Dr. Afshan Anjum Dr. Richard Repass</p>	<p><i>Repeat Testosterone Related Exacerbation of Psychosis in a Schizoaffective Transgender Male Undergoing Masculinization Therapy</i></p> <p>Introduction: Physicians increasingly utilize masculinizing hormone therapy, though not yet FDA approved, in the transition process of transgender men. Research suggests testosterone can be safe and effective, however more data is required to understand its impact on specific patient populations.</p> <p>Case Presentation: A 40-year-old female-to-male transgender patient with a history of schizoaffective disorder depressive type, delusion disorder, anxiety, and gender dysphoria undergoing gender transition presented to the ER in fall 2019, due to an altercation with a housemate at an assisted living facility.</p> <p>The patient had been living in his chosen gender role as a male since 2016, with his mood and psychosis symptoms well controlled on his medication. In summer 2017, he began testosterone therapy of 50 mg IMI once a week. In winter 2018, he required hospitalization for a mental health emergency where he exhibited persecutory delusions, paranoia, and auditory hallucinations. He was stabilized with psychotropic medications, but his testosterone medications were inadvertently held, and was discharged to an assisted living facility. After discharge, he restarted his testosterone injections.</p> <p>During his most recent hospitalization he presented with worsening psychosis. Substance use and organic causes were ruled out. Weekly testosterone injections continued during this hospitalization and the patient’s symptoms were refractory to all psychopharm interventions. Upon discussion with the gender transition clinic and patient, the treatment team agreed to discontinue testosterone injections. The patient’s psychotic and mood symptoms improved and he was discharged on risperidone, clonidine, paliperidone, and sertraline.</p> <p>Discussion: Special considerations are needed when considering hormone therapy in female-to-male gender transition. In a transgender male undergoing cross-sex treatment, testosterone induces virilization and suppresses feminizing characteristics. Literature is replete with reports on estrogen’s protective effects on the onset of schizophrenia in women. First episodes of psychosis in women tend to occur a decade later than in men. In our perimenopausal patient, the etiology of psychosis could be due to low estrogen level or exogenous testosterone treatment. Psychosis that occurs following hormone manipulation has been reported to be resistant to usual antipsychotic regimens, which was true in our case.</p>

	<p>This case poses several questions</p> <ol style="list-style-type: none"> <li>1) What are the mechanisms by which testosterone can induce psychosis? Are they direct or indirect?</li> <li>2) Should estrogen be considered as an adjuvant treatment in these cases, further capitalizing on its psycho protective effects? Is it ethical to treat transgender men with feminizing hormones?</li> <li>3) Should clozapine be considered as an option for treatment-resistant psychosis cases in this population? Is it OK to burden the patient with the plethora of side effects and monitoring that comes along with clozapine?</li> <li>4) What supports or therapies should be offered to patients whose transition treatment is interrupted or discontinued?</li> </ol>
<p><b>Ellen Spartz</b> Stephen John Christopher Sullivan</p>	<p><i>A Case of Multifactorial Hyponatremia</i></p> <p>Introduction: Hyponatremia is a relatively common laboratory finding. The causes of hyponatremia span many different organ systems, leading to a wide diagnostic differential. A thorough history, physical exam, and laboratory evaluation with serum sodium, serum osmolality, urine sodium, and urine osmolality can aid in identifying the etiology.</p> <p>Case presentation: A 65-year-old female presented with 4 days of nausea, vomiting, malaise, and 2 weeks of worsening fatigue. She also reported decreased appetite, no bowel movements, and increased volume of urination. The patient had a throbbing headache that was unlike previous headaches as well as visual disturbances. She recently completed treatment with isoniazid for latent tuberculosis but had no other significant medical history.</p> <p>On initial presentation, she was hypotensive at 99/69 with otherwise normal vital signs. Her physical exam was notable for dry mucous membranes with no other abnormalities. Laboratory evaluation showed serum sodium of 116, serum osmolality of 244, urine sodium of 82, urine osmolality of 473, cortisol of 1.3 and ACTH of 38. She was also found to have an elevated prolactin at 71, a low free T4 of 0.49, and TSH of 3.31. Her potassium, serum glucose, and creatinine were within normal limits. Given her neurological symptoms and persistent vomiting, a non-contrast CT was obtained and demonstrated a sellar/suprasellar mass. Further imaging with MRI showed an enhancing 2.7 x 2.0 x 3.7 cm sellar/suprasellar mass with extension into the left sphenoid sinus.</p> <p>The patient's serum sodium improved after receiving 1L of normal saline upon presentation. Once a presumed diagnosis of SIADH was established, the patient was placed on fluid restriction and was started on hydrocortisone and levothyroxine. This resulted in gradual improvement of serum sodium over the next few days. Given concern for a pituitary macroadenoma causing these symptoms, the patient underwent surgical resection of the sellar/suprasellar mass. She continues to take hydrocortisone and levothyroxine. On follow-up, the patient reports no symptoms and has a serum sodium of 140.</p> <p>Discussion: The patient's hyponatremia was likely multifactorial. The pattern of laboratory findings suggests a diagnosis of SIADH versus adrenal suppression. The patient's increase in serum sodium following administration of corticosteroids is consistent with adrenal suppression. We suspect that the pituitary macroadenoma compressed the pituitary such that production of ACTH and TSH were compromised. Also, disturbances in the central nervous system can lead to SIADH. Finally, the initial increase in sodium with fluid</p>



	<p>administration suggests a component of volume depletion. In conclusion, we propose that a systematic approach is helpful when approaching patients with hyponatremia. Laboratory evaluation can distinguish causes such as primary polydipsia, heart failure, SIADH, and adrenal insufficiency. Additionally, in patients presenting with neurological symptoms such as headache and visual changes, it may be appropriate to obtain brain imaging.</p>
<p><b>Simon Yang</b> Dr. Austin Cudak Dr. Peter Lund</p>	<p><i>Atypical Presentation of Mantle Cell Lymphoma Mimicking Pancreatic Adenocarcinoma</i></p> <p>Introduction: While the majority of solid pancreatic neoplasms are primary exocrine pancreatic cancer or neuroendocrine tumors, a minority are rare and can pose a diagnostic challenge. A comprehensive evaluation of extra-pancreatic findings can speed diagnosis.</p> <p>Case Presentation: A 56-year-old male with no significant medical history presented with one month of intermittent epigastric abdominal pain, nausea and a 10 pound weight loss. He initially presented to clinic where an abdominal ultrasound noted a 9cm pancreatic head mass with extrahepatic biliary dilation and borderline gallbladder wall thickening and he was referred for admission.</p> <p>On admission he was afebrile and hemodynamically stable. Laboratories were notable for WBC 2.8, Hgb 8.3, platelet 68, CRP 2.73, lipase 85.2, elevated metamyelocytes of 4.4, low normal B12 of 412, and positive H. pylori antigen. CT scan confirmed a 10cm mass enveloping the pancreas. Further evaluation of pancytopenia with bone marrow biopsy revealed mantle cell lymphoma blastic variant with Ki-67 proliferation index of 20%. Subsequent tissue biopsy noted effacement of pancreatic architecture with lymphoid appearing cells positive for CD20 B-cell marker and CD5 seen in the germinal center and mantle zone.</p> <p>During his subsequent stay, he developed jaundice with elevated LFTs in an obstructive pattern. Repeat ultrasound and PET scan showed a hypermetabolic 10-11cm pancreas-encasing mass narrowing the common bile duct with dilated common hepatic duct and intrahepatic biliary duct as well as intense heterogenous uptake in the bone marrow. A biliary stent was placed by ERCP and jaundice improved. He was then placed on RCHOP (rituximab, cyclophosphamide, doxorubicin, vincristine, and prednisone) with plans to follow with autologous stem cell transplant.</p> <p>Conclusion: Mantle cell lymphoma - a type of primary pancreatic lymphoma - is known for its heterogenous presentations in a variety of extra-nodal.<sup>1,2</sup> This case illustrates how uncommon types of pancreatic cancer can present similarly to pancreatic adenocarcinoma, which represents approximately 85% of cases.<sup>3</sup> Although primary pancreatic lymphomas are rare, our main takeaway is that a broad differential should be considered for pancreatic tumors especially if there are other clinical clues, such as pancytopenia seen with this case, as it can result in far different treatment and prognosis.</p> <p>Reference</p> <ol style="list-style-type: none"> <li>1. Arieira, Catia, et al. "Primary colon mantle lymphoma: a misleading macroscopic appearance!." <i>Revista Espanola de Enfermadades Digestivas (REED)</i> 111.12 (2019): 965-968.</li> <li>2. Iqbal, Madiha, et al. "Intraocular involvement of mantle cell lymphoma: a case report and literature review." <i>Hematology/Oncology and Stem Cell</i></li> </ol>

	Therapy (2019). 3. Ilic, Milena, and Irena Ilic. "Epidemiology of pancreatic cancer." World journal of gastroenterology 22.44 (2016): 9694.
<b>Residents</b>	
<b>Quality Improvement - Residents</b>	
<b>Emily Westergard</b> L. Patel L. Wohlwend M. Ornes	<p><i>Improved Inpatient Appropriate VTE Prophylaxis Rates Through Use of An Electronic Medical Record Intervention</i></p> <p>Background: Venous thromboembolism (VTE) is a major health issue, carrying a high mortality rate and significant economic toll within the United States healthcare system. Despite the prevalence of VTE, clinicians have struggled with appropriately prescribing prophylactic agents in the inpatient setting despite multiple risk assessment models and guidelines. To help improve appropriate VTE prophylaxis rates within the Allina Healthcare system, a Padua Scoring Tool was incorporated into admission order sets in September 2019.</p> <p>Methods: Internal medicine resident admissions at Abbott Northwestern Hospital were randomly sampled from both February to July 2019 and 2020. Admission orders were then assessed for appropriate selection of VTE prophylaxis and compared pre- and post-addition of the Padua scoring tool. Retrospective calculation of the Padua, IMPROVE, and IMPROVE Bleed scores was used to define appropriate VTE prophylaxis. Rates of appropriate prophylaxis, frequency of inappropriate prophylactic agent use, and omission of prophylaxis were then compared.</p> <p>Results: Appropriate VTE prophylaxis rates increased from 64.5% to 76.7% post-intervention. Inappropriate sequential compression device (SCD) prescribing worsened from 51% to 78%. Inappropriate use of heparin improved from 43% to 18%. Inappropriate use of enoxaparin remained low at 2% and 3%, respectively. Rate of inappropriate lack of prophylaxis remained stable at 6%.</p> <p>Conclusion: Introduction of the Padua scoring tool to admission order sets was associated with a significant improvement in appropriate VTE prophylaxis and inappropriate heparin use overall, however, there was an unexpected increase in inappropriate SCD use.</p>
<b>Research - Residents</b>	
<b>Natashay Bailey</b> Dr. Allyson Palmer Dr. Laura Greenlund	<p><i>Comparison of D-dimer Values for Acute Deep Venous Thrombosis vs Alternative Diagnoses in Patients undergoing Lower Extremity Ul</i></p> <p>Introduction: Lower extremity deep venous thrombosis (DVT) is a common diagnosis in ambulatory, critically ill, and post-surgical patients. Laboratory testing, in particular d-dimer values, and ultrasonography are useful in the diagnosis of DVTs. Typically, d-dimer is used to screen low and intermediate pre-test probability patients to determine the need for ultrasound. There is little in the medical literature describing the incidence of alternative diagnoses observed on lower extremity ultrasound and the corresponding d-dimer test results in patients being evaluated for suspected acute DVT. In this study, we identify patients with ultrasound identified alternative diagnoses and</p>

	<p>characterize the corresponding d-dimer levels in comparison to patients with acute DVT.</p> <p>Methods: We conducted a retrospective, single health system study using chart analysis, focusing on lower extremity ultrasounds between July 2017 and July 2019. Data was collected from both ambulatory and hospitalized patients across three Mayo Clinic regions in the United States (Arizona, Florida, and Rochester). We assessed patient characteristics, d-dimer levels detected by high sensitivity immunoassay, and incidence of acute DVT and alternative diagnoses on ultrasound. Cases in which patients underwent screening for surgical planning, had limited sonography, had known DVTs within 6 months, and did not have d-dimer as part of the evaluation were omitted. Overall, 2,377 cases fulfilling study criteria were analyzed.</p> <p>Results: Among 2,377 patients that underwent both d-dimer and lower extremity ultrasound, acute DVT was identified in 8.8% while alternative diagnoses were seen in 30.2% of patients. The most common alternative diagnoses were popliteal cyst (n= 243, 10.2%), edema (n= 149, 6.3%), post-thrombotic changes (n= 91, 3.8%), chronic DVT (n= 78, 3.3%), SVT (n=38, 1.6%), and sub-acute DVT (n= 27, 1.1%). The mean d-dimer level in acute DVT case was 6889.5 ng/mL (SEM 623.6 ng/mL) versus 2180.3 ng/mL (SEM 165.7 ng/mL) with no pathology. In alternate diagnoses, mean d-dimer values with SEM were 2217.3 +/- 257.6 ng/mL in popliteal cyst, 3286.1 ± 488.0 ng/mL in edema, 1142.6 ±181.2 ng/mL in post-thrombotic changes, 1385.4 ± 272.2 ng/mL in chronic DVT, 6099.9 ± 1782.9 ng/mL in sub-acute DVT, and 5276.1 ± 1716.4 ng/mL in SVT. Interestingly, in those with d-dimer values below 500 ng/mL, the typical low and intermediate pre-test probability cutoff where patients would not require ultrasound, the incidence of acute DVT was 2.2% (12 of 547 cases) vs 10.8% (197 of 1830 cases) in patients with d-dimer over 500 ng/mL.</p> <p>Conclusion: In patients, undergoing d-dimer testing and lower extremity ultrasound to rule out acute DVT in the Mayo Clinic network, the incidence of acute DVT was low compared to alternative diagnoses (8.8% vs 30.2%). Alternative pathologies such as sub-acute DVT and SVT had d-dimer values comparable to acute DVT. In contrast, pathologies such as post-thrombotic changes and popliteal cyst, d-dimer values were similar to cases with no pathology. Future studies should seek to validate these findings with a prospective design.</p>
<p><b>Megan Covington</b>  Dr. Philip Young  Dr. Melanie Bois  Dr. Joseph Maleszewski  Dr. Kyle Klarich</p>	<p><i>The Clinical Impact of Cardiac Fibromas</i></p> <p>Introduction: Cardiac fibromas are rare tumors that can present with a variety of clinical findings. Previously they have been described as having the potential to cause significant morbidity and mortality, including inflow and outflow obstruction, arrhythmias, and sudden cardiac death. There has not been a large review of cases focusing on the clinical aspects of cardiac fibromas since 1994. The purpose of this study is to provide an updated analysis of their clinical impact. This will allow for better understanding of these tumors, lead to clinical awareness, and reveal important factors for consideration when diagnosing and determining treatment options.</p> <p>Methods: A retrospective case series was conducted from a tertiary care institution to identify cases of cardiac fibroma (1970-2020). Mayo Clinic's radiology and pathology archives were reviewed, and patient cases were</p>

	<p>included if cardiac fibroma was diagnosed via imaging results or pathology report. Patient cases were reviewed and examined for demographics, symptomatology, location, heart rhythm data, radiographic findings, pathology findings, interventions, and outcomes.</p> <p>Results: Twenty patients (11 female) with a median age of 23.5 years (1 day - 72 years) were identified through institutional archives as having a cardiac fibroma. Presenting symptoms included ventricular tachycardia (VT), dyspnea, palpitations, syncope, angina, heart failure, and emboli. VT was noted in six patients (30%), all of whom experienced associated symptoms such as palpitations, syncope/presyncope, and cardiac arrest. New murmur was also common, noted in five patients (25%). Older patients were more likely to be asymptomatic (49-72 years). One patient had Gorlin’s syndrome. Tumors were most commonly located in the left ventricle (14/20, 70%). Eighteen out of twenty were initially diagnosed with imaging, twelve of whom were later confirmed via resection. Fourteen patients underwent resection, four of whom required complex operations including artery graft, valve repair, reconstruction of the aortic root and ventricular septum, and amputation of LA appendage. Six of these patients had delayed intervention ranging from 6 months to 7 years. One patient required a second surgical resection. One operative death occurred. Six patients were managed with clinical follow-up and imaging surveillance. Three were asymptomatic, one experienced heart failure, and two suffered from arrhythmias. Both of the latter patients required antiarrhythmics, and one ultimately required ablation and pacemaker placement. One was lost to follow up.</p> <p>Conclusion: Cardiac fibromas are typically considered to affect a pediatric population; however, this study demonstrated a significant prevalence in adults, and should therefore remain on the differential in the assessment of cardiac masses. VT and associated symptoms were common at initial presentation. Combined imaging modalities are sensitive in the diagnosis of cardiac fibroma. Surgical resection is overall successful in symptomatic patients, and clinical follow-up with imaging surveillance may be appropriate management for asymptomatic patients.</p>
<p><b>Siva Kamal Guntupalli</b>  Yosuf Subat  Pavol Sajgalik  Matthew Hainy  Kim Chul-Ho  Bruce Johnson  Thomas Allison  Kaiser Lim  Alexander Niven</p>	<p><i>Aerosol and Droplet Particle Generation During Forced Expiration for Peak Flow Testing</i></p> <p>Introduction: Peak flow meters are routinely used by asthmatic patients to assess airflow limitation. In order to ensure proper technique, patients are often asked to perform peak flow maneuvers in the presence of physicians, nurses, respiratory therapists, and other healthcare workers, potentially creating the infection transmission risk given the ongoing COVID epidemic. We aimed to develop a pragmatic approach to quantify particle generation during peak flow testing to better understand the risk of this procedure with the goal of informing institutional infection control and mitigation strategies.</p> <p>Methods: For this pilot study, a tightly sealed room was connected to two portable fans with HEPA filters, allowing for nearly particle free room. We measured the concentrations of the generated particles after peak flow testing on 5 healthy volunteers using five different peak flow meters (Respironics, Philips, Clement Clarke, Respironics low range, and Mogaghan) using Fluke and PTrak particle counters. Baseline concentrations of the existing particles in a standard pulmonary function testing room were also measured for comparison with the testing environment.</p>

	<p>Results: Higher mean particle concentrations were observed with peak flow testing compared to unmasked tidal breathing. Ultrafine particles (0.02-1 micron) were generated in higher proportions compared to particles of other sizes (&gt;1 micron). Mean particle concentration was lowest with the use of Respironics peak flow meter (1.25±0.47 particles/cc). Comparable mean particle concentrations were observed with Philips (3.06±1.22), Clement Clarke (3.55±1.22), Respironics low range (3.50±1.52), and Mogaghan (3.78±1.31) peak flow meters. When compared to the mean concentration of the generated ultrafine particles (0.02-1 micron) by masked (0.22±0.29 particles/cc) and unmasked (0.15±0.18) tidal breathing, mean particle concentrations were higher after use of peak flow meters.</p> <p>Conclusions: We developed a pragmatic approach to quantify small particle generation after peak flow testing. Although all peak flow meters demonstrated increased mean particle concentration, differences were small compared to the mean particle concentrations found in the ambient clinical environment.</p>
<p><b>Fredrick Ogugua</b> Dr. Abdulrahman Gamam Dr. Jorge Reyes</p>	<p><i>Associations of Implantable Cardiac Defibrillator placement in Cardiac Sarcoidosis: An insight from the National Inpatient Sample</i></p> <p>Introduction: There is sparse literature detailing the indications for implantable cardiac defibrillator (ICD) placement specific to hospitalized patients with cardiac sarcoidosis (CS). Using the National Inpatient Sample (NIS), we aim to identify patient characteristics and comorbid conditions associated with ICD placement in patients with CS.</p> <p>Methods: We queried the 2016-2017 NIS database to identify all admissions age 18 years or older with a discharge diagnosis of CS who did not have a prior history of ICD placement. We accounted for survey design and built a multivariable logistic regression to identify predictors of ICD placement in CS patients. Variables of interest included age, race, sex, and validated codes for multiple comorbidities.</p> <p>Results: An estimated 1,180 patients with CS without prior ICD placement were hospitalized from 2016 to 2017. Among them, 35% had an ICD placed during that hospitalization. CS patients who had ICD placement were more likely to be male (72% v. 48%, OR 4.00 {1.92-8.36}, p&lt;0.01). There were no significant differences in age or race regarding ICD placement. CS patients with ICD placement were more likely to be obese (35% v. 24%, OR 3.00{1.46-6.15},p&lt;0.01), have hypertension (71% v. 68%, OR 2.68{1.20-5.99}, p&lt;0.01) and have a valvular disease (20% v. 16%, OR 4.06{1.25-13.2}, p&lt;0.01).</p> <p>Conclusion: In our retrospective cohort of patients with CS without prior ICD placement, we identified male sex, comorbid obesity, hypertension, and valvular disease to be predictors of ICD placement. It is unclear if the association with valvular disease signifies advanced cardiac sarcoidosis, as sarcoid lesions involving cardiac papillary muscles can precipitate valvular disease. Further research is needed to determine the clinical significance between the aforementioned comorbidities, cardiac sarcoidosis and ICD placement.</p>
<p><b>Jessica Padniewski</b> Dr. Linh Ngo</p>	<p><i>Gazing Into The Crystal: Current Status of Gout Care Among Different Specialties At A Tertiary Academic Center</i></p>

	<p>Background/Purpose: Gout is the most common type of inflammatory arthritis, affecting about 4% of the U.S. population and results in an estimated 6 billion dollars in healthcare costs yearly. Annual gout management costs anywhere from \$172 to \$6179 according to a meta-analysis performed by Rai et al. Despite available treatment guidelines published by the American College of Rheumatology (ACR) and European League Against Rheumatism (EULAR), deficiencies in the treatment of gout remain prevalent. The ineffective treatment of gout results in significant functional decline, increased morbidity, and significant economic burden. A study performed by Harrold et al. in 2013 found that about half of primary care providers reported optimal gout treatment practices. In an attempt to evaluate the efficacy of gout management and adoption of the 2012 ACR gout guidelines, we performed a retrospective cohort study at Hennepin Healthcare, a tertiary academic referral center.</p> <p>Methods: IRB approval was obtained for this retrospective cohort study. All outpatient encounters with ICD-10 diagnosis code of M10.9x for gout between July 1, 2018 through June 31, 2019 at Hennepin Healthcare were pulled from a virtual data warehouse. A total of 485 outpatient encounters and 297 unique patients were identified. Patients who had greater than or equal to 8 points in the 2015 ACR Gout Classification criteria were excluded from this descriptive analysis. A total of 226 unique patients met criteria and were enrolled into the study. Chart review of enrolled patients was completed and data regarding comorbidities, outpatient clinic type, basic demographics, baseline uric acid, urate lowering therapy and follow up frequency were recorded into a HIPAA compliant REDCap database and a descriptive analysis was done.</p> <p>Results: In summary, of patients noted to have uric acid levels &gt; 6.0 mg/dL, about 51.6% started uric acid prophylaxis. Of patients with gout and uric acid levels &gt; 6.0 mg/dL, about 44% eventually met uric acid level goals, while about 55% did not. For those who achieved uric acid goal, the average number of days to goal was 134 days.</p> <p>Conclusion: Despite the ACR Gout Guidelines being published in 2012, our data indicates adherence to its major principles is still lacking at our tertiary academic center. A large percentage of patients above uric acid goals do not receive flare prophylaxis, average days between follow up for titration of uric acid lowering therapy is almost half a year, and the majority of patients with gout did not reach uric acid goals. Further intervention is needed to improve adoption of guideline principles of gout care.</p>
<p><b>Ka Bao Vang</b>  Dr. Kathleen Culhane-Pera  Dr. Luis Martin Ortega  Txia Xiong  Carin Northuis  Pilar de la Parra  Dr. Kamakshi Lakshminiarayan</p>	<p><i>Mobile Health Technology for Hypertension Management in Hmong and Latino adults: Empowerment or Entrapment?</i></p> <p>Background: Hypertension is a major risk factor for stroke and heart attack. Unfortunately, more than 40% of adults with hypertension have inadequate blood pressure control. Patients from minority groups, including Hmong and Latino communities, have worse HTN control. Home blood pressure self-monitoring can improve hypertension control, particularly in times of restricted clinic visits during COVID-19 pandemic. Mobile health technology (mHealth) can facilitate this self-monitoring. However, it is unknown if mHealth will be accepted by minority patients.</p> <p>Objective: Identify Hmong and Latino adults' perspectives about a mHealth based care model for high blood pressure management involving self-</p>

	<p>monitoring and responsive medication adjustment by a provider team.</p> <p>Methods: We enrolled 25 Hmong and 25 Latino participants with hypertension from an urban federally qualified health center serving low income patients. We obtained demographic data, collected responses about hypertension knowledge, and conducted open-ended interviews to identify participants' perspectives about the mHealth based hypertension care model.</p> <p>Results: Participants were 56% female with a mean age of 56 years. Most (88%) were immigrants and most (96%) had lived in the United States (US) for 10 years or longer. Half (52%) spoke and read English and 100% of Latino and 76% of Hmong were literate in their own language. In addition to hypertension, participants reported comorbidities including diabetes mellitus (48%), elevated cholesterol (54%) and depression or anxiety (48%). While most participants (98%) agreed that lowering high blood pressure decreased the risk of strokes, heart attacks and kidney failure, there were gaps in medical knowledge. Three major themes emerged from the interviews regarding the perception of the mHealth based care model: 1) Using mHealth technology could be useful especially if assistance was available to patients with technological challenges; 2) Knowing blood pressures could be helpful to patients who agreed with doctors' medical diagnosis and prescribed treatment; 3) Transmitting blood pressures to the clinic and their responsive actions could feel empowering for patients; however, the sense of increased surveillance could feel entrapping to some patients.</p> <p>Conclusion: In general, Hmong and Latino adults responded positively to the mHealth based hypertension care model and felt that it could be empowering since it could increase patient-provider communication without burden of clinic visits and could increase involvement in BP control for those who agree with the medical model of hypertension. Some participants expressed concern that the mHealth model could be entrapping as it could breach patient privacy, interfere with patients' lifestyle choices, and curtail patient autonomy. In a shared decision-making approach with patients (and possibly their family members), health care systems and clinicians should explore potential issues of empowerment and entrapment when offering an mHealth care model in practice.</p>
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**Clinical Vignette- Residents**

<p><b>Omar Abdullahi</b> Dr. Breanna Zarmbinski Dr. Clara Zamorano</p>	<p><i>A Rare Case of Macrophage Activation Syndrome (MAS)</i></p> <p>Case Presentation: MAS is a subtype of hemophagocytic lymphohistiocytosis (HLH) and is a syndrome of immune hyperactivity and cytokine storm. Primary HLH is mainly seen in pediatrics and is associated with multiple gene mutations. In contrast, secondary HLH is often driven by secondary inflammatory causes, most commonly viral illness, malignancy, or autoimmune diseases. MAS has no preceding viral illness, new medication, or malignancy. Highest incidence is with systemic juvenile idiopathic arthritis in children and with RA in adults.</p> <p>A 28-y.o. female presented with 5 day history of headache, nausea, vomiting, fatigue, body aches and maculopapular rash not involving the palms or soles. On admission, she was afebrile, hypotensive, and tachycardic. Labs notable for elevated lactate, procalcitonin and LFTs. UA and CXR were unremarkable. Her symptoms were concerning for bacterial meningitis and multiorgan system dysfunction, and she was treated with broad spectrum</p>
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	<p>antibiotics and fluids. On day 2, repeat CXR showed new bilateral airspace infiltrates with interstitial edema at the lung bases, concerning for flash pulmonary edema. The patient subsequently went into shock and further respiratory distress, so her fluids were stopped, pressors were started and she was intubated. She also had worsening anemia and thrombocytopenia thought to be secondary to acute viral illness or newly diagnosed autoimmune flare. Autoimmune workup showed strongly positive sjogrens markers (Ro52 antibody&gt;1,685.3, Ro60 antibody&gt;1,374.8, SS-B antibody&gt;1,550.0). Further workup notable for elevated ferritin at 17,000 (lower with steroid) with triglycerides elevated at 389. These lab findings, along with the clinical picture, were concerning for HLH. She had a peripheral smear which showed no hemolysis. Bone marrow biopsy was highly suggestive of HLH. Heme/Onc consult initiated intrathecal methotrexate, IV etoposide, and dexamethasone. She clinically improved, was successfully extubated and discharged to home within several weeks.</p> <p>Conclusion: This case illustrates that the clinical presentation of HLH can mimic acute infection with multiorgan failure. These patients, unlike those with infection, will be unresponsive to antibiotics. Early diagnosis is of critical importance considering adult patients have a 31-58% survival rate if treatment is initiated promptly. Without treatment mean survival is 3-5 weeks with mortality approaching 100%. Hence, start workup early with triglycerides and ferritin levels if HLH is suspected.</p>
<p><b>Nadia Akhiyat</b> Dr. Brett Grieb Dr. Mustaqueem Siddiqui</p>	<p><i>A Rare Neoplasm Masquerading as Pulmonary Embolus</i></p> <p>Introduction: Pulmonary artery sarcoma is a rare malignant neoplasm that may be difficult to distinguish from pulmonary arterial embolism. Initial misdiagnosis is common and often delays therapeutic intervention. Distinguishing key features are necessary for earlier diagnosis and improved clinical outcomes. We present a case of pulmonary artery sarcoma presenting as hyperfibrinolysis in the absence of identifiable clot formation.</p> <p>Case Description: A 44 year old male with no known comorbidities initially presented with recurrent gross hematuria and cough with hemoptysis associated with 30lbs unintentional weight loss and night sweats. Initial CT chest without contrast demonstrated scattered non-specific pulmonary nodules. Comprehensive bleeding evaluation revealed evidence of dysfibrinogenemia with D-dimer &gt; 30,000, decreased alpha-2 plasmin inhibitor, and markedly elevated soluble fibrin monomer. Other coagulation studies were normal. Ultrasound of the extremities was negative for overt clot formation. CT abdomen and pelvis was negative for occult malignancy. Thorough urologic evaluation revealed an AV malformation of the right renal artery which was embolized. BAL was negative. A tranexamic acid taper resolved hematuria and reflected improvement in fibrinolysis labs. Our patient however continued to experience B-symptoms. He again developed hemoptysis 3 months following his initial presentation. A CT chest with IV contrast was obtained and was interpreted to demonstrate an extensive right pulmonary embolus involving the entire right pulmonary artery, proximal left pulmonary artery, and encroachment upon the left main coronary artery. A PET-CT revealed intense FDG uptake within the expansile mass. Pathology from an endobronchial ultrasound biopsy revealed spindle cell sarcoma. This patient successfully underwent radical surgical resection with planned chemo-radiation therapy. Hyperfibrinolysis was ultimately attributed to underlying sarcoma.</p>



	<p>Discussion: Pulmonary artery sarcoma is often misdiagnosed as pulmonary embolism. Key distinguishing features include an atypical appearance of a pulmonary artery mass on CT imaging without peripheral emboli, cough, hemoptysis, and clinical presentation suggesting underlying malignancy. This case illustrates such key features in addition to the importance of considering occult malignancy in the setting of idiopathic hyperfibrinolysis in the absence of clot formation.</p>
<p><b>Chineze Akusoba</b> Dr. Brenden Ingraham Dr. Adam Sawatsky</p>	<p><i>Gerbode Defect: A Rare Complication of Tricuspid Valve Endocarditis</i></p> <p>Introduction: Infective endocarditis can be associated with a multitude of complications including heart failure, perivalvular abscess, septic embolization and mycotic aneurysm. The likelihood of complications depends on a variety of factors, including comorbidities, pathogen, and duration of illness prior to therapy. We describe a case of methicillin-resistant staphylococcus aureus (MRSA) tricuspid valve endocarditis that resulted in a ventricular septal defect creating a communication between the left ventricle (LV) and right atrium (RA), also known as the Gerbode defect.</p> <p>Case Presentation: A 50-year-old man with a history of metastatic colon cancer status post sigmoidectomy and pelvic radiation complicated by ureteral strictures requiring bilateral nephrostomy tubes was admitted with hematuria and dyspnea following recent diagnosis of MRSA bacteremia and tricuspid valve endocarditis.</p> <p>The patient was hospitalized 3 weeks prior to admission with MRSA bacteremia secondary to the nephrostomy tubes. He underwent a transthoracic echocardiogram (TTE) which demonstrated a 1.5 cm vegetation on the anterior leaflet of the tricuspid valve with moderate tricuspid regurgitation. A CT Chest revealed multifocal thoracic septic emboli. He was discharged on a 6-week course of IV vancomycin via a peripherally inserted central catheter (PICC). Unfortunately, he developed worsening dyspnea and significant hematuria requiring numerous red blood cell transfusions and was re-hospitalized.</p> <p>On arrival, he was afebrile and hemodynamically stable. Exam was notable for a 3/6 pansystolic murmur best heard at the lower left sternal border. Laboratory evaluation revealed acute anemia with hemoglobin 6.0 (13.2 – 16.6 g/dL), leukocytosis of 10.2 (3.4 - 9.6 x 10<sup>9</sup>/L) and CRP 41.2 (≤ 8.0 mg/L). Blood cultures were negative. He underwent repeat TTE, which revealed resolution of the tricuspid vegetation, however there was a new, large atrio-ventricular mycotic aneurysm that ruptured into the RA resulting in a left to right shunt between the LV and RA. Transesophageal echo (TEE) confirmed this finding. He underwent right heart catheterization notable for oxygen step-up in the low right atrium of 14% and elevated cardiac index of 4.5 (2.5 – 4.0 L/min/m<sup>2</sup>) with moderate left-to-right shunting. Cardiovascular Surgery recommended elective surgical intervention for closure of the defect.</p> <p>Discussion: The Gerbode defect is a communication between the LV and RA. It can be congenital or acquired as the result of endocarditis, prior cardiac surgery, myocardial infarction, or trauma. Diagnosis is generally made by TTE. Surgical and percutaneous transcatheter closure are options for management of the defect depending on the patient's symptoms, magnitude of shunt, anatomical feasibility, and comorbidities.</p>
<p><b>Hadiyah Audil</b></p>	<p><i>Copper Deficiency-Induced Anemia and Neutropenia</i></p>

<p><b>Dr. Animesh Pardanani</b></p>	<p><b>Introduction:</b> The prevalence of obesity has been estimated at 13% worldwide and nearly 40% in the US. Bariatric surgery is effective for obese patients and is becoming more prevalent; in 2017, approximately 580,000 people underwent bariatric surgery, with 228,000 cases being performed in the US alone. Such procedures include Roux-en-Y gastric bypass (RYBG), wherein a small proximal gastric pouch is created and anastomosed to the small intestine. This bypassing of the alimentary tract predisposes to micronutrient and mineral deficiencies. Guidelines therefore recommend that patients undergo routine post-operative monitoring for nutritional deficiencies and daily supplementation with a minerals-containing multivitamin, calcium, and vitamins D and B12.</p> <p><b>Case Description:</b> A 49-year-old female presented with two weeks of progressive dyspnea. She was status post RYGB 18 years ago, and was on daily B12 and folate supplementation only. Labs on admission were remarkable for macrocytic anemia (Hgb 6.2, MCV 105.2) and neutropenic leukopenia (WBC 1.7, ANC 240); on chart review, CBC from nine months prior was normal. Physical exam was unremarkable and review of systems was negative for blood loss and constitutional B symptoms. She was given one unit of packed red blood cells with appropriate response in hemoglobin and some symptom improvement; Hematology was consulted due to concern for myelodysplasia. Peripheral smear showed slight anisopoikilocytosis. Evaluation for GI blood loss, hemolysis, liver disease, and hypothyroidism was unremarkable. Although B12 and folate were normal, serum copper was &lt;0.10 mcg/mL (normal 0.75-1.45 mcg/mL). As she had an inappropriately-normal reticulocyte count, a bone marrow biopsy was performed. Pathology revealed panhyperplasia, cytoplasmic vacuolization in myeloid precursors, increased ring sideroblasts, and no increase in blasts. She was started on IV copper replacement and discharged with outpatient copper infusions.</p> <p><b>Discussion:</b> Copper deficiency occurs in 10-15% of patients following gastric bypass surgery, but remains under-recognized. Further risk factors for acquired hypocupremia include other malabsorptive conditions, total parenteral nutrition lacking sufficient copper, excessive zinc ingestion, and chronic dialysis. Clinical manifestations include anemia, neutropenia, and neurologic deficits (such as ataxia, myeloneuropathy, and cognitive deficits), the latter of which was not seen in our patient but often mimics symptoms of B12 deficiency. Serum copper levels can confirm the diagnosis and bone marrow biopsy also has diagnostic utility, with abnormalities including erythroid hyperplasia, vacuoles in myeloid precursors, iron-containing plasma cells, and ringed sideroblasts. Treatment typically relies on copper repletion. Importantly, although labs may resemble iron deficiency anemia, iron supplementation can cause excess iron to compete with copper absorption and worsen hypocupremia. With the rising prevalence of bariatric surgeries, copper deficiency remains an important pathophysiologic process to consider and diagnose in the correct clinical context.</p>
<p><b>Madeline Barnes</b> Dr. Ronald Reilkoff</p>	<p><i>Diagnostic Challenges in Detecting Leptospirosis</i></p> <p><b>Introduction:</b> Leptospirosis has a varied clinical course with most cases resulting in a mild/self-limited or subclinical presentation, but some are severe resulting in potentially fatal outcomes with liver, kidney and pulmonary failure. The wide-ranging clinical course with the relative rareness seen in Minnesota makes leptospirosis a diagnostic challenge.</p>

	<p>Case Presentation: A previously healthy 49-year-old male presented to the emergency department with fever, myalgias, malaise, and vomiting and found to be in new atrial fibrillation with rapid ventricular response and hypoxic respiratory failure with labs showing acute renal failure, acute hepatitis and troponin elevation. He was admitted to the ICU due to pressor requirement in presumed septic shock due to a respiratory pathogen given chest x-ray with bilateral disease and started on vancomycin, zosyn and doxycycline. Initial infectious workup was negative including RVP, legionella, strep pneumo and COVID-19 thus it was broadened to include tick born illnesses, fungal and viral infections, parasites, and rare bacterial such as leptospirosis and tularemia given his exposure with goats, donkeys, rats and sewage. He continued to decline requiring a bronchoscopy revealing diffuse alveolar hemorrhage and transfer to center with liver transplant capabilities on day 3 of hospitalization. At admission to the liver transplant center, repeat cultures were collected and antimicrobials were broadened to penicillin, ceftriaxone, doxycycline, and ambisome. On day 6 of hospitalization, the repeat cultures grew yeast while all other testing continued to come back negative for infectious causes. As more infectious tests came back negative including leptospirosis antibody testing and the inflammatory markers finally decreased, it was decided to discontinue penicillin on day 8. The same day the culture growing yeast speciated to <i>Candida albicans</i> resulting in ambisome being switched to micafungin. The patient continued to slowly improve from day 8 with extubation occurring on day 12. Throughout that time, he continued to receive doxycycline, ceftriaxone and micafungin and the entire infectious workup came back negative. It was unclear how long to keep the antibiotics given unknown etiology leading to the decision to send the Karius testing on day 13. The Karius test came back positive on day 15 for <i>Leptospira</i>, he completed a 16-day course of doxycycline and ceftriaxone. He continued to improve and was discharged on day 20 from the hospital.</p> <p>Conclusion: This case illustrates the difficulty in diagnosing leptospirosis. Specifically, that IgM can be negative for the first 7 days of illness as in this patient's case and repeat testing should always be consider if there is a high enough clinical suspicion.</p>
<p><b>Scarlett Cao</b> Dr. Daniel Pfeifle Dr. Timothy Aksamit</p>	<p><i>Hodgkin's Lymphoma; An Unusual Presentation with Budd-Chiari Syndrome</i></p> <p>Introduction: Budd-Chiari Syndrome (BCS) is a rare group of disorders characterized by hepatic venous outflow obstruction. It often can present non-specifically with symptoms including abdominal pain, ascites, and hepatomegaly. Most commonly, this is caused by hypercoagulable states resulting in occlusion of the venous outflow tract, but in rare cases can result from direct vascular compression or invasion associated with malignancy. Here, we describe BCS in a 37-year-old as a complication from a mediastinal mass due to Hodgkin's lymphoma, resulting in symptomatic obstruction and acute liver failure.</p> <p>Case Presentation: A 37-year-old female presented to the emergency department for acute onset shortness of breath and abdominal pain. Past medical history was significant for a recently found large 8.3 cm mediastinal mass, concerning for lymphoma, with biopsy results pending at presentation. On presentation, she had acutely elevated liver function testing including ALT of 855, AST of 1817, alkaline phosphatase of 142, total bilirubin of 4.1, and an elevated INR of 3.4. Notably, she also had a lactate of &gt;18. She underwent CT of the chest and abdomen due to concern for an ischemic process, which showed the large mediastinal mass encasing and occluding the IVC at the</p>

	<p>level of the diaphragm in addition to a new large thrombus within the intrahepatic IVC. She was started on a heparin infusion for initial treatment and admitted to the ICU.</p> <p>She was noted to have rapidly worsening liver enzymes during the first 24-hours of hospitalization. As the obstructing mass was suspected lymphoma, she was started on high-dose IV methylprednisolone to reduce tumor burden and help vascular flow. She was taken urgently for angioplasty and stent placement in the IVC, with measured improvement in hepatic to atrial gradient.</p> <p>Her liver function testing significantly improved with intervention. Biopsy results taken previously from the mediastinal mass demonstrated nodular sclerosing Hodgkin’s lymphoma, and with stabilization of her condition, she subsequently started chemotherapy during her hospitalization.</p> <p>Highlighted Points: This case highlights a rare etiology of Budd-Chiari Syndrome (BCS), resulting from vascular compression and thrombus formation secondary to Hodgkin’s lymphoma. BCS is most commonly due to intravascular thrombosis predisposed by hypercoagulable states—myeloproliferative disorders account for 50% of primary BCS—and less commonly due to malignancy-associated vascular compression or invasion.</p> <p>Conclusion: Symptoms of Budd-Chiari are often non-specific, requiring a high level of clinical suspicion to identify. The overall clinical course depends on the acuity and extent of vein occlusion, ranging from chronic asymptomatic to fulminant disease. For management, systemic anticoagulation remains the foundation of medical therapy. With severe disease, interventional approaches can be considered to improve hepatic outflow or reduce sinusoidal pressure, among them balloon angioplasty with or without stenting, placement of TIPS, or in rare occasions, liver transplantation.</p>
<p><b>Monazza Chaudhry</b> Dr. James Leatherman</p>	<p><i>Pulmonary Blastomycosis: A Familiar Foe in the Midwest</i></p> <p>Introduction: Pulmonary blastomycosis is one of the endemic fungal mycoses more commonly seen in immunocompromised individuals in the Midwestern United States. This case describes a subclinical infection masquerading as a lung abscess in a young, healthy individual.</p> <p>Case description: A 35 year old male presented to the emergency department with a 6 day history of mild fevers, cough, and myalgias. Initial vitals were significant for a temperature of 38.5 degrees Celsius and a WBC of 14. CXR was significant for a left sided opacity. Treatment for community acquired pneumonia was initiated and he was admitted for overnight observation. Over the next 72 hrs, he continued to be febrile and profusely diaphoretic with labs showing an up trending WBC up to 22, despite being optimized on broad spectrum antibiotics. Further chest imaging revealed a large necrotic nodular infiltrate initially concerning for a lung abscess. Upon further evaluation, it was noted that the patient had gone on a short hiking trip in Wisconsin two months prior and he had been feeling unwell for the past 2 weeks. Initial sputum cultures showed no growth. As the clinical suspicion was exceedingly high for a fungal etiology, further sets of induced sputum cultures were collected. Within 3 hours, budding yeasts were visualized.</p> <p>Conclusion: This case illustrates the importance of maintaining a high index</p>

	<p>of clinical suspicion and the judicious use of clinical judgement in an underrecognized presentation of a well-known, yet highly fatal, endemic disease.</p>
<p><b>Chansong Choi</b> Andrew Greenlund</p>	<p><i>An Unusual Tale of Small Bowel Edema</i></p> <p>Introduction: Angiotensin converting enzyme inhibitors (ACEI) is a common class of drug utilized to treat hypertension and other cardiovascular conditions such as heart failure. (1) Its mechanism involves the renin-angiotensin-aldosterone pathway as it inhibits the breakdown of bradykinin. This results in vasodilation, increased postcapillary venule permeability, and plasma extravasation to surrounding tissues. (1) Angioedema is rather infrequent adverse effect of the use of ACEI, as it is estimated to occur in 0.1-1% patients taking ACEI. (1,2) Classically, it is reported to involve the mucus membranes of the oropharynx, periorbital region, and upper airway potentially resulting in asphyxia. Less described is the manifestation of visceral angioedema involving the gastrointestinal (GI) tract, which is an under-recognized phenomenon. (1–3)</p> <p>Case Presentation: A 99-year old female with history of HTN on lisinopril presents to the ED with 1-day history of nausea, abdominal pain, and emesis. Physical exam was benign at the time of presentation. Laboratory evaluation was notable for leukocytes 11.7, hemoglobin 13.0g/dL, lipase 28, and unremarkable chemistry panel. Plain abdominal XR showed nonspecific bowel gas pattern. CT abdomen/pelvis revealed abdominal ascites in addition to wall thickening and edema involving loops of distal small bowel. Subsequent work up to assess for underlying autoimmune, infectious causes including celiac serologies, C4 and CRP level, QuantiFERON-TB were obtained which were negative. During hospitalization, lisinopril was discontinued in the setting of precipitous drop in hemoglobin concerning for GI bleed. Repeat CT scan in the setting of acute anemia 28 hours later showed decreased abdominal wall ascites with resolving ileal wall thickening and edema. There was no evidence of acute GI bleed on imaging, and hemoglobin drop was attributed to dilution. She was noted to have remarkable improvement in her symptoms with resolution of ileal wall edema and inflammation after the withdrawal of lisinopril.</p> <p>Conclusion: This case highlights an interesting case of visceral angioedema secondary to lisinopril use resulting in case of ileal edema and inflammation. Visceral angioedema is a rare phenomenon with 44 cases reported in the literature from 1982 to 2016. Most reported cases involve the use of either lisinopril or enalapril. (4) Patients usually present with nonspecific symptoms such as abdominal pain, nausea, emesis, or diarrhea. Anatomic location of visceral edema among reported cases has been variable, from antrum of the stomach to isolated segments of the small bowel. It is usually treated with supportive management including intravenous fluid replacement, antiemetics, and discontinuation of the offending agent. (2) Given medication induced visceral angioedema is a rare entity, clinicians should consider it as a diagnosis of exclusion and should first explore and rule out other differential diagnoses as vasculitis, small bowel lymphoma, and inflammatory bowel disease such as Crohn’s disease. (2)</p>
<p><b>John Christensen</b> Dr. Somaira Nowsheen Lauren Webb</p>	<p><i>Bartonella henselae endocarditis presenting as anemia, splenomegaly, and renal failure in an immunocompetent young man</i></p> <p>Case Presentation: A man in his mid-forties presented to our hospital with two</p>

<p>Dr. Maryam Mahmood</p>	<p>months of fever with chills, severe recurrent transfusion-dependent anemia, splenomegaly, and renal failure. He had no significant past medical history, and took no medications; he was in his usual state of health until approximately six months prior to presentation. He had no history of cardiac valvular disease or immunosuppression. He had had a very thorough workup with his local health care providers, including hematologists, nephrologists, infectious disease specialists, and rheumatologists. Investigations at our hospital demonstrated infection-associated glomerulonephritis and small, dense, thickened, sessile lesions on both the atrial and ventricular surfaces of the mitral valve; infectious workup ultimately revealed positive Bartonella serology. The patient was treated with a prolonged course of antimicrobials and ultimately underwent surgical replacement of his infected valves.</p> <p>Conclusion: Bartonellosis, caused by gram-negative intracellular facultative bacteria, can lead to severe complications, such as anemia, fever, hepatosplenomegaly, and endocarditis. Mortality approaches 40% without treatment. Therefore, prompt diagnosis and intervention are essential. Diagnosis of Bartonellosis relies on a combination of histological and microbiologic evaluations, including serological testing, culture, and nucleic acid amplification testing (NAAT). Conventional culture methods are not ideal for diagnosis as currently available tests have low sensitivity.</p> <p>Our case highlights the importance of considering Bartonellosis as a potential diagnosis even in immunocompetent individuals who have signs and symptoms suggestive of infective endocarditis, especially as the delay in diagnosis for our patient led to a prolonged clinical course with recurrent transfusions, kidney injury, and profound functional impairment prior to definitive treatment.</p>
<p><b>Molly Clark</b> Dr. James Campbell Dr. Nadeem Chaudhary</p>	<p><i>Breast Cancer Presenting as Painful Jaundice</i></p> <p>Introduction: Breast cancer is one of the most common forms of cancer affecting women, with 1:8 women being affected by breast cancer in their lifetime [3]. However, breast cancer metastasizing to the pancreas is very rare.</p> <p>Case Presentation: A 46 year old female presented to the ED with jaundice and right upper quadrant and epigastric pain radiating to her back, with 6 months of associated progressive fatigue, loss of appetite, nausea, vomiting, dark urine, and numbness in her toes. Of note, she also reported a small lump in her right breast that she had noticed about one year ago. Ultrasound of the abdomen revealed a large pancreatic mass and dilation of the bile ducts and pancreatic duct. Subsequent CT abdomen/pelvis with IV contrast done for further characterization of the masses showed a right breast mass, pancreatic head mass, bilateral adrenal masses, right pararenal mass, multiple mesenteric implants, innumerable subcutaneous masses, and mild retroperitoneal lymphadenopathy; no hepatic lesions were seen. Endoscopic ultrasound done for tissue biopsy revealed a large hypoechoic and heterogeneous round mass in the head of the pancreas with irregular outer margins causing obstruction and upstream dilation of the common bile duct. A similar appearing, but smaller, mass was seen in the left adrenal gland. Fine needle biopsy of the adrenal and pancreatic masses revealed signet ring morphology typical of pleomorphic lobular carcinoma and chromogranin expression more suggestive of pleomorphic lobular carcinoma. The tumor cells stained positive for GATA3 (the transcription factor binding to DNA sequence “GATA”) and estrogen receptors (ER+) supporting a breast primary. She was diagnosed with pleomorphic lobular carcinoma of the breast. While inpatient, she underwent</p>

	<p>ERCP for decompression of the biliary tree; however, the ampulla was unable to be located due to extrinsic pressure on the duodenal wall, thus percutaneous biliary drainage was performed for decompression of the “trapped” gallbladder. She was treated with 10 days of ceftriaxone (and later cefpodoxime) and metronidazole, was started on tamoxifen and goserelin, and plans were made for palliative radiation therapy.</p> <p>Conclusion: This case represents a rare case of breast cancer presenting as painful jaundice secondary to biliary obstruction from a large pancreatic metastasis. Invasive pleomorphic lobular carcinoma is an aggressive form of lobular carcinoma and more likely to have distant metastases and a lower median survival than invasive ductal carcinoma[2]; however pancreatic metastases are very rare[1]. When distinguishing between a primary pancreatic malignancy and a distant metastatic breast cancer, it is important to use immunohistochemical staining for GATA3, as this has high sensitivity for breast carcinoma[1].</p>
<p><b>Maros Cunderlik</b>  Dr. Alec Bunting  Dr. Amelia Krug  Dr. Fredrick Ogugua  Dr. Michele LeClaire  D. Abby Montague</p>	<p><i>A Case of Massive Caffeine Overdose With Refractory Shock Requiring Extracorporeal Membrane Oxygenation Support.</i></p> <p>Caffeine is ubiquitous in our society with 62% of the US population consuming caffeinated beverages daily and caffeine-containing compounds being increasingly used in energy drinks and supplements due to its desirable psychostimulant properties at low doses (&lt; 400mg). While life-threatening caffeine overdoses are rare, massive ingestions above the established lethal doses (150-200mg/kg, serum concentrations above 80µg/mL) have been reported with most deaths attributed to arrhythmias and cardiovascular collapse due to a unique caffeine toxicity profile.</p> <p>Here we present a case of a 34-year-old female with intentional massive caffeine overdose after ingestion of a guarana supplement containing approximately 20g of caffeine - twice the upper limit of the lethal dose. Shortly after the ingestion, the patient was altered requiring intubation for airway protection. She was also hypotensive with mean arterial pressures of approximately 40 mmHg and heart rhythm of wide complex tachycardia despite three cardioversion attempts. At the outside institution, the patient remained hemodynamically unstable despite aggressive medical interventions including amiodarone, intralipids, norepinephrine, vasopressin, and esmolol drips. She was subsequently transferred to our institution for consideration of veno-arterial extracorporeal membrane oxygenation support (VA ECMO) to facilitate emergent hemodialysis. On arrival, the patient was in severe multifactorial cardiogenic, vasodilatory and hypovolemic shock. She also had multiple metabolic derangements and had experienced status epilepticus. After stabilization with multiple pressors including angiotensin II infusion, electrolyte replacement, volume resuscitation and antiarrhythmic infusions she underwent VA ECMO cannulation and emergent hemodialysis with gradually improving hemodynamics over next 24 hours. On hospital day two, the patient was weaned off all ECMO and all vasoactive medications with complete recovery of cardiac function. Patient’s neurologic function also gradually improved and she was extubated on hospital day six. With continued supportive care, the patient achieved complete cardiovascular and neurologic recovery, she was discharged to the inpatient psychiatric unit seventeen days after the admission.</p> <p>In conclusion, we present a case of severe caffeine toxicity due to the intentional dietary supplement overdose resulting in severe multifactorial</p>

	<p>shock requiring VA ECMO resulting in eventual complete cardiovascular and neurologic recovery. Initial serum caffeine level was found to be 425 µg/mL. This is to our knowledge the highest reported caffeine serum level where the patient survived the ingestion.</p>
<p><b>Michael Dietrich</b></p>	<p><i>Malignancy Masquerading as Sinusitis</i></p> <p>Introduction: Malignant melanoma is extremely variable in initial presentation which can make early diagnosis and treatment challenging. Malignant melanoma of the nasal mucosa can present very similarly to sinusitis, which can add further difficulty to accurate diagnosis.</p> <p>Case Presentation: An 87 year old male with a past medical history of only hypertension noticed blood in his nasal mucus after blowing his nose. He had no significant history of allergies, atopy, epistaxis, nasal trauma or chronic rhinosinusitis. Bleeding continued to worsen over the next few months and he developed new nasal congestion despite multiple nasal decongestants. He presented to an urgent care clinic and was prescribed an anti-histamine with continued vasoconstrictors/decongestants for presumed chronic sinusitis. However, he represented to outpatient clinic 1 week later due to worsening of his symptoms, including new feelings of nasal obstruction.</p> <p>Laboratory studies including complete blood count, coagulation studies, and erythrocyte sedimentation rate were ordered and within normal range. Computerized tomography (CT) sinus without contrast showed an expansile mass in the right posterior nasal cavity. The patient was referred to otolaryngology who performed rhinoscopy with biopsies of the mass. Follow up magnetic resonance imaging (MRI) to better characterize the lesion redemonstrated a mass within the posterior nasal cavity which extended into the nasopharynx, without extension through the right sphenopalatine foramen. Initial frozen pathology showed poorly differentiated malignant neoplasm. Positron emission tomography (PET)-CT scan did not show evidence of regional or metastatic disease. Therefore, patient was taken for endoscopic resection about 7 days after initial diagnosis. Final pathology showed extensively necrotic 4.5cm malignant mucosal melanoma of the right nasal septum with extension into the right middle turbinate.</p> <p>Resection was performed without complication and he also underwent adjuvant chemotherapy and radiation. However, 4 months later a follow up PET-CT scan showed right cervical nodal metastasis which was confirmed to be malignant melanoma on biopsy. Surgical modified neck dissection was performed, lymph nodes positive for metastatic malignant melanoma. Again adjuvant immunotherapy and radiation were performed. Unfortunately, repeat PET-CT scan showed recurrent disease locally in the neck and evidence of metastatic disease in the abdomen. The patient decided not to pursue further systemic treatment.</p> <p>Conclusion: This case demonstrates the variability in presentation of malignant melanoma. Here we see nasal mucosal malignant melanoma initially presenting as blood tinged mucus and symptoms of nasal congestion. Therefore, it is important to keep a wide differential for symptoms of sinusitis, especially given that mucosal melanoma can be a very aggressive subtype of melanoma.</p>
<p><b>Kiril Dimitrov</b> Dr. Veronika</p>	<p><i>Recognizing Cytokine Release Syndrome and Neurotoxicity as Side Effects of CAR-T Therapy: Mimics of Sepsis and Stroke</i></p>



<p>Bachanova</p>	<p>Introduction: Chimeric Antigen Receptor T-cell Therapy (CAR-T) has been an important breakthrough in treatment of several hematologic malignancies. CAR-T cells are autologous T lymphocytes genetically modified to express novel protein to facilitate targeting of tumor cells. Several CAR-T products are now FDA approved and there is increasing recognition of their unique side effects. More prevalent application of CAR-T will require hospitalists to differentiate sequelae from other common conditions seen in cancer patients.</p> <p>Case Presentation: A 56-year-old female with Diffuse Large B-Cell Lymphoma received lymphodepleting chemotherapy followed by CAR-T19 cell therapy axicabtagene ciloleucel (Yescarta) for relapsed disease. On Day +6 patient developed neutropenic fevers to 101.6°F and hypotension that was responsive to IV fluids. BMP, CBC with differential, blood cultures, chest X-ray, urinalysis/culture, and inflammatory markers (IL6, Ferritin, and CRP) were obtained. CXR was negative for infection and blood/urine cultures showed no growth. CRP and IL-6 levels were elevated. Differential diagnosis of elevated temperature and hypotension in a post CAR-T patient includes sepsis and cytokine release syndrome (CRS). Patient was treated with IV antibiotics, but fever persisted with intermittent hypotension. In the setting of CRS Grade 2, patient received one dose of Tocilizumab and transferred to the ICU for potential pressor support. Fevers and hypotension resolved the same day and she became hemodynamically stable. She then presented with mild intention tremor and trouble writing. Patient subsequently developed acute left forearm numbness, impaired attention, confusion, and global aphasia but her level of consciousness was not impaired. Considerations at that time were acute stroke vs Immune effector-Cell Associated Neurotoxicity (ICANS) Grade 3 from CAR-T therapy. Repeat MRI was without stroke. EEG showed mild-moderate encephalopathy. Treatment included IV dexamethasone and prophylactic Keppra. Patient promptly responded and within 24 hours her symptoms improved. She was discharged in the next few days on a steroid taper and with Grade 0 CRS/ICANS.</p> <p>CRS is characterized by rapid release of pro-inflammatory cytokines including IL-6 and IFN-<math>\gamma</math>, occurring 2-5 days after CAR-T infusion, and is triggered by activated CAR-T cells. Clinical manifestations include high fever, myalgias/arthralgias, hypotension, dyspnea, and capillary leak. Grading for CRS (1-4) includes varying degrees of fever, hypotension, and hypoxia. Treatment includes supportive cares with fluids, antipyretics, and administration of IL-6 receptor antibody, Tocilizumab. Vasopressors and ventilatory support may be required.</p> <p>Conclusion: Immune effector Cell-Associated Neurotoxicity Syndrome (ICANS) pathophysiology is distinct from CRS. Symptoms include headaches, tremors, impaired attention, lethargy, aphasia, agraphia, confusion, seizures, and increased intracranial pressure. Grading for ICANS includes varying degrees (1-4) of decreased level of consciousness, seizures, motor weakness, and increased cerebral edema. Steroids are the mainstay of neurotoxicity treatment and yield quick improvement if instituted early. For grade 4 ICANS, decrease of ICP with mechanical ventilation, mannitol/acetazolamide may be required, and neurological consultation is recommended.</p>
<p><b>Christopher Dinh</b> Dr. Hannah Nordhues</p>	<p><i>A Woman with Urothelial Carcinoma and Abdominal Pain</i></p> <p>Case Presentation: A 73-year-old female with a past medical history significant for metastatic high-grade urothelial carcinoma of the right kidney</p>

presented with three weeks of progressive abdominal pain, dyspnea, and urinary symptoms. Evaluation revealed total calcium of 12.4 mg/dL, ionized calcium of 6.21 mg/dL, and elevated liver enzymes of AST 66, ALT 60, and alkaline phosphatase 227. CT chest, abdomen, and pelvis revealed marked progression of the liver, lung, and small bowel metastases. She was admitted to the hospital for further management.

Additional laboratory evaluation was significant for normal vitamin D levels of 36 ng/mL, appropriately suppressed parathyroid hormone (PTH) of 8.6 pg/mL, and elevated parathyroid hormone-related peptide (PTHrP) of 8.6 pmol/L. Her abdominal pain was multifactorial from both her significant malignant disease burden and symptomatic hypercalcemia. Her hypercalcemia was treated with IV fluids and zoledronic acid, with partial improvement of her abdominal pain. She was subsequently discharged from the hospital with close oncologic follow-up.

Discussion: Hypercalcemia is a common clinical scenario with a variety of symptoms and etiologies. Hypercalcemia affects numerous organ systems including renal, musculoskeletal, gastrointestinal, neuropsychiatric, and cardiovascular.

A systematic approach to hypercalcemia is essential in making the correct diagnosis. Although many etiologies for hypercalcemia exist, primary hyperparathyroidism and malignancy account for 90% of all cases. Initial evaluation includes repeat calcium (corrected total calcium or ionized calcium), intact PTH, and vitamin D levels. Additional studies depend on underlying clinical suspicion and may include PTHrP for humoral hypercalcemia of malignancy, 1,25-dihydroxyvitamin D for granulomatous diseases, or serum and urine protein electrophoresis for plasma cell disorders including multiple myeloma. Common medication culprits include thiazide diuretics, lithium, PTH-analogs, and excess vitamin D supplementation, so medication reconciliation is essential.

Malignant hypercalcemia is seen in up to 20-30% of patients with malignancy and portends a poor prognosis. The three major mechanisms of malignant hypercalcemia include humoral production of PTHrP, osteolytic skeletal metastases, and 1,25-dihydroxyvitamin D production via 1-alpha-hydroxylase. In rare case reports, hypercalcemia may be driven by tumor production of intact PTH. In this case, the patient's progressive urothelial carcinoma led to PTHrP production.

Regardless of the etiology, initial treatment for severe or symptomatic hypercalcemia begins with high-volume crystalloid fluid resuscitation at approximately 200-300 mL/hour. Depending on the underlying etiology, additional treatment agents may include calcitonin, denosumab, bisphosphonates, glucocorticoids, or dialysis in refractory cases. Identification and treatment of the underlying etiology is essential.

Conclusion: Hypercalcemia is a frequently encountered clinical problem and warrants systemic evaluation starting with repeat calcium, PTH, and vitamin D levels. Ninety percent of cases are caused by primary hyperparathyroidism or malignancy. Severe or symptomatic hypercalcemia is treated with intravenous fluid resuscitation, multiple adjunctive agents, and correcting the underlying etiology.

**Michael Downey**

*Masquerading Hearts: Stress Cardiomyopathy Masking Apical Variant*

<p>Dr. Brandon Boyle Dr. Andrew Olson</p>	<p><i>Hypertrophic Cardiomyopathy</i></p>
	<p>Case Presentation: A 72 year old female presented with recurrent chest pain after an angiographic supported diagnosis of stress cardiomyopathy 1 month prior. On readmission, evaluation was notable for new electrocardiographic findings of T-wave inversions most pronounced in anterio-apical leads and echocardiogram demonstrating recovered ejection fraction but new apical hypertrophy – all consistent with hypertrophic cardiomyopathy. Cardiac MR confirmed hypertrophy without evidence of late enhancement. The patient was admitted for ongoing cardiac monitoring without incident and was eventually discharged on ACEi and beta-blocker therapy with serial echocardiogram and cardiology follow up. At 4 weeks follow-up, patient remained asymptomatic with consistently recovered ejection fraction and hypertrophic cardiac apex.</p> <p>Discussion: This case covers two distinct cardiomyopathies both presenting in the same patient. Intriguingly, these pathologies cover both overlapping and distinct learning points. First, stress cardiomyopathy can have recurrent pain following initial presentation. Second, in all patients with stress cardiomyopathy, a follow up TTE is recommended to assess not only recovery of ejection fraction but also for unmasking of other potential rare cardiac abnormalities obscured by diffuse process of stress cardiomyopathy (e.g. hypertrophic cardiomyopathy, underlying regional WMA). Third, both stress cardiomyopathy and hypertrophic cardiomyopathy, individually, are at risk for developing left ventricular outflow obstructive shock which must be recognized early as it is traditional management of hypotension / distributive shock will only worsen these patients. Finally, this case serves as a general reminder that patients may have multiple pathologies, all at different stages, wherein one can transiently obscure the other. Making time and serial evaluations a powerful diagnostic tool of the internist.</p>
<p><b>Nouran Felo</b> Dr. John Bundrick</p>	<p><i>Parsonage-Turner Syndrome or Neurolymphomatosis?</i></p> <p>Introduction: Neurolymphomatosis is a rare manifestation of non-Hodgkin’s lymphoma in which malignant lymphoma cells infiltrate the central or peripheral nervous system, mimicking Parsonage –Turner Syndrome (PTS). PTS, or brachial neuritis, is a rare disorder with symptoms of progressive weakness and sensory abnormalities in the proximal upper extremities secondary to trauma, post viral illness, or multiple other etiologies. It is important to differentiate between the two for treatment and prognosis.</p> <p>Case Presentation: A 73-year-old man presented to the hospital with progressively worsening asymmetric weakness of his upper and lower extremities with paresthesia, difficulties performing activities of daily living, dyspnea, and hypoxia 5 months after recovering from a viral illness. A year prior, the patient developed weakness, increased falls, and changes in his behavior, leading to a diagnosis of mild cognitive impairment with concern for early-onset Alzheimer’s and was started on Aricept.</p> <p>On admission, he was found to have both upper and lower motor neuron signs, bulbar and diaphragmatic weakness, and a notable decrease in range of motion and strength of his right upper extremity. Given his constellation of symptoms, there was concern for PTS versus ALS. CSF panel showed elevated protein and EMG was consistent with axonal polyradiculoneuropathy which ruled out ALS. PET-CT showed uptake in the paranasal sinuses, adrenal glands, brachial plexus, sacral nerves, and liver. It also showed</p>

	<p>lymphadenopathy in the inguinal, pelvic sidewall, and upper retroperitoneal stations. Additionally, he had brachial nerve enhancement on MRI.</p> <p>Ground-glass opacities of the lung, enhancement of paranasal sinuses, and polyneuropathy in the setting of a positive c-ANCA and PR3 were concerning for a vasculitic process. Further evaluation included an immunologic workup and a nasal cavity mucosal biopsy which were negative. The positive c-ANCA and PR3 antibody were thought to be falsely elevated in the setting of a potential malignancy.</p> <p>Left inguinal lymph node biopsy showed Diffuse Large B-cell Lymphoma (DLBCL) with prominent intravascular infiltrates, a germinal center B-cell phenotype without MYC gene rearrangement. He was subsequently transferred to the hematology service to initiate MR-CHOP chemotherapy.</p> <p>Discussion: This complex case highlights the importance of maintaining a broad differential diagnosis and enlisting a multi-disciplinary approach to ensure best care for the patient. The patient’s presentation with asymmetric weakness, focal neurologic deficits, and laboratory abnormalities led us in many directions, with the ultimate diagnosis being neurolymphomatosis in the setting of intravascular DLBCL and falsely elevated vasculitis labs. Neurolymphomatosis is rare and frequently misdiagnosed; however, the treatment plan is curative, whereas PTS would be treated symptomatically. Good patient outcomes and quality of life are the goal, and although the diagnosis was not clear at first, we were able to ensure our patient received the right treatment for his condition.</p>
<p><b>Ellen Gardner</b>  Dr. Fatima Hassan  Dr. Ruth Verner  Dr. David Griffin</p>	<p><i>Mad as a Hatter: the Infectious Quandary</i></p> <p>Introduction: Histoplasma capsulatum is a dimorphic fungus found in soil contaminated with bird or bat droppings. It is found worldwide, most commonly in the Central United States. Histoplasmosis is usually self-limiting in immunocompetent individuals, typically causing isolated pneumonia. Presented is a case of disseminated histoplasmosis in an immunocompetent patient.</p> <p>Case Presentation: 58 year old male with history of hairy cell leukemia (treated with cladribine; in remission since 2017) presented for evaluation of syncope. Six months prior to hospitalization, he had been evaluated for odynophagia, which had been attributed to periodontal disease and rhinosinusitis. He was treated with antibiotics and prednisone. After no improvement, further imaging demonstrated epiglottitis leading to epiglottitis biopsy showed non-necrotizing granulomas with yeast consistent with histoplasmosis, but no cultures were obtained. He was prescribed a course of oral fluconazole. Approximately 15 weeks later, he presented to the ED with complaints of weight loss, headache and syncope and found to be encephalopathic. He was admitted for syncope evaluation.</p> <p>On first day of hospitalization, he exhibited psychotic features and became febrile, thus started on empiric coverage for bacterial and viral CNS infection. CSF was obtained with glucose 25, protein 80, WBC 60 (68% PMN and 14% mono), RBC 5; negative gram stain, cytology, infectious and paraneoplastic panel. Infectious disease was consulted to discuss the previous epiglottitis biopsy. Their impression was that yeast forms are common in oropharynx, lack of sufficient evidence to diagnose histoplasmosis, and no risk factors for disseminated histoplasmosis, thus toxicities of amphotericin outweighed</p>

	<p>benefit of empiric treatment. A brain MRI showed enhancing nodules of the cerebral and cerebellar hemispheres suspicious for brain parenchymal involvement of leukemia. Hematology was consulted with impression that recurrence of hairy cell leukemia was unlikely and more likely was new primary malignancy or side effect from cladribine. No evidence of occult neoplasm was found on further investigation.</p> <p>Additional CSF obtained for flow cytometry showed protein 88; glucose 12; WBC 256 (86% PMN) and negative for malignancy. Broad work-up continued leading to biopsy of new oral ulcers with preliminary pathology showing budding yeast concerning for histoplasmosis.</p> <p>Patient was started on amphotericin. His CSF and serum resulted several days after initiation of therapy with highly positive histoplasmosis. With appropriate treatment, his mental status returned to baseline over next few days.</p> <p>Discussion: This case highlights the importance of considering disseminated histoplasmosis in endemic areas as cause of acute encephalopathy. While typically included on the differential of those with traditional risk factors, dissemination fungal infections can occur in the immunocompetent patient. With enough supportive evidence of disseminated histoplasmosis empiric amphotericin should be initiated, as waiting for confirmatory results would lead to delay in appropriate treatment.</p>
<p><b>Jennifer Gile</b>  Dr. Chineze Akusoba  Dr. Douglas Challener  Dr. MarkENZler</p>	<p><i>Hemophagocytic Lymphohistiocytosis Caused by Disseminated Histoplasmosis</i></p> <p>Case Presentation: A 43-year-old female with medical comorbidities including multifocal choroiditis with panuveitis on adalimumab and mycophenolate was hospitalized locally with nausea, vomiting, fevers, headaches and night sweats of 2 weeks duration. Laboratory workup was significant for pancytopenia, transaminitis and elevated inflammatory markers. CT abdomen/pelvis revealed hepatosplenomegaly with diffuse hepatosteatosis. CT Chest demonstrated a rounded 1.4 cm ground-glass opacity in the left upper lobe and mediastinal adenopathy. A peripheral smear demonstrated burr cells and smudge cells. Initial ferritin was &gt;5000 mcg/L (normal 11-307 mcg/L). Antinuclear antibody, double-stranded DNA, antineutrophil cytoplasmic antibodies and rheumatoid factor were negative. Lumbar puncture revealed elevated opening pressure and protein. Cerebrospinal fluid testing including cell count, glucose, protein, JC virus/Lyme PCR /West Nile virus/ HHV6 PCRs was negative/normal. Testing for tick borne illness, viral hepatitis, CMV and EBV were negative. The patient was transferred to our institution for further workup. Blastomyces, Bartonella, and Coxiella serologies were obtained and negative. Histoplasma urine antigen was &gt;23 ng/mL (normal 0-0.1 ng/mL) confirming our suspicion that the patient had disseminated histoplasmosis (DH). Subsequent fungal blood cultures grew Histoplasma capsulatum and Histoplasma serology antibody complement fixation serologies were positive: yeast 1:128, mycelial 1:8 and both H &amp; M bands were present. Repeat ferritin was &gt;11,000 mcg/L. Hematology was consulted with concern for hemophagocytic lymphohistiocytosis (HLH) secondary to DH infection since she fulfilled 5 of 8 diagnostic criteria for HLH: fever <math>\geq 38.5^{\circ}\text{C}</math>, splenomegaly, <math>\geq 2</math> cytopenias, ferritin level &gt;500 ng/mL, and hypofibrinogenemia. Bone marrow biopsy demonstrated normocellular bone marrow and hemophagocytosis of red cell precursors and white cells and yeast forms were visualized on silver stain. The patient was diagnosed with HLH secondary to DH in the setting of immune</p>

	<p>deficiency. She received high-dose IV steroids for treatment of HLH and IV liposomal amphotericin B for induction treatment of the histoplasmosis with an excellent clinical response.</p> <p>Discussion: HLH is a syndrome of pathologic immune activation and is classified as primary or secondary. Secondary causes of HLH include malignancies and infections associated with Epstein Barr Virus and H. capsulatum. Diagnosis of HLH is suspected based on presentation and laboratory abnormalities, as no single definitive diagnostic test exists. Initial treatment of HLH involves the use of steroids to block the hyper-inflammatory response and treatment of the underlying process if one is found.</p> <p>DH has emerged as one the second most common infectious cause of HLH in some case series. The patient was exposed to H. capsulatum during her childhood in which she lived on a farm in the Midwest. Her presentation is consistent with recrudescent histoplasmosis given her immunocompromised status. Histoplasmosis should be suspected in patients with risk factors for disease, fever, fatigue, weight loss, cytopenias, adenopathy, splenomegaly and elevated liver function tests.</p>
<p><b>Stephanie Grach</b> Rohit Divekar</p>	<p><i>An Unfortunate Minnesotan: Diagnosis and Management of Cold Urticaria</i></p> <p>Introduction: Cold urticaria is a rare allergic reaction. Individuals may experience hives (urticaria), erythema, pruritis, or angioedema on exposure to cold weather, fluids, or objects. Initial management involves use of antihistamines scheduled and as needed. Epinephrine auto-injectors should be prescribed to all cold urticaria patients in case of a life-threatening exposure.</p> <p>Case Presentation: A 27 y.o. female presented to clinic for recurrent pruritic rash. She first noticed itchiness in her palms 3 months prior while holding cold cans. 1 month prior to presentation, she was holding a bottle of water in her arm when she began to have not only itching, but also swelling and erythema at the location of the water bottle. This phenomenon was reproducible with pressing a cold spoon against her skin. During cooler months, she began experiencing a generalized warmth and erythema, accompanied by hot, red, painful ears for 1 hour on one occasion. Additional symptoms included tingling at the lips with use of a metal straw, and chest pain following the ingestion of ice cream. She started taking cetirizine 10 mg daily which reduced erythema and swelling, though some pruritus persisted.</p> <p>Diagnostic Findings: Cryoglobulins, protein electrophoresis, lactate dehydrogenase, creatinine, CBC, CRP, and ALT were obtained and unremarkable. Patient photographs confirmed the localized nature of swelling in response to cold contact. Given her presentation and negative laboratory workup, she was diagnosed with cold urticaria.</p> <p>Management: The patient was educated on cold avoidance and high-risk activities, including consumption of cold food or beverages, use of cold IV fluids, and participation in cold water activities such as polar plunges. Medical therapy for prevention was described, and she opted to continue cetirizine 10mg daily with plan to increase to cetirizine 10mg twice daily if needed. An epinephrine auto-injector was prescribed in case of emergencies.</p> <p>Discussion: Cold urticaria is a form of inducible physical urticaria in which exposure to cold substances, objects, or air can result in a localized or</p>

	<p>systemic reaction. It is a rare condition with estimated incidence of 0.05%, with approximately 50% of cases improving or resolving within 5 years of onset. The diagnosis can be made via ice cube testing, in which ice is held against the skin: if swelling and erythema arise within minutes of exposure, the patient is deemed to have cold urticaria. In this case, the patient had performed similar maneuvers with positive results, which was sufficient. Antihistamines are first-line for preventative medical therapy. In cases of refractory symptoms, prednisone, omalizumab, or cyclosporine may be considered. Throat or whole body exposures to extreme cold may be fatal without treatment; as such, an epinephrine auto-injector should be prescribed on diagnosis. Patients should receive education on how to reduce risk of exacerbations through reducing exposures.</p>
<p><b>Alexandra Hall</b></p>	<p><i>Broadening the Differential Diagnosis for Ahock: An atypical cause of obstruction</i></p> <p>Introduction: Shock represents a physiologic state on the pathway to cardiovascular failure. As such, outcomes are highly dependent on early detection and targeted treatment of the underlying condition. While septic shock is the most common subset, it is imperative to consider alternative pathologies. Here we present a patient who developed shock due to inferior vena cava obstruction secondary to extensive DVTs.</p> <p>Case Presentation: A 56 year old male with a history of stroke, prior deep vein thromboses and pulmonary emboli (on chronic anticoagulation), hypertension, and diabetes presented with progressive pelvic pain and hematuria. Symptoms of straining and pelvic discomfort had begun two weeks prior. He was prescribed antibiotics for prostatitis in clinic soon after symptoms began and had a foley catheter placed in the following days for urinary retention. The patient returned to seek medical care when his pain began worsening with walking and after he experienced a single episode of hematochezia. Initial vital signs were within normal limits: temperature 97.7, heart rate 72, blood pressure 100/66, respiratory rate 16, oxygen saturation 96% on room air. Mild generalized pelvic tenderness was present on exam. Labs were notable for acute kidney injury and subtherapeutic INR. He was started on antibiotics, anticoagulation, and IV fluids. Over the next 12 hours he developed numbness in his lower extremities, became progressively hypotensive (76/47), and was found to have a lactic acidosis. Subsequent CTA of his chest/abdomen/pelvis revealed nonocclusive pulmonary emboli in his lungs and high suspicion for IVC thrombus. Lower extremity ultrasound was notable for extensive occlusive DVT extending from the distal IVC through both common iliac veins to the calves. The patient underwent bilateral lower extremity and IVC thrombectomy as well as removal of an infrarenal IVC filter with resolution of his hypotension.</p> <p>Conclusion: Undifferentiated shock warrants a rapid and thorough workup to optimize health outcomes. While empiric treatment of sepsis, when considered a fitting possibility, is a common initial first step, further investigation is needed in many cases. Obstructive shock is a type of hypodynamic shock resulting in increased systemic vascular resistance and decreased cardiac output. While this is most commonly associated with pulmonary embolism, pericardial tamponade, and tension pneumothorax, catastrophic venous occlusion can cause this physiology as well. Complete obstruction of the inferior vena cava, for instance, represents a significant obstacle that effectively decreases venous return and preload, thus impairing cardiac output and effective arterial blood volume. In such cases, timely</p>

	removal of the thrombus is crucial. Furthermore, the identification of predisposing factors is indicated for secondary prevention.
<p><b>Naima Hashi</b> Dr. Sagar Rakshit Dr. Konstantinos Leventakos</p>	<p><i>Etomidate: Bridge to Adrenalectomy in Paraneoplastic Cushing's Syndrome</i></p> <p>Introduction: Ectopic production of adrenal corticotropin (ACTH) can cause Cushing syndrome. Patients typically present with indolent muscle weakness, hypertension, central obesity, osteoporosis, hypokalemic alkalosis, and hyperglycemia. Paraneoplastic Cushing's syndrome is a rare cause of endogenous hypercortisolism and is seen in up to 5% of patients with small cell lung cancer. Patients can present with severe and life-threatening hypercortisolism. Medical therapy for severe hypercortisolemia is challenging and options are limited.</p> <p>Case Description: A 67-year-old woman with known extensive-stage small cell lung cancer was admitted to the hospital with severe hypertension, hypernatremia, hypokalemia, and hyperglycemia. Labs revealed an elevated ACTH of 145 (7.2 – 63 pg/mL) and morning cortisol of 46 (4.8 – 20 mcg/dL) which were consistent with ectopic Cushing's syndrome secondary to small cell lung cancer.</p> <p>She was started on ketoconazole 400 mg 3 times daily. Her blood pressure, electrolytes, and blood glucose normalized within 3 days on treatment. Morning cortisol was 20 mcg/dL at the time of discharge. Unfortunately, she was re-hospitalized 3-weeks later with similar symptoms while still on treatment with ketoconazole. Morning cortisol was now notably elevated at 50 mcg/dL. After failing medical therapy, adrenalectomy was recommended for definitive management. Surgery had to be delayed by 5 days due to the use of apixaban. There was significant concern that she would rapidly deteriorate prior to surgery.</p> <p>The decision was made to start her on an etomidate infusion as a bridge to surgery. She was transferred to the ICU and started on etomidate 0.04 mg/kg/hr with a goal cortisol of 18-29 mcg/dL. Cortisol levels were monitored every 6 hours. She reported no sedating effects from the etomidate and her am cortisol after 24 hours reduced to 21 mcg/dL. Her symptoms improved with the reduction of cortisol and she was able to undergo bilateral adrenalectomy. She was discharged in stable condition and is currently on treatment with hydrocortisone and fludrocortisone.</p> <p>Discussion: Etomidate is an imidazole derivative similar to ketoconazole that decreases steroidogenesis in the adrenal gland. Etomidate is an effective medication for patients who are refractory to ketoconazole and who present with life-threatening sequela associated with Cushing's syndrome. Etomidate infusion rapidly reduces cortisol level helping to stabilize patient's symptoms, and hopefully bridge them to surgery. We suggest starting an initial dose of 0.04 mg/kg/hr and titrating the dose based on daily morning cortisol levels.</p>
<p><b>Ashley Hickman</b> Dr. Carl Andersen Dr. Kelley Dages</p>	<p><i>Is That Possible? A Presentation of Erythrocytosis</i></p> <p>Introduction: Interpreting abnormal lab values is an everyday job of the Internist. Erythrocytosis, an isolated elevation of red blood cells, can occur in a variety of conditions encountered in the primary care setting. A relative erythrocytosis is due to volume depletion. An absolute increase in red blood cell mass can be due to a genetic mutation in red blood progenitor cells (primary) or due to another medical condition (secondary). Secondary</p>



	<p>erythrocytosis is caused by conditions including smoking, cardiopulmonary disease, erythropoietin-producing tumors, testosterone replacement, and living at high altitudes. This case presents a patient with a significant elevation in hematocrit and venous thrombosis, which calls into question a primary versus secondary cause of erythrocytosis. This case also demonstrates a laboratory phenomenon illustrating the often multifactorial nature of a smoker's polycythemia.</p> <p>Case Presentation: A 43 year-old male presented to clinic with a hemoglobin of 23.9 g/dL (13.2 - 16.6 g/dL) and hematocrit of 66.4% (38.3 - 48.6 %). He smoked 1 pack per day for 26 years, had active alcohol abuse, and a BMI of 18. He had a ruddy appearance and oxygen saturations of 96% on room air. An erythropoietin level was 6.7 mIU/mL (2.6 - 18.5 mIU/mL). He was suspected to have a secondary erythrocytosis due to smoking; however, it was unclear if this alone could result in such a significant hematocrit. An overnight oximetry showed a low baseline with periodic desaturations. One month later, he was hospitalized with pancreatitis and found to have an acute mesenteric vein thrombosis. Lab work on admission showed a hemoglobin/hematocrit of 22.4 g/dL /61.2%. Due to the significant hematocrit with the presence of thrombus, he underwent investigation for polycythemia vera. JAK 2 V617F Mutation Analysis and JAK 2 Exons 12-15 Genetic Alteration Analysis were negative. During the four-day hospitalization, his hematocrit decreased by 20% with fluid resuscitation and smoking cessation. On day of discharge, hemoglobin/hematocrit was 17.4 g/dL /49.1%.</p> <p>Conclusion: This case illustrates several common causes of erythrocytosis including smoking, pulmonary gas exchange dysfunction, volume depletion, and sleep-related breathing disorder. Surprises seen with this case include the degree of hematocrit elevation and abnormal overnight oximetry despite a low suspicion for sleep-related breathing disorder. Also seen was the red herring of venous thrombosis, which was more likely related to pancreatitis and tobacco use rather than erythrocytosis. Smoker's polycythemia is a commonly recognized phenomenon that presents as a mixed relative and absolute polycythemia. Due to the relative component, there is typically a &gt;4% decrease in hematocrit within days of smoking cessation. Treatment of secondary erythrocytosis is aimed at correcting the underlying cause. Routine phlebotomy is not recommended in secondary erythrocytosis because, in contrast to primary erythrocytosis, the risk of thrombosis is not thought to be elevated by the polycythemia itself.</p>
<p><b>Alexander Hines</b> Dr. Chansong Choi</p>	<p><i>Capecitabine Induced Vasospastic Angina</i></p> <p>Introduction: Coronary vasospastic angina is defined as angina which is (1) nitrate responsive (2) accompanied by transient ECG changes in at least two contiguous leads and (3) produced by coronary artery spasm, defined as transient total or subtotal occlusion of coronary artery (&gt;90% constriction). Persistent or prolonged vasospasm can induce ischemia, potentially leading to type II myocardial infarction (MI) in some patients. Type II MI is attributed to an imbalance between oxygen supply and demand, not due to acute atherothrombotic coronary artery disease. Ischemic effects vary significantly according to the magnitude of the stressor, potential underlying coronary artery disease and cardiac structural abnormalities, and the presence of non-cardiac comorbidities.</p> <p>Case Presentation: 75 year old woman with recently diagnosed rectal adenocarcinoma (Stage T3b, N+) presented to the Emergency Department</p>

	<p>with 3 day history of pressure like substernal chest pain with radiation to her right arm and diaphoresis. On the morning of her presentation, she developed sudden shortness of breath and substernal chest pain upon climbing a flight of stairs. She received her first dose of radiosensitizing capecitabine (500mg TID) two days prior to onset of initial symptoms. Blood assay was notable for hs-Troponin level of 12 with further elevation to 36 at 2-hour recheck. EKG showed no aberrant rhythm and no concerning ST segment changes. Coronary angiography revealed mild coronary artery atherosclerosis (most notable for 50% obstruction of distal LAD) without evidence of any culprit lesions. She was diagnosed with vasospastic angina secondary to capecitabine and discharged home on long acting nitrates and with same-day oncology clinic follow-up.</p> <p>Conclusion: This case portrays the importance of developing a broad differential diagnosis when encountering patients presenting with angina. Chest pain in younger persons, those without significant coronary artery disease risk factors, or angina triggered in the context of recent addition of one or more medications should prompt clinicians to consider non-CAD etiologies of angina. Finally, clinicians should be aware of effects of capecitabine and its rare but clinically significant association with vasospastic angina. Capecitabine, an oral prodrug of 5-fluorouracil (5-FU), is thymidine phosphorylase-activated fluoropyrimidine carbamate which undergoes conversion to 5-FU following three enzymatic cascades. Bioavailability after oral administration is reported close to 100 percent. It is typically associated with GI and dermatologic adverse effects, but cardiotoxicity has been described in few case reports. Capecitabine is suggested to reduce the risk of chest pain by half compared to 5-FU (approximately 1-18% in exposed patients). Presentation can vary from resting or exertional dyspnea to abnormal noninvasive stress testing. The chest pain often has a quick onset as it relates to changes in molecular signaling pathways involved in vascular smooth muscle tone with subsequent changes in vasoconstriction and vascular reactivity.</p>
<p><b>Jason Hoard</b> Dr. Kristina Krohn Dr. Stephen Selinsky</p>	<p><i>Thromboembolism in the Setting of Systemic Bacillus Cereus Infection</i></p> <p>Introduction: Bacillus cereus is a Gram-positive spore-forming rod widely found in the environment. It commonly causes foodborne gastrointestinal disease but is also known to cause more invasive disease including endophthalmitis, endocarditis, bacteremia, and severe pneumonia.</p> <p>Case Presentation: A 15-year-old male with obesity was admitted to the hospital with four weeks of intermittent fevers, emesis, and dyspnea. He was found to have right lower leg pain and swelling, and an ultrasound revealed a right lower extremity deep vein thrombosis. Chest CT with angiography demonstrated extensive bilateral pulmonary embolisms, left lower lobe consolidation, and bilateral pleural effusions. He was started on a heparin infusion, as well as ceftriaxone and azithromycin. Bilateral chest tubes were placed to drain the effusions. An extensive infectious workup was initially negative. However, a Karius molecular diagnostic test returned positive for B. cereus in the blood, and pleural fluid was positive for B. cereus ribosomal DNA. His antibiotics were changed to vancomycin, and he improved clinically. The patient also underwent hematologic and rheumatologic evaluation due to his apparent hypercoagulable state. This included a bone marrow biopsy, which was negative for malignancy. His workup also included genomic sequencing, which demonstrated that the patient was heterozygous for a Factor V gene mutation. He was transitioned to enoxaparin</p>

	<p>for the venous thromboembolisms and was discharged on oral linezolid to complete a 6-week treatment course. The patient’s thromboembolisms were ultimately attributed to his Factor V heterozygosity in the setting of obesity and a significantly proinflammatory state due to systemic <i>B. cereus</i> infection with bacteremia and complicated pneumonia.</p> <p>Conclusion: This case illustrates the potential for severe, systemic disease due to <i>B. cereus</i>, the role of molecular diagnostic testing to identify pathogens when an infectious process is suspected, and the importance of testing for hypercoagulable disorders in young patients with unexplained thromboembolic events.</p>
<p><b>Danielle Hron</b> Dr. Charles Billington</p>	<p><i>Take the High-Road: Navigating Post-Roux-en-Y Bypass Hypoglycemia and Psychiatric Illness</i></p> <p>Introduction: Post-Roux-en-Y gastric bypass hypoglycemia is an incompletely understood complication. Diagnosis and treatment become increasingly complex in the context of comorbid psychiatric conditions. As clinicians, it can be difficult to separate bias from objective assessment, especially when questions of somatization, illness anxiety, or factitious disorders are raised.</p> <p>Case Presentation: A 47 year old woman with history of chronic pain, depression, GERD requiring Roux-en-Y gastric bypass (RYGB) &amp; Nissen fundoplication, and post-RYGB hypoglycemia requiring stomach-remnant gastric tube placement presented with recurrent abdominal pain around her G-tube site and hypoglycemia.</p> <p>Previous workup confirmed endogenous hyperinsulinemia without insulinoma. Acarbose, a somatostatin analog, and dietary changes did not sufficiently control her hypoglycemia. She could not take diazoxide or calcium-channel blockers due to sulfa and nifedipine allergies. A trial of pasireotide seemed promising, though she had difficulty accessing this medication. Pharmacologic treatment was not only difficult in this patient, but she also received multiple surgical procedures including transoral outlet reduction and feeding tube placement in the gastric remnant. Despite these efforts, she continued to have recurrent hypoglycemic episodes.</p> <p>On admission, vitals were stable and labs remarkable for hypoglycemia. She was started on IV dextrose. Imaging confirmed correct positioning of her G-tube without signs of abscess. Shortly after admission, her G-tube mysteriously fell out. Interventional Radiology replaced the G-tube. Labeled as having “Munchausen Syndrome,” the interdisciplinary team believed she was manipulating her G-tube to obtain more procedures and surreptitiously using medications to lower her blood glucose. Her primary Medicine team planned to discharge her the following day, instructing her to resume continuous tube feeds for hypoglycemia.</p> <p>A new team assumed care on her intended day of discharge. They reviewed her hypoglycemia history and confirmed her endogenous hyperinsulinemia. With the assistance of her primary Endocrinologist, they re-started pasireotide. This allowed her to safely discharge after weaning intravenous dextrose and tube feeds. On 6 month follow up, her glycemic control was better than it had ever been.</p> <p>Conclusion: This case demonstrates the impact that cognitive and personal bias can have when caring for patients with post-RYGB hypoglycemia and</p>

	<p>comorbid psychiatric conditions. It was easy for various medical teams to dismiss significant health issues as psychogenic in nature and pass off meaningful hypoglycemia as surreptitious. Diagnostic error occurred in this case due to anchoring and confirmation cognitive biases, preventing investment in better management strategies. Without the involvement of a new team who objectively re-evaluated her condition, this patient might have continued to bear the burden of tube feeding and debilitating hypoglycemic episodes for the foreseeable future.</p>
<p><b>Danielle Hron</b></p>	<p><i>Knowledge Gap: Propylene Glycol Toxicity as a Rare Etiology of Severe Anion-Gap Metabolic Acidosis</i></p> <p>Case Presentation: A 51-year-old man with history of alcohol use, delirium tremens, and compensated alcoholic cirrhosis presented to the emergency department with vomiting and intoxication. On exam, he was hypotensive, tachycardic, and had psychomotor agitation requiring sedation which lead to intubation. Initial labs were significant for elevated blood ethanol level, significant metabolic acidosis with anion gap out of proportion to severe lactic acidosis, elevated osmolar gap, and acute non-uremic renal failure with no crystals on urinalysis. He was given empiric fomepizole in the emergency department for high suspicion of toxic alcohol ingestion. Upon admission, his wife gave collateral history that he worked in food service but had recently become unemployed, which lead to depression and increased alcohol intake. She did not personally witness him ingest anything other than ethanol. A volatile alcohol panel was sent which showed positive levels of propylene glycol and acetone the following morning.</p> <p>His propylene glycol level 12 mg/dl. Toxicity can occur in levels greater than 10mg/dl. Serum levels generally peak 1 hour following ingestion, so his initial concentration was likely much higher given the delay in testing. Much like ethanol, propylene glycol is thought to demonstrate zero order kinetics. Based on an ethanol level on presentation of 0.26g/dL, that had decreased to only 0.16g/dL at the time of the toxic alcohol panel was drawn, it can be inferred that propylene was likely also more elevated at time of presentation.</p> <p>Furthermore, the administration of fomepizole likely increased the amount of time spent with toxic serum propylene glycol levels. Unlike more common toxic alcohols, the parent alcohol of propylene glycol is the source of toxicity rather than metabolites. Propylene glycol is metabolized into l-lactate and d-lactate. Most lab assays only detect l-lactate, so his unmeasured d-lactic acidosis likely accounted for an ongoing anion gap that was out of proportion to measured l-lactic acid level.</p> <p>He met all suggested criteria for toxicity based on serum propylene glycol level, initial bicarbonate level, anion gap, elevated osmolality, and elevated osmolar gap. Additionally, he experienced multisystem organ failure is a consequence of toxicity. Due to anuric renal failure, severe acidosis, and propylene glycol toxicity, hemodialysis was indicated. His blood pressures did not tolerate intermittent dialysis and he was transitioned to continuous renal replacement therapy. He had worsening shock/sepsis like syndrome which also is associated with propylene glycol toxicity that was refractory to four pressors resulting in shock liver. His wife made the decision to transition to comfort-oriented care, and the patient expired shortly thereafter.</p>
<p><b>Ryan Huynh</b> Dr. Anya Jamrozy</p>	<p><i>Lupus Incognito</i></p>

Introduction: Systemic lupus erythematosus (SLE) can affect any organ of the body. Perhaps due to increased physician awareness and enhanced testing, the manifestation of neuropsychiatric symptoms related to the autoimmune disease appears to be increasing.

Case Description: A 39-year-old woman with history of lupus and hypertension presented with witnessed sudden abnormal limb motions with mouth-foaming and left eye vision changes and elevated blood pressures to 233/149mmhg. Other vitals were stable; physical exam was notable for swollen fingers/toes, confusion, and left-eye blurriness. CT head was unremarkable; MRI head showed subacute lacunar infarcts within the semiovale/corona-radiata bilaterally, an isolated microbleed of the central pons, and periventricular hyperintensities. Labwork demonstrated troponin 0.148, lactate 2.4, ESR 48, and thrombocytopenia. Patient was started on labetalol and nicardipine infusions and admitted to Neuro-ICU. EEG monitoring revealed two subacute seizures and evidence of diffuse slowing. Levetiracetam was initiated.

Workup for secondary hypertension was negative including non-remarkable renal-ultrasound, urine-toxicology, metanephrines, and aldosterone. Transthoracic-echocardiogram showed a tethered mitral valve with severe regurgitation, concerning for endocarditis. One of two blood cultures grew coagulase-negative-staphylococcus, but this was ultimately deemed a contaminant and follow-up cultures had no further growth. Her vision changes were monitored with serial evaluations without indications for intervention. Follow-up CT angiogram head demonstrated subtle luminal narrowing in two vessels that clinically correlated with vasculopathy. With clearing encephalopathy, patient revealed she recently discontinued all prescribed medications and started following a juice-oriented diet in a self-guided treatment plan. A lumbar puncture (LP) was offered but ultimately declined. Subsequent labs were consistent with vasculitis including positive cardiolipin IgA, ANA, beta-2-glycoprotein (IgG, IgA), Anti-ds-DNA, and low complement (c3, c4). Transesophageal echocardiogram showed vegetations on the mitral valve consistent with lupus-associated Libman-Sacks endocarditis. Hydroxychloroquine and high dose steroids were initiated for treatment of lupus cerebritis. After further clinical improvement, the patient was discharged in stable condition with hydroxychloroquine, amlodipine, chlorthalidone, lisinopril and further steroid infusions.

Discussion: Although the presentation was initially consistent with hypertensive encephalopathy, the patient's eventual CT-angiogram and recent history of noncompliance supported a clinical diagnosis of lupus cerebritis. This diagnosis was a unifying etiology for the patient's seizures, neurologic symptoms, vasculopathy/hypertensive emergency, and optic neuropathy. An LP, if she had consented for one, would have likely yielded further elevated markers/neuroproteins. Other clues included nonspecific findings that in hindsight are consistent with lupus cerebritis, including the periventricular hyperintensities on MRI and diffuse slow-wave activity on EEG.

Conclusion: SLE may present with a multitude of neuropsychiatric symptoms, which are often difficult to distinguish from symptoms of other concurrent medical problems (i.e. anchoring on hypertensive encephalopathy in this case) or functional syndromes. As there is not a singular definitive test for lupus cerebritis, this case illustrates a diagnostic workup that supports the diagnosis.

**Qiuyu Jin**

*It's Not Always the IBD - Delayed Diagnosis of Histoplasmosis in a Patient*

<p>Dr. Daniel Childs</p>	<p><i>with Crohn's Disease</i></p> <p>Introduction: Oropharyngeal lesions are commonly seen in the ambulatory primary care setting. In patients with systemic illnesses, such as those with Inflammatory Bowel Disease, it is always important to think of direct oral manifestations of their disease as well as indirect complications such as malignancy. However, given their frequent use of immunosuppressive medications, opportunistic infections must also be kept in mind. Here we present a case of histoplasmosis masquerading as Crohn's Disease progression.</p> <p>Case Presentation: A 71 year old woman with Crohn's Disease, well-controlled on adalimumab, presented to clinic with 6 months of progressive oral ulcerations. Initially, she had a single lesion on her right lower gingiva and was referred to her local dentist and periodontist. An initial biopsy showed ulceration with microbial colonies. She underwent gingival flap surgery, but did not improve. In fact, she developed worsening ulceration involving her lower gingivolabial sulcus and sublingual area, although did not develop aphthous ulcers. With concern for oral manifestations of Crohn's Disease, she was prescribed corticosteroids but experienced worsening pain, fevers, chills, and unintentional weight loss. She also developed anemia and leukopenia. Ultimately, after about six months with progressive oropharyngeal and systemic symptoms, the patient was referred to oral dermatology. An oral mucosal swab with fungal culture was performed to rule out Candidal infection, but grew <i>Histoplasma capsulatum</i> in fungal culture. Subsequent gingival biopsy showed granulomatous inflammation with budding yeast. Blood and urine histoplasma antigen were positive. Abdominal MRI showed numerous splenic nodules suggestive of infection. Infectious Diseases was consulted and the patient was diagnosed with disseminated histoplasmosis and adalimumab discontinued. The patient underwent treatment with liposomal amphotericin B and itraconazole. She had complete symptom resolution and completed 1 year of itraconazole treatment. Adalimumab continued to be held. At 1 year follow up the patient remained asymptomatic and did not require further Histoplasmosis treatment or immunosuppression for Crohn's Disease.</p> <p>Discussion: Here, we share a case from a patient who presented with painful oral ulcerations that were the primary manifestation of her disseminated histoplasmosis. This manner of presentation is at particular risk for delay in diagnosis or misdiagnosis because of the broad differential for oropharyngeal ulcerations. However, with more frequent use of tumor necrosis factor (TNF) antagonists for a variety of medical conditions, we are seeing more cases of disseminated fungal infections that can manifest in this manner. It is not routine practice for otolaryngologists, oral and maxillofacial surgeons, or dentists to perform biopsies with fungal stains and cultures, but more prompt recognition of this entity by internists in the primary care setting would certainly improve outcomes in this potentially fatal disease. Oropharyngeal and disseminated histoplasmosis should be considered in immunocompromised patients presenting with new oral ulcerations.</p>
<p><b>Jeremiah Johnson</b> Dr. Peter Lund</p>	<p><i>Feeling Like a Clot: The Consequences of Delayed Treatment and Recognition of Thrombotic Thrombocytopenic Purpura (TTP)</i></p> <p>Learning points: - Thrombotic thrombocytopenic purpura (TTP) should be considered for patients with microangiopathic hemolytic anemia (MAHA) and</p>

	<p>thrombocytopenia.</p> <ul style="list-style-type: none"> <li>- Neurologic, cardiac, gastrointestinal, and renal findings can be indications of TTP.</li> <li>- The PLASMIC score can estimate the probability of TTP and guide therapy.</li> <li>- TTP is a clinical diagnosis, treatment should not be delayed while awaiting ADAMTS13 results.</li> </ul> <p>Introduction: Thrombotic Thrombocytopenic Purpura (TTP) is a microangiopathic hemolytic anemia caused by a severe reduction in ADAMTS13, a protease responsible for cleaving von Willebrand factor (vWF). Uncleaved vWF can accumulate in the microvasculature, creating platelet rich thrombi causing multi-organ system microinfarction. Without timely recognition and intervention, TTP has a high risk of mortality.</p> <p>Case Presentation: A 56-year old woman presented with chest pain, Hgb of 9.9, platelets of 56, creatinine of 1.58, and troponin I of 1.364. She was seen the day previous with the same complaint and similar labs but EKG, CT PE, and echocardiogram were unremarkable. She left AMA prior to additional workup. On this second presentation, EKG showed ST elevations but CT angiography was normal. Platelets and Hgb continued to downtrend. A peripheral smear showed normocytic anemia with schistocytes but she again left AMA.</p> <p>One day later, she re-presented with abdominal pain, anemia, and worsening thrombocytopenia and creatinine. A bone marrow biopsy showed normal hematopoiesis. A repeat peripheral smear showed polychromasia and schistocytes. Hgb and platelets decreased to 7.9 and 21, and creatinine increased to 1.88. Concerned for TTP, hemolytic labs showed LDH of 1122, total bilirubin of 3.2, and undetectable haptoglobin. Her PLASMIC score estimated a 72% chance of severe ADAMTS13 deficiency. ADAMTS13 assay was ordered and plasma exchange and treatment with IV steroids, Rituxamab, and Caplacizumab were initiated prior to receiving results. Before plasma exchange could be started, she experienced seizure-like activity requiring intubation. MRI brain showed diffusion abnormalities within the right parietal lobe thought to represent small subacute infarctions. Hgb, platelets, and LDH improved with treatment and she was extubated after two days. ADAMTS13 returned &lt; 5%. Cardiac MRI showed segments of myocardial edema likely due to microangiopathic thrombosis. She displayed no residual neurologic deficits at discharge and continued pheresis as an outpatient.</p> <p>Discussion: MAHA and thrombocytopenia should always raise clinical suspicion for TTP. Neurologic, cardiac, gastrointestinal, and renal abnormalities can be indications for TTP but are non-specific. The PLASMIC score, using platelet count, evidence of hemolysis, presence of cancer, history of organ/stem cell transplant, MCV, INR, and creatinine can be used to risk stratify individuals into low, intermediate, or low risk groups for TTP. It is a useful adjunct to clinical decision making as TTP is primarily a clinical diagnosis and initiation of treatment should not be delayed while waiting for ADAMTS13 results.</p>
<p><b>Sywia Kaeding</b></p>	<p><i>Irreversible Brain Injury from Hyperammonemia following Roux-en-Y Gastric Bypass</i></p> <p>Introduction: Roux-en-Y gastric bypass can be associated with severe, fatal hyperammonemia. This complication is relatively rare and occurs at variable intervals following the procedure, presenting even in the absence of</p>

	<p>underlying cirrhosis.</p> <p>Case Presentation: A 58 year old female, who had prior history of alcohol and opioid use and who had undergone roux-en-y gastric bypass sixteen years prior, presented from a rehabilitation after she was noted to be more somnolent than normal after taking her morning medications. At baseline, she was alert and oriented, and this decline in mental status was abrupt. On evaluation, she displayed myoclonic movements in her upper extremities. Whereas her ammonia levels on prior presentations had been normal (including an ammonia level measured under two months ago), the ammonia level on admission was over 200. Due to her severely depressed mental status and inability to protect her airway, patient was transferred to the intensive care unit. She underwent hemodialysis for elevated ammonia levels. Despite the correction of ammonia levels, patient remained encephalopathic. She was also started on rifaximin and lactulose. Hospitalization course also notable for convulsive status epilepticus and resultant respiratory arrest. As part of her work-up for encephalopathy, patient underwent liver biopsy and evaluation for urea cycle disorders. There was no cirrhosis on liver biopsy, and ornithine transcarbamylase deficiency was ruled out. She also underwent numerous EEG and MRI brain studies. Her neurological exam at roughly six weeks from her elevated ammonia showed her to be in unresponsive wakefulness syndrome. She had evidence of central pontine osmotic demyelination, likely from her elevated ammonia. Her likely chance of any further improvement was felt to be very low, as she demonstrated minimal improvement. Examination remained very poor; patient remained nonverbal and was unable to follow commands. Following a care conference, patient was transitioned to comfort cares. After a hospital stay of 59-day duration, she was discharged to a hospice facility.</p> <p>Conclusion: This case of irreversible brain injury from hyperammonemia, occurring several years after Roux-en-Y gastric bypass, demonstrates the rapid time course in which encephalopathy can develop. Given the poor prognosis of this complication, conversations regarding detection and management are worthwhile.</p>
<p><b>Brittany Kimball</b> Dr. Ryan Kelly</p>	<p><i>You, Me, and Hep C: An Internist's Role in the Silent Epidemic</i></p> <p>Case Presentation: A 33-year-old man with a history of opioid use disorder (on Suboxone) and chronic hepatitis C virus (HCV) presented to a primary care clinic. He had contracted HCV via IV drug use. His HCV was first discovered in 2017 via screening based on risk factors. Since then, he'd enrolled in the clinic's Suboxone program and had been abstinent from injection drug use for &gt;6 months. At the time of his visit, he had a viral load by PCR of 3,767,337 IU/L and was found to have genotype 1A. He was HIV negative, and had appropriate antibodies to hepatitis A and B. His fibrosis-4 score was 0.61. Based on this information, an 8 week course of glecaprevir/pibrentasvir (Mavyret) was prescribed. 12 weeks after completing treatment, an HCV viral load showed no detectable HCV, confirming cure.</p> <p>Discussion: This case is a simple but elegant example of how HCV can—and should—be successfully detected and treated in the primary care setting. Hepatitis C induced cirrhosis is the leading indication for liver transplantation in the US adult population and accounts for one third of patients who develop hepatocellular carcinoma. 2.7 million Americans are estimated to be living with hepatitis C virus (HCV), and despite the emergence of highly effective direct acting antiviral agents against the disease, only 432,000 people have</p>



	<p>begun treatment for chronic HCV. What primary care providers may not know is that combatting this epidemic is in our hands. Thanks to legislative changes in the state of Minnesota that went into effect January 1, 2020, this disease, once relegated to the realm of specialists, can now be treated successfully in the primary care setting under many circumstances. Through this case, we will discuss the public health implications of this policy change for Minnesota primary care providers, as well as the steps needed to select appropriate patients to treat for chronic HCV in primary care.</p>
<p><b>Thomas Klink</b> Dr. Talitha Wilson Dr. Michael Aylward</p>	<p><i>Under the Guise of Edema: An unusual presentation of statin-associated necrotizing autoimmune myopathy</i></p> <p>Introduction: Statin-associated necrotizing autoimmune myopathy is a rare side effect of statins that can occur after months to years of use. It is typically characterized by progressive proximal muscle weakness, elevated creatine kinase, and presence of anti-hydroxy-3-methylglutaryl-CoA reductase autoantibodies. Extra-muscular symptoms are seldom reported.</p> <p>Case Presentation: A 64 year old female with a seven year history of left lower extremity lymphedema presented as a new patient to a primary care clinic for evaluation of worsening left leg swelling, night sweats, chills, and 15 kg weight loss over the prior six months. On exam she had non-pitting edema of her left lower extremity to her mid back, with her left leg being approximately twice the size of her right leg. She was uninsured so a stepwise workup was initiated to reduce cost. Initial labs demonstrated normocytic anemia and mildly elevated transaminases. A CT abdomen and pelvis showed no evidence of malignancy, only subcutaneous fat stranding of lower extremities, left worse than right.</p> <p>One month later she was no longer able to walk secondary to pain and swelling of her left leg. She also reported new early satiety and epigastric pain. Additional workup included left lower extremity doppler, chest x ray, right upper quadrant ultrasound, thyroid studies, inflammatory markers, and infectious studies; this was revealing only for an erythrocyte sedimentation rate of 58 (normal 0-30). She was started on furosemide and dexlansoprazole for symptomatic relief.</p> <p>By her next visit, her left leg swelling and epigastric had significantly improved, although she was still unable to walk. On her 5th clinic visit, she had notable proximal muscle weakness in upper and lower extremities with intact distal muscle strength. Creatine kinase level was 12,930. An urgent rheumatology referral was placed for presumed polymyositis and her atorvastatin and furosemide were stopped.</p> <p>She was seen in rheumatology clinic one week later and was directly admitted to the county hospital for pulse steroids, hydration, and muscle biopsy. Her hospital course was complicated by new onset atrial fibrillation, but ultimately she discharged home and muscle biopsy showed anti-hydroxy-3-methylglutaryl-CoA reductase positive necrotizing myositis. Rheumatology continued a treatment course of steroids and later started rituximab infusions. Approximately five months after admission, she is now able to complete all activities of daily living on her own, although requires some assistance with walking.</p> <p>Discussion: This case highlights the challenge of diagnosing a rare disease based on an unusual presentation in a busy primary care setting. Her workup</p>

	<p>was likely influenced by anchoring bias in the face of alarming peripheral edema and constitutional symptoms. On a broader scale, lack of insurance and a fragmented medical system undoubtedly affected her care.</p>
<p><b>Jonathan Knott</b> Dr. Alexander Theofiles Dr. Reema Tawfiq</p>	<p><i>The Pan-Positive Presentation: A Case of Granulomatosis with Polyangiitis</i></p> <p>Introduction: Granulomatosis with polyangiitis (GPA) is a systemic necrotizing vasculitis, affecting the small and medium-sized vessels and is often associated with cytoplasmic ANCA (c-ANCA). Clinical manifestations of GPA are broad which leads to difficulty in early diagnosis. As a result, several sets of diagnostic criteria have been developed. Early diagnosis is important to adequately manage the disease and avoid potentially lethal complications.</p> <p>Case Presentation: A 58-year-old female with a history of psoriasis vulgaris and prior tobacco use presented with neck pain that radiated to her shoulders and bilateral upper extremities, unintentional weight loss, intermittent fevers, right-sided wrist pain, hand swelling, and left-sided upper chest pain. Further history revealed a predilection for nose bleeds, occasional sinus congestion, morning joint stiffness and swelling, predominantly in her hands. She also reported red, irritated eyes with intermittent photosensitivity.</p> <p>Physical exam revealed dried blood in her nasal turbinates bilaterally, bilateral conjunctival injection and a 1 cm aphthous ulcer under her tongue. The right hand was remarkable for erythema and edema of the 3rd MCP joint and painful passive range of motion of the right wrist. Arm abduction was limited by pain past 45°. During the hospitalization, she was intermittently febrile.</p> <p>The patient was recently evaluated for chest pain at a different institution and underwent a CT chest which showed a 2.1 x 1.5 cm soft tissue density at the apex of the left lung with adjacent pleural thickening. We pursued a biopsy of the lung mass and pathology revealed necrotizing granulomatous inflammation. Laboratory workup revealed CRP 235.5, c-ANCA of 1:512, and anti-PR3 antibodies &gt;8 (positive). Anti-MPO, p-ANCA, and anti-GBM antibodies were negative. Urinalysis revealed microscopic hematuria without proteinuria or RBC casts. Creatinine was within normal limits. CT urogram showed no evidence of structural causes of hematuria. A diagnosis of GPA was supported by 14 points on the ACR/EULAR 2017 criteria with ≥5 points needed for diagnosis. She was started on induction therapy with IV methylprednisolone and transitioned to oral prednisone which resulted in a significant improvement in her symptoms. Due to her evidence of renal involvement, she was later started on rituximab as an outpatient.</p> <p>Conclusion: This patient’s presentation is an excellent example of the broad clinical manifestations of GPA. Patients typically present with nonspecific symptoms such as fever, malaise, weight loss, arthralgia and myalgia. There are various classification algorithms that can be used to assist with the diagnosis of GPA. One limitation is that older algorithms lack the ability to distinguish between GPA and microscopic polyangiitis. We employed the use of the ACR/EULAR 2017 provisional classification criteria for GPA. Treatment of GPA with organ involvement includes induction therapy with glucocorticoids in addition to rituximab or cyclophosphamide.</p>
<p><b>Bryan Koenig</b> Dr. Amy Holbrook</p>	<p><i>When Vitamin Replacement isn’t all “Hocus Pocus”</i></p> <p>Introduction: Anemia is one of the most commonly encountered lab</p>

	<p>abnormalities, and can be associated with significant morbidity and mortality. Cobalamin (Vitamin B12) deficiency can lead to red blood cell destruction through a well-documented process of progressive defective DNA repair, cytoplasmic and nuclear desynchrony, and cell maturation arrest. This process leads to intramedullary hemolysis, a condition characterized by destruction of red blood cells via apoptosis within the bone marrow. As an easily reversible cause of anemia, proper recognition, identification, and differentiation from other etiologies of bone marrow failure is imperative.</p> <p>Case Presentation: A 77 year old woman with a history of Roux-en-Y gastric bypass in 2001 presented with 3 days of chest pain and dyspnea. She denied any signs or symptoms of obvious blood loss. Her vitals and exam were notable only for pale skin and conjunctiva. Her CBC showed pancytopenia with macrocytosis: Hgb 5.9, MCV 125, WBC 1.8, Plt 113. Her hemolysis labs were positive, with an undetectable haptoglobin, LDH of 4883, and uric acid of 11.2. Her reticulocyte count was elevated to 3%, but in the setting of her significant anemia her reticulocyte production index of 0.52 represented overall bone marrow hypoproduction. Initial concern was for a hematologic malignancy, and a bone marrow biopsy was arranged for the following hospital day.</p> <p>Her vitamin B12 returned the next day and was undetectable. Peripheral smear noted marked macrocytic anemia with associated moderate absolute neutropenia with hypersegmented neutrophils and mild left shift, findings consistent with vitamin B12 deficiency. Bone marrow biopsy noted hypercellular bone marrow for age with 95% cellularity (expected ~ 30%) with trilineage hematopoiesis. There was significant dysmorphic change in many erythroid precursors and rare megakaryocytes, and 3% blasts. Cytogenetic evaluation showed a normal karyotype. There was no evidence of iron deficiency. The complete findings were most consistent with severe vitamin B12 deficiency.</p> <p>On further questioning, patient had noted not taking her recommended vitamin supplements as she was told, “vitamins and minerals are just hocus pocus.” She received 2 units pRBC with near complete resolution of her symptoms. She was treated with subcutaneous vitamin B12 replacement therapy. Her pancytopenia resolved with a hemoglobin of 12.4 within one month following hospital discharge.</p> <p>Conclusion: This case exemplifies the potential for a significant vitamin B12 deficiency to give rise to a life-threatening anemia. Roux-en-Y gastric bypass leads to decreased vitamin B12 absorption, and without supplementation can cause severe vitamin B12 deficiency. This in turn can cause pancytopenia, and in extreme deficiency, can lead to intramedullary hemolysis from apoptosis of unstable red cells that are unable to traverse into the peripheral blood.</p>
<p><b>Robert Kraft</b> Dr. Floranne Ernste</p>	<p><i>Chronic Abdominal Pain: “Sjö” Me the Diagnosis</i></p> <p>Introduction: Sclerosing mesenteritis is a rare, inflammatory, fibrotic disease affecting the small bowel mesentery. It is most commonly found in patients with prior abdominal surgeries or malignancies, and, in rare occasions, a manifestation of autoimmune diseases. This case illustrates an unusual presentation of undiagnosed Sjögren Syndrome presenting as sclerosing mesenteritis.</p>

	<p>Case Presentation: A 60-year-old female, non-smoker, with medical comorbidities including chronic dysphagia, hypertension, and obesity, presented with a chief complaint of dull, constant, left upper quadrant abdominal pain for the past eight months. Associated symptoms included bloating and constipation. Relevant surgical history included laparoscopic cholecystectomy and total hysterectomy. Review of systems was positive for fatigue, dry eyes, dry mouth requiring consumption of “tons of water” daily, and left-sided facial swelling; negative for night sweats, fevers, or unintentional weight loss. Physical examination revealed vital signs within normal limits, enlarged and non-tender parotid glands bilaterally, dry oral mucous membranes, and soft abdomen with a palpable, firm, irregular mass in the left upper quadrant. There was no hepatosplenomegaly. A CT abdomen and pelvis with IV contrast revealed mesenteric lymphadenopathy and mesenteric fibrosis consistent with a diagnosis of sclerosing mesenteritis. An excisional lymph node biopsy was performed and showed a reactive lymph node without findings of malignancy. Relevant laboratory data that returned negative or normal included monoclonal gammopathy screen, kappa/lambda free light chains, leukemia/lymphoma immunophenotyping by peripheral flow cytometry, ds-DNA antibodies, complement levels, IgG4 level, and CA-125. Further laboratory workup revealed strongly positive antinuclear antibody titer, along with positive anti-Ro and anti-La antibodies. Because of the clinical history and diagnostic findings, a bilateral parotid gland ultrasound and minor salivary gland biopsy were performed. Ultrasound revealed hypoechoic lesions throughout the parotid glands suggestive of Sjögren Syndrome. Minor salivary gland biopsy was positive for severe lymphocytic sialadenitis (focus score &gt;3 per 4 mm<sup>2</sup>) confirming the diagnosis of Sjögren Syndrome. She was started on prednisone and began to show improvement in her symptoms.</p> <p>Discussion: Sclerosing mesenteritis is a rare, inflammatory, fibrotic disease affecting the small bowel mesentery. The pathogenesis of the disease is unclear, but it is most commonly seen in patients with prior abdominal surgeries, malignancies, and autoimmune diseases. The majority of patients present with abdominal pain with nearly half having a palpable abdominal mass. CT imaging can show several different patterns including soft tissue mass lesions, fat ring sign, tumor pseudocapsule, calcifications, and hazy appearance of the mesentery. Typical therapies used for treatment include corticosteroids, azathioprine, and tamoxifen. A high index of suspicion for malignancy should be considered if sclerosing mesenteritis is seen on imaging; however, if malignancy workup returns negative, autoimmune diseases such as Sjögren Syndrome as a potential etiology should be investigated.</p>
<p><b>Brooke McDonald</b> Dr. Matthew Prekker</p>	<p><i>Pyelonephritis-Induced Septic Thrombophlebitis Leading to Cardiopulmonary Complications in a 46-Year-Old Diabetic</i></p> <p>Introduction: Septic thrombophlebitis can occur in any vein as a complication of invasive medical procedures or by hematogenous spread from nearby non-vascular structures. Typically bacteremia is caused by <i>Fusobacterium necrophorum</i> or <i>Staphylococcus aureus</i>. Common locations include the internal jugular vein as seen in Lemierre’s disease, but have also been described in pelvic, portal, and sinus veins.</p> <p>Case Presentation: A 46-year old male with past medical history significant for T2DM, HTN, and prior CVA presents to outside hospital for weakness, nausea, and elevated blood glucose for the past three weeks. He reports</p>

	<p>limited access to insulin for the past eight months. He is a mechanic and recently suffered blunt torso trauma involving a car falling onto his right flank at work but he did not seek medical care at that time. At presentation he was normotensive, afebrile and tachycardic with unremarkable physical exam.</p> <p>Initial labs were significant for leukocyte count 38 cells/mm<sup>2</sup>, hemoglobin of 9.6 g/dL, potassium of 6.5 mEq/L, bicarbonate of 13 mEq/L, beta-hydroxybutyrate of 5.4 mmol/L, glucose of 721 mg/dL, and troponin of 0.477ng/L. Initial imaging obtained for trauma workup included CT chest/abdomen/pelvis with peripheral cavitory pulmonary nodules with small bilateral effusions, bilateral pyelonephritis with intraparenchymal abscess in left kidney, nonocclusive thrombus in left renal vein, and moderate pericardial effusion. Diabetic ketoacidosis protocol and broad-spectrum antibiotics were initiated and he was transferred to our hospital's intensive care unit for concern of developing cardiac tamponade physiology. Infectious disease and cardiology were consulted. Blood cultures, urine cultures and pericardiocentesis fluid were notable for growth of methicillin sensitive staphylococcus aureus. TEE was without evidence of valvular vegetations or endocarditis. Hemoglobin A1c was &gt;15%. HIV, RPR, hepatitis panel, fungal serologies were unremarkable. Quantiferon-TB gold was indeterminate.</p> <p>Discussion: In this case, uncontrolled type 2 diabetes led to diabetic ketoacidosis and likely some degree of immunosuppression, leading to MSSA bacteremia and ascending urinary tract infection, renal abscess, and eventually renal vein thrombosis. The septic renal vein thrombophlebitis embolized to the lungs and also caused the rare complication of purulent pericarditis (his pericardial fluid gram stain and culture revealed MSSA as well). He was transitioned to cefazolin for six weeks of treatment and therapeutically anticoagulated with unfractionated heparin. Patient has been improving clinically and remains hospitalized as he completes 6-week course of antibiotics and is transitioned to 3-month course of rivaroxaban for anticoagulation.</p> <p>Conclusion: When originally approaching a patient with multiple pulmonary nodules and renal mass, it is critical to maintain a broad diagnosis. Acute pyelonephritis leading to septic thrombophlebitis can present with several complications including pulmonary septic emboli, pericardial effusion, hypoxia, sepsis, or death. Treatment includes intravenous antibiotics, anticoagulation, and possible surgical intervention to control the source of the infection. Rapid diagnosis is necessary to prevent high mortality.</p>
<p><b>Nicole McLaughlin</b> Dr. Emily Leasure</p>	<p><i>A Case for a Patient Exam</i></p> <p>Introduction: Dizziness is a common presentation in primary care and is described by our patients in multiple ways from vision changes to abnormal sensations of the body to a sense of spinning. Nystagmus, or regular rhythmic oscillation of the eyes, may be present on exam when evaluating a patient with dizziness. When nystagmus is present, exam findings can suggest either a central or peripheral process causing the disturbance, which guide the differential diagnosis and next steps in evaluation.</p> <p>We present a case of a 33-year-old male who presented to his primary care clinic with vision changes for the past 6 weeks associated with a sense of dizziness. He described a sensation of his “eyes moving back and forth.” Exam revealed nystagmus in eccentric gaze that would eventually slow down and stop. About 2 minutes later, the nystagmus would recur but in the</p>

	<p>opposite direction. Additional exam findings included nystagmus with straight ahead gaze and bilateral vertical skew deviation, causing concern for a central etiology of his vertigo. No other deficits or abnormalities were noted on the patient’s neurologic examination. He underwent further evaluation with an MRI of the head, MRA of the neck and follow up with outpatient neurology. Imaging did not reveal an etiology including a clear demyelinating pattern, mass, or infarct. He was also evaluated by a specialist in Vestibular and Eye Movement disorders where he was diagnosed with acquired periodic alternating nystagmus (PAN). Further work up for an underlying etiology is still underway, however lumbar puncture with CSF analysis, testicular ultrasound, serum paraneoplastic panel, autoimmune panel, underlying infectious markers, and oligoclonal bands have been unremarkable.</p> <p>Conclusion: PAN is a rare type of horizontal nystagmus that reverses its direction at variable intervals of seconds to minutes. It can be congenital or acquired, and in the acquired adult form, it has a characteristic oscillation cycle of 2 to 4 minutes. Due to the changing nature of the direction of nystagmus, the key to diagnosis requires the clinician observe the eye movements for several minutes or the change in direction will be missed. As in our patient’s case, once PAN is diagnosed, evaluation for possible causes of PAN should explore autoimmune, paraneoplastic, inflammatory, demyelinating, metabolic and infectious conditions. Once found, treatment of the underlying condition is a primary goal. Baclofen can be effective in managing symptoms of idiopathic PAN. Additional treatment options include retrobulbar botulinum toxin injections and amantadine therapy.</p>
<p><b>Morgan Mensink</b></p>	<p><i>Abdominal Pain in the Early Bird</i></p> <p>Introduction: Ascariasis is one of the most common parasitic infections in humans worldwide and caused by the <i>Ascaris lumbricoides</i> roundworm. While majority of infections are found in developing countries, with increasing immigration and worldwide travel we are likely to see more cases in non-endemic areas such as the United States. The roundworm typically resides in the small intestines however complications such as acute cholecystitis, cholangitis, pancreatitis, and liver abscesses can occur when the adult worm migrates into the biliary system.</p> <p>Case Presentation: We report a case of a 50-year-old woman presenting with progressive nausea, vomiting, and epigastric pain with initial laboratory and imaging concerning for gallstone pancreatitis. Further investigation with EUS revealed a roundworm in the gastric antrum, identified as <i>Ascaris lumbricoides</i>. Patient was treated with albendazole. The patient had subsequent admission for cholangitis related to distal biliary stricture secondary to initial helminth infection.</p> <p>Conclusion: This case describes an uncommon etiology of acute interstitial pancreatitis and complications of <i>Ascaris</i> infection.</p>
<p><b>William Minter</b> Dr. Frank Brozovich</p>	<p><i>Don’t Trust the Troponin?</i></p> <p>Case Presentation: A 55 year old Ethiopian male presented in December with recent 20lb unintentional weight loss and 3 days of fevers, dyspnea, and cough. He was febrile and hypoxic, requiring 4L O2 by nasal cannula. His history is notable for steroid-responsive interstitial lung disease with polyclonal hypergammaglobulinemia of unclear etiology. His disease was previously controlled with prednisone but he had not taken prednisone for</p>

	<p>over 6 months. Initial testing revealed hyperproteinemia with elevated protein gap (7.9 g/dL), elevated AST (66 U/L), significant troponinemia (troponin T (TnT) 1679 ng/L that up-trended to 1871 ng/L at 2 hours), and positive influenza A PCR. CT imaging was notable for progression of diffuse bilateral ground-glass opacities. One year prior while on high dose prednisone, the patient had been hospitalized for syncope and was found to have elevated TnT (173 ng/L), and underwent diagnostic angiography which was negative for obstructive coronary artery disease. Severe influenza A myocarditis was suspected, but cardiac MRI showed only mild pericarditis. Although serology for HIV, HCV and HBV was positive, testing by PCR was negative. Due to the second episode of discordance between TnT and other clinical data, as well as discordant viral testing, troponin assay interference by a heterophile antibody was suspected. Troponin I (TnI) was obtained and was elevated, but to a lesser degree. Serial dilution of both TnT and TnI samples was non-linear. Commercially available heterophile antibody blocker reagent significantly reduced TnI with a smaller effect on TnT, and completely corrected viral immunoassays, confirming the presence of a heterophile antibody. In this case, the presence of a heterophile antibody resulted in extensive, unnecessary serum testing, coronary angiography, and significant emotional burden to the patient due to prolonged hospitalization and concern about contracting multiple infectious diseases. Heterophile antibodies interfere with immunoassays by binding the capture and tracer antibodies, typically of mouse origin, producing a false positive signal. Patients suspected to be at risk for this phenomenon are those treated with biologicals with chimeric or humanized Fc-regions (ximab/zumab), seropositive rheumatic disease, and those with exposure to animal antigens.</p> <p>Conclusion: In this case, the patient developed interstitial lung disease after coming to America and working at a meat processing plant. The degree of his troponin elevation this admission was thought secondary to higher titers of heterophile antibody from uncontrolled auto inflammatory disease with a contribution from influenza A pericarditis.</p>
<p><b>Leah Mische</b> Dr. Hannah Nordhues</p>	<p><i>Diarrhea followed by Back Pain: Atypical Presentation of an Aortic Mycotic Aneurysm</i></p> <p>Introduction: Abdominal aortic aneurysms are most frequently attributed to atherosclerosis. Rarely, these aneurysms can be secondary to infections, most commonly due to endocarditis embolic vegetations or hematogenous spread in the setting of a diseased aorta.</p> <p>Case Description: A 66-year-old woman with rheumatoid arthritis (on Methotrexate) and dyslipidemia presented to an outside hospital with two days of low back pain and 19 days of non-bloody diarrhea in the setting of recent travel to Mexico. Initial evaluation of symptoms was delayed due to COVID-19.</p> <p>Her vitals were notable for mild hypothermia (35.9°C) and subsequent fever (38.3 °C). Labs were notable for WBC: 8.4, lactate: 0.5, ESR: 40 and CRP: 130.2. CT Angiogram demonstrated a 5mm luminal ulceration of the upper abdominal aorta with extensive surrounding inflammation, concerning for a mycotic aneurysm and mild thickening of the aortic valve.</p> <p>Exam was notable for left lower quadrant pain to palpation and a I/VI systolic murmur heard diffusely. She denied ever having used methamphetamines or cocaine and had a 0.5 pack year smoking history. The differential diagnosis</p>

	<p>included an inflammatory arteritis versus a mycotic aneurysm. Autoimmune serologies were all negative and Rheumatology indicated low suspicion for an active autoimmune process given the absence of recent RA flare and atypical imaging findings.</p> <p>Infectious Disease raised concern for a Salmonella mycotic aneurysm, and subsequently stool cultures and blood cultures identified Salmonella enterica. She was started on Ceftriaxone when susceptibilities demonstrated pan-sensitivity. Transesophageal echocardiogram to rule out endocarditis demonstrated a sclerotic aortic valve without vegetations.</p> <p>The aneurysm continued to grow despite appropriate medical therapy and Vascular Surgery performed an extremely high-risk surgical resection including replacement of the affected aorta and reconstruction of the SMA, renal arteries and celiac artery. Other than a transient AKI, she recovered well from surgery.</p> <p>Cultures of the mycotic aneurysm grew Salmonella enterica. She completed a six-week course of IV Ceftriaxone followed by 4 months of amoxicillin-clavulanate. Post-operative surveillance imaging showed widely patent vessels without evidence of persistent infection.</p> <p>Discussion: We describe a case of an immunocompromised woman presenting with a mycotic aneurysm secondary to Salmonella enterica. Bacterial aortic mycotic aneurysms are most common in patients with atherosclerotic plaque and hematogenous or embolic spread of Staphylococcus aureus or Salmonella enterica. Our patient likely contracted Salmonella during her trip to Mexico and developed bacteremia in the setting of an immunocompromised state and delayed treatment due to COVID-19 with subsequent inoculation of a diseased aorta. The treatment of aortic mycotic aneurysms almost always includes surgical management as the risk of death exceeds 80% in those managed medically alone. An aortic mycotic aneurysm can present as diarrhea followed by back pain and early identification is key.</p>
<p><b>Sneha Mohan</b> Dr. John Matulis III</p>	<p><i>When HbA1c Doesn't Tell the Whole Story: Large Vessel Vasculitis Manifesting with Elevated Glycosylated Hemoglobin Values</i></p> <p>Case Report: A 65 year old female with long-standing Insulin dependent Diabetes presented for routine follow-up, reporting slight fatigue attributed to poor sleep and a busy work schedule. Physical exam was unremarkable. Initial laboratory results revealed elevated Hemoglobin A1c (HbA1c) of 8.9%, discordant from measured self-monitored glucose values ranging from 100-150 mg/dl (correlating with HbA1c of 6-7%). Fructosamine level of 304 mcmol/L (correlates to HbA1c of approximately 7.5%) confirmed this discordance. An etiology for altered red blood cell turnover was sought. Additional tests showed normocytic anemia with Hemoglobin of 10.9 g/dl, elevated inflammatory markers suggestive of inflammatory anemia with Erythrocyte Sedimentation Rate (ESR) of 114 mm/h (ref: 2-22 mm/h) and C-Reactive Protein (CRP) of 75.2 mg/L (ref: &lt;=8.0 mg/L); and mild iron deficiency evidenced by Ferritin of 176 mcg/L (ref: 11-307 mcg/L), Transferrin saturation of 14% (ref: 14-50%) and TIBC of 182 mcg/dl (ref: 250-400 mcg/dl). Rheumatology recommended CT Angiography (CTA) of thoracic aorta, and autoimmune panel. Autoimmune panel, which included mildly elevated Rheumatoid factor of 16 IU/ml (ref &lt;15 IU/ml), Anti-nuclear Antibody of 0.5 U (ref &lt;1 U), cyclic citrullinated peptide at &lt; 15.6 U (ref &lt;20</p>



	<p>U) and negative ANCA, was unrevealing. CTA of the thoracic aorta revealed extensive mural thickening of the thoracic and abdominal aorta including the proximal great branches, suggestive of Giant Cell Arteritis. Prednisone was initiated at 40 mg, along with oral iron. On follow up at 1 month, she stated improvement in fatigue with labs revealing reductions in ESR to 14 and CRP to 16.2. HbA1c and fasting blood glucose demonstrated concordance with correction of her inflammation.</p> <p>Discussion: Interpretation of HbA1c values should consider other physiologic factors which may alter results. Fructosamine levels can help to confirm discordance between serum blood glucose and HbA1c. Falsely elevated HbA1c from decreased red cell turnover may arise from iron deficiency or reduced availability of functional iron as seen in inflammatory anemia. In these circumstances, thorough evaluation is warranted to identify the underlying inflammatory condition.</p>
<p><b>Jwan Naser</b>  Dr. Sorin Pislaru  Dr. Andy Boggust  Dr. Mayra Guerrero</p>	<p><i>Came with TIA, diagnosed with STEMI</i></p> <p>Introduction: Elderly patients, women, and diabetics tend to have atypical presentation of acute coronary syndrome (ACS) that can manifest as epigastric pain, nausea, vomiting, dyspnea, confusion, syncope or other symptoms.</p> <p>Case presentation: An 87-year-old woman with history of hypertension, hyperlipidemia, dementia, and chronic kidney disease presented to the emergency department (ED) with syncope. While the patient does not remember details, per report, she had transient drooping of one side of her face as well as unilateral weakness with uncertain laterality. In the ED, she had no focal neurological signs or symptoms. The patient was borderline hypotensive with blood pressure of 92/57. The patient described a vomiting episode the day before. She denied nausea, chest pain, shortness of breath, or palpitations in the ED. Computed tomography of the head showed no hemorrhage. Electrocardiogram (ECG) revealed ST segment elevation in the anterolateral leads. Troponins were markedly elevated. On further questioning about cardiac symptoms, the patient endorsed having had diffuse mild chest “soreness” at the time of vomiting that then localized to the right side, and “did not last long”. ST elevation myocardial infarction (STEMI) of late presentation was diagnosed and the patient received loading dose of Aspirin and Clopidogrel, intravenous heparin, and underwent emergency cardiac catheterization procedure.</p> <p>Coronary angiography revealed severe single vessel coronary artery disease involving the left anterior descending artery (LAD) with 50% proximal stenosis and 100% mid LAD occlusion with ruptured atherosclerotic plaque and thrombus as well as 99% diffuse stenosis in the distal LAD. There were no concerns for intracoronary embolism. Percutaneous coronary intervention (PCI) with balloon angioplasty of mid LAD was performed successfully but no stent was placed due to small caliber vessel and diffuse disease not suitable for stenting. Despite revascularization with angioplasty of culprit lesion, flow into distal LAD was not restored due to very severe diffuse disease. Transthoracic echocardiography revealed a new decrease of ejection fraction to 38% as well as new regional wall motion abnormalities but no intracardiac thrombi. Telemetry throughout the hospitalization showed sinus rhythm. The patient was discharged on Aspirin, high dose Atorvastatin, Lisinopril and Clopidogrel and arranged to follow up with plans of initiating Metoprolol given low blood pressure during her hospital stay.</p>

	<p>Discussion: A high index of suspicion for ACS is required in elderly patients who present to the ED with acute symptoms that may include confusion, syncope, focal neurological symptoms, or nausea and vomiting, and ECG should be mandatory even in the absence of chest pain, palpitations, or dyspnea. In patients with STEMI and focal neurological symptoms, in addition to diffuse atherosclerosis in the coronary and carotid arteries predisposing to both and the need to investigate for underlying atrial fibrillation, other important considerations in the differential diagnosis include intracardiac thrombi embolising to both arterial systems as well as massive STEMI resulting in ventricular hypokinesia and thrombus formation with subsequent embolization.</p>
<p><b>Nathan Nesbitt</b> Elizabeth Kelsey Dr. Diego Suarez</p>	<p><i>An Unusual Case of Prostatitis</i></p> <p>Introduction: Prostatitis is the most common urological diagnosis in men under age fifty. Approximately 1-10% of cases are due to acute bacterial prostatitis, with most others consisting of chronic inflammatory/non-inflammatory prostatitis. Typical presentation of acute bacterial prostatitis follows a urinary tract infection due to a urinary pathogen or urethritis due to a sexually transmitted pathogen (&gt;97% of cases together). Here, we present a case of acute bacterial prostatitis secondary to Salmonella enterica, an exceedingly rare cause of this disorder, precipitated by acute infectious enteritis.</p> <p>Case Presentation: A 38 year old gentleman was initially evaluated by video visit for five days of acute diarrhea. The patient initially developed body aches and chills, followed by the onset of diarrhea at a rate of 2-3 stool episodes per hour with associated lower abdominal cramping and bloating and tenesmus, small amounts of hematochezia, as well as low grade fevers. The patient's children had similar symptoms which had resolved. SARS Coronavirus-2 nasopharynx testing was unremarkable. Infectious enteritis was suspected, and enteric pathogen panel revealed Salmonella enterica. Antibiotics were deferred.</p> <p>One week later, he presented in person to primary care clinic for dysuria and hematuria that had onset 24 hours prior. He denied any concern for sexually transmitted infections, and his abdominal bloating had improved. No one at home was experiencing similar symptoms. Urinalysis and urine cultures were obtained to evaluate potential causes of urinary tract infection (UTI). He was initiated on trimethoprim-sulfamethoxazole to cover both salmonella and the common causes of UTI. A urine culture returned two days later revealing Salmonella (non-Typhi), and blood cultures were negative.</p> <p>The patient called in with worsening symptoms and infectious disease was consulted. The decision was made to investigate further with a CT pelvis with IV contrast, and switch from trimethoprim-sulfamethoxazole to levofloxacin daily due to suspicion of prostatitis. Imaging findings revealed asymmetric heterogeneous hyper enhancement in the left prostate without discrete fluid collection indicative of infectious prostatitis. The infectious disease consultant recommended continued levofloxacin therapy for six weeks for further management.</p> <p>Discussion: While acute bacterial prostatitis is frequently caused by urinary tract pathogens, a case due to acute enteritis is extremely rare. Potential pathophysiological explanations include lymphatic travel from the rectum or hematological transport in the setting of systemic infection. The key point in</p>

	<p>this case is recognizing that the urinary symptoms subsequent to enteritis may be prostatic in origin, because antibiotic selection for prostatitis is important due to the poor penetration of most antibiotics into the prostate and because a lengthy antibiotic course is required to adequately treat bacterial prostatitis. Thus, this case is illustrative of the value of clinical judgement in avoiding premature closure on a condition that masqueraded as a UTI.</p>
<p><b>Daniel Pfeifle</b> Dr. Anna Subramaniam</p>	<p><i>Acute-Li or Chronical-Li toxic? Why not both?</i></p> <p>Background: Lithium remains the mainstay treatment for patients with bipolar mood disorders, both for acute manic episodes and preventing relapses of manic attacks. The mechanism of action for lithium in treatment of mood disorders is not entirely known. Lithium toxicity can be due to either excessive intake or due to impaired excretion. In this case, we present a patient who presented with acute lithium toxicity due to sequela from chronic lithium toxicity.</p> <p>Case Presentation: A 21 year old woman presented with excessive thirst and urination concurrent with 6 months of worsening nausea, vomiting, and fatigue. Her past medical history was notable for autism spectrum disorder and bipolar disorder, for which she had been managed with lithium. The lithium dose had been recently decreased from her longstanding stable regimen to avoid toxicity in the setting of worsening nausea and vomiting. The patient was found to have a supratherapeutic lithium level (1.8 mmol/L) despite the decreased dose. She was referred to the ED, where she was tachycardic, tachypneic, and had myoclonic jerks. Labs were remarkable for hypernatremia and low urine osmolality. Nephrology was consulted given concern for nephrogenic diabetes insipidus (NDI). Water deprivation test was performed, demonstrating a partial NDI likely secondary to her chronic lithium use.</p> <p>Psychiatry and Neurology were consulted for assistance in removing lithium from her regimen. Unfortunately, due to failed trials of other medications in the past, significant changes were not able to be made, so the lithium dose was further decreased.</p> <p>Multiple medication regimens to address the NDI were trialed during the hospital stay. Ultimately the most effective regimen was amiloride/HCTZ combination therapy, which was started prior to dismissal. With hypotonic fluids, lithium dose decrease, and the addition of the amiloride/HCTZ therapy, the patient's tachycardia and lithium level improved, as did her nausea, vomiting, and fatigue.</p> <p>Discussion: This case highlights the importance of recognition regarding both the acute and chronic effects of lithium on patients, as well as the treatment options for NDI, particularly in the setting of chronic lithium use. Patients with elevated lithium levels may be asymptomatic, or experience symptoms of acute lithium toxicity ranging from vomiting and diarrhea to ataxia, falls, and altered level of consciousness. Chronic toxicities from lithium use include hypothyroidism, sinus node dysfunction, or nephrogenic diabetes insipidus (NDI), where it is the most common cause of drug-induced NDI. In our patient, the development of NDI from chronic lithium use lead to polyuria, and ultimately to a dehydrated state causing acute elevations in her lithium level. This acute lithium toxicity manifested as gastrointestinal symptoms, further worsening her dehydration. After fluid resuscitation and additional therapies targeting her NDI, her lithium level decreased and her acute symptoms dissipated.</p>

<p><b>Kate Richards May</b> Dr. Amy Holbrook</p>	<p><i>A Cryptic Cause of Ataxia</i></p> <p>Introduction: Infections are a major cause of morbidity and mortality in immunocompromised patients. Although the majority are due to bacterial infections, the prevalence of invasive fungal infections is increasing. The rising incidence should raise providers' suspicion for these infections when evaluating immunocompromised patients.</p> <p>Case Presentation: A 70-year-old woman with multiple myeloma undergoing treatment and paroxysmal atrial fibrillation on anticoagulation was evaluated for two weeks of headaches, nausea and unsteady gait. Her presentation was initially attributed to subacute cerebellar strokes found on brain MRI. The patient was continued on warfarin therapy due to presumed cardioembolic strokes. Several weeks later, however, the patient developed worsening symptoms and confusion. Repeat MRI revealed numerous lesions in the bilateral cortex, basal ganglia and cerebellar regions with diffuse luminal irregularity of the intracranial vasculature. Lumbar puncture showed cloudy CSF, opening pressure 20, glucose 3, WBC 22, protein 68. Cultures were positive for yeast with CSF cryptococcal antigen titer of 1:2560. CSF analysis and imaging were consistent with cryptococcal meningoencephalitis in the setting of immunosuppression from multiple myeloma and treatment with lenalidomide. HIV testing was negative. Induction therapy was initiated with IV liposomal amphotericin B and flucytosine. Additionally, the patient required multiple therapeutic lumbar punctures due to clinical signs of increased intracranial pressure and ultimately required lumbar drain placement.</p> <p>Unfortunately, the patient made minimal clinical improvement despite treatment and became progressively more somnolent. On hospital day ten the patient developed massive bilateral strokes and passed away shortly thereafter.</p> <p>Conclusion: This case illustrates the importance of maintaining a high index of suspicion for atypical infections in immunocompromised patients. Invasive fungal infections like Cryptococcus can present insidiously and progress rapidly causing significant morbidity and mortality. The prevalence of immunocompromised patients is increasing as the indications for immunosuppressive treatments are expanding. Therefore, it is important to consider invasive fungal infections when evaluating the immunocompromised patient.</p>
<p><b>Anthony Robateau</b> Dr. Babusai Rapaka Dr. Xiao Jing Wang</p>	<p><i>An Unusual Presentation of Acute Pancreatitis</i></p> <p>Case Presentation: A 75-year-old woman presented to the emergency department with 2 weeks of non-radiating epigastric pain and nausea. Her medical history was notable for collagenous colitis treated with budesonide, hypertension, and remote cholecystectomy for cholecystitis. She reported approximately 6 oz of daily alcohol consumption with no tobacco or illicit drug use.</p> <p>Her vital signs were significant for a blood pressure of 215/105 mmHg and was otherwise within normal limits. Physical exam was remarkable for epigastric tenderness. Initial lab workup revealed Hgb of 10.0 g/dL with unremarkable metabolic and liver chemistries. Triglyceride level was 164 mg/dL, and lipase of 101 U/L. CT imaging revealed diffuse inflammatory</p>

	<p>changes of the pancreas with an inferior pancreaticoduodenal artery pseudoaneurysm. Minutes after finishing the CT, patient became unresponsive and hypotensive. Repeat CT imaging after fluid resuscitation revealed a large amount of intraperitoneal blood. This prompted emergent interventional radiology guided coil embolization. Angiogram during the procedure revealed segmental narrowing with areas of ectasia of the right hepatic arterial vasculature concerning for vasculitis</p> <p>After the patient was stabilized, she developed melanic stools and dysphagia with pills. Esophagogastroduodenoscopy revealed circumferential ischemic appearing esophagitis of the distal 3rd of the esophagus as well as ischemic appearing duodenitis with ulceration consistent with recent embolization. With the constellation of abnormal angiography, hypertension, and ischemic esophagus it was hypothesized the patient's pancreatitis was secondary to polyarteritis nodosa (PAN). Her ischemic esophagitis was likely sequelae of her hypotensive episode, though underlying vasculitis may have contributed to its development. IG4, antinuclear antibodies, cryoglobulins, compliment levels and anti-neutrophil cytoplasmic antibodies were negative. HIV, hepatitis B and C were negative as well.</p> <p>Conclusion: PAN is a form of medium vessel vasculitis characterized by transmural inflammation of the vessel walls, which can cause to luminal narrowing or lead to formation of aneurysms. The formation of these structures can ultimately lead to ischemia. Pancreatic presentation of PAN is rare, and few cases have been reported and prognosis tends to be poor [1,2]. Fortunately, our patient had a remarkable recovery with conservative measures, and PAN directed therapy was deferred to the outpatient setting.</p> <p>References:  1. Suresh, E., et al., Acute pancreatitis with pseudocyst formation in a patient with polyarteritis nodosa. J Rheumatol, 2005. 32(2): p. 386-8.  2. Flaherty J, Bradley EL 3rd. Acute pancreatitis as a complication of polyarteritis nodosa. Int J Pancreatol. 1999 Feb;25(1):53-7.</p>
<p><b>Malvi Savani</b>  Dr. Dr. Katti  Woerner  Dr. Lihong Bu  Dr. Mark  Birkenbach  Dr. Keith Skubitz</p>	<p><i>Pegylated liposomal doxorubicin-induced renal toxicity in retroperitoneal liposarcoma</i></p> <p>Introduction: Doxorubicin is one of the most active drugs for sarcoma. Pegylated liposomal doxorubicin (PLD) is a unique formulation of doxorubicin, which carries a more favorable toxicity profile in comparison with free doxorubicin. The main toxicity of PLD is hand-foot syndrome. Unlike free doxorubicin, PLD is unlikely to cause alopecia, nausea, myelosuppression, or cardiotoxicity. Additionally, no premedications are required.</p> <p>Case Presentation: We describe the case of a 50-year-old man with advanced retroperitoneal liposarcoma who developed irreversible PLD-associated progressive renal failure requiring chronic hemodialysis due to a thrombotic microangiopathy. No cardiotoxicity was noted 84 months after he first initiated PLD.</p> <p>Conclusion: This case describes a lesser-known toxicity of PLD and may be a toxicity of long-term treatment with other liposomal drugs.</p>
<p><b>Kirsten Shaw</b>  Dr. Anya Jamrozy</p>	<p><i>A Little Fatigue, A Lot of T3</i></p>

	<p>Introduction: Wilson’s Temperature Syndrome (WTS) is a constellation of nonspecific symptoms, some of which include a low-normal body temperature, headaches, and fatigue, that was rejected by the American Thyroid Association as a valid medical diagnosis in 2005. Originally proposed in 1990 by Dr. E Denis Wilson, the etiology was theorized to be an impaired ability to convert T4 to T3, although this was not corroborated in thyroid lab abnormalities. Despite being publicly rebuffed as a true medical diagnosis, WTS has continued to gain traction amongst certain alternative medical groups.</p> <p>Case Presentation: A 58-year old woman who had recently undergone an extensive workup for fatigue with her primary care doctor, presented to the ED for chest pain and lightheadedness. Her troponin was found to be elevated to 3.7 ng/mL, and her BNP was elevated to 4,568 pg/mL. She had no prior cardiac history, and had recently had an ECHO with normal findings. Physical exam revealed a euvolemic appearing woman without jugular venous distention or peripheral edema. There was no clubbing or cyanosis of her peripheral extremities. Her heart rate and rhythm were within normal limits. Her EKG did not have any acute ST changes, and her CXR was unremarkable. She was given aspirin and started on therapeutic heparin. Upon admission to the medical floor, a point-of-care bedside ultrasound by internal medicine revealed hypokinesis of the ventricular apex, raising the question of a stress cardiomyopathy. Formal ECHO revealed an EF of 30% with hypokinesis of the entire apex and mid ventricle, once again thought to be suspicious for a stress cardiomyopathy. She underwent coronary angiogram which revealed completely normal coronary arteries. Given her new cardiomyopathy without a coronary artery culprit lesion, the patient underwent a cardiac MRI which confirmed a stress cardiomyopathy of the mid-ventricular variant. Meanwhile, laboratory workup was completed which revealed a TSH that was undetectable, a T4 that was low at 0.53 ng/dL, and a T3 that was severely elevated to 6.37 pg/mL. Patient disclosed that she had been diagnosed with WTS by a naturopathic nurse practitioner, and had been started on Liothyronine (T3) 5mcg BID, which was up-titrated to 40mcg BID over the past 6-8 weeks prior to her hospital admission. The patient's stress cardiomyopathy was presumed to be due to exogenous thyrotoxicosis. She was strongly encouraged to cease taking her thyroid supplement, and within four weeks, her EF had improved to 40% upon repeat ECHO.</p> <p>Conclusion: This case highlights the importance of asking about natural medicines/supplements while gathering a patient history, the significant medical complications that supplements can cause, and the importance of physician awareness of alternative medicine diagnoses and treatment regimens that can cause harm to patients.</p>
<p><b>Danielle Shilling</b> Dr. Christine Klassen</p>	<p><i>MRI in Occult Primary Breast Cancer (OPBC): A Case Complicated by Paraneoplastic Syndrome</i></p> <p>Introduction: Adenocarcinoma in one or more axillary lymph nodes without clinical, ultrasonographic, or mammographic evidence of primary disease in the breast is defined as occult primary breast cancer (OPBC). OPBCs account for less than one percent of all breast cancers, and rates are declining due to improvements in mammographic techniques, as well as the introduction of breast MRI. This report reviews the case of a woman with OPBC presenting with neurological paraneoplastic syndrome and current evidence on the utility of breast MRI in detecting primary lesions in cases of biopsy proven OPBC.</p>

	<p>Case Presentation: A 49-year-old female with a 2 year history of progressive neurological symptoms found to have CSF positive for amphiphysin antibodies presented to the breast clinic for evaluation of right axillary lymph node biopsy demonstrating estrogen receptor (ER)/progesterone receptor (PR) positive, HER2/Neu negative metastatic ductal carcinoma of the breast. A screening mammogram performed 1 week prior to her biopsy was negative. A subsequent PET/CT demonstrated hypermetabolic right axillary lymph nodes leading to core biopsy. Subsequent breast MRI was unable to identify a primary lesion. The patient underwent right axillary lymph node dissection with pathology demonstrating 15 of 18 lymph nodes positive for metastasis ranging 3-21 mm in size with extranodal extension and lymphovascular invasion. The patient's neurological symptoms began to improve following this surgery, and she is currently being managed with chemotherapy (adriamycin/cyclophosphamide followed by paclitaxel) and whole breast and axillary radiation.</p> <p>Discussion: Breasts were some of the first organs imaged with MRI, as early as the late 1970s. Breast MRI today is perhaps most recognized as a screening tool for patients at high risk for breast cancer or with dense breast tissue; however, the utility of breast MRI in the setting of OPBC is still being studied. In one case series, Stomper et. al. found that breast MRI led to identification of primary lesions in 2 of 8 patients, while De Bresser et al. in their systemic review found suspect lesions in 36-86% of patients with OPBC. This review determined the pooled sensitivity of breast MRI to detect the primary lesion in OPBC was 90%, but pooled specificity was only 31%. While primary lesions still escape detection despite the addition of MRI, and MRI increases the chances of false positive findings, De Bresser et al. found that 35% of OPBC patients found to have a primary lesion on MRI were able to be treated with breast conserving surgery over mastectomy, suggesting MRI should be standard of care in OPBC.</p>
<p><b>Lynn Sipsey</b> Dr. Breanna Zarmbinski</p>	<p><i>A Curious Case of Paralysis: Enteric infectious Botulism</i></p> <p>Introduction: Botulism is a rare but life-threatening cause of paralysis. Intoxication with botulinum neurotoxin naturally occurs through ingestion, wound infection, or intestinal colonization.</p> <p>Case presentation: A 40 year old female with a history of Crohn disease, on infliximab, s/p partial colectomy and small bowel resection, with high ostomy output, on magnesium supplement, presented with a two day history of progressive bulbar weakness with slurred speech, difficulty managing her secretions, and double vision. She denied recent infection, ostomy output changes, tick bites, or ingestion of any home canned foods. Workup revealed normal electrolytes, vitamin B12 level, extensive cerebral spinal fluid studies, and neuroimaging. During her admission, a descending flaccid paralysis progressed from her neck to her proximal upper extremities, diaphragm, and eventually lower limbs, though sensorium was preserved. In week two, her pupils became unreactive. Further workup included negative myasthenia gravis panel and Lyme and West Nile Virus PCR. EMG was consistent with a neuromuscular junction process. Given her presentation and history of autoimmune disease, she was treated for seronegative myasthenia gravis with Intravenous Immunoglobulin (IVIG) and Methylprednisolone. She did not improve significantly and was ultimately discharged to a long-term care facility on a ventilator with a tracheostomy and a gastrostomy tube. During her stay she did not clinically improve. Additional workup was sent with stool sample evaluating for botulism PCR, which resulted positive. This was</p>

	<p>confirmed with a mice lethality assay and was found to have Botulinum Toxin B and F. The case was discussed with the Center for Disease Control which initially recommended against administration of antitoxin, however given a suspicion of intestinal colonization, antitoxin was administered. Additionally, a course of penicillin, metronidazole, and vancomycin enemas were administered, per the recommendations of Infectious Disease consultants. Fecal transplant was considered, but lack of donor material in the setting of COVID-19 was prohibitive. She improved significantly and no longer requires a ventilator and has progressively improving strength.</p> <p>Discussion: Intestinal overgrowth by C Botulinum is a rare presentation of a rare intoxication, but when it occurs it is almost exclusively in patients with GI tract abnormalities. In 2017, only 1% of 141 lab confirmed cases of botulism were suspected adult colonization. In adults, Botulism toxin infection presents with bulbar weakness and descending paralysis secondary to neuromuscular junction pathology, similar to myasthenia gravis. However, myasthenia gravis does not present with autonomic symptoms such as dilated pupils. Myasthenia gravis is treated with steroids, IVIG, and plasmapheresis.</p> <p>Conclusion: CDC recommends initiating treatment at the initial clinical suspicion before laboratory confirmation. However, it must be understood, administration of antitoxin does not reverse paralysis, but halts further progression.</p>
<p><b>Allison Smith</b></p>	<p><i>Erythema Multiforme as Presenting Rash in Kawasaki Disease in an Adult</i></p> <p>Case Presentation: A 19 year old male without significant past medical history presented to the emergency department after developing conjunctivitis followed by a diffuse, full body maculopapular rash involving the palms and soles. He was found to be febrile with associated mucositis. Biopsy of the rash was consistent with erythema multiforme. The remainder of his infectious work-up was negative. Overall, diagnosis was most consistent with Kawasaki’s disease, meeting all of the major diagnostic criteria. Diagnostic criteria include otherwise unexplained fever for 5 days and at least 4 of the following: bilateral conjunctivitis, oral mucosa findings, erythema of the palms and soles, polymorphic rash, and cervical lymphadenopathy. He was treated with IVIG, methylprednisolone, and aspirin. Cardiac evaluation with cardiac CTA revealed no coronary artery aneurysms and transthoracic echo has remained normal with serial evaluation. He clinically improved with treatment without further recurrence or complications.</p> <p>Discussion: Kawasaki disease (also known as mucocutaneous lymph node syndrome) is a medium-sized vessel vasculitis that is most often seen in children under 5 years old and is rarely reported in healthy adults. There are no diagnostic histologic findings with Kawasaki’s disease, but erythema multiforme has been described in the literature. As this is typically a disease of childhood, it is important to continue to recognize its incidence into adulthood, particularly as early diagnosis and treatment predicts better prognosis.</p>
<p><b>Nuttavut Sumransub</b>  Dr. Paari Murugan  Dr. Shelly Marette  Dr. Denis Clohisy  Dr. Keith Skubitz</p>	<p><i>Multiple tumors in a patient with familial chordoma</i></p> <p>We describe a 67-year-old woman who developed an undifferentiated pleomorphic sarcoma in her thigh. She had a very rapidly progressive course of disease with pulmonary metastases despite the treatment with standard chemotherapy regimens and passed away 8 months from diagnosis with</p>



	<p>pulmonary complications. Her medical history was remarkable in that she had a sphenoid-occipital chordoma at age 39 and later developed multiple other tumors throughout her life including Hodgkin lymphoma, and squamous cell carcinoma and basal cell carcinoma of the skin. She had a family history of chordoma and her family underwent extensive genetic study in the past and were found to have a duplication of the TBXT gene, a gene encoding transcription factor brachyury. This transcription factor has been found to associate with tumor progression, treatment resistance, and metastasis in various epithelial cancers, and might play roles in tumorigenesis and aggressiveness in this patient with multiple rare tumors; targeting this molecule may be useful for some malignancies.</p>
<p><b>Benjamin Swart</b> Dr. Allison Smith, Dr. Zaki Abou Zahr</p>	<p><i>Not Just Another Pair of Diabetic Feet: A Severe Case of Keratoderma Blenorrhagicum</i></p> <p><i>Introduction:</i> Reactive arthritis is one of the family of seronegative spondyloarthropathies which can present with a variety of extra-articular findings. This vignette highlights a severe case of keratoderma blenorrhagicum, a psoriasiform cutaneous manifestation of reactive arthritis which affects approximately 10% of those with the disease.</p> <p><i>Case Presentation:</i> A 61-year-old male was transferred from a rural hospital to HCMC for evaluation of a traumatic cerebellar hemorrhage. Upon admission he was found to have extensive hyperkeratotic lesions on his bilateral feet. Further questioning revealed he had a history of reactive arthritis, diagnosed when he was 22. He endorsed intermittent flares of mild hyperkeratotic skin lesions on his fingertips and toes since the time of diagnosis, some greater than 10 years apart. These had typically been attributed to onychomycosis or diabetic dermopathy, with no consistent subspecialty follow up. His most recent flare had started approximately 5 weeks prior to admission and was accompanied by diffuse arthritis, most notably of his knees. Further physical examination revealed similar hyperkeratotic lesions on his fingertips and genitals, as well as right knee effusion with enthesitis and non-traumatic rupture of the patellar tendon. Laboratory analysis revealed HLA-B27+, ESR 49 mm/hr, CRP 130 mg/L, with negative ANA, ANCA, CCP, and rheumatoid factor. There was no laboratory evidence of sexually transmitted infections or enteric pathogens. He was started on a regimen of topical keratolytics, emollients, and corticosteroids with oral cyclosporine and was later transitioned to adalimumab for long-term management. The lesions were largely resolved in one week and he remains free of flares at his most recent follow-up.</p> <p><i>Discussion:</i> Keratoderma blenorrhagicum is a clinical diagnosis based on identifying the characteristic skin lesions, typically on the tips of the fingers or the feet, in a patient with the classic symptoms of reactive arthritis. Reactive arthritis is typically triggered by infection with sexually transmitted or enteric pathogens, most commonly <i>Chlamydia trachomatis</i> or <i>Shigella</i> species, though half of newly diagnosed cases have no clear infectious source. Oral antibiotics can be used to treat the underlying infection in patients with a clear source, otherwise NSAIDs, systemic glucocorticoids, oral DMARDs, and injectable biologics are also used. A review of the literature and internet image search underscores the severity of this patient's disease – there are few other documented photos reflecting such extensive and destructive pedal keratoderma. This case also highlights the availability and recency bias that can cloud a clinician's thinking when confronted with pieces of information that fit best into common syndromes. A brief history, even for the most</p>

	<p>menial complaints, can sometimes lead to key information that can greatly change a patient's course of care.</p>
<p><b>Absar Tahir</b> Dr. Ellie Berbari</p>	<p><i>Not a Typical Atypical Pneumonia</i></p> <p>Case Presentation: A healthy 38 year-old gentleman with no significant medical comorbidities presented to the ED with several days of sore throat, myalgias, cough, sputum production, dyspnea and fever. Chest X-ray initially showed a right middle lobe infiltrate. He was treated for community acquired pneumonia with ceftriaxone and azithromycin and sent home. He returned the next day with ongoing symptoms in addition to a new erythematous rash on the chest, red and matted eyes, facial swelling, and mucosal sloughing. He was also found to have some scattered erythematous skin lesions with central clearing. He was not exposed to any medications associated with Stevens-Johnsons syndrome (SJS). A broad infectious workup was obtained, including negative nasopharyngeal extended respiratory pathogen PCR panel (testing for adenovirus, coronavirus, rhinovirus/enterovirus, metapneumovirus, influenza, parainfluenza, RSV, Bordetella pertussis, Chlamydia pneumonia, and Mycoplasma pneumoniae), Streptococcus pyogenes PCR, MRSA nasopharyngeal swab, Mycoplasma pneumoniae serum IgG and IgM, HSV 1/2 serum IgG plus cutaneous and oral swab PCR, and HIV antibody screen. Mycoplasma pneumoniae sputum PCR was also obtained and returned positive.</p> <p>Skin biopsy was obtained with pathology showing subepidermal separation resulting from a vacuolar interface dermatitis with apoptotic keratinocytes. The patient was diagnosed with Mycoplasma pneumoniae pneumonia complicated by Mycoplasma induced rash and mucositis (MIRM). He was treated with antibiotics, oral prednisone with taper, triamcinolone 0.1% cream, lidocaine swish-and-spit, and artificial tears. He made a full recovery and was discharged to home.</p> <p>Conclusion: MIRM is a mucocutaneous finding associated with Mycoplasma pneumoniae infections. It has historically been considered to be a subtype of SJS and Toxic Epidermal Necrolysis (TEN), though it has been increasingly recognized as a unique entity with similar pathologic and clinical appearance. MIRM tends to be associated with less cutaneous involvement and a much more favorable prognosis than SJS/TEN. On average, 2-3 mucosal sites are typically affected, most commonly with oral (94%), ocular (82%), and urogenital (63%) involvement. As in this patient, oral lesions include hemorrhagic crusting of the lips and erosions on the tongue and buccal mucosa. Ocular findings include bilateral, purulent conjunctivitis, photophobia, and eyelid edema.</p> <p>Treatment of MIRM is largely supportive and includes treatment of the associated Mycoplasma pneumoniae infection. Corticosteroids may be trialed, though there is little evidence to support their use. Dermatology and Ophthalmology consultations can be valuable for diagnostic clarification and treatment assistance.</p>
<p><b>Alex Tarabockia</b> Dr. Christopher Wittich</p>	<p><i>Leukemia Cutis: An Extramedullary Leukemia in a Patient with Bone Marrow Failure</i></p> <p>Introduction: Leukemia Cutis (LC) is an extramedullary form of leukemia that presents most often as a nodular or plaque like rash affecting the superficial and deep dermal layers of the skin. Rarer presentations include erythematous</p>

	<p>macules, blisters, and ulcers. We present a case of LC in a patient with longstanding hematological abnormalities including thrombocytopenia diagnosed 25 years ago which has now progressed to bone marrow failure with myelofibrosis and pancytopenia.</p> <p>Case Description: The patient is a 70-year-old man with past medical history of mild diastolic heart failure, prior tobacco use, mild COPD, hypertension, dyslipidemia, and myelofibrosis with pancytopenia requiring supportive transfusions who presented for evaluation of a new rash over the past one month. Review of systems was significant for arthralgias over a several month period treated with an increasingly higher dose of corticosteroids. Physical exam was significant for scattered, erythematous, tender, and rubbery with palpation randomly distributed over the upper upper extremities, scalp, chest, and thighs. The sizes ranged from 1-4 centimeters. Given the atypical distribution he was referred to dermatology and he underwent biopsy. The pathology was consistent with leukemia cutis, prompting further evaluation with bone marrow biopsy and translocation analysis. These additional tests showed no transformation to acute leukemia, hypocellularity, and 2-3 % blasts. He was started on ruxolitinib for myelofibrosis therapy.</p> <p>Discussion: LC occurs is an extramedullary form of leukemia most commonly seen in AML and CLL (3-30%). The majority of patients diagnosed with LC have a known prior diagnosis of leukemia (55-77%), others are diagnosed with leukemia simultaneously (23-38%), and a very small minority of patients are aleukemic (7%). The latter, like our patient, almost always go on to develop leukemia over the span of a couple of months to years. Unfortunately the prognosis is quite grim with an average life expectancy of 1-3 months at time of diagnosis. The most classic physical exam finding is a nodular or plaque like rash. In rare cases it can present as erythroderma, ulcers or blisters. The differential is vast and includes non-malignant and malignant skin disorders. Non-malignant mimickers may include erythema nodosum, syphilis, psoriasis, drug eruption, viral infection, ulcering of various origins, and blistering diseases. Malignant mimickers may include Sweet's Syndrome, primary systemic amyloidosis, dermatomyositis, neurofibromatosis, lymphoma, and primary tumor invasion of the skin. The appearance of a plaque like or nodular rash that does not follow a classic distribution of any of the above, particularly in a patient with constitutional symptoms or a prior malignancy, should prompt a more extensive workup including blood work, dermatological evaluation, skin biopsy, and bone marrow biopsy if warranted. The treatment of all paraneoplastic skin disorders, including LC, requires treatment of the underlying malignancy.</p>
<p><b>Reema Tawfiq</b>  Dr. Jonathan Knott  Dr. Alexander Theofiles</p>	<p><i>A Shawl Only Steroids Can Remove: A Unique Case of Dermatomyositis</i></p> <p>Introduction: Dermatomyositis (DM) is an inflammatory myopathy characterized by proximal skeletal muscle weakness, muscle inflammation, and distinctive skin manifestations. Anti-nuclear matrix protein 2 (NXP-2) is a less common and newly recognized auto-antibody specific to idiopathic inflammatory myopathies. Anti-NXP-2 was previously associated with dermatomyositis in children, but more recently has been identified in adult patients with a highly characteristic clinical presentation of DM.</p> <p>Case Presentation: A 61-year-old man consulted his doctor for bilateral arm and leg pain, proximal muscle weakness, and a rash on the chest and face. After completing a short course of oral steroids, his symptoms worsened and he presented to urgent care. He was found to have elevated serum creatinine</p>

	<p>kinase (CK) and was admitted for treatment of rhabdomyolysis. Common autoimmune antibodies were within normal limits. Despite aggressive hydration and pain medication, his symptoms continued to worsen. His CK and liver enzymes continued to trend up and he was transferred for further evaluation and management of presumed inflammatory myopathy versus steroid-induced myopathy. He presented with severe myalgia, skeletal muscle weakness, and skin findings that included a heliotrope rash, Gottron papules, the shawl sign, and the V sign. Laboratory studies showed markedly elevated CK with abnormal liver transaminases, aldolase, ESR, and CRP. Complete blood cell count, creatinine, TSH, anti-nuclear antibodies, antibodies to extractable nuclear antigens, and HMG-CoA reductase antibodies were normal or negative. An extended myositis marker panel (MyoMarker 3) was sent and pending during the hospital stay. Electromyography was positive for evidence of myopathy, but surgical muscle biopsy was indeterminate. The patient was diagnosed with rhabdomyolysis secondary to dermatomyositis. Given dermatomyositis's high association with malignancy, a thorough work-up, including Positron-Emission Tomography, was done and was unrevealing.</p> <p>Treatment was initiated immediately with aggressive IV fluid hydration and IV methylprednisolone, which was transitioned to oral prednisone. Unfortunately, while on oral steroids his symptoms worsened and CK increased. He was transitioned back to IV methylprednisolone for 3-days and treated with 5-days of intravenous immunoglobulin (IVIg). His strength and symptoms began to improve, and his CK trended down. Before dismissal from the hospital, CK was below 5,000, IV fluids were discontinued, and he was transitioned to oral prednisone and mycophenolate with plans for close rheumatology follow-up and continued outpatient malignancy surveillance.</p> <p>The MyoMarker 3 panel later returned positive for anti-NXP-2, which supported the diagnosis of a unique subtype of severe DM.</p> <p>Conclusion: Autoantibodies, including anti-NXP-2, have been identified as specific markers for DM and are associated with unique clinical subsets of DM. Although valuable in making the diagnosis and monitoring clinical manifestations, antibody testing is not required to diagnose dermatomyositis if other diagnostic criteria are met.</p>
<p><b>Lauren Thornton</b></p>	<p><i>LAST but not the least</i></p> <p>Introduction: Local anesthetic-induced systemic toxicity (LAST) is a rare but well documented phenomenon. Regional pain management has become more popular as it is a favorable option to minimize the use of opioids for pain control. As this therapy becomes more widely utilized, it is important to know the presentation, pathophysiology, and management of LAST.</p> <p>Case description: A 44 year old woman was admitted after an out of hospital PEA arrest with prolonged ACLS, and was intubated for airway protection. She was found to have multiple bilateral rib fractures secondary to chest compressions on admission imaging. Fortunately, her clinical course was favorable and her mental status was intact. As ventilator weaning trials were pursued, she was only able to take rapid shallow breaths due to pain. The primary team spoke with the regional anesthesia team to pursue bilateral erector spine plane/multiple intercostal nerve blocks with ropivacaine for directed pain control. The patient initially tolerated the bolus doses of this medication, however within minutes developed bradycardia and hypotension and ultimately went into a pulseless electrical activity. ACLS was initiated,</p>

	<p>and point of care ultrasound revealed dilated left atrium and left ventricle, as well as severely decreased biventricular function compared to a formal transthoracic echocardiogram completed two days prior which showed preserved EF of 52% and no chamber enlargement. During the code blue, the patient received intravenous lipid emulsion infusion as a reversal agent. ROSC was achieved and fortunately patient's neurologic status remained intact. Ultimately, she received percutaneous tracheostomy and was discharged to continue ventilator weaning.</p> <p>Discussion: This report describes a severe case of local anesthetic-induced systemic toxicity. As use of regional pain control becomes more common, it is important for the medical team to be aware of adverse effects and management. The use of intravenous lipid emulsion has been well studied as a reversal agent of local anesthetics in the setting of cardiotoxicity, however further studies are required to determine optimal timing and dosage of this therapy.</p> <p>References:</p> <ol style="list-style-type: none"> <li>1. Butterworth JF., 4th Models and Mechanisms of Local Anesthetic Cardiac Toxicity. Reg Anesth Pain Med. 2010; 35:167-76</li> <li>2. Corman SL, Skledar SJ. Use of lipid emulsion to reverse local anesthetic-induced toxicity. Ann Pharmacother. 2007 Nov;41(11):1873-7. Doi: 10.1345/aph.1K244. Epub2007 Sep 25. PMID: 17895327.</li> <li>3. Kosh MC, Miller AD, Michels JE. Intravenous lipid emulsion for treatment of local anesthetic toxicity. Ther Clin Risk Manag. 2010;6:449-451. Published 2010 Oct 5. Doi: 10.2147/TCRM.S11861.</li> </ol>
<p><b>Alessandra Tomasi</b> Ashley Jacobson Cameron Wangsgard</p>	<p><i>Bannwarth Syndrome and Complete Heart Block: An Unusual Case of Disseminated Lyme</i></p> <p>Case Presentation: A 35-year-old man with no medical history presented with diffuse generalized pain and trismus. Originally from Guatemala, he moved to Minnesota one year ago and works on a dairy farm. He was in his normal state of health until one month prior to presentation, when he noted erythema on his left toe that subsequently spread throughout his foot. At this time he also developed fevers. One week following resolution of the rash, he began to experience sore throat, neck pain, and upper and mid back pain. He presented to the Emergency Department in July for evaluation of worsening pain, new odynophagia, and an inability to open his mouth fully which resulted in a 5-10 pound weight loss. On arrival, vital signs were notable for a heart rate between 40-60 and a temperature of 37.8oC. Physical exam showed a diaphoretic male in mild distress due to pain. He had generalized tenderness to palpation of the neck, shoulders, and upper and mid back, and significantly limited range of motion of the neck and mouth. CBC revealed a mild neutrophil-predominant leukocytosis. CT of the soft tissues of the neck was obtained due to odynophagia and was unremarkable. ECG showed complete heart block. Tick-borne antibody panel showed a positive Borrelia burgdoferi antibody serology, and confirmatory immunoblot testing was positive for Lyme-specific IgM and IgG. Lumbar puncture was also performed. CSF analysis showed lymphocytic-predominant pleocytosis, mildly elevated spinal fluid protein, and reactive Lyme CSF IgG. Blood cultures remained negative. The patient was admitted to the hospital and symptoms improved with IV ceftriaxone and oral doxycycline. Within the initial hours of his hospitalization after initiation of IV ceftriaxone, complete heart block transitioned to a Mobitz II rhythm. The patient never required cardiac pacing. His leukocytosis down-trended and he remained afebrile through the duration</p>

	<p>of his hospital stay. He was discharged on day 8 in stable condition. Subsequent ambulatory cardiac monitoring with BodyGuardian showed complete resolution of his heart block to a normal sinus rhythm. This case illustrates an example of Bannwarth Syndrome, an uncommon manifestation of disseminated Lyme disease characterized by painful radiculopathy, neuropathy, and motor and facial weakness. Our patient also exhibited signs and symptoms more frequently associated with Lyme disease including erythema migrans, aseptic meningitis, and complete heart block. However, his chief complaint of diffuse pain, trismus, and weight loss suggested an additional manifestation of this neuroinvasive infection. Interestingly, Bannwarth Syndrome is common in Europe and is less prevalent in North America, though clusters of the disease have previously been reported in the Midwestern United States. It is speculated that the underlying pathophysiology is a vasculitic process due to immune infiltration, resulting in axonal degeneration. Treatment with parenteral antibiotics typically leads to complete resolution.</p>
<p><b>Christine Wagner</b> Dr. Pezhman Roohani</p>	<p><i>A Rare Presentation of a Rare Disease: Creutzfeldt-Jakob Disease Presenting as Status Epilepticus</i></p> <p>Introduction: Creutzfeldt-Jakob disease (CJD) is a rapidly progressive spongiform encephalopathy, occurring one per 1,000,000 people worldwide per year. Myoclonus and neuropsychiatric symptoms at presentation are common, whereas seizures reportedly are exceedingly rare. Establishing a clinical diagnosis prior to the advanced stages of the disease requires a thoughtful exam and early identification of characteristic imaging abnormalities.</p> <p>Case Presentation: A 53-year-old male with no past medical history presented to a local hospital with dysgraphia and mild cognitive changes. MRI showed multiple abnormalities reported to appear consistent with embolic strokes. Echocardiogram showed no abnormalities. An internal loop recorder was placed and he was discharged. In the following weeks he presented to his primary physician on three occasions, each time describing dysgraphia and memory changes. He noted he had been working with occupational therapy but felt “things had stalled and even gone backward”.</p> <p>The patient presented to our hospital demonstrating a clinical picture called Gerstmann’s Syndrome (agraphia, acalculia, finger agnosia, and inability with left right differentiation) and right sided myoclonus. MRI showed cortical diffusion restriction bilaterally along the cortical ribbon only, a finding that can be described with post ictal changes. EEG revealed status epilepticus. He was started on multiple antiepileptic drugs to which seizures were refractory. Autoimmune encephalopathy panel and lumbar puncture were normal. Studies for other infectious and malignant etiologies remained negative. He progressively declined, requiring intubation for airway protection. IVIG and corticosteroids provided no benefit. Given MRI findings and suspicion for CJD, the CSF, RT-QuIC test to detect prion protein was sent that ultimately returned positive. Now, MRI, EEG and CSF were consistent with CJD. He was extubated to comfort cares and expired 3 days later.</p> <p>Discussion: This case highlights an unusual presentation of CJD. The presence of status epilepticus in this patient provided an alternative diagnosis and explanation for the MRI findings, clinical picture, and cognitive decline, making the true diagnosis elusive. CSF protein markers can help aid in the diagnosis. RT-QuIC assay, for detecting prion proteins, is now included in the</p>

	<p>diagnostic criteria given its high sensitivity (80%) and specificity (100%). MRI stands as the most useful radiographic study where lesions accepted can be found in the striatum, the thalamus, and along the cortical ribbon (cerebral or cerebellar). This case brings about the question of whether CJD with seizure is indeed more common than we think. Given the right clinical context, CJD ought to be a consideration in patients presenting with focal status epilepticus.</p>
<p><b>Jordyn Walter</b> Dr. Meghana Ghattu Dr. Lucas Ramsey</p>	<p><i>What goes around comes back around</i></p> <p>Introduction: Reactivation of previously indolent diseases can be seen in post viral syndromes while the immune system is recovering. Most commonly cited are reactivation of the herpes family of viruses. In the time of the COVID pandemic, COVID-19 has been thought to cause an immunocompromised state leading to reactivation of previous diseases.</p> <p>We report a case of a previously healthy 41-year-old female who presented with painful cervical lymph nodes and discomfort swallowing. Weeks prior to presenting, she developed fever and upper respiratory symptoms at which time she tested positive COVID. She was managed conservatively and recovered quickly without requiring hospitalization. Two weeks after her COVID diagnosis, she presented with the aforementioned complaints. Rapid strep and heterophile antibodies were negative, and she was diagnosed with a general lymphadenitis and tonsillitis. She was prescribed Augmentin and, days later, erupted in a rash thought to be a drug reaction. Antibiotics were changed to Azithromycin. A few days later, she had progressive painful lymphadenopathy with recurrent fevers and presented to the hospital. She had a leukocytosis to 18 with elevated inflammatory markers. Her mononucleosis test was negative. Infectious disease was consulted out of concern for tuberculosis. A CT chest/abdomen/pelvis was done that did not show further lymphadenopathy or pulmonary lesions. She then decompensated and was transferred to the ICU for hypotension. Broad-spectrum antibiotics were started and her condition improved. AFB smears came back negative. A lymph node FNA was performed which eventually revealed a necrotizing lymphadenitis with EBV inclusion bodies consistent with a reactivated EBV infection, most likely triggered by her recent COVID infection. Her latent disease state was supported by positive VCA IGG and EBNA IGG. She was then discharged with infectious disease follow up in stable condition.</p> <p>Conclusion: EBV can reactivate to lytic stage in immunocompetent patients, but rarely causes symptoms. Systemic infection, including mild COVID-19 infection can cause a relative immunosuppressed state, which may lead to a symptomatic reactivation of indolent EBV infection. The heterophile antibody for detecting EBV is highly sensitive (85%) but has a high false negative rate in early infection. EBV specific antibodies are highly sensitive (97%) and specific (94%) for primary infection, although VCA-IgM may be negative in late primary infection or reactivation. IgG avidity can help differentiate past infection with acute infection.</p>
<p><b>Elizabeth Wendt</b></p>	<p><i>Subacute Presentation of Transverse Myelitis secondary to Neuromyelitis Optica</i></p> <p>Introduction: This case explores a subacute presentation of transverse myelitis secondary to neuromyelitis optica (NMO) in a patient with waxing and waning neurological symptoms throughout hospitalization. The case emphasizes the importance of thorough and frequent neurological</p>

	<p>examination by the hospitalist physician and discusses utility of frequent measurement of inflammatory markers such as CRP in acute and subacute presentations of possible inflammatory pathology.</p> <p>Case Presentation: Patient is a 43-year-old woman with PMH of rheumatoid arthritis, migraine with aura associated with intermittent right leg numbness, and depression/anxiety who presented with thoracic back pain and nausea. Initial exam with intact cranial nerves, decreased sensation to light touch over RLE in no particular distribution, 4/5 strength in RLE, no midline spinal tenderness. Initial laboratory workup with unremarkable CBC, chem, d-dimer, troponin, and CRP. Over hospital days 1-4, patient with waxing and waning RLE weakness, development of point spinal tenderness in thoracic region, and new onset urinary retention. CT negative for cervical, thoracic, lumbar spinal pathology. On hospital day 5, patient with strikingly abnormal neurological examination including right gaze nystagmus, 4/5 strength with R hip flexion/extension, absent vibratory sensation in bilateral big toes, brisk reflexes throughout, and patchy reduced sensation up to the thorax without clearly discernable spinal level. MRI obtained and with increased T2 signal indicating long segment central cord involvement (C1-T10). Repeat CRP now with marked abnormality at 39 (reference &lt;5 mg/dL). Given the subacute nature of symptom development, presentation most concerning for transverse myelitis with demyelinating, infectious, or other autoimmune/rheumatologic etiology (multiple sclerosis, systemic lupus erythematosus, hypothyroid, neuro-Behcet's, anti-NMDA encephalitis); less likely compressive. MRI brain without MS findings and cerebrospinal fluid with lymphocytic pleocytosis, often consistent with NMO. Ultimately, CSF was NMO/aquaporin 4 antibody positive. Patient started on pulse steroids and IVIG with subsequent improvement in her neurologic examination. Patient transitioned to oral steroids and transferred to long-term rehabilitation facility with functional improvement and discharge in 1.5 months.</p> <p>Discussion: Key lessons from this case include:</p> <ol style="list-style-type: none"> <li>1. The importance of thorough, daily neurological exams, especially in patients with complex or evolving neurological complaints. Brisk reflexes, presence of a spinal level, and reduced vibratory sensation individually or together require prompt evaluation by neurology subspecialist if available and timely assessment with MRI.</li> <li>2. Utility of serial CRP in patients with evolving neurological complaints. CRP concentrations change rapidly within 6-8 hours of injury, peak at 48 hours. CRP is both more sensitive and specific than ESR to detect acute phase inflammation in patients with undiagnosed conditions. Because it is an acute phase reactant, serial measurements in the setting of progressively worsening neurological examination with concern for inflammatory pathology may have been helpful.</li> <li>3. Waxing and waning neurological symptomatology should not decrease clinician suspicion for serious neurological pathology.</li> </ol>
<p><b>Kristen Westenfield</b> Dr. Christine Wagner Dr. Kevin Harris</p>	<p><i>Acute Myopericarditis Due to Campylobacter Jejuni</i></p> <p>Introduction: Campylobacter jejuni is a common cause of diarrheal illness in the United States with more than 1.3 million people affected annually. The most common route of transmission is fecal-oral with an incubation period of two to three days followed by development of symptoms including diarrhea, abdominal pain, fever and vomiting. The infection is generally self-limiting, though complications including Guillain-Barre syndrome, reactive arthritis, and rarely myopericarditis have been reported.</p>



	<p>We report a case of <i>C. jejuni</i> infection in a healthy middle-aged female who presented to the emergency department with intermittent central chest pressure that radiated to the jaw. Five days prior to admission, she attended the Minnesota State Fair where she ate a variety of items including chicken quesadillas. The following day she developed abdominal pain with vomiting and diarrhea. Laboratory data revealed an elevated Troponin-I of 4.427 ng/mL. Initial EKG showed normal sinus rhythm without significant ST changes. She was started on a heparin infusion and transferred to a tertiary hospital for management of non-ST segment elevation myocardial infarction. Upon arrival, a repeat EKG demonstrated dynamic changes with new mild ST elevation in the inferior and lateral leads with diffuse PR depression. Repeat troponin was 5.954 ng/mL. Transthoracic echocardiogram (TTE) showed mild hypokinesis of the septum and mid-anterior wall. Cardiac magnetic resonance imaging (CMR) was performed and was consistent with myopericarditis. Stool pathogen multiplex panel PCR returned positive for campylobacter. Treatment with azithromycin was initiated. She was discharged on colchicine for myopericarditis. At three month follow-up, CMR with significant improvement in degree of delayed enhancement in all affected areas with normal EF. In this case, clinical suspicion favored pericarditis due to the infectious prodrome, lack of significant coronary risk factors and the diffuseness of the EKG changes.</p> <p>Discussion: This case demonstrates a rare complication of <i>C. jejuni</i> infection resulting in myopericarditis. In this case, clinical suspicion favored pericarditis due to the infectious prodrome, lack of significant coronary risk factors and the diffuseness of the EKG changes. While myopericarditis is not uncommon, <i>C. jejuni</i> associated myopericarditis is rare and identification of this disease is important to initiate treatment and to facilitate appropriate monitoring.</p>
<p><b>Gregory Wieland</b></p>	<p><i>An Uncommon Cause of Septic Emboli</i></p> <p>Introduction: Respiratory distress with hemodynamic instability is a common presentation for respiratory infections and sepsis. This clinical vignette details a 55-year-old male presenting to the emergency department with respiratory distress and hypotension following a recent sore throat.</p> <p>Case Description: A 55-year-old male with unknown past medical history presented to the emergency department from an outside hospital with eight days of a sore throat accompanied by throat foreign body sensation, one day of shortness of breath, and septic shock. At the outside emergency department, blood pressure was 87/40 mmHg, heart rate 125 bpm, temperature 38.4° Celsius, respiratory rate 40/minute, and oxygen saturation 89% on room air. Physical exam was notable for normal mentation and warm, well perfused extremities. Initial laboratory values were notable for a white blood cell count of 15,420 with 86% neutrophils and platelets of 11,000; lactate of 8.0 mmol/L; venous blood gas with pH of 7.10, pCO<sub>2</sub> of 38 mmHg, and HCO<sub>3</sub> of 12 mEq/L; and creatinine of 5.3 mg/dL. Upon arrival to the outside hospital he was placed on BiPAP. He was initiated on intravenous fluids, norepinephrine, vancomycin, and piperacillin/tazobactam and was transferred to our hospital for a higher level of intensive care.</p> <p>Upon arrival, he immediately underwent intubation due to increased work of breathing despite BiPAP. Bedside ultrasound demonstrated hyperdynamic cardiac function without evidence of vegetation and revealed a right internal</p>

	<p>jugular venous thrombus. Neck CT confirmed this thrombus as well as a right tonsillar phlegmon invading into the deep neck space to the carotid sheath. Chest CT showed multiple bilateral septic emboli. These findings together were most consistent with Lemierre syndrome. Blood cultures returned positive for <i>Fusobacterium necrophorum</i> and <i>Streptococcus constellatus</i>. Vancomycin and piperacillin/tazobactam were initially continued with resolution of the septic shock. However, over the course of the next 2 weeks, the patient continued to require high levels of oxygen support. He developed worsening renal failure and was initiated on continuous renal replacement therapy. His course was further complicated by a cardiac arrest due to respiratory failure on hospital day 13, and he developed worsening multiorgan failure after this. With his family it was decided to transition to comfort focused cares on hospital day 18, and he expired shortly after.</p> <p>Discussion: This case represents Lemierre syndrome, defined by septic thrombophlebitis of the internal jugular vein. Though uncommon in the age of antibiotics, it should be suspected when sepsis is present following recent pharyngitis and when septic emboli are present without evidence of infective endocarditis. It is most commonly caused by <i>Fusobacterium necrophorum</i>, but may be caused by other organisms and can be polymicrobial. Early initiation of broad-spectrum antibiotics with anaerobic coverage is the mainstay of treatment, and mortality increases when this is delayed.</p>
<p><b>Talitha Wilson</b> Dr. Jacob Ortiz</p>	<p><i>'There is a pill for that': Encapsulating peritoneal sclerosis as a treatable cause of intestinal obstruction and malnutrition</i></p> <p>Introduction: Encapsulating peritoneal sclerosis (EPS) is a clinical syndrome in which excessive peritoneal fibrous tissue forms around the intestinal tract. It is classically associated with long term peritoneal dialysis, but rarely, it has been seen in patients with organ transplantation, end stage liver disease, and calcineurin inhibitor therapy. Over time EPS causes intestinal encasement and obstruction; ultimately it can lead to deadly malnutrition and sepsis. Treatment with immunosuppressive medications and tamoxifen has been shown to improve survival for patients with peritoneal dialysis related EPS. We report a case of EPS outside of the context of peritoneal dialysis that was effectively treated with tamoxifen plus prednisone.</p> <p>Case Description: A 49 year-old man presented with severe malnutrition due to intractable vomiting and 20lb weight loss over the prior two months. Four years ago he underwent liver transplant complicated by renal failure requiring intermittent hemodialysis and gastrostomy tube dependence. On exam he had diffuse mild abdominal tenderness. He was diagnosed with a partial small bowel obstruction thought to be secondary to adhesions from prior abdominal surgeries and recurrent <i>clostridium difficile</i> colitis. He went for a revision to a gastrojejunostomy tube and was able to tolerate uptitration of jejunal feeds. Given the degree of malnutrition and multi-organ disease, palliative care was consulted for possible transition to hospice.</p> <p>During a readmission for weakness from malnutrition, a CT showed bowel wall thickening with encapsulated appearance without frank obstruction. The medical teams suspected EPS but he had never received peritoneal dialysis so it was considered less likely. He eventually tolerated feeds at goal and was discharged after a prolonged hospital stay. During a third readmission in two months time for sepsis secondary to colitis he failed to improve despite antimicrobial therapy. The hospitalist and nephrology teams decided to initiate a trial of medical therapy for EPS with prednisone 40mg and</p>

	<p>tamoxifen 10mg twice daily. Over the next four months the patient's body mass index increased from a nadir of 16.4 to 19.5 and serum albumin increased from 1 to 2.3 g/dL on an oral diet with nighttime tube feeds. A repeat CT scan showed untethering of the intestines.</p> <p>Discussion: This case illustrates the diagnostic challenges of EPS in the setting of atypical risk factors, relatively low prevalence in general medicine wards, and complicated history. However it also shows that tamoxifen and prednisone helped minimize complications in a patient who had never received peritoneal dialysis. Overall, intestinal obstruction management in the hospital is common and complex. EPS is a relatively rare cause, but it deserves the attention of clinicians as conservative medical management with tamoxifen and prednisone can have a lasting improvement in quality of life.</p>
<p><b>Luke Wohlwend</b> Dr. Peter Lund</p>	<p><i>A Rare Complication from a Commonly Prescribed Drug</i></p> <p>Introduction: Statin induced autoimmune myopathy (SIAM) is a rare but serious complication of statin therapy that can occur during any time of use. It should be considered in patients who develop proximal muscle weakness and elevated creatinine kinase levels while taking statin therapy.</p> <p>Case Presentation: A 59 year old man presented to the hospital with 1 month of progressive proximal muscle weakness. His symptoms started after a fall progressing to inability to stand from sitting, inability to climb stairs and inability to wash his hair. He denied any skin changes, rashes, joint swelling, fatigue, or weight loss. Exam was notable for severe proximal upper and lower extremity weakness with preserved distal strength.</p> <p>Laboratories revealed significant elevation of CK to 8967. CRP and ESR were minimally elevated and ANA, RNP, Anti-smooth muscle antibody, and Sjogren's antibodies were negative.</p> <p>Prior to admission, the patient was evaluated by neurology with EMG which revealed diffuse fibrillations and muscle disease pattern. Neurology directed the patient to admission for IVIG therapy given EMG findings and progressive symptoms with concern for inflammatory myositis. On hospital day 2 a muscle biopsy was obtained followed by initiation of solumedrol 1 gram daily.</p> <p>Review of home medications revealed that the patient had been on atorvastatin for the prior seven years without issue. Anti HMG-CoA reductase IgG was collected to evaluate for statin induced autoimmune myopathy.</p> <p>Anti HMG-CoA reductase antibodies returned strongly positive and muscle biopsy showed mild necrotizing myopathy, securing the diagnosis of SIAM. With these findings he was started on a prolonged steroid taper, IVIG every other week, and azathioprine as an outpatient. Slow and steady improvement has occurred over 2 months but to date resolution has been incomplete.</p> <p>Conclusion: This case represents a rare but devastating complication of statin therapy. The more common statin induced myopathy occurs in 1 of 10,000 individuals and is treated with cessation of statin therapy. SIAM is seen in 1 of 100,000 individuals and can progress to bulbar or respiratory symptoms if untreated. Cessation of statin therapy is inadequate for SIAM and immunomodulation is required. Typical therapy included high dose</p>

	<p>corticosteroids and IVIG with adjuvant therapies such as methotrexate, azathioprine, mycophenolate or rituximab selected based on clinical characteristics. Response to therapy is variable and prolonged or indefinite therapy can be required. Though the majority of myalgias in patients on statin therapy are mild and in many cases unrelated to the statin, severe myositis is possible and requires thorough investigation and intensive therapy.</p>
<p><b>Adam Wolfe, MD, MPH</b>  Dr. Brandon Wiley, Dr. David M. Tierney, Dr. Catherine A St. Hill, Claire S. Smith, MS, Jesse Abelson, Dr. Roman Melamed</p>	<p><i>Change in left ventricular outflow tract velocity time integral after tPA for pulmonary embolism</i></p> <p>Introduction: Pulmonary embolism (PE) causes almost 30,000 deaths annually in the United States. Guidelines recommend tissue plasminogen activator (tPA) for high risk and select intermediate-high risk PE. Echocardiogram (echo) parameters have been used to risk stratify PE patients and predict benefit from tPA. The left ventricular outflow tract velocity time integral (LVOT VTI) is a quantitative echo measure directly related to stroke volume that has been used to predict prognosis, inversely correlates with risk of death and decompensation from PE, is independent of ventricular geometry and wall motion abnormalities, and can identify normotensive patients at high risk of decompensation. The changes in LVOT VTI before and after tPA administration have not been described.</p> <p>Methods: Retrospective observational cohort including all patients admitted (2012-2019) to a 670-bed tertiary care center for high or intermediate-high risk PE who received tPA and underwent echo before and after tPA administration. Pre- and post-tPA LVOT VTI values were compared. Change in qualitative right ventricular (RV) function and size, tricuspid annular plane systolic excursion (TAPSE) and estimated pulmonary artery (PA) pressure were also analyzed. Paired t-tests were used to determine significance.</p> <p>Results: 34 patients had LVOT VTI measurements before and after tPA administration. 91.2% had intermediate-high risk PEs. Median LVOT VTI improved from 0.14m on echo 1 to 0.20m on echo 2 (35.5%, p&lt;0.0001). Median estimated PA pressure improved from 45.3 to 33.9mmHg (-28.0%, p=0.0295). Median TAPSE improved from 1.65mm to 2.10mm (41.2%, p=0.0007). RV size and function normalized in 33% and 46% of patients respectively. LVOT VTI normalized despite ongoing RV dysfunction in 10 patients (29.4%).</p> <p>Discussion: Among people with high and intermediate-high risk PE, LVOT VTI increased significantly following tPA and mirrored improvement in RV size and function, PA pressure and TAPSE. Normalization of LVOT VTI may be a more sensitive indicator of recovery than visually estimated RV dysfunction. The LVOT VTI is easily obtained and has the potential to identify high-risk PE patients and to monitor for improvement in cardiac function following tPA.</p>
<p><b>Qiaonan Zhong</b>  Dr. Qiuyu Jin  Dr. Anna Subramaniam  Dr. Mandeep Singh</p>	<p><i>Capecitabine induced coronary vasospasm mimicking ST-elevation myocardial infarction</i></p> <p>Introduction: 5-fluorouracil (5-FU) is a very common chemotherapy agent with known significant cardiac toxicity. In patients with ischemic heart disease risk factors, acute coronary syndrome / myocardial infarction can occur. Chest pain also occurs in patients without underlying coronary artery disease. The exact mechanism remains under debate, but coronary vasospasm is commonly considered. Capecitabine, an oral prodrug of 5-FU, has also had similar adverse effects described.</p>

Case Presentation: The patient is a 39 year old male with rectal cancer undergoing neoadjuvant radiation and chemotherapy with capecitabine. He is otherwise healthy and has no personal or family history of early coronary artery disease. Two days after capecitabine initiation, the patient developed left-sided chest pain and pressure, initially on exertion but later at rest as well. Five days after capecitabine initiation, he presented to a local emergency room. Initial ECG was unremarkable. Chest CT angiogram was negative for pulmonary embolism. The patient was sent for stress echocardiogram. After receiving dobutamine, he developed severe crushing chest pain with accompanying diffuse ST elevation and T wave inversions. However, the echocardiogram showed no wall motion abnormalities and troponins remained normal. Nevertheless the patient was given aspirin, clopidogrel, and heparin bolus and was admitted to cardiology. He was started on IV nitroglycerin, which resolved his pain. Nitroglycerin was gradually weaned without recurrence of pain. ECG changes normalized entirely. He underwent a cardiac CT which showed widely patent, anatomically normal coronary arteries. Overall, it was felt that given his lack of cardiac risk factors and the reassuring echocardiogram and cardiac CT, as well as timing with exposure to capecitabine, his chest pain was most likely similar to 5-FU related coronary vasospasm. The patient was switched to an alternative agent and did not have further episodes of chest pain.

Discussion: This case highlights the importance of general medical providers to keep in mind side effects of common chemotherapeutic medications. Due to his severe chest pain the patient was not evaluated by his oncologist, but rather in emergency medicine, internal medicine, and cardiology. Given the concerning ST elevation on ECG, the patient was appropriately treated for ACS and coronary angiogram was planned. However, he was at such low baseline risk and his other studies (echocardiogram, negative troponins) that we did not feel his presentation would be consistent with a STEMI. We instead ruled out ACS with cardiac CT, which is a useful tool in this setting.