2021 Abstracts

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1School of Medicine, Medical College of Wisconsin, Milwaukee, WI
2Division of General Internal Medicine, Department of Medicine; Center for Advancing Population Science; Medical College of Wisconsin, Milwaukee, WI

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Case Based Vignettes
Introduction: Rapid accumulation of fluid within the pericardial space can lead to life-threatening cardiac tamponade. In the modern antibiotic era, purulent pericardial effusions are rare. MRSA purulent pericardial effusions are rarely described in the literature without risk factors such as immunosuppression, recent thoracic surgery, and prior pericardial disease.

Case Description: An 87-year-old male with DM2, HTN, PVD, and atrial fibrillation was admitted for several weeks of drainage from chronic right lower extremity and sacral wounds with known ischial osteomyelitis along with months of progressive immobility. Initial diagnostic studies showed polymicrobial wound cultures including MRSA and pseudomonas without bacteremia. After surgical consultation, surgery was deferred in place of a 7-day antibiotic course, wound cares, and transfer to an inpatient rehab unit for physical therapy. Two months into rehabilitation the patient required ICU transfer for atrial fibrillation with rapid ventricular response, hypotension, and fever from MRSA bacteremia. He was stabilized with vancomycin, amiodarone, and apixaban. Three days later, the patient became acutely lethargic, hypotensive, and anemic requiring vasopressor support. Exam was notable for altered mental status, fatigue, an irregularly irregular rhythm, and cool extremities. CT abdomen/pelvis discovered pericardial effusion with subsequent TTE demonstrating tamponade physiology. TTE 3 days prior to acute decompensation did not show evidence of pericardial effusion. Emergent pericardiocentesis was performed with drainage of 450cc serosanguinous fluid growing 1+ MRSA on culture (cloudy, red, cell count 7,900, 92% PMNs, 0.4% Hct, RBC 48,000). Pericardial window was deferred in favor of high dose colchicine due to clinical instability. The patient initially improved and was weaned off vasopressors, though began showing signs of frank aspiration and family elected to pursue comfort cares with death shortly thereafter.

Discussion: Despite adequate treatment of MRSA bacteremia with vancomycin, the patient developed life-threatening cardiac tamponade secondary to MRSA purulent pericardial effusion days after initiating therapy. Few case reports have previously documented this condition, which requires a high level of suspicion and prompt intervention. This case serves to demonstrate that despite adequate treatment, significant complications can arise.
Background: Tubulointerstitial nephritis and uveitis syndrome is a rare disease, which manifests with bilateral non-granulomatous anterior uveitis in combination with tubulointerstitial nephritis.

Case: Our case involves a 43 year-old female patient with past medical history of asthma, bipolar disorder, obstructive sleep apnea, type II diabetes, and vitamin D deficiency who presented to the emergency department with worsening eye pain and blurry vision of four days duration with associated clear discharge and moderate bilateral photophobia. Patient was diagnosed with anterior uveitis and prescribed prednisolone eye drops. Lyme disease, tuberculosis, syphilis and sarcoidosis were ruled out with laboratory evaluation. Patient was found to have elevated creatinine as well as proteinuria. Upon consultation with nephrology, further rheumatologic evaluation, including complement levels and anti-nuclear antibody, as well as infectious evaluation, including hepatitis and HIV, were performed. Renal biopsy was performed, which showed interstitial inflammation and no evidence of glomerular injury. In combination with anterior uveitis diagnosed earlier, TINU was diagnosed. Prednisone and trimethoprim-sulfamethoxazole, for PJP prophylaxis, was initiated.

Discussion: Diagnosis involves excluding multiple autoimmune processes, including sarcoidosis and systemic lupus erythematosus, as well as infectious processes, including HIV and hepatitis. Among patients attending uveitis clinics, prevalence of TINU is < 0.1 to 2%. Paucity of data regarding this disease process negatively impacts the determination of genetic and environmental influences. Underlying pathophysiology for this disease is not well understood. Modified C-reactive protein may be implicated as this protein is found in both renal tubular cells and the uvea. In a recent study, mCRP was found to be higher in patients with TINU syndrome and late-onset uveitis when compared to drug-induced interstitial nephritis1. Close follow-up with ophthalmology and nephrology is essential in these patients as uveitis often recurs after corticosteroid withdrawal; chronic kidney damage can ensue if there is not sufficient monitoring2.
Abstract: Herpes simplex virus-1 (HSV-1) can reactivate in the central nervous system (CNS), leading to acute encephalitis. In this care report, we discuss an 83-year-old female who was found unresponsive at home. She had been undergoing therapy with a T-cell costimulatory molecule blocker, abatacept, for rheumatoid arthritis (RA) in the months leading up to her presentation. Initial MRI brain without contrast showed subtle restricted diffusion in left medial occipital and restricted diffusion of left insula and left greater than right medial temporal lobes. Cerebrospinal fluid (CSF) studies on day of admission confirmed a diagnosis of HSV-1 encephalitis (HSE). Acyclovir was started empirically immediately after lumbar puncture. Subsequent MRI brain on day 7 of hospitalization revealed worsening swelling of the mesial temporal lobes and extension into the insula and lentiform nuclei bilaterally with areas of developing hemorrhagic necrosis. This is consistent with a diagnosis and natural course of HSE. Healthcare providers should have an awareness of acute encephalitis related to reactivation of human herpes viruses while being treated with abatacept and patients receiving abatacept should be adequately educated.
DIAGNOSTIC CHALLENGES DURING A PANDEMIC
Chiamaka Ogemdi Isiguzo, DO; Henok Hardilo, MD; Julia Usatinsky, MD
Aurora Health Care Internal Medicine Residency Program, Milwaukee, WI

Introduction: SARS-CoV-2 has been implicated as the culprit in multi-organ dysfunction. Its ability to affect different organ systems has led to it being at the top or only differential for new patient presentations who simultaneously have been diagnosed with COVID-19 infection and/or have suffered recent COVID-19 illness. This narrowing of differentials can contribute to diagnostic errors during a pandemic.

Case Presentation: We report a case of ANCA-Vasculitis in a 52-year-old female with a recent diagnosis of COVID after presenting with myalgias, fatigue, headaches and fevers. After several weeks of unresolving symptoms, she presented to her PCP and was found to have wheezing on examination and infiltrates on the chest x-ray and treated with steroids and antibiotics. Routine labs were ordered which demonstrated creatinine of 1.58 and hemoglobin of 10.3 (no baseline was available). Despite treatment, patient’s symptoms persisted which prompted an ED visit. Her symptoms were attributed to “long COVID” and she was sent home without further work-up. She presented back to the ED 5 days later with continued symptoms and was once again discharged without further work-up. Patient continued to have persistent myalgias, fevers and dyspnea, and her creatinine continued to worsen, which prompted her PCP to directly admit her to the hospital for further work-up. She was ultimately found to have ANCA-vasculitis and started on steroid and rituximab regimen with improvement in her symptoms and stabilization of kidney function.

Discussion: Work-up of this patient was complicated by cognitive bias as her complaints were attributed to the COVID syndrome. This report aims to address the pervasiveness of anchoring bias present during a pandemic, and to alert clinicians to the importance of broader differential diagnosis, to minimize diagnostic errors.
WHEN BROAD SPECTRUM ANTIBIOTICS FAIL: COINFECTION WITH LYME’S DISEASE AND HUMAN GRANULOCYTIC ANAPLASMOSIS MASQUERADING AS SEPTIC SHOCK

Nicole Krolak, DO; Somto Nwaedozie, MD; Prathima Gopinath, MD

Department of 1Med-Peds, 2Internal Medicine Marshfield Medical Center, Marshfield, WI

Introduction: Midwestern United States is endemic with Lyme disease, the most common tickborne infection in North America transmitted by *Ixodes scapularis* tick which can also transmit *Anaplasma phagocytophilum*, the pathogenic cause of human granulocytic anaplasmosis [HGA]. Patients can present with a coinfection in about 5% of cases which can alter the clinical presentation, course and severity.

Case presentation: A 75 year old male presented to the ER with a history of fever, cough, and shortness of breath. On examination, he was febrile, tachycardic, tachypneic with oxygen saturation of 80% on room air. He was confused, generally weak, and had diminished breath sounds bilaterally. He had a normal WBC, lymphopenia, moderate thrombocytopenia, elevated lactate and inflammatory markers. His chest xray showed bilateral perihilar and bibasilar opacities. He was treated empirically for community acquired pneumonia with ceftriaxone and azithromycin but continued to have worsening hypoxia over the following days until he required intubation. Antibiotics were broadened to Piperacillin-Tazobactam and Vancomycin but he continued to have worsening hypoxia requiring intubation and hypotension requiring multiple vasopressors. Follow-up labs showed significant anemia, and DIC and the he required cryoprecipitate, platelets and pRBC transfusions. He also required CRRT for severe AKI. Further work-up with for tickborne illnesses was positive for Lyme IgM and IgG bands, and Anaplasma by PCR. Doxycycline was initiated with discontinuation of oral antibiotics. Within 48 hours of initiation of doxycycline, his mental status improved, he was extubated, his kidney function recovered, and DIC resolved. With improvement in his clinical status, he was discharged after 18 days of hospitalization.

Discussion: This case highlights the necessity of having a high index of suspicion for tickborne disease in the appropriate epidemiological context as an etiology for septic shock, especially when conventional broad-spectrum empiric antibiotics fail to achieve the anticipated clinical response.
A 50 year old male with recently diagnosed seronegative rheumatoid arthritis on sulfasalazine presented with 1 month history of intermittent fevers, generalized malaise, headaches, cough, and diarrhea. Exam revealed erythema of the malar cheeks, with subsequent development of erythroderma and partially blanching macules of his extremities by HD2. CT C/A/P and PET scans were notable for new splenomegaly and diffuse lymphadenopathy. Labs showed new transaminitis, hyperbilirubinemia, ferritin>6000, LDH>1300, sIL2R>2400, and bicytopenia. Multiple biopsies of his skin, bone, and lymph nodes were taken, with findings suggestive of a reactive process, and dermatopathology consistent with DRESS. He met 4 out of the 8 diagnostic criteria for HLH and his RegiSCAR score of 5 made diagnosis of DRESS probable. Therefore his constellation of clinical features were suggestive of DRESS/HLH overlap syndrome, he was started on IV dexamethasone and transitioned to prednisone on discharge. At 2 week follow up, he had continued improvement in his rash with complete resolution of his constitutional symptoms.

DRESS is a T-cell mediated hypersensitivity reaction while HLH is characterized by hyperactivation of hemophagocytic macrophages. Given that both processes involve immune dysregulation and frequently present with constitutional symptoms, rash, and multiorgan involvement, it could be suggestive of HLH and DRESS lying on a spectrum of immunological hypersensitivity. Both are managed with similar therapeutic interventions, meaning that cases of DRESS and HLH as an overlapping syndrome could be underestimated by individual cases resolving before the other can be co-diagnosed. While the reported number of overlap syndrome cases is rare, clinicians should be prompted to further evaluate for a co-diagnosis, if each individual syndrome fails to improve on standard therapies.
ON PINS AND NEEDLES: PARESTHESIAS, FATIGUE, AND WEIGHT LOSS IN A 29 YEAR-OLD

Kirsten Lipps, MD; Kevin McKown, MD
University of Wisconsin Hospital and Clinics, Madison, WI

Introduction: Sarcoidosis is a systemic disease, which presents with neurologic manifestations in 5 to 10 percent of affected individuals. Both central and peripheral nervous systems may be involved, resulting in a wide range of neurologic deficits. Due to low disease prevalence and non-specific symptoms, the diagnosis of neurosarcoidosis may be challenging.

Case: A 29 year-old man presented with progressive, ascending paresthesias for 3 weeks with associated fatigue and weight loss. The patient denied other neurologic symptoms and lacked objective deficits on physical examination. Initial laboratory evaluation was notable for elevated inflammatory markers. MRI spine demonstrated an expansile abnormality in the spinal cord intramedullary space from C4 to T2. MRI head revealed multiple non-specific abnormalities involving the subarachnoid space, lateral ventricles, orbital cavities, cranial nerves, and cerebellum. Cerebrospinal fluid analysis was notable for mononuclear cell pleocytosis and elevated protein. MRI spine scout images revealed bilateral hilar lymphadenopathy. CT chest confirmed this abnormality, in addition to demonstrating perilymphatic nodularity. Further diagnostic evaluation revealed no evidence of infection, hematologic malignancy, or demyelinating disorder. Rheumatologic studies were also unremarkable. The patient’s constellation of symptoms and imaging abnormalities prompted a clinical diagnosis of sarcoidosis with pulmonary and neurologic involvement. The patient was initiated on high-dose glucocorticoids with plan to initiate a steroid-sparing agent at a later date.

Discussion: The differential diagnosis for new-onset neurologic symptoms is broad. Neurosarcoidosis should be considered in patients presenting with non-specific and/or multiple neurologic concerns. Though tissue biopsy remains the gold standard for diagnosis, clinical features may be sufficient to initiate treatment, particularly in those with systemic disease and/or characteristic imaging abnormalities. Glucocorticoids are first line for treatment of neurologic sarcoidosis, with steroid-sparing agents used as an adjunct to mitigate adverse effects associated with chronic steroid therapy while preventing relapse. With treatment, prognosis of neurologic sarcoidosis is favorable, with greater than one-half of patients achieving complete remission.
Introduction: Diffuse cortical necrosis is very rare in developed countries and responsible for only 1-2% of Acute Kidney Injury (AKI) cases and it is usually irreversible. Atypical hemolytic uremic syndrome (aHUS) causing renal cortical necrosis is extremely rare. Most cases of aHUS are caused by genetic mutations.

Case Presentation: 69-year-old female with a PMHx of breast cancer s/p left mastectomy admitted for cough, watery diarrhea, fatigue and diffuse maculopapular rash of 3 days duration. She reported having cough and chest congestion for 2 weeks prior to presentation and was recently treated with prednisone but denied recent NSAIDS use. Admission WBC 35K, creatinine 0.77(GFR >90), U/A with hematuria, proteinuria, and pyuria. On second day of hospitalization, she developed AKI. Despite adequate hydration, patient became anuric and her GFR continued to rapidly worsen with creatinine 4.55(GFR 11) and 7.72(GFR 5) on third and sixth day of hospitalization respectively. Labs were notable for negative ANA with Reflex and ANCA(MPO/PR3). Further labs revealed elevated LDH(2173), low C3(20) and C4(2.5); however, haptoglobin, platelet, and hemoglobin were normal and no schistocytes on peripheral smear. Direct Coombs test was negative; reticulocyte count and ADAMTS 13 activity were normal. Atypical HUS genetic test was positive. Oral steroid was started and then HD initiated given no recovery in renal function. Kidney biopsy showed extensive cortical coagulative necrosis with vascular fibrin deposit consistent with diffuse cortical necrosis. Patient was discharged with outpatient HD(MWF).

Discussion: This case demonstrates aHUS without hemolytic anemia or thrombocytopenia causing irreversible renal cortical necrosis. She has genetic predisposition to aHUS precipitated by severe sepsis causing diffuse renal cortical necrosis.
**ISCHEMIC COLITIS IN A PATIENT WITH A PACEMAKER**

*Jon Schrope, BS¹; Jeremy Smith, MD²*

¹University of Wisconsin School of Medicine and Public Health, Madison WI.
²University of Wisconsin Department of Medicine, Madison WI

**Background:** Non-occlusive mesenteric ischemia commonly occurs from transient hypoperfusion of watershed regions of the colon, classically manifesting as crampy post-prandial pain with diarrhea and hematochezia. The most typical precipitating factor is shock. Here, we report a case of severe ischemic colitis not visible by CT on admission in a patient with a pacemaker experiencing chronic hypotension likely related to cardiac dysynchrony due to pacing.

**Case:** A 75 year old male with a history of non-ischemic cardiomyopathy with LVEF of 35%, paroxysmal atrial fibrillation, and nearly incessant ventricular tachycardia status post two ablation procedures presented to the ED with a nine month history of severe post-prandial abdominal pain with associated constipation, nausea, vomiting and a sixty-pound weight loss. His blood pressure on admission was 105/67, and his systolic blood pressures over the next 5 days ranged between the mid-70s and high-80s. Abdominal exam yielded tenderness to light palpation of the peri-umbilical region, epigastrium and right upper quadrant with guarding but without rebound tenderness. Hemoglobin was 10.5 and he had a mild leukocytosis of 12.3. Abdominal CTA was unrevealing. EGD and colonoscopy showed evidence of diffuse inflammation, with colon biopsy consistent with ischemic injury. MRA showed no evidence of mesenteric atherosclerosis or vasculitis. It was determined that the pain began after his most recent VT ablation, which resulted in pacemaker dependence, and he was diagnosed with GI ischemia attributable to chronic severe hypotension secondary to iatrogenic dysynchrony from the pacemaker. His symptoms improved on initiation of midodrine.

**Discussion:** Iatrogenic dyssynchrony leading to decreased cardiac performance is a known phenomenon, however it very rarely presents as bowel hypoperfusion and with such severe symptomatology. While rare, it is important that physicians are aware of this possible complication of cardiac pacing devices.
Cerebrotendinous Xanthomatosis (CTX) is a rare, autosomal recessive disease which results from a defect in bile acid synthesis causing accumulation of cholesterol and cholestanol in nearly all organ systems. The principle enzyme deficiency is sterol 27-hydroxylase which leads to formation of xanthomas, nodules, and plaques in the central nervous system, eyes, tendons, skin, lung, and bones. CTX occurs in less than 5 per 100,000 people. Patients may present with intractable diarrhea, premature cataracts, and progressive neurologic dysfunction including intellectual decline, and spasticity starting in late childhood. We present a unique case presenting with chronic diarrhea in an adult patient.

A 54 year old female presented to the gastroenterology clinic with a several year history of diarrhea and ataxia. Family history included a degenerative familial ataxia in immediate relatives. Over the last ten years she had worsening ataxia requiring wheelchair due to falls and spasticity. She also had poor oral intake, and difficulty with activities of daily living. Exam was unremarkable except for difficulty with speech. Infectious workup for diarrhea, celiac testing, and colonoscopy were unremarkable. When her brother was diagnosed with CTX disease, she underwent genetics evaluation, measurement of cholestenol levels which returned as total bile acids greater than 15 µmol (normal < 10), 8(9)-cholestenol 54.53 µg/mL (0-0.3), and cholestanol 24.71 µg/mL (0.86 – 3.71) consistent with Cerebrotendinous Xanthomatosis. She was treated with chenodeoxycholic acid 250 mg three times daily following which she started noticing improvement in her appetite, started gaining weight with improvement in diarrhea and cognitive function.

Chronic diarrhea in CTX often presents early in the clinical course in older childhood. The underlying pathophysiology in CTX is not completely understood but likely related to excess bile alcohols in the gastrointestinal tract causing diarrhea and deficiency of bile acids causing steatorrhea. Ideally treatment is started in childhood to prevent neurologic complications which are not completely reversible in adulthood with treatment. Chenodeoxycholic acid is believed to replace the bile alcohols produced in CTX and help with diarrhea, but this mechanism is not entirely known.
Research Based Vignettes
Background: Diversifying the medical workforce is increasingly recognized as essential to addressing health disparities. Pursuant to this goal, the Department of Medicine at the University of Wisconsin (UW) established the Diversity, Equity, and Inclusion Task Force in the spring of 2020. Particular attention was given to the development and implementation of strategies for diversifying the internal medicine residency training program without using demographic quotas. These strategies were implemented during the recruitment of the UW 2021 internal medicine intern class, and outcomes were retrospectively analyzed following the match.

Methods: The pre-intervention group consisted of all active UW categorical internal medicine residents during the 2021 recruitment season (3 resident classes spanning 2018-2020). The post-intervention group consisted of the newly matched UW 2021 categorical internal medicine intern class. The demographics of these groups were categorized according to the Association of American Medical Colleges (AAMC) race and ethnicity classifications. The proportion of historically underrepresented minority (URM) residents was compared between groups using the chi-squared test.

Results: The pre-intervention group was characterized by 83.5% (71/85) residents who self-identified as “White” and only 2.4% (2/85) residents who self-identified as URM. These were both far above and below the respective national averages of 50.1% and 21.6% published by the AAMC for all active MD internal medicine residents in 2019. The post-intervention group was characterized by 58.6% (17/29) residents who self-identified as “White” and 37.9% (11/29) who self-identified as URM. The proportion of URM residents was significantly increased in the post-intervention group compared to the pre-intervention group (p < 0.05).

Conclusion: Significant progress can be made in the diversification of residency training programs in a single application cycle through strategic interventions in conjunction with departmental and institutional support. The use of demographic quotas is neither recommended nor necessary.
**Introduction:** Increasing relative dose intensity (RDI) in obese women with breast cancer is important because obese patients have both high risk for low RDIs and worse outcomes than normal weight patients. Utilizing dose-dense chemotherapy has previously been found to improve outcomes for breast cancer patients. This study sought to understand the interaction between BMI and dose-dense regimens and its possible effects on RDI.

**Methods:** We examined a cohort of patients treated at a multispecialty academic cancer center for whom chemotherapy was guideline-recommended during the study period: triple negative or HER2-positive subtypes, and either positive lymph node metastases or tumor size > 1.0 cm.

We obtained administration dates and doses for doxorubicin, cyclophosphamide, paclitaxel, docetaxel, and carboplatin. Other variables included BMI categories (non-overweight, overweight, obese), race, HER2 status, stage, and Elixhauser co-morbidity score. Logistic regression models were performed to examine associations of each variable with our primary outcome (RDI<85%) and a combined chemotherapy adherence outcome of whether she had either a low RDI, delay, or discontinuation. Separate models were run analyzing the interaction between dose-dense and BMI.

**Results:** We identified 244 eligible patients, of whom 117 (48%) received a dose-dense regimen. Mean BMI (SD) was 29.9 (7.6) and 43.6% of patients in the dose-dense group were obese. The odds of having a low RDI is 2.3 times higher if the patient was obese (CI:1.051-5.097; P=0.0371). However obese patients receiving dose-dense chemotherapy had a 91% decreased risk of having a low RDI (CI: 0.031-0.257).

**Conclusions:** This research provides significant support in use of dose-dense regimens amongst obese patients for maintenance of a high relative dose intensity.
PREDICTORS OF PERMANENT PACEMAKER PLACEMENT FOLLOWING TRANS-CATHETER AORTIC VALVE REPLACEMENT. THE MARSHFIELD CLINIC HEALTH SYSTEM (MCHS) EXPERIENCE

Somto Nwaedozie\textsuperscript{1}, Javad Najjar Mojarrab\textsuperscript{1}, Romel J. Garcia-Montilla\textsuperscript{2}
Department of \textsuperscript{1}Internal Medicine, \textsuperscript{2}Surgical Intensive Care Unit, Marshfield Medical Center, Marshfield, WI

\textbf{Background:} Conduction or rhythm abnormalities are some of the short-term complications following Trans-catheter aortic valve replacement (TAVR) with about one quarter of cases requiring permanent pacemaker implantation (PPM). Factors like age, conduction abnormalities, self-expandable valve type are recognized as predictors of PPM but others like QRS duration, prolonged QTc and supraventricular arrhythmias have been poorly studied.

\textbf{Objective:} To determine the predictors of PPM requirement following TAVR in MCHS.

\textbf{Method:} Retrospective study that identified patients with TAVR between January 1, 2012 to December 31, 2019 at the MCHS. The group was dichotomized into 2 cohorts- those with post TAVR PPM and without PPM, both were followed for one year. Pre- and post-procedural data were abstracted.

\textbf{Results:} Out of the 357 patient that met inclusion criteria, 57 [16\%] had a PPM within the 1 year follow-up. Baseline demographics, valve type and cardiovascular risk factors were similar except for type II DM which was more prevalent in the PPM cohort [59.6\% versus 40.7\%; \( P= 0.009 \)]. The PPM cohort had a significantly rate of pre-procedure right bundle branch block, prolonged QRS and QTc interval, and a marginally higher rate of supraventricular arrhythmias. Unadjusted odds ratio for PPM was significant for prior conduction defects [2.88; \( P<0.001 \)], prolonged QRS [3.44; \( P<0.001 \)] and prolonged QTc [2.94; \( P<0.001 \)]. After model adjustment for baseline demographics and cardiovascular risk factors, the odds ratio was significant for supraventricular arrhythmia [2.12, \( P = 0.047 \)] and prolonged QRS [3.22, \( P = 0.035 \)] but marginally significant for prolonged QTc [1.99, \( P= 0.074 \)].

\textbf{Conclusion:} Presence of supraventricular arrhythmia, prolonged QRS and prolonged QTc may be predictive of post TAVR PPM requirement.
Background: The COVID-19 pandemic has disproportionately impacted minority communities, evidenced by higher rates of infection, hospitalization, and mortality. Worse COVID-19 outcomes in minorities communities are inextricably linked to the pervasive effect of structural racism. Structural racism is embedded in the fabric of our systems of housing, education, employment, earnings, benefits, credit, criminal justice, and health care, ultimately manifesting in the creation and persistence of health inequities. Since the beginning of the U.S. vaccine rollout, we have seen a lack of equitable access to the vaccine, especially for people of color. Equitable COVID-19 vaccine access is essential to ending the COVID-19 pandemic and protecting the health of Black and other socially vulnerable populations. However, inequitable access to digital health information and patient portals place these populations at a disadvantage when vaccine access is driven by electronic outreach, perpetuating health inequities.

Setting/Program Description: An urban academic General Internal Medicine (GIM) practice that serves a high proportion of Black and socially vulnerable patients. Patients 65 and older in this clinic without patient portal access are more likely to be Black, rely on public insurance, or live in zip codes with high social vulnerability based on the social vulnerability index. The health system deployed a method of patient outreach largely driven by patient portal notification and scheduling while the GIM practice mobilized community health workers and students to engage in telephonic outreach to patients aged 65 and older without patient portal access.

Program Evaluation: Our team provided outreach to 1007 GIM clinic patients from February 11 to March 9, 2021. During that time, 865 GIM patients completed their first dose of the COVID-19 vaccine at a Froedtert & MCW vaccination clinic; one-third of these had been contacted through the outreach program. A pre-post analysis showed the following changes in the demographics of patients vaccinated at our clinic: Hispanic from 2% to 4%, non-Hispanic White from 88% to 50%, non-Hispanic Black from 7% to 42%, other 3% for both, EMR inactive from 3% to 37%, and high disparity zip code from 14% to 43%.

Discussion: In Wisconsin, Black populations have received 3% of total vaccinations, but are responsible for 7% of cases, 6% of deaths, and comprise 6% of total the population. Milwaukee County has a higher percentage of Black (27.2%) people. Black people have only received 14% of total vaccine doses, and residents in high social vulnerability zip codes have received a lower percentage of the vaccine as of May 10th, 2021. Our outreach intervention has improved access to COVID-19 vaccines for individuals aged 65 and older without patient portal access and, as a result, improved equity in vaccine access for black and socially vulnerable populations; it has also provided a unique and valuable experience for medical students.
Displayed Posters
1) RARE CASE OF INSULIN-DERIVED AMYLOIDOSIS THAT APPEARED AS ECTOPIC BREAST TISSUE
Abdul-Rahman K. Abdel-Reheem; Malek E. Ayoub; Evelyn Chan, MD
Medical College of Wisconsin, Milwaukee, WI

Insulin-derived amyloidosis (AIins) is a rare localized dermatological condition associated with insulin injections. A 53-year-old woman with a history of T1DM, Graves’ disease, and a thyroid nodule came to the clinic to establish care. The patient complained of painless, palpable bilateral abdominal masses that the patient said have been there for a long time. An abdominal CT scan with contrast was performed and reported small para midline foci of soft tissue attenuation with associated calcifications that were highly suggestive of breast tissue. The patient then underwent an ultrasound to further evaluate the masses. The ultrasound found that, although these are likely related to the injections performed in these areas, other etiologies such as ectopic breast tissue or malignancy cannot be excluded. Therefore, the patient underwent an ultrasound-guided biopsy where pathology confirmed the diagnosis of amyloidosis. Biopsy samples were sent to Mayo clinic for mass spectrometry and results indicated amyloidosis: Alns type. Further evaluation was done including UPEP, SPEP, and multiple myeloma screening to investigate if systemic disease was present. These studies were unremarkable, and the patient elected to have the amyloidomas removed. Patient was also instructed to rotate injection sites for insulin and avoid injecting in the amyloidomas.

Less than 100 cases of AIins have been reported in literature. Given the widespread use of insulin, it is likely that insulin-derived amyloidosis is largely underreported. It is important for clinicians to consider it as a possible differential, especially in patients on insulin that present with masses at their injection sites. Although it is a localized disease, it is also important to rule out other serious conditions such as multiple myeloma, systemic amyloidosis, and other malignancy such as in this patient. Insulin-derived amyloidosis can be difficult to recognize in most diabetic patients due to several overlapping signs and symptoms with systemic amyloidosis and diabetic complications such as peripheral neuropathy, diabetic nephropathy, and hypertrophic cardiomyopathy. Since Alns type amyloidosis normally occurs at the site of insulin injection, it is recommended to educate patients on routinely changing the site of injection to avoid the formation of amyloid fibrils. Treatment of insulin-derived amyloidosis includes avoidance of injection at the sites of amyloidosis, local treatment, and surgical excision of amyloidomas. Insulin-derived amyloidosis is a rare condition usually observed at the sites of injection. Although it is only a localized process, it is important to evaluate to rule out other conditions such as malignancy, systemic disease, and ectopic tissues. A biopsy with mass-spectrometry is usually diagnostic. These cases should be continued to be reported to better characterize this disease.
2) INTERESTING PRESENTATION OF RHABDOMYOLYSIS

Emily Adochio; Truman Landowski; Gregory Capelli
Medical College of Wisconsin, Milwaukee, WI

Introduction: In patients presenting with elevated creatinine kinase (CK) the differential diagnosis can be broad. Elevated CK can also be found in many conditions such as inflammatory myositis, infectious myositis, and can be caused by certain drugs. Screening for all these diseases is important in patients where the cause of the elevated CK is unclear – especially if there is concern for dermatomyositis or polymyositis as these diseases can be associated with underlying malignancy [2]. Our case highlights the unique work up for screening patients with elevated CK.

Case: The patient is a 77-year-old male with a past medical history significant for atrial fibrillation on warfarin and hyperlipidemia on a statin who presented for a month of weakness. The patient had a viral illness approximately 3 months prior to admission. He noted his shoulder girdle was weak. His labs on presentation revealed elevated CK ~18,000, LDH ~1000, K 5.3, Cr 6.68, Alk phos 176, AST ~700, ALT 337, INR 8.8, CRP 7.6, ESR 50. Urine on admission appeared orange/dark red. Given the laboratory findings and clinical history there was concern for rhabdomyolysis. The patient was started on aggressive IV hydration, however his CK remained elevated. The persistently elevated CK despite fluid resuscitation and elevated inflammatory markers led our team to pursue further rheumatology/inflammatory myositis work up. In collaboration with Rheumatology the patient’s work up included: an autoimmune myositis pane, a MRI of his leg, and a muscle biopsy. His autoimmune/inflammatory panel was negative (including: ANA, dsNDA, anti-smith, RNP, SSA/SSB, anti-Jo-1, mitochondrial antibody, HMG-CoA reductase, and myositis panel). The MRI of his left leg was concerning for a myositis. However, a muscle biopsy of his left quadricep was consistent with rhabdomyolysis. The patient’s statin was discontinued. He continued to be treated with IV fluids and by discharge his labs had mostly normalized aside from a CK of 440. His condition was thought to be rhabdomyolysis due to viral illness and less likely to be due to an underlying autoimmune disease.

Discussion: This case demonstrates the unique work up that is needed in patients where there is concern for an underlying rheumatologic/ inflammatory myositis. Specifically, patients who are suspected to have myositis should be screened for dermatomyositis and polymyositis as these are associated with underlying malignancy. About 80% of patients with dermatomyositis and polymyositis have elevated ANA however, myositis-specific autoantibodies are only present in 20-40% of patients [3]. Therefore, a consultation to a Rheumatologist is often needed.
Introduction: Hemolytic anemia is due to premature destruction of red blood cells. One common etiology is autoimmune hemolytic anemia (AIHA), triggered by IgG or IgM antibodies. A Direct Antiglobulin Test (DAT) is used to detect these autoantibodies. Here, we present an intriguing case of DAT negative autoimmune hemolytic anemia.

Case: A 50-year-old Mexican male with a history of hypertension and diabetes presented with 3 weeks of dyspnea on exertion, fatigue, and fever. He was mildly tachycardic, had conjunctival pallor, and a grade 2 systolic murmur. Labs were notable for: hemoglobin-7, total bilirubin-2.1/direct-0.5, haptoglobin<15, LDH-600, normal vitamin B12, folate and iron studies. ECG and CXR were unremarkable. Peripheral blood smear was without schistocytes. DAT was negative. He was empirically started on prednisone 70mg daily for a DAT negative AIHA. A “super Coombs” test revealed an IgA autoantibody mediated AIHA. His outpatient course was complicated by a decreased hemoglobin despite prednisone; thus, Rituximab was given. He received appropriate vaccinations in anticipation of possible splenectomy. The prednisone was ultimately tapered, and hemoglobin remained stable three months later, without undergoing a splenectomy.

Discussion: A small fraction of AIHA has been linked to IgA autoantibodies which are not routinely detected unless a wider panel is tested for using the “super Coombs” test. IgG AIHA, the most common etiology, is insidious whereas our patient showcased a more acute and severe presentation. Other cases in literature had a similar progression, were steroid refractory, transitioned to cytotoxic or monoclonal antibody medications and eventually led to a splenectomy. Thus, emphasizing the need to include anti-IgA in the standard DAT test to shorten the time to diagnosis, reduce cost and number of tests. Additionally, a modified treatment protocol may be required to identify steroid refractory cases early. Further research is needed to properly characterize this rare cause of AIHA.
Gallard’s “military aneurysm of the stomach” first described in 1884, now known as Dieulafoy’s lesion is a rare vascular abnormality of the GI tract mucosa that has been implicated in severe cases of gastrointestinal bleed and as in our case, hemorrhagic shock. Given its fatal consequences, it is imperative that Dieulafoy’s lesions be included in the differential diagnosis for any GI bleed. Our patient is a 35 year old alcoholic male with known grade 1 varices who was admitted to the ICU 3 times in the last two months with acute blood loss anemia. During all three admissions he presented with hematemesis, melena and syncope. Prior to these two months, he had a hemoglobin of 14mg/dl and on presentations it was at 7.5 mg/dl which barely moved up beyond 9mg/dl during these two months. On all three occasions his initial MAPs were below 65 mmHg which improved with blood product and crystalloid resuscitation. During the 1st hospitalization, esophagogastroduodenoscopy showed erosive esophagitis and portal hypertensive gastropathy without evidence of recent or active bleeding. 2nd EGD done a month after the 1st one showed resolution of erosive esophagitis and 1 column of subtle esophageal varices completely flattened with air insufflation. EGD 10 days later revealed completely flattened 1 column of esophageal varices previously seen but also the culprit for his acute blood loss anemia on two occasions and hemorrhagic shock this time: an actively spurting Dieulafoy lesion in the cardia of the stomach. This lesion was treated with 2 cook clips. Patient had good post procedure recovery with improved hemoglobin and his shock resolved. The idea behind this case report is to highlight the importance of broadening differentials in a patient with known alcoholism, alcoholic liver disease and coagulopathy. In this case one tends to believe that patient has upper gastrointestinal bleed from known varices, or esophageal mucosal tears from vomiting/retching or from known portal hypertensive gastropathy in the setting of coagulopathy. Dieulafoy’s lesions are rare, probably because they are underdiagnosed and as evident in our case their diagnosis continues to be a dilemma because our patient was probably bleeding from that one lesion all along which was never really discovered up until his 3rd EGD.
5) CARDIAC TAMponade AND INFECTED PLEURAL EFFUSION IN ACUTE PANCREATITIS

Maleeha Ajmal, MD; Muhammad Gilani, MD; Gul Nawaz, MD
Marshfield Clinic Health System, Marshfield, WI

We present a case of 60 year old man with gallstone acute pancreatitis with pancreatic necrosis complicated by cardiac tamponade which was initially noninfectious. Two weeks later, the pericardial fluid was positive for polymicrobial infection suggesting spread of infection from gastrointestinal source. Literature suggests that pleural effusions, ascites and pericardial effusions occur commonly in acute pancreatitis in decreasing order of frequency but cardiac tamponade is fairly rare. Moreover, no case report was found to suggest direct microbial invasion of the pericardial space by gastrointestinal flora. In our patient, acute pancreatitis was managed with IV hydration, early enteral feeding and pain control with expected improvement in symptoms. Infected pericardial effusion with cardiac tamponade was treated with pericardial window, fluid drainage and long term antibiotics and antifungals with a favorable outcome. Our case highlights the importance of further investigation when pericardial effusion persists despite improvement in acute pancreatitis.
Acute pancreatitis can result in fat necrosis, typically occurring in the peri-pancreatic region within weeks to months and it generally appears as a low attenuation collection, with minimal heterogeneity. There are no specific imaging features that can diagnose retroperitoneal fat necrosis which may imitate other entities including certain malignancies, which may lead to invasive studies for diagnosis. Herein, we present a case of extensive retroperitoneal fat necrosis beyond the peri-pancreatic region that developed 10 days after an episode of acute pancreatitis.
Background: Angelman syndrome (AS) is a disorder of genetic imprinting that affects 1 in 12,000-20,000 people. It is characterized by severe intellectual disability, inappropriate laughter, ataxia, and seizures. AS may manifest behaviorally with hyperactivity, aggression, and anxiety. Aggression worsens with age. We present a case of hypersexuality successfully treated with testosterone suppression.

Case Presentation: A 16-year-old male with AS presented with new-onset of hypersexual stimulation, frustration, and aggressive behavior, including grabbing and hitting his penis repeatedly. After extensive evaluation, it was postulated that this could be due to his rise in testosterone during puberty. His serum testosterone at this time was 550 ng/dL, so it was elected to treat him with a 3 month depot leuprolide acetate (GnRH agonist) injection. He initially became more symptomatic (presumably from the testosterone flare), but then was noted that he no longer grabbed, pinched, or scratched, and he focused better. A repeat testosterone was at castrate levels. This treatment was continued for 9 years, at which point, insurance would no longer cover this treatment and bilateral orchiectomy was considered. The Ethics Committee was consulted due to the controversial nature of orchiectomy in a patient unable to consent, and they deemed it reasonable to pursue an orchiectomy, given the patient’s total dependence on his parents for activities of daily living with no future fertility concerns. A bilateral orchiectomy was successfully performed with no adverse events.

Discussion: AS has been associated with aggression that worsens with age. Here we present a case of post-pubertal aggression and hypersexuality in an AS patient responsive to medical castration. His symptoms were found to mirror his testosterone levels. Leuprolide may be a good initial treatment for this rare manifestation of AS. Orchiectomy may be considered as definitive treatment in some AS patients with hypersexuality and aggression.
Objective: Previous studies have found higher rates of heart disease and worse mental health outcomes among individuals residing in rural areas. To our knowledge, no research has used county-level data to measure the effect of “ruralness” (the degree to which a county is rural) on heart disease and mentally unhealthy days while controlling for other sociodemographic factors. This study analyzes the effect of ruralness on heart disease death rates and the average number of mentally unhealthy days on a county-level.

Study Design: Linear regressions were performed using county-level data to analyze the effect of “Ruralness” on heart disease death rates and mental unhealthiness while controlling for confounding variables. Geographic analysis was also used.

Results: Higher rural-urban continuum codes predict lower rates of cardiac mortality (β=-.075 deaths per 100,000 people/continuum code, t=-4.36, p<.001) and fewer mentally unhealthy days (β=-.265 monthly mentally unhealthy days/continuum code, t=-16.45, p<.001).

Conclusion: Being from a rural area correlates with lower rates of heart disease death and mental unhealthiness after controlling for sociodemographic confounders. This adds nuance to the previously reported trend of heart disease being more prevalent in rural areas.
Presented is a case of a 66 year old female with medical history of allergic rhinitis who presented with hypoxic respiratory failure, acute renal failure, subacute sinus pain, and epistaxis. The patient reported to have been taking 2.4g of IBU daily for three weeks. Initial concerns were for renal failure requiring dialysis and a possible pneumonia. Notable on exam was significant nasal crust- ing. Laboratory studies revealed a creatinine of 10, a normocytic anemia with a hemoglobin of 7.6, all other labs on admission were within normal limits. CXR on admission showed bilateral pulmonary infiltrates. Peculiar was the present- ing sinus pain in the past one month and three day history of epistaxis. These symptoms along with renal failure and pulmonary infiltrate raised concern for a vasculitis process. ESR and CRP were tested and returned highly elevated. A biopsy of the nasal mucosa was obtained and revealed small- and medium- caliber vasculitis with vague granulomatous inflammation. The diagnosis of Granulomatosis with Polyangiitis was made, the patient was started on high dose steroid treatment and Rituximab for induction therapy. Plasma exchange was not performed due to recent published data from the PEXIVAS trail where ESKD and death were not reduced with the use of plasma exchange.
10) PRIMARY MALIGNANT MELANOMA OF THE GASTROESOPHAGEAL JUNCTION TREATED WITH IMMUNOTHERAPY: A CASE REPORT

Nabil Attlassy; Abiye Agbeh; Rohan Patnaik
Medical College of Wisconsin, Milwaukee, WI

Introduction: Primary Malignant Esophageal Melanoma (PMME) constitutes 0.1% -0.5% of all primary malignant esophageal neoplasms. Melanocytes are present within the squamous epithelium of the esophagus in the stratum basale layer with melanocytosis rare within the esophagus. PMME is aggressive and has a poor survival rate and 80% of patients have metastatic disease at diagnosis. Resection surgery is usually first-line treatment for localized PMME but recurrence rates remain high. Tumor-specific immunotherapy has shown promising results. We report a case of PMME with metastasis to the liver treated with immunotherapy.

Case Presentation: A 66-year-old female with 2 months of progressive dysphagia and 3 episodes of hematemesis the previous night. Endoscopic examination showed a hypervascular distal esophageal mass. Biopsy was positive for S-100, SOX-10, HMB-45 and showed rare mitotic figures with scattered pigment, consistent with melanoma. The patient was scheduled for esophagectomy initially, but instead pursued immunotherapy after liver metastasis was diagnosed during pre-op MRI. Immunotherapy consisted of 8 cycles of pembrolizumab followed by 4 months nivolumab and ipilimumab. The patient remains in remission three years after completing immunotherapy.

Discussion/Conclusion: Our patient was diagnosed with PMME of the distal esophagus with metastasis to the liver, a presentation that typically has a poor prognosis. Despite this, remission was achieved with immunotherapy without surgical intervention. Only a small number of cases of PMME treated with immunotherapy have been reported, one showcasing tumor stabilization following several cycles of therapy with eventual metastasis, while our patient had a stable response to treatment. Further exploration of medical management with immunotherapy should be conducted as it represents an alternative treatment for patients who do not have the option of surgical management.
Blastomycosis commonly presents as pulmonary infection but may occasionally manifest systemically. The non-specific clinical presentation and radiological findings in soft tissue and osteo-articular blastomycosis may often cause delay in diagnosis and treatment, even in endemic areas.

A 56-year-old female Wisconsin resident with a complex past medical history presented with a chief complain of right ankle pain and swelling. During her first hospitalization for acute heart failure, she was found to have pulmonary nodules. Excision biopsy showed granulomatous fungal lesions and she was started on voriconazole. At the same time, she had worsening cellulitis of her right leg over the ankle joint. Treated for bacterial cellulitis, her symptoms regressed, and she was discharged. However, she returned to ER with worsening RLE symptoms. She endorsed erythema, swelling, and numbness from foot to mid-shin with no fever. Systemic physical exam was unremarkable except for erythema and edema of the anterior lower leg from the distal foot to the mid shin, tender to light palpation, and 2cm intact blister to lateral malleolus. Labs showed elevated CRP (28.3 mg/L) and ESR (119 mm/hour) without leukocytosis. CT scan of the right lower extremities showed elongated abscess (24 cm) associated with the ankle involving peroneal compartment, anterior and deep/superficial posterior compartments, and osteomyelitis of the distal fibular with tendon involvement. She was started on Cefepime and Vancomycin and continued voriconazole. Her urine Blastomyces antigen level was reported high (14.7 ng/mL) with new RLE abscess and early osteomyelitis, clinicians were concerned for disseminated Blastomycosis with osteo-articular involvement. Therapy was continued on Voriconazole, and IV Liposomal Amphotericin B initiated. Right ankle aspiration was performed, growing budding yeast. Patient remains inpatient for ongoing definitive surgical management of the osteoarticular disease.

Blastomycosis dermatitidis is a systemic pyogranulomatous infection that occurs when aerosolized hyphae are inhaled and transform into yeast form in the lungs at body temperature. Most cases are reported from endemic areas including Arkansas, Louisiana, Michigan, Minnesota, and Wisconsin. While infections are typically self-limited, chronic infections may present usually limited to the lungs. Rarely, disseminated infection involving extrapulmonary sites may be seen. Ulcerative cutaneous lesions start as papules that eventually develop into ulcers with erythema, swelling and heaped-up borders, leading to frequent misdiagnosis as bacterial cellulitis. Osteoarticular presentation is the next common, causing osteomyelitis, as seen in our patient. Guidelines recommend therapy with amphotericin B followed by itraconazole for outpatient therapy. Formal incisional biopsy with frozen section followed by multiple excision and debridement is essential for source control and to prevent further dissemination. We present this case to raise awareness amongst health care providers about the signs and symptoms of blastomycosis, allowing for early diagnosis and initiation of appropriate treatment to prevent systemic dissemination.
**Introduction:** Vaping is known to cause lung injury and the case presentation could be quite similar to COVID-19 pneumonia infection during the initial presentation. Here we present a case of EVALI who was initially thought to have COVID-19.

**Case Presentation:** A previously healthy, 20-year-old Caucasian male presented with worsening dyspnea after being diagnosed with pneumonia a week ago, and failed outpatient treatment with azithromycin. He denied sick contacts or recent travel; he endorsed vaping nicotine and THC oil once a week socially. On presentation, he was febrile to 100.8F, tachypneic to RR of 40, and required 4L of O2 on an oxygen mask. He tested negative for COVID-19 one week prior to presentation and in the ED; however, initial CXR demonstrated diffuse bilateral airspace interstitial opacity, and the inflammatory markers, such as ferritin, LDH, CRP were elevated, which was concerning for possible false-negative COVID-19 infection. He also had leukocytosis and elevated procalcitonin, which are suggestive of possible CAP, and he was started on ceftriaxone. Eosinophilia present on admission and considered ANCA vasculitis as possible differential diagnoses as well. The AHRF got worse, needed optiflow 35% O2, and 40% FiO2, and he was started on methylprednisolone. ID and Pulmonary were consulted, and he underwent chest CT, which demonstrated bilateral ground-glass opacities, and BAL and transbronchial biopsy – negative COVID, extended viral NAAT, and cytology. The remaining labs from the initial workup: sputum culture, blood cultures, COVID antibody, Flu NAAT, serum IgE, urine antigens for Legionella, Histoplasma, Blastomyces, Aspergillus, Mycoplasma NAAT, Chlamydia NAAT, Quantiferon gold test, HIV, ANA, p-ANCA, and c-ANCA all came back negative. He was diagnosed with e-cigarette/vaping-associated lung injury (EVALI) as a diagnosis of exclusion and continued on methylprednisolone. He was hospitalized for 7 days total and went home on oxygen. He was off oxygen on pulmonary follow-up appointment, and he has been continuing prednisone taper.

**Discussion:** The prevalence of vaping has significantly increased among teenagers and young adults. As of 2020, 2807 cases of EVALI have been reported to the CDC. Among these reported cases, 66% were male, and 37% of patients were 18 to 24 years old. Typical presentations for EVALI include fever, non-productive cough, dyspnea, leukocytosis which would usually indicate possible PNA. It is important for clinicians to consider EVALI by obtaining a thorough social history regarding vaping use in the last 6 weeks, especially when young patients fail to improve after initiating antibiotics therapy. The exact cause of EVALI is not known, but possible causes include vitamin E acetate as well as those that contain vape juices with THC. Due to the rapid decline in respiratory failure, it is important to start corticosteroid treatment early. Radiographic imaging typically reveals a bilateral ground-glass opacification, which has also been demonstrated in COVID-19 infection. Some specific findings of Chest CT abnormalities in COVID-19 are often bilateral, have a peripheral distribution, and involve the lower lobes. Infectious workup usually will be unrevealing, and while BAL is not necessary to confirm EVALI, it may help to rule out other infectious causes.
Introduction: Severe acute respiratory syndrome coronavirus 2 (SARS-COV-2) is the pathogen responsible for the COVID-19 pandemic which has caused over 179 million cases and 3.8 million deaths around the world by June 2021. While SARS-COV-2 typically presents with respiratory symptoms like pneumonia, pathology in many other organ systems has been well documented. Cardiac complications of severe SARS-COV-2 have been increasingly identified in the literature. Here, we present the case of acute pericarditis following aggressive SARS-COV-2 treatment in a kidney transplant recipient which was managed with colchicine and aspirin.

Case Presentation: We present the case of a 71-year-old woman with a past medical history significant for systemic lupus erythematosus, focal segmental glomerulosclerosis, status post renal transplant 15 years prior, and type-2 diabetes mellitus. She was diagnosed with SARS-COV-2 pneumonia which was managed with 1-week course of 6mg daily dexamethasone and remdesivir. She represented to the emergency department the next day with shortness of breath, altered mental status, and chest pain which was exacerbated on inspiration. Given her constellation of clinical findings, she was admitted to the hospital with presumptive pericarditis secondary to SARS-COV-2 infection. Management was achieved with 650mg aspirin three times daily and 1.2mg colchicine twice daily. Her cognition and chest pain markedly improved after initiation of these therapies along with supportive treatment. Colchicine was reduced to 0.6mg twice daily after one day. She was discharged two days after treatment in stable condition with 0.6mg colchicine twice daily for three months and 650mg aspirin three times daily to be tapered over two to four weeks.

Conclusion: Acute pericarditis is a rare complication of SARS-COV2. A high clinical suspicion should be noted in patients with recent SARS-CoV2 positive assays in correlation with elevated troponins and ECG changes. Cardiac injury is hypothesized to occur secondary to immune response rather than direct toxicity. Treatment is currently case by case due to a lack of standardized therapy. Early hospitalization and adjuvant remdesivir, aspirin, and colchicine were successful for our patient.
14) A CASE OF POST-STEMI VENTRICULAR FREE WALL RUPTURE CONFIRMED ON AUTOPSY

Trevor Birkey
Department of Medicine, Medical College of Wisconsin, Milwaukee, WI

Post-STEMI complications comprise a heterogenous group of disorders, of which perhaps the most lethal complication is mechanical rupture. This condition involves the tearing of infarcted, necrotic tissue with typical presenting findings of PEA arrest and cardiac tamponade from hemopericardium leading to cardiogenic shock and death.

A 70-year-old female was admitted to the hospital after 12 hours of constant, aching chest pain and discovered to have inferior STEMI on EKG. The patient had no previous cardiac history with a normal TTE and stress test 3 years prior, though had cardiac risk factors of hypertension, hyperlipidemia, and an extensive smoking history. Physical examination at the time of presentation was unremarkable. Relevant abnormal labs at the time of presentation include troponin-I of 16.69 and hemoglobin 11.2. 12-lead EKG demonstrated ST elevations in 2, 3, and aVF with q waves, ST depressions in V2, and T wave inversions in V4-V6. The patient was appropriately loaded with aspirin, Brilinta, and given a heparin bolus before timely transfer to the cath lab with successful placement of a DES to a 90% proximal stenosis in the left circumflex artery. Post-procedurally she was transferred to the ICU in stable condition with resolution of chest pain and initiation of standard post-STEMI treatment. Approximately 6 hours following the catheterization, a code was called for PEA arrest. Telemetry review demonstrated progressive bradycardia over the preceding 2-3 minutes, with the patient having been seen normal 10 minutes prior during routine neurologic check. ACLS protocol was initiated and followed for roughly 30 minutes with 6 doses of epinephrine administered and intubation performed prior to termination of resuscitative efforts. The patient remained in PEA throughout the duration of the code. Pathologic examination during an autopsy demonstrated a 2 cm slit-like tear in the inferior left ventricular myocardial free wall along with hemopericardium.

This case demonstrates an uncommon complication of STEMI. Free wall rupture requires a high level of suspicion and prompt diagnosis for consideration of surgical intervention if in the proper setting, though the exact technique remains controversial. In hospital-mortality remains high despite therapeutic strategy.
Introduction: A wide range of complications from COVID-19 have been reported since the first described cases in December 2019, including cardiac complications.

Case Presentation: A 71-year-old female with systemic lupus erythematosus complicated by focal segmental glomerular sclerosis status-post kidney transplant presented with worsening left-sided chest pain after receiving treatment for COVID-19 pneumonia at an outside hospital. Her physical exam revealed reproducible chest pain and a friction rub on cardiac auscultation, and ECG revealed sinus tachycardia and diffuse ST-segment elevations in leads II, III, aVF and V1-V6. After subsequent echocardiogram demonstrated no wall-motion abnormalities, she was diagnosed with acute pericarditis likely secondary to viral infection with COVID-19. She was successfully treated with aspirin and colchicine for 90 days without complications.

Discussion: NSAIDs and colchicine are mainstays in the treatment of acute pericarditis. Treatment for this patient presented a potential challenge given this patient’s immunosuppressive regimen included tacrolimus. Tacrolimus has been associated with increased risk of colchicine toxicity-ranging from mild gastrointestinal side effects to fatal myotoxicity and rhabdomyolysis--when administered concomitantly due to its interference with colchicine metabolism. However, there has been at least one other case of acute pericarditis secondary to COVID-19 infection in a kidney transplant patient on tacrolimus successfully treated with colchicine without complications. Colchicine and aspirin also proved to be effective in treating our patient’s case of COVID-19-associated pericarditis.

Conclusion: This case report has implications for future treatment of renal transplant patients with COVID-19-related pericarditis and emphasizes the need for research into the pathophysiology of pericarditis in the context of COVID-19, including risk factors and treatment.
16) AN UNUSUAL SITE OF RESIDENCE FOR FUSOBACTERIUM NUCLEATUM

Bryanna Buchman; Pinky Jha, MD
Medical College of Wisconsin, Milwaukee, WI

Introduction: *Fusobacterium nucleatum* is a Gram-negative anaerobe that has proven difficult to culture and is often missed upon initial screening. This fastidious organism is typically found as an oral commensal and periodontal pathogen. FadA is the key virulence factor that allows this organism to adhere and invade systemically. Well documented systemic effects of this commensal turned pathogen include adverse pregnancy outcomes including preterm labor and preeclampsia, as well as GI disorders such as colorectal cancer and inflammatory bowel disease.

Case: A 77 year-old male presented with the primary complaint of abdominal pain and diarrhea. Patient reported 3-5 loose stools without melena beginning when he was on vacation in Mexico where he consumed the water. In Mexico, he was diagnosed with Salmonella and given Ciprofloxacin with no improvement. An abdominal CT revealed a large hepatic abscess and patient was started on Ceftriaxone/Flagyl. The hepatic abscess was drained using Interventional Radiology guidance. Patient was discharged with IV Zosyn pending drainage culture results.

Cultures came back positive for *Fusobacterium nucleatum*. Four weeks later at a follow-up visit, fever and abdominal pain had improved. Labs showed decreased WBC and platelet count. A repeat abdominal CT showed an improved abscess. The patient was still tolerating the antibiotics well.

Discussion: Here we report a rare case of liver abscess to increase awareness among practitioners to consider this diagnosis. This is an atypical presentation and location of *Fusobacterium nucleatum* in a patient who presented with 3-5 episodes of loose stools and abdominal pain while vacationing in Mexico, prompting his early return. This organism is difficult to culture which most likely explains the incorrect initial diagnosis of Salmonella and no symptom improvement with Ciprofloxacin. Only after an abdominal CT, was an abscess noted and subsequently drained to determine the offending agent. Perhaps as our diagnostic and laboratory technology continue to improve, we will find more often that *F. nucleatum* is the cause of other systemic symptoms and conditions not yet recognized in the literature.
Malignant peripheral nerve sheath tumors are classified as soft tissue sarcomas that sometimes arise from pre-existing neurofibromas or normal nerves. Up to 50% of peripheral nerve sheath tumors occur in patients with neurofibromatosis type 1 (NF1). These tumors are most commonly found in the extremities and less commonly in the head and neck. Prognosis is poor, especially in tumors exceeding 5 cm in size and those tumors which are associated with NF1.

Case: A 21 year-old female with a past medical history of NF1 and a malignant nerve sheath tumor, presented with the primary complaint of shortness of breath and worsening palpitations. A chest x-ray obtained in the emergency department showed near-complete opacification of the left hemithorax causing a collapse of the left lung and right mediastinal shift. The patient was admitted and a chest tube was placed in attempts to drain the pleural effusion. Scant drainage was reported with minimal clinical improvement. The patient underwent a left thoracoscopy and a biopsy was taken of the pleural mass. Biopsy results were consistent with a malignant peripheral nerve sheath tumor. Surgical consultation revealed no viable surgical options and no bronchi suitable for stent placement. Oncology trialed Ifosfamide/Etoposide as chemotherapy, but patient experienced toxicity and was subsequently given methylene blue for several doses. Patient’s condition deteriorated and she was transferred to the ICU where she was intubated. A CT showed near collapse of her left lung, with the mass continuing to push on the left atrium and other mediastinal structures. Patient expressed her desire for life sustaining measures and tracheostomy was placed. She later changed her wishes and was transferred from life-supporting measures to palliative care where she passed.

Discussion: Here we report a case of a malignant peripheral nerve sheath tumor arising in the left hemithorax causing significant lung atelectasis and mediastinal structure dysfunction. This report can help increase awareness to providers that these tumors are not only limited to the extremities. The patient presented with classic signs of a pleural effusion, but only after chest tube placement and drainage was it discovered that an abscess was the most likely culprit. Biopsy results of a peripheral nerve sheath tumor were epidemiologically consistent with the patient’s past medical history of NF1, but the location of the tumor is unique. Lack of surgical and chemotherapy options fashioned a tragic ending to this case.
18) LEPTOMENINGEAL RECURRENCE OF TESTICULAR DIFFUSE LARGE B CELL LYMPHOMA
Anna Luise Calderon, BS; Nabeel Khan, MD; Pinky Jha, MD
Medical College of Wisconsin, Milwaukee, WI

Introduction: Diffuse large B-cell lymphoma (DLBCL) is the most common form of non-Hodgkin lymphoma (NHL). Rarely, it presents as primary testicular lymphoma, an aggressive form that represents 1% of all NHL cases. Testicular DLBCL frequently recurs in the CNS, which has resulted in increased CNS prophylaxis with rituximab and radiation therapy.

Case: An 83-year-old male with history of testicular DLBCL, Lyme disease, and CHF who presented with new onset progressive lower extremity weakness. Over the past 3 weeks he had progressive generalized weakness, fatigue, and back pain. In the week leading up to admission he had significantly increased weakness in his legs. The patient also reported an approximate 10-pound weight loss, and his wife reported transient confusion. On admission, he had 5/5 strength in his upper extremities bilaterally and 1/5 weakness in his lower extremities bilaterally. He was alert and oriented and had no other focal neurological deficits besides CN 6 and 7 deficits that had been identified at a previous encounter. Laboratory findings were significant for hyponatremia that resolved with normal saline. Shortly after admission he developed urinary retention. Due to these symptoms, continued weakness, and concern for recurrence of his cancer, thoracic and lumbar MRIs were completed and found significant focal leptomeningeal lymphoma involvement at L2-L3 and milder involvement along the cauda equina. CT showed no significant lymphadenopathy or evidence of disease outside the CNS. Subsequent lumbar puncture showed an elevated white blood cell count and protein, and cytology was consistent with DLBCL. MRI showed new involvement of the posterior lateral ventricles. He was started on high dose dexamethasone and methotrexate, and subsequently received rituximab and radiation to the brain and spine. His weakness improved with treatment and physical therapy and he was discharged to subacute rehab.

Discussion: Here we discuss the case of a patient with recurrent testicular DLBCL with CNS involvement who presented with progressive weakness. Testicular DLBCL frequently recurs in the CNS, however patients typically present with focal CNS deficits or headaches. Rituximab has been shown to improve outcomes of testicular DLBCL, however as prophylaxis it has not shown to decrease CNS recurrence. It is therefore important to consider CNS involvement when a patient with history of testicular DLBCL presents with findings, however atypical, consistent with CNS recurrence.

References:
**Background:** Hospital readmissions are a common, costly problem. Readmissions occurring within a few days of discharge are more likely due to a problem from the patient’s original admission and may be preventable by interventions in the hospital setting.

**Objective:** To determine causes of readmission within 72 hours and identify indicators of readmission during the index admission.

**Design:** Patients readmitted within 30 days were previously interviewed by a social worker regarding reasons for readmission and their perspective on what might have prevented it. These answers were abstracted from charts of patients readmitted within 72 hours and compared with physician notes. If patients were identified as potentially benefitting from a longer hospitalization, their index admission was reviewed for indicators of readmission.

**Setting, patients:** 131 patient readmissions within 72 hours between 2/1/2019-6/7/2019 in a healthcare system containing an academic medical center.

**Measures:** Reasons for readmission, potential preventive measures, and indicators of readmission were independently reviewed by two authors then grouped into common themes by consensus.

**Results:** Most patients were readmitted for infection related, cardiac or pulmonary reasons. Extending the initial admission was the most common factor suggested by both patients and physicians to prevent readmission. Of 70 patients who may have benefited from a longer admission, indicators included patients not returning to their baseline health status, inadequate management of a known issue, or new symptoms developing during the index admission.

**Conclusions:** Patients should be evaluated for indicators of readmission which may help guide decisions to discharge patients and decrease rates of 72-hour readmissions.
Introduction: Lyme disease is a tick-borne illness which can affect multiple organ systems, and symptoms vary based on clinical stages. Patients classily present with dermatologic findings of erythema migrans, but initial presenting symptoms can vary depending on the staging of the disease. Lyme disease can progress to cause life-threatening conditions.

Case: A 71-year-old female with a past medical history of chronic myelomonocytic leukemia presented in the winter with a 3-day history of right eye swelling and conjunctivitis. On initial presentation, the patient had right-sided 2-mm proptosis, chemosis, and mild edema surrounding right eye. Extraocular motility, visual fields, visual acuity, intraocular pressure were intact, and the patient did not experience eye pain. Cranial nerve exam did not display any deficits. CT and MRI of the head and orbits displayed right scleritis, episcleritis, possible evidence of cellulitis without abscess formation, mild retrobulbar edema which did not involve the orbit, and mild proptosis. CT angiogram of the head was unremarkable. Initial labs were significant for leukocytosis (32.7), elevated CRP (1.10) and ESR >119. The patient had a positive ANA but negative dsDNA. ACE, Antichromatin, Ribosomal, Centromere, SCL-70, Smith, RNP, Anti Jo, SSA/SSB, RF, and IgG4 antibodies were not detected or were within normal limits. Lyme IgG and IgM antibodies were both positive. Chart review revealed prior presentation to dermatology 6 months prior for evaluation of a pink indurated 5cm multifocal noncircular rash on the right buttock. Biopsy of the rash displayed findings of superficial and deep lymphocytic infiltrate with numerous eosinophils consistent with an arthropod bite. Due to high inflammatory markers, the differential included orbital inflammatory syndrome therefore the patient was started on IV methylprednisone. The patient’s eye edema, proptosis, scleritis, and episcleritis resolved during 5-day hospitalization. She was discharged on a prednisone taper. However, she was lost to follow-up.

Discussion: The diagnosis of Lyme disease without initial findings of erythema migrans or an obvious clinical backstory of tick exposure is difficult due to the myriad of symptoms and variable clinical findings that Lyme disease can cause. Early localized Lyme disease has been known to manifest with ocular symptoms. This case demonstrates that later stages of Lyme disease can manifest as serious ocular findings, including orbital inflammation. This illustrates the importance of evaluating for Lyme disease in patients who present with ocular complaints, high inflammatory markers, and otherwise negative rheumatologic and infectious evaluation.
Introduction: Drug induced liver injury (DILI) is a common cause of elevated liver chemistries. Presentations can range between asymptomatic and fulminant hepatic failure. The list of products that can cause DILI continues to expand. Diagnosis of DILI is often challenging as it requires ruling-out more common causes of liver injury, detailed discussions with patients, and sometimes a liver biopsy.

Case: An 89-year-old female with a medical history of depression, breast cancer, and Hodgkin lymphoma presented with a one-week history of generalized weakness and failure to thrive. The patient was a poor historian and unable to identify a single chief complaint. She stated that her only home medication was acetaminophen, which she took very rarely for pain. She denied any alcohol or recreational drug use. Review of systems and physical exam were unremarkable, and exam was negative for hepatomegaly, splenomegaly, peripheral edema, jaundice, and asterixis. Initial laboratory data was significant for an ALT of 882, AST of 353, alkaline phosphatase of 272, and total bilirubin of 2.2. Additionally, Hepatitis A, B and C panel negative, ASMA negative, ANA positive, no presence of serum acetaminophen, negative ceruloplasmin, INR of 1.1. Ferritin was elevated at 1,519mg/dL, however it was within normal limits on all prior labs. Right upper quadrant abdominal ultrasound and CT abdomen did not display any evidence for cirrhosis or anatomic abnormalities in the liver or biliary tree. After additional discussions with the patient’s daughter, her primary caregiver, it was found that the patient was also taking daily curcumin supplementation. Curcumin was not given during her hospitalization, and although no interventions outside of physical, occupational, and nutritional therapy were taken during the admission, the patient’s liver chemistries quickly began to downtrend. The patient was discharged with instructions to discontinue curcumin supplementation, and on one month follow up, the patient’s liver chemistries had fallen to within normal limits.

Discussion: The diagnosis of DILI requires in-depth patient interviews, exclusion of underlying liver disease, and in some cases, by liver biopsy. This case outlines the importance of considering non-stereotypical pharmaceuticals and supplements in the evaluation of suspected DILI. This case also illustrates the importance of gathering history from multiple sources in patients who are poor historians, and the importance of re-interviewing patients throughout a hospitalization. In this case, these extra measures allowed the team to make a diagnosis of DILI without the need for more invasive or costly laboratory tests or procedures.
22) ICD-PACEMAKER LEAD ASSOCIATED GROUP B STREPTOCOCCUS ENDOCARDITIS

Medhavani D. Chandra¹; Javad Najjar Mojarrab²; Ateeq Rehman²

¹Department of Med-Peds, ²Department of Internal Medicine, Marshfield Clinic, Marshfield, WI

Group B streptococcus in adults may cause skin and/or soft tissue infections, bacteremia without a focus, pneumonia, osteomyelitis, and rarely, meningitis or endocarditis, both of which are associated with higher morbidity and mortality especially in the elderly and immunocompromised. The presence of endocavitary cardiac devices has been noted to be a predisposing factor for endocarditis due to β-hemolytic streptococci, which may present acutely or subacutely, and associated mortality as high as 40%.

An 86-year-old male with ischemic cardiomyopathy with an ejection fraction of 27% and biventricular pacemaker-ICD presented to the ER for lower extremity edema and generalized weakness. He had undergone radiation and surgical dissection for squamous cell carcinoma of the head, which resulted in a left periauricular wound with exposure down to the bone but with no signs of infection on physical exam. Based on initial evaluation, he was diagnosed with acute heart failure exacerbation and was treated accordingly. On day 4 of hospitalization, he abruptly deteriorated with fever and markedly elevated inflammatory markers. This rapid onset sepsis lead to sustained hypotension needing vasopressor support in the ICU. Two sets of blood cultures were positive for GBS. A transthoracic echocardiogram (TTE) showed a linear, mobile echodensity associated with the tricuspid valve versus ICD/pacemaker leads. A transesophageal echocardiogram (TEE) showed a very long, highly mobile echodensity measuring several centimeters that seemed to be attached to the coronary sinus pacemaker/ICD-lead. He met Duke’s criteria for infective endocarditis and was treated with an antibiotic. The patient underwent a transvenous ICD lead and device extraction with the placement of a temporary transvenous pacemaker.

This case illustrates the potential risk factors that may lead to pacemaker-associated GBS infection. It also signifies the use of TEE over TTE in diagnosing lead vegetations. Pacemaker lead infection is a life-threatening condition and warrants removal of all foreign material by either thoracotomy or intravascular extraction, depending on the size of the vegetation, state of the tricuspid valve, and clinical status of the patient.
23) REGIONAL CITRATE AND SYSTEMIC HEPARIN ARE ADEQUATE TO MAINTAIN FILTER HALF LIFE FOR COVID-19 PATIENTS ON CRRT

Cassandra Chiao, MD; Hilary Faust, MD; Tripti Singh, MD
Department of Medicine, University of Wisconsin- Madison School of Medicine and Public Health, Madison, WI

Purpose: The aim of our study is to compare clotting of CRRT filters in patients with COVID-19-associated AKI vs. septic shock-associated AKI.

Methods: Retrospective single center study of adult patients with COVID-19 infection compared to those with septic shock admitted to the ICU at a tertiary university hospital April-October 2020. We used independent t-test and chi square test to determine statistical significance of CRRT filter clotting and related factors in COVID-19 patients compared with septic shock patients in the ICU. Time to event data was analyzed with Kaplan-Meier curves. Analyses were performed on Microsoft Excel and MedCalc.

Results: A total of 27 ICU patients with AKI requiring CRRT were included in the study, 13 with COVID-19 infection and 14 with septic shock. The mean half-life of CRRT hemofilter was similar in COVID-19 patients compared to non-COVID-19 patients (27.4 hours vs 27.5 hours, p=0.79). The number of CRRT hemofilter changes per day were also similar in both groups (0.6 filter changes per day, p=0.84). However, significantly more patients with COVID-19 were on systemic heparin compared to the non-COVID-19 patients (69% vs 13%, p= 0.02).

Conclusions: We found that COVID-19 patients with AKI requiring CRRT had similar CRRT hemofilter half-life compared with sepsis-associated AKI patients with use of regional citrate anticoagulation and systemic heparin use. Further studies are needed to find which methods of anticoagulation are optimal in patients with COVID-19 infection with AKI requiring CRRT.
24) ILLUSTRATING THE MECHANISM OF SOCIAL SUPPORT IN PATIENTS WITH PANCRATIC CANCER

Meghan Conroy¹, Samih Thalji, MD²; David Nelson, PhD, MS³
¹Medical College of Wisconsin, Milwaukee, WI; ²Department of Surgery, ³Dept of Family and Community Medicine, Milwaukee, WI

Background: Pancreatic cancer is an aggressive disease with high morbidity and mortality. It is known that social determinants of health play a role in pancreatic cancer incidence and outcomes, however, there has been little research into how social determinants impact patients’ personal experiences of illness and quality of life. Social support is defined as actual or perceived assistance provided by social networks. In the setting of breast cancer, social support improves patient’s quality of life. However, the impact of social support on quality of life has not been explored in patients with pancreatic cancer. The objective of this study was to gain insight into the mechanisms through which social support may influence quality of life experiences in the pancreatic cancer patient population.

Methods: Semi-structured interviews were conducted with patients with pancreatic cancer. Perceptions of social determinants and social support in the context of their disease were discussed and analyzed using a grounded theory approach.

Results: Analysis of five interviews thus far resulted in two primary types of social support associated with decreased feelings of stress and health outcomes. First, social networks provided tangible support in the form of transportation, finances, liaison services, meals, and caretaker duties. Second, social networks provided emotional/psychological support. This type of support was instrumental in allowing patients to not worry and feel comforted throughout their treatment process and beyond. Both types of social support were related to decreased feelings of stress.

Conclusions: The presence and recognition of social determinants is an important consideration for the health of patients and positive outcomes. For example, one patient said that if he didn’t have a good support system in place, he would not have gone through chemotherapy or surgery. Social support is essential to many patients throughout cancer diagnosis, treatment, and recovery. It is important to identify the mechanisms through which social support may improve both treatment outcomes and quality of life in patients with serious illnesses like pancreatic cancer. There is a need to build an intentional system that connects resources around social determinants and social support to patients with pancreatic cancer. This research study will inform future interventions to improve outcomes and support quality of life.
25) CANCER CELLS CAN CONSUME GLUCOSE TOO

Kevin Credille
Medical College of Wisconsin, Milwaukee, WI

An 86-year-old female with a history of chronic obstructive pulmonary disease, hepatocellular and basal cell carcinoma, and latent tuberculosis infection (treated in 1979) presented with a femoral fracture and pleural effusion. A twenty-pound weight loss was discovered over the course of the prior year without any associated fevers. Results from the thoracentesis during her admission showed a cloudy red-amber fluid with elevated lactate dehydrogenase (LDH), moderately elevated protein, and extremely low glucose level less than 2 mg/dL consistent with an exudate. Bacterial analysis showed a negative gram stain and culture negative for aerobic, anaerobic, fungal, and acid-fast organisms, ruling out bacterial or mycobacterial etiology. Finally, cytology and flow cytometry returned findings consistent with a large B-cell lymphoma.

Diagnosis of pleural effusion requires thorough history taking, thoracentesis and systematic approach to analysis of the pleural fluid. Tests routinely performed on pleural fluid include cell count, cell differential, pH, protein, LDH, and glucose. Characterization of the pleural fluid based on Modified Light’s Criteria as either a transudate or an exudate will further help in narrowing differentials. Typically, a very decreased pleural fluid glucose level narrows the differential diagnosis to an empyema or rheumatoid pleurisy. Malignant pleural effusions commonly have an exudative presentation with a glucose reading in the normal or moderately elevated range, but occasionally can present in the low range (30 to 60 mg/dL). Only one case study has previously described a malignant pleural effusion with a very low glucose level less than 30 mg/dL of glucose. The mechanism of low pleural glucose is poorly understood but presumed to be due to both increased glucose uptake by leukocytes and cancer cells and impaired glucose transport across the pleural membrane secondary to fibrosis and tumor presence. Although it is rare, B-cell lymphoma can present as an exudative pleural effusion with very low glucose level. Diagnostic vigilance with recognition of this possibility is critical to appropriate management.

This case demonstrates the potential capability of malignant cells to metabolize glucose as rapidly as bacterial and inflammatory cells in pleural bacterial infection and rheumatoid pleurisy. The clinical implication from this case is that malignancy should remain on the differential diagnosis in patients even with pleural fluid analysis showing a glucose level less than 30 mg/dL, which may possibly serve to reduce excessive usage of antibiotics in this scenario.
Background: Pentosan polysulfate sodium (PPS) is an oral medication used for the chronic management of interstitial cystitis (IC), a syndrome of chronic pelvic pain and urinary urgency that primarily affects women. Although associated with few adverse effects, recent studies suggest an ocular toxicity associated with long-term use. We used multivariable logistic regression models to investigate this relationship in a large, cross-sectional cohort of patients with IC.

Methods: The TriNetX Research Network, a database of more than 60 million patients from over 50 healthcare organizations, was queried on February 26th, 2020 for 31,562 adults with IC. Logistic regression models were fitted to determine the odds of retinopathy diagnosis associated with PPS while adjusting for known risk factors, with 494 patients excluded for missing age or sex. The outcome variable was a binary value informed by the presence of any retinopathy diagnosis that may have been used to document PPS-related maculopathy: non-exudative or exudative age-related macular degeneration, drusen, hereditary retinal dystrophy, toxic maculopathy, and unspecified macular degeneration.

Results: The study included 2,492 men and 29,068 women with an average age of 54.6 (17.0 SD) years. A total of 6,261 (19.8%) patients had at least one order for PPS. Average duration of PPS therapy was 1.27 (2.25 SD) years. Multivariable logistic regression analysis revealed a statistically significant increase in the odds of retinopathy associated with PPS duration (OR=1.13 per year, 95% CI 1.08-1.18, p<0.001), hydroxychloroquine duration (OR=1.20 per year, 95% CI 1.11-1.28, p<0.001), age (OR=1.08, 95% CI 1.07-1.09, p<0.001), history of smoking (OR=1.40, 95% CI 1.15-1.70, p<0.001), essential hypertension (OR=1.89, 95% CI 1.54-2.32, p<0.001), diabetes mellitus (OR=1.28, 95% CI 1.05-1.55, p=0.01), and significant kidney disease (OR=1.49, 95% CI 1.23-1.81, p<0.001). Repeat multivariable regression with PPS duration as a categorical variable in one-year increments revealed a significant association starting at 6 years of PPS use (OR=3.11, 95% CI 1.34-6.31, p=0.004).

Conclusion: After adjusting for covariates known to increase the risk of retinopathy, there was a statistically significant increase in the odds of retinopathy diagnosis corresponding with duration of PPS therapy. These findings strengthen the body of evidence suggesting an ocular toxicity related to PPS.
Introduction: Legionnaire’s disease is caused by inhalation or aspiration of water droplets containing the opportunistic Gram-negative bacilli species, *Legionella*. Infection classically presents as an atypical pneumonia with accompanying diarrhea and hyponatremia, while hepatic and renal involvement are rare. Here, we present a case of *Legionella* pneumonia with acute hepatitis.

Case: A 61-year-old female with a history of anemia and hypertension presented with several days of loose stools, poor oral intake, increasing shortness of breath, and productive cough following known COVID-19 exposure while traveling in another state. She was tachycardic, but was otherwise hemodynamically stable, afebrile, and saturating appropriately on room air with audible rhonchi. ED workup was significant for a WBC count of 34,000/mL, sodium of 132 mEq/L, potassium of 2.6 mmol/L, creatinine of 1.5 mg/dL, ALT of 406 U/L, and AST of 474 U/L. CXR was consistent with pneumonia and empiric ceftriaxone and azithromycin were started. CT Abdomen and Pelvis, COVID-19 testing, and urine/blood cultures were unremarkable. She was continued on azithromycin monotherapy following positive urine *Legionella* antigen testing. The patient was admitted for IV rehydration, electrolyte replacement, and comprehensive work-up, which revealed no other apparent cause for her acute hepatitis. Her symptoms improved throughout admission and she was discharged on the fifth day with a clear lung examination, resolved acute kidney injury, down-trending transaminases, and plans for outpatient monitoring.

Discussion: This case aims to raise awareness amongst clinicians about the rare occurrence of *Legionella* induced hepatitis and to consider it when evaluating any patient with a combination of pulmonary and gastrointestinal symptoms. This is especially important now given the overlap in presentations between Legionnaire’s and COVID-19. Diagnosis is made by the urine antigen test or sputum culture, but Legionnaire’s is often under-diagnosed and underreported. Given the potential for multiorgan involvement, we recommend clinicians have a low threshold for laboratory monitoring to prevent progression to acute organ failure. Comprehensive acute liver injury testing may not be necessary if transaminases improve with antibiotic initiation but can provide support that *Legionella* is the likely cause of their acute hepatitis. Early identification of Legionnaire’s disease and its less-common presentations can reduce morbidity and mortality.
Introduction: Drug induced lupus is an autoimmune disorder triggered by several known medications. Of these medications, tumor necrosis factor (TNF)-alpha inhibitors, including infliximab, have been reported to cause anti-tumor necrosis factor-alpha induced lupus (ATIL). The estimated incidence of ATIL is 0.19-0.22% for infliximab. Of these cases, only 9% present with either pleural or pericardial effusions, making this adverse event exceedingly rare.

Case: A 73-year-old female with a past medical history of diabetes mellitus type II and complicated Crohn’s disease presented to the emergency department with a two-day history of pleuritic chest pain. The chest pain was located across the lower part of her chest bilaterally; it was very intense, did not radiate, and was made worse by sitting up and leaning forward. She denied fever, chills, orthopnea, cough, dyspnea, edema, and rashes. She underwent a CT of the chest and the abdomen which revealed moderate-sized pleural effusions and a pericardial effusion, and she was subsequently admitted to hospitalist service. Inpatient echocardiogram, stress test, and CT angiography of the chest were all performed and revealed no abnormalities aside from the known pericardial and pleural effusions. Since the patient had been taking Infliximab for one year prior to control her Crohn’s disease, a diagnosis of drug-induced lupus was considered. Serology was performed, and ANA and anti-dsDNA antibodies were both newly positive at this time. Per rheumatology recommendation, Infliximab was held, which lead to gradual improvement of her clinical symptoms within several days. Her pericardial and pleural effusions gradually resolved, and her lupus antibodies subsequently became undetectable with prolonged discontinuation of infliximab.

Discussion: The diagnosis of drug induced lupus requires a high index of suspicion; furthermore, the diagnosis of ATIL presenting with pericardial and pleural effusions remains highly elusive. Consider ATIL as an etiology in patients with pericardial and/or pleural effusions not deemed to be cardiogenic, inflammatory, neoplastic, or infectious in nature. The temporal relationship between the introduction of TNF-alpha inhibitors and the development of serologic and clinical manifestations should prompt suspicion for ATIL.
Introduction: Cardiac amyloidosis is a condition in which abnormal amyloid fibril proteins are deposited in the myocardium. Occasionally, patients with cardiac amyloidosis demonstrate an atypical pattern of infiltration with asymmetric septal thickening and a left ventricular outflow tract (LVOT) gradient with systolic anterior motion (SAM) of the mitral valve resembling obstructive hypertrophic cardiomyopathy (HCM). We present a case of cardiac light-chain amyloidosis and LVOT obstruction successfully treated with alcohol septal ablation (ASA).

Case: A 70-year old man with a history of multiple myeloma was admitted for evaluation and management of acute decompensated heart failure. He had received a diagnosis of cardiac amyloidosis six years prior after presenting with lower extremity edema, palpitations, and several episodes of pre-syncope, and had been followed by cardiology with serial transthoracic echocardiograms (TTE). He was clinically stable up until several months prior to admission, during which time he had multiple admissions for acute decompensated heart failure. TTE at that time demonstrated progression of left ventricular wall thickness up to 22 mm and worsening LVOT obstruction with a peak gradient of 70 mmHg. The decision was made to proceed with percutaneous ASA. Following the procedure, the patient reported significant improvement in his dyspnea. At three-month follow-up, he continued to have improvement in his symptoms and a repeat TTE demonstrated an LVOT gradient of 20 mmHg and improvement in the SAM of the mitral valve.

Discussion: Rarely, patients with cardiac amyloidosis have isolated wall thickening and evidence of LVOT obstruction that resembles that of obstructive HCM. Septal reduction therapy is the treatment of choice for symptomatic obstructive HCM refractory to medical therapy, however, there is a lack of data regarding whether patients with cardiac amyloidosis and LVOT obstruction benefit from similar treatment strategies. ASA is a technique that is used in the treatment of obstructive HCM and involves the injection of alcohol into the septal perforator coronary arteries creating a small, controlled myocardial infarction. This case demonstrates that ASA is a technically feasible and effective procedure for relieving LVOT obstruction in cardiac amyloidosis and should be considered as a treatment option in patients whose symptoms are refractory to medical therapy.
Background: Pemigatinib is a fibroblast growth factor receptor (FGFR) kinase inhibitor used in unresectable and metastatic cholangiocarcinoma. It is known to cause hyperphosphatemia leading to alterations in serum calcium concentrations and soft tissue mineralization. Optimal management for these adverse events are unknown.

Case: A 40-year-old female with metastatic FGFR2-BICC1 Fusion cholangiocarcinoma treated with pemigatinib presented during cycle two with painful skin thickening of her bilateral axillae. Her serum phosphate (which was normal prior to starting pemigatinib) increased to 8.5 mg/dL and serum calcium increased to 12.4 mg/dL. An empiric trial of topical steroids was initiated without symptomatic improvement. A skin biopsy confirmed the clinical suspicion for calcinosis cutis secondary to pemigatinib. She was started on sevelamer 800 mg TID for hyperphosphatemia. She then experienced further skin eruptions in the popliteal fossa. Given her progressive skin lesions, pemigatinib was held. Zoledronic acid 4 mg IV was given for hypercalcemia. Extrapolating from the management of calcinosis cutis in the setting of dermatomyositis, she was started on diltiazem 120 mg daily and topical sodium thiosulfate. Her lesions improved with the above interventions. Full dose pemigatinib was restarted without exacerbation of her calcinosis cutis. Her cancer has responded to treatment with a sustained partial response.

Discussion: Calcinosis cutis associated with hyperphosphatemia is a known but rare side effect of FGFR inhibitors. Although hypocalcemia is more common, soft tissue calcifications can be seen in intertriginous areas with both hypercalcemia and hypocalcemia. There is limited data to support treatment of calcinosis cutis in this setting. Cessation of pemigatinib was recommended so symptoms could be controlled. Treatment was resumed following improvement in her electrolytes and skin lesions. This case demonstrates a multifaceted approach that was needed to manage hypercalcemia, hyperphosphatemia, and cutaneous symptoms while also balancing the importance of limiting treatment disruptions in the setting of metastatic cholangiocarcinoma.
**Case:** A 64 year old female with a history of asthma and Primary Biliary Cholangitis post liver biopsy 10 days prior presented to the ED with 5 days of intermittent abdominal pain. Pain was waxing and waning without obvious triggers and was localized primarily to the epigastrium with some right upper quadrant component. The patient reported nausea, denied vomiting, and uncertainty if related to meals due to lack of appetite. In the ED, initial vitals were: Temperature 98.6 F, Blood Pressure 149/72, Heart Rate 68, Respiratory rate 16. Notably, there was negative rebound, guarding, and Murphy’s sign. Labs notable for no leukocytosis (Leukocytes 6.7), Hemoglobin 13.6, Alkaline Phosphatase 280 (baseline 130s-160s), Aspartate Aminotransferase 204 (previously in the 30s), Alanine Aminotransferase 376 (previously in the 50s), and total bilirubin 2.7 (previously <0.4). Lipase was elevated at 192. Computerized tomography scan of the abdomen/pelvis showed a distended thick-walled gallbladder with hyperattenuating contents which could represent sludge, pus, or blood, as well as mild pelvic free fluid. A Right upper quadrant ultrasound showed echogenicity material within the gallbladder lumen without shadowing, likely related to hemorrhage. Once admitted, an MRCP was done with GI that showed hemorrhagic cholecystitis without CBD blockage so ERCP was not recommended. HIDA scan was also performed which showed absent gallbladder filling consistent with acute cholecystitis and general surgery performed urgent cholecystectomy.

**Discussion:** Hemorrhagic cholecystitis is an extremely rare form of cholecystitis with an inconsistent presentation and multiple causes. Notable causes include transmural necrosis secondary to calculus cholecystitis, rupture of cystic artery pseudoaneurysms with hemobilia, and blunt trauma. Pt risk factors include use of anticoagulants, kidney disease, COPD, and chronic steroids. Patients always present with abdominal pain, many with nausea and vomiting, some with fever and leukocytosis, and only some with hyperbilirubinemia. Additionally, the degree of bleeding does not typically lead to hemodynamic instability but that is a possibility and should determine the course of treatment. Severe complications include peritonitis, hemoperitoneum, and hematemesis. Computerized Tomography, which is superior at differentiating calculous cholecystitis from hemorrhagic cholecystitis, and ultrasound are the typical imaging modalities used for diagnosis. Definitive treatment is typically urgent cholecystectomy.
Abstract: Point of care ultrasound (POCUS) is convenient and relatively inexpensive, raises confidence in clinical decision making, improves patient and provider relationships by bringing provider to bedside, improves safety of bedside procedures, and expedites and improves accuracy of diagnoses. More and more Internal Medicine residency programs are attempting to create a structured POCUS curriculum with clear proficiency goals. Studies suggest that portable ultrasound simulator may provide equivalent training in comparison to traditional live instructor and model training. Goals of this program were to 1) Solidify fundamentals of US 2) Create a sustainable template for curriculum 3) Improve resident confidence through integration of ultrasound simulator, online modules and self-assessments, didactic lectures, direct patient scanning, and live model training 4) Establish a POCUS lecture series 5) Track progress and identify obstacles along the way 7) Use pre-test and post-test to assess success of program. Methods included developing a curriculum divided into independent study and group study. Independent study utilized SonoSim learning modules. The academic year was divided into “blocks” and each “block” had corresponding assignments (Figure 1). A SonoSim representative assisted in tutorial. Group study curriculum included a weekly 5 minute review of ultrasound, small group learning with live ultrasound models, small group learning ad libitum. Competency assessments were performed by pre-test and a post-test. Reminder emails were utilized in attempts to improve participation. Exclusion from analysis: residents who participated in LIVE pocus classes in past or who did not attempt both competency tests. Total participation: 10 PGY-1, 8 PGY-2, 5 PGY-3 for a total 23 residents. Results showed that all 3 cohorts of residents improved their scores, yet the comfort level of residents increased at a greater rate than resident scores. Participation was less in more senior residents compared to interns. Average number of resources used were 2-3, even though 2 residents expressed “I have yet to touch an ultrasound this year”. In conclusion, COVID-19 pandemic had a significant impact on the execution of group study. Resident participation was poor overall and there were problems with enforcing the curriculum as proposed. Possible contributing problems are 1) resident-led nature of curriculum 2) viewed as “not required by ACGME”. Considering the value of this competency, we propose POCUS be foundation of all programs in future.
33) UNANTICIPATED RADIOGRAPHIC FINDING IN PRE-OPERATIVE PATIENT

Maria Georgen\textsuperscript{1,2}; Stefanie DiGiandomenico\textsuperscript{1,2}; Andrew Rosenblum\textsuperscript{1,2}

\textsuperscript{1}Medical College of Wisconsin Affiliated Hospitals, Milwaukee, WI
\textsuperscript{2}Clement J. Zablocki VA Medical Center, Milwaukee, WI

Lanthanum carbonate is a commonly prescribed oral phosphate binder in patients with advanced renal insufficiency. Lanthanum is a natural earth metal and can appear as radiopaque deposits within the gastrointestinal tract on imaging. Here we describe a case of a patient admitted with unstable angina who, pre-operatively, was incidentally found to have multiple small radiopaque densities throughout his colon. This case illustrates the importance of including lanthanum carbonate in the differential diagnosis of radiopaque foreign objects, especially in patients with renal insufficiency. Early recognition of this association may prevent costly use of medical resources for a relatively benign ailment.
34) OVERDIAGNOSIS OR JUST UNLUCKY?
Georgescu, Abigail, MS4; Al Homssi, Amer, MD
Medical College of Wisconsin, Milwaukee, WI

Introduction: Screening tests, though integral to preventive medicine, carry risks like any procedure in which a patient participates. While the goal of adequate screening is the highest sensitivity and specificity possible, they are not without the risk of overdiagnosis. In this case, we describe an eventful month-long hospital stay after a screening chest X-ray (CXR) for a young patient.

Case Description: An otherwise healthy 24-year-old African American female was transferred to our hospital after a failed diagnostic bronchoscopy at an outside institution. The bronchoscopy was done to biopsy a left hilar mass noted on a screening CXR and a subsequent chest CT scan. The patient had no respiratory or constitutional symptoms with a normal pulmonary examination and just finished quarantine for asymptomatic COVID-19 infection. During her 29 days of hospitalization, she underwent extensive workup for the mass: negative broad infectious and rheumatologic testing, multiple bronchoscopies, endoscopies, and a robotic-assisted thoracotomy. After multiple inconclusive biopsies, histopathology of the biopsy obtained from the thoracotomy demonstrated extralobular sequestration versus congenital pulmonary airway malformation. However, the patient experienced multiple complications of interventions: bleeding from the site of biopsy requiring post-procedure intubation and intensive-care unit stay, mainstem bronchus occlusion secondary to mucous plugging requiring clearance by bronchoscopy, and post-obstructive pneumonia. Eventually, she was transferred back to the original facility with a possible superimposing fungal infection for which she was started on treatment and a possible diagnosis of one of the following rare conditions: congenital pulmonary airway malformation (CPAM), fibrosing mediastinitis, or extra lobular sequestration.

Discussion: The receipt of any positive result on a screening test can have negative psychosocial effects on patients and, as demonstrated in this case, can lead to arguably avoidable physical complications. This risk of overdiagnosis should be part of the discussion regarding whether to screen. Clinicians should be cognizant of the significance of screening tests when planning subsequent workups. On the other hand, holding one of the rare diagnoses can affect plans for treatment, surveillance, and prognosis which could justify the extensive workup done despite the not uncommon complications the patient suffered from during her unexpected long hospital stay.
Median arcuate ligament syndrome (MALS), also known as Celiac artery compression syndrome is clinically characterized by triad of weight loss, postprandial abdominal pain and sometimes abdominal bruit. It is more common in women between the ages of 40 and 60s. Exact mechanism is unknown and MALS is diagnosis of exclusion.

42-year-old female with PMH of Chronic shoulder pain and chronic intercostal who presented with chronic recurrent diarrhea and chronic recurrent postprandial abdominal pain that resulted in significant weight loss in 2 to 3 weeks. Extensive work-up including lactate, lipase, stool studies, EGD, colonoscopy was unremarkable. Biopsies from EGD showed chronic duodenitis. Patient’s previous CT abdomen pelvis showed focal narrowing of 50% celiac artery origin at diaphragmatic crus so CTA abdomen pelvis was done which showed 60 to 70% stenosis of celiac artery during expiration suggestive of median arcuate ligament syndrome. Gastric emptying study showed significant gastroparesis. Patient was started on TPN due to poor oral intake. General and Vascular surgery recommended follow-up in outpatient to do further evaluation for median arcuate ligament syndrome.

Inspiratory and expiratory Vascular imaging (CTA/MRA) with worsening of stenosis on expiration is used as definitive diagnostic criteria for MALS. Celiac artery decompression is mainstay of treatment in symptomatic patients in which laparoscopic approach is preferred over open surgery. Persistent or recurrent symptoms are managed with percutaneous or surgical revascularization. Gastroparesis has also been reported in patient with MALS.
Introduction: Anti-Synthetase Syndrome (ASS) is a rare, multi-systemic autoimmune entity which can manifest with myositis, arthritis, Raynaud’s phenomenon, and interstitial lung disease. ASS is supported by a positive anti-Jo1 antibody and muscle biopsy. Many patients with Anti-Synthetase Syndrome suffer from additional rheumatologic disorders, including Rheumatoid Arthritis (RA). RA can also present with ILD, which can create a complicated picture for a patient suffering from an overlap syndrome of both disorders.

Case Presentation: A 60-year-old male presented to the office after an abnormal chest CT.

A year prior to presentation, the patient was suffering from DIP/PIP swelling, and dry, flaky skin. He was diagnosed with Anti-Synthetase disorder after presenting with the classic “Mechanic’s hands” dermatitis specific to the diagnosis of ASS. His antibody panel was positive for elevated Anti-Jo1 >8.0, elevated rheumatoid factor at 82, and CCP antibodies at 120. His CT chest showed subpleural reticulation and ground glass opacities, suggesting ILD. The patient was started on Mycophenolate Mofetil and was advised to follow up with Pulmonology for continued work-up. He eventually returned with increased joint swelling and stiffness. After thorough review of previous testing, the patient was diagnosed with an overlap syndrome: Rheumatoid Arthritis and Anti-Synthetase Syndrome. A high-resolution CT of the lungs was repeated, due to worsening SOB and medication non-compliance, which showed a progressive pulmonary fibrosis with usual interstitial pneumonia (UIP). This UIP pattern is seen in both RA and ASS, and importantly, in an overlap syndrome of both disorders.

Discussion: Although both syndromes can independently cause ILD, this case shows a rare overlap of two separate rheumatologic disorders resulting in a devastating pulmonary outcome. Both disorders cause a similar UIP picture in this patient. Anti-Synthetase Syndrome, albeit a rare disorder, can be hidden behind the common diagnosis of RA.
**Objectives:** Physicians struggle to diagnose Alzheimer’s disease in its early stages. The forefront of Alzheimer’s disease pathology and research is based on the amyloid hypothesis, which has faced criticism in recent years. Its debate has led researchers to consider other avenues of pathogenesis. For this retrospective study, the aim is to determine if the baseline concentration of a novel Alzheimer’s disease biomarker, symmetric dimethylarginine, is as effective as a baseline plasma biomarker concentration of amyloid β-peptide 42, the current standard, in discriminating prodromal Alzheimer’s disease patients from normal controls.

**Methods:** To investigate our hypothesis, a total of 146 prodromal Alzheimer’s disease patients and 108 controls were analyzed. Demographic data, magnetic resonance imaging, fluorodeoxyglucose-positron emission tomography scans, and plasma biomarker concentrations of symmetric dimethylarginine and amyloid β-peptide 42 were obtained from the Alzheimer’s Disease Neuroimaging Initiative database. Multivariable regression analyses were conducted to determine if either biomarker could discriminate between patients that will develop Alzheimer’s from those that will not.

**Results:** Both amyloid β-peptide 42 (p= 0.0751) and symmetric dimethylarginine (p=0.891) levels showed no statistical difference in being able to discriminate between control and prodromal patients. Age was found to be a confounding variable for symmetric dimethylarginine in both control (r=0.400, p < 0.00001) and prodromal Alzheimer’s disease patients (r=0.387, p < 0.00001), as well as for amyloid β-peptide 42 in both control (r=0.155, p=0.0549) and prodromal Alzheimer’s disease patients (r=0.214, p-value=0.0262). Associations between biomarkers and magnetic resonance imaging and fluorodeoxyglucose-positron emission tomography scans were only significant for amyloid β-peptide 42 in control patients.

**Conclusions:** We were unable to find evidence to support the usage of symmetric dimethylarginine as a novel biomarker predictor for Alzheimer’s disease pathogenesis as a single measurement, though amyloid β-peptide 42 was also nondiscriminatory. These results confirm with other investigations that there is insufficient evidence in utilizing baseline plasma amyloid β-peptide 42 in the diagnosis and risk assessment of Alzheimer’s disease as a single measurement.
Introduction: Systemic lupus erythematosus (SLE) is a chronic autoimmune disease with multi-organ involvement. SLE activity involving GI system is relatively rare but can be severe and life-threatening. Here we present a case of Lupus enteritis manifesting as acute abdominal pain, nausea, vomiting and diarrhea in a patient with previously diagnosed SLE.

Case Presentation: 29 y/o female, diagnosed with SLE in 2012 when she presented with polyarthralgia, myalgia, fatigue, occasional fevers, photosensitive rash consistent with Lupus on biopsy, positive ds-DNA, leukopenia and hypocomplementemia, has been on Hydroxychloroquine and Mycophenolate since 2012. 4 months ago, she presented with acute abdominal pain associated with nausea, vomiting and diarrhea along with flare up of photosensitive rash. Workup revealed leukopenia, elevated CRP, hypocomplementemia, new onset proteinuria, hematuria and characteristic CT findings of bowel wall thickening and edema. EGD with biopsies revealed active duodenitis. Her clinical presentation in the setting of underlying SLE raised a strong suspicion for Lupus enteritis and she was treated with high dose steroid therapy, resulting in rapid clinical response and complete resolution of symptoms.

Discussion: Lupus enteritis is a rare and severe complication of SLE. Clinical picture is non-specific with abdominal pain being cardinal symptom and no single biologic finding can be considered pathognomonic for Lupus Enteritis. CT scan has become gold standard test with characteristic finding of “Target sign”. Our patient most likely had Lupus enteritis based on clinical presentation and CT findings. Lupus enteritis is typically steroid responsive with excellent prognosis but failure to treat early could lead to life-threatening complications. Continued investigations into the pathogenesis and treatment of SLE related GI involvement are needed to improve mortality and morbidity outcomes for patients with SLE.
Introduction: Heparin-induced thrombocytopenia (HIT) is a life-threatening complication of heparin exposure. Reports have established a HIT-mimicking thromboembolic disorder without prior heparin exposure, known as Spontaneous HIT syndrome. Here we present a case of acute thrombocytopenia and b/l adrenal hemorrhage presenting 1 week post total knee arthroplasty (TKA).

Case: 70 y/o male who underwent left TKA 1 week ago and discharged on aspirin postoperatively for DVT prophylaxis, presented with acute abdominal pain, N/V. Of note, he received Moderna vaccine 4 weeks prior and had no previous heparin exposure. Workup revealed acute thrombocytopenia with platelets at 93K and CT evidence of b/l adrenal hemorrhages followed by subsequent diagnosis of acute LLE DVT and b/l PE. IVC filter was placed prophylactically. Next day, platelets dropped down to 35K, had positive HIT antibodies and positive Serotonin Release Assay (SRA). Based on above presentation, spontaneous HIT was suspected as he did not have prior heparin exposure except receiving 1 heparin flush during IVC filter placement. He was treated with Bivalirudin and IVIG.

Discussion: Joint replacement surgery, particularly TKA, has been reported to be a common trigger for spontaneous HIT, where exposure to knee cartilage glycosaminoglycan could possibly trigger anti-PF4/heparin antibodies resulting in platelet activation, although pathophysiology remains unclear. Recently, COVID19 adenoviral vector vaccines have been associated with Vaccine-induced immune thrombotic thrombocytopenia (VITT) presenting as thrombocytopenia, atypical thrombosis, positive PF4 dependent enzyme-immunoassays, similar to spontaneous HIT. Our patient had acute thrombocytopenia and thrombosis with positive HIT antibodies without prior heparin exposure, presenting 1 week post TKA, 4 weeks post Moderna vaccine, raising suspicion for spontaneous HIT vs VITT. Continued investigations are required to understand the pathogenesis. Treatment includes Non-heparin anticoagulation and IVIG.
Covid-19 has had a significant impact on society, the economy, and even our approach to medicine. Although the pandemic has challenged the globe, vaccines have been reducing the transmission and symptomatic impact of Covid-19, providing hope for a return to normalcy. Vaccines are a safe and recommended approach to combatting the pandemic, but nevertheless do not come without the potential for adverse reactions and outcomes as discussed here.

A 36-year-old man with a history of tobacco use and prediabetes presented to the emergency department with diaphoresis, headache, and substernal chest pain one day after receiving the second Moderna COVID vaccine. On physical exam, the patient was in no acute distress with regular peripheral pulses, clear lung sounds, and no abnormalities noted on cardiac auscultation. EKG revealed ST segment elevations in anterolateral leads with bedside point of care ultrasound showing apical wall motion abnormalities. Baseline high-sensitivity troponin returned at 500, with a peak of >2500 and resolution thereafter. Subsequent coronary angiography demonstrated non-obstructive coronary arteries however mid-LAD was noted to have sluggish TIMI 3 flow. LV angiogram confirmed apical hypokinesis raising concern for Takotsubo cardiomyopathy. Patient was admitted for further work-up and management. Cardiac MRI completed the following day interestingly revealed subepicardial to mid-myocardial delayed gadolinium hyperenhancement extending from the anterolateral wall to the inferolateral wall and apex, consistent with myocarditis. Patient was discharged on baby aspirin, metoprolol succinate 25 mg and lisinopril 5 mg. He was given nicotine replacement therapy and advised to follow-up with repeat MRI. Five weeks after discharge, follow-up cardiac MRI showed near resolution of myocardial inflammation, noting basal subepicardial delayed enhancement in the same distribution as prior. Patient continues to follow with outpatient cardiology and has had minimal symptoms since this time.

This case illustrates the potential development of acute myocarditis status-post COVID vaccination. Although there are many viral etiologies of myocarditis, the patient in this case had no previous exposure outside of his recent vaccination. Additionally, it was initially thought that this patient had Takotsubo cardiomyopathy given consolation of findings on invasive cardiac imaging. However, there was significant, patchy enhancement seen on CMR which is consistent with myocarditis. As clinicians, it is important to recognize this potential side-effect from COVID vaccination, particularly in those with more severe cardiac histories and comorbid factors.
Among the thrombophilias, Factor V Leiden (FVL) is the most common, seconded by a mutation in prothrombin 20210 (PTM). Carriers of both FVL and PTM are called compound heterozygotes; the prevalence is rare, 1:1000 in the general population and 1-5% among DVT patients. Compound FVL/PTM heterozygotes have an estimated 3-4 times higher risk of initial VTE than heterozygous carriers of either FVL or PTM. Low prevalence renders population-level studies difficult, hindering clinical understanding of the disease and identification of therapeutic targets.

Our patient was a 40-year-old man with a history significant for superficial venous thrombosis and varicose veins, who underwent left saphenous vein radiofrequency ablation in 2015. In December 2020 he developed erythema, warmth, and swelling in his left lower leg, with pain behind the knee and heaviness in his hip. He had no recent surgeries, hospitalizations, travel, or immobilization. Doppler ultrasound revealed an age-indeterminate occlusive thrombus within a duplicated mid-femoral vein, and superficial thrombophlebitis of the calf. He was initiated on apixaban and referred to Hematology.

Given his lack of identifiable DVT risk factors, young age, and new femoral DVT, a hypercoagulable workup was done, revealing compound heterozygosity for FVL and PTM. Indefinite long-term anticoagulation with apixaban was recommended; repeat ultrasound showed a resolving femoral thrombus. He was also counseled on the risk of inheriting both FVL and PT mutations in 1st-degree relatives.

Of FVL patients, 8-14% have a coexisting inherited thrombophilia, such as a protein C or antithrombin deficiency. In homozygous FVL patients, the risk of clotting is increased up to 80-fold from the normal population. Among PTM homozygotes, the estimated risk of VTE is about 33%. In compound FVL/PTM heterozygotes, thrombosis risk is 3-4 times higher than those with a single variant. These patients, especially with VTE risk factors such as obesity, immobilization, or pregnancy, are usually prophylactically anticoagulated. Diagnosis is imperative, as patients should be on long-term anticoagulation. Future studies are needed on the efficacy of various anticoagulants in compound heterozygotes, and to determine if there is a potential indication for genetic screening in families for VTE prevention.
42) DIAGNOSIS OF UNCOMMON ADAMANTINOMA IN OLDER PATIENTS
Henok Hardilo, MD; Chiamaka Ogemdi Isiguzo, DO; Biana Leybishkis, MD
Aurora Health Care Internal Medicine Residency Program, Milwaukee, WI

Introduction:
Adamantinoma is a very rare, low-grade, primary malignant tumor of the bone, with an indolent course affecting mainly the long bones. It accounts for only 0.1-0.5% of all primary bone tumors.

Case Presentation: A 70-year-old female patient presented to the clinic with progressive, sharp (9/10 severity), non-radiating, left mid leg pain over two weeks duration. The pain was aggravated by weight bearing and improved with rest. The patient denied other symptoms or similar episodes in the past. She had a past medical history of chronic pain syndrome and osteoarthritis. On physical examination, she appeared in distress from pain. There was point tenderness on the left upper tibia with localized swelling. X-ray showed a large 5.6 cm x 3cm x 2.3 cm. destructive bone lesion involving the proximal tibial diaphysis. MRI showed a minimally displaced pathologic fracture through the mass. A total body PET scan revealed intense uptake in the left tibial lytic lesion with no additional areas of abnormal uptake. Fine needle aspiration biopsy of the lesion was consistent with adamantinoma. The patient underwent a successful left tibia wide excision and intramedullary rodding with left lower extremity reconstruction. Follow up imaging after several months revealed stable results and no further recurrence.

Discussion: Careful history taking, physical examination and appropriate imaging are vital for early diagnosis and effective treatment of uncommon diseases, such as adamantinoma. This case report demonstrates that in patients with preexisting chronic pain and osteoarthritis, consideration of uncommon diagnosis is very important for early detection and successful treatment.
**Introduction:** Nonbacterial thrombotic endocarditis (NBTE) is an uncommon condition, usually arising in the setting of a hypercoagulable state, typically cancer or systemic lupus erythematosus. Unless embolization occurs, NBTE is often clinically silent. Vegetations in NBTE are often found on an undamaged mitral or aortic valve.

**Case:** An 86-year-old woman with a history of insulin-dependent diabetes, hypertension, chronic kidney disease, vascular disease including cerebrovascular accident, hyperlipidemia, coronary artery disease status post coronary artery bypass graft, carotid endarterectomy, and pacemaker placement for sick sinus syndrome presented complaining of headache and dizziness. Medications included digoxin, clopidogrel, metoprolol, rosuvastatin, and insulin. Except hypotension (77/44 mmHg), the patient’s vitals were within normal limits. Physical exam was significant for a mid-systolic murmur and a clean-appearing ruptured right tympanic membrane. Head and neck CT scans showed chronic total occlusion of the left vertebral artery, bilateral moderate to severe subclavian artery stenosis, post-surgical changes from prior carotid endarterectomies, and no acute pathology. Complete blood count was unremarkable; basic metabolic panel showed baseline elevations of creatinine and glucose consistent with her chronic diseases. High-sensitivity troponin was mildly positive on admission; it was trended and remained unchanged. Chest X-ray, electrocardiogram, and pacemaker evaluation were unremarkable. A normal saline bolus and aspirin were administered for hypotension and positive troponins; blood pressure quickly normalized. Two blood cultures were drawn, and one grew S. hominis. Echocardiography showed a normal ejection fraction, patent foramen ovale (PFO) with left-to-right shunt, poly-valvular regurgitation, and a linear density of 2.1 by 0.5 cm on the mitral annulus concerning for vegetation. Vancomycin infusion was started for presumed infectious endocarditis. A second and third set of blood cultures remained negative. Another echocardiogram showed no changes. Originally cultured *S. hominis* was attributed to contamination; vancomycin infusion was halted, and the patient was discharged. At follow-up, additional blood cultures were negative and echocardiography redocumented the vegetation without significant change.

**Discussion:** This case illustrates the potential for culture-negative valvular vegetation without underlying hypercoagulable state. It is unclear what role vascular disease, dehydration, and PFO played in the etiology of the vegetation, although previous reports describe endovascular injury as a precipitating factor in NBTE. Endothelial injury in vascular disease is well-characterized, but less is known of cardiac remodeling due to endothelial damage in endocarditis. NBTE should be considered in cases of sterile endocarditis where there is significant endovascular disease.
Introduction: Chronic eosinophilic pneumonia is an idiopathic disease first described in 1969 and is characterized by dyspnea, alveolar infiltrates, and eosinophilia.

Case Presentation: This is a 19-year-old female with a history of vaping and marijuana use who presents with two months of progressive shortness of breath, chest tightness, and intermittent urticaria. She was started on albuterol and fluticasone inhalers as well as cetirizine, without significant improvement in her symptoms. After one month, a CBC w/ differential and chest x-ray were performed which demonstrated peripheral eosinophilia of 35% with an absolute eosinophil count of 5,030 and a focal nodule airspace opacity, respectively. A CT chest was performed one week later, which showed scattered tree-in-bud nodules superimposed on patchy areas of groundglass attenuation with evidence for air trapping within the lung bases. Pulmonary consultation was obtained and bronchoscopy was performed which demonstrated a normal airway exam other than mild erythema throughout. Bronchoalveolar lavage lavage demonstrated 80% eosinophils and she was diagnosed with chronic eosinophilic pneumonia (CEP). She was started on 40 mg prednisone daily and continued on fluticasone and albuterol inhalers with significant improvement in her respiratory symptoms. After six weeks of therapy, CT chest demonstrated near complete resolution of groundglass opacities. CBC w/ differential demonstrated improved but ongoing peripheral eosinophilia with 5% eosinophils (absolute eosinophil count 740) after three months of steroid therapy and 5% eosinophilia (absolute eosinophil count 590) after five months of steroid therapy. Her clinical course has been complicated by significant weight gain, but her respiratory symptoms have improved. Steroids have been tapered to 15 mg daily, with plans to continue prolonged taper over 9-12 months since diagnosis unless side effects prove intolerable, in which case a biologic such as mepolizumab or benralizumab will be considered.

Discussion: Even in the midst of a respiratory pandemic, and especially in the setting of peripheral eosinophilia, clinical suspicion must remain high to promptly diagnose CEP. The mainstay of treatment for CEP is corticosteroids and patients typically have rapid and dramatic improvement in their symptoms.
Introduction: The immunomodulating properties of cytomegalovirus (CMV) are well-described. We report a case of CMV infection in a patient with a prior heart transplant complicated by invasive pulmonary aspergillosis (IPA).

Case Presentation: This is a 68-year-old female with a history of non-ischemic cardiomyopathy status-post heart transplant two years prior to presentation (on immunosuppression) who presented with a three-week history of shortness of breath, fatigue, cough, and diarrhea. On physical exam, she was afebrile, normotensive and tachycardic to the 120s, with saturations in the low 90s on 6L oxygen via nasal cannula (NC). Her pulmonary exam was notable for crackles in the bilateral bases. Her abdomen was soft and nontender to palpation. Labs demonstrated WBC 2.3, hemoglobin 9.1, and platelets 119. Chest CT demonstrated diffuse cellular bronchiolitis pattern, most consistent with infection. A bronchoscopy was considered, however progressive hypoxia and escalating high flow nasal cannula (HFNC) requirement precluded this at the time of initial consult. Ultimately, CMV PCR returned positive, initially at 114,815 copies and peaking at 954,993 copies. She was started on IV ganciclovir. Despite this treatment, her respiratory status worsened and she required 60L 100% FiO2 via HFNC. Sputum fungal culture then grew aspergillus fumigatus consistent with IPA and she was started on posaconazole. She initially improved and was able to tolerate 3-4L via NC. However, her supplemental oxygen requirements again increased to 6L and imaging demonstrated enlarging consolidations. Bronchoscopy at that time revealed thick mucus plugging in the left lower lobe and bronchoalveolar lavage was notable for a positive galactomannan index (1.3). She was discharged on IV ganciclovir infusions (with a prolonged valganciclovir course to follow) for primary CMV, and posaconazole for 6-12 months pending clinical improvement for IPA.

Discussion: Co-infections of the immunosuppressed patient can cause significant morbidity and mortality. Increased prevalence of viral, fungal, and bacterial infections is particularly well documented in patients with CMV infection. Prompt diagnosis and appropriate treatment are paramount.
Head and neck abscesses may present insidiously as simple infections such as acute otitis externa. A seventy-one-year-old female with severe depression requiring vagal nerve stimulator, tobacco use, and recent tooth extraction presented to the ED with worsening left-sided otalgia, hearing loss, tinnitus, headache, and new localized facial edema despite recent outpatient treatment for otitis externa with topical antibiotics and steroids. Exam was significant for left-sided erythematous facial edema and exquisite pain to palpation of the left temporomandibular joint (TMJ). Lab studies showed leukocytosis and elevated inflammatory markers. CT imaging indicated otomastoiditis, left TMJ septic arthritis, developing subdural empyema, and possible osteomyelitis of the skull base. She was started on broad-spectrum antibiotics. Attempts to aspirate the TMJ were unsuccessful though a biopsy of necrotic tissue was obtained, and a tooth was also extracted. Interval MRI showed tissue changes concerning for a developing temporal lobe abscess, which was surgically drained. Otolgia, trismus, and facial edema improved throughout this time, though hearing loss persisted, and she developed new aphasia thought due to associated vasogenic edema. Temporal abscess cultures were negative, left otorrhea culture grew 1+ *Streptococcus constellatus*, and left perimandibular cultures grew 1+ *Streptococcus anginosus*. Antibiotics were narrowed from cefepime, vancomycin, and metronidazole to ceftriaxone, which was continued outpatient for a total antibiotic course of thirty-eight days from abscess drainage with subsequent MRI showing decrease in size of postoperative cavity. Residual sequelae nine months later included mixed hearing loss and arthrosis of the left TMJ. Persistent worsening of an initially simple-appearing infection should prompt further investigation and consideration of patient’s history and risk factors, in this case recent dental extraction and ongoing cigarette use. In otitis externa, severe pain correlates with infection severity. Close proximity of anatomic spaces involving the oral cavity, ear, and skull base should guide both investigation and ultimate selection of antimicrobials for an intracranial abscess such as the one in this case.
Stress cardiomyopathy is an acute variant of dilated cardiomyopathy with underlying pathophysiology triggered by intense stress or emotional experiences. In the immediate setting, it may initially masquerade as acute coronary syndrome (ACS). A sixty-eight-year-old female was at the hospital bedside of her recently deceased husband when she became acutely short of breath with associated severe chest pain. She was found to be hypoxic requiring 10LNC, and hypertensive. Initial high sensitivity troponin and NT-pro-BNP were both significantly elevated. A subsequent EKG showed ST elevations in V2 and V3 with bedside ultrasonography demonstrating global wall motion abnormalities. Medical management for ACS was started, and she was intubated for acute hypoxic respiratory failure. Transthoracic echocardiogram demonstrated severe LV systolic dysfunction with wall akinesis and hypokinesis localized to apical and distal left ventricle. Emergent right heart catheterization showed diffusely elevated left- and right-sided filling pressures with reduced cardiac index, and left heart catheterization showed no evidence of obstructive CAD. She was admitted to the ICU where she required initial diuresis as well as two days of vasopressor and inotropic support. Course was complicated by tracheobronchitis requiring antimicrobial treatment. Within several days, cardiac index and filling pressures measured by Swan-Ganz catheter improved dramatically. Serial EKGs demonstrated persistent QT prolongation and T wave inversions, which improved over time as well. Repeat TTE at a three-month interval demonstrated LVEF of 74% and completely recovered systolic function. Stress cardiomyopathy remains a unique pathologic entity among acute cardiovascular insults that is managed supportively to apprehend any complications related to myocardial stunning, which overlap significantly with those of ACS. As in this case, broadly elevated filling pressures and absence of significant obstructive CAD virtually cement the diagnosis, though the initial presentation and history are usually suggestive. QT prolongation is a characteristic and increasingly recognized sequel that should be monitored closely as some patients, especially with incomplete functional recovery, may develop acquired long QT syndrome putting them at greater risk for sudden arrhythmia.
Introduction: Multisystem Inflammatory Syndrome (MIS-A) is a rare complication in adults involving systemic inflammation manifesting in the respiratory, gastrointestinal, renal, cardiovascular, and dermatological symptoms. Since 2020, there has been a greater frequency of clinical cases highlighting the association of MIS-A with the COVID-19 pandemic. Here we present a case of a young man with MIS-A associated with COVID-19.

Case: A 28-year-old male patient with a COVID-19 diagnosis from a month prior and no PMHx was transferred for non-ischemic cardiomyopathy and HF with reduced EF of 30-35%, leukemoid reaction, and concern for MIS-A. The leukemoid reaction is presented as erythematous patches on the bilateral axilla, hips, and lateral upper legs; biopsy was consistent with viral exanthem and suspected inflammatory response resulting from leukocytosis. Dermatology and infectious disease were consulted.

Lab examinations revealed elevated levels of ferritin (1558 ng/mL); procalcitonin (3.49ng/mL); D-dimer (1.44 mg/L); high sensitivity troponin (182 ng/ml); ALT (122 unit/L); CRP (10.3mg/dL). CBC indicated elevated WBC count and marginally lower Hb. SARS-CoV-2 Abs was reactive, disqualifying this patient from convalescent plasma therapy and remdesivir administration. IV antibiotics were initially given due to high procalcitonin but subsequently discontinued after ID evaluation. Cardiac MRI displayed diminished left and right ventricular systolic function, with left more acutely impacted, small pericardial and pleural effusion and consolidation in the left lower lobe, likely due to pneumonia. CTPE was negative for PE and without pulmonary edema. ECG indicated sinus tachycardia. Patient gradually improved resulting in discharge on day 3. Patient was also prescribed Lisinopril and Toprol for management of cardiovascular condition.

Discussion: This case provides insight for clinicians around the comorbidities associated with COVID. Mechanism of action of MIS-A is unclear. COVID-19 is often thought to be relatively asymptomatic amongst pediatric patients, however amongst adults, the symptomatic nature of a COVID-19 infection can cloak the advancement of other related ailments. MIS-A’s heterogeneous clinical presentation makes it acutely prone to underdiagnosis, especially within the context of a global pandemic. Furthermore, understanding the pathology fueling the association between MIS-A and COVID-19 has great potential of uncovering further details about the intricacies of the human immune system.
**49) CASE OF SPONTANEOUS CELIAC ARTERY DISSECTION**

Aashish Katapadi, MD; Louie Kostopoulos, MD

Aurora Health Care Internal Medicine Residency Program, Milwaukee, WI

**Introduction:** Spontaneous isolated celiac artery dissections (SICAD) are rare manifestations of non-atherosclerotic peripheral arterial disease with variable clinical presentations. Few previous literatures inform our current understanding of its history and management strategies. We present a case of a healthy middle-aged male without major risk factors diagnosed with SICAD and his clinical course.

**Case Presentation:** A previously healthy 46-year-old male with dyslipidemia presented to his primary care physician’s office with sudden-onset epigastric pain with diaphoresis and nausea, resolved prior to visit. After continued discomfort with empiric PPI therapy, he subsequently presented to the ED with severe pain after exercise. On presentation, BP was elevated, and he reported mild epigastric tenderness. EKG was unremarkable and labs noteworthy for mildly elevated CRP, fibrinogen, and D-dimer; lactate levels remained normal. Contrast-enhanced abdominal CT demonstrated non-atherosclerotic dissection with sub-total occlusion of the celiac axis extending into the hepatic and splenic arteries. He remained hemodynamically stable and DAPT and Heparin was started. He was also started on beta-blocker therapy. Arterial doppler study confirmed findings. Work up for autoimmune and connective tissue disorders were negative. He was discharged with limited exercise restriction, gradually lifted with close follow up and repeat imaging.

**Discussion:** SICAD is a rare cause of abdominal pain. Risk factors are equivalent to other types of dissection; observed incidence is greater in males. Clinical spectrum varies from completely asymptomatic to epigastric pain and nausea. Findings on either abdominal duplex ultrasound or CT angiography, often establish the diagnosis; definitive diagnosis is often established with invasive arteriography. In hemodynamically stable patients, a conservative approach with anti-thrombotic therapy and serial surveillance has been suggested, with potential endovascular repair. Unstable patients require urgent endovascular or open surgical repair. Our patient continues to do well with conservative management.
50) MDA-5 DERMATOMYOSITIS WITH SYSTEMIC LUPUS ERYTHEMATOSUS OVERLAP SYNDROME: A LITTLE KNOWN CLINICAL ENTITY
Jonathan Katz, MD; Sean O’Neill, MD
University of Wisconsin Department of Medicine, Madison, WI

Introduction: Dermatomyositis (DM) is an autoimmune condition characterized by inflammation of the skin and musculature leading to the development of dermatologic abnormalities and proximal symmetric weakness. While these manifestations are typical of classical DM, several DM disease phenotypes have been identified with the clinical variants associated with different autoimmune markers of the disease. Additionally, DM is known to present in conjunction with other autoimmune diseases leading to a variety of overlap syndromes.

Case: 38 yo Female G3P2 of Chinese descent is referred from rheumatology clinic to the ED with four months of hair loss, arthralgias, weakness, and weight loss. On exam, she was profoundly weak and her skin was notable for an erythematous flaky rash which had started on her face and slowly spread over her upper arms. Labs where notable for high ferritin, pancytopenia, elevated CK, AST, positive SSA, Smith, Ribonuclear protein, MDA-5 antibodies, low C3 and C4. She was started on high dose steroids with improvement and discharged home. Several weeks after her initial hospitalization, she returned to the ED following a witnessed seizure. She was febrile and hypoxic requiring intubation and transfer to the ICU. She received antibiotics, high dose steroids, and was started on intermittent plasma exchange with IVIG. Bone marrow biopsy obtained showed increased hemophagocytosis. Now meeting 5/8 criteria for HLH she was started on IV cyclophosphamide, anakinra and rituximab. Shortly after she began to show improvement in her C3/C4 levels as did her clinical status. Unfortunately, on hospital day 10 she became febrile and hypotensive, with cultures notable for candida and on HOD 16 she passed away.

Discussion: Of the various antibodies associated with DM, Anti-melanoma differentiation–associated gene 5 (MDA-5) was identified in 2005 and found to be associated with a unique form of DM characterized by a rapidly progressive interstitial lung disease. Since it’s identification, MDA-5 DM has been examined multiple times in the literature, however there are scarce reports of MDA-5 DM overlap syndromes. Due to this paucity, little is known of the natural disease course, complications or optimal treatments for these syndromes. Here we present a case of Refractory MDA-5 DM with Systemic lupus erythematosus overlap syndrome complicated by rapidly progressive ILD and macrophage activation syndrome. This case serves to add to the growing body of literature around this little-known clinical entity.
51) SHOOTING SUBOXONE: SHOOTING YOURSELF IN THE FOOT AND THE HEART

Kidus Kebede, MD; Betelhem Yifra, MD; Colleen Nichols, MD
Aurora Health Care Internal Medicine Residency Program, Milwaukee, WI

Introduction: The United States is experiencing an unprecedented opioid pandemic. Several placebo-controlled studies document the general efficacy of buprenorphine for opioid use disorder. Suboxone, the combination of buprenorphine and naloxone, is a unique formulation designed to prevent intravenous or intranasal abuse of buprenorphine.

Case Presentation: A 34 year old female presented with fever, chills, and skin rash of 2 weeks duration. She has history of heroin use disorder but has been clean and maintained on Suboxone for the last 4 years. She is happily married and works as a consultant for a multimillion-dollar company.

Initial lab evaluation showed markedly elevated ESR of 128 CRP of 16.6. Blood culture showed Pseudomonas Aeruginosa, and patient was started on cefepime and ciprofloxacin. Her echocardiogram showed vegetations involving the aortic valve with mild to moderate aortic insufficiency. She underwent minimally invasive bioprosthetic aortic valve replacement via hemi sternotomy. The source for Pseudomonas Aeruginosa was suspected to be a right hip wound. Patient later reported injecting Suboxone for years over the right hip wound. MRI of the hip showed no evidence of osteomyelitis or abscess. The patient remained in the hospital to finish her IV antibiotic course at which point ID transitioned her to oral ciprofloxacin to continue for an additional week. Her PICC line was removed, and she was discharged in stable condition.

Discussion: This case illustrates the complex nature of addiction. We need to further understand the neurobiological process and take advantage of this knowledge to test new targets for therapy. The fight against addiction can only be won when our strategies reflect the full complexity of the disease.
We report the case of an 83-year-old female with a past medical history of type 2 diabetes mellitus, thyroid cancer, chronic lymphedema, hypertension, hyperlipidemia, congestive heart failure and chronic kidney disease (CKD) who presented to the emergency department with a 2–3-month history of left leg pain. She noted that the interior aspect of her left thigh had been erythematous, irritated and painful for the past few months. She also had a 5-6 cm wound with eschar formation in the same area. Of note, our patient had recurrent admissions for treatment of cellulitis of her left thigh over the last 3 months. Her initial workup consisted of a doppler ultrasound of the lower left extremity which was negative for deep vein thrombosis. X-ray of the left femur showed non-specific fat stranding in the soft tissues. She then had a CT performed on her left lower extremity which showed no subcutaneous free air. She was given a dose of linezolid in the Emergency Department and admitted for presumed cellulitis. The next hospital day, dermatology was consulted who performed a punch biopsy of the wound on the left thigh which showed epidermal and fat necrosis with thrombi and calcified elastic fibers, which is consistent with a diagnosis of calciphylaxis. She was started on treatment with sodium thiosulfate inpatient with directions to continue intravenous infusions on an outpatient basis. Throughout this hospital course, she also had recurrent hypocalcemia treated with supplementation with oral and IV calcium.

Calciphylaxis, also known as calcific uremic arteriopathy, is a rare disorder that is characterized by calcium deposition in arterioles leading to skin and subcutaneous ischemia and necrosis. It is mostly seen in patients with end-stage renal disease. Incidence in patients without CKD or End Stage Renal Disease (ESRD) has not been reported. A systemic review in 2008 found 36 cases of non-uremic calciphylaxis in the literature (1). Here we report another case of non-uremic calciphylaxis in a patient with presumed etiology of CKD. The classic presentation of calciphylaxis involves skin lesions frequently described as reticular purpura, plaques or nodules. Calciphylaxis commonly involves the cutaneous and subcutaneous layers of regions with more adipose tissue such as the thigh, as in our patient. These lesions progress quickly to ulceration with black painful eschar. The mainstay of treatment is wound care and metabolic control. Sodium thiosulfate is sometimes utilized as was in our patient, but the efficacy has not been clearly demonstrated. Morbidity and mortality are high in calciphylaxis, as these wounds heal poorly, and frequently become secondarily infected. Infection is the leading cause of death in these patients.
53) A CASE REPORT OF ERDHEIM-CHESTER DISEASE

Alexander Kerschner; Keowa Bonilla, MD
Medical College of Wisconsin, Milwaukee, WI

Introduction: Erdheim-Chester disease (ECD) is a rare disease with approximately 1500 published cases worldwide. This disorder is classified as non-Langerhans cell histiocytosis, a malignancy of myeloid progenitor cells. Mutation of BRAFV600E is present in most cases, activating the RAS/RAF/MEK signaling pathway resulting in inflammation, fibrosis, and organ damage. Clinical manifestations commonly involve the long bones, cardiovascular system, retroperitoneum, lungs, central nervous system, and skin.

Case Description: A 76-year-old female presented to the Emergency Department with diarrhea. Computed tomography imaging revealed enlarged bilateral kidneys with perinephric fat stranding, along with moderate pericardial and pleural effusions. A transthoracic echocardiogram the following month showed a large pericardial effusion with tamponade physiology prompting hospital admission. A pericardial window performed during admission revealed lymphohistiocytic infiltration consistent with ECD. A perinephric mass biopsy exhibited similar results consistent with ECD. A positron emission tomography scan showed hypermetabolic tissue along the aorta, pericardium, and kidneys. Lytic lesions were found in the sacrum and right humeral head. Magnetic resonance imaging of the brain showed punctate foci consistent with histiocytosis.

Discussion: This case provides additional data to assist with the diagnosis and clinical picture of ECD. Around 70% of reported cases are in male patients with an average age of 48-56 years and thus our patient falls outside the common demographics. ECD is a difficult diagnosis due to the complex involvement. It can be suspected in patients with unexplained bone pain along with associated systemic symptoms. Diagnosis is typically made on histopathology of cutaneous lesions; however, our patient lacked cutaneous symptoms necessitating an invasive biopsy. Molecular testing of BRAFV600E is another hallmark of disease for which our patient tested positive. Our patient was started on Vemurafenib, the mainstay treatment for BRAFV600E positive patients. There is currently no cure for ECD, but targeted therapies provide hope in slowing disease progression.
A 17-year-old male without any previous medical issues presents to the emergency department (ED) with bilateral leg pain and weakness. He had progressive lower extremity weakness for the last month without a known inciting event and for the past week he required the use of a wheelchair in school due to an inability to ambulate. On a detailed review of systems, the patient endorsed a five-pound weight loss and a non-painful, non-pruritic rash over the anterior aspect of his lower extremities bilaterally. The patient denied substance and medication use. He was otherwise developmentally normal and attended his assigned grade without additional assistance or an individualized education plan.

Physical examination revealed generalized tenderness to palpation of his deltoid and quadriceps muscles. He had 4/5 strength in bilateral deltoid, triceps, hip flexor, and knee extensor muscle groups. He had a slow, wide-based tandem gait. His upper and lower extremity reflexes and cranial nerves were intact. On skin examination there were scattered perifollicular petechiae on his bilateral lower extremities.

Initial laboratory studies included a microcytic anemia with a hemoglobin of 9.5 g/dL (normal: 12.5-16.1). TSH and ANA was within normal limits. Urine drug screen was negative for illicit substances. Initial imaging included bilateral hip and pelvic x-rays which were unremarkable. An MRI of the spine showed multifocal abnormal enhancing hyperintensities within the posterior spinous processes of the lower cervical, midthoracic, and lower lumbar spine as well as at the sacrum and sacroiliac joint. Dermatology performed a punch biopsy of the right lower extremity rash which revealed perifollicular extravasated erythrocytes and associated lymphocytes surrounding a somewhat tortuous hair follicle on histopathology.

A review of the patient’s dietary history was significant for a limited and restricted dietary intake to mainly processed carbohydrates. Further vitamin evaluation was obtained and resulted with a vitamin C level which was undetectable (normal: 0.2-2.1mg/dL).

Given the patient’s clinical presentation as well as imaging and histopathological skin biopsy findings in the setting of a known dietary restriction with low vitamin C plasma concentration, without other laboratory or imaging findings to suggest additional cause, the patient was diagnosed with scurvy secondary to an avoidant restrictive food intake disorder (ARFID) in an otherwise developmentally appropriate patient.
55) BULLET IN HEAD CAUSING LEAD ASSOCIATED PSYCHOSIS AND NEUROPATHY

Adam Koraym, BS; Pinky Jha, MD
Medical College of Wisconsin, Milwaukee, WI

Introduction: Elevated lead levels, even those below 5 μg/dL (50 ppb), impair cognition. Symptoms of mildly elevated lead levels include abdominal pain (“lead colic”), arthralgia, myalgia, irritability, and depression. Very high blood lead levels (>100 mcg/dL) present risks for more serious central nervous system effects such as encephalopathy (coma, seizures, delirium) as well as persistent cognitive impairment after recovery. Complete blood count (CBC) may be useful for patients with extensive lead exposure. There may also be basophilic stippling in patients who have been significantly poisoned for a prolonged period.

Case Description: A 25 Y male with no known past psychiatric medical history and recent history of a gunshot wound (1/15/21) admitted from a Detention Facility to Froedtert Hospital on 4/22/2021 with altered mental status. Over the next few weeks, the patient violated his parole and was detained, when corrections officers noticed that the patient had increasing agitation, aggression, suicidal ideation, and memory impairments in the 3 days prior to admission. Bedside officers reported that the patient was very paranoid the day prior to admission; while in CT room he was looking suspiciously around and appearing paranoid; officers had to reassure him for him to agree to scan. Per primary team, this morning patient briefly awakened and asked if provider was trying to kill patient. The patient was reporting having generalized pain in his muscles and joints, abdominal pain, and neuropathy bilaterally in his legs. Head imaging revealed bullet fragments embedded within the right temporal bone with trace middle cranial fossa pneumocephalus and artifact limited evaluation of the adjacent parenchyma.

Discussion: Even though this patient’s blood lead level is only 20, the manifestations are present, and given the proximity of the source of the lead to the central nervous system, it is not implausible that many of his symptoms are neurologic/psychiatric. Lead poisoning should therefore remain on the differential for patients with nonspecific, unexplained symptoms who have any retained bullets.
**Introduction:** Neurosarcoïdosis occurs in <10% of patients with sarcoïdosis and presents a diagnostic challenge given the risks associated with direct neural biopsy necessary for definitive diagnosis. Most patients will only have a probable or possible diagnosis of neurosarcoïdosis whenever extra-neural tissue diagnosis including from skin, lymph nodes, or lung is feasible alongside clinical improvement following steroid administration. Here, we describe a case of a rare acute-stroke like presentation of possible neurosarcoïdosis.

**Case:** A 59-year-old female with history of seizure disorder and alcohol abuse was hospitalized for amnesia following a house fire. On admission vitals were within normal limits (WNL) and there were no focal neurologic deficits. Laboratory results showed pancytopenia (hemoglobin 8.3 g/dL, leukocyte count 3,400 cells/µL, platelets 32,000 cells/µL). Carbon monoxide, TSH, B12, folate, and methylmalonic acid were WNL and urine drug screen was negative. CT head showed no acute findings. Continuous EEG started on admission was unremarkable. Two days into admission, the patient suddenly developed dysarthria, aphasia, and left upper extremity and bilateral lower extremity weakness. Stat CT head showed no acute abnormalities, but repeat continuous EEG showed multiple focal subclinical seizures. Levetiracetam was started. Brain MRI showed subtle acute vs. subacute infarcts and leptomeningeal enhancements. Further stroke workup was unremarkable. Infectious workup including CSF studies was negative. ANA screen by indirect immunofluorescence was positive (1:160, speckled pattern), however, solid phase ANA assay and complement levels were normal. SLE was ruled out. CT chest/abdomen/pelvis showed enlarged calcified mediastinal and hilar lymph nodes. Methylprednisolone was started for presumed neurosarcoïdosis with significant improvement in mentation. Hilar lymph node biopsy demonstrated rare possible granuloma formation with no fungal or acid-fast bacilli; thus, the patient’s final diagnosis was possible neurosarcoïdosis.

**Discussion:** Granulomatous inflammation of the perivascular distribution in the brain can cause vasculopathy or seizure as was noted in our patient. Although acute stroke-like symptoms are very rare and seizures are present in only 14% of patients with neurosarcoïdosis, it is important to include neurosarcoïdosis in the differential diagnosis as the treatment is different.
Introduction: Dermatomyositis (DM) is a rare inflammatory myopathy characterized by symmetric proximal muscle weakness and cutaneous manifestations. Despite the 6-fold higher risk of malignancy, there is no consensus on how aggressively cancer screening should be pursued for DM patients.

Case: The 83-year-old male patient with a history of DM, CAD, and prostate cancer presented with a one-week history of dyspnea on exertion. CT angiography showed pulmonary emboli and a large right pleural effusion. Therapeutic anticoagulation was started. The patient underwent a thoracentesis with removal of 1.9L of exudative pleural fluid. Cytology showed primary lung adenocarcinoma indicating Stage IV lung cancer. Due to reaccumulation of fluid, a PleurX catheter was placed.

The patient was diagnosed with DM six months prior. At that time, he had been following up with a pulmonologist for two pulmonary nodules (in RUL and LLL), first noted almost two years prior. Image-guided biopsy of the LLL abnormality was recommended nearly a year prior but the patient elected to monitor radiographically. Follow-up imaging nine months prior showed a slight increase in size of the LLL density and decrease in size of the RUL density, and a repeat CT chest was scheduled. At the time of DM diagnosis, no further cancer workup was pursued, and a six-month follow-up CT chest was recommended.

Of note, the patient was found to have positive TIF1-gamma antibodies. Anti-TIF1-gamma is seen in 18-23% of patients with DM and is associated with a positive predictive value of 58% and a negative predictive value of 95% for cancer-associated DM.

Discussion: After DM diagnosis, our patient did not undergo adequate cancer screening, including aggressive follow-up on previously noted lung lesions that could have led to an earlier diagnosis of his cancer. This case highlights the importance of a thorough cancer workup in DM patients with positive TIF1-gamma antibodies. How aggressively cancer screening should be pursued for DM patients is up for debate. A guideline with recommendations for cancer screening after DM diagnosis is needed, especially as most malignancies present within two years of DM diagnosis.
Introduction: Pancreatic Panniculitis is a rare skin manifestation presenting with many deep, red migratory nodules, mainly on the lower extremities. Etiology is unclear yet correlates with inflammation and elevated pancreatic enzymes. Here we present a case of panniculitis associated with pancreatitis.

Case: A 63-year-old female with PMHx of HTN, chronic pancreatitis with pseudocyst, SMV thrombosis, gastric outlet obstruction and alcohol use disorder was transferred for loose non-bloody stools and progressive nodular pruritic and tender skin lesions of the bilateral upper and lower extremities with serosanguinous discharge. The lesions were purple, 4-cm long by 2-cm wide with red halos. Bactrim, vancomycin 500mg, piperacillin and tazobactam displayed minimal improvement and tissue biopsy was consistent with pancreatic panniculitis. Blood examinations showed elevated serum amylase and lipase levels of >3000 UI/L; hemoglobin level of 9.4 g/dL with a MCV of 94 fl. CT pancreas showed no signs of acute pancreatitis. ERCP was performed and pancreatic sphincterotomy and pancreatic duct stent was placed. Consequently, amylase and lipase fell to the 400s. Status post, patient was stable. A day later, the patient was declining, displaying hypotension down to 70s systolic and hypoglycemia. The patient was afebrile, tachycardic and saturating in the 90s. Blood cultures were positive for E. coli, yielding broad spectrum antibiotics. Patient was provided supportive care and on day 12, patient’s health deteriorated and went into PEA; patient was pronounced deceased.

Discussion: This case aims to raise awareness amongst clinicians to consider panniculitis as a manifestation of pancreatitis. The release of pancreatic enzymes may be involved in increasing the micro-vascular permeability allowing hydrolysis of neutral fat. The resultant glycerol and fatty acids lead to fat necrosis, resulting in erythematous and edematous lesions. The pathogenic role of pancreatic lipase is supported by the finding of the enzyme and anti-lipase monoclonal antibodies within the necrotic tissue. Kin lesions precede the symptoms of underlying diseases and the interval between cutaneous findings and discovery of abdominal disorders goes from 1 to 7 months, an early and proper diagnosis being thus crucial. A deep skin biopsy that includes the subcutaneous tissue is mandatory for the diagnosis. Clinically, it is often difficult to make a differential diagnosis between the various types of panniculitis: erythema nodosum, lupus panniculitis, panniculitis in sarcoidosis, and erythema induratum/nodular vasculitis.
Introduction: Aspergillus is an opportunistic fungus that demonstrates septate hyphae with acute angle branching. A brain abscess due to Aspergillus can be due to direct or hematogenous spread from a local or distant site of infection. In this case specifically, a frontal and ethmoidal sinus infection likely led to direct invasion of the frontal lobes.

Case: A 73-year-old female with a past medical history of poorly controlled Type II Diabetes Mellitus and arthritis treated with steroids. Three months prior to admission, she developed copious, thick, yellow-green nasal discharge, headaches, and right eye blindness. She traveled from Mexico to Milwaukee since these symptoms persisted, and upon arrival was admitted. CT head scan without contrast showed a likely brain abscess located in the anterior cranial fossa associated with bilateral frontal bones and ethmoid plate, imaging also demonstrated opacification of both frontal sinuses. To remove the abscess, a bicoronal bifrontal craniotomy with pericranial flap was performed. CALCOs from the surgical abscess were 1/4 positive for septate hyphae concerning for Aspergillus. The patient was treated with antifungals – amphotericin and voriconazole – which she would continue for 3-6 months post-surgery.

Discussion: Here we report a case of a bilateral frontal brain abscess due to Aspergillus. The abscess likely developed due to the longstanding history of sinusitis and immunosuppression from steroid use. The patient’s presentation first was thought to be solely due to sinusitis (three months of nasal drainage and headache), however following additional imaging elucidated brain involvement. As the CT scan showed, an infection in the frontal/ethmoidal sinuses can spread to the frontal lobes, either by direct or hematogenous spread. Brain abscesses due to Aspergillus are rare and an example of an opportunistic infection. This cause should be included in the differential diagnoses in patients presenting with longstanding severe sinusitis symptoms, especially in those with current or past history of immunosuppression (which is a well-known risk factor for opportunistic infections). It has also been reported that patients with hyperglycemia (such as from poorly controlled diabetes) show increased rates of mortality due to brain abscesses. This highlights another important reason to keep Aspergillus brain abscesses on the differential in a patient with a history of poorly controlled type 2 diabetes mellitus, as seen here. This case demonstrates a unique intertwining of the risk factors of immunosuppression and hyperglycemia in the development of an Aspergillus brain abscess, and the importance of not ruling out potential diagnoses simply because of their rarity.
Introduction: Frontotemporal dementia (FTD) is a neurocognitive disorder with a spectrum of distinct disease states with behavioral, cognitive, speech, and motor features. We present a case of new amyotrophic lateral sclerosis (ALS) in a patient with an established diagnosis of behavioral variant-FTD representing a refined diagnosis of frontotemporal dementia with motor neuron disease (FTD-MND).

Case Description: A 71-year-old man with bv-FTD presented to clinic for evaluation of new falls. The first reported symptoms of emotional outbursts, flawed decision making, and sexual addiction occurred in 2001, while neuropsychological testing and imaging made a formal diagnosis in 2016. His current behaviors are controlled on risperidone 1mg BID. Over the past eight months, he went from regular bike rides on his own to requiring a walker for ambulation. He had multiple falls at his care facility over the past two months. On exam, cranial nerves were intact, with no tongue atrophy or fasciculations and preserved vertical gaze. Shoulder girdles, calf, and intrinsic hand and feet muscles were atrophied. Ankle dorsiflexion strength was 2/5 on left and 3/5 on right, while rest of strength testing was normal. Fasciculations were visible in deltoid, pectoralis, biceps, and thigh muscles. The triceps reflex was 3+ with trace ankle jerks while other reflexes were normal. Steppage gait and left foot drop were present. He was unable to walk on heels or toes. These findings are new compared to prior. Sensation and coordination testing was normal. Electromyography results revealed both acute and chronic denervation across various muscle groups. The exam and EMG confirmed ALS. Two months after presentation to clinic, he was confined to a wheelchair.

Discussion: This case shows the complexity of diagnosing dementia-related diseases, specifically within the FTD spectrum. Diagnostic features may take time to reveal themselves which can delay appropriate treatment. The FTD spectrum consists of bv-FTD, primary progressive aphasia, corticobasal syndrome, progressive supranuclear palsy, and FTD with motor neuron disease (FTD-MND). Each of these sub-diagnoses has unique clinical features, however there is overlap as well. This makes accurate diagnostic criteria critical. Ultimately, this patient was diagnosed with FTD-MND after many years of an established diagnosis of bv-FTD. Regular screening for features of the specific sub-diagnoses within the FTD spectrum may achieve an accurate diagnosis and appropriate treatment earlier.
61) A CASE OF SECONDARY HYPERTENSION: MISSED MORE OFTEN THAN NOT IN PRIMARY CARE

Kirsten Lipps, MD; Christine Kolehmainen, MD, MS; Laura Phillips, MD

University of Wisconsin Hospital and Clinics, Madison, WI
William S. Middleton Memorial Veterans Affairs Hospital, Madison, WI

**Introduction:** Approximately 45 percent of adults in the United States have hypertension, and nearly one-half of these individuals have elevated blood pressure despite treatment. Primary aldosteronism is a common secondary cause of elevated blood pressure, with an estimated prevalence of 5 to 10 percent in hypertensive populations. This increases to 20 percent in patients with resistant hypertension. However, primary aldosteronism is underdiagnosed in primary care, which contributes to excess cardiovascular and cerebrovascular morbidity and mortality.

**Case:** A 65 year-old woman with hypertension and obstructive sleep apnea presented to primary care with long-standing resistant hypertension despite adherence to three anti-hypertensive medications. The patient was started on chlorthalidone and developed severe hypokalemia and metabolic alkalosis. Review of the electronic health record revealed that the patient experienced severe hypokalemia in setting of thiazide diuretic initiation ten years earlier. Evaluation for primary aldosteronism revealed plasma aldosterone/renin ratio 40.7 (reference range [ref.] 0.9 – 28.9). Serum renin activity was normal, albeit at the lower limit. Primary aldosteronism was suspected, and the patient completed a confirmatory 3-day oral sodium load. Day 3 urine aldosterone was 9.5 mcg/24 hours (ref. < 12 mcg/24 hours). This was non-diagnostic due to the presence of serum hypokalemia, which suppresses aldosterone secretion and may result in a false negative. The patient was presumed to have primary aldosteronism, and computed tomography of the abdomen revealed bilateral adrenal adenomas and unilateral adrenal hyperplasia. The patient declined further workup with adrenal vein sampling and was initiated on medical therapy with spironolactone.

**Discussion:** Current guidelines recommend screening for primary aldosteronism in specific populations, including adults with resistant hypertension, hypertension and spontaneous or diuretic-induced hypokalemia, and hypertension and obstructive sleep apnea. Yet, it is estimated that only 2 to 13 percent of eligible individuals are properly evaluated. Improving adherence to screening guidelines for primary aldosteronism in the primary care setting is essential to not only appropriately manage hypertension but also to mitigate excess risk of cardiovascular and cerebrovascular disease, which is independent of blood pressure control.
62) HIGH-INTENSITY VERSUS MODERATE- OR LOW-INTENSITY STATIN THERAPY FOR PRIMARY PREVENTION OF CARDIOVASCULAR DISEASE IN HIGHER-RISK ADULTS: THE MULTI-ETHNIC STUDY OF ATHEROSCLEROSIS (MESA)

Kirsten Lipps, MD; Yacob G. Tedla, PhD; Adam D. Gepner, MD
University of Wisconsin Hospital and Clinics, Madison, WI

**Background:** Statin therapy is a cornerstone of atherosclerotic cardiovascular disease (ASCVD) prevention. In contrast to strong evidence supporting high-intensity statins for secondary ASCVD prevention, the importance of statin intensity for primary prevention is not well understood. This study evaluated the association of statin intensity with the development of ASCVD in a multi-ethnic population of adults at higher risk of CVD.

**Methods:** Total CVD and coronary heart disease (CHD) events were prospectively evaluated in MESA participants who were free from CVD at baseline, at higher risk for CVD, and on statin therapy during the study (2000 – 2017). Mutually exclusive groups were Diabetes Mellitus (DM), High-risk (Pooled Cohort Equation (PCE) estimated 10-year ASCVD risk > 20%) without DM, and Intermediate-risk (PCE 10-year ASCVD risk 7.5 ¬– 20%) without DM. The association of statin intensity (high vs. moderate/low) with total CVD and CHD events was analyzed using Cox regression, accounting for time-varying covariates of cardiovascular risk factors, use of non-statin lipid lowering medications, and field center.

**Results:** We included 2,288 adults (53% female, 55% non-white), who were a mean (standard deviation) age of 68.5 years (9.1) in the analysis. At baseline, 31% had DM, 30% were High-risk, and 39% were Intermediate-risk. During follow-up, 255 participants used high-intensity statin at any time point. High-intensity statin therapy was not associated significantly with lower risk of total CVD or CHD events in any higher-risk group as compared to moderate-/low-intensity statin.

**Discussion:** To our knowledge, this is the first study to evaluate the association of statin intensity with the development of ASCVD. Our results indicate that in adults at higher-risk of ASCVD, high-intensity statin therapy was not associated with lower risk of total CVD or CHD events, as compared to moderate- or low-intensity statin. This study was limited by small numbers of events in all higher-risk groups. However, the direction of hazard ratios consistently suggests CVD benefit with high-intensity statin therapy. Further research, with a larger cohort, will help establish the association of intensity of statin therapy with development of ASCVD.
Introduction: Patients with acute encephalopathy, especially when unaccompanied, leave clinicians with little to no medical history. This case illustrates the importance of keeping a wide differential early on with concomitant thorough analysis of objective findings in patients with acute encephalopathy.

Case: An 87-year-old female with history of grave’s disease status-post thyroid ablation and neurocognitive deficit was brought in by her son for acutely altered mental status (AMS). Her son briefly stated he found her slumped over on a toilet with black stool inside before having to leave.

On admission, the patient was hypotensive to 80’s over 40’s, tachypneic between 26-32 but saturating 98% on room air and afebrile. She was disoriented to time, person, and place with slurred speech. Lungs were clear. She had mild lower abdominal tenderness and frank melena on rectal exam. Labs were significant for leukocytosis 13.4, hemoglobin 10.4 (baseline 9’s), Creatinine 1.2 (baseline 0.5), bicarbonate 14, anion gap 23, and lactic acid 4.1. Urine drug screen and CT head were unremarkable. CT abdomen/pelvis revealed acute uncomplicated sigmoid diverticulitis. 1 unit packed red blood cells and IV fluids were given. Cefepime and Flagyl were started.

Unfortunately, she continued to have tachypnea despite clear lungs and normal saturations. Venous blood gas (VBG) revealed pH of 7.52, pCO2 of 21, and Bicarb of 17. Salicylate level was markedly elevated at 66.8. Patient was transferred to the ICU and sodium bicarbonate drip was started. Once family was finally reached, they confirmed they found a nearly empty bottle of aspirin next to the patient. This was likely an accidental overdose in response to her abdominal pain.

Discussion: Without a history, metabolic disturbances, and etiologies such as aspirin overdose can go overlooked. This patient had exaggerated tachypnea for the degree of sepsis; VBG confirmed respiratory alkalosis which triggered the aspirin level. When patients have an anion gap metabolic acidosis, salicylate levels must be obtained for rapid treatment to improve mortality and reduce hospital length of stay.
The Moderna COVID-19 vaccine is a recently approved vaccine to prevent COVID-19 infections and complications.

**CASE 1:** An 86-year-old woman with no known neurological nor psychiatric history was hospitalized 7 days after receiving her first Moderna COVID-19 vaccine administration. She had suddenly developed acute confusion with visual hallucinations and left frontal headache two days before admission. The patient had never used tobacco, alcohol, nor any other illicit drugs. On admission, her vital signs were within normal limits (WNL). There were no focal neurological deficits (FND) noted. Laboratory studies were significant for inflammatory markers. Metabolic and infectious workup were nonexplanatory. CSF studies and appropriate head imaging were non-explanatory. Continuous EEGs demonstrated non-convulsive focal status epilepticus treated with Lorazepam and Fosphenytoin and with Levetiracetam on discharge. 1 month after hospitalization, she was seen in an outpatient neurology clinic with return to baseline neurologic status.

**CASE 2:** A 73-year-old man with a history of Crohn’s and no known neurological history nor psychiatric history was hospitalized 21 days after receiving his first Moderna COVID-19 vaccine. He was recently hospitalized for 2 days following 10 days of staring episodes, restlessness, and cognitive deficits that started 7 days after his Moderna COVID-19 vaccine. The patient had never used tobacco and does not use any other illicit drugs. The only new medication was a prednisone 40 mg taper that was started 5 weeks ago for a Crohn’s flare. Prednisone was discontinued at the prior 2-day hospitalization. While at home, he developed hallucinations and worsening confusion. On admission 2 days later, all vital signs were WNL. There were no FND. Laboratory studies were unremarkable except for a mild AKI. Metabolic and infectious studies were nonexplanatory.

CT head and MRI brain showed no acute findings. Continuous EEG demonstrated non-convulsive status epilepticus, which was treated with Lorazepam and Levetiracetam loading and maintenance. 2 months after hospitalization, the patient was seen remotely in outpatient neurology clinic. The patient and wife stated he was at baseline neurological status.

**Discussion:** Seizures as a complication of vaccinations have been reported before. This is the second report of non-epileptic seizures after COVID-19 vaccine.
Background: Two-thirds of clinical trials never meet their enrollment goals. Not only do patients lose the opportunity to undergo life-altering therapies, but also patients with lower socioeconomic status or without access to large academic medical centers are disproportionately impacted. In particular, African American patients are 5.7 times more likely to die from COVID-19 in Wisconsin, while making up less than 20% of major clinical trial cohorts. Concurrently, the prominence of COVID-19 trials has revealed a need to address gaps in knowledge about the benefits and risks of participating in experimental treatments; the urgency to engage patients in clinical trials has never been greater. Our team seeks to develop an anonymous web-based search tool for patients to discover clinical trials for COVID-19 with unintimidating language adapted by healthcare professionals.

Methods: After observing many physician-patient trial recruitment conversations, we compiled the information that patients most often request to make an informed decision. Information from selected clinical trials was extracted from the clinicaltrials.gov database. Complex concepts, such as mechanism of action and prior clinical safety data, were distilled into a unique library of easily understood concepts, completely eliminating medical jargon. We created a simple search engine website and distributed it using social media.

Results: At the peak of the COVID pandemic, the website received 2,400 and 2,800 views in the months of April and May of 2020, respectively. Of the individual study pages, information regarding hydroxychloroquine studies and plasma donation received the most unique views. No identifying information was collected to protect patient privacy.

Conclusion: A patient-centered clinical trial recruitment strategy can alleviate disparities in clinical trial recruitment. We identified privacy and ease-of-use as pillars to developing a best-in-class solution. Continuing to engage patients and tracking the impact of our website remains a challenge with an anonymous platform.
66) A CASE OF TRICKY PLEURAL FLUID
Maxwell Machurick, MD; John McCartney MD
University of Wisconsin Hospital & Clinics, Madison, WI

Introduction: Pleural fluid cytology is a commonly used test to assess for the presence of malignancy in pleural effusions. Recent studies have found the sensitivity to be between 60-70% for all malignancies and up to 80% in adenocarcinoma. However, as with all objective testing, this data must be interpreted in the appropriate context of the patient’s presentation and associated clinical findings. This component is critical for accurate diagnosis, patient management, and avoidance of medical errors.

Case: The patient is a 60-year-old male with 35 pack-year smoking history who presented to the emergency department with increased cough and chest discomfort over several weeks. Chest imaging revealed complicated left pleural effusion and he underwent thoracentesis and chest tube placement. Fluid was neutrophilic, exudative, and with a low glucose concerning for empyema; 16S ribosomal DNA was positive for Prevotella orvis. With the history of poor dentition and known reflux disease, the patient was felt to have an aspiration pneumonia with subsequent anaerobic empyema. The patient improved with IV antimicrobial therapy and was discharged after with plans for an extended course of antibiotics and follow-up imaging. Shortly after discharge, cytology from the original pleural fluid returned positive for small cell carcinoma. Meanwhile, he continued oral antibiotic therapy and was no longer suffering from fevers, chills, or night sweats. A repeat chest CT was performed approximately one month after initial presentation and demonstrated resolving mediastinal lymphadenopathy and only minimal nodular densities in the left lower lobe consistent with resolving infection. Bronchoscopy and left lower lobe tissue sampling were performed but did not reveal any evidence of malignancy. As the pleural cytology result did not seem consistent with the clinical presentation, Pathology compared the two samples and molecular analysis from the small cell cancer cytology did not match the specimens collected during bronchoscopy. It is thought the original cytology results represented contamination from specimen handling in the lab and had no other connection to our patient.

Discussion: This case demonstrates the importance of interpreting test results in context of the patient’s presentation and other objective findings. The incidence of false positive pleural fluid cytology is exceedingly rare, with estimates around 0-1%. Though the process of clarifying these results required multiple testing modalities, we were ultimately able to demonstrate a processing error that led to a “false positive” cytology result.
There is a high need for compassionate care for critically ill COVID-19 patients, their loved ones, and front-line caregivers, especially those requiring ECMO. Patients afflicted with severe COVID-19 infection on ECMO suffer from difficult symptom burden including anxiety, dyspnea, and loneliness that requires multi-modal management with both pharmacologic and non-pharmacologic efforts. Family members and healthcare team members witnessing and participating in caring for these patients often experience feelings of helplessness, anxiety and depression. Rather than providing a concert of familiar songs, a music vigil offers a quiet space for reflection, rest and, sometimes, for finding meaning. In March of 2020, we recognized the need for this service within the COVID-19 patient population at Froedtert Hospital, Milwaukee, WI. A group of harpists, highly trained palliative musicians, meets virtually together weekly to discuss the possibility of bringing virtual music sessions to ICU COVID-19 patients. The harpists came from all across the United States, Canada, and Australia, and many have decades of experience offering live music in medical settings. These palliative musicians work in the field of music thanatology which is music that is prescriptive for the patients’ physiologic needs or stressors, many of which are external factors beyond their control. In August of 2020, a plan for offering a harpist on-call Monday through Friday to play music sessions for patients with COVID-19 was brought to the nursing leadership team of the Cardiovascular ICU (CVICU) and gained approval for pilot. A secure virtual platform that worked for both the musicians and the clinicians was identified and a process for obtaining consent. A daily communication process of the on-call harpist for the day contacting the CVICU Nurse and obtains consent to set up the music session on the IPAD at the bedside. In our experience of over 130 sessions, music delivered via an electronic device can address the issue of reaching critically ill, isolated patients with a non-pharmacologic symptom management modality that has the potential to improve their experience of anxiety, dyspnea and loneliness, as well as to bring comfort to family members who may be present and the staff caring for them. Musicians who may be very remote to the physical location of the patient can be brought to their bedside through the creative use of a secure, virtual platform. Music can benefit critically ill patients, their loved ones, and the staff who care for them. Given the virtual nature of the sessions, this type of non-pharmacologic intervention has the potential to be reproducible at other medical institutions and clinical settings.
68) A RARE CASE OF SEVERE HYPERNATREMIA INDUCED
RHABDOMYOLYSIS

Dawit Major, MD; Mekbib Onkiso, MD; Lamya Boujelbane, MD
Aurora Health Care Internal Medicine Residency Program, Milwaukee, WI

Introduction: Electrolyte abnormalities are a prominent feature of rhabdomyolysis, and some electrolyte disorders can be a cause of rhabdomyolysis. Rhabdomyolysis caused by severe hypernatremia (>160 mmol/l) was reported only in few case reports.

Case Presentation: We present a 35-year-old female with PMHx of developmental delay, blindness, and Type II DM (Diabetes Mellitus) presented with fever, fatigue, and lethargy.

Per caregiver, patient had decreased oral intake of 5 days duration. No vomiting or diarrhea reported. On physical exam, patient was severely dehydrated and lethargic. PR 114/min, BP 114/63 mmHg and temperature 37.4 C. Labs were remarkable for sodium 190 mmol/l, potassium 3.8 mmol/l, creatinine 1.75 mg/dl, WBC 14K, platelet 79K, creatinine kinase 12,700 IU/l, serum osmolality 425 mOsm/kg, and urine osmolality 499 mOsm/kg. Blood glucose level in 500’s units with no metabolic acidosis, improved to 100’s - 200’s units after insulin administration. PT/PTT were normal and urine analysis was unremarkable. Patient was resuscitated with IV normal saline and D5W fluids with improvement of sodium to 160 mmol/l. 2 days after admission, CPK increased to 82,000 later to 174,000, accompanied by worsening renal function. Urine was dark. Hospital course was further complicated by acute hypoxemic respiratory failure and hypovolemic shock for which patient was sedated, intubated, and put on pressors. Need for hemodialysis initiation was discussed with family. However, the family opted for comfort care. Patient extubated and subsequently expired with the patient’s family at bedside.

Conclusion: This case demonstrated severe hypernatremia causing rhabdomyolysis, subsequently leading to multiple organ failure and death. The exact mechanism is unknown but thought to be due to hyperosmolar state which impairs sodium calcium transport, causing an activation of protein kinases, subsequently leading to muscle lysis/rhabdomyolysis.
This report examines the case of a 61-year-old man who was admitted to the hospital with three weeks of progressively worsening leg swelling and dyspnea on exertion. Physical exam on presentation was significant for jugular venous distention to the patient’s mid-neck, crackles at the bilateral lung bases, and 2+ bilateral lower extremity pitting edema. Laboratory workup revealed nephrotic range proteinuria, an albumin of 2.0 g/dL, and a persistent coagulopathy with an INR of 2.0 and PTT of 36 seconds. A CT chest with contrast revealed a single subsegmental pulmonary embolism as well as two enlarged lymph nodes flanking the common hepatic artery with presumed hemorrhage into the nodes. Further hematologic workup revealed normal PT and PTT mixing studies, a normal fibrinogen, and low factor X activity. Serum protein electrophoresis showed no monoclonal protein but revealed a kappa to lambda ratio of 2.63. The rest of his serology did not reveal any causes of nephrotic syndrome. He then received prothrombin complex concentrate to safely undergo a kidney biopsy and endoscopic ultrasound with fine needle aspiration of his abdominal lymphadenopathy. His kidney biopsy revealed AL kappa restricted amyloidosis and his lymph node biopsy was negative for lymphoma. He then underwent a bone marrow biopsy which showed a positive congo red stain with 3.6% kappa restricted plasma cells. He recently started treatment with daratumumab, cyclophosphamide, bortezomib, and dexamethasone (Dara-CyBorD) with the hope of an autologous stem cell transplant in the future.

This case provides an example of how amyloidosis can cause both nephrotic syndrome and factor X coagulopathy as well as how to approach its workup. It also highlights emerging therapies for amyloidosis with Dara-CyBorD. Lastly, it emphasizes the importance of maintaining a broad differential diagnosis, as this patient presented with signs and symptoms that suggested heart failure.
70) SYSTEMIC SCLEROSIS, SEVERE MALNUTRITION, AND SMALL BOWEL OBSTRUCTION: WHY TOTAL PARENTERAL NUTRITION SHOULD BE AN EARLY CONSIDERATION

Ben Massat, DO; James McCarthy, MD
Medical College of Wisconsin, Milwaukee, WI

Systemic sclerosis can cause vascular endothelial damage and fibrosis involving nearly all aspects of the gastrointestinal track. This can lead to esophagitis, gastroparesis, small bowel dysmotility, small intestinal bacterial overgrowth, chronic intestinal pseudo-obstruction, and malnutrition among other complications. We present a case in which a 62-year-old woman with a history of diffuse cutaneous systemic sclerosis developed severe gastrointestinal involvement, leading to multiple small bowel obstructions and severe malnutrition. Our patient’s case is unique as she suffered from two separate instances of mechanical small bowel obstruction without volvulus, rather than pseudo-obstruction as is typically seen in systemic sclerosis patients. Maintaining adequate nutrition also proved extremely difficult due to our patient’s candida esophagitis, gastroparesis, and small bowel dysmotility. She could not tolerate nasogastric feeds and developed her second small bowel obstruction shortly after starting nasojugal feeds. Ultimately, shortly after starting total parenteral nutrition, our patient developed pulmonary edema and died shortly after going into pulseless electrical activity cardiac arrest.

This case underscores the importance of evaluating for true small bowel obstruction in patients with systemic sclerosis prior to treating for pseudo-obstruction. It also demonstrates that in patients with nutritional deficiencies secondary to systemic sclerosis with gastrointestinal involvement, early initiation of total parental nutrition should be strongly considered as it may be difficult to maintain adequate nutritional support with enteral feeds alone given the ability of systemic sclerosis to adversely affect nearly all areas of the gastrointestinal tract. This case also shares experience that may influence clinical practice in a population of patients difficult to study in formal clinical trials.
**Introduction:** Pernicious anemia is a rare autoimmune disorder characterized by destruction of gastric parietal cells or antibodies against intrinsic factor. This glycoprotein is essential for vitamin B12 absorption in the terminal ileum. As a result, this condition leads to B12 deficiency and can manifest as ineffective erythropoiesis and/or myelin synthesis.

**Case:** A 49-year-old male with history of coronary artery disease, ischemic cardiomyopathy, aortic stenosis, and COPD presented with profound fatigue, dyspnea, abdominal discomfort, and bilateral foot numbness. Initial labs were notable for WBC 1.7 K/uL, MCV 113 fL, Hgb 4.9 g/dL, Plt 79 K/uL, LDH 3808 U/L, haptoglobin <8 mg/dL, and absolute reticulocyte 10 K/uL. Peripheral blood smear revealed hypersegmented polymorphonuclear leukocytes without circulating blasts. His severe pancytopenia with macrocytic anemia was concerning for a myelophthisic process such as acute lymphoma or myelodysplastic syndrome with acute myelofibrosis. Bone marrow biopsy was performed, and aspirate pathology was interpreted as “markedly hypercellular marrow with trilineage hematopoiesis showing marked erythroid hyperplasia and striking megaloblastic features, 3% blasts; findings consistent with megaloblastic anemia.” Subsequent measurement of vitamin B12 level was <170 pg/mL; folate was within normal limits. Isolated B12 deficiency led to the discovery of positive intrinsic factor antibodies, consistent with a diagnosis of pernicious anemia. He was started on subcutaneous B12 1,000mcg daily injections and counseled on the importance of screening for GI malignancies with EGD and colonoscopy. Several months after initiation of a B12 supplement, his symptoms had completely resolved with normalization of blood counts.

**Discussion:** Presentations of low vitamin B12 can range greatly from asymptomatic to life-threatening anemia, pancytopenia, or neuropathy/myelopathy. Investigation of B12 and folate should occur in the evaluation of pancytopenia to prevent unnecessary and invasive diagnostics. Pernicious anemia is one of the most common causes of severe B12 deficiency. This disorder is associated with a significantly increased risk of concomitant autoimmune conditions, gastric adenocarcinoma, carcinoid tumors, and myeloid malignancies. Benefits of endoscopic surveillance in this population have not been established; however, it is generally recommended to perform endoscopy within 6-12 months of diagnosis due to the elevated incidence of malignancy.
**Introduction:** Nephrotic syndrome is a disorder of the kidney with a broad range of etiologies including infectious, neoplastic, cytokine mediated, idiopathic, and amyloid deposition. Traditionally, nephrotic syndrome presents with proteinuria and edema. It may be complicated by hypogammaglobulinemia and hypercoagulability due to serum protein loss. When amyloidosis is the cause of nephrotic syndrome it is typically due to a plasma cell dyscrasia, malignancy or a chronic inflammatory state.

**Case:** A 61-year-old male with a history of diabetes mellitus, diabetic gastroparesis, and hypertension presented for shortness of breath, worsening abdominal pain, and progressive swelling in his abdomen and legs over months. He was recently discharged from an outside hospital, where he was diagnosed with gastroparesis and noted to have an elevated INR. At intake, he was afebrile and his vital signs were stable. On initial exam, he was noted to have pitting edema of his lower extremities, abdominal distension, and periumbilical ecchymosis. A contrast enhanced chest CT demonstrated a subsegmental pulmonary embolism. CT abdomen/pelvis demonstrated a large soft tissue density, presumed to be a hemorrhagic lymph node, which was similar in size to a mass seen six days prior on a CT scan obtained at the outside hospital. Anticoagulation was held due to concern for intraabdominal bleeding. Urinalysis was ordered and revealed significant urine protein, raising concern for nephrotic syndrome. Bleeding workup showed an INR of 2.0. Further investigations during hospitalization included a renal biopsy which showed AL amyloidosis, an abdominal lymph node biopsy which showed hemorrhage, and a factor X activity of 8%, consistent with primary amyloidosis. The patient was discharged following improvement of his abdominal pain and swelling and advised to see a hematologist to undergo bone marrow biopsy to assess for hematologic malignancy.

**Discussion:** Here we report a case of amyloid nephropathy that presented with an anti-coagulated state secondary to acquired factor X deficiency, causing a hemorrhagic lymph node and physical exam findings consistent with retroperitoneal bleeding. Typically, a pro-coagulable state occurs during nephrotic syndrome. However, this case demonstrates the possibility of primary amyloidosis leading to concurrent factor X deficiency and amyloid nephropathy, resulting in a paradoxically increased bleeding risk in the setting of nephrotic syndrome. This presentation introduced a complex decision regarding use of anticoagulation, which was ultimately not used.
Non ischemic cardiomyopathy (NICM) is an interesting diagnosis as it provides a large differential and usually affects a younger patient population when compared to ischemic cardiomyopathy. There are a few medications that are typically associated with NICM including chemotherapy agents however atypical antipsychotics are a rare reported possible cause.

We present a case of a 46-year-old female with a past medical history of bipolar disorder, hypothyroidism, interstitial cystitis and IBS who presented to the Emergency Department (ED) with a chief complaint of a 1-month history chest pain and shortness of breath that has worsened the week prior to presentation. ED workup was unremarkable including a non-ischemic EKG with normal QTc, negative troponin, and normal chest radiograph. Patient was admitted to the general medicine floor and a transthoracic echocardiogram (TTE) was performed showing a moderately increased left ventricular cavity size with an ejection fraction of 30-35%. Patient denied any family history of NICM, CHD, or genetic disorders with cardiac defects. Denied history of any recent viral illnesses. Routine work up for NICM was negative including ferritin, ESR/CRP, hepatitis panel, rheumatoid factor, ACE level, ANA, aldolase, and urine drug screen. TSH upon admission was within normal range with stable levothyroxine dose. Ischemic evaluation with left heart catheterization was unrevealing for coronary artery disease. Cardiac MRI was unable to be obtained given patient had a bladder stimulator in place for her interstitial cystitis. Reviewing her home medications, patients psychiatric regimen included bupropion 450 mg daily, clonazepam 1 mg BID, lamotrigine 400 mg QHS, and quetiapine 600 mg QHS. Given patient’s negative work up, her quetiapine was weaned with the assistance of mental health. At her outpatient follow up 6 months later in addition to goal directed medical therapy her LVEF recovered to 46-51%.

Per literature review, there have been a few case reports of quetiapine associated cardiomyopathy with the common characteristics being women less than 50 years of age and medication dosage greater than 400 mg. Although a cardiac MRI was unable to be obtained, the leading hypothesis remains her cardiomyopathy was secondary to high dosing of quetiapine. As the atypical antipsychotics become more common in outpatient management of chronic disorders and inpatient use of delirium, this is a side effect that should have closer surveillance in the medical community.
74) TRANSCATHETER AORTIC VALVE REPLACEMENT FOR SEVERE AORTIC STENOSIS IN A TRANSPLANTED HEART
Fekadesilassie Moges, MD; Ankoor Biswas, MD; Suhail Allaqaband, MD
Aurora Health Care Internal Medicine Residency Program, Milwaukee, WI

Introduction: Heart transplantation has become the definitive treatment for end stage heart failure. As the long-term survival of heart transplant recipients is improved, the incidence of post-transplant valvular heart disease is growing. Tricuspid regurgitation is the most common valvular heart disease following transplant, while left-sided valvular disease is less frequent. Transcatheter aortic valve replacement (TAVR) has become an alternative to surgical aortic valve replacement in high-risk patients with severe aortic stenosis (AS).

Case Presentation: A 54-year-old patient with history of orthotopic heart transplantation 25 years prior for ischemic cardiomyopathy as well as kidney transplantation twice (30 years and 9 years prior) was admitted to the hospital with decompensated heart failure. Mild AS had first been noted 3 years prior. An updated echocardiogram revealed severe calcific AS with a valve area of 0.8 square centimeters. Aortic valve (AV) peak velocity was 4.6 m/s, with a mean gradient of 53 mmHg. Pre-TAVR cardiac computed tomography was avoided as the patient had chronic kidney disease stage 4 in a transplanted kidney. Instead, a preoperative transesophageal echocardiogram was utilized for valve sizing. The patient underwent transfemoral TAVR with a successful placement of a 29 mm Evolut PRO + valve. A month after the procedure, echocardiography revealed AV mean gradient of 10 mmHg and AV area of 1.9 square centimeters with trivial paravalvular regurgitation.

Discussion: Aortic valve stenosis is rare following cardiac transplantation. However, with improving long-term survival rates after transplant, AS may be seen with increasing frequency in the future. Our case demonstrates that TAVR is an effective and safe alternative in managing severe AS in a transplanted heart, even decades after transplantation.
**75) METASTATIC LUNG CANCER PRESENTING AS AN ACUTE ABDOMEN**

**Fekadesilassie Moges, MD; Ankoor Biswas, MD**

Aurora Health Care Internal Medicine Residency Program, Milwaukee, WI

**Introduction:** Lung cancer is one of the leading causes of cancer-related deaths in the world. A large proportion of patients present with metastatic disease and the predominant sites of spread include brain, liver, adrenal glands, and bone. Gastrointestinal (GI) metastasis from primary lung carcinoma is rare; the small bowel is most commonly involved. Furthermore, GI perforation as a result of metastatic lung cancer is extremely rare.

**Case Description:** A 58-year-old male patient with advanced non-small cell lung carcinoma (NSCLC) presented with sudden onset of diffuse abdominal pain, nausea and vomiting. The patient had been receiving Pembrolizumab as palliative immunotherapy for biopsy-proven metastatic lung cancer to the right iliac bone. On examination, he had involuntary guarding in the abdomen with rebound tenderness. Abdominal x-ray revealed large amount of free intraperitoneal air. He underwent emergent exploratory laparotomy.

A 2 cm x 2.5 cm perforation at the proximal jejunum with an adjacent mesenteric lymph node enlargement was found. A liter of intestinal contents was suctioned out and the perforated segment was resected with an adequate surgical margin, with an end-to-end jejunal anastomosis made to restore intestinal continuity. Histopathology of the jejunal specimen demonstrated metastatic poorly differentiated NSCLC which was similar to the findings from the fine needle aspirate of the right ilium soft tissue mass. The patient was transferred to the intensive care unit following surgery. His hospital course was complicated by aspiration of GI contents during intubation prior to the surgery. His condition improved gradually, and he was eventually discharged in stable condition.

**Conclusion:** Acute abdomen due to metastatic lung cancer is rare and contributes to a poor prognosis. Small bowel metastases should be considered in a lung cancer patient presenting with an acute abdomen.
Cardiac amyloidosis is a process in which abnormally folded proteins are deposited within cardiac tissue, leading to cardiac dysfunction as deposition progresses. While there are multiple etiologies for cardiac amyloidosis, deposition of the protein Transthyretin is becoming an increasingly recognized cause of amyloid cardiomyopathy. There are various forms of transthyretin amyloid cardiomyopathy (ATTR-CM), including hereditary forms as well as the more common wild-type form. With the rise of more accurate and non-invasive methods for diagnosis, ATTR-CM has been identified as more prevalent than previously thought. There are, however, various ‘red flag’ symptoms and other laboratory and imaging clues that should raise clinicians’ suspicion for ATTR-CM and even prompt more in-depth screening and investigation. As there is now an approved pharmacologic treatment agent for ATTR-CM, recognition and diagnosis of this disease is even more critical. In this case, an 84 y/o male with non-specific cardiorespiratory symptoms underwent in-depth workup with eventual diagnosis of ATTR-CM, and it provides a good example of how various non-specific clues that taken together should prompt a deeper investigation into possible ATTR-CM.
Introduction: The CDC recognized AIDS in 1981 when MSM contracted Pneumocystis carinii pneumonia and Kaposi sarcoma. Since then, more AIDS-defining illnesses have been described. The diagnosis of abdominal pain and diarrhea in newly diagnosed HIV presents a significant challenge.

Case Presentation: JM is a 31-year-old male who presented with a one-week history of worsening right lower quadrant abdominal pain and associated non-bloody diarrhea. He had no known medical history and a negative HIV test 3 years prior. CT revealed abnormal peritoneal thickening, mesenteric lymphadenopathy, and small bowel thickening. Enterotoxigenic Escherichia coli was found in the stool. HIV viral load was 70,143 copies/mL with an absolute CD4+ count of 172 cell/μL. A biopsy of an inguinal lymph node was unremarkable. Infectious disease initiated valganciclovir, ART, and a course of ciprofloxacin. His diarrhea resolved, and his abdominal pain waxed and waned until three months later when he returned to the hospital. Repeat CT showed worsening nodular disease. Liver biopsy confirmed the diagnosis of BL with c-MYC rearrangement. He has completed two cycles of DA-R-EPOCH without complication.

Discussion: ART has not decreased the incidence of BL making it the most common, 10-20% lifetime risk, NHL of ADC. The incidence of NHL has decreased by over 110-fold since the advent of ART. BL incidence is bimodally distributed decreasing when CD4+ cell count <50 cells/μL, unlike other NHLs whose incidence is conversely related to CD4+ count. BL presents earlier than other NHLs and has a rapid, one-to-two-day doubling time leading to rapid detection. In HIV-associated NHL, 30-50% of patients present with extra-nodal gastrointestinal involvement with more advanced Ann Arbor staging. This case demonstrates the importance of the following points: 1) The rapid progression of BL following HIV seroconversion, 2) The complexity in evaluating acute gastrointestinal complaints in a patient with HIV, recognizing ADCs and opportunistic infections.
It is very rare for disseminated histoplasmosis to present with gastrointestinal involvement without any pulmonary symptoms in the presence of advanced pulmonary histoplasmosis. Our patient is a 64 year old immunosuppressed female on methotrexate and Adalimumab for Rheumatoid Arthritis who presented with diarrhea. After other causes of diarrhea were ruled out, she underwent diagnostic colonoscopy which showed white patches in the colon, biopsy confirmed the diagnosis of colonic histoplasmosis. Interestingly, she later developed dyspnea with acute hypoxic respiratory failure. This prompted evaluation with a Chest CT. Chest CT showed bilateral pulmonary opacities. Urine was positive for histoplasma antigen and a diagnosis of disseminated histoplasmosis was made. She was initially treated with amphoteric B which was later switched to Itraconazole for one year. Due to varied and nonspecific presentation of disseminated histoplasmosis, the index of suspicion remains low with disseminated histoplasmosis being underdiagnosed, therefore, it is imperative for clinicians to keep histoplasmosis in the differentials for any immunocompromised patient presenting with diarrhea.
This is an interesting case of a 62-year-old male initially admitted with septic shock secondary to colitis. The patient remained encephalopathic following resuscitation so head imaging was obtained. CT and MRI demonstrated multiple intracranial lesions infiltrating the hypothalamus and corpus callosum. Lab evidence was consistent with secondary adrenal insufficiency, central diabetes insipidus, and central hypothyroidism. Endocrinology was consulted and hormone replacement started. Patient’s mentation slowly improved with hormone repletion. He underwent a brain biopsy with pathology coming back as Diffuse Large B-Cell Lymphoma (DLBCL). Hematology was consulted and workup revealed no evidence of DLBCL outside of the CNS. However, he was found to have an indolent peripheral chronic lymphocytic leukemia on peripheral smear and flow cytometry. Patient underwent high dose intrathecal methotrexate, cytarabine, and dexamethasone treatment prior to discharge. DLBCL accounts for 30-40% of adult Non-Hodgkin Lymphomas. However, primary CNS DLBCL comprises less than 1% of those cases. To have invasion affecting the pituitary axis is even more rare and has only been reported in a handful of cases. These cases are typically treated with high-dose intrathecal methotrexate and dexamethasone. Close endocrine evaluation and appropriate hormone replacement is also essential for these rare patients.
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**Introduction:** Diffuse large B cell lymphoma (DLBCL) typically presents as a symptomatic mass with majority of patients presenting with advanced stage DLBCL. Symptoms can be non-specific, and only 30% of patients have the classic systemic “B” symptoms of fever, weight loss, and night sweats. This case highlights the importance of high clinical suspicion and value of CT imaging to aid in the diagnosis of DLBCL.

**Case:** A 57-year-old male with a history of tobacco use presented with a 10-month history of chronic cough and a 3-day history of shortness of breath. He also reported occasional post-tussive emesis, dysphagia, and a 20-pound weight loss. Three months prior to the current presentation, his primary care provider ordered PFTs which showed a mixed restrictive/obstructive pattern; he was prescribed a combination LABA-ICS inhaler. One week prior to presentation, he returned to his primary care provider for progressive worsening of cough. A CT chest was ordered; however his dyspnea worsened prior to his scheduled CT scan, and he presented to the emergency department. On evaluation, he was afebrile, tachycardic, tachypneic, and hypoxic with SpO2 of 80% on room air. He was placed on CPAP with improvement of SpO2 to 98%. Physical exam was notable for diaphoresis and respiratory distress with accessory muscle use and diffusely coarse lung sounds. Laboratory evaluation revealed white cell count 14.1 K/uL, venous pH 7.28, and venous pCO2 58 mmHg. Chest radiograph showed increased mediastinal lymphadenopathy with mild mass effect on the trachea and a right lower lobe consolidation. CT chest showed extensive bulky lymphadenopathy and a large subcarinal mass with central gas and debris consistent with contained esophageal rupture as well as severe mass effect on the trachea, carina, and proximal airways. The patient was intubated and admitted to the ICU. He developed septic shock with mediastinitis requiring urgent thoracotomy, washout of the chest, and esophageal stent placement. Biopsy of the mediastinal mass was performed with pathology revealing a T-cell/histiocyte-rich large B-cell lymphoma. He improved clinically, was extubated, and transitioned to general care for chemotherapy initiation.

**Discussion:** DLBCL is an aggressive disease that often presents at an advanced stage. High clinical suspicion and early imaging are important when patients present with nonspecific symptoms secondary to mass effect, such as chronic cough. Earlier detection of disease helps reduce the risk of complications. To our knowledge, this is the first report of esophageal perforation as a complication of primary mediastinal DLBCL.
Introduction: Fanconi’s syndrome is by wasting of glucose and electrolytes that are typically reabsorbed by the proximal convoluted tubule (PCT) of the nephron. Since this portion of the nephron is so active, it is also prone to damage when its metabolic demands are not met. Here we describe a case of a man with a likely preexisting Fanconi’s syndrome who developed profound glucose and electrolyte wasting after receiving FOLFOX which is not associated with Fanconi’s syndrome.

Case description: An elderly gentleman with a past medical history of stage IV colon cancer presented with weakness and fatigue. Patient completed one cycle of FOLFOX approximately one week prior to hospitalization with oxaliplatin. Blood studies were significant for hypoalbuminemia at 1.7 g/dL, and normal electrolytes. Urinalysis had large amount of glucose and protein as well as elevated sodium and potassium. History was evaluated extensively for causes of Fanconi’s syndrome and was negative for lead poisoning, animal bites, or topic medications.

Conclusion: Adult–acquired Fanconi syndrome is a rare condition generalized by wasting of amino acids, glucose, phosphate, uric acid and ions from the proximal renal tubule. The diagnosis of Fanconi syndrome could be made by the findings of aminoaciduria, hypophosphatemia and findings of renal tubular acidosis (type 2). The pathophysiology of Fanconi syndrome lies in the defect in the PCT. While most cases of adult onset Fanconi’s syndrome is associated with monoclonal gammopathy, in our case there was no evidence of monoclonal gammopathy.
Diagnostic tests are typically used to confirm or refute clinical suspicion of disease but may be over-utilized due to several factors like widespread availability, convenience and patient expectations. Ulysses syndrome can be an effect of unnecessary investigations or incorrect interpretation of test results. Clinical estimation of pretest probability prior to diagnostic evaluation is valuable in avoiding this prevalent syndrome.

A 61-year-old female with a past medical history of hypertension, hyperlipidemia, type II DM and obesity presented to her PCP for a routine health visit. She complained of fatigue. She denied chest pain, shortness of breath, and any other cardiac symptoms. Her examination was unremarkable except for a grade 2/6 systolic ejection murmur at the apex. Her metabolic profile, thyroid function test, and EKG were unremarkable. Resting echocardiography did not reveal cardiac, valvular lesions. She was evaluated with a stress EKG with echocardiography due to her cardiac murmur and multiple risk factors. Rest and stress EKG showed no ischemic changes. Stress echocardiography showed normal global LV systolic function without wall motion abnormalities but the function did not increase with the stress. Due to this equivocal report, cardiac coronary angiography was done and showed non-obstructive calcified and noncalcified plaque in the proximal and mid LAD. As a result, a cardiac catheterization was done and revealed no hemodynamically significant CAD warranting intervention.

This middle-aged woman with multiple cardiac risk factors but no specific cardiac symptoms had a low pretest probability of significant CAD. As a result, further evaluation beyond medical management and risk factor modification was of low value. The unnecessary testing predisposed her to significant risks and increased healthcare costs. Determining her pretest probability of disease would have limited her diagnostic work-up and would have significantly reduced the risks and unwarranted costs.
COVID-19 is associated with systemic inflammatory response and coagulopathy which are oftentimes more prothrombotic than hemorrhagic. In severe cases, DIC can occur which can constitute a challenge in determining optimal anticoagulation strategies. TEG is a noninvasive test that measures the dynamics of clots formation, stabilization and dissolution and therefore gives an insight into patients’ hemostatic state which can effectively inform anticoagulation decisions.

68-year-old male with unremarkable past medical history was admitted to the medical ICU on account of acute hypoxemic respiratory failure from COVID-19 pneumonia. On initial admission, he had elevated inflammatory markers, normal platelets, fibrinogen and an initial elevated D-dimer of 3927ng/mL. Chest CT angiogram was negative for PE but revealed multifocal diffuse ground-glass opacities. He received Remdesivir, Tocilizumab, steroids, empiric antibiotics and prophylactic heparin. 48hrs later, his labs revealed D-dimer of over 35,000, high INR of 1.5, low serum fibrinogen of 50mg/dl and low platelet count of 109,000mcL. This profile raised a clinical suspicion of DIC which put him at an increased risk of bleeding from anticoagulation. A TEG study was done which revealed a low R time (4.2 min) that suggested a hyper-coagulable state, a high K time (4.8 min), low alpha angle (43.4) due to hypofibrinogenemia, low MA (44.8 mm) due to thrombocytopenia, and a normal LY 30. Based on the TEG profile, he received cryoprecipitate and anticoagulation was continued due to his prothrombotic state. Further evaluation revealed that he had developed acute bilateral lower extremity DVT, and as a result, he was commenced on therapeutic anticoagulation.

The clinical utility of TEG in forming anticoagulation therapy decisions in coagulopathic medical ICU patients has become manifest in the COVID-19 era because unlike other traditional hematologic tests, it quantitatively measures the real-time dynamics of hemostasis which can best inform anticoagulation decisions. Its clinical use in DIC may continue beyond the COVID-19 era.
Introduction: Catatonia is a behavioral syndrome marked by an inability to move and function normally. It can be caused by several underlying psychiatric and medical conditions. The incidence of catatonia has primarily been researched in patients with underlying acute psychiatric conditions to date. However, there is concern that it often goes unrecognized and is underdiagnosed, leading to the false conclusion that it is a rare condition.

Case: A 32 year old woman with a significant past medical history of renal transplant on immunosuppression and type 2 diabetes initially presented to the hospital with decreased PO intake and worsening diarrhea. Workup was initially concerning for post transplant lymphoproliferative disorder as the patient had extensive lymphadenopathy, but later was found to have disseminated tuberculosis. Prior to confirming the diagnosis, the patient became critically ill with acute hypoxic respiratory failure requiring transfer to the ICU and eventual intubation. Once TB was confirmed and the patient was started on RIPE therapy, she improved and was extubated. Over a 2-3 day period following extubation, she progressively became more unresponsive, though vital signs remained stable. She was immobile, mute, would not respond to stimuli, resisted passive movement, eyes were fixed and staring, and she was not sleeping. Extensive workup was done for her altered mental status including: negative EEG, negative brain MRI, no metabolic explanation, thorough medication reconciliation, normal lumbar puncture, and even negative test for acute intermittent porphyria. Eventually, a “Lorazepam challenge” was trialed and in less than 10 minutes the patient was spontaneously moving her extremities and partially responding to some commands. Scheduled Lorazepam was continued and over the next 2-3 days she returned mostly to her neurologic baseline.

Discussion: Traditionally, catatonia is thought to be more common among patients with psychiatric histories and diagnoses. However, there appears to be increasing acknowledgement that it can occur, and even be underdiagnosed in patients with no psychiatric history. This case serves as a reminder to consider the diagnosis of catatonia in a patient with altered mental status after other etiologies have been ruled out. It will also increase awareness of the “Lorazepam challenge” test that can help quickly confirm the diagnosis of catatonia in appropriate circumstances.
85) POSSIBLE SHORT TERM HEIGHTENED HYPERCOAGULABILITY IN PATIENTS WITH INDETERMINATE CARDIOLIPIN IGM LEVELS FOLLOWING COVID-19 VACCINATION

Kishan Patel, BS; Adam Koraym, BS; Pinky Jha, MD
Medical College of Wisconsin, Milwaukee, WI

Introduction: Patients with the antiphospholipid antibody syndrome (APS) have an increased risk for recurrent miscarriages, myocardial infarction, venous thrombosis, thromboembolism, thrombocytopenia, stroke, and other vascular coagulabilities. ACA antibodies are quite common in the general population and are not always associated with APS. Studies indicate that there is a higher prevalence of IgM positives than IgG in the general population, in fact, the IgM isotype is frequently considered as transient and not significant for APS diagnosis when isolated. In this case report, we will explore the possibility of patients with mildly elevated IgM anticardiolipin developing blood clots with the COVID vaccine for months possibly.

Case report: A 74 Y male with no significant past medical history presented to the ED with a chief complaint of sudden onset flank pain. Multiple diagnoses were considered in the care and evaluation of this patient. He was seen immediately upon arrival and thoroughly examined. It was noted that he had taken his second dose of the Moderna COVID-19 vaccine six weeks prior to this episode. He was afebrile and his vitals were within normal limits. A peripheral IV was established and labs were drawn. He was given fentanyl for pain. Blood and urine labs showed no evidence of infection. CT showed multiple infarcts but without a clear etiology. Anti-Phospholipid Antibodies were measured, with cardiolipin IgM being 15 MPL being the only positive finding (normal limit: 0-12).

Discussion: It should be noted that the purpose of this case report is not to undermine the necessity of continued vaccination efforts in the COVID-19 pandemic, but to highlight a possible rare complication in a very small subset of patients who have no other clear etiologies of thrombotic events. In the setting of a recent COVID vaccine, and no other clear source of the clots, the combination of a COVID vaccine in patients with mildly elevated anticardiolipin antibodies may pose a slight increase in the risk of developing blood clots.
Successful, long-term weight loss solutions are needed to address the obesity crisis in the United States. Bariatric surgery is an option to help people lose weight and maintain a healthier lifestyle. Cognitive behavioral therapy (CBT) has been used to improve outcomes after bariatric surgery. We hypothesized that patients who underwent a CBT course will have higher self-reported confidence levels compared to patients who did not participate in the course.

Methods: A two-part prospective cohort study of CBT course participants and bariatric surgery patients between August 2019 and May 2021 was conducted. The CBT course participants were evaluated with a pre- and post-course test with overall test score being the primary outcome. All bariatric surgery patients completed a self-reported confidence level survey, and the total confidence level score was used as the primary outcome. Patients were also evaluated for participation in the CBT course and time interval to/from the bariatric procedure.

Results: There were 145 pre-course tests and 88 post-course tests collected. Patients scored an average of 7.18% higher after completing the CBT course (0.7179 vs. 0.7897, p<0.05). There were 310 self-reported confidence level surveys collected. The overall self-reported confidence level survey score ranged from 10-40 points with 40 points corresponding with the highest confidence rating for all scenarios. The average overall confidence level score increased by 1.71 points when comparing patients before and after surgery (32.88 vs. 34.57, p<0.05). In addition, patients who underwent the CBT course reported a 4.38% higher overall confidence level compared to those without CBT (34.39 vs. 32.64, p<0.05).

Conclusion: Overall, there is a trend of increasing confidence levels as patients approach their surgery date and a further increase in confidence in the post-operative period. The higher post-course test scores suggest that the CBT course participants are meeting the learning objectives of the course. Patients who underwent a CBT course had a higher self-reported confidence level compared to patients who did not have CBT. This suggests that CBT courses in the pre-operative period may improve bariatric surgery outcomes given the enhanced self-efficacy.
87) LGI1 LIMBIC ENCEPHALITIS: A RARE CAUSE OF COMMON CHIEF COMPLAINT

Alana Petrassi; Miranda Brown; Amro Abdelghani
Medical College of Wisconsin, Milwaukee, WI

Introduction: Though a very common complaint in the emergency department, altered mental status has a heterogeneous list of etiologies. Defined as an alteration in emotional, intellectual, or psychological functioning, the differential can be separated into primary neurological, toxic/metabolic, and infectious. When initial investigation fails to elucidate a diagnosis, symptoms and exam findings should guide testing for less common etiologies.

Case Description: A 71-year-old Caucasian male with a history of atrial fibrillation, hypertension, hypothyroidism, and pulmonary embolism presented to an outside ED reporting 50 daily episodes of dizziness. Neurological exam and ECG findings were benign. Two months later, the patient was admitted due to confusion, paranoia, and insomnia. His sodium was 126 but quickly corrected. CT and MRI as well lumbar puncture were unremarkable.

After discharge, patient’s daughter sought a second opinion. At this time, the patient had asynchronous, hypnic jerking of the extremities. Repeat MRI demonstrated a new area of hyperintensity in the left hippocampus. An autoimmune encephalitis panel demonstrated the presence of Leucine-Rich Glioma Inactivated 1 Protein-IgG (LGI1). The diagnosis of limbic encephalitis (LE) was confirmed. A five-day course of steroid therapy was initiated.

Discussion: Here we detail a common presentation of a relatively rare disease: LGI1 positive limbic encephalitis. LGI1 LE is an inflammatory process localized to the limbic system caused by production of antibodies against leucine-rich glioma inactivated 1 protein. Common presenting symptoms for LGI1 LE are hyponatremia and faciobrachial dystonic seizures. LGI1 also has a strong association with malignancy.

With this patient’s initial complaint of dizziness followed by his deteriorating mental status, hyponatremia, and dystonic jerks, his presentation was a classic picture of LGI1 LE. Although this may not be on the initial differential for altered mental status, this process and other autoimmune or paraneoplastic conditions should be considered in the advanced workup.
Introduction: With an estimated 14,000 cases in 2015, anticholinergic toxicity is a common condition that can arise from many sources. Symptoms can include fever, anhidrosis, nonreactive mydriasis, and psychosis. Diagnosis is made from a mix of classic symptomology and a known exposure to an anticholinergic substance. However, anticholinergic toxicity should be considered in any patient with altered mental status and accompanying symptoms consistent with toxicity.

Case Description: A 76-year-old male with a past medical history of Type 2 DM, HLD, and HTN presented with weakness. Symptoms began abruptly after eating a hamburger with abdominal pain, emesis, and diarrhea. He soon became confused, diaphoretic, and incontinent of bloody urine. He presented to the ED and was febrile to a Tmax of 104.5°F, tachycardic, and tachypneic. On physical exam, he was oriented only to self and situation. His lungs were clear bilaterally and a cardiac exam showed tachycardia without abnormal heart sounds. Labs were remarkable for a lactic acid of 6.3 (ref range: 0.5-2.0 mmol/L), and UA positive for leukocyte esterase. Head CT was negative for an acute intracranial process and chest X-Ray showed a left lung base patchy density. He was started on IV vancomycin, azithromycin, and cefepime for suspected sepsis. Within 24 hours, he showed signs of improvement. He was afebrile, his lactic acid had trended down to 3.1, and his altered mental status had completely resolved. However, he did have ongoing leukocytosis with WBC 23. It was then discovered that he had been using Round Up, an herbicide containing glyphosate, before symptom onset. A physical exam showed dirt caked under his fingernails, providing a likely source of oral transmission with the consumption of the hamburger. After a discussion with poison control, the patient was able to be discharged as toxicity wanes within 72 hours, he was afebrile, and tolerating a diet.

Discussion: Anticholinergic toxicity can be difficult to diagnose without known exposure. In addition, spring and summer months often increase exposures to anticholinergic drugs used in gardening products and pesticides making it more important to consider as a differential diagnosis. In this case, our patient had symptoms that pointed towards possible sepsis including his leukocytosis, elevated lactic acid, and his UA and CXR results. However, further clinical assessment was needed as the quick resolution of symptoms and altered mental status did not correlate with sepsis. Utilization of detailed history taking and poison control services proved to be key in finding the correct diagnosis and providing the best follow-up care.
Background: Although *Staph aureus* is the most common cause of vertebral osteomyelitis, other rare causal pathogens including *Mycobacterium bovis* have been reported. Spinal tuberculosis (TB) is rare in the United States, with an estimate of 1 case per year for every 2 million individuals. Bacille Calmette-Guerin (BCG) vaccine is a live, attenuated form of *Mycobacterium bovis* utilized in the United States for its unique utility in the treatment of bladder cancer. We report an extremely rare case of BCG vaccine-induced TB Osteomyelitis and extensive destruction in the lumbar spine, along with novel application of temporary percutaneous instrumentation and bone reconstitution with anti TB antibiotics.

Case Presentation: A 77-year-old male patient with past medical history significant for bladder cancer treated with intravesical BCG vaccine presented with severe lower back pain and lumbar radiculopathy. The patient was confirmed to have *Mycobacterium bovis* TB osteomyelitis and anti-TB regimen was initiated. Due to degenerative stenosis, he had Lumbar L1-5 decompression surgery. Further degenerative changes resulted in a 31.4° kyphotic deformity at L1 and L2, 11.9° lumbar lordosis, and 42% and 41% destruction in L1 and L2 respectively. In hopes for vertebral reconstitution, the patient underwent a posterior percutaneous fixation with navigated pedicle screws and rods at T12-L3, with correction of the kyphotic deformity. After this surgery 21% and 27% volume was restored back to L1 and L2 respectively.

Discussion: The BCG vaccine is a live, attenuated form of *Mycobacterium bovis* commonly used for bladder cancer treatment. A systematic review found 25 reports of spinal TB related to intravesical administration of BCG vaccine for bladder cancer. Traditionally, vertebrectomy and reconstruction has been the mainstay option for extensive destructive TB Osteomyelitis in the thoraco-lumbar spine. Minimally invasive percutaneous instrumentation alone, without fusion, appears to allow for excellent bone reconstitution and stabilization and is a viable option for this pathology.
Introduction: Cauda equina syndrome is caused by severe compression of nerve roots in the thecal sac and presenting as most commonly back pain with either unilateral or bilateral radiation of pain, weakness, or sensory loss. Bladder dysfunction may or may not be present. A neoplastic process can contribute to compression of the epidural spinal cord and common malignancies being prostate, breast, lung, and multiple myeloma. MRI imaging of choice for imaging diagnosis and management.

Case Presentation: 73-year-old male with a history of hypertension, benign hematuria, CKD stage III, an active smoker was being followed up by PCP for subacute lower back pain following a fall, worsened with movement radiating down to bilateral thighs posteriorly to the level of the knee, also aggravated towards the end of the day. Evaluation with MRI revealed severe spinal canal stenosis along with impingement of cauda equina nerve root by infiltrative osseous processes likely concerning the metastatic disease. Immediate lab work-up including CBC, CMP, PSA, serum protein electrophoresis, and the light chains were requested, which revealed an abnormally elevated PSA of 5494 NG/mL that revealed high suspicion of metastatic prostate cancer with clinical signs of cauda equina syndrome with bilateral lower extremity weakness and pain.

Oncology, radiation oncology, neurosurgery were consulted and the patient was treated with dexamethasone, radiation therapy with significant improvement in symptoms was observed. He followed up with oncology outpatient and received firmagon, denosumab, docetaxel that was discontinued due to complications. He is currently on abiraterone and prednisone as well for maintenance.

Discussion: Spinal cord lesion can result in permanent neurologic deficits including quadriplegia if left untreated, thereby immediate evaluation and intervention are recommended. One of the causes can be a neoplastic process that can lead to nerve root compression.

An evaluation for emergent surgical decompression in patients with rapidly progressive cauda equina syndrome with bladder dysfunction is performed. As the patient was diagnosed with metastatic prostate cancer in the setting of elevated PSA, dexamethasone, gonadotropin-releasing hormone antagonist (degarelix) that rapidly decrease his testosterone levels along with palliative radiation was given that decreased the size of lesion mass with improvement for the patient.
Abstract: A 53-year-old male with recent known exposure to COVID-19 presented with fever, cough, and shortness of breath. Five nasopharyngeal samples were tested with RT-PCR and resulted negative for COVID-19, but a bronchoalveolar sample was tested with RT-PCR and resulted positive. At present, the most used detection method for COVID-19 is a nasopharyngeal sample tested with RT-PCR, but bronchoalveolar lavage with RT-PCR is considered the standard diagnostic test for COVID-19. Case reports demonstrate a degree of discordance between nasopharyngeal and bronchoalveolar lavage samples. Patients shedding the virus in their lower respiratory tract may not be ideal candidates for nasopharyngeal/oropharyngeal testing, and bronchoalveolar lavage sample with RT-PCR may be the best-suited test for these patients. This case highlights the importance of collecting a sample by bronchoalveolar lavage to diagnose COVID-19 in patients with symptoms of COVID-19 when nasopharyngeal/oropharyngeal swabs are negative.
92) LEGIONNAIRES’ DISEASE IN A PATIENT WITH WELL-CONTROLLED HIV

Fernando Garcia-Ramirez; Brian Quinn
Medical College of Wisconsin, Milwaukee, WI

**Introduction:** AIDS-defining illnesses classically present in patients with CD4+ cell counts below 200 cells/mm$^3$. Nevertheless, HIV-infected individuals, even with high CD4+ cell counts, are at a higher risk of developing opportunistic infections than the general population. It is therefore surprising that the incidence of Legionella pneumonia, an opportunistic infection also known as Legionnaires’ disease, remains relatively low in HIV-positive patients, ranging from less than one to eight percent of pneumonia cases in retrospective case series. The following case demonstrates a rare but pathognomonic presentation of Legionella pneumonia in an HIV-positive individual despite a well-controlled viral status.

**Case Presentation:** A 47-year-old Hispanic man with a past medical history significant for HIV-1 on TRIUMEQ admitted to the internal medicine service with a three-day history of fever, chills, night sweats, intermittent shortness of breath, pleuritic chest pain, and diarrhea. Upon admission, the patient had a low-grade fever of 99.3 °F, borderline hypotensive, and tachycardic on arrival. Lungs were clear to auscultation on physical examination. Notably, the patient had an undetected HIV viral load of <20 HIV RNA copies/mL and a CD4+ cell count of 990/mm$^3$ seven months prior to admission. Overall, his HIV has been well-managed with antiretroviral therapy (ART) since his initial diagnosis at age 28. Initial labs in the ED were significant for leukocytosis (WBC = 11.5 x 10$^3$/μL) and neutrophilia (ANC = 8.68 x 10$^3$/μL). A basic metabolic panel obtained upon admission was significant for mild hyponatremia (Serum Na$^+$ = 133 mEq/L). Chest X-rays obtained in the ED identified consolidation in the lateral aspect of the left midlung consistent with pneumonia. The patient was initiated on empiric treatment for community-acquired pneumonia. The patient’s symptom profile, along with his HIV status, neutrophilia, hyponatremia, and lobar infiltrate on chest X-ray immediately raised concerns for Legionnaires’. A urine antigen test resulted positive for *L. pneumophila*. The patient was discharged to home in improved and stable condition with a seven-day course of daily oral azithromycin 500 mg.

**Discussion:** Legionella pneumonia in HIV-positive patients remains rare but can be more severe in those with untreated or poorly managed HIV. This case highlights how 1) HIV-infected individuals are at a higher risk of developing opportunistic infections regardless of CD4+ cell counts, and 2) close monitoring and management of HIV with ART is imperative for best possible outcomes during coinfections.
Pulmonary hypertension (PH) affects 1% of the global population, including 10% of people over 65 and ~50% of people with Congestive Heart Failure (CHF). Pulmonary hypertension (PH) is classified into 5 clinical subgroups: pulmonary arterial hypertension (PAH), PH due to left-sided heart disease, PH due to chronic lung disease, PH due to chronic thromboembolic disease (CTEPH), and PH with idiopathic or multifactorial etiology.

A 75 Y female with a PMH including severe COPD on 6-7L home O2 and MAC infection, presented with 5 days worsening exertional dyspnea, increased O2 requirement, and chest “tightness” with exertion. She was afebrile, HR 87, RR 36, BP 153/103, SaO2 98% on 5L NC. Physical exam was significant for respiratory distress with mild accessory muscle use, without wheezing or crackles, and 1+ peripheral edema to just above the ankles. Labs included a troponin of 43 and BNP of 11,319 (increased from 3,344 one month prior). Initial diagnosis was new onset CHF exacerbation, and the patient was treated with IV furosemide.

A CT PE scan showed centrilobular emphysema and non-TB MAC that progressed from nodules to thick-walled cavitary lesions. The CT scan was negative for VTE, significant pulmonary congestion or acute PE, though V/Q scan is gold standard for CTEPH. After 2L of diuresis, an echocardiogram showed an elevated PA pressure of 82 mmHg. Mean PA systolic pressure increased from 30 to 38 mmHg from the patient’s most recent RHC (10/2019). The PVR also increased from 5 to 11 Woods units.

Though the patient has severe COPD, her PH was most likely due to group 1 rather than group 3 PH, as the PA pressure increased beyond what would be expected in one year solely from COPD. The patient was started on a vasodilator used in group 1 PAH.

This case highlights the difficulties of diagnosing pulmonary hypertension. PH is often initially mistaken for a CHF exacerbation. In addition, the etiology of PH can be difficult to diagnose. However, accurate diagnosis and classification is essential for effective therapy, as treatment can be targeted to the specific type of PH. Reclassifying this patient as group 1 PAH opened medication options that are efficacious in group 1, but not group 3 PH.
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94) ACUTE WORSENING OF ATYPICAL PARKINSON’S SYNDROME AFTER RECEIVING THE SECOND DOSE OF THE MODERNA COVID-19 VACCINE
Rachel Russell; Brian Quinn, MD
Medical College of Wisconsin, Milwaukee, WI

Atypical Parkinson’s syndromes are a set of neurodegenerative conditions defined by the presence of classical Parkinson’s disease symptoms (bradykinesia, tremors, rigidity, and postural instability) in addition to various other unrelated issues. We present the case of a 71-year old male who had a one-year history of weakness and upper extremity tremors that rapidly worsened after receiving the second dose of the Moderna Covid-19 vaccine. The patient had no known medical history as he had not visited a physician in over 20 years. He reported an approximately 12-month history of resting and action hand tremors (right-side more than the left), a decline in fine motor movements (primarily related to handwriting), and progressive gait changes leading to increased instability and 2-3 “minor” falls at home. Upon physical examination, it was noted that the patient had a resting tremor, severe rigidity, and bradykinesia with the right side significantly more affected than the left. Neurology was consulted and a full workup was performed including a brain MRI, a spine MRI, and an EMG. The only clinically-significant findings were those of the EMG which demonstrated evidence of a mild, diffuse, chronic lower motor neuron process affecting the right cervical, thoracic, and lumbosacral regions. Extensive metabolic, rheumatologic, and neurotoxic laboratory assessments were also completed. The only abnormalities identified were a positive Antinuclear Antibody test with elevated Topoisomerase antibodies. Consistent with the clinical picture of an asymmetric atypical Parkinson’s syndrome, Neurology recommended that the patient be initiated on carbidopa/levodopa treatment with baclofen. The patient was discharged from the hospital to a rehabilitation center for continued strength and conditioning. At his one-month follow-up the patient had demonstrated minimal response to these interventions. It is clear that our patient had underlying signs of an Atypical Parkinson’s syndrome prior to his presentation. We are uncertain as to what degree the Covid-19 vaccine impacted the patient’s condition but would like to recognize the significant temporal association between the administration of the vaccine and the abrupt worsening of our patient’s symptoms.
Spontaneous bacterial peritonitis (SBP) is a severe complication of ascites, often seen in advanced hepatic disease. As causative organisms are typically E. coli, Klebsiella, or other gram-negative enterics, third-generation cephalosporins are the treatment of choice. In SBP unresponsive to conventional treatments, other etiologies must be considered.

Our patient was a 71-year-old female presenting for rectal pain and abdominal distension, with a history significant for Crohn disease on azathioprine, and nodular regenerative hyperplasia with portal hypertension. One month prior, she was evaluated for a fever of unknown origin; a CT-A/P showed colitis. Antibiotics were ineffective, but prednisone abated her fever. Shortly after, she developed worsening abdominal distension. Paracentesis removed 5.5L of cloudy-yellow fluid with WBCs over 5000, 86% PMNs. Cephalexin was initiated. 9 days later she developed the presenting symptoms. Repeat paracentesis yielded 600ml of cloudy-yellow fluid with a WBC count of 2800, 82% PMNs. WBCs were elevated from 7.6 to 17, but BMP and transaminases were unremarkable. CT-A/P revealed cirrhotic changes, ascites, and new 2.5cm air-fluid level contiguous with the posterior anus and extending to the gluteal cleft, suspicious for a perianal abscess. Ascitic fluid yielded gram-positive rods; culture grew cephalexin-resistant Corynebacterium. She was switched to vancomycin but developed diarrhea. Improving ESR/CRP made a CD flare unlikely, and she was C.-diff-NAAT-positive but toxin-negative, suggesting an antibiotic-induced diarrhea.

The Corynebacterium was suspected to be an abscess contaminant; reanalyzing the culture showed Listeria. Further history revealed she owned a dairy farm, and her husband recently had a GI illness. She likely contracted enteric Listeria that seeded her peritoneal fluid. Ampicillin was initiated; repeat paracentesis showed a recovering WBC count with negative gram stain and culture. She was discharged on amoxicillin.

Listeria is a rare cause of SBP, with only 98 cases reported between 1977-2015. Its presentation resembles other pathogens, identification of risk factors such as ascitic or blood cultures with gram-positive rods, immunosuppression, exposure to farm animals or contaminated foods, and iron overload, is crucial. Estimated mortality is 30%. Listeria is resistant to some cephalosporins; ampicillin, with an aminoglycoside if tolerated, is the treatment of choice. Given recurrence up to 70% in one year and innate fluoroquinolone resistance, trimethoprim-sulfamethoxazole prophylaxis is indicated after an initial episode of Listeria SBP.
POEMS syndrome is a rare paraneoplastic syndrome named for its constellation of polyneuropathy, organomegaly, endocrinopathy, monoclonal protein, and skin changes. A proliferative plasma cell disorder, it has a multisystem presentation and chronic cytokine overproduction. It often presents in the 5th or 6th decade, mostly in males. Presentations vary widely, making it extremely difficult to diagnose. A chronic progressive distal, sensorimotor polyneuropathy in conjunction with a monoclonal plasma cell dyscrasia suggests POEMS.

A 61-year-old man with a past medical history significant for a 2-year history of bilateral lower extremity weakness and a cerebral meningioma presented with anorexia, intermittent dysphagia, and early satiety with intermittent vomiting and diarrhea over the past 3 months, with a 20-pound weight loss. Prior to admission he underwent extensive workup for similar symptoms. His creatinine was elevated, and troponins were high despite a normal EKG. A CT A/P was obtained due to vomiting, showing expansile lesions of the left ischium, acetabulum, and ilium, with small-volume ascites and splenomegaly. In lieu of these findings and noted bilateral lower extremity neuropathic weakness, we obtained further testing to corroborate suspicions of a plasma cell disorder. Labs were significant for IgG lambda with M spike, low cortisol, low testosterone and elevated TSH. Protein electrophoresis was positive for bi-clonal lambda, and his VEGF levels were elevated to 377. Bone marrow core biopsy indicated malignancy. Given his polyneuropathy, organomegaly, endocrinopathy, IgG lambda, skin changes and extravascular fluid overload, POEMS syndrome was diagnosed. He was initiated on Lenalidomide and Dexamethasone, and eventually received a stem cell autologous bone marrow transplant.

Given his non-specific presentation, and the confounding factor of a known meningioma causing hypopituitarism, diagnosing POEMS required extensive workup. A contrast CT demonstrating bone lesions suspicious for myeloma-type disease was crucial, pairing his endocrinopathy and neuropathy with a plasma cell dyscrasia. Although POEMS is exceedingly rare, accurate diagnosis is vital, as treatment requires a multidisciplinary approach. While high-dose chemotherapy-conditioned autologous stem cell transplantation is the gold standard treatment for POEMS syndrome, patients who are diagnosed in a late stage of the disease are not candidates, underscoring the need for early identification of the disorder.
Introduction: Hydralazine is commonly associated with drug-induced lupus but there is another more seldom seen condition associated with the use of hydralazine. Our case depicts a patient treated with hydralazine for several years leading to microscopic hematuria and worsening renal function with a negative urologic workup prior to hospitalization found to have hydralazine induced antineutrophil cytoplasmic antibody (ANCA) ANCA vasculitis.

Case Presentation: A 65-year-old male with a past medical history of essential hypertension and chronic kidney disease stage II who presented to the hospital with fatigue, dyspnea, and arthralgias. Pertinent laboratory findings included creatinine of 8 mg/dL, large blood on urinalysis, antinuclear antibody titer of 1:1280 with homogeneous staining pattern, myeloperoxidase antibody >8.0, and proteinase-3 antibody >8.0 all highly suspicious for hydralazine induced ANCA vasculitis. Hydralazine was discontinued and patient was started on corticosteroids, plasmapheresis, and a rituximab infusion. The patient’s symptoms and renal function improved leading to discharge on prednisone with plans to complete further rituximab infusions and plasmapheresis sessions as an outpatient.

Discussion: Our patient had been receiving hydralazine for 2.3 years prior to presentation at doses of up to 300 mg per day. Hydralazine induced ANCA vasculitis is seen on average 4.7 years after drug initiation with an average daily dose of 142 mg (1). The pathogenesis is unidentified but it is hypothesized that hydralazine collects inside neutrophils causing apoptosis and allowing for antibody formation against exposed antigens. This case demonstrated the importance of recognizing hydralazine induced ANCA vasculitis in order to stop hydralazine and start immunosuppressive therapy.
A 63-year-old male farmer from central Wisconsin presented with a week-long history of chest pain and fevers. He was found to be bradycardic to 45 bpm but hemodynamically stable. Exam revealed a rash consistent with erythema migrans, EKG demonstrated first-degree heart block. A clinical diagnosis of Lyme disease was made, and empiric treatment was initiated with ceftriaxone. Lyme EIA resulted positive shortly thereafter. Two hours following presentation, he developed second-degree type II AV block followed by complete heart block but otherwise remained hemodynamically stable. Overnight, he developed fever, diaphoresis, and rigors consistent with the Jarisch-Herxheimer reaction. On HD1, he began to have episodes of asystole with an unreliable escape rhythm, requiring transcutaneous followed by transvenous pacing and semi-permanent pacemaker (semi-PPM) placement. On HD4, Lyme western blot resulted positive. After 1 week of antibiotic therapy, his primary rhythm was first-degree AV block, and the semi-PPM was removed. He was transitioned to doxycycline and discharged with a 30-day event monitor. At one-month follow-up, he had resolution of symptoms with return to normal sinus rhythm without evidence of arrhythmia or AV block on the event monitor.

Caused by the spirochete *B. burgdorferi* and transmitted in the saliva of the *I. scapularis* tick, Lyme disease is the most common vector-borne infection in the US. There are 3 phases of Lyme disease: early localized, early disseminated, and late disease. Diagnosis is a two-tiered approach consisting of screening EIA followed by confirmatory western blot. The diagnosis may be made clinically in patients from an endemic area presenting with consistent history and exam findings, diagnostics should not delay treatment in these cases. In early disseminated disease, carditis is caused by bacterial invasion of the myocardium and may cause high-grade AV block requiring IV antibiotics, cardiac telemetry, and temporary pacing if hemodynamic instability develops. Death may occur if treatment is delayed. Ceftriaxone is first-line and should be administered until resolution of high-grade AV block is observed, at which time patients may be transitioned to doxycycline to complete a 28-day total course of antibiotics. The Jarish-Herxheimer reaction is classically observed in the treatment of syphilis but may also be seen in the treatment of non-treponemal spirochete infections. As demonstrated by this case, with appropriate management, Lyme carditis is a reversible disease with a favorable prognosis.
Introduction: Ocular syphilis can involve virtually all ocular structures, and uveitis and chorioretinitis are the most frequent manifestations. Here, we present an interesting case of ocular syphilis in an HIV-positive male.

Case Description: A 52-year-old male with a history of recent COVID-19 infection and HIV on triple therapy presented to outpatient neuro-ophthalmology clinic for 8 weeks of worsening right eye vision. Ocular exam showed evidence of ocular inflammation, including uveitis. Extensive lab workup demonstrated reactive serum RPR and treponema antibodies. Thus, the patient was admitted for evaluation of neurosyphilis. Due to additional concerns for a space-occupying lesion and neuromyelitis optica spectrum disorder, he underwent MRI/MRV brain/orbit prior to lumbar puncture for further evaluation. Imaging was negative, while CSF analysis demonstrated pleocytosis with elevated protein and reactive VDRL, confirming the diagnosis of neurosyphilis. The patient was treated with IV crystalline penicillin for 14 days, followed by IM benzathine penicillin for 3 weeks, as his diagnosis was considered late-latent syphilis. Optic disc edema and positive MOG antibody were suggestive of additional contribution from MOG-associated optic neuritis (MOG ON), which was thought to be a sequela of his syphilis infection or recent COVID-19 infection. As vision did not improve on high-dose IV methylprednisolone, there was concern for steroid-unresponsive MOG ON or acute HSV/VZV retinitis, unmasked by steroids in an immunosuppressed individual. The patient required stopping of steroids and a course of IV immunoglobulin (IVIG) and acyclovir. Ultimately, he reported a gradual improvement in vision prior to discharge.

Discussion: Syphilis should always be considered in the differential diagnosis of ocular inflammation in both HIV-negative and HIV-positive patients. Vision loss and ocular complications are more common among HIV-negative patients due to delay of diagnosis. This highlights the importance of early diagnosis by screening for visual complaints in syphilis-positive patients and, conversely, screening for syphilis in high-risk patients with visual symptoms (e.g., men who have sex with men [MSM], HIV-positive patients, or patients with multiple/anonymous sexual partners). In patients with syphilis and ocular complaints, immediate ophthalmologic evaluation should be considered. Since syphilis is often curable, early diagnosis is key, as accurate diagnosis and treatment prevent further spread of the infection and its complications.
Background: Approximately 27% of older adults 65 years and older have diabetes and are at significant risk for complications, institutionalization, and mortality. Diabetes Self-Management (DSM) is a key part of diabetes care and requires knowledge, skills, and an understanding of lifestyle behaviors, which are often accessed from a number of sources outside of the diabetes care team. Understanding how sources of diabetes knowledge impacts DSM behaviors among older adults has important implications for diabetes care for this vulnerable population.

Methods: Data from 1,901 adults from the Health and Retirement Survey (HRS) 2003 diabetes cohort was used. The outcome, DSM behaviors included medication adherence, diet, and blood sugar testing. The primary independent variable was source of diabetes information, use of television, internet, newspaper or magazines, books, or friends. Survey logistic regression models tested the likelihood of performing recommended DSM behaviors, and whether source of information was associated with DSM behaviors controlling for demographics.

Results: Mean age was 70 years, 76% were non-Hispanic White, and 44% had a high school education. Internet as a source of diabetes information was significantly related to lower odds of medication adherence (OR=0.37;CI=0.21-0.65). Television (OR=0.68;CI=0.51-0.91), newspapers/magazines (OR=0.55;CI=0.42-0.73), and friends (OR=0.62;CI=0.47-0.82) as sources of diabetes information were related to lower odds of following a recommended diet, compared to not using these sources for diabetes information. Books were related to higher odds of eating a healthful diet (OR=1.74;CI=1.22-2.49) and higher odds of testing blood sugar (OR=1.53;CI=1.04-2.25).

Conclusions: Overall, sources of diabetes information had a differential relationship with DSM behaviors. Patients reporting the use of books as a source of diabetes information had significantly higher likelihood of following a healthful diet and were more likely to test their blood sugar. Incorporating effective
Pericardial effusion is defined as the presence of more than 150 to 200 cc of fluid in the pericardium. It can lead to life-threatening cardiac tamponade that compresses the heart. Physical signs are nonspecific, most patients without a hemodynamically significant pericardial effusion have no symptoms. Most commonly diagnostic studies start with EKG which shows low voltage QRS complexes, but it is not specific to patients with effusion. Small to moderate effusions might not show up on chest x-ray. Additional imaging usually echo in patients with suspected pericardial disease can be helpful. These approaches can delay the diagnosis. Bedside ultrasound can be used for diagnosis of pericardial effusion quicker than other diagnostic imaging technologies. We are presenting a 63-year-old patient with history of untreated hypertension who came in with nonspecific complaints of bilateral lower extremity edema and oliguria of 2 weeks duration. A bedside ultrasound in emergency department revealed a large pericardial effusion with signs of hemodynamic compromise. Further echocardiographic imaging was performed which revealed a very large circumferential pericardial effusion, urgent pericardiocentesis was performed which drained 1.8L of hemorrhagic fluid. Cytology of pericardial effusion revealed malignant cells derived from poorly differentiated metastatic adenocarcinoma which was strongly positive for epithelial specific antigen/epithelial cell adhesion molecule. This case highlights the importance of bedside ultrasound in quickly diagnosing pericardial effusion, and further study to evaluate the diagnostic performance of this rapid imaging modality for patients with suspected pericardial effusion. Expediting time to diagnosis and initiation of management in pericardial effusion with tamponade physiology is very crucial, it not only decreases morbidity and mortality but also it is very crucial in improving overall patient outcomes. Point-of-care ultrasound plays an important role in initial evaluation and prompt management of patients presenting with cardiovascular symptoms, it narrows down the differential diagnosis, guides in initiation of goal-directed therapy and consultations required.
Introduction: Renal-limited ANCA-positive vasculitis is an exceedingly rare condition with involvement of other organ systems becoming a distinct possibility with disease progression. A rising creatinine with hematuria should be treated as a medical emergency when developing over days to weeks.

Case Presentation: A 27-year old male presented to urgent care with vague abdominal pain, ten-pound weight loss, a petechial rash on the abdomen and feet, anxiety and occasional copper-colored urine. These symptoms occurred one month after an assumed viral upper respiratory infection (URI) and two weeks after receiving the second dose of an mRNA COVID vaccine. Initial workup revealed a serum Creatinine of 9.0. He was admitted to the hospital and further investigation revealed microscopic hematuria, nephrotic-range proteinuria, and positive anti-PR-3 Antibody. The patient underwent renal biopsy which showed crescentic glomerulonephritis with 18% of glomeruli demonstrating either global or segmental scarring, as well as thin basement membrane nephropathy. He was initiated on high dose steroids, IV fluids and Rituximab resulting in slight improvement in serum Creatinine to 7.3. He was discharged with a plan for continued immunosuppression and close monitoring of kidney function.

Discussion: This case illustrates a patient diagnosed with rapidly progressive glomerulonephritis due to two separate pathological processes given evidence of glomerular scarring due to vasculitis as well as thin basement membrane nephropathy. Furthermore, it remains unclear whether the COVID vaccine is implicated in this diagnosis given the recency of administration to the patient’s presentation as well as the preceding viral URI.
103) DO NOT FORGET TO SAVE YOUR HEART

Karan Bir Singh\textsuperscript{1}; Manoj Reddy Somagutta\textsuperscript{1}; Aman Kalra\textsuperscript{1}; Mahavir Singh\textsuperscript{2}

\textsuperscript{1}Avalon University School of Medicine, Willemstad, Curacao
\textsuperscript{2}University of Louisville School of Medicine, Louisville, KY

Introduction: Alzheimer’s disease (AD) is the most common cause of dementia in the elderly, however; the clinical association of cardiovascular conditions with that of mild cognitive impairment (MCI) and AD are not well understood. Recently, heart failure (HF) has been reported as a significant risk factor for AD, and the mechanism is believed to be the reduced cerebral perfusion and impaired neurohormonal activation leading to impaired clearance of the amyloid-beta (Aβ) and hyperphosphorylation of tau protein. These biochemical perturbations result into the formation of Aβ plaques and neurofibrillary tangles.

Objective: To investigate the causal relationships, the associated risk factors and prognostic role of HF and AD types, if any, between AD and HF patients.

Methods: Databases such as PubMed, Google Scholar and PubMed Central were systematically searched for literature published within the last 10 years, incorporating the keywords, “Alzheimer’s disease and heart failure.” Original research articles were included in this study with inclusion of individuals >45 years old.

Results: Ten pertinent studies were analyzed. One study investigates the association between subclinical cardiac dysfunction and AD biomarkers and found that left ventricular ejection fraction (EF) was linked to more robust evidence of tau phosphorylation and neurodegeneration among older adults with normal cognition. A Swedish study in 775 HF patients noted HF with preserved EF was the most common HF type. In a Danish study, risk of all-cause dementia was increased among HF patients compared to general population [HR 1.21, 95%, (CI) 1.18-1.24], especially in patients under age 70. In another study, 12 months after diagnosis of HF, there was notable decline in physical activity, contributing to worsening cognition and cerebral perfusion, increasing the risk for MCI and subsequent AD. The Framingham Study demonstrated decreased myocardial function being the hallmark for HF and linked associated lower brain volume, the hallmark of AD. Surprisingly, none of the aforementioned studies remarked on the association between the various HF types or patient’s survival with AD.

Conclusion: HF is a risk factor for the development of AD. Although the association at a deeper level remains unknown, it appears to be the decreased CBF and associated comorbidities such as atherosclerosis. Evidence indicates management of HF, and its risk factors reduce the risk of neurocognitive degeneration and subsequent AD.
104) STATIN INDUCED NECROTIZING MYOPATHY
Courtney Smith, M4; Amro Abdelghani
Medical College of Wisconsin, Milwaukee, WI

**Introduction:** Statin induced muscle related side effects range from myalgia, muscle atrophy and inflammation, to rhabdomyolysis and renal failure. Patients with statin induced myopathy have proximal, symmetric weakness with or without tenderness, functional impairment such as rising from a sitting position or climbing stairs. Patients may or may not have elevated CK, though severity of disease and response to therapy can be monitored by CK level. Most patients have onset of symptoms within weeks to months of starting statin treatment, but it can happen at any time.

**Case Description:** A 61-year-old African American female presents with progressive bilateral lower extremity weakness that acutely worsened requiring the use of a walker for ambulation and inability to comb her hair. EMG findings demonstrated severe, patchy irritable myopathy in the upper and lower limbs. Patient was started on solumedrol 1 g for 5 days, and then prednisone 60 mg for 4-6 weeks. HMGCR antibodies were positive. On admission her CK was 11,350, which decreased to 2800.

A 62-year-old Caucasian male with history of CABG, presents with history of acute worsening of painless symmetric weakness in his proximal lower limbs requiring the use of a wheelchair. Prior to admission he had an elevated CK of 8788, positive HMGCR antibody and had stopped statin therapy and started a high dose steroid taper. A muscle biopsy was done outpatient, which showed as active necrotizing myopathy. CK on admission was 115. Because he had already been treated with a course of steroids, IVIG was started. Repeat EMG during admission revealed that the myopathy has responded to steroids. Patient will continue to get IVIG for 4-6 months.

**Discussion:** Here we report two cases of statin induced autoimmune necrotizing myopathy in patients on statin therapy. Statin therapies are common in primary care as a first line agent for hyperlipidemia and elevated ASCVD risk. With statin induced myalgias, patients can trial a lower intensity statin or lower dose without concern. However, there is a high risk of symptom relapse with necrotizing myopathy. This should be a discussion between the patient and physician about risks vs. benefits especially in patients with significant cardiovascular disease.
EMERGENCY DEPARTMENT UTILIZATION
Kaitlyn Sonnentag
Medical College of Wisconsin, Green Bay, WI

Introduction: Past research looking at emergency department (ED) utilization found key reasons why patients come to the ED for non-emergent conditions are:
1. Lack of knowledge about affordable and convenient care outside of the ED.
2. Patients’ perceptions of the acuity of their conditions being inconsistent with perceptions of the ED providers.

Purpose:
1. To investigate the reasons for usage of the Bellin ED in Green Bay, WI for non-emergent conditions.
2. To provide patients with information on when they should seek care at an alternative healthcare facility and specific alternatives for care in the area. The ultimate reason is to decrease the number of patients presenting to the ED so that patients with emergent conditions can get faster and better care.

Methods:
Patients who received an acuity level of 4 or 5 by ED providers were considered for the survey. At the end of the survey, patients were asked if they would like an informational handout on criteria for seeking care at an ED, urgent care, or primary care facility as well as addresses of these facilities in the area.

Results:
• 75% of patients rated their problem as more severe than providers did.
• 87% stated that they would use reliable alternatives to getting care outside of the ED if these existed.
• 50% stated they wanted the informational handout.

Conclusion:
• Patients’ perceptions of the acuity of their conditions are inconsistent with providers’ perceptions.
• There is lack of knowledge about alternatives to the ED.
• Patients would be willing to use alternatives to the ED if they knew when and where to go.
• With educating our patients, we could reduce the number of people who use the ED for non-emergent conditions and have more resources for patients with emergent conditions.
Introduction: Behçet’s syndrome is a rare, chronic inflammatory disorder characterized by a myriad of systemic symptoms, most notably including recurrent oral aphthous and genital ulcers. Ocular manifestations, gastrointestinal disease, neurological disease, pulmonary disease and arthralgias can also commonly be seen. The etiology, although unclear, is thought to be consistent with an all-vessel vasculitis with resultant widespread systemic inflammation.

Case: A 34-year-old African American male with a past medical history pertinent for Insulin Dependent Diabetes Mellitus, hypertension, and learning disability who presented with complaints of malaise, nausea, vomiting, and abdominal pain. He was admitted to the inpatient hospital team for management of ketoacidosis (likely mixed diabetic and starvation) and treated conservatively with resolution of his vomiting and abdominal pain. Concurrently, the patient was noted to have intermittent fevers with minimal response to acetaminophen. General infectious workup was negative. Given unclear etiology, a deep chart review was performed, unveiling a prior admission in 2016 for DKA in which the patient was noted to have a scrotal ulcer. Biopsy results demonstrated lymphocytic vasculitis for which he had no follow up or treatment. On a more complete physical exam, two aphthous ulcers in the patient’s mouth and one nontender scrotal ulcer were identified. Rheumatology was consulted and confirmed the likely diagnosis of an active Behçet’s syndrome flare. He was started on 0.6mg of colchicine twice a day which led to the resolution of his fevers and established with outpatient follow up.

Discussion: This case highlights a unique presentation of Behçet’s flares as recurrent DKA admissions. Inflammatory syndromes such as Behçets lead to increased stress and cortisol production, which not only increase gluconeogenesis and lead to hyperglycemia, but can also result in inflammation giving rise to insulin resistance. An alternative proposal could be that the hyperglycemia and subsequent glucose uptake by endothelial cells can cause an inflammatory state secondary to the production of reactive oxygen species. Whether this would be significant enough of a stimulus to cause a flare is not well documented but would be useful in the clinical management of diabetic patients with chronic inflammatory disorders and decreasing recurrent flares.
Case: We present a case of a 72-year-old male with a past medical history significant for CKD 3, emphysema, HTN, hypocalcemia, tobacco use, and OSA who presented with severe weakness and a fall that occurred as he was lifting a package. The patient stated that this muscle weakness had been progressing over the past 2-3 months, to a point where he was barely able to get up from his chair. The patient denied any fevers, vomiting, trauma, diuretic, or statin use during this time. Laboratory tests on admission showed various electrolyte abnormalities including an elevated creatine kinase (12820) and severely low potassium (1.6). ECG displayed flattened T waves and abnormal U waves. He was admitted to the inpatient team for management of his severe hypokalemia and rhabdomyolysis. Further chart review revealed a prior diagnosis of hyperaldosteronism with patient documenting noncompliance with spironolactone and potassium prior to his symptom’s onset. The patient was started on aggressive IV and PO potassium chloride supplementation, IV fluids, and spironolactone. With this treatment, his potassium level increased, CK normalized, and his weakness improved.

Impact/Discussion: Rhabdomyolysis is a dangerous condition in which skeletal muscles are severely damaged, causing toxic metabolites to be released into circulation. It is most commonly the result of traumatic, muscle compression, or drug/toxin effects. However, rhabdomyolysis caused by hypokalemia is an extremely rare clinical manifestation that has been documented in the literature. Primary hyperaldosteronism (PHA) is defined as excess production of the hormone aldosterone in the setting of low plasma renin. In PHA, inappropriately elevated secretion of aldosterone leads to increased sodium absorption and potassium excretion. Hypokalemia caused by PHA (which is less common compared to normokalaemia in this condition) can result in chronic weakness, pain, and fatigue. In rare instances, severe potassium excretion can result in rhabdomyolysis. We described a case of severe hypokalemia due to a patient’s noncompliance with his potassium and spironolactone for his primary hyperaldosteronism.

Conclusion: This is a unique presentation of primary aldosteronism that would be useful in the clinical management of patients with hypokalemia-induced rhabdomyolysis with an unclear etiology. Additionally, this case emphasizes the importance of considering hyperaldosteronism as the cause of hypokalemia induced rhabdomyolysis.
Introduction: Enterococcus faecalis is a gram positive, facultative anerobic cocci usually associated with community-acquired endocarditis, nosocomial urinary tract infections, and biliary tract infections. E. faecalis is rarely associated with septic arthritis, which is usually caused by Staphylococcus aureus, Streptococcal species, and Neisseria gonorrhea.

Case: Patient is a 65-year-old female with end stage renal disease on hemodialysis who presented with worsening bilateral knee pain and swelling. Patient was previously hospitalized one week prior for left knee pain and swelling, at which time a left knee arthrocentesis showed Calcium Pyrophosphate Deposition (CPPD) crystals and Enterococcus faecalis which was thought to be contaminant due to negative gram stain. Patient was thus treated with intra-articular injections of triamcinolone for presumed pseudogout- but pain returned causing her to present again. Imaging showed significant osteoarthritis and R knee effusion. Repeat L and R knee arthrocentesis was again positive for CPPD crystals. Fluid was highly inflammatory with WBC 21,110 and 90% PMNs. Synovial fluid cultures also showed E. faecalis growth. Given concern for septic arthritis, patient underwent bilateral incision and drainage. Blood cultures were drawn to rule out a hematological source given patient was dependent on hemodialysis which were also positive for E. faecalis. Thus, patient was started on vancomycin and underwent a transesophageal echocardiography for definitive rule out of endocarditis, which showed no vegetations or evidence of endocarditis. Permcath was exchanged after a 72 hour line holiday, and catheter tip was sent for culture, showing no growth. Patient completed a six-week course of vancomycin treatment without any complications, and left knee pain which improved overtime with increased ambulation. Repeat blood cultures showed no growth.

Discussion: Here we present a case of bilateral E. Faecalis septic arthritis with concurrent E. faecalis bacteremia. Though E. faecalis is rarely associated with septic arthritis, it is possible that a hematogenous source may have been the source of infection given patients long term dependence on hemodialysis. Furthermore, given that E. faecalis is rarely the causative pathogen for septic arthritis optimal therapy is not known, but this case demonstrated that 6-week course of vancomycin is effective. E. Faecalis was also sensitive to ampicillin and penicillin, however, due to fluid restrictions in an ESRD patient, vancomycin was preferred.
Introduction: Intracranial hypotension typically occurs spontaneous and is typically marked by an orthostatic headache, diffused meningeal enhancement on brain MRI and low cerebrospinal fluid pressure. Though a rare cause, spinal chiropractic manipulation may lead to mechanical disruption of the spinal dural thecal sac leading to a subsequent loss of cerebral spinal fluid (CSF).

Case: Patient is a 53 yo male who presented with a 2 week history of double vision and persistent headache. Patient initially developed severe neck pain leading him to seek chiropractic spinal manipulation. 4 days later patient developed a headache worsened with prolonged standing or sitting, and double vision with an inability to abduct left eye. Patient’s ophthalmologist sent him to the emergency room where workup for vertebral dissection, brain aneurysm and stroke were negative. Fluoroscopy guided lumbar puncture was done which showed an opening pressure of <3 cm H2O, and sufficient sample for CSF studies could not be obtained. MR imaging of the brain was consistent with diffused pachymeningitis, no evidence of brain stem infarcts, or orbital masses were seen. MR of the spine showed diffused dural thickening and enhancement with engorgement of the dural venous plexus- consistent with intracranial hypotension. Subsequently a CT cisternogram and myelogram showed findings concerning for a T1-T4 dural tear. Ophthalmology conferred that left abducens nerve palsy was likely due to the pachymeningitis secondary to the CSF leak from the thoracic spinal dural tear. Patient then underwent an epidural blood patch during his hospitalization which initially provided relief for the headache. However, one week after discharge, his headache returned and consequently patient underwent another epidural blood patch. Gradually overtime, patient noticed an improvement in his headache, resolution of left abducens nerve palsy and subsequent improvement in double vision.

Discussion: Here we present a rare case of intracranial hypotension following spinal chiropractic manipulation, as majority of reported cases of intracranial hypotension occur spontaneously with no clear etiology. This case is also demonstrates that a CSF leak can cause left abducens nerve palsy, likely due to the caudal displacement of the brain that occurs with intracranial hypotension placing traction on the abducens nerve.
Introduction: Hyponatremia is a common lab abnormality with a broad differential. While steroids commonly cause adrenal insufficiency, inhaled glucocorticoids are often overlooked. We present a case of hyponatremia secondary to adrenal insufficiency from inhaled fluticasone.

Case Description: A 67-year-old female with a history of 10+ years of hypertension (controlled on HCTZ and losartan) and mild intermittent asthma on long term fluticasone-salmeterol and fluticasone nasal spray presented for a routine physical. She reported feeling well and had no complaints. Vitals and physical exam were unremarkable while routine labs revealed hyponatremia (Na 131) and hypochloremia (92); potassium and creatine were normal. Her sodium had ranged from 127-133 and chloride 89-95 since 2014, the earliest records on file. Hyponatremia was assumed to be secondary to HCTZ which was replaced by amlodipine. Two months later, follow-up labs showed persistent hyponatremia and hypochloremia. AM cortisol was low (1.8) and subsequent ACTH stimulation test showed an inappropriate response. A Chest X ray was normal while an MRI revealed slight right sided asymmetry of the pituitary gland with a possible 6 mm focus thought secondary to normal variation. Ultimately, she was diagnosed with central adrenal insufficiency secondary to adrenal suppression from her inhaled corticosteroid. Prolonged exposure to inhaled fluticasone resulted in atrophy of ACTH-dependent adrenal cells. Fluticasone-salmeterol was discontinued and the patient was placed on a steroid taper with improvement in her hyponatremia (134) and AM cortisol (5.6).

Discussion: Inhaled glucocorticoid use is a rare cause of adrenal insufficiency with about 1-2% of individuals on inhaled glucocorticoids developing this complication. Red herrings are thus common when evaluating patients for the etiology of hyponatremia with thiazide diuretics contributing in 11% of patients. The risk of adrenal insufficiency with inhaled glucocorticoids varies based on medication dose, administration form and duration and there is a weak association between adrenal insufficiency and higher dose and duration. Medications that inhibit metabolism of glucocorticoids through their effects on CYP3A4, such as diltiazem, itraconazole, and grapefruit juice increase the risk of adrenal insufficiency and management of this condition involves ruling out any secondary causes as well as a steroid taper.
Background: Acquired syphilitic hepatitis is generally an overlooked cause of hepatitis particularly in immunocompetent individuals. Hepatic involvement in syphilis patients is rarely described, with incidence ranging from 0.2 to 3%. We present a case of a healthy immunocompetent male with an elevated LFT who was found to have syphilis.

Case: 27-year-old male with no past medical history presented due to abdominal pain for 2-3 weeks. It was located in the right upper and lower quadrants with radiation to the back. He reported intermittent chills and fatigue. He denied alcohol, drugs and herbal products use. His exam was remarkable for right sided tenderness without rebound. His workup revealed normal CBC. His LFTs were elevated (AST 169, ALT 271 ALK Phos 377) with normal bilirubin. His Lipase was normal. His CT scan abdomen was unremarkable with normal liver and gall bladder, however, there were findings of abdominal and pelvic lymphadenopathy. His Hepatitis A, B, C and HIV (including HIV RNA) was negative. His ANA was negative and ASMA was mildly elevated at 20. EBV and CMV were ruled out as well. His RPR was reactive with reactive TPP IGG-IGM Ab. During hospital course his LFTs trend was worsening. He was managed as secondary syphilis due to additional history of penile lesion and chancre on exam along with abdominal lymphadenopathy. He was given 2.4 million units of Benzathine penicillin. Upon follow up after one week, he reported complete resolution of his symptoms. His LFTs were normalized on repeat check.

Conclusion: Given significant morbidity associated with a missed diagnosis, syphilitic hepatitis should be considered as a part of work up for evaluation of elevated LFTs in an appropriate clinical setting. This case also highlights the importance of obtaining a sexual history and performing genital exam.
112) ANAPHYLACTOID COMPLICATION WITH PLASMA EXCHANGE THERAPY IN MANAGEMENT OF ANTIPHOSPHOLIPID SYNDROME

Ivana Surjancev, MD; Jon Arnason, MD
University of Wisconsin Hospitals and Clinics, Madison, WI

Introduction: Plasma exchange therapies are used often in rheumatologic or hematologic diseases as adjuvant or independent therapeutic treatment modalities. While generally regarded as safe, severe complications can occur including cardiovascular events, respiratory events, and anaphylactoid reactions (estimated incidence of 0.2-0.25% in studied cases).

Case: A 43 year old male with anti-phospholipid syndrome (APS), type 2 heparin induced thrombocytopenia (HIT), and deep venous thromboembolisms presented with diffuse arthralgias and fevers. Notably, patient had recent hospitalization for lower extremity retiform purpura related to his severe antiphospholipid syndrome which continued to be difficult to treat due to his history of HIT and no associated secondary autoimmune etiologies on prior lab evaluations. Infectious work up was unrevealing, and symptoms were ultimately attributed to breakthrough of APS given recurrence of retiform rash during admission. Despite immunosuppressive therapies with high dose intravenous steroids, rituximab, and anticoagulation with fondaparinux (later transitioned to argatroban for concerns of treatment failure), the patient’s symptoms progressed. He had worsening leg pain with rash and acute hypoxic respiratory failure requiring transfer to the intensive care unit for intubation. Bronchoscopy was completed and consistent with diffuse alveolar hemorrhage, raising concerns for progression to catastrophic APS. Labs were notable for low complement levels and a five day course of plasma exchange therapy (PLEX) was initiated with extubation after the second session. Patient developed lip swelling with the third cycle and an anaphylactoid reaction characterized by oropharyngeal edema, rash, and difficulty breathing during his fourth cycle (despite premedication with dexamethasone and diphenhydramine). An epinephrine injection was given with resolution of symptoms. PLEX was discontinued and patient was discharged home on steroid taper and scheduled rituximab infusions.

Discussion: The patient had recurrent admissions and sadly developed avascular necrosis of his knee from ongoing high dose steroids and poorly controlled APS. Patient is now on sirolimus and hydroxychloroquine for management. There is a paucity of literature regarding how to safely trial or resume PLEX after anaphylactoid reactions. For this patient, this means lifelong therapies with immunosuppressive agents with significant side effects and potential for further adverse outcomes while PLEX remains unavailable.
113) AN ATYPICAL CASE OF HEMOLYTIC UREMIC SYNDROME

Steven J. Szlembarski, DO; Douglas W. White, MD
Gundersen Health System, La Crosse, WI

Abstract: This case report takes a look at a likely case of atypical hemolytic uremic syndrome (aHUS). aHUS is a chronic, relapsing disorder characterized by the triad of anemia, thrombocytopenia and acute renal failure. It is a rare disorder accounting for 1-2 cases/million inhabitant of USA. In order to make the diagnosis one must rule out STEC, ADAMTS13 deficiency and secondary causes of TMA. aHUS can be caused by genetic mutations of complement proteins or autoantibodies to complement Factor H leading to complement-mediated endothelial damage. In this case report we take a look at an 85 year-old woman who presented with confusion, falls and lower extremity edema. On admission and during the course of her hospital stay she developed bilateral toe lesions progressing to gangrene requiring bilateral transmetatarsal amputations. The patient also had AKI, anemia with schistocytes, thrombocytopenia and autoimmune workup with weak positive ACL IgM and low C3/C4. The patient’s workup effectively ruled out STEC, TTP and secondary causes of thrombotic microangiopathy, making aHUS a more likely culprit. The patient will require additional workup including analysis of complement genes and Factor H autoantibody to confirm the disease. In conclusion, aHUS is a dysregulation of the alternative complement pathway with high mortality rate after the first episode. A majority of aHUS cases have mutations in one or more complement proteins and some have complement Factor H autoantibodies. Treatment with the C5 inhibitor - Eculizumab - is an effective treatment of aHUS resulting in hematologic normalization, improvement of GFR and reducing the need for hemodialysis.
114) A CASE OF VACCINE-INDUCED PERIMYOCARDITIS ASSOCIATED WITH REDUCED LVEF

Ran Tao, MD; Nicole Reilly, MD
University of WI Hospital and Clinics. Madison, WI

Background: Myocarditis is defined as inflammation of the myocardium. It can be caused by infectious or noninfectious etiology. There have been case reports of vaccines-induced myocarditis, notably by smallpox vaccine, but there is a lack of consensus regarding its treatment.

Case: A 21-year-old male with ADHD (on Adderall) presented with one day of substernal chest pain. Pain is not exertional or pleuritic. The patient received the 2nd dose of Pfizer-BioNTech COVID vaccine 4 days prior to his presentation. He reported fevers and headaches 48-72 hours after the vaccination, which spontaneously resolved. Admission EKG showed diffuse ST segmental elevation. Troponin was elevated to 3.82 ng/mL (normal < 0.03). C reactive protein 3.6 mg/dL (normal <1.0). Nasopharyngeal PCR for COVID-19 was negative. Cardiac MRI was performed on hospital day 1, which showed global left ventricular hypokinesis with an LVEF of 30.4% (RVEF 42.4%). There is a nonischemic pattern of myocardial late gadolinium enhancement and T2 hyperintense signal throughout the septum and partially involving the anteroseptal and inferoseptal walls. Patchy pericardial enhancement was also seen. The finding is consistent with myopericarditis. Endomyocardial biopsy and cardiac cath were not performed. His chest pain resolved on hospital day 2. He was started on colchicine and as-needed NSAIDs. Steroids were deferred due to resolution of his symptom. He was also started on metoprolol succinate and sacubitril-valsartan for reduced EF. Adderall was held on discharge. He is discharged on hospital day 2 and to follow up with cardiology on an outpatient basis.

Discussion: There have been case reports of myocarditis following the COVID vaccination. One source cited the incidence to be around 1/100,000, and it is more common among people under 30 years old. Although endomyocardial biopsy is still considered as the gold standard, CMR has emerged as an attractive alternative for diagnosis of myocarditis. Lake Loise Criteria, recently updated in 2018, can be used to support the diagnosis of myocarditis. It includes edema (T2-weighted imaging), hyperemia (early gadolinium enhancement), and necrosis/fibrosis (late gadolinium enhancement). There are no current recommendations regarding the management of vaccine-associated myocarditis. Most treatment has focused on anti-inflammatory and GDMT if LVEF is reduced.
115) IDENTIFICATION AND TREATMENT OF LEMIERRE’S SYNDROME IN A YOUNG OTHERWISE HEALTHY CAUCASIAN MALE
Francis Tongpalad, BS; Pinky Jha, MD; Nabeel Khan, DO
Medical College of Wisconsin, Milwaukee, WI

Lemierre’s syndrome is a rare infectious thrombophlebitis of the internal jugular vein that typically occurs after Fusobacterium infection of the peritonsillar tissue or palatine tonsils. There are no formal guidelines for anticoagulation therapy once patients are discharged from the hospital. This case highlights the need for formalized clinical guidelines to help clinicians choose the best anticoagulation therapy for their patients.

Patient is a 21-year-old Caucasian male with no significant past medical history who presented to the ED with 7 days of fever, vomiting, and abdominal pain admitted for management of acute hypoxemic respiratory failure, septic thromboemboli of the L internal jugular vein, acute liver injury, and monitoring of cardiac, hematologic, and electrolyte status. Blood cultures were positive for Fusobacterium. He was found to be in septic shock and intubated. CT chest showed multiple bilateral cavitary lesions and a loculated R pleural effusion concerning for empyema. CT neck found thrombophlebitis with partial occlusion of the L internal jugular vein. He was started on IV vancomycin, cefepime and azithromycin. Five days later, a new L-sided pleural effusion was seen on ultrasound and he underwent thoracentesis with L chest tube placement and removal of 1L of serosanguinous fluid. Despite this, his respiratory status continued to decline. CT PE was negative for acute PE but revealed a loculated R pleural effusion. CT surgery was consulted and performed full decortication of the R lung with placement of 2 R chest tubes. He was then started on IV Unasyn. Repeat CT neck showed worsened thrombophlebitis of the L internal jugular vein causing complete occlusion spanning the mandibular angle through the thyroid cartilage levels. He was started on a heparin drip. Over the next few days, his bilateral pleural effusions resolved and chest tubes were removed. His oxygenation status improved, he was extubated, and weaned off of supplementary oxygen. Hematology was consulted, who recommended a 3-month course of either warfarin or DOAC therapy. ID was consulted, who recommended starting oral Augmentin after discharge to complete 4 weeks from time of lung decortication. The patient was subsequently discharged.

Although anticoagulation therapy is recommended if internal jugular vein thrombophlebitis worsens, there is no consensus on the best anticoagulation therapy after discharge. This highlights the urgency to develop guidelines for prophylactic anticoagulation once patients leave the hospital. This will save medical systems money, prevent adverse clinical outcomes, and save lives.
116) CYTOMEGALOVIRUS: NOT JUST A DISEASE FOR IMMUNOCOMPROMISED PATIENTS
Sarah Torres, BS; Adam Koraym, BS; Pinky Jha, MD
Medical College of Wisconsin, Milwaukee, WI

Introduction: The morbidity and mortality associated with cytomegalovirus (CMV) infection in immunocompromised patients such as patients with HIV and transplant recipients on immunosuppressants are well known. CMV in immunocompromised patients can cause severe disease, either by reactivation of latent CMV infection or by primary CMV infection. However, relatively little attention has been paid to the morbidity and mortality that CMV infection may cause in immunocompetent patients. In immunocompetent patients, primary CMV infection typically manifests as an undifferentiated viral syndrome or is expressed as a mononucleosis-like syndrome. CMV infections in immunocompetent hosts have traditionally been considered to have a benign with a self-limited course, nevertheless, this may not always be the case as this report will show.

Case Description: An 86-year-old immunocompetent female presented to the ED with bright red blood per rectum. She was treated with IV antibiotics and was managed using conservative therapy. However, because of her ongoing GI bleeding, and an elevated monocyte percentage (20%) on differential, a flexible sigmoidoscopy with biopsy taken of sigmoid colon and of rectum was done. Biopsies showed ulcerated colonic mucosa with underlying inflamed granulation tissue and marked reactive epithelial changes. Rare positive cells were identified on CMV immunostaining. HIV NAAT was non-reactive, CD4 count was within normal limits, and CMV NAAT was positive. While it is rare to see CMV colitis in an immunocompetent patient, given the severity of the disease and findings on colonoscopy with report of CMV on pathology, it was agreed that we would opt to treat for CMV colitis. Treatment consisted of PO Valganciclovir 900mg two times a day followed by maintenance dosing.

Discussion: While numerous studies about severe CMV disease in immunocompromised patients have been done, there have been relatively few large cohort studies evaluating the incidence and symptomology of severe CMV disease in immunocompetent patients, which while rare, should not be uncharted.
117) WHEN 3 ORGANS FAIL TOGETHER: A DISSECTION OF CARDIO-RENAL-HEPATIC SYNDROME

Danielle N. Villa; Chad Wenzel, MD
Medical College of Wisconsin, Milwaukee, WI

**Introduction:** Vascular congestion due to a deterioration in cardiac function can manifest as heart, liver and renal failure. Challenges lie in diagnosis, ruling out concomitant pathology, and optimizing treatment. Optimization of cardio-renal-hepatic syndrome management can prove difficult as diuretics can improve congestion at the expense of worsening organ perfusion. While using diuretics to treat a patient with worsening renal function may seem counterintuitive, for patients who are hypervolemic with poor cardiac output, diuretics are the mainstay of treatment due to their ability to decrease renal venous pressure.

**Case:** A 75-year-old female with a history of heart failure with reduced ejection fraction of 33% and CKD Stage III presented to an outside hospital with weakness and body aches. Labs revealed acute kidney injury on CKD Stage III, mild transaminitis, elevated INR of 5.0 and lactic acid of 2.9. Workup for acute viral diseases, hemochromatosis, Wilson’s disease, autoimmune hepatitis, alpha-1-antitrypsin, and hypergammaglobulinemia were negative. Abdominal ultrasound and CT scan demonstrated ascites but did not demonstrate any structural causes for transaminitis. Her diuretics and warfarin were held. While her creatinine improved, her transaminases and INR continued trending upward. The patient was transferred to our academic center. Her ALT was subsequently 314, AST 542, alkaline phosphatase 377, and INR was 9.8. Her creatinine was 3.05 (baseline was 1.5), FeNA was 0.2%, pro-BNP 26,900, and LDH was 716. She was initiated on a regimen of metolazone, bumetanide and IV vitamin K. The patient’s INR corrected quickly, and her transaminases and creatinine steadily improved over several days of ongoing diuresis. She was discharged with an ongoing home diuretic regimen.

**Discussion:** This case illustrates the importance of recognizing acute decompensated heart failure that can lead to a decline in liver and kidney function. This patient’s combination of broad transaminase and Pro-BNP elevation with her symptoms suggested a mixed picture of cardiorenal syndrome and congestive hepatopathy. Cardio-renal-hepatic syndrome should be considered in patients with elevated bilirubin, aminotransferases, alkaline phosphatase, INR, and creatinine and a FeNA under 1% in conjunction with heart failure. Loop diuretics are considered first line therapy for patients with volume overload due to heart failure. Diuretics improve cardiac output by decreasing preload and improve GFR by reducing renal venous pressure. In a patient with a history of heart failure who presents with signs of liver and renal failure, it is vital to consider that the source of multiorgan failure may be acute decompensated heart failure and resulting volume overload.
Introduction: Clostridium perfringens bacteremia is a rare syndrome accounting for less than 1% of all bloodstream infections with a significant mortality rate ranging from 27% to 58%. Given the severity of C. Perfringens bacteremia, prompt clinical recognition is necessary. Here we present a case of C. Perfringens bacteremia in the setting of necrotic empyema.

Case: A 55 year old male with no reported medical history presents to an outside hospital with hypotension and hypoxia in the setting of subacute malaise and fatigue for the past month. Labs were notable for a lactic acid of 6.6, white blood cell count of 26.3 and creatinine of 3.59. Given his respiratory distress patient was intubated which was complicated by subsequent PEA arrest. ROSC was obtained, and patient was transferred to our ICU. CT Chest showed bilateral pulmonary emboli, bilateral hydro pneumothoraces, right sided necrotic focus with a pleuroparenchymal right middle lobe fistula and a complex right pleural fluid collection, portal vein thrombus, and a splenic infarct. He had three chest tubes placed for his pleural fluid collections and pneumothoraces. Blood cultures and pleural fluid cultures were obtained, growing gram + rods with eventual speciation to C. Perfringens. ECG showed a LBBB and TTE was obtained which showed a large LV thrombus and a hypokinetic left ventricle with an ejection fraction of 15%. Thoracic Surgery was consulted for consideration of decortication but given his critical illness and multiorgan failure surgery was deferred. Patient underwent bronchoscopy with BAL which was unrevealing. TEE was obtained which showed no evidence of endocarditis but large apical and septal thrombi. A family meeting was held and given patients poor prognosis and extended hospitalization, comfort cares were initiated. Ultimately the nidus for this patient C. Perfringens bacteremia was thought to be likely his necrotic empyemas in the setting of a more indolent process (likely undiagnosed malignancy or prior myocardial infarction) which had been causing his subacute symptoms, thromboemboli, and heart failure.

Conclusion: This case illustrates the complexity and severity of severe Clostridium Perfringens bacteremia leading to multiorgan failure. Sources of infection are typically in the GI tract, but can be elsewhere such as in this case. Early recognition, antibiotics, and source control are essential in treatment.
119) WAUPACA COUNTY (WISCONSIN) MEDICAL TRANSPORT LOCATION AND COST DISPARITIES
Matthew Waldrop
Medical College of Wisconsin, Green Bay, WI

Introduction: Non-Emergency Medical Transportation (NEMT) is transportation service for patients with barriers getting to medical appointments. Many rural areas lack transportation options. 6% of federal transit resources supports rural public transportation, representing 19% of the U.S. population. 46% of Waupaca County, Wisconsin residents reported being late or missing appointments because of a lack of reliable transportation. 87% needed alternative transportation options 1+ times per month. 42% cited medical appointments as top reason they used alternative transportation options. In 2019, 41,097 NEMT trips were taken in Waupaca County.

Methods: Surveys were administered to transportation organizations in Waupaca County via phone call. Survey questions included: contact information, clientele, service hours, vehicle number and type, service area, payment methods, limitations, and required certification.

Results: The map shows the layout of NEMT and healthcare systems in Waupaca County. Cost is increased further from NEMT organizations and healthcare systems. Examples based on most likely transportation service utilized for a 1-hour appointment at the city of Waupaca’s main clinic: Within the city of Waupaca - $22.50, Ogdensburg to Waupaca - $42.50, Big Falls to Waupaca - $96.00

Conclusions: In Waupaca County, NEMT organizations are located near the 3 towns with the largest population densities. Healthcare systems are more widespread than NEMT organizations in Waupaca County. Many rural Waupaca County residents are greater than 15 miles from the nearest NEMT organization. No areas in Waupaca County completely lack options for NEMT, however cost is increased further from NEMT organizations and healthcare systems.
**Introduction:** EBV and CMV are highly prevalent herpesviruses that commonly present in childhood and adolescence. While many EBV and CMV infections are subclinical, primary infections caused by both viruses result in symptoms typically consisting of rhinorrhea, tonsilitis/pharyngitis, lymphadenopathy, splenomegaly and/or malaise. EBV and CMV can cause fever of unknown origin in the adult population, and rarely, adult patients can present with co-infection.

**Case:** A 49 year old man with minimal past medical history was admitted from the ER with 25 days of fever, rigors, cough, and fatigue with a largely negative outpatient work-up prior to presentation. At the beginning of his illness, he reported a sore throat for three days at the same time as his 10 month old twins had colds. His fever persisted reaching a maximum of 102.4F and was associated with rigors and night sweats. He presented to the ER after 18 days of illness. He had a negative covid-19 PCR, negative influenza and RSV swabs, normal chest x-ray, and negative blood cultures. CBC, ESR, and BMP were within normal limits. The patient was treated symptomatically and discharged from the ER. He subsequently saw his primary care provider who prescribed doxycycline. He also tested negative for Lyme IGG at that time. The patient presented for a third time, and a more extensive fever of unknown origin work-up was pursued including flow cytometry and a wide range of infection titers. Labs were notable for a negative heterophile antibody test but significantly elevated CMV IgG, CMV IgM, CMV quantified viral load, EBV VCA IgM, EBV VCA IgG, EBV Nuclear antigen, EBV early antigen antibody, EBV quantified viral load. Based on the patient’s pattern of results, he was felt to have primary CMV with reactivated EBV. During admission the patient continued to have nightly fevers reaching > 102F with associated night sweats. He was discharged with ID follow-up, and at his follow-up appointment, all of his symptoms and mild lab abnormalities had resolved.

**Discussion:** Both EBV and CMV can present as fever of unknown origin in adults. This case demonstrates the utility of CMV and EBV quantification in the setting of a negative heterophile antibody test. Notably, this patient had a positive EBV nuclear antigen level which is often predictive of past infection as it takes several months to form. Therefore, this patient likely has primary CMV infection from his young children with EBV reactivation. Regardless, isolated fever is a unique presentation of both CMV and EBV viremia.
Sarcoidosis is a poorly understood inflammatory disorder characterized by the deposition of epithelioid granulomas in the absence of diseases such as malignancy, tuberculosis, or fungal infections. While the pulmonary system is involved in around 90% of sarcoidosis cases, other organ systems such as skin, spleen, stomach, small bowel, bone, peripheral lymph nodes, and liver can also be involved.

A 56-year-old Caucasian male with a past medical history of type 2 diabetes presented to clinic with multiple chronic symptoms including nightly fevers, fatigue, dysphagia, 35-pound weight loss in the last 3 months, and a 5 year history of cough that was previously investigated with a negative chest CT. Initial lab work was significant for elevated Alk Phos 236 (nml 40-129). ALT, AST, CBC, electrolytes, creatinine were all within normal range. A CT scan of the abdomen was obtained and showed multiple liver lesions, splenomegaly, innumerable osteolytic osseous lesions and associated mesenteric and retroperitoneal lymphadenopathy consistent with metastatic disease. However, CT-guided liver biopsy showed numerous, non-necrotizing granulomas, negative for malignancy. The differential included infectious etiology vs. liver involvement in systemic granulomatous disease (sarcoidosis). Granuloma characteristics were not consistent with autoimmune hepatitis or primary biliary cirrhosis, and although toxins were considered, patient denied any herbal supplements or over the counter medications.

Social history revealed multiple potential infectious exposures. He lived in the Mississippi River Valley but summered in New Orleans including shortly after Hurricane Katrina. He visited a family member in western Arizona years ago shortly before his cough began. He worked as a barn painter and had exposures to paints, chemicals, mold, dust, mice, ticks, bats, and farm animals. Extensive infectious workup was negative for Tuberculosis, Bartonella, Brucella, Q fever, Syphilis, Coccidioidomycosis, Histoplasmosis, Blastomyces, Cryptococcus, Candida, Aspergillus, Pneumocystis, and HIV. Blood cultures showed no growth. With infection ruled out, patient was diagnosed with Hepatic Sarcoidosis and started on high-dose prednisone with the intent to transition to methotrexate or a TNFi.

This case illustrates a peculiar presentation of sarcoidosis with clinically significant hepatic involvement, which is estimated to occur in less than 20% of cases.
Intro: Immune thrombocytopenia (ITP) is a disease process mediated by antibodies which target platelets for destruction and damage megakaryocytes to limit their production. While most cases are idiopathic, vaccines such as the MMR have been noted to be associated with ITP. There have been few reported cases of apparent secondary immune thrombocytopenia following COVID-19 vaccination with both the Moderna and Pfizer vaccinations. Most patients in those case studies responded to treatment with corticosteroids and IVIG. However, ITP secondary to the newly released COVID-19 vaccination is an evolving topic with few cases reported to date. This report demonstrates a rare case of COVID-19 vaccine-induced severe ITP which was refractory to first line treatment.

Case Description: A previously healthy 61-year-old female presented ten days after receiving her second dose of the Moderna COVID-19 vaccination with a two-day history of petechia on her legs, wet purpura on the buccal mucosa and tongue, and light vaginal bleeding. Patient reported no personal or family history of autoimmune or hematologic disorders and denied recent illnesses or sick contacts. Labs on admission were significant for isolated thrombocytopenia with otherwise normal CBC.

The patient was refractory to initial ITP treatment, including IVIG, dexamethasone, NPlate, and platelet infusions, with platelets remaining undetectable for eleven days. Patient was discharged with a platelet count of 18k/L but returned one week later with a platelet count under 5. Vincristine was started and the patient saw rapidly increasing platelet levels within two treatments. This case report demonstrates secondary refractory ITP due to the Moderna COVID-19 vaccination.

Conclusion: There have been increasing reports of associated ITP two weeks following COVID-19 vaccinations. As previously described in the literature, COVID-19 infections are associated with increased coagulation, thrombocytopenia, and thrombosis. Thus, it is important to further investigate any association of hematologic pathologies such as ITP with the COVID-19 vaccines. This case shows an important description of the presentation of vaccine induced ITP. It also demonstrates a case of refractory ITP which did not respond to first line treatment with IVIG and steroids.
Background: The COVID-19 pandemic has disproportionately impacted minority communities, evidenced by higher rates of infection, hospitalization, and mortality. Worse COVID-19 outcomes in minorities communities are inextricably linked to the pervasive effect of structural racism. Structural racism is embedded in the fabric of our systems of housing, education, employment, earnings, benefits, credit, criminal justice, and health care, ultimately manifesting in the creation and persistence of health inequities. Since the beginning of the U.S. vaccine rollout, we have seen a lack of equitable access to the vaccine, especially for people of color. Equitable COVID-19 vaccine access is essential to ending the COVID-19 pandemic and protecting the health of Black and other socially vulnerable populations. However, inequitable access to digital health information and patient portals place these populations at a disadvantage when vaccine access is driven by electronic outreach, perpetuating health inequities.

Setting/Program Description: An urban academic General Internal Medicine (GIM) practice that serves a high proportion of Black and socially vulnerable patients. Patients 65 and older in this clinic without patient portal access are more likely to be Black, rely on public insurance, or live in zip codes with high social vulnerability based on the social vulnerability index. The health system deployed a method of patient outreach largely driven by patient portal notification and scheduling while the GIM practice mobilized community health workers and students to engage in telephonic outreach to patients aged 65 and older without patient portal access.

Program Evaluation: Our team provided outreach to 1007 GIM clinic patients from February 11 to March 9, 2021. During that time, 865 GIM patients completed their first dose of the COVID-19 vaccine at a Froedtert & MCW vaccination clinic; one-third of these had been contacted through the outreach program. A pre-post analysis showed the following changes in the demographics of patients vaccinated at our clinic: Hispanic from 2% to 4%, non-Hispanic White from 88% to 50%, non-Hispanic Black from 7% to 42%, other 3% for both, EMR inactive from 3% to 37%, and high disparity zip code from 14% to 43%.

Discussion: In Wisconsin, Black populations have received 3% of total vaccinations, but are responsible for 7% of cases, 6% of deaths, and comprise 6% of total the population. Milwaukee County has a higher percentage of Black (27.2%) people. Black people have only received 14% of total vaccine doses, and residents in high social vulnerability zip codes have received a lower percentage of the vaccine as of May 10th, 2021. Our outreach intervention has improved access to COVID-19 vaccines for individuals aged 65 and older without patient portal access and, as a result, improved equity in vaccine access for black and socially vulnerable populations; it has also provided a unique and valuable experience for medical students.
A previously healthy 57-year-old man with a history of chronic sinusitis who received the Ad26.COV2.S vaccination 13 days ago without incident presented to the emergency department with left lower extremity swelling and pain, left medial thigh ecchymosis, transient episodes of right hand paresthesia and weakness, severe headache with transient blurry vision, and progressively worsening dyspnea with minimal exertion. These symptoms began the day after endoscopic sinus surgery with septoplasty, turbinate reduction was completed 5 days ago with the first post-surgery symptom being headache. The initial evaluation at another hospital showed normal hemoglobin with severe thrombocytopenia (platelet count, 6,000/mm3). D-dimer was significantly elevated at 114,166 ng/mL FEU, fibrinogen was low at 174 mg/dL, and APPT was within normal limits. SARS-CoV-2 RNA was not detected on RT-PCR assay of a sample obtained with a nasopharyngeal swab. Imaging studies showed left lower extremity deep venous thrombosis, bilateral pulmonary embolism, left frontoparietal subarachnoid hemorrhage and bilateral lacunar cerebellar infarcts. Hematology, concerned for ITP, administered dexamethasone 40 mg IV and 10 mg orally, and 2 units of platelets. The patient was then transferred to our Neuroscience Intensive Care Unit.

The timing of the Ad26.COV2.S vaccine (4-30 days previously), thrombocytopenia, and further workup including extensive occlusive thrombus throughout the superior sagittal sinus and positive PF4 ELISA were consistent with a diagnosis of thrombosis and thrombocytopenia syndrome. Starting on hospital day (HD) two, treatment included a non-heparin anticoagulation bivalirudin (HD2-5), methylprednisolone 1g (HD2-4), plasmapheresis (HD2), and intravenous immune immunoglobin 1 g/kg (HD2-3). On HD5, a prednisone taper starting at 40mg was initiated, and bivalirudin was switched to rivaroxaban, starting with a loading dose of 15mg twice daily for 21 days, and then a maintenance dose of 20mg daily. The patient was stable without further bleeding events and discharged to outpatient hematology follow-up.

As of May 7, 2021 in the United States, 28 cases of TTS following Ad26.COV2.S vaccination (22 female, 6 male) have been reported out of 8,739,657 total shots given, 19 of which had cerebral venous sinus thrombosis (CVST). To our knowledge, there are only 2 published cases of a male presenting with TTS, and this is the 1st case report of a male with TTS and CVST following Ad26.COV2.S vaccination. TTS is an extremely rare condition, and timely and accurate diagnosis and treatment of TTS will improve patient outcomes. While TTS is a clinically serious and potentially life threatening condition, the FDA and CDC have conducted extensive, thorough, and transparent safety reviews and risk/benefit analyses that demonstrate that while there is likely an association between Ad26.COV2.S vaccination and TTS, the risk is very low and the benefits outweigh the risks.
125) CARBAPENEM RESISTANCE IN A CASE OF DISSEMINATED NOCARDIA WALLACEI
Mir Zulqarnain, DO; Ajaya Sharma, MD; Amro Abdelghani, MD
Froedtert & Medical College of Wisconsin, Milwaukee, WI

**Background:** *Nocardia* is a gram-positive, filamentous rod that largely causes opportunistic infections of the skin, lungs, and CNS in immunocompromised hosts. The vast majority of Nocardia species are susceptible to carbapenems. Here, we present a case of carbapenem resistant disseminated Nocardia Wallacei in an immunocompromised renal transplant patient.

**Case:** A 56-year-old female with a history of renal transplant was admitted with pleuritic chest pain and found to have 3 cm lung mass that was biopsied with post-discharge cultures growing Nocardia Wallacei. She was started oral TMP-SMX by outpatient infectious disease however developed skin lesions, worsening headache, nausea, and weakness requiring re-admission. Upon this admission, exam was notable for multiple tender nodules that were new from past admission, as well as new left upper extremity dysmetria and persistent non-focal generalized weakness. Brain MRI was done which revealed numerous ring-enhancing lesions concerning for brain abscesses. The patient was started on high-dose IV Bactrim, Imipenem & Linezolid. Her headache, nausea, weakness, and skin lesions improved slightly, but persisted despite this antibiotic regimen. Culture susceptibilities resulted showing sensitivities to linezolid, ceftriaxone, and TMP-SMX, but resistance to imipenem. The patient was transitioned to CNS dosing of IV ceftriaxone in place of imipenem and this led dramatic improvement in her symptoms. The patient was discharged home with 6 weeks of ceftriaxone and one year of oral TMP-SMX.

**Discussion:** This case demonstrates the classic features of nocardial infections and their predilection to cause disseminated disease in immunocompromised hosts. Nocardia Wallacei is an emerging isolate that has shown resistance to sulfonamides in the past, however, carbapenem resistance has not been extensively reported. The susceptibility profile seen in our case, suggests that it is prudent to cover suspected cases with multiple classes of antibiotics until final sensitivities result.