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2018 Abstracts

Clinical Vignette and Research Competition

63rd Annual Wisconsin Scientific Meeting
Wilderness Resort, Wisconsin Dells, Wisconsin

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Case Based Vignettes

AN ATYPICAL CASE OF ACUTE RENAL FAILURE

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Introduction: Hemolytic uremic syndrome (HUS) is characterized by hemolytic anemia, thrombocytopenia, and renal failure. Atypical or complement-mediated HUS is caused by dysregulation of the alternative complement pathway. We present a case of hereditary atypical HUS caused by a gain-of-function mutation in the gene encoding complement factor 3 (C3).

Case: A 48-year old male with no significant past medical history presented with a 4-day history of abdominal pain, nausea, vomiting, and diarrhea. Physical exam was remarkable for hypertension but was otherwise normal. Laboratory evaluation revealed hemoglobin 10.3 g/dL, platelets 63 K/uL, and creatinine of 14.2 mg/dL. The patient was urgently started on intermittent dialysis. Subsequent workup revealed elevated total bilirubin, elevated LDH, undetectable haptoglobin, elevated reticulocyte count, negative direct Coomb's, low C3 levels, normal ADAMTS13 activity, peripheral smear with moderate schistocytes, and stool negative for Shiga toxin. A kidney biopsy was performed which revealed evidence of thrombotic microangiopathy. The patient was started on plasma exchange for presumed atypical HUS. His kidney function and hemolysis failed to improve, so the patient was initiated on eculizumab. With this, plasma exchange was ultimately discontinued but he remained dialysis-dependent. Genetic studies later revealed elevated levels of soluble C5b-C9 membrane attack complex (MAC) and a heterozygous C3 p.Lys65Gln mutation associated with hereditary atypical HUS.

Discussion: Atypical HUS results from uncontrolled activation of the alternative complement pathway with increased formation of the C5b-9 MAC. Hereditary atypical HUS may result from a gain-of-function mutation in a complement effector gene or a loss-of-function mutation in a complement regulatory gene. The C3 mutation detected in this patient was predicted to lead to decreased binding of the regulatory protein complement factor H resulting in impaired inactivation of C3b. Eculizumab is an anticomplement agent that targets the C5 portion of MAC and can be used to supplement plasma exchange in the treatment of atypical HUS.

SUSAC SNOWBALLS: AN OFTEN MISSED DIAGNOSIS

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INTRODUCTION: A new and relatively unknown syndrome is attacking young persons. The typical presentation is generally non-specific and can include encephalopathy, migraine, weakness, fatigue, vision and hearing loss, and tinnitus. Often misdiagnosed as atypical multiple sclerosis (MS) or Meniere's disease, diagnosis of this rare syndrome is essential, as early and aggressive treatment is required to prevent devastating long-term consequences.

CASE REPORT: A 29 y.o. Caucasian female presented to clinic with paroxysmal, vertical double vision, hand numbness/tingling, palpitations and transient memory impairment lasting 5-10minutes. Basic initial work-up was benign. She returned to clinic two weeks later with transient facial flushing, fingers that turn purple/white, and continued transient forgetfulness. Inflammatory work-up was benign. MRI head was ordered. The following day the patient presented to the ED with a new non-pruritic, non-painful, abdominal rash. Skin biopsy obtained and revealed non-specific findings, consistent with perivascular dermatitis. The patient's MRI returned showing multiple demyelinating lesions within the corpus callosum, suspected to be due to MS. While in the ED, the patient experienced new, non-painful, partial left-sided vision loss. She was found to have a left-sided branch retinal artery occlusion (BRAO). She was started on high-dose oral prednisone and discharged home. Five days later, she presented to the ED with partial right-sided vision loss. Evaluation revealed a right-sided superior nasal BRAO, while on high-dose prednisone. Rheumatology was consulted and upon review of MRI, suspected a rare syndrome known as Susac Syndrome. The patient was started on daily IV methylprednisolone, mycophenolate mofetil, and IVIG infusions every two weeks.

DISCUSSION: Susac Syndrome is a rare disorder characterized by: encephalopathy, branch retinal artery occlusion, and hearing loss. These symptoms are due autoimmune endotheliopathy of the brain, retina, and inner ear. It often affects young women between the ages of 20-40, but has occurred in individuals ranging in age from 9 to 72. Women are three times more likely than men to aquire it. There are four registries in the world for this disease. It is diagnosed with characteristic MRI findings revealing corpus callosum "snowballs", BRAO, and audiogramabnormalities. Optimal treatment is still debated, but steroids, IVIG, and mycophenolate mofetil are the agents most often used. Early diagnosis and aggressive treatment are needed, as deficits can be irreversible.

GLYCEMIC CONTROL GONE WILD

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A 25 year old male with pertinent history of type 1 diabetes mellitus presented to the hospital with diabetic ketoacidosis and liver transaminases greater than 40 times the upper limit of normal. His diabetes was poorly controlled; this was his fourth episode of diabetic ketoacidosis in the previous 12 months. Liver function studies, including bilirubin and INR, were only mildly elevated. He did not show signs of fulminant liver failure. He endorsed mild right-upper quadrant abdominal pain and displayed hepatomegaly on physical exam. A broad workup in the hospital including diagnostic studies for viral hepatitis, autoimmune hepatitis, acetaminophen toxicity, Wilson disease, muscle disorders, and malignant infiltration did not reveal a source. A liver biopsy was performed due to persistent elevation of transaminases revealing accumulation of glycogen within hepatocytes suggesting a diagnosis of glycogenic hepatopathy due to poorly controlled type 1 diabetes mellitus. The patient's transaminitis resolved rapidly with improved glycemic control.

Glycogenic hepatopathy is a syndrome in type 1 diabetics characterized by marked elevations in serum transaminases and hepatomegaly. It is strongly associated with poor glycemic control with recurrent diabetic ketoacidosis in type 1 diabetes, often with hemoglobin a1c greater than 11.0. Glycogenic hepatopathy is not known to lead to adverse outcomes long-term; however, it can be considered a marker of poor glycemic control in type 1 diabetes. Glycogenic hepatopathy should be considered with elevation in liver transaminases and right-upper quadrant abdominal pain in type 1 diabetics.

FEELING BLUE: A CASE OF DIGITAL ISCHEMIA

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The etiology of digital cyanosis requires a broad differential diagnosis, including thromboembolism, infection, Raynaud's and vasculitis. The case presented here illustrates an unusual case of digital ischemia.

A 66 year old male with a history of hypertension, hyperlipidemia, multiple sclerosis, rheumatoid arthritis, and newly diagnosed esophageal intra-mucosal adenocarcinoma presented to an outpatient GI lab for planned endoscopic mucosal resection. In the pre-operative area, his oxygen saturation was low so he was admitted for evaluation of hypoxia. Upon arrival, his fingers appeared gray and were cool to touch. Earlobe measured SpO2 was normal. Further questioning revealed months of fatigue, joint aches, progressive hearing loss, bilateral foot numbness, progressive muscle weakness, and left foot drop. On admission, vitals were unremarkable, CBC showed a leukocytosis to 17.3 and thrombocytosis to 505. CRP was elevated to 26.7 mg/dL, ESR to 124, and CK to 541. The patient's fingers turned dark blue with areas of black near the fingertips. Lesions resembled Janeway lesions however blood cultures were negative and TTE was without vegetations. MR brain revealed acute scattered infarcts. Fundoscopic exam was negative for Roth spots. Biopsy of a digit lesion was performed and empiric high dose steroids with IV Solumedrol 500mg were started for concern of vasculitis. Biopsy confirmed a small and medium vessel vasculitis. The digital ischemia progressed despite steroids prompting urgent plasmapheresis. Paraneoplastic panel, ANA, P-ANCA, anti-myeloperoxidase, Cryoglobulins, Hepatitis B, C, HIV, Lyme, FISH/flow cytometry, JAK2, SPEP were all unremarkable. C-ANCA was positive to 1:320 and Anti Proteinase 3 Antibody was markedly positive to 56 unit/mL, clenching a diagnosis of granulomatosis with polyangiitis (GPA). Patient underwent a total of 6 sessions of plasmapheresis, cyclophosphamide therapy, and prolonged steroid taper with stabilization of his digital dry gangrene.

Diagnosing GPA can be difficult when the typical sites of involvement such as the sinuses, lungs, and kidneys are not involved. The initial symptoms of the disease are often non-specific findings of low-grade fevers, fatigue, weight loss, night sweats. Peripheral nervous system involvement occurs in 15% of patients. CNS involvement occurs in 7-11% of patients. Digital ischemia has rarely been described in GPA.

MYOCARDITIS AND COMPLETE HEART BLOCK AFTER ADMINISTRATION OF PEMBROLIZUMAB

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Introduction: The development of immune checkpoint inhibitors has led to important advances in therapy for solid tumor cancers. Serious immune related adverse events are uncommon but can be life-threatening. Thus, early recognition and treatment is paramount for these patients. We describe a 63-year-old male who developed pembrolizumab induced myocarditis and complete heart block.

Case description: A 63-year-old male was diagnosed with esophageal adenocarcinoma 2 years earlier and was previously treated with combination of chemoradiation and esophagectomy. He was recently started on pembrolizumab as part of a clinical trial due to disease progression. He received a total of two doses, the last one 3 days prior to hospitalization. He described progressive fatigue, palpitations and shortness of breath two days prior to admission. He was found to be alternating between an accelerated idioventricular rhythm at approximately 100 BPM and complete heart block with a ventricular escape around 60 BPM, but hemodynamically stable. Troponin was markedly elevated at 49.71 ng/ml and transthoracic echocardiogram showed mild global left ventricular systolic dysfunction. A cardiac MRI did not demonstrate significant late gadolinium enhancement. Cardiac catheterization showed no angiographic evidence of coronary artery disease and endocardial biopsy revealed a lymphocytic myocarditis with scattered eosinophils. He was started on high dose of methylprednisolone IV and infliximab. His conduction abnormalities did improve over the course of the hospitalization, however he continued to show idioventricular rhythm. He was discharged 6 days later with planned cardiology follow up. Unfortunately, the patient died due to cardiac arrest about a month after discharge from the hospital.

Discussions: Adverse autoimmune events due to immune checkpoint inhibitors are wide ranging and usually well tolerated. The mechanism is thought to involve aberrant activation of autoreactive T cells. However, serious life-threatening adverse have been reported, including fulminant myocarditis. Optimal treatment remains unknown, but high dose corticosteroids, infliximab, mycophenolate and anti-thymocyte globulin have all been utilized. Currently there are no guidelines for monitoring for serious adverse events. Further characterization of these serious immune related adverse events is needed.

A RARE CASE OF COINFECTION WITH JAMESTOWN CANYON AND POWASSAN VIRUS CAUSING MENINGOENCEPHALITIS

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BACKGROUND: Arboviruses are viruses that are transmitted through the bites of mosquitoes, ticks, or sandflies. Recently, Jamestown Canyon and Powassan viruses have emerged as increasingly important arboviruses that can cause human disease in North America.

CASE DESCRIPTION: A 79-year-old woman with history of being in the farm and having tick and mosquito bite was admitted to outside facility (OSF) with complaints of generalized weakness and headache. She also developed fever and became confused. With concern for meningitis she had Lumbar Puncture (LP) which showed lymphocytic pleocytosis with normal glucose and increased protein. She was treated for meningo encephalitis with acyclovir, ampicillin, ceftriaxone and vancomycin. However she continued to remain encephalopathic and was transferred to our hospital. On arrival to our ICU patient was drowsy. She mumbled but was not able to give any history. She opened her eyes when spoken to and was moving all her extremities. GCS was 11. Cranial Nerves were intact without signs of meningeal irritation. She was continued on acyclovir and ceftriaxone. CT and MRI head performed in the OSF were unremarkable for any acute change. CSF studies repeated in our hospital were negative for WNV, HSV, Enterovirus, VDRL, Lyme, and TB. Tests were also negative for vasculitis and paraneoplastic process. Patient was started on steroids for presumed immune mediated process. Work up was done for occult malignancy with CT Chest, Abdomen and Pelvis which were negative. Cerebral angiogram showed no signs of vasculitis. Patient showed some improvement with steroids however she was having a waxing and waning course. At the time of discharge (3 weeks hospital stay) she was about 90 percent back to her baseline. Her CSF arbovirus result eventually came back (CSF samples had to be sent to UW Madison) positive for Jamestown Canyon and Powassan virus. Jamestown Canyon IgM Ab was 16.366 and Powassan IgM Ab was 16.672. To our knowledge this is the first reported case of co infection with Jamestown Canyon and Powassan virus in Wisconsin.

DISCUSSION: Given the increased incidence of Jamestown Canyon and Powassan virus in recent years in Wisconsin, clinicians should be aware of their potential resultant illnesses. Although most viral infections result in mild illness, this case highlights that these viruses can cause severe neuroinvasive disease. Patients who seek medical care for febrile or encephalitic clinical symptoms and who have possible or known exposures to mosquito or tick vectors should be considered for arboviral testing. Unfortunately, there are currently no proven disease-modifying therapies for these arboviral diseases, so treatment is largely supportive.

WEAKNESS, SLURRED SPEECH, AND HEADACHE; COMMON SYMPTOMS, UNCOMMON DIAGNOSIS

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Primary CNS vasculitis is a rare disease. It's varied presenting symptoms, very low prevalence, and non-specific symptoms pose a diagnostic challenge to providers. Average time to diagnosis is greater than 1 year, potentially with grave consequences.

A 60-year old previously healthy male presented with left sided weakness and headache that worsened gradually over a week. He demonstrated inferior quadrantanopsia. CT and CTA of the head were negative. MRI showed slight enhancement thought secondary to artifact on the right, not anatomically correlated with clinical findings. He had normal white count and ESR of 30. Lumbar puncture was performed and the patient was placed on Vancomycin, Ceftriaxone, Ampicillin, Acyclovir, and Dexamethasone. CSF showed mild pleocytosis with 12 TNC's. Viral PCR, cytology, and cultures were negative. His symptoms completely resolved the following day and he was discharged with a 1 week steroid taper.

He presented again with the same symptoms on day 5 of his taper, now with much worse right-sided, temporal headache. Repeat ESR was 12. Temporal artery biopsy showed vaso vasorum vasculitis. Repeat MRI demonstrated clear right leptomeningeal enhancement. We resumed high dose steroids.

Biopsy of the leptomeningeal tissue as well as cortex showed Granulomatous Angiitis of cerebral cortical arteries with extensive mural fibrinoid necrosis. Immunohistochemical stains revealed extensive vascular and parenchymal accumulation of Beta-amyloid, these findings are consistent with Cerebral amyloid angiopathy-related inflammation (CAA-ri)/ ABeta-Related Angiitis (ABRA). Chest/Abdomen/Pelvis CTA was free of small vessel disease. The patient was placed on Cyclophosphamide and his symptoms completely resolved.

This case shows the need to broaden differential diagnosis when common diagnoses are excluded. It also demonstrates a rare facet of an already rare disease with an extracranial manifestation of temporal artery microvascular disease. Early recognition of this disease is crucial to maintaining function and reducing mortality.

ATYPICAL PRESENTATION OF DISSEMINATED BLASTOMYCOSIS

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An 85-year-old male from Wisconsin presented with four days of sharp left foot pain that was erythematous and warm to the touch. Pain was around the midfoot with no signs of skin penetration or scratches and no pruritus. The patient reported an injury to his ankle three months prior to admission. He also described himself as an avid gardener. An x-ray showed no osseous abnormalities. The patient was initially started on Keflex for presumed cellulitis then transitioned to IV Vancomycin due to lack of clinical response. The foot pain persisted. A bone scan was completed with findings concerning for osteomyelitis. An MRI was unable to be obtained due to a non-MRI compatible ICD. A CT scan revealed two rim-enhancing fluid collections measuring 5-7mm each in the plantar surface of the midfoot consistent with micro-abscesses. Osteomyelitis could not be ruled out at that time. ESR and CRP were found to be 118 mm/h and 173 mg/L respectively. The patient was discharged on IV Vancomycin for presumed osteomyelitis.

The patient returned six days after discharge with increasing left foot pain, swelling and erythema, and inability to bear weight on the foot. Two days prior to re-admission, the patient noted several 0.3-1cm turbid, pustular lesions on his hands as well as a 3x2 cm tender, erythematous shin lesion. A scraping of the lesion sent for culture did not demonstrate any growth. A punch biopsy was obtained due to progression of the lesions.

One week into his second admission he developed progressive hypoxia over a 48-hour period requiring 3-5L of oxygen via nasal cannula. Audible wheezing was noted on physical exam. A chest x-ray was obtained and showed diffuse reticulonodular lung lesions. A chest CT demonstrated diffuse, bilateral ground glass opacities. Urine blastomycosis and histoplasmosis were sent and found to be positive for blastomycosis. Final results of the punch biopsy were also consistent with blastomycosis. A seven-day course of Amphotericin B was initiated due to presence of systemic blastomycosis. With treatment, patient returned to room air with improvement of the left foot pain, shin lesion, and hand bullae. Patient was subsequently placed on an extended course of oral itraconazole.

This case illustrates the potential for non-respiratory symptoms as the primary presentation for disseminated blastomycosis in an immunocompetent patient. Although respiratory distress is the predominant sign of blastomycosis, it is crucial to recognize other systemic signs such as osteomyelitis and skin findings that might alert a provider to begin treatment for blastomycosis.

HYPERCALCEMIA IN A PATIENT WITH PNEUMOCYSTIS PNEUMONIA

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INTRODUCTION: Hypercalcemia is a potentially life-threatening condition that has many possible etiologies. Appropriate workup is needed given the breadth of causes for hypercalcemia and its physiological consequences. Recognition of granulomatous inflammation as an etiology is important, especially as the use of immunosuppressive agents becomes more prevalent.

CASE: A 74-year-old woman with a history of kidney transplant due to hypertensive nephropathy was found to have a non-hemolyzed calcium value of 17.5 mg/dL and CMV viremia on outpatient labs. On admission, the patient endorsed increased fatigue, mild dyspnea, constipation and decreased appetite over recent weeks. While inpatient, the patient's calcium was rechecked to be 14.0 mg/dL. PTH levels were obtained, which were found to be low at 21 pg/mL, while 1,25 dihydroxyvitamin D was high at 126.0 pg/mL. The patient was treated with intravenous fluid, furosemide, and valganciclovir while causes of her hypercalcemia were worked up. PTHrP was undetectable, while CBC, ALP and TSH values were within normal limits. Hypogammaglobulinemia was reported on SPEP, while results from UPEP, serum light chains and EBV by PCR were unremarkable. A CT scan of the patient's chest, abdomen, and pelvis was obtained to investigate for malignancy. The imaging studies showed diffuse ground-glass opacities in the patient's lungs bilaterally. Histoplasmosis antigen, galactomannan, and 1,3-ß-D-glucan studies were ordered to investigate for infectious etiologies. While the fungal studies were pending, the patient's calcium decreased to 11.0 mg/dL and the patient was discharged. Days later the 1,3-ß-D-glucan study was positive, and an outpatient bronchoscopy was scheduled. Biopsy samples were found to be positive for Pneumocystis jirovecii. The patient was instructed to return for inpatient management, where her calcium was rechecked to be 12.9 mg/dL. The patient was treated with Bactrim and prednisone, along with continued valganciclovir, and later discharged on Bactrim and furosemide. Her serum calcium returned to normal levels on outpatient monitoring. At this time, she is awaiting a follow-up appointment for her SPEP result.

DISCUSSION: This case illustrates the potential of Pneumocystis jirovecii pneumonia as being a direct, or contributing, cause of hypercalcemia in patients. Given the multiple etiologies of hypercalcemia, a thorough diagnostic approach is important to consider based on the patient's clinical scenario.

AN UNUSUAL PRESENTATION OF WERNICKE'S ENCEPHALOPATHY AFTER BARIATRIC SURGERY

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Introduction: The use of bariatric surgery for obesity management has increased recently, so has the incidence of thiamine deficiency. Clinicians should be aware of its uncommon presentations and complications. This case demonstrates an unusual manifestation of thiamine deficiency after sleeve gastrectomy.

Case Description: A 34-year-old female presented to the emergency department (ED) with intractable nausea, vomiting, paresthesias, blurred vision, and generalized weakness. She had a sleeve gastrectomy four months prior and had been to multiple EDs for these symptoms without relief. Upon this admission, exam was notable for bilateral horizontal nystagmus without ophthalmoplegia, bilateral lower extremity sensory loss, and intermittent confusion. Esophagogastroduodenoscopy revealed chronic gastritis. She was started on IV proton pump inhibitor, multivitamin, calcium citrate, vitamin D, and sublingual B12 without relief of her symptoms. Brain MRI revealed T2 mild enlargement of the mamillary bodies and potentially more subtle involvement of the hypothalamus. Given her history, exam and MRI findings, Wernicke's encephalopathy was suspected and IV thiamine was started (thiamine level came back low at 17 nmol/l). All symptoms resolved except weakness. MRI of lumbar spine demonstrated L5-S1 left posterior disc bulge impinging on left descending S1 nerve root. EMG showed absent sensory nerve potentials. Serum ceruloplasmin, RPR, TSH, folate, B12, zinc, copper, SPEP, and ANA were within normal limits. She continued to work with therapy and was discharged home with home therapy.

Discussion: Thiamine deficiency occurs following bariatric surgery due to persistent vomiting or change of gut flora. Wernicke's encephalopathy typically presents with the classic triad of ataxia, delirium, and ophthalmoplegia and is often only considered when the patient is an alcoholic. This patient presented with paresthesias, bilateral lower extremity weakness, and horizontal nystagmus. Bariatric surgery was the cause of her thiamine deficiency. Clinicians need to be aware of atypical presentations of Wernicke's encephalopathy and any condition resulting in poor nutritional status, including bariatric surgery, places patients at risk. Patients need to be educated about this potential complication and have appropriate supplementation prescribed after surgery.

WHAT'S IN COMMON BETWEEN MUSCLE BREAKDOWN AND BLEEDING DISORDER – A CASE STUDY

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Introduction: Synthetic marijuana has recently been reported to cause life-threatening coagulopathy by addition of Vitamin K antagonists.

Case Presentation: A 37-year-old male with history of well controlled HIV disease on HAART presented with severe rhabdomyolysis and acute kidney injury. He denied traumatic events or any other known triggers. He denied illicit drug or alcohol use. Despite aggressive hydration, patient's CPK continued to rise for several days though his kidney function returned to baseline. Since there were no identifiable factors that may have contributed to the development of rhabdomyolysis, patient's HIV medication (elvitegravir/cobicistat/

emtricitabine/tenofovir combination - Genvoya) was held as a possible culprit. Patient was discharged home once CPK remained on the downtrend.

The patient presented back to the hospital 7 days after discharge with gingival bleeding, gross hematuria, and flank pain. In addition to urinalysis significant for blood, lab testing revealed undetectable INR and PT as well as PTT of 174. Patient denied any new medications aside from restarting HAART 3 days prior to admission. He denied any illicit drug use. Further testing showed severe deficiency of vitamin K dependent factors and negative mixing study (no inhibitor). Patient was treated with FFP and Vitamin K with improvement in coagulation studies, but without additional FFP both PT and PTT soon rose again and remained prolonged until they normalized by day 8 of admission. Genvoya was held initially but then restarted in consultation with ID as there have been no reported cases of coagulopathy induced by this medication or any of its components.

Conclusion: It is remarkable how our patient developed severe rhabdomyolysis followed by life-threatening coagulopathy without any attributable cause. Both conditions had prolonged, fluctuating course. Case reports suggest that these conditions may be seen in patients who have been exposed to Synthetic marijuana. Anticoagulant screen revealed presence of Brodifacoum, a Vit-K antagonist found within rat poison but more recently reported in cases of synthetic marijuana.

A CASE SERIES: HERPES ZOSTER MENINGITIS IN IMMUNOCOMPETENT PATIENTS

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Abstract: Herpes zoster (HZ) manifesting as acute meningitis in a young, healthy, immune-competent individual is extremely rare. HZ reactivation that manifest into meningitis is a disease process that preferentially affects patients with compromised immune systems. The reactivation of HZ accounts for 2.5-11% of cases involving aseptic meningitis. Here we report 2 healthy, immunocompetent males in their 20's who developed HZ meningitis with a characteristic skin rash in a craniocervical dermatomal distribution and one 60 year old immunocompetent female with a disseminated zoster rash and meningitis. All were treated with IV acyclovir followed by transition to PO valacyclovir. HZ meningitis is an underreported and unrecognized diagnosis. There is no published data to support the use of acyclovir to modify the disease course of HZ meningitis. Therefore, young age, immunocompetence, and the late development of a HZ rash, in uncomplicated cases, should merit consideration for supportive treatment in individuals that meet the above criteria. There is a need for more study to establish treatment guidelines.

Introduction: Viral infections account for the most common form of meningitis, which is characterized as inflammation of the layers of tissue overlying the brain and spinal cord.1 Herpes Zoster is the least common viral etiology for meningitis and infection tend to remain latent in cranial nerves, dorsal roots, and autonomic ganglia with the ability for reactivation. The cell-mediated immune system eradicates the initial viral infection, producing memory cells that prevent re-infection in healthy individuals. Reactivation of HZ presents as painful, dermatomal, vesicular papules with a variety of complications.[1] Risk factors for reactivation include advanced age, immune suppressed state, trauma, malignancy, disorders of cell-mediated immunity, and chronic lung or kidney disease.[2] Our series describe two cases of young immunocompetent males who developed HZ meningitis with a characteristic reactivation of the virus in a dermatomal distribution on the head and one immunocompetent female who developed disseminated HZ and meningitis.

Conclusion: Central nervous system involvement is an uncommon sequela following VZV infection but can be a potentially life-threatening complication. The early recognition of neurological involvement should prompt acute and appropriate antiviral treatment. Furthermore, HZ meningitis is a rare complication of varicella infection. Low prevalence may be attributed to a lack of recognition and reporting of this disease.

Research Based Vignettes

CLINICAL AND SOCIODEMOGRAPHIC PROFILE OF PATIENT WITH THYROID STORM: A NATIONAL INPATIENT SAMPLE DATABASE ANALYSIS FROM 2012-2014

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Introduction/Background: Thyroid storm is a rare, life threatening complication of thyrotoxicosis. It is associated with significant mortality if not treated on time. The clinical outcome of these patients has not been evaluated in general practice on a national scale. The aim of our study was to find the clinical characteristics and outcomes of patients with thyrotoxicosis using the National Inpatient Sample (NIS) database.

Methods: We selected adult patient (≥18 years of age) diagnosed with thyrotoxicosis utilizing the NIS database between 2012 to 2014 The NIS is the largest all-payer inpatient care database in the United States, containing data on more than seven million hospital stays. All categorical variables, including demographic and comorbidity variables, were analyzed using Pearson's chi-square, while statistical analysis was done using SAS v9.4. The level of significance was chosen for $p \leq 0.05$.

Results: In total, 1966 hospitalizations with a primary or secondary diagnosis of thyrotoxicosis were identified. The mean age was 44.4+/-18.0, 524 were males (26.7%), 818 (43.6%) were Caucasians and 864 (44.0%) were less than 30 years old. Overall, inpatient mortality rate was 4.2%. On univariate analysis, age ≥ 60 years, male gender, electrolytes disturbance and those with known pulmonary or neuromuscular disease were found to be factors that were associated with significantly higher rates of in-hospital mortality.

Discussion: This large retrospective study utilizing a large national database from the United States showed an overall mortality rate of 4.2 % in patients with thyroid storm. It highlights the importance of early diagnosis and prompt treatment and close clinical surveillance, especially in those who are elderly, male, have electrolytes disturbances or have known pulmonary or neuro-muscular disease. Our study is large and comprehensive and is comparable to the general US estimates but is limited by lack of prospective study design.

PREDICTORS OF RESPONSE TO PLATINUM-BASED CHEMOTHERAPY IN METASTATIC CASTRATE-RESISTANT PROSTATE CANCER (MCRPC)

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Background: Aggressive variant PC (AVPC) is a well-described subtype of PC which portends a poor prognosis. Studies have shown an increase in progression-free survival(PFS) when Pt is added to taxane-based treatment in AVPC. Clinical features including visceral metastasis, lytic bone lesions, low PSA, short responses to androgen suppression and high Gleason score at diagnosis predict response to upfront docetaxel (D) and Pt (Aparicio et. al. Clin Cancer Res 2013). Our objective was to study the effect of baseline characteristics on PFS with D+Pt in D-pretreated CRPC.

Methods: A retrospective review of D-pretreated mCRPC men who received D (60-75 mg/m2) and carboplatin (C, AUC 4-5) at our institution between 2008- 2015 were included in this analysis. All patients had metastatic CRPC, were heavily pre- treated (median treatments = 4) and received at least one cycle of D+C. Numerical data was analyzed using Student's T-test; binary data was analyzed with Z-proportions test.

Results: 28 patients were identified. The median age was 60 yrs(48-73); median PSA at diagnosis was 21ng/dl(0.3-5000); median PSA at the start of D+C was 115.32 ng/dl(0.65-1395). The response rate was 60.7% and the median time to response was 1 mo. The median reduction in PSA was 48.13%. The median PFS was 6 mo, and median OS was 10 mo. Common treatment – related side effects included grade 1 fatigue (82.14%), pancytopenia (42.86%), and febrile neutropenia (10.71%). Correlation of baseline characteristics to PFS was evaluated. Short response to ADT < 1 yr ($p < 0.05$), prior response to D (PFS > 6 months) ($p = 0.037$), and presence of visceral mets ($p < 0.05$) predicted better response to D+C. Age ($p = 0.27$), Gleason score at diagnosis ($p = 0.08$), performance status at start of therapy ($p = 0.27$), PSA at diagnosis ($p = 0.35$), and PSA at start of D+C ($p = 0.420$) did not predict response.

Conclusions: D+C is effective and safe in D pretreated CRPC. Response to prior D, short response to ADT, and the presence of visceral metastasis predicted response to D+C. These are features of AVPC. Prospective studies to validate our findings and identify molecular characteristics of Pt responders are needed in this heavily pre-treated "real world" patient population.

TRENDS OF ALL-CAUSE MORTALITY BY SEVERITY OF HYPONATREMIA AMONG HOSPITALIZED PATIENTS IN A TERTIARY REFERRAL HOSPITAL OVER A FIVE YEAR PERIOD

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Gap: Hyponatremia is common among hospitalized patients. Patients with severe symptomatic hyponatremia (serum sodium≤120 mEq/L) are often aggressively treated to prevent mortality but little is known about mortality from moderate and mild degrees of hyponatremia.

Methods: We obtained data from 76,323 patients with 137,354 hospitalizations from January 1, 2012 to December 31, 2016 at a tertiary referral hospital in Central Wisconsin. Of these, we examined data from 16,115 unique patients with 24,631 hospitalizations who had admitting serum sodium <135 and/or prior history of hyponatremia. We classified them into four categories based on their admitting serum sodium as mild (130-134), moderate (125-129), profound (<125) degrees of hyponatremia, and the 'hyponatremia history' group with a serum sodium that was normal (≥135) during their index hospitalization. We obtained their vital status (alive or deceased) at 6 month intervals from the end of study period on June 30, 2017, and December 31, 2017.

Results: Hyponatremia occurred in 17.9% of total hospitalizations during the study period. Of the 24,631 hospitalizations in the 16,115 patients studied, there were 19,814 (80.4%), 2,544 (10.3%), 614 (2.5%) and 1,659 (6.7%) hospitalizations with mild, moderate, or profound degrees of hyponatremia and the 'hyponatremia history' group, respectively. Proportion of all-cause deaths at 6 months and 1 year later for mild hyponatremia were 37.3% and 39.7% respectively, for moderate hyponatremia were 45.6% and 48.6% respectively, for profound were 50.2% and 53.9% respectively, but were 60.9% and 63.8% for the hyponatremia history group respectively. These results were robust when broken down by year, follow up time (6 months versus 1 year) and gender.

Conclusions: A large number of all-cause mortality including cardiovascular disease, stroke, cancer, liver cirrhosis deaths were occurring to a significant proportion even in patients with mild and moderate degrees of hyponatremia. This may be occurring because milder degrees of hyponatremia are not as aggressively treated as profound hyponatremia as they are not commonly thought to be associated with neurologic and/or fatal sequelae. Surprisingly, a prior history of hyponatremia showed a trend towards worse mortality outcomes compared with current hyponatremia even when sodium levels were normal in the index hospitalization. These findings will need to be confirmed with additional research and explore potential mechanisms that contribute to death in those with milder forms of hyponatremia, and whether correcting sodium levels in those with milder degrees of hyponatremia may prevent further deaths in the future.

Displayed Posters

1) NITROFURANTION INDUCED METHEMOGLOBINEMIA

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Introduction: Acquired methemoglobinemia can occur from a variety of medications and ingestions. Exposure to the offending agent will alter the hemoglobin by oxidizing ferrous iron to ferric iron which is unable to reversibly bind oxygen. If the level of methemoglobin reaches a critical threshold causing impaired oxygen delivery this can be a severely detrimental or even fatal illness.

Case: 25 year old female presenting with dysuria, fever, and flank pain concerning for pyelonephritis. Her initial dysuria developed five days prior and she was prescribed nitrofurantoin for a urinary tract infection through an online medical service. She additionally was taking phenazopyridine for symptom relief. Her symptoms continued to worsen and she developed dark urine. She presented to emergency services where initial evaluation included fever, hypoxia requiring supplemental oxygen, elevated white blood cell count, and urinalysis concerning for infection. Given her prior history of myasthenia gravis and worsening hypoxia there was concern for potential myasthenia gravis crisis so she was given an extra dose of pyridostigmine before transfer to our facility for admission. On arrival she was with worsening hypoxia saturating at 82% on non-rebreather mask. Arterial blood gas (ABG) revealing methemoglobin level elevated at 15.8%. Since this level was <20 and she was asymptomatic she was treated with supportive cares of oxygen and intravenous fluids. Her labs additionally revealed mild hemolysis with elevated reticulocyte and low haptoglobin so testing for glucose-6-phosphate dehydrogenase (G6PD) was not obtained as would potentially be falsely low. She recovered to baseline after treatment for pyelonephritis with cephalosporins. She was discharge home with close hematology follow up for evaluation of G6PD.

Discussion: Methemoglobinemia can be accurately diagnosed with an arterial blood draw. At high levels (>20) or in symptomatic patient treatment with methylene blue or ascorbic acid should be initiated as severity of impaired oxygen delivery can be fatal. However, one significant contraindication for use of methylene blue is G6PD deficiency so all patients should be further evaluated for this following the acute episode.

2) ACUTE SUPPURATIVE THYROIDITIS IN DISGUISE

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Acute suppurative thyroiditis (AST) is a rare life-threatening emergency accounting for 0.1-0.7 % of all thyroid pathology. The thyroid is resistant to infection due to increased iodine concentration, robust blood/lymphatic supply and an impervious capsule. Without treatment mortality is 12%.

We report a case of 55-year-old Hmong male with long standing goiter and chronic hepatitis B, who developed AST of left lobe and Streptococcus constellatus bacteremia. He presented with a four-day history of sore throat, neck pain, fever, chills and myalgias. He was seen twice for same complaints: initially treated for influenza with Oseltamivir & later with oral prednisolone for severe pharyngitis. His symptoms (except fever which subsided with prednisolone) worsened, causing troublesome dysphagia. He had no stridor or dysphonia. Upon his third visit he appeared toxic, was tachycardic, and had a large, firm, tender thyroid, left lobe larger than right, with pitting neck edema. Radiograph of his neck revealed stable calcified rim in the anterior neck with tracheal deviation. New soft tissue gas was noted along the anterior margin. CT revealed a large left thyroid nodule with irregular rim enhancement, internal gas and inflammation without evidence of tracheal / esophageal fistula. Labs were significant for leukocytosis with neutrophilia and monocytosis. Electrolytes and TSH were within normal limits. The thyroid abscess was aspirated and sent for culture and cytology. Empiric antibiotic therapy with Zosyn was initiated. US guided core biopsy excluded anaplastic carcinoma. ½ blood culture bottle yielded Streptococcus constellatus. Aspirate of the thyroid yielded mixed aerobic and anaerobic flora consistent with oropharyngeal flora (Streptococcus mitis / oralis / constellatus / gordoni). The patient improved clinically and completed four-week course of antibiotics. He underwent elective left hemi thyroidectomy and isthmusmectomy four months later. Final tissue sample was negative for malignancy.

In our case we emphasize the fact that initial presentation of AST, although rare, may mimic symptoms of acute pharyngitis, delaying diagnosis. Initial presentation may be precipitating factor, such as URI and presumptive therapy (such as steroids) may even intensify the underlying infection. There should be an enhanced index of suspicion in patients with preexisting thyroid disorder or immunosuppression.

3) DELAYED DIAGNOSIS OF HODGKIN LYMPHOMA

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Introduction: Hodgkin lymphoma is a malignant mass arising from B cells composed primarily of reactive cells and accounts for approximately 10% of all lymphomas. There is a bimodal age distribution with most of the cases seen in patients under 20 and greater than 65 years of age. We present a patient who does not fall in this epidemiological cohort who repeatedly interacted with the health care system with an enlarging neck mass over a two year time period.

Case Description: A 55-year-old morbidly obese female presented with an enlarging neck mass. She initially presented to her primary care physician two years ago with complaints of several weeks' history of enlarging left-sided neck mass. She was given a clinical diagnosis of a left anterior neck lipoma. She was seen on five occasions over the next two years for routine annual visits and outpatient complaints. On each occasion, a decision was made to monitor the neck mass. Review of her chart revealed a decline in weight from 397 pounds to 320 pounds over the same time period. She eventually presented to our hospital with complaints of fatigue, unintentional weight loss, cough, progressive weakness and numbness of bilateral legs and hands, and recent falls.

Laboratory studies were significant for progressive anemia (hemoglobin 7.9 g/dL). Physical exam revealed an impressive 'lemon-sized' mass in left supraclavicular region. Initial chest x-ray showed a mass-like opacity in left hilum. Computed tomography revealed a 7.2 cm by 3.3 cm anterior mediastinal mass encasing blood vessels and extending into the neck with critical contact of major structures, along with marked cervical lymphadenopathy. Biopsy revealed multiple fragments of fibrous tissue and skeletal muscle with an infiltrative inflammatory proliferation composed of an admixture of histiocytes, lymphocytes, occasional eosinophils and rare plasma cells. Diagnosis was confirmed to be Hodgkin lymphoma with paraneoplastic syndrome. ABVD chemotherapy was initiated.

Discussion: Any painless, progressively enlarging supraclavicular mass needs further evaluation, particularly if other concerning symptoms such as weight loss and fatigue are present. Special attention should be paid to capture unintentional weight loss in morbidly obese patients.

4) REITER SYNDROME SECONDARY TO DISSEMINATED GONOCOCCAL INFECTION

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Introduction: Reactive arthritis is a seronegative spondylarthropathy most commonly associated with preceding gastrointestinal or urogenital infection. Reiter syndrome is a specific presentation of reactive arthritis that includes the classic triad of urethritis, arthritis, and conjunctivitis.

Case Description: A 29-year-old male with past medical history of hypertension and disseminated gonococcal infection requiring four weeks of IV Ceftriaxone presented with increasing back and bilateral knee pain, along with penile discharge persistent since initial gonorrhea diagnosis. Patient states he had similar pains before which resolved while he was on antibiotics, and they restarted a few days after the course was completed, approximately two weeks prior to this admission. He presented to the ED due to progressively worsening knee pains, left more than right, to the point where he was unable to ambulate. Of note, patient's right knee had been previously diagnosed with septic gonococcal arthritis, along with reactive arthritis in right elbow and third metacarpophalangeal joint. Labs revealed no leukocytosis, ESR 98, CRP 12.90, Chlamydia negative, Gonorrhoeae positive (followed by negative result on repeat). IV Ceftriaxone was restarted due to suspected treatment failure, and the Infectious Diseases team was consulted. Based on patient's prior aggressive antibiotic therapy, lack of sexual intercourse since treatment and inconclusive lab results, ID recommended to discontinue Ceftriaxone. Due to patient's ongoing penile discharge and new onset arthritis in lower extremity, Rheumatology team was consulted to assess for reactive arthritis. Mild conjunctivitis was appreciated on exam, along with balanitis. Synovial fluid evaluation from left knee showed 5150 WBC and negative gram stain, favoring inflammatory versus infectious etiology. This constellation of symptoms substantiated diagnosis of Reiter Syndrome and patient was started on Indomethacin, Sulfasalazine, and Methotrexate.

Discussion: Though Reiter syndrome has historically been interchangeable with reactive arthritis, it is rare for it to classically present with that specific triad of symptoms. Thus, it is important to consider reactive arthritis, even in patients with coexisting septic gonococcal arthritis who have received prior aggressive antibiotic therapy.

5) KAPOSI STRIKES BACK: HOW A ONCE-COMMON CAUSE OF DYSPNEA IN HIV RESURFACED

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Introduction: The incidence of Kaposi Sarcoma (KS) in AIDS population has decreased significantly in the era of antiretroviral therapy (ART). Clinicians are not fully familiar with its pulmonary presentations which might lead to delay of diagnosis and treatment.

Case Description: A 59-year-old Caucasian male, HIV-positive for >20 years, was diagnosed with right leg KS by biopsy 15 years ago. His course was complicated with ART refusal/interruption, consenting to ART only from 2006-12, during which HIV was suppressed and KS regressed and stabilized. He was lost to care from 2013-18. He was admitted in April 2018 with respiratory distress that required ICU and brief intubation. He was found to have Pneumocystis jiroveci pneumonia by bronchoalveolar lavage, CD4 of 5, and viral load of >6 log. The patient was discharged home with 4L of oxygen and to complete the TMP/SMX course and referred to outpatient infectious disease to resume ART. Three days after clinic and recommendation to resume ART, he was readmitted complaining of worsening dyspnea. Shortly after admission, respiratory failure developed necessitating ICU transfer. Bronchoscopy showed normal airway anatomy and appearance. Lung biopsy revealed spindle cell proliferation with positive herpesvirus-8(HHV-8) stain. Liposomal doxorubicin was administered. Unfortunately, the patient continued with refractory hypoxia and was made comfort care by family.

Discussion: KS, a low-grade vascular tumor associated with HHV-8, is the most common neoplasm in HIV patients. Studies reveal that environmental stressors such as hypoxia can stimulate its progenesis and oncogenesis activity. Despite the decline in the KS in recent years, pulmonary KS cases have been linked to interruption or lack of HIV care. The biology of KS and the importance of maintaining linkage to care to control and prevent KS complications will be reviewed.

6) CONTROLLING SCABIES IN A RESOURCE LIMITED SETTING

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Introduction: Scabies is a skin condition caused by *Sarcoptes scabiei* infestation. Crowded human populations with limited access to water, like those frequently found amongst displaced persons (DP), makes eradication of the infestation particularly difficult. The following outlines a novel intervention to effectively control scabies in resource limited conditions.

Discussion: The ongoing violent conflicts in the Middle East have resulted in millions of DP with Syrians making up 22% of the global total 22.5 million registered refugees. The living conditions of these individuals are often cramped making transmission of scabies easy. Prolonged direct physical contact with infected individuals represents the primary mode of transmission. However, infested bedding and clothing where hot water or heated dryers are unavailable make fomite driven re-infection likely and eradication particularly difficult. Five cases of scabies were diagnosed with classic history and examination findings in a Greek refugee camp housing approximately 600 DP. Three of the five cases were severe with crusted genital lesions, also known as Norwegian scabies. Standard recommended therapy includes treatment of the affected individual and close contacts with permethrin 5% cream. Clothing and linens should then be heat treated with a hot wash and heated dryer. Severe cases with crusted lesions or institutional outbreaks should be treated aggressively with topical permethrin plus oral ivermectin. Unfortunately, we were unable to obtain sufficient amounts of permethrin or ivermectin which often occurs in resource limited settings. Further complicating the matter, spare mattresses could not be sourced, and heat treatment of clothing/bedding was not possible due to camp facilities.

Intervention: Affected individuals were isolated and instructed to place all their belongings/bedding into plastic bags which were then sealed. Patients were then treated with topical benzoyl benzoate 25% emulsion daily. On day two their mattresses were wrapped with clear plastic film and they were given new clothing and bedding. All patients repeated benzoyl benzoate application seven days after initial treatment. Those with crusted lesions were given permethrin 5% cream to apply directly to the lesions daily for 14 days. All patients had complete resolution of symptoms without re-infection.

7) HEMOPTYSIS, HOARSENESS, AND HORNER SYNDROME

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Case: A 71-year old male with limited medical contact presented with a one-day history of hemoptysis. The evening prior to presentation, he reported hemoptysis of ~100 mL which resolved spontaneously. Upon further questioning, the patient endorsed a 40-pound weight loss, progressive dysphagia, and voice hoarseness over the past year. His physical exam was remarkable for right-sided ptosis, anisocoria with miosis of the right pupil, and a palpable right-sided neck mass. Initial laboratory workup was unremarkable. A CT of the neck and chest was performed which revealed a large paratracheal mass originating from the right lobe of the thyroid gland with extension into the mediastinum with tracheal and esophageal invasion. Ultrasound-guided biopsy of the mass was performed with pathology returning consistent with papillary thyroid carcinoma (PTC) with tall cell features. Given the size and location of the mass, surgical resection was not initially pursued. He completed radiation therapy with plans for surgical tumor debulking prior to radioactive iodine treatment.

Discussion: PTC accounts for approximately 85% of all thyroid tumors. Most patients present with a solitary thyroid nodule discovered incidentally on physical exam and generally have a favorable prognosis. The tall cell variant is defined as harboring a population of columnar-appearing neoplastic cells and is associated with a more aggressive clinical course. They can present with findings attributable to tumor extension into surrounding structures such as involvement of the thoracic sympathetic trunk causing Horner syndrome. Recurrent laryngeal nerve compression can lead to vocal cord paralysis and hoarseness. Tracheal erosion can cause respiratory compromise and hemoptysis. Control of hemoptysis in this setting can be challenging, as embolization involves accessing the vessels of the thyrocervical trunk. Treatment of PTC is surgical resection with radioiodine therapy and systemic chemotherapy reserved for patients with late-stage disease.

Conclusions: While 10-year overall survival remains high, this case illustrates the aggressive nature of the tall cell variant of PTC and the potential for local invasion.

8) A COMPLICATED CASE OF MRSA BACTEREMIA

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Introduction: MRSA bacteremia, including infective endocarditis, carries a high mortality rate. The patients who fail initial therapy with vancomycin require salvage therapy.

Case Description: A 33-year-old male with Hepatitis C and heroin use disorder was recently diagnosed with MRSA bacteremia likely secondary to IV drug use, infective endocarditis with vegetation on the tricuspid valve, and scattered septic pulmonary emboli with cavitation. He was initially treated with IV vancomycin and then transitioned to weekly oritavancin. However, he only received 2 infusions. On admission, his vitals were significant for a pulse of 116 and a temperature of 102.0°F. Exam was significant for a II/VI systolic ejection murmur, bilateral lower extremity edema, and an erythematous rash over the right lower leg. Blood cultures grew MRSA and his WBC was 18.1. Patient was started on vancomycin and ID was consulted. The bacteria was found to have a vancomycin AUC/MIC <400, so he was switched to daptomycin and ceftaroline. The organism was then found to be not susceptible to daptomycin. Therefore, patient's antibiotic treatment changed to daptomycin, ceftaroline, and linezolid. Blood cultures remained positive for six days after admission. TEE showed a large vegetation on the tricuspid valve with moderate tricuspid regurgitation, thoraco-lumbar spine MRI showed multi-level osteodiscitis without spinal abscess, and cervical spine MRI showed a phlegmon or septic embolus within the paraspinal musculature abutting right articular pillar at C5-C6 and multiple patchy abnormal enhancements throughout the cervical paraspinal musculature suggestive of septic emboli. CT surgery was consulted; he is not a surgical candidate due to his active drug use and hemodynamic stability despite the size of the vegetation. The patient will continue on the above antibiotics for at least 6 weeks. Patient was discharged home with plans for continued IV antibiotics via home health and weekly blood draws in an infusion clinic to monitor for bone marrow suppression and bacteremia. He has follow-up with his previous rehabilitation program.

Discussion: MRSA bacteremia, including infective endocarditis, carries a high mortality rate, with up to 50% of patient failing initial therapy with vancomycin. We report a case of complicated right-sided infective endocarditis by MRSA successfully treated with a unique antibiotic regimen.

9) THOUGHT ONE, TURNED OUT TO BE BABESIA

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A 45 year old male with a past medical history of Multiple Sclerosis (MS) and Optic Neuritis presented to the hospital with hematuria, dysuria, fatigue, falls, night sweats and chills. He denied epistaxis, melena, hematochezia, or hematemesis. He was previously treated with two doses of Orelizumab 4 months ago for MS. A week prior to hospitalization, he traveled to Nevada. When returning to Wisconsin, he fell down multiple times when exiting the plane, without hitting his head or losing consciousness. He went to Neurology Clinic the next morning. Lab work was remarkable for hemoglobin of 12.9 g/dL, hematocrit of 37.7%, and platelet count of 46 10e3/uL. In the same day, he went to the ED with vitals remarkable for heart rate of 113 beats per minute. Lab work was remarkable for haptoglobin below 10 mg/dL, lactate dehydrogenase of 771 unit/L, d-dimer of 6.3 mg/L FEU, hemoglobin of 12.1 g/dL, platelet count of 51 10e3/uL, AST of 60 unit/L, Alkaline phosphatase of 132 unit/L. ADAMTS13 activity, fibrinogen, total bilirubin, direct bilirubin, and reticulocyte count were within normal limits. Direct antiglobulin test was negative. Peripheral blood revealed no schistocytes or spherocytes.

Patient then admitted to hospital. While hospitalized, patient was febrile multiple times with highest peak temperature of 103 F. Hematology and infectious disease were also consulted. Brain magnetic resonance imaging was done, with no indication of new multiple sclerosis lesions and no findings suggestive of PML. Infectious disease workup was negative for HIV, Hepatitis C, EBV, CMV, Influenza A&B, Parainfluenza, rhinovirus, adenovirus, RSV A&B, Coccidioides, Blastomyces, Histoplasma, atypical pneumoniae, Ehrlichia, and Lyme disease.

Initially the hematologic abnormalities were of unclear etiology. Work was done ruling out MS exacerbation, PML, thrombotic microangiopathy, disseminated intravascular coagulation, autoimmune hemolytic anemia. Multiple infectious causes were ruled out over course of hospital stay. Empiric babesia treatment of atovaquone and azithromycin was started. Blood smears specific for babesia was ordered and it came back positive. During treatment, symptoms slowly improved until patient was near baseline. He was no longer febrile with labs slowly improved including platelet count of 139 10e3/uL on the day of discharge. Patient was discharged on continued treatment after last blood smear was negative.

This case illustrates when having a patient with hemolysis and thrombocytopenia, it is important to keep babesia higher on your differential diagnosis, even if babesia itself may be rare.

10) INTIMATE PARTNER VIOLENCE (IPV)

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Introduction: Intimate partner violence (IPV) is a growing problem in the United States. Research found individuals who experience IPV may become psychologically disturbed, which correlated with an increased risk of chronic disease and disability. Inversely, positive thoughts and wellness activities are correlated with improved health.

Objective: This study investigated the potential effect of exercise as a primary intervention to minimize the risk of chronic disease.

Methods: Women residing at a domestic violence shelter participated in a weekly wellness program and were instructed to complete a Rand-36 Short Form Health Survey (SF-36) before and after completing each walk or run for a minimum of 20 minutes for 1-10 weeks on their own time. In conjunction to this, weekly discussion groups were held with a focus on mental health.

Results: Over three months, 23 women were enrolled in the program. The scores for all participants in all health areas were averaged and categorized as pre, or post intervention. They were then compared to the Rand average and standard deviation. In all areas, the participants had lower health scores than the Rand Medical Outcomes Study population. Post scores were higher than the pre-survey scores in all areas, but no statistical difference was found when using a paired t-test.

Conclusion: Most participants expressed a desire to be more active, healthier and lose weight. The quality of the data assessing the effectiveness of the intervention may be compromised by subjects self-reporting exercise. Due to the independent nature of the project, there is an increased compromise to the quality of the data in assessing the effectiveness of the intervention. However, the participants expressed that the program added value to their lives. Future directions include repeating the program with a larger population and developing a companion program for children to increase participation and retention of Women with children.

11) CHD-SQUARED: CONGENITAL HEART DISEASE (CHD) AND COMPLEX HEMATOLOGIC DECISION MAKING IN A PREGNANT PATIENT

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Introduction: Adult patients with Fontan physiology are complex and pose unique dilemmas as they are prone to complications: this is compounded in pregnancy.

Case Report: A 19 year-old G2P1001 with past medical history significant for pulmonary atresia with intact intraventricular septum status post Fontan presented to the emergency department with dyspnea and chest pain. Her current pregnancy was complicated by a subchorionic hematoma. On presentation, she was tachycardic and dyspneic; imaging confirmed multiple pulmonary emboli. She was placed on a heparin drip and admitted; shortly thereafter, vaginal bleeding developed. The drip was paused and urgent obstetric confirmed a viable intrauterine pregnancy with worsening of the hematoma. She was switched to enoxaparin prior to discharge home. Unfortunately, the patient presented two weeks later with vaginal bleeding. Her subchorionic hematoma had worsened and she miscarried shortly thereafter.

Discussion: Patients with Fontan physiology are at risk for multiple long-term complications including protein losing enteropathy, cirrhosis, and recurrent arrhythmias. One of the major complications seen in these patients is a chronic prothrombotic state, secondary to chronically abnormal levels of protein C, protein S, and antithrombin III. Pregnancy in patients with CHD poses another clinical dilemma. There is a known risk to both the mother and fetus, which prompted the creation of the modified World Health Organization (WHO) system, which places patients into different risk categories based on severity of the underlying cardiac condition and accounts for all known maternal risk factors including other co-morbidities. Categories range from I (no increased risk) to IV (Pregnancy contraindicated secondary to extremely high risk). The patient in the vignette had a WHO score of III, which portends a significantly increased risk of maternal morbidity and mortality, and expert counseling is required.

Conclusion: Patients with Fontan physiology are at increased risk for a variety of long-term complications—including thrombus—as well as significantly increased risk for morbidity and mortality in pregnancy. Pregnant patients should be risk-stratified with the modified WHO system and counseled appropriately.

12) DISSEMINATED BLASTOMYCOSIS IN AN IMMUNOCOMPETENT HOST

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Introduction: Blastomycosis dermatitidis, a dimorphic fungus endemic to the Great Lakes and Ohio-Mississippi River valleys, most often presents as an isolated pulmonary infection following inhalation of conidia spores. However, dissemination through hematogenous spread is a known serious complication.

Case: A 61-year-old male with ESRD presented with a two week history of fever, diffuse pustular rash, and cavitating right upper lobe mass. His rash began on the forehead and progressively spread downwards with enlarging nodular lesions. A punch biopsy of the rash obtained during a recent outpatient dermatology visit showed broad-based budding. On admission, the patient tested positive for Blastomycosis urinary antigens. A chest CT showed a right upper lobe cavitary lesion and hilar lymphadenopathy. A CT abdomen and pelvis showed a loculated fluid collection in the periumbilical region near the patient's PD catheter site. Peritoneal fluid showed greater than 500 WBCs. The patient was placed on IV liposomal Amphotericin B. Blood and peritoneal cultures drawn on admission later resulted positive for Blastomycosis. On Day 3 of hospitalization, a rapid response was called for increasing oxygen requirements and the patient was transferred to the intensive care unit. The patient's respiratory status continued to decline leading to intubation. He was eventually extubated and transferred to the floor. A Brain MRI was obtained given progressive confusion on the floor. Multiple new infarcts in both cerebellar hemispheres, the right frontal lobe, and left corona radiata were seen. The patient's mental status returned to baseline over the next two weeks. He completed 4 weeks of liposomal Amphotericin B and was transitioned to oral Voriconazole for a total of 12 months. He was discharged home on day 30. The patient has outpatient follow-up scheduled for the removal of his PD catheter.

Discussion: This case highlights the importance of early diagnosis and treatment of patients with Blastomycosis. Delays in diagnosis increase risk for hematogenous spread to other organ systems, including the Central Nervous System (CNS), and increased mortality rates. Although fungal cultures can take upwards of two weeks to result, patients can be quickly and easily screened using urinary antigen testing. Once there is sufficient clinical suspicion for infection with Blastomycosis, treatment should be initiated with liposomal Amphotericin B. After stabilization, patients with suspected CNS disease should be transitioned to Voriconazole due to its ability to permeate the blood-brain barrier. In addition, patients with indwelling foreign bodies, such as central lines or catheters, should have these removed or replaced once medically stable to prevent re-infection.

13) CRYPTOCOCCAL MENINGITIS IN PATIENT WITH ALCOHOLIC CIRRHOSIS

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Background: Cryptococcal meningoencephalitis via infection from *Cryptococcus neoformans* is a grave opportunistic infection often associated with severe immunodeficiency, most classically in untreated HIV/AIDS patients with a CD4 count < 100 cells/uL. In fact, 80-90% of all patients with cryptococcal meningitis have AIDS. Despite this association, it is critical to recognize that HIV-seronegative etiologies of this infection occur. Hematologic malignancy, chronic glucocorticoid therapy, immunosuppression following organ transplantation, sarcoidosis, and advanced liver disease have all been shown to be implicated in HIV-seronegative cryptococcal infections.

Case: A 31 year-old woman PMH significant for alcoholic cirrhosis (MELD 16, Child Pugh class C) presented to the Emergency Department (ED) with chronic headache for 1 month and associated subjective fevers, photophobia, phonophobia, nausea/vomiting, and nuchal rigidity. MR brain demonstrated leptomeningeal enhancement and lumbar puncture was positive for cryptococcal antigen and C. neoformans NAAT. An HIV screen was non-reactive. She was initiated on induction therapy with amphotericin and flucytosine. During induction therapy, she became progressively pancytopenic so was transitioned from flucytosine to high-dose fluconazole. Her thrombocytopenia continued to progress so amphotericin was discontinued. After five weeks of continued therapy, the patient left AMA with a platelet count of 9,000. She returned to the ED five days later with intractable epistaxis, an undetectable platelet count, and hemoglobin of 5.7. She was intubated for airway protection. Given her severe pancytopenia, a bone marrow biopsy was obtained which demonstrated significant hypocellularity (<10%) and hemophagocytosis consistent with hemophagocytic lymphohistiocytosis versus acute marrow damage due to flucytosine. After 2 months of treatment patient was stable for discharge on maintenance fluconazole. Serial analysis of cryptococcal antigen titer revealed improvement in underlying infection.

Conclusion: While cryptococcal meningoencephalitis is most commonly associated HIV/AIDS, recognition that HIV-seronegative etiologies occur should prompt evaluation for deficiencies in cell-mediated immunity. Management of cryptococcal meningoencephalitis includes induction therapy with amphotericin and flucytosine for 2-6 weeks, followed by consolidation and maintenance therapy with fluconazole for one year.

14) DLBCL PRESENTING IN SPLENIC RED PULP AND BONE MARROW

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Splenic involvement of diffuse large B-cell lymphoma (DLBCL) is relatively common, but most cases manifest as part of advanced, secondary disease. Primary splenic DLBCL is a rare lymphoma presenting as one or more nodules in the splenic white pulp, but red pulp is usually not involved.

A 67 year old man presented to the emergency department with complaints of fatigue, weakness, and abdominal pain. He was noted to have new onset normocytic anemia to 10.0 g/dL. Hemolysis labs, B12, and folate were within normal limits and peripheral smear showed no signs of hematologic malignancy. FIT testing and EGD were both negative for bleed. CT abdomen/pelvis was significant for hepatosplenomegaly without evidence of mass. He was discharged home but returned 10 days later with Hgb decreased to 8.1g/dL. Bone marrow biopsy was performed at this time and showed increased iron stores with slightly hypercellular marrow at 40% and mild erythroid hyperplasia. No dysplasia, significant blasts, lymphocytic infiltrate or ring sideroblasts were observed. Flow cytometry of the bone marrow revealed a small 1.6% B-cell population (CD19+, CD20+, CD5-) which were of normal size and which expressed lambda light chain surface immunoglobulin in a monoclonal fashion. Karyotype revealed three cells with clonal abnormalities suggestive of B-cell lymphoma including deletion 6q, trisomy 18, and t(3;22). t(;22) may represent BCL6;IgL translocation so FISH for BCL6 was done confirming BCL6 rearrangement in 5% of cells. Liver biopsy revealed diffuse necroinflammatory changes and a perivenular and periportal lymphoid infiltrate suspicious for diffuse large B-cell lymphoma. Shorty after liver biopsy, the patient began to clinically decline and hemoglobin dropped from 8.0g/dL to 4.0 g/dL He was taken for emergent exploratory laparotomy due to concern for splenic hemorrhage and underwent splenectomy. Pathology of the spleen revealed diffuse infiltrates of medium to large atypical cells with many mitotic figures. The atypical cells were positive for CD20, PAX-5, BCL-2, BCL6 and MUM-1, and negative for CD138, cyclin D1 (bcl-1), CD10, CD5, and CD3 consistent with a diagnosis of DLBCL. Unfortunately, the patient was not clinically stable enough to initiate chemotherapy and he ultimately passed away from distributive shock and multirorgan failure.

This case may represent a distinct subtype of DLBCL with primary extranodal presentation in the spleen and bone marrow. Recognition of this entity is critical for delivering appropriate treatment in a timely fashion to prevent significant morbidity or mortality as seen in this patient.

15) A TYPICAL PRESENTATION OF CLASSICAL HODGKIN LYMPHOMA; NOT AS STRAIGHTFORWARD AS EXPECTED

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Hodgkin lymphoma (HL) is relatively uncommon. The most common presentation of HL is a painless enlarged lymph node, either in the supraclavicular or cervical area. Even in advanced disease, fewer than 50% of patients present with fever, weight loss, and sweats.

A 31-year-old woman new to Wisconsin presented to a podiatrist for swelling in her third toe and an intermittent rash. The podiatrist recommended that she establish with a primary care provider for further evaluation. At her initial PCP appointment, the patient had no constitutional symptoms, and ROS was negative. Physical examination revealed an enlarged supraclavicular lymph node, which the patient reported had been biopsied on two occasions over the past three years, each time with benign pathology. Plain radiographs of the toe were unremarkable, and initial lab evaluation demonstrated normal CBC, CRP, and ESR. Outside records were obtained, and referrals were made to ENT and rheumatology.

A month later, the patient returned with bilateral lymphedema to the mid-shins, left worse than right. She continued to deny any constitutional symptoms, and ROS remained negative. Her rash had improved and was now limited to her shins. Repeat labs showed a normal CBC, but ESR and CRP were now elevated. Chest radiograph was significant for a left hilar mass. Core needle biopsy of the supraclavicular node was repeated, and the patient was diagnosed with classical HL. PET-CT demonstrated significant disease with hepatic and bone marrow involvement.

Bilateral lower extremity edema in an otherwise healthy adult can be a challenging symptom to diagnose. This case illustrates the persistence needed to make a difficult diagnosis, particularly when following one's clinical intuition means repeating biopsies or other tests that have been normal in the past. HL often presents with painless adenopathy without classical B symptoms, which can make it challenging to diagnosis.

16) APPROACH TO MIGRATORY ARTHRITIS WITH EVANESCENT RASH IN A MIDDLE-AGED FEMALE.

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Adult Still's Disease (ASD) is a systemic inflammatory condition that presents with migratory arthritis; high-spiking, intermittent fevers; and an evanescent, salmon-colored rash. ASD is a rare condition of unknown origin, estimated to affect 1:100,000 individuals annually. Given the broad spectrum of differentials that present with similar rheumatologic symptoms; especially in the Midwest population, and the rarity of this disease, the diagnosis of ASD may be delayed or missed entirely.

A 43-year-old woman presented to the Emergency Department with right foot pain and history of intermittent, large joint migratory arthritis, without rash. Examination revealed a swollen second toe with normal X-ray. She was discharged with Tylenol. Ten days later she returned to the Emergency Department with intermittent pruritic rash and debilitating myalgias of 3 weeks duration. Exam revealed slightly raised, pink, non-blanchable maculopapular rash on her trunk and arms; attributed to work-related chemical exposure. She was subsequently prescribed Prednisone that she did not utilize. Four days later, she presented to Urgent Care with continued intermittent myalgia, arthralgia, and malaise. Skin and joint exam were normal. Lab work-up revealed: CRP 6.9 mg/dL, ESR 35 mm/hr, CK 15 U/L, and WBC 32.6 x 10^3/uL with neutrophil predominance. Lyme and tick borne panel were negative. Patient was admitted to the hospital where inguinal lymphadenopathy was noted and joint exam was normal despite complaints of arthralgia. Chest X-ray was normal and ANA, RF, CCP, blood culture, and repeat Lyme serology were negative. The following day, arthralgia persisted and she developed a fever of 103°F. Physical exam remained benign and additional lab work revealed ferritin of 1108 ng/mL, which increased to 12,499 ng/mL three weeks later. Given the above, the patient met Yamaguchi criteria for ASD and was discharged on scheduled naproxen and prednisone, with Anakinra added one month later. She continued to have arthralgia, myalgia and malaise without reappearance of rash, so methotrexate was added.

This case is a prime example of the difficulty in diagnosing ASD and the importance of correlating migratory arthritis, evanescent rash, and intermittent fever. The general internist should keep in mind the triad of symptoms associated with ASD and care should be taken to obtain necessary lab work and prompt referral to Rheumatology to expedite treatment.

17) RECURRENT ASCITES AS AN INITIAL MANIFESTATION OF MULTIPLE MYELOMA

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Abstract: Ascites is a rare initial presentation of Multiple Myeloma and commonly noticed during the course of the disease in patients with advanced and recurrent multiple myeloma. Literature review suggests presence of ascites is a poor prognostic marker and few cases were reported ascites and peritoneal carcinomatosis as initial presentation of Multiple Myeloma. We report a case of 75 year old male who presented with recurrent ascites, omental nodules and splenic nodules as an initial presentation of myeloma. Protein electrophoresis showed IgA Myeloma band. Metastatic workup revealed wide spread disease involving right pulmonary hilum, celiac axis, rectum. Bone marrow biopsy confirmed 27% of plasma cells. Cytogenetics was positive for 17p deletion. He tolerated induction chemotherapy and received autologous stem cell transplantation. However he relapsed within 6 months after transplantation indicating poor prognosis associated with ascites and peritoneal carcinomatosis.

18) A SUPRISISING CASE OF KIKUCHI-FUJIMOTO DISEASE

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Introduction: Kikuchi-Fujimoto disease is a rare, benign and self-limited syndrome characterized by regional lymphadenopathy with tenderness, predominantly in the cervical region, usually accompanied by mild fever and night sweats. It is important to consider it as differential diagnosis in a patient with cervical lymphadenopathy, fever and rash.

Case presentation: A 54-year-old African American female with medical history of hypertension presented with fevers, chills, generalized weakness, headache, nausea, diarrhea, dizziness and painful neck swelling for 3-4 weeks. She reported developing a generalized pruritic skin rash about one week prior. On admission, she was in mild distress due to pruritus, but afebrile. Her exam was notable for tender lymphadenopathy throughout the right side of her neck, a diffuse maculopapular rash over the torso and extremities, and RUQ tenderness. Laboratory results showed elevated creatinine of 2.14 mg/dl, WBC count of 1.7, AST 105, ALT 75 and lipase 65. CT scan of Abdomen/Pelvis showed multiple enlarged retroperitoneal and pelvic lymph nodes, suspicious for lymphoma, 1.9 cm cystic lesion in right adnexa, and multiple bilateral lobar hepatic cysts. She was started on supportive care with IV hydration and antipruritic agents. Extensive infectious disease workup only yielded positive IgG and IgM antibodies for HSV-1 and HSV-2 and an elevated erythrocyte sedimentation rate (ESR). A cervical lymph node biopsy showed necrotizing lymphadenitis, favoring a diagnosis of Kikuchi-Fujimoto lymphadenopathy. Skin biopsy showed dermatitis consisting of lymphocytes and histiocytes with scattered necrotic keratinocytes. Patient was discharged home with supportive care.

Discussion: Here we present a rare case of Kikuchi-Fujimoto disease likely related to HSV infection. Kikuchi-Fujimoto disease is an uncommon, idiopathic, self-limited cause of lymphadenitis. Exact pathogenesis is unknown, but viral or autoimmune cause has been suggested. The most common clinical manifestation is cervical lymphadenopathy, with or without systemic signs and symptoms. Clinically and histologically, the disease can be mistaken for lymphoma or systemic lupus erythematosus (SLE). It almost always runs a benign course and resolves in several weeks to months. Disease recurrence is unusual, and fatalities are rare, although they have been reported. The most common presenting features are cervical lymphadenopathy, leukopenia, elevated ESR, and elevated liver enzyme levels. Fever is also a common feature and should be on the differential in cases with fever of unknown origin. Lymph node biopsy is confirmatory with necrosis, karyorrhectic debris, and the presence of crescentic histiocytes and plasmacytoid monocytes. It is also found to be associated with EBV, parvovirus, HHV-6, and rarely HSV. Treatment is supportive with NSAIDs. Steroids are indicated for severe disease.Its recognition is crucial especially because this disease can be mistaken for SLE, malignant lymphoma, lymph node TB, or rarely adenocarcinoma. Clinicians' and pathologists' awareness of this disorder may help prevent misdiagnosis and inappropriate treatment.

19) THYROTOXIC PERIODIC PARALYSIS PRESENTING AS PAROXYSMAL DIFFUSE HYPOKALEMIC WEAKNESS

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Introduction: Thyrotoxic periodic paralysis is a rare disorder characterized by episodic hypokalemia and diffuse paralysis.

Case Report: A 29-year-old Asian male with a one-year history of recurrent generalized muscle weakness presented to the emergency department with whole body paralysis. The patient's symptoms began abruptly that morning, when he was unable to stand and exit a train due to lower extremity weakness, which generalized over the course of one hour. He reported a one-year history of previous episodic muscle weakness lasting several hours, which last occurred three months prior. He reported palpitations, sharp chest pain, and mild shortness of breath. Additionally, he endorsed a 20-30 lb. weight loss throughout the past year.

Upon presentation patient was tachycardic and tremulous. Physical exam was notable for a diffusely enlarged, non-tender, non-nodular thyroid, decreased muscle strength and diminished deep tendon reflexes in both lower extremities, with marked lower-extremity proximal muscle weakness and tremors, and marked truncal weakness with an inability to support a seated posture. He also had a pruritic, urticarial skin rash on his trunk and bilateral upper and lower extremities. In the emergency department, his serum potassium was 1.9 mmol/L and magnesium level was decreased. An electrocardiogram showed sinus tachycardia. Potassium was replaced and his weakness quickly resolved after correction of electrolytes. Patient's symptoms were suspected to be due to periodic hypokalemic paralysis. Thyroid function test showed low TSH and elevated free thyroxine (FT4). Both thyroid peroxidase (TPO) antibody level and TSH receptor antibody level were elevated. Patient was started on methimazole 20 mg twice-daily and propranolol. While in the hospital, he developed a pruritic skin rash consistent with acute urticaria, likely related to his autoimmune thyroid disease. Patient was started on antihistamine and his rash improved. He was discharged home with the diagnosis of thyrotoxic periodic paralysis secondary to Graves' disease.

Discussion: Thyrotoxic periodic paralysis (TPP) is a disorder most commonly seen in Asian men aged 20-40, and is characterized by episodic hypokalemia and diffuse paralysis. The condition primarily affects the lower extremities and is secondary to thyrotoxicosis. Hypokalemia in TPP is thought to occur as a result of thyroid hormone sensitization of the Na+/K+-ATPase and an intracellular potassium shift. The history of acute weakness in the setting of these metabolic abnormalities in a young Asian male is classic for thyrotoxic periodic paralysis. Excess thyroid hormone activates beta adrenergic receptors, which subsequently activate the Na/K ATPase and causes a net intracellular potassium shift, resulting in muscle hyperpolarization and transient paralysis. Treatment of TPP includes prevention of this potassium shift via nonselective beta-blockade, careful repletion of potassium, and treatment of the underlying thyroid disease. Early recognition of this disorder is crucial, to prevent rebound hyperkalemia with aggressive potassium repletion and to initiate appropriate treatment protocols.

20) PERCEPTION OF JUNIOR FACULTY OF GENERAL INTERNAL MEDICINE REGARDING MENTORING SCHOLARY PROJECTS AND FACULTY DEVELOPMENT.

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BACKGROUND: Studies have shown that mentorship has an important influence on personal development, career guidance, career choice, and research productivity, including publication and grant success. Mentoring is often identified as a crucial step in achieving career success, but it is challenged by increased clinical, administrative, research, and other educational demands on medical faculty. Since clinician educators rarely receive training on the mentoring process, they are often poorly equipped to face challenges when taking on mentoring responsibilities. Mentoring skills are valuable assets for academic medicine faculty, who support academic excellence including scholarly productivity of the next generation of physicians. Therefore, it is important to identify perception of faculty members earlier in their career to design interventions that will facilitate scholarship and career development. This study assesses junior faculty's perceived benefits, challenges, and barriers regarding mentoring medical students and residents through the process of writing and presenting case reports.

METHODS: A Qualtrics survey was emailed to 50 assistant professors in the Division of General Internal Medicine at the Medical College of Wisconsin. All of the questions in the survey were focused on obtaining the junior faculty's perceptions of factors regarding the mentoring of medical students and residents through the process of writing and presenting scholarly projects, its benefits, and the perceived challenges and barriers. Responses were obtained on a five-point Likert scale. We performed conventional quantitative analysis on all the responses and analyzed the data as respective proportions. A comparison between the responses of those who have and have not presented case reports previously was performed using a chi-square test or Fischer exact test.

RESULTS: Of the 34 junior faculty (30 academic hospitalists and 4 primary care physicians) who completed the survey, 47% said they have not mentored a medical student or resident in writing and presenting scholarly projects including case reports in regional or national meetings. Eighty-two percent of surveyed faculty had completed scholarly projects either during their medical school or residency training. Of the faculty who had mentored medical students and residents, 68% had mentored 1-3 projects, and 25% had mentored more than 6 projects. The top three perceived benefits of mentoring learners in scholarly projects included enhancing curriculum vitae (CV) (100%), building educator portfolio for promotion (94%), and improving critical thinking skills (88%). The main perceived barriers included lack of resources or formal training for mentoring learners (85%), lack of time due to clinical and administrative responsibility (82%), and lack of knowledge about opportunities for mentorship and faculty development (79%). More than 76% faculty reported lack of structured mentorship and curriculum as a barrier to mentoring learners in scholarly activities. 97% of the faculty reported the need for protected time and more than 70% of the faculty perceived the need for structured peer mentorship, training/workshops, and incentives to promote scholarship and faculty development. In subgroup analysis, a higher percentage of female faculty (60%) were involved in mentoring students and residents compared to males (33%). However, this difference was not statistically significant (P=0.17, Fischer's exact test).

CONCLUSION: Our study shows that the majority of junior faculty (early career hospitalist and primary care providers) reported having not mentored medical students or residents in the process of writing or presenting any scholarly projects including case reports, despite having previous experience of scholarly projects during their medical or residency training. Mentoring medical students and residents has great benefits and barriers. Junior faculty see professional values in mentoring, as this can enhance their CV and their educator portfolio for promotion. A lack of resources, formal training and protected (non-clinical) time for academic pursuits due to clinical and administrative responsibility were identified as the major barriers to mentorship. Our findings highlight the need for future studies on developing multifaceted interventions to facilitate structured mentorship among faculty and learners.

21) AN UNUSUAL CAUSE OF TENSION PNEUMOTHORAX

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A tension pneumothorax is a life-threatening condition when air progressively accumulates in the pleural cavity and is unable to escape. Due to a unidirectional valve mechanism during normal rhythmic breathing, a mass effect on the effected lung compresses mediastinal and intrathoracic structures. Rapid recognition and treatment of tension pneumothoraces is imperative to avoid imminent cardiopulmonary arrest. This case highlights an unusual presentation of a spontaneous tension pneumothorax in the setting of parenchymal necrosis secondary to pulmonary infarction. A 69 year-old man with a history of advanced heart failure, on treatment for invasive aspergillosis, with known bilateral multiple pulmonary emboli with secondary infarcts due to right ventricular implantable cardioverter defibrillator lead thrombus, presented with sudden onset of right sided chest pain, cough and shortness of breath. The physical examination revealed, bilateral ronchi, diminished breath sounds, hyperresonance, and decreased tactile fremitus on the right side. Computed tomography (CT) scan of his chest in the emergency department revealed right sided tension pneumothorax and left mediastinal shift with collapse of the right lung. It was also reported that the right lower lobe pulmonary infarct had become necrotic with direct communication into the pleural space. Cardiothoracic surgery was consulted and placed a chest tube. His symptoms significantly improved and were confirmed with serial chest x-rays. He was discharged home on long-term anticoagulation and continued a three-month treatment of pulmonary aspergillosis with Isavuconazole. Spontaneous tension pneumothorax in the setting of parenchymal necrosis secondary to pulmonary infarction is a rare presentation. There was a 6-month time lag between the diagnosis of pulmonary embolism (PE) and pneumothorax in this patient. This is longer than the reported time lag of days to weeks for reported cases of pneumothorax secondary to PE. While pulmonary infarction is seen in up to 10% of patients with pulmonary embolism, formation of a pneumothorax appears to be extremely uncommon. We found 2 reported cases in the literature over the last 15 years. Although progressive parenchymal necrosis can occur over a number of weeks in patients with pulmonary infarcts, the cumulative damage to the lung parenchyma can culminate to a deadly apex in hours. Hence, the finding of pulmonary infarctions on CT should raise clinical anticipation for possible tension pneumothorax, especially in patients with additional lung co-morbidities. This is vital since the hemodynamic compromise caused by a tension pneumothorax in a diseased lung can be rapidly fatal. In addition, CT angiography to look for pulmonary emboli can be considered in patients with spontaneous pneumothorax of unknown cause who have additional risk factors for venous thromboembolism.

22) PATIENT-DIRECTED CRYOTHERAPY AS A CAUSE OF PERIPHERAL NEUROPATHY

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The peripheral nervous system is susceptible to a wide variety of toxic, inflammatory, and infectious insults. Often, the etiology of new neuropathy is evident, as in cases of longstanding diabetes mellitus or neurotoxic chemotherapy. However, in an otherwise healthy patient the broad differential for peripheral neuropathy can make definitive diagnosis elusive.

A 53-year-old very physically active female consulted her physician for progressive lower extremity pain of 7 weeks duration. The pain localized to her right medial ankle with associated numbness and tingling in her medial arch. She had also recently developed pain in her left foot. The presumed diagnosis was overuse injury and she was instructed to decrease her high-impact exercises and "ice as she sees fit". Over the course of 8 weeks, she was seen by podiatry, sports medicine, primary care, and an orthopedic surgeon without diagnosis and with continued symptoms progression. More intensive workup was initiated. Electromyography revealed very mild mononeuropathies of the right tibial nerve which was deemed nonspecific. An MRI of both ankles and feet demonstrated no acute nerve or soft tissue injury. Lab evaluation was normal including ANA, RF, SPEP, fasting glucose, TSH, CK, B12, BMP, and CBC. Symptoms continued to worsen and a repeat electromyography was done one month after initial. This revealed multiple absent sensory evoked potentials and motor unit loss in intrinsic foot muscles innervated by the tibial and peroneal nerves. She was started on gabapentin for symptom control and returned to see neurology. At this neurology follow-up visit, 4 months after her initial presentation, the patient offered up that she had been icing her lower extremities continuously for two or more hours each day since initiation of symptoms. She was instructed to stop cryotherapy and monitor her symptoms. With the cessation of icing, her symptoms resolved over the course of a year and she was able to return to her normal activities pain free.

This case illustrates the potential for significant neurologic injury with patient-directed cryotherapy. Cryotherapy after an acute sports injury has become a standard practice, and neuropathy is a known potential consequence. Treatments should therefore be limited to 20 minutes. This case also serves as a reminder that patients often treat symptoms in the home before presentation to a physician's office. In cases such as this, eliciting patients' home remedies can be instrumental to making an elusive diagnosis.

23) A CURIOUS CASE OF THROMBOTIC OCCLUSIVE VASCULOPATHY

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Case: A 52-year-old Caucasian female with medical history of recurrent deep vein thrombosis on warfarin for over 10 years and type II diabetes mellitus, presented as a transfer from outside hospital for further evaluation of necrotic abdominal wall wounds thought to be secondary to warfarin.

She initially presented for evaluation of worsening abdominal wall necrotic rash that started one month prior as purpura over the umbilical region. She was treated with doxycycline for presumed panniculitis without improvement. Her lesions worsened over next few days to become necrotic and extended to her upper thighs. She was switched to dabigatran for fear of warfarin skin necrosis, but the lesions continued to spread despite medication change, and was transferred to our facility. A skin exam was notable for multiple tender abdominal 20-30 cm interconnected retiform purpuric plaques with firm necrotic centers. The borders appeared less inflamed, with evidence of minimal re-epithelialization at base. On the left lower posterior leg were two 5-6 cm retiform purpuric patches, the superior lesion had central necrotic eschar.

Extensive hypercoagulability workup was notable for positive lupus anticoagulant and heterozygous prothrombin gene mutation. A full thickness abdominal skin biopsy revealed full thickness skin necrosis with intravascular thrombosis (dermal and pannicular) and panniculitis that was negative for calcium deposits. Due to the clinical picture and biopsy results the underlying cause of ischemic skin necrosis was determined to be caused by thrombotic vasculopathy syndrome secondary to her prothrombin gene mutation and lupus anticoagulant positivity. She was treated with enoxaparin with improvement in wound appearance. Despite this, she developed new retiform purpuric lesions. The most recent biopsy showed thrombosis and necrosis which were more advanced in comparison to prior tissue samples.

Discussion: Here we present a rare case of thrombotic occlusive vasculopathy. This patient's clinical presentation is very similar to calciphylaxis, making this a challenging case to diagnose. Her prothrombotic state also raises suspicion for antiphospholipid syndrome (APS). In the setting of APS, it is rare to present with such extensive subcutaneous thrombosis and necrosis. In this case, it was crucial to obtain a biopsy and hypercoagulability workup to establish a diagnosis of thrombotic occlusive vasculopathy. Both APS and calciphylaxis are associated with significant morbidity and mortality. It is important to rule out calciphylaxis as the medical management is distinct in the use of sodium thiosulfate, compared to anticoagulation for the treatment of thrombotic occlusive vasculopathy. Clinicians should consider thrombotic occlusive vasculopathy associated with underlying prothrombotic state as part of the differential diagnosis in patients presenting with a similar rash.

24) ADULT ONSET STILL'S DISEASE IN PATIENT WITH MACROPHAGE ACTIVATING SYNDROME

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INTRODUCTION: Adult onset Still's Disease (AOSD) is a serious but manageable condition that can rarely present in patients with previous diagnoses of macrophage activating syndrome (MAS). In this case, a patient was admitted to Froedtert Hospital with a classical presentation of both diseases.

CASE DESCRIPTION: A 19-year-old female with past medical history of MAS presented to the ED with abdominal pain, nausea, hematemesis, and jaundice. Symptoms had been occurring for several days, and she had also described recent fevers. On physical exam, the patient had visible scleral icterus, tenderness to palpation in bilateral upper abdominal quadrants, but notably no hepatomegaly. Vital signs showed slight fever (100.3 ºF) and tachycardia (127 bpm). On admission, the patient was found to be pan-cytopenic with elevated AST, ALT, and serum ferritin. The patient received work-up while inpatient to assess whether she could start tocilizumab treatment for AOSD; this included a liver biopsy conducted by hepatology. During her time in the hospital, rheumatology was also consulted to manage her AOSD. Ultimately, the patient was discharged to await results of liver biopsy from pathology. Biopsy showed clear macrophage phagocytosis of erythrocytes, confirming her prior diagnosis of MAS, but no acute processes preventing her from receiving immunotherapy.

DISCUSSION: AOSD can prevent with a variety of symptoms, most commonly daily fever, effervescent salmon pink rash, and liver involvement. Treatment with tocilizumab, an anti-IL6R humanized monoclonal antibody, is often utilized to prevent length of time required on corticosteroids. MAS is seen in a minority of AOSD cases and first line treatment is high dose corticosteroids. In cases of AOSD and MAS, it is important to consult hepatology and rheumatology to rule out liver damage prior to starting immunotherapy, which can potentially be hepatotoxic.

25) LEMIERRE'S SYNDROME

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Lemierre's syndrome is characterized by infectious involvement of the carotid sheath vessels with bacteremia. It typically arises from an infection in the oropharynx, most commonly caused by fusobacterium. It can also arise from infections of the ear, mastoid bone, sinuses, or salivary glands. Diagnosis is typically made based on blood culture or purulent material expressed from the site. CT and Ultrasonography showing internal jugular vein thrombosis can also be used in conjunction with blood cultures for diagnosis.

Case Description: A 20 year old African American female was admitted with complaint of shortness of breath that was preceded three weeks prior with symptoms of fever, chills, nausea, diarrhea and sore throat which resolved spontaneously without treatment. Due to the patient's shortness of breath and persistent non-productive cough, workup revealed a WBC 20.9 and elevated d-dimer. A CT-PE showed no pulmonary embolisms and TEE performed for concern for septic emboli was also was negative. The patient was diagnosed with suspected pneumonia and was treated empirically with vancomycin, piperacillin/tazobactam, and azithromycin. Blood and sputum cultures were both negative. An ultrasound of the neck revealed a focal (1-2 cm), nonocclusive thrombus in the left internal jugular vein which lead to the diagnosis of Lemierre's syndrome. Over the course of her stay her shortness of breath improved and was later discharged on amoxicillin/clavulanate.

Discussion:

Here we present a previously healthy 20-year-old African American female who was initially diagnosed with pneumonia and later found to have non-occlusive thrombus in the internal jugular vein consistent with Lemierre's syndrome. Complete Lemierre's syndrome typically has the characteristic IJV thrombosis in addition to the fusobacterium infection as was seen with this patient. The negative blood cultures seen here are likely due to the initiation of antibiotic treatment prior to obtaining blood cultures. The seemingly nonspecific constitutional symptoms of low grade fever, myalgias, and congestion can often mislead a provider to the diagnosis of pharyngitis. This often leads to delayed treatment and further complications. It is thus crucial for providers to be aware of the possibility of Lemierre's syndrome especially in a patient presenting with sore throat so that this diagnosis can be considered and treated.

26) DIFFUSE BLISTERING RASH: ATYPICAL PRESENTATION OF BULLOUS PEMPHIGOID

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Bullous Pemphigoid is an autoimmune blistering disease that most commonly arises in adults greater than 60 years old. It is characterized by subepithelial blister formation and deposition of immunoglobulins and complement within the epidermal basement membrane zone. Blisters are commonly found on the trunk, extremities, flexures, and inguinal folds and are usually preceded by a prodromal phase consisting of pruritic, eczematous, urticaria-like skin lesions. This is a case of a 54-year-old male with no significant past medical history who presented with a worsening diffuse pruritic blistering rash for one month with increased fatigue and lymphadenopathy. Abnormal laboratory studies included elevated white blood count with absolute eosinophilia count of 1.5, C-ANCA 1:160, IgE 1714, and CRP 5.16. Normal studies included Hepatitis B&C panel, HIV, RPR, stool ova/parasites, CMP, B12, and FISH panel. Biopsy of lesions showed linear deposits of IgG and C3 at the basement membrane zone as well as deposits of IgA, C3, and Fibrin in the blood vessels within the upper dermis. Patient was started on prednisone and mycophenolate mofetil and demonstrated immediate improvement in symptoms and progression of disease. Diagnosis of bullous pemphigoid is based on the presence of characteristic symptoms and physical findings and is confirmed by biopsy results. Treatment is largely based on disease severity and consists of topical and systemic steroids, immunomodulatory, and antibiotic therapy.

27) MENTAL HEALTH INDICATORS IN ASIA PACIFIC ECONOMIC COOPERATION (APEC)

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Introduction: APEC (Asia Pacific Economic Cooperation) is a consensus-based inter-governmental voluntary forum for 21 Pacific Rim member economies that promotes free trade throughout the Asia-Pacific region. The "*APEC Roadmap to Promote Mental Wellness in a Healthy Asia Pacific (2014-2020)*"1 was strategically approved in 2014 in order to support and complement the World Health Organization's Mental Health Action Plan by promoting awareness of and increased services for mental health within its economies.

Methods: The 2014 WHO Mental Health Atlas contains information collected from 171 countries that responded to a survey conducted by the Department of Mental Health and Substance Abuse, WHO in collaboration with its six WHO Regional Offices. Data provided for APEC economies was organized around a subset of four queries in order to see how the APEC region compared to WHO regions that contain a mix of low, lower- middle, upper middle and high income countries. The APEC economy-specific queries included: availability of mental health data, sources of funding for mental health services, the composition of mental health workforces and suicide rates.

Discussion: The WHO media center fact sheet on suicides states that close to 800,000 suicides occur each year and in 2015 75% of global suicides occurred in low- and middle income countries. In this study the three APEC members with the highest suicide rates were all high income economies. The observation of high rates of psychiatrist providers in APEC may reflect cultural preferences. Because APEC does not include any low-income countries, this may lead to a much higher perceived percentage of psychiatrist providers.

Conclusion: APEC is committed to promoting and supporting implementation of the World Health Organization Mental Health Action Plan. APEC has committed its resources, political and financial capital to promote and facilitate advances in mental health services as a unique and critically important measure of economic development. The participation of such a high profile multinational economic organization as APEC in the mental health arena is unique and historic, but its ability to substantively impact the global problem of mental illness remains to be seen.

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28) PARENTAL PERCEPTION OF CARE FOR INFANTS WITH FEVER DURING HOSPITALIZATION

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Background/Objectives: As part of the national collaborative, Project REVISE (Reducing Excessive Variability in Infant Sepsis Evaluation), we have evaluated many aspects of care around infants with fever, including appropriate work-up and length of stay (LOS). Objective: As part of our local work, we sought to understand parental perceptions about the reason for hospitalization, anticipated LOS, and preference for time of discharge.

Design/Methods: The study group included parents of infants admitted to one acute care unit of the Children's Hospital of Wisconsin. Parents of infants meeting inclusion criteria for Project REVISE (non-complex, well-appearing infants, ages 7-60 days, evaluated for fever without a source) were eligible for an interview. A 5-question structured interview was given to parents by one study team member over a 6-month period, between June and November 2017. Interviews were conducted on any day of hospitalization. Responses to the following 3 question topics will be discussed: reason for and length of hospitalization and discharge time preference.

Results: The interview was completed by 24 parents. One parent was non-English-speaking (language: Karen), and the remaining 23 were English-speaking. Bacterial cultures were positive for 3 infants (2 E. Coli, 1 GBS urinary tract infections (UTI)), viral PCR were positive for 8 infants (6 Enterovirus, 2 Parechovirus), and no organisms were found in 14 infants. The average LOS for all infants with negative cultures was 47 hours. 87% (21/24) of parents correctly identified their infants' reason for hospitalization as fever. The remaining 13% correctly provided established diagnoses (2 UTI; 1 meningitis). 29% (7/24) of parents anticipated a 36-hour LOS, 50% (12/24) a 48-hour LOS, and 21% (5/24) other. 50% (12/24) of parents preferred morning discharge, 42% (10/24) overnight discharge, and 8% (2/24) no preference.

Discussion: The results demonstrate that additional education from the healthcare team is needed in both emphasizing fever as the reason for the baby's hospitalization as well as the target goal of 36-hour LOS if bacterial cultures are negative, based on current supporting evidence. Optimized education for all parents of infants awaiting results will then allow them to better predict their LOS and determine if their preferences for overnight or morning discharge could be accommodated within 36 hours. Overnight discharge could then be considered based on these preferences and institutional practices.

29) A CURIOUS CASE OF CONCURRENT MUCOUS MEMBRANE PEMPHIGOID AND ERYTHEMA MULTIFORME

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INTRODUCTION: In medicine, it is a rarity to come across blistering skin conditions, let alone two concurrent conditions in the same patient. Here, we report a unique case of concurrent mucous membrane pemphigoid (MMP) and erythema multiforme (EM). This case demonstrates the importance of keeping a broad differential to diagnosing multiple clinical entities

CASE PRESENTATION: A 69-year-old male with a history of recently diagnosed Mucous Membrane Pemphigoid (MMP) presented to the Emergency Department with increased pain, blistering and edema of his bilateral feet and hands one day after rituximab infusion. He was recently diagnosed with biopsy-proven MMP by dermatology after several months of work-up for oral, genital, and conjunctival ulcers by other specialties. His outpatient regimen included prednisone 80 mg, azathioprine, doxycycline, and rituximab. Presenting exam showed several large, tense blisters on his feet, mucosal lesions, and multiple healing erosions and blisters of the extremities, flanks, axillae and abdomen. He was admitted for an acute flare of MMP, with plans to treat with IV steroids and pain control. The day after admission, he developed new painful oral lesions and targetoid lesions of axillae, palms and soles. Azathioprine was held, and an HSV NAAT from an oral blister was positive. Punch biopsy showed findings consistent with Erythema Multiforme (EM.) Valtrex therapy was started for HSV, and high-dose steroids, doxycycline, and pain medication were continued for MMP. Patient improved and was discharged with dermatology follow-up.

DISCUSSION: MMP and EM are rare, clinically distinct entities. MMP is a chronic autoimmune disease of the basement membrane, whereas EM is an acute, immune-mediated condition most commonly associated with Herpes Simplex Virus (HSV) infection. The majority of MMP cases involve oral mucosa and conjunctiva, but approximately 25% have skin involvement characterized by scattered, tense bullae arising from an erythematous base, leaving behind erosions once ruptured. Management involves immunosuppression with steroids, azathioprine, and antibiotics for superimposed infection. The lesions of EM are characterized by edematous papules resembling insect bites that progress to the well-known targetoid lesions. Up to 70% of cases have mucosal involvement. Management involves treatment of underlying etiology; i.e., antivirals for HSV or stopping an offending medication. Interestingly, our patient tested negative for serum HSV immunoglobulins and PCR one month prior, likely developing HSV while immunosuppressed from MMP treatment. This case exemplifies that patients can have new processes develop and shows the importance of maintaining a broad differential to ensure proper diagnosis and management

30) A RARE CASE OF SELECTIVE IGM DEFICIENCY IN AN OTHERWISE HEALTHY ADULT

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Case Presentation: 59 yo Caucasian male presented from an outside hospital (OSH) with primary complaint of painful "leg abscesses" of 1 wk duration. Patient was afebrile, vital signs stable and in no acute distress. PMHx significant for recurrent scrotal cellulitis for the past few years, but this was improving when the leg sores appeared. PMHx negative for diabetes, autoimmune conditions, or hospitalization for abscesses. Physical exam unremarkable except for painful eschars 1-4cm in diameter on bilateral lower extremities. Initial labs were unremarkable. General surgery was consulted to incise and drain the lesions, but US showed no fluid to drain. OSH vancomycin was continued per ID consult recommendations. Blood cultures from OSH and those obtained during this admission showed no growth at 5 days. Wound swabs from both hospitals grew MRSA. Dermatology was consulted and obtained tissue sample of the wounds. Due to rapid appearance of eschars of unclear etiology, Immunology was consulted for possible immunodeficiency. Quantitative serum IgM was low at 11mg/dL (ref range: 40-230) with IgG, IgA, and IgE normal. Repeat IgM level was significantly low at 12mg/dL. ABO typing showed patient was B+, thus isohemogglutinins were ordered. This patient had undetectable Anti-A2 (<1:2) and deficient levels of Anti-A1 (1:2) IgM with expected Anti-B (<1:2) (ref ranges: after 6-9 months of age, titers ≥ 1:4 are considered normal in ABO O patients and <1:2 in AB pts). All other immunodeficiency labs were negative. Patient received vancomycin for 10 days and eschars improved. He was discharged home on doxycycline with Immunology and Dermatology follow-up appointments.

Impact: Here we present a rare case of MRSA eschars and chronic skin infections due to Selective IgM Deficiency (SIGMD). To date, there are no reported cases of SIGMD presenting as diffuse eschars. Abnormal MRSA skin presentations in a healthy adult should trigger further investigation for underlying diseases, including primary immunodeficiencies. We are reporting this case to increase awareness among clinicians to consider Selective IgM Deficiency in patients with recurrent or unusual skin infections.

Discussion: SIGMD is a rare immunodeficiency in which patients have normal T-cell immunity, produce normal levels of IgG and IgA, but produce IgM 2 SD below the mean. This can predispose patients to unusual or recurrent infections. However, patients may be asymptomatic. It is detected by low IgM levels and low isohemogglutinins titers (predominantly IgM isohemogglutinins produced in patients with A or B blood). Our patient has low IgM levels and does not produce adequate levels of Anti-A immunoglobins for his blood type. We report a case of SIGMD in an adult who primarily presented with multiple bilateral lower extremity eschars.

31) METOLAZONE INDUCED ACUTE INTERSTITIAL NEPHRITIS

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Case Presentation: A 86 year old female with a past medical history significant for CKD stage IV presented with lethargy, rash and lower extremity edema. The patient was recovering in a rehab facility from a recent hospitalization due to an acute kidney injury (AKI) when she developed increased lower extremity edema. In response, furosemide was increased and metolazone was added. Metolazone was discontinued three days later due to worsening renal function. Three days later, furosemide was increased again and metolazone was restarted. The patient then developed a diffuse rash two days later, which prompted her medications to be held. She presented to the ED one week later following an office visit with her nephrologist for concern of persistent rash and worsening renal function. On admission, physical exam revealed a diffuse eczematous rash involving her back, arms and legs. Lab findings were suggestive of AKI due to acute interstitial nephritis (AIN) given FENa of 1.5% and white blood cell clumps on urinalysis. Dermatology and nephrology were involved and based on clinical presentation and lab findings, patient was diagnosed with AIN likely related to metolazone. The offending agent was stopped prior to admission, and she was started on oral prednisone and topical clobetasol cream. Her renal function and dermatitis rapidly improved on this treatment regimen and a renal biopsy was deferred.

Discussion: Acute interstitial nephritis is a hypersensitivity reaction involving the interstitium and tubules resulting in acute renal failure. While AIN can be caused by infections or autoimmune disorders, it is most commonly the result of drug therapy. Antibiotics account for up to half of AIN cases but many other drugs have been implicated in causing AIN including NSAIDs, loop diuretics, thiazide-type diuretics, allopurinol and PPIs. Classically, patients were described as presenting with rash, fever and eosinophilia days to weeks after beginning a new medication. In reality only 10% of patients present with this classic triad of symptoms. Many patients present with nonspecific signs and symptoms of acute renal dysfunction including nausea, vomiting and malaise. Although our patient was on three medications known to cause AIN (allopurinol, furosemide, metolazone), metolazone was likely the offending agent given this patient had extensive history with allopurinol and furosemide without reported adverse reactions.

CONCLUSION: Here we report a case of AIN in a patient who presented with rash and worsening renal function approximately two weeks after beginning metolazone. To date, there are no documented case reports on metolazone induced AIN. The purpose of this case report is to increase awareness among clinicians to consider metolazone as a medication that can induce AIN.

32) BORDETELLA PERTUSSIS IN A FOUR TIME KIDNEY TRANSPLANT RECIPIENT: A CALL FOR IMMUNIZATION PROGRAMS AT TRANSPLANT CENTERS

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Pertussis, or whooping cough, is a highly contagious respiratory illness caused most frequently by Bordetella pertussis. Clinical presentation ranges in severity, but life-threatening illness disproportionately affects children and immunocompromised individuals. Acellular vaccines for pertussis have been available for decades, and they are recommended throughout the lifespan. We describe a renal transplant patient who presented with respiratory distress and dry cough as manifestations of co-infection with Bordetella pertussis and Bordetella parapertussis/ bronchiseptica. The goal of this report is to highlight the importance of immunization programs at transplant centers, which are in the unique position to care for patients both with end-stage organ disease and in the post-transplant setting.

33) PAINLESS PURPLE PLAQUES, AN INITIAL PRESENTATION OF KAPOSI SARCOMA

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Introduction: Purple patches and plaque like lesions are commonly associated with palpable purpura and steer the differential diagnosis towards small vessel vasculitides, TTP or cryoglobulinemia. However, in a patient with high risk behaviors and without regular medical care, it is important to remember to keep Kaposi's sarcoma on the differential especially since early lesions may be mistaken for angiomas, dermatofibromas or even nevi. Even in a post HAART world there are millions of uninsured and under-insured patients without access to regular care or screening who still may present with an AIDS defining illness.

Case Summary: Patient is a 41 year old M who presented to the ER with 2 months of subjective fevers, chills, night sweats and a rash on his chest and shoulder. Patient is MSM who has had two lifetime sexual partners, both male and without regular condom use. On arrival his Temp was 99.7F, HR 106, BP 120/74 and RR 17. His exam showed no oropharyngeal lesion and no palpable cervical, axillary, or inguinal lymphadenopathy. On his chest, left palm and back were multiple irregularly shaped purple plaque-like lesions on his skin. They were non-tender and not pruritic. His initial labs were notable for a WBC 3.4 but on the differential his absolute lymphocyte count was within normal limits. HIV1 antibody test came back positive and his viral load was 242,000. His CD4 count was 34. He was admitted for infectious work-up given his subjective fevers, and night sweats; but it was negative. A skin biopsy confirmed the diagnosis of Kaposi's Sarcoma. He appeared to only have cutaneous involvement of Kaposi's at the time of diagnosis and was started on HAART medications with good response. He followed up with Dermatology outpatient, where he underwent cryotherapy and was started on topical imiquimod as an outpatient.

Discussion: Kaposi sarcoma is considered an AIDS defining illness caused by HHV8, which is This patient had not had any regular screening medical care for over 10 years and as such his HIV diagnosis went undiagnosed and as such had progressed to AIDS. Though a biopsy is not necessary to make a diagnosis of Kaposi's, it can be useful for confirmation. It is important to consider the possibility of visceral involvement in the lungs or intestines. As far as treatment, watchful waiting can be appropriate as most lesions regress with HIV treatment. However, if patient prefers there are options including cryotherapy, excision, radiation, laser therapy, intralesional vs systemic chemotherapy, topical agents, and immunomodulators such as IFN-alfa.

34) CMV INDUCED TERMINAL ILEUM STRICTURE PRESENTING AS A HIGH GRADE SMALL BOWEL OBSTRUCTION

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Introduction: Cytomegalovirus (CMV) colitis is typically found in patients that are immunocompromised and is rarely found in immunocompetent patients. CMV ileitis has been primarily described in AIDS patients and is found in 4% of CMV cases affecting the GI tract. In this case, we present an immunocompetent patient who presented with a high grade small bowel obstruction (SBO) that was secondary to CMV ileocolitis causing a terminal ileum stricture.

Case Description: A 60-year-old immunocompetent, HIV-negative black male presented with small bowel obstruction. CT scan of the abdomen showed circumferential wall thickening of the terminal ileum (TI). Colonoscopy was unrevealing due to incomplete bowel prep. He was deemed a poor candidate for surgery given severe cardiomyopathy. Outpatient colonoscopy was planned for after completion of two weeks of prednisone for presumed Crohn's disease. The patient had persistent abdominal pain requiring readmission. Colonoscopy showed ulcerations and erosions in the TI and cecum with a TI stricture that could not be transversed. Biopsies were taken and showed crypt distortion and ulceration; immunohistochemical staining was positive for Cytomegalovirus (CMV). Patient underwent bowel resection, and gross specimen further confirmed viral cytopathic change with multiple superficial ulcerations and vasculature containing endothelial cells with prominent viral inclusions positive for CMV.

Discussion: CMV ileitis is rare in immunocompetent patients. A systematic review of immunocompetent patients with CMV in any system in 2008 described 60 patients with CMV colitis. There is only one other case report of a patient with a CMV –induced colonic stricture presenting as an acute intestinal obstruction in an immunocompetent adult. This is the only case of CMV terminal ileocolitis with primary ileal involvement that caused acute intestinal obstruction in an immunocompetent adult.

35) MULTI-DRUG RESISTANT PSEUDOMONAS PNEUMONIA SUPERIMPOSED ON A MYCOBACTERIUM AVIUM COMPLEX INFECTION

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Introduction: Necrotizing pneumonia is often caused by organisms such as S. aureus and *K. pneumoniae*. In the immunosuppressed, *P. aeruginosa* and other gram-negative rods are the most common cause. For opportunistic organisms such as *Pseudomonas*, early CT imaging and frequent microbiologic testing are critical for determining effective treatments.

Case: A frail 54-year-old Caucasian woman with a history of rheumatoid arthritis treated with DMARDs and centrilobular emphysema secondary to nicotine dependence, presented with a rapidly destructive pneumonia from a community hospital. Her primary provider prescribed a course of Azithromycin and Prednisone, but her condition worsened leading to hemoptysis, hypotension, and dehydration warranting hospital admission. Her WBC count was 17.6 x 109 cells/L and CT confirmed a necrotizing pneumonia in her left upper lobe, superior left lower lobe, and infiltrates in the right lower lobe. Sputum cultures grew pan-sensitive *Pseudomonas* and *M. avium* complex species. Despite treatment, she continued to deteriorate with a complete collapse of her left upper lobe, worsening anemia and increasing *Pseudomonas* resistance. She was later started on Azithromycin, Ethambutol, and Rifabutin. The cardiothoracic surgery team believed she was not a surgical candidate due to her infectious burden. Upon transfer to our institution, ventilation-perfusion scans and pulmonary function tests confirmed no perfusion in the left upper lobe, overall decreased spirometry values, and a low DLCO. With this information, she was confirmed a candidate for a left upper lobectomy.

Discussion: The increasing resistance of *P. aeruginosa* during treatment signaled a high risk of morbidity and mortality. Frequent sputum samples and susceptibility testing are essential to ensure that patients receive the most active treatment. The one-month delay in initiating MAC therapy in this patient likely contributed to her weight loss and eventual respiratory failure. The cure rate for pulmonary MAC infection is greater than 90% if cultures remain negative for 12 months on therapy. Her underlying lung disease and immunosuppression increased her risk for acquiring a necrotizing pneumonia that could have led to her demise if a second opinion was not sought at our institution.

36) VITAMIN B12 DEFICIENCY: A CASE REPORT OF BONE MARROW SUPPRESSION

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Introduction: Vitamin B12 (Cyanocobalamin) deficiency is a common vitamin deficiency that can present with a range of clinical manifestations, including pancytopenia. This case illustrates the presentation of a common vitamin deficiency as pancytopenia that can cause higher morbidity in otherwise healthy patients.

Case Description: A 61-year-old female presented to the emergency department (ED) with generalized weakness. She recently moved from Puerto Rico and has medical history of chronic anemia, required blood transfusions in the past. She denied any melena, easy bruising or neurological symptoms. Initial work up in the ED showed significant pancytopenia with WBC count 0.5, Hemoglobin 4.3, and platelet count 37 K. MCV was elevated at 123, peripheral blood smear showed hyper-segmented neutrophils. She received

2 units of packed RBCs and was admitted to the hospital. Hematology was consulted for further work up of significant pancytopenia. Bone marrow biopsy was pursued, which showed markedly hypercellular bone marrow consistent with megaloblastic anemia in the setting of vitamin B12 deficiency. No obvious myelodysplasia or malignancy noted. B12 level was found to be <45 pg/ml (normal levels-211-911 pg/ml), folate level was normal. Patient had no symptoms of malbasorption, and did not follow a vegetarian diet. Work up for pernicious anemia, including both parietal cell and intrinsic factor antibodies was negative. Patient was treated with a course of intramascular injections of vitamin B12. Follow up with Hematology as outpatient showed significant improvement in the cell count and clinical symptoms.

Conclusion: Although vitamin B12 is a common deficiency, a clinician must be aware of the rare manifestations of this condition to adequately manage the patients. Including it the differential diagnosis, work up of pancytopenia is essential for the early management.

37) PSEUDOLEADS ON TRANSESOPHEAGL ECHOCARDIOGRAM

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INTRODUCTION: Infections involving implanted cardiac devices (ICDs) are associated with high morbidity and mortality rates. When ICDs become infected, complete removal of the device generator and leads is required to curtail the dangerous consequences of infection. Following removal of infected ICDs, unaccounted-for structures, described as 'freely floating, mobile, echodense masses have been reported on follow-up echocardiograms. The etiology of these masses has been discovered as fibrin casts that have formed around the metallic implanted cardiac device leads. Cardiac *pseudolead* casts that remain after lead extraction have implications for worsened clinical outcomes and increased mortality.

CASE REPORT: A 66-year-old Caucasian male with history of ICD placement 5 years prior, whom had recently undergone pacer generator exchange, presented with AMS and high-grade fever of 103oF. Purulent fluid expression was noted from the site of his recent generator exchange. Incision and drainage of the pacer pocket was urgently completed. The ICD was removed with cultures taken that returned growing MRSA, for which he completed a 30-day course of IV antibiotics. The patient returned two months later with leukocytosis, fever of 101.00F, and thoracic spinal pain. Blood cultures were drawn and returned growing MRSA. Transesophageal ECHO was completed revealing; three mobile vegetations noted on the right side of the heart. Follow-up chest radiograph and chest CT scan revealed no evidence of retained foreign bodies. The previous ICD removal procedure was discussed with the operating physician; reporting that all leads had been safely removed intact. Without radiopaque objects seen on follow-up chest radiograph or CT scan, it was determined that the unaccounted-for objects were in fact, pseudoleads. Pt was treated with prolonged IV antibiotics, similar to treatment course for native valve endocarditis.

DISCUSSION: Pseudoleads are associated with an elevated risk for recurrent infections and worsened clinical outcome. Discerning metallic presence of unaccounted-for-structures as seen on TEE can be accomplished by noting an absence of radiopaque objects on follow-up CXR or CT scan. Management of pseudoleads has been debated and likely falls to clinical judgment. Previous cases of surgical extraction have included; large and overtly mobile pseudoleads with elevated embolic risk. A prolonged course of IV antibiotics is warranted for pseudolead cases.

38) A CASE OF MARENTIC ENDOCARDITIS AND PARANEOPLASTIC THROMBOEMBOLISM

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Cancer induced nonbacterial thrombotic endocarditis (NBTE) formally called marantic endocarditis is a rare complication and may preceed the diagnosis of an occult malignancy. It must be considered in patients with unprovoked venous thromboembolism.

A 51-year-old female presented with sharp left sided chest pain and numbness of left arm which started while she was carrying laundry upstairs. She had a recent history of air travel in past 6 weeks and medications included estrogen-containing oral contraceptive pills. She did not have a history of Intravenous drug use or family history of coagulopathy/thromboembolism. Physical exam was unremarkable except for tachycardia. Electrocardiogram revealed T wave inversions in lateral leads and poor R wave progression. Labs showed elevated troponin T which trended upwards. Her d-dimer was elevated. Abnormal compression and filling defect were noted on DVT Ultrasound. A coronary angiogram was obtained due to worsening cardiac enzymes which showed occluded distal D1 branch not amenable to intervention. 2D Echo was negative for patent foramen ovale. She was discharged home on medical management for non-ST elevation myocardial infarction and anticoagulation with Apixaban for deep venous thrombosis and pulmonary embolism. Two weeks later, she returned to the emergency department with intermittent neurological symptoms including word finding difficulty, dysgraphia, anomic aphasia and transient numbness in bilateral hands. MRI head revealed multiple small infarcts of varying ages involving multiple vascular territories. Transesophageal Echocardiogram was obtained revealing a 0.5 x 0.6 cm echogenic globular mass attached to the mitral valve leaflets. Blood cultures returned negative. Further workup with CT revealed enlarged mediastinal, abdominal and pelvic lymph nodes. Biopsy of the lymph node showed metastatic cancer of mullarian origin.

Non-bacterial thrombotic endocarditis (NBTE) was first described by Zeigler in 1888, which is a disease characterized by the presence of vegetations on cardiac valves consisting of fibrin and platelet aggregates and are devoid of inflammation or bacteria. It is a separate entity to culture-negative endocarditis which is due to infectious etiologies that are not readily identified on regular culture media. The major clinical manifestations result from systemic emboli rather than valvular dysfunction itself. A workup for a hypercoagulable state, disseminated intravascular coagulation, malignancy and systemic lupus erythematosus should be performed in every case of suspected NBTE. Treatment consists of management of underlying condition. Anticoagulation is required to prevent thromboembolism and valvular surgery is occasionally needed in selected cases.

39) MYOCARDIAL BRIDGE AS A CAUSE OF ACUTE MYOCARDIAL INFARCTION

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Patients with myocardial bridging are commonly encountered clinically and may present with exertional symptoms of myocardial ischemia, syncope, and even sudden death. Careful evaluation using an array of noninvasive and invasive diagnostic modalities can be used to evaluate patients who are symptomatic.

A 55 year old male patient with no known coronary artery disease, presented with exertional shortness of breath and vague symptoms of generalized weakness. The exam revealed irregularly irregular heart rate and bilateral crackles. Electrocardiogram revealed ST elevation in anterior and lateral leads. Cardiac catheterization was performed revealing a small caliber left anterior descending vessel exhibiting severe spasm in the setting of a long intra-myocardial segment extending almost from proximal to entire mid segment of the vessel. The vessel diameter increased with diastole and decreased with systole. There was some resolution of spasm and ST elevation with intracoronary nitroglycerin with consequent improvement in vessel diameter. Transthoracic echocardiogram showed abnormal apical wall motion consistent with findings on coronary angiogram. Cardiac MRI was obtained showing near transmural delayed enhancement of the mid to apical anterior wall suggestive of myocardial infarction.

Myocardial infarction with normal coronary arteries is a conundrum with multiple etiologies. Myocardial bridging is an inborn coronary abnormality which is usually being. Symptoms usually start in the third decade. Pathophysiology includes a segment of a major epicardial coronary artery called the 'tunneled artery', that goes intramurally through the myocardium. The muscle covering the artery is called 'myocardial bridge'. It is most common in mid left anterior descending artery. Frequency reported varies from 1.5 to 16. It should be considered in patients who have angina but lack risk factors or have normal coronaries at coronary angiography. Angiographic diagnosis is based on a change in diameter of more than 70% during systole, creating a significant "milking effect". The myocardial bridge is a systolic angiographic event and it acquires clinical relevance only in particular conditions, such as tachycardia. Tachycardia can provoke an ischemic effect by shortening the diastolic phase and increasing the importance of systolic blood flow. First Line treatment includes Beta Blocker and non-dihydropyridine calcium channel blockers. Caution should be exercised in using nitrates which can exacerbate ischemia from bridging by causing reflex tachycardia. For select patients refractory to intensified medical therapy, surgical intervention, or less preferably percutaneous intervention, can be considered.

40) DISSEMINATED BLASTOMYCOSIS IN THE IMMUNOCOMPETENT: A CASE REVIEW

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INTRODUCTION: Blastomycosis is a fungal infection endemic to states bordering the Mississippi and Ohio rivers and the Great Lakes with the Milwaukee River Watershed being hyper-endemic. Infection is most commonly suspected with pulmonary findings on radiography with or without symptoms. Skin lesions are the most frequent extrapulmonary finding of infection. Disseminated blastomycosis, particularly involving the central nervous system, is rarely seen in the immunocompetent, but high index of suspicion should be maintained.

CASE PRESENTATION: A 57-year-old female with history of bipolar disorder and native to Milwaukee, Wisconsin presented to her primary care physician with 2-weeks of dizziness, falls, and headaches. MRI revealed 2 cerebellar lesions concerning for malignancy, and the patient was admitted. She had no history of immunodeficiency, and HIV testing was negative. Admitting physical exam was notable for dysmetria, gait instability, and disorientation. Skin exam showed a 2.3 cm violaceous nodule on the abdomen. She underwent craniotomy with resection of the cerebellar masses; dermatology biopsied the skin lesion. CT of the chest, abdomen, and pelvis showed no evidence of pulmonary disease or intraabdominal pathology. Biopsy specimens showed blastomycosis. She was started on amphotericin therapy with plan to transition to voriconazole.

DISCUSSION: Blastomycosis is caused by inhalation of the fungal spores. Manifestations are divided into pulmonary and extrapulmonary. More than half of infected individuals are asymptomatic. Symptomatic patients usually present with acute or chronic pneumonia. Extrapulmonary disease typically affects the skin, bones, and genitourinary tract. Rarely, CNS involvement can lead to meningeal signs or focal neurologic deficits. Nervous system infection is frequently seen in HIV/AIDs patients; less than five percent of immunocompetent hosts suffer CNS disease. Definitive diagnosis requires culture with growth of *B dermatitidis* but is supported by direct visualization of characteristic budding yeast, imaging, or antigen testing. Mild disease without CNS involvement is treated with oral azole therapy. In moderate to severe disease or in those with CNS lesions, amphotericin therapy is warranted, followed by itraconazole. Ninety percent of patients are cured of disease with appropriate treatment. Poorer prognosis is predicted in immunocompromised individuals and those with CNS disease.

41) EVALUATING END OF LIFE REGRET THROUGH COMMUNITY INTERVENTION

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Introduction: Increasingly, patients and providers alike are interested in achieving a "Good Death", a concept that focuses on completion and meaningfulness at the end of life (1). This project aimed to address this by evaluating end of life regret.

Objective: Using the Stanford Life Review templates (2), a freely accessible public resource, I hypothesized that guided writing would help participants address regret while they were still healthy.

Methods: I organized three workshops with 47 total participants aged 61 to 89. Each participant took a pre-survey assessing the participant's current level of regret. Subsequently, I led the course which utilized guided writing prompts on topics ranging from remembering key moments to apologizing for past mistakes. After, I asked participants to complete a post-survey.

Main Results: In our pre-course survey, I found that 49.9% of participants feel guilty about unresolved disagreements with loved ones. Further, 47.8% of participants agree that they find themselves regretting life choices. In our post survey, 77.8% of participants indicated that writing letters would help them plan for the future and 80.4% of participants planned on sharing their letters.

Conclusion: These results indicate that unresolved disagreements and negative life choices are the largest area of regret among the elderly population sampled. The positive post-survey responses highlight that writing exercises could help address these areas, potentially reducing suffering at end of life.

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42) STATIN-INDUCED NECROTIZING AUTOIMMUNE MYOPATHY

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Statins are widely used for primary and secondary prevention of cardiovascular events. The spectrum of statin-associated muscle adverse events includes myalgia, myopathy, myositis, rhabdomyolysis and myonecrosis. Here, we present a rare case of statin-induced necrotizing autoimmune myopathy.

A 76 year-old man was admitted for diffuse myalgias and proximal muscle weakness, with a creatine kinase (CK) of 14,000. He started atorvastatin 2 years previously for peripheral vascular disease. His statin was discontinued and he was discharged to a rehabilitation center. He was readmitted 3 months later with worsening symptoms. He denied fever, dysphagia, shortness of breath or rashes. CK was 8960. Thyroid, parathyroid function and electrolytes were normal. Infectious workup and myositis antibody panel was negative. Right thigh muscle biopsy showed necrotizing myopathy, without inclusion bodies on electron microscopy. Antibodies against 3-hydroxy-3methylglutaryl-coenzyme A reductase (HMGCR) were elevated >200 AU/ml (normal <20AU/ml). A diagnosis of statin-induced necrotizing autoimmune myopathy was made and prednisone 1mg/kg/d was initiated. The patient also received intravenous immunoglobulin (further doses held due to rash) and azathioprine. He currently takes prednisone and azathioprine and is able to ambulate independently.

The incidence of statin-induced necrotizing autoimmune myopathy is about 2-3 new cases among 100,000 patients exposed to a statin. The presence of antibodies against HMGCR is a diagnostic clue and should prompt further workup with muscle biopsy to confirm diagnosis. Although rare, internists must have high suspicion for this condition in patients that do not improve rapidly following withdrawal of the statin. In patients with high cardiovascular risk, alternative therapies like proprotein convertase subtilisin/kexin type 9 (PCSK9) inhibitors may be considered. Early recognition and institution of immunosuppression along with rehabilitative therapies may be beneficial in treating this condition.

43) UNUSUAL TISSUE- UNUSUAL ISSUE. PANCREATIC HETEROTOPIA PRESENTING AS GASTRIC OUTLET OBSTRUCTION.

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Gastric outlet obstruction can be seen in malignancy, peptic ulcer disease, Crohn disease, caustic ingestion, large polyps, gastric bezoars and Bouveret syndrome. Herein, we report a rare case of pancreatitis occurring in heterotopic pancreas presenting as symptomatic gastric outlet obstruction in a young adult.

A 27 year old previously healthy Caucasian female presented with 3 week history of burning epigastric pain, nausea, early satiety and constipation (without obstipation). Abdominal exam revealed tenderness in epigastric and right upper quadrant area without a palpable mass. Laboratory workup including liver function tests, lipase and sonogram were unremarkable. A CT scan showed a 5cm cystic mass arising from the gastric antrum resulting in pyloric stenosis and partial gastric outlet obstruction.

Esophagogastroduodenoscopy demonstrated a single, inflamed, umbilicated lesion measuring 30mm in diameter in the prepyloric region suspicious for aberrant pancreas (pancreatic rest). Endoscopic Ultrasound (EUS) revealed no clear cystic lesion. Fine needle aspiration(FNA) was consistent with pancreatic heterotopia with acute inflammation. The patient was treated conservatively and discharged to home with advice to maintain hydration and avoid alcohol.

Pancreatic heterotopia (pancreatic rest) is a congenital anomaly defined as pancreatic tissue lying at a non- physiological site without any vascular or anatomic continuity to pancreas proper. They are usually asymptomatic and only incidentally detected upon pathophysiological exam or autopsy (incidence -0.5%-13.0%). They typically appear as an umbilicated, submucosal mass in the gastric antrum on upper endoscopy although they can be found in any location in the stomach or proximal duodenum. Diagnosis is typically based off of a classic appearance, but in the case of complications or atypical location, EUS guided evaluation and FNA is required. Although rare, pancreatic heterotopia must be included in the differential diagnosis of a submucosal prepyloric mass in young patients even without elevated lipasemia.

44) DYSPHAGIA AS PRESENTING FEATURE OF DERMATOMYOSITIS

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Introduction: Dermatomyositis is an idiopathic inflammatory myopathy that typically causes proximal muscle weakness and features classic skin findings, usually with onset in fifth or sixth decade of life. Affected individuals will often have weakness symmetrically in the deltoids or hip flexor muscles. Likewise, the striated muscles of the upper esophagus can be affected leading to trouble swallowing and increased risk of aspiration. The most common cutaneous changes include a heliotrope rash on the upper eyelids and Gottron's papules located over the dorsal aspects of the interphalangeal joints. Rashes located on the chest, back, face, scalp, and hips may also be present.

Case: This is a case of a 79-year-old who initially presented to an outside hospital with complaints of a sore throat and dysphagia for 2 weeks. Patient was also found to have heliotrope rash, Gottron's papules, and Shawl sign. In addition, patient also stated that he could not lift his arms above his head due to weakness. EMG directed muscle biopsy taken from the patient's right deltoid showed findings consistent with a nonspecific, active and chronic inflammatory myopathy with perifascicular atrophy, features consistent with dermatomyositis. Stains more specific for dermatomyositis were ordered. Patient was initially treated with high dose methylprednisolone, and IVIG was given for a five-day course to help with the patient's swallow function. Finally, azathioprine was prescribed based on the severity of the patient's illness. Patient demonstrated improvement in proximal arm weakness, but there was minimal improvement in swallow function, necessitating PEG tube placement.

Discussion: This is an interesting case where development of dysphagia in the setting of rash prompted further work up including muscle biopsy leading to diagnosis of dermatomyositis. Patients with undiagnosed dermatomyositis typically present with complaints of proximal arm or leg weakness along with a rash, but it is important to highlight that a patient's primary muscle weakness may be in the pharyngeal muscles. Therefore, it is important to keep dermatomyositis as a differential diagnosis in a patient presenting with dysphagia in setting of skin rash.

45) HITTING THE LAD WITHIN THE RADIATION FIELD

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Radiation therapy to the chest is a known risk factor for coronary disease. We describe a case of acute coronary syndrome in a young female with a history of radiation therapy to her right breast two years prior.

A 47-year-old woman presented to the with two days of worsening substernal chest pain. Her past medical history was significant for obesity (BMI of 33 kg/m2) and right sided ductal carcinoma in situ (DCIS) status post lumpectomy and radiation therapy two years prior. Pain was described as an intermittent pressure without radiation or associated symptoms when it began. Pain persisted for longer periods and was associated with flushing, diaphoresis and radiation along her left arm, causing her to seek medical care. An electrocardiogram (EKG) done at an outpatient clinic showed anterolateral T wave inversions. She was given 324mg aspirin and sublingual nitroglycerin before being taken to the emergency department (ED).

On arrival, the patient was afebrile, pulse rate 81 beats per minute, blood pressure 146/77 mmHg, respiratory rate 16 per minute and was saturating 97% on room air. Physical exam revealed an obese female with normal first and second heart sounds and regular rate and rhythm. No lower extremity edema or jugular venous distension was noted. Chest pain was not reproducible on palpation and lungs were clear to auscultation. Initial EKG in the ED showed normal sinus rhythm with anterolateral T wave inversions. Blood work was drawn and treatment for unstable angina started with dual antiplatelet therapy and a heparin infusion. Serial EKGs showed dynamic anterolateral ST-T segments. Serial troponins were undetectable and basic metabolic panel was within normal limits. Left heart catheterization with coronary angiography revealed mild to moderate systemic hypertension, a mildly elevated left ventricular end-diastolic pressure and an eccentric, tubular 90% stenosis of the proximal left anterior descending artery (pLAD) with TIMI 3 flow. The remaining coronaries had mild luminal irregularities. The pLAD lesion was treated with a drug eluting stent resulting in improved angiographic appearance and continued TIMI 3 flow.

The increased risk of coronary disease with radiation therapy has been demonstrated to be dose-dependent. The risk of developing a major coronary event is reported to increase by approximately 7-8% per unit of radiation. This risk is increased when combined with underlying risk factors for heart disease. With evidence of cardiac changes as early as >6 months post treatment, physicians should be aware of radiation exposure as an important risk factor among younger patients without known atherosclerotic disease or frequently encountered risk factors such as family history, high blood pressure, diabetes mellitus or smoking.

46) FATAL HYPERMAGNESEMIA FROM ACCIDENTAL OVERDOSAGE WITH EPSOM SALT IN A PATIENT WITH NORMAL RENAL FUNCTIONS.

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Background: Generally, it is very rare for clinically significant hypermagnesemia to develop in an individual with normal renal functions as the renal handling of serum magnesium is a very potent process and it has the capacity, under conditions of hypermagnesemia, to completely block Mg (magnesium) reabsorption from the thick ascending limb of Henle. Therefore, hypermagnesemia usually arises in the setting of renal failure.

Case presentation: We present a very rare case of 40 years old African American obese female with prior normal renal functions, who presented post-cardiac arrest following accidental overdosage with Epsom salt. The patient was initially given supportive therapy and was later considered for the dialysis despite normal renal functions, as serum Mg levels kept on creeping up and clinical status kept on deteriorating continuously.

Conclusion: A seemingly harmless magnesium containing OTC (Over-The-Counter) can potentially be lethal, and such consequences must always be taken into account when using such medications for a prolonged period of time.

47) IATROGENIC CELLULITIS FROM PNEUMOCOCCALVACCINATION: RAISING THE QUESTION OF ARTHUS REACTION

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Introduction: Inpatient vaccination through standing order programs represent a public health initiative to improve immunization rates in an at-risk population and overall herd immunity. The incidence of harms associated with this practice is largely thought to be clinically insignificant. There have been rare case reports of immunogenic adverse effects previously linked to vaccines, particularly tetanus, rabies, pneumococcal and hepatitis B mostly within the pediatric literature. We describe a case of readmission that was directly caused by inpatient pneumococcal vaccination.

Case: A 46-year-old man with history of pontine brain abscesses presented with severe headache and fever found to have new enlarged enhancing lesions within the central pons and right thalamus. He was started on IV ertapenem, trimethoprim-sulfamethoxazole, and linezolid with overall improvement in the recurrent abscesses. On the day of discharge, he received the PPSV23 vaccine. Two days later, he developed fever of 103°F, chills, nausea, vomiting and acute-onset left shoulder erythema around the vaccination site with significant pain exacerbated by movement, prompting readmission. Relevant lab results included worsening leukocytosis and inflammatory markers with WBC increased from 8K/uL to 33.6K/uL, CRP from 1.6mg/dL to 30.5mg/dL and ESR from 23mm/hr to 47mm/hr. Due to concerns for potential septic arthritis based on physical exam, he underwent arthrocentesis which revealed total cell count of 33 and was negative for crystals. Ultrasound of the shoulder demonstrated findings of cellulitis without abscess or subcutaneous gas. Blood and synovial cultures remained negative. His antibiotics were switched to vancomycin and cefepime for presumed cellulitis with rapid improvement in his pain and range of motion over 24 hours. He was discharged on his home antibiotic regimen for the underlying central nervous system infection.

Discussion: The development of cellulitis from inadequate sterile technique during vaccination suggests the transmission of a very large inoculum of bacteria. However, this is a surprising finding in a patient on numerous antibiotics with staphylococcal and streptococcal activity. Given the unlikeliness of this clinical scenario, providers should consider the possibility of a noninfectious Arthus reaction in their differential.

48) RHEUMATOID ARTHRITIS: AN UNUSUAL PRESENTATION

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Introduction: Rheumatoid arthritis is a common chronic systemic inflammatory disorder that most commonly affects joints causing progressive symmetric erosive destruction of cartilage and bone. Although it primarily affects joints, it also causes extra articular manifestations in about 40 percent of patients with RA. Of all the extra articular manifestations, pulmonary involvement is the most common and is major contributor to morbidity and mortality.

Case: A 64 year-old Caucasian gentleman with history of smoking, presented to ED with, worsening shortness of breath, mild cough, malaise, and fatigue for 4 days and fever for 1 day. Denied any chills/rigors or upper respiratory symptoms. Denied exposure to sick contacts or travel history. About two months ago, he had polyarthritis which was treated with NSAIDS and a short course of steroids. Physical exam relevant for low grade fever, hypoxia and bibasilar crackles. Blood work notable for elevated ESR with negative infectious work up. CT chest showed a small pleural based pulmonary nodule and bilateral ground glass opacities. He was empirically started on antibiotic. However with worsening hypoxia, he was evaluated with bronchoscopy. BAL showed neutrophil predominance. Cultures were negative. Serology was significant for very high titers of anti CCP antibodies. He was diagnosed to have rheumatoid arthritis with interstitial lung disease. Subsequently he was started on immunosuppressive treatment. Patient showed improvement of symptoms and was discharged home. He was maintained on long term steroid and rituximab. Currently patient is symptom free.

Discussion: There are a variety of pulmonary manifestations of rheumatoid arthritis- interstitial lung disease (ILD), pleural thickening and effusions, vasculitis and pulmonary hypertension. Pathogenesis of which is secondary to chronic inflammation, infection or immune modulating medications. High resolution CT scan forms the basis of diagnosis, though surgical biopsy is gold standard. Treatment with anti-inflammatory and / or immunosuppressive agents is recommended. This case illustrates the rare unusual presentation of rheumatoid lung disease, highlighting prompt identification of lung disease and close monitoring during therapy.

49) REMITTING SERONEGATIVE SYMMETRICAL SYNOVITIS WITH PITTING EDEMA (RS3PE) SYNDROME

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Introduction: Remitting seronegative symmetrical synovitis with pitting edema (RS(3)PE) syndrome is a rare inflammatory arthritis, characterized by symmetrical distal synovitis, pitting edema of the hands and feet, absence of rheumatoid factor, and favorable response to glucocorticoids.

Case: A 73-year-old male patient with a significant past medical history of focal encephalomalacia, chronic right shoulder discomfort, and left hip pain, came in with a chief complaint of bilateral hand and foot swelling. Patient was not on any medications at the time of presentation. Patient noticed some swelling over the past couple of months which gradually got worse over the past couple of weeks before presentation. The swelling was localized to the hands as well as the feet. Patient described pain along with the swelling. He also did complain of morning stiffness with the swelling. He denied any fevers or chills. On exam there was bilateral 2+ pitting edema of both hands just off MCPs and MTPs along with mild tenderness.

Results: Lab tests showed elevated ESR of 42 with an elevated CRP of 2.4. CCP as well as rheumatoid factor was negative. Other lab tests showed a low albumin of 3.8 and slightly low hemoglobin at 12.7. Bilateral hand x-rays showed severe bilateral hand swelling along with chondrocalcinosis and osteoarthrosis. He was started on a tapering dose of prednisone with an excellent response.

Discussion: Although there are overlapping clinical manifestations among RS3PE, elderly-onset rheumatoid arthritis, and polymyalgia rheumatica, RS3PE has distinct characteristics. RS3PE can be associated with neoplasia and various rheumatic conditions, suggesting that it may be heterogeneous. It can be considered a paraneoplastic rheumatic disease. Treatment for this syndrome includes low-dose corticosteroid preparations or hydroxychloroquine. Signs and symptoms usually remit within 18 months after initiation of treatment.

50) M3 HOSPITALIST ROTATION EDUCATION

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Background: With recent restrictions on resident work hours, many more academic programs are developing "non-covered" hospitalist service similar to the community model. At many academic institutions, hospitalists are increasingly acting as the teaching attendings on inpatient general medicine rotations. There are many published studies on medical students experience on a traditional ward rotation with a team including house staff and medical students. But there have been few studies demonstrating perceived benefits of hospitalist faculty on medical education in academic hospitals.

Methods: A Qualtrics survey was emailed to 53 third year medical students at the Medical College of Wisconsin who had completed 4 weeks rotation with hospitalist. All of the questions in the survey were focused on obtaining the students's perceptions about rotation with hospitalists, its benefits, and the perceived challenges and barriers. It also included questions about preferred number of patients and type of cases assigned to them. Responses were obtained on a five-point Likert scale. Survey questions were multiple choice but allowed for comment submission. 49 of 53 (92.5%) total eligible students responded to the survey.

Results: 3.6 was the average number of patients responders thought they should be assigned, with the mode being 3. 0% of responders thought that one patient was the maximum number they should care for, 6% thought *two* patients, 48% *thought three* patients, 36% *thought* four, and 10% *thought* five. Majority of students reported the opportunity for one-on-one interactions with several attendings in a month of rotation as a benefit. Other perceived benefits included exposure to unique patient care styles of different attendings, exposure to large number of diverse pathology, autonomy/increased level of independence The main barriers expressed by the students pertained to the challenge of scheduling dedicated teaching time while attending physicians are busy with patient care and variability in teaching styles.

Conclusions: Our medical students perceive educational benefits to rotation with hospitalist attendings on medicine ward. But there are some barriers that indicates the need for innovation. Third year medical students on a hospitalist internal medicine service think that they should ideally follow 3-4 patients on average. Several comments have indicated variability due to previous clinical experience or the complexity of the patients being cared for. Further study will be necessary to identify an optimal curriculum for such a rotation and to evaluate the impact on students' career choices and perceptions of the field of internal medicine.

51) A CAT CHASE GONE WRONG: COMPLICATIONS FROM STRESS – INDUCED CARDIOMYOPATHY

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Introduction: Stress-induced cardiomyopathy is an increasingly diagnosed syndrome of transient left ventricular systolic and diastolic dysfunction following physical or emotional stress. Echocardiographic findings typically show apical ballooning and hyperkinetic basal walls which take the shape of an octopus trap, also known as "takotsubo" in Japanese. In most cases, patient's symptoms and function resolve with supportive care.

Case: A 69-year-old female with hypertension, hyperlipidemia, and diabetes mellitus type II presented to the emergency department with acute onset chest pain radiating to her neck and arm. Her symptoms developed shortly after chasing her cat who had escaped the house. EKG showed non-specific ST-segment changes, in normal sinus rhythm. Her troponin was elevated. Coronary angiography revealed normal coronary arteries. Echocardiogram showed reduced left ventricular ejection fraction of 31% with apical hypokinesis and preserved basal function. A diagnosis of takotsubo stress induced cardiomyopathy was made. She was diuresed with an intravenous loop diuretic and started on a beta-blocker and an angiotensin-converting-enzyme inhibitor. Two days later, her symptoms had resolved and she was discharged home. Shortly after returning home, she developed acute onset right sided weakness and expressive aphasia consistent with left middle cerebral artery embolic stroke. Tissue plasminogen activator was given and mechanical thrombectomy was performed. Repeat EKG showed new atrial fibrillation with rapid ventricular response. An echocardiogram did not show a thrombus or shunt, but did show improvement of her ejection fraction. She was started on oral anticoagulation for atrial fibrillation. Several weeks later, she continued to have severe aphasia and right sided extremity weakness.

Discussion: Stress-induced cardiomyopathy is a relatively new diagnosis first described in 1990. As a result, there is a lack of randomized data that helps guide management and prevent complications. General principles are to provide supportive care that align with current heart failure. However, this case highlights the need for more specific recommendations regarding issues such as anticoagulation. For our patient, anticoagulation was not pursued as she was in normal sinus rhythm at discharge and did not have a thrombus on echocardiogram. Hopefully, the future will provide us with more evidence based guidelines to prevent complications of this syndrome.

52) A CASE OF 5-FLUOROURACIL INDUCED CORONARY VASOSPASM

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5-Fluorouracil is a commonly used adjunctive chemotherapy in the treatment of colorectal cancers. A rare but serious complication of 5FU is cardiotoxicity associated with coronary vasospasm, ventricular dysrhythmia, and myocardial infarction.

69 year old man with history notable for metabolic syndrome but no known coronary artery disease and recent diagnosis of stage IIIB rectal adenocarcinoma status post diverting ileostomy presented to the ED with substernal chest pain in the setting of continuous 5-FU infusion that was started 2 days prior to admission. ROS was negative for recent changes in activity tolerance, dyspnea on exertion or viral URI symptoms. On exam, he was hemodynamically stable, afebrile, and without evidence of elevated JVP/ murmurs/crackles/lower extremity edema. Serial EKGs showed nonspecific T wave abnormalities and diffuse ST changes likely to be early repolarization (as seen in previous EKGS). However, there were some dynamic but nonspecific ST segment elevation and T wave inversions in the anterior leads while having pain. Multiple sublingual nitroglycerin doses failed to provide pain relief and nitroglycerin infusion was initiated. Troponins x3 was negative. TTE showed normal diastolic and systolic function (LVEF 60-65%) without any evidence of valvular disease or regional wall abnormalities. However, given his risk factors for CAD and refractory pain, he underwent a coronary angiography that showed one vessel disease of the RCA (60-70% stenosis) and non-obstructive disease in the LAD. No stents were placed and patient was managed medically with aspirin, atenolol, atorvastatin, and isosorbide mononitrate. Lisinopril and spironolactone were held due to pre-renal AKI. Patient remained pain free for the next 72 hours without any telemetry events and was discharged to home with oncology follow up. Patient's symptom was most likely due to coronary vasospasm induced by 5FU given the temporal relation of events.

The pathophysiology of 5-FU induced cardiotoxicity remains unclear and likely multifactorial. Recommended management include 1) prompt cessation of the drug, 2) administration of either nitrates or calcium channel blockers, and 3) monitor cardiac enzymes, and proceed with coronary angiography in cases of refractory pain, hemodynamical or electrical instabilities.

53) ACUTE B SYMPTOMS IN TICK-BORNE ILLNESS

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Persistent fevers despite extensive initial work up pose diagnostic challenges. Diagnoses are particularly difficult if the underlying condition presents without classic signs.

The patient is a 48 year old male with no past medical history, recent travels to Mexico, Colorado and northern Wisconsin, who presented to the Emergency Department for an 11 day history of ongoing fevers. Symptoms started with a stiff neck, body aches, and a temperature of 104F. He had drenching night sweats and 10 pound weight loss over the last 2weeks. He denied any rash, cough, shortness of breath, abdominal pain, diarrhea, vision changes, mentation changes. He had sought out another ED 1 week prior, laboratory studies pertinent for thrombocytopenia 122 and mild transaminitis. A mononucleosis test, Lyme IgM/IgG, West Nile test, and blood cultures were negative. Urinalysis and Chest X-ray were normal. He was diagnosed with an unspecified viral infection and sent home. Upon admission to our facility, his temperature was 102.2F, had no neck stiffness, no joint swelling, neurologic exam unremarkable. Skin exam revealed a new rash of non-tender, flat, lightly erythematous ovoid/circular lesions roughly 10cm in diameter, some coalescing throughout his back, flanks, abdomen, and thighs. He had a leukocytosis of 15.5, normal hemoglobin and platelets. LFT's were unremarkable. Lactic acid was normal. ECG, chest xray, and urinalysis were normal. Blood cultures were negative. CT chest/abdomen/pelvis was unremarkable. Re-examination of the patient revealed the lesions had developed central clearing. Lyme IgM antibody was positive, clinching the diagnosis of Lyme's disease. He was treated with Doxycycline, fevers and rash resolved within 48 hours of treatment.

Lyme's disease prevalence is on the rise. Its manifestations go beyond the well-known single erythema migrans lesion. When early localized disease is present, fatigue, myalgia, headache, stiff neck, and fevers are observed. Although the classic EM lesion occurs in about 80% of cases, it can occur 3-30 days post-bite and only an estimated 25% of patients recall the lesion. EM lesions often lack the central clearing and are uniformly erythematous. Lyme IgM takes 7-10 days before becoming positive. In early disseminated disease, multiple EM lesions can arise. At this stage, neurologic and cardiac findings can occur. Recognizing Lyme's disease and treating early can prevent disease progression and complications.

54) APPROACH TO THE HIV+ PATIENT WITH A VAGUE HISTORY DUE TO PSYCHIATRIC COMORBIDITIES

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CASE: A 65-year-old man with past medical history of HIV type B and delusional projection disorder presented with non-bloody diarrhea and fatigue of unknown duration. Review of symptoms was positive for diarrhea, abdominal pain, fever, chills, weight loss, nausea, and shortness of breath with a nonproductive cough. Patient was a vague historian and had known delusions regarding the etiology of his HIV transmission. He was homeless and denied sexual activity for over 2 years. Previously on Stribild© (ART) but discontinued three years ago due to access difficulties. Last CD4 count 1 year prior (off ART) was 67 with a viral load of <20. Initial vitals were unremarkable. Bilateral bibasilar rales and rhonchi in left lower quadrant were noted on physical exam. Basic labs were unremarkable apart from a K+ of 2.8. UA, C diff., and influenza were negative. Diagnostic workup consisted of both pulmonary and GI components. Lactate dehydrogenase was 213U/L, which was not suggestive of disseminated histoplasmosis or pneumocystis jiroveci pneumonia (PJP). A bronchoscopy with bronchoalveolar lavage (BAL) was performed in anterior segment of right lower lobe and showed probable pneumonia. Direct fluorescent antibody testing on BAL was positive for PJP. Cytomegalovirus (CMV) PCR of the BAL was also positive. Stool cultures were negative for cryptosporidium, giardia, microsporidia, salmonella, shigella, campylobacter, and E.coli O157. Stool calprotectin did not indicate an inflammatory pathology such as inflammatory bowel disease. Cytomegalovirus (CMV) DNA was significantly elevated in the blood. Given a lack of other infectious etiologies, diarrhea was attributed to CMV. By the time of discharge, diarrhea was resolved. Absolute CD4 count was 33 cells/uL with a viral load of 31,300 copies/mL. He was discharged with TMP-SMX, Genvoya©, and acyclovir. DISCUSSION: Our case exemplifies the impact that psychiatric comorbidities can have on the ability of a provider to perform an adequate history and, which can incite a circuitous diagnostic course. The presentation of a HIV positive patient with presumed immunodeficiency broadens both the differential diagnoses and the possible workup. With the advent of ART, HIV has joined the rank of manageable and chronic illnesses. However, because of an increased susceptibility to psychiatric comorbidities, the reliability of history needs to be carefully assessed as noncompliance to ART merits a more thorough work up.

55) ACUTE LIVER INJURY: NOT ALWAYS AN "AUTO"MATIC DIAGNOSIS

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Introduction: Autoimmune hepatitis is a less frequent cause of acute liver injury. It typically presents with aminotransferase elevations or a cholestatic picture for which the differential is broad. We present a case of autoimmune hepatitis in which the patient had multiple potential causes of liver injury, highlighting the importance of including autoimmune hepatitis in the differential of acute liver injury.

Case: A 53-year-old female with a history of HIV on ART, depression, and hypothyroidism who presented with 3 months of fatigue, malaise, and abdominal discomfort. She switched from fluoxetine to duloxetine about 3 months ago and attributed her symptoms to stress from her father's recent death. A month prior to admission she was found to have mildly elevated aminotransferases and an elevated total bilirubin and alkaline phosphatase. This was initially attributed to duloxetine and with low suspicion for ART playing a role because her HIV had remained suppressed on this regimen for many years. She was transitioned to fluoxetine, but her liver function tests did not improve, so approximately 2 months later she was admitted. After a normal liver ultrasound, she was found to have a positive ANA and anti-smooth muscle antibody. Liver biopsy confirmed a diagnosis of autoimmune hepatitis. She was started on prednisone and transitioned to azathioprine, with concordant improvement in her liver function tests.

Discussion: The most common causes of acute liver injury in adults are viral and drug-induced hepatitis. The initial symptoms of acute liver injury are nonspecific, including fatigue, nausea/vomiting, and abdominal pain. The diagnostic work-up for acute liver injury is largely based on the history, since physical exam findings tend to be nonspecific and generate a broad differential. This case demonstrates the importance of a thorough laboratory evaluation for acute liver injury. Despite a suspected drug-induced cause based on history, laboratory evaluation suggested autoimmune hepatitis, which was confirmed on liver biopsy. Autoimmune hepatitis is most commonly characterized by antibodies to ANA and/or smooth muscle. Patients with autoimmune hepatitis can present on a spectrum, from asymptomatic to acute liver failure. While this disease is usually confined to the liver, it can be associated with other autoimmune conditions. Treatment of autoimmune hepatitis is important in order to prevent progressive liver disease.

56) RENAL INJURY IN HEPATIC DISEASE, THE ROLE OF GOLDEN CASTS.

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A 49 year old male with no past medical history initially presented with a chief complaint of jaundice. His exam was noticeable for pronounced jaundice, spider angiomas, and ascites, he demonstrated minimal encephalopathy, He was admitted and diagnosed with severe alcoholic hepatitis. A broad workup for alternative causes of liver disease did not reveal an additional etiology. Lab work was notable for severely elevated bilirubin, (>30mg/dL), normal synthetic function and normal renal function. He was discharged on prednisolone and lactulose with hepatology follow-up. The patient was re-admitted two weeks later with a chief complaint of fatigue. Lab work done on re-admission continued to demonstrate findings consistent with acute on chronic liver failure with persistently elevated Tbili (37.7mg/dL),and new worsening synthetic function with INR 3.2 and albumin 2.7g/dL. Re-admission labs also were notable for new onset renal failure with BUN/Creatinine of 90/3.2 mg/dL, at discharge two weeks ago he had normal values, 13/0.73mg/dL. He was given normal saline and albumin for fluid resuscitation, without improvement in renal lab abnormalities. Renal ultrasound did not demonstrate nephrolithiasis or hydronephrosis. Urine microscopy was significant for the finding of golden tubular casts consistent with a diagnosis of cholemic nephrosis, also known as bile cast nephropathy (microscopy pictures included). Cholemic nephrosis is a under recognized etiology of renal failure in patients with hepatic disease. It is a tubular nephropathy that can be seen in any cause of hyperbilirubinemia. The pathophysiology is thought to be a consequence of bilirubin cast formation secondary to prolonged hyperbiliurinemia. Bilirubin and bile salts that compose the casts are directly toxic to tubular epithelium. The goal of treatment is normalization of hepatic function when possible or addressing alternative causes of hyperbilirubinemia. The focus of the poster/vignette would be to increase recognition of cholemic nephrosis as a unique etiology for kidney failure in patients with liver disease, outside of the often-diagnosed pre-renal azotemia or hepatorenal syndrome.

57) HEMOPTYSIS AND DYSPHAGIA: UNIFYING THE DIANGOSIS

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A 70 year old male with a past medical history of hypertension, hyperlipidemia, diabetes, alcoholic cirrhosis, pulmonary embolism, Barrett's Esophagus, and 40 pack year smoking presented to the VA ED with one month of progressive dysphagia and regurgitation, one week of hemoptysis, and 30 pounds of weight loss over 6 months. Initial vitals were unremarkable. Exam revealed jaundice and distended epigastric veins. Labs revealed baseline platelets (91), normal LFTs, INR, and albumin with mild tBili elevation (2.1). CT-PE showed new marked circumferential wall thickening of mid-esophagus with proximal esophageal distension and compression of both mainstem bronchi, descending thoracic aorta, and left atrium. Bilateral noncalcified pulmonary nodules and associated enlarged mediastinal lymph nodes were also seen. Liver ultrasound showed cirrhotic changes and a new 1.9 cm indeterminant right hepatic lobe hypoechoic lesion with benign enhancement on CT triple phase and normal AFP. EGD scope was unable to pass the mid-esophagus due to structuring. Biopsy revealed poorly differentiated squamous cell carcinoma. A PET scan showed a 9.4 cm lesion in mid to distal esophagus representing a malignant tumor with mediastinal, abdominal, and pulmonary nodal metastases, as well as vertebral, iliac, and soft tissue metastases to right forearm, left lower chest wall, and left upper thigh. A gastrogafrin study was negative for TEF. Bronchoscopy showed mucosal tumor invasion with 20% luminal narrowing and evidence of recent bleeding in left mainstem bronchus. A palliative esophageal stent was placed. He received 8 palliative radiation treatments and passed away 3 weeks after presentation.

Discussion: This case encouraged us to create unifying differential diagnoses for problems involving multiple organs with seemingly different etiologies. This patient had risk factors for GI and pulmonary pathologies (smoking, alcohol use, PE, Barrett's). Initially a thorough differential for dysphagia, regurgitation, and hemoptysis was developed. A unifying diagnosis was realized more quickly by choosing a test in initial survey to help elucidate both hemoptysis and dysphagia. This case highlights need for recognition of aggressive and uncommon distant metastases in ESCC and their associated symptoms. ESCC typically presents late-stage with variable, discordantly minimal symptoms. The patient presented as stage IV with uncommon soft tissue and thoracic metastases (6% and 21% of ESCC mets, respectively) after only one month of symptoms and no evidence of ESCC on CT chest one year prior. His death was likely due to bronchial hemorrhage following radiation.

58) UNCOMMON PRESENTATION OF AN UNCOMMON COMPLICATION OF GRANULOMATOSIS WITH POLYANGIITIS

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Introduction: Granulomatosis with polyangiitis (GPA) is one of the anti-neutrophil cytoplasmic autoantibody (ANCA)-associated vasculitidies (AAV). GPA is a necrotizing vasculitis principally affecting small to medium-sized vessels. Typically, this disease involves many organ systems. The lungs are one of the more common systems damaged in GPA. Diffuse alveolar hemorrhage (DAH) is a possible complication of GPA involvement of the lungs. DAH is only seen in 5-10% of patients with GPA. Furthermore, only about one third of these patients do not have hemoptysis on presentation.

Case: A 67 year old male with a past medical history significant for atrial fibrillation (not on anticoagulation), Raynaud's phenomenon, and recently diagnosed GPA presented to an outside hospital (OSH) with one day of persistent lightheadedness. He also reported accompanying hoarseness, the sensation of "a tickle in my throat", wheezing, fatigue, and generalized weakness. He denied hemoptysis, shortness of breath, chest pain, nausea, vomiting, and trauma. At the OSH he was found to have a fever, leukocytosis, hemoglobin of 7.7g/dL, perihilar lung opacification on chest x-ray, and a urinalysis with 76-100 red blood cells. The patient was treated with levofloxacin, linezolid, IV fluids, and was given 2 units of packed red blood cells. He was then transferred to the medical ICU at Froedtert for further care. Repeat imaging showed worsening perihilar lung opacification and a bronchoscopy confirmed DAH. The patient was treated with plasmapheresis and high dose steroids for a presumed GPA flare-up. After a couple of days in the medical ICU, the patient was stable and transferred to a medicine team on the hospital floor. There, he received one treatment of rituximab, continued to undergo plasmapheresis, and transitioned to a lower dose of steroids. After finishing five courses of plasmapheresis, the patient's hemoglobin was stabilized, he was asymptomatic, and was subsequently discharged.

Discussion: Here we report a case of DAH in the setting of a GPA flare-up and the absence of hemoptysis. This patient received the diagnosis of GPA just a couple weeks before presenting with DAH, and was being treated with high dose steroids and rituximab at the time of presentation. Rituximab was chosen because the patient is PR3-ANCA positive. There is a growing recognition that ANCA type (PR3-ANCA or MPO-ANCA) has more prognostic and clinical importance than the type of AAV the patient has (microscopic polyangiitis versus GPA).

59) AN INTERESTING CASE OF ABDOMINAL PAIN

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A 65 year old male with a past medical history of paroxysmal atrial fibrillation/atrial flutter, coronary artery disease, and diverticulosis presented to the emergency department with a complaint of new onset abdominal pain. He reported the pain had begun two days prior to presentation, describing it as sharp and constant, located in the left lower quadrant and flank without radiation. He reported nausea and multiple episodes of non-bilious, non-bloody emesis since the onset of the pain. He denied hematuria, melena, or hematochezia. He reported that he had not taken any medications, including warfarin, for 2 days prior to admission due to feeling poorly, but reported that he was previously taking all medications as prescribed. On presentation, his blood pressure was elevated to greater than 200 systolic. Exam was significant for irregular heart rhythm with regular rate and intermittent runs of tachycardia. His left lower quadrant was tender to palpation, with no guarding or rebound tenderness. Labs drawn at the time of presentation were significant for a creatinine of 2.05 with a previous baseline of 1.3, lactate of 3.3, white blood cell count of 14.3 with neutrophil predominance and a subtherapeutic INR of 1.45. A urinalysis showed moderate blood. Initially, a non-contrast CT was obtained due to his acute kidney injury, which did not show any acute intra-abdominal process. After fluid resuscitation, due to continued high suspicion for a serious abdominal pathology, a CT angiogram was completed, which showed a left renal artery embolus causing near complete left renal infarct. He was admitted for further care. Due to the time course of his symptoms, he was not a candidate for any surgical revascularization and was medically managed. He was started on a heparin drip with a subsequent bridge to warfarin. His creatinine steadily improved throughout his admission.

A renal artery embolus is the result the occlusion of arterial blood flow to renal parenchyma from a distant location. Common symptoms of a renal artery embolus include acute onset of abdominal or flank pain, often associated with fever, vomiting and acute elevation in blood pressure. Patients most often are older and have a history of atrial fibrillation, though valvular disease, cardiomyopathy and ischemic heart disease are possible predisposing conditions as well. This case illustrates that paroxysmal atrial fibrillation without therapeutic anticoagulation can lead to emboli that may travel to distant locations that are less commonly recognized, such as the renal artery. While renal artery embolus is an uncommon diagnosis, with a concerning history and physical exam, a high index of suspicion should be present. Appropriate imaging should be obtained to identify the correct diagnosis. Treatment involves anticoagulation with consideration of thrombolysis.

60) HEADACHE: A RARE PRESENTATION OF MILLER FISHER SYNDROME

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Introduction: Miller Fisher is a known variant of Guillain Barre Syndrome with the triad of ophthalmoplegia, areflexia and ataxia. This case will discuss a rare presentation with a severe headache.

Case Presentation: A 51 year old male initially presented with blurry vision and a severe occipital headache with no papilledema. Patient endorsed having an upper respiratory-like illness 1-2 weeks prior to symptoms onset. Initial head CT was negative. The lumbar puncture was performed, and noted to have an opening pressure of 320 mmHg. Other values were normal. Serum studies were positive for GQ1B antibody. This gave the diagnosis of Miller Fisher Syndrome. IVIG treatment was initiated and symptoms largely improved, including the headache. Severe headache is usually not a symptom of Miller Fisher Syndrome. In a case series including 27 patients with Miller Fisher Syndrome, only two reported a headache. One study proposed that an increase in CSF protein could lead to outflow obstruction, increasing ICP and leading to headaches. However our patient's CSF protein was 45 mg/dl. Another potential explanation could be injury to the ventral and dorsal roots of cranial nerves by antibodies to GD3 and GD1b. A different study discovered that along with antibodies to GQ1b, in rare cases patients also develop antibodies to GD3 and GD1b. With such a small percentage of patients being positive for the antibodies to GD3 and GD1b, it was theorized that this could be the reason for the headache. Unfortunately, in our case, both GD1b and GD3 antibodies were negative.

Discussion: Headaches are a rare symptom of Miller Fisher with unknown pathophysiology. More research is needed, especially in antibody related nerve damage. Nonetheless, physicians need to identify this symptom and know headaches are usually self-limiting.

61) DECORTICATE POSTURING SECONDARY TO HEPATIC ENCEPHALOPATHY: A CASE REPORT AND REVIEW OF THE LITERATURE.

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Hepatic encephalopathy is a state of brain dysfunction due to liver impairment or portosystemic shunting. Signs of hepatic encephalopathy range from decreased short-term memory to coma in the severe acute setting. Only a few case reports exist demonstrating the rare presentation of decorticate or decerebrate posturing with hepatic encephalopathy. The pathophysiology of such reversible posturing is unknown, but is associated with abnormal respiratory patterns and poor prognosis.

The following case is of a 31 year old woman with Child-Pugh B liver disease secondary to Hepatitis C virus and chronic alcoholism who presented with decorticate posturing due to decompensated acute hepatic encephalopathy 1 day post-EGD with variceal banding. The patient was treated for hepatic encephalopathy with complete resolution of neurologic findings. Unfortunately, she decompensated to end-stage liver disease with a poor prognosis barring liver transplant.

The clinical and pathological findings of this case are presented and the limited literature on this subject is further discussed.

62) IDIOPATHIC HYPEREOSINOPHILIC SYNDROME PRESENTING AS GASTROINTESTINAL SYMPTOMATOLOGY

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Introduction: Hypereosinophilic syndrome (HES) is a rare condition that occurs when overproduction of eosinophils leads to tissue infiltration and possible organ damage in the absence of secondary causes. Several organ systems can be affected, resulting in unpredictable presentations and challenging diagnoses.

Case Description: A 34-year-old female presented to a GI clinic with a 1-week history of diarrhea, nausea and RUQ abdominal pain. A RUQ ultrasound showed gallbladder wall thickening but no signs of dilatation or gallstones. Labs were significant for elevated LFTs of AST 141, ALT 235, and Alk Phos 256, and a WBC of 16.8 with an absolute eosinophil count (AEC) of 3.5. A peripheral blood smear confirmed the presence of leukocytosis with eosinophilia. A HIDA scan and MRCP were performed, which showed a diffusely thickened gallbladder with normal function. Three days after her initial presentation, her LFTs had risen to AST 312, ALT 483 and Alk Phos 386, her AEC had increased to 7.9, and her INR was 2.3, necessitating direct admission to the hospital. A transvenous liver biopsy was performed with concern for acute liver failure, which revealed prominent eosinophilic inflammation, raising suspicions for HES. Hematologic etiologies were ruled out with flow cytometry, FISH and a peripheral smear, while autoimmune etiologies were deemed unlikely with a negative ANCA and no signs of vasculitides. Infectious causes were not likely with negative O&P tests and negative strongyloides IgG. She was started on a 60mg daily course of oral prednisone and responded drastically. Her AEC dropped to 0.28 after one dose and her LFTs began to trend downward after subsequent doses.

Discussion: This case shows an example of idiopathic HES with gastrointestinal involvement, one of many presentations in which this disorder can appear. Because idiopathic HES is a diagnosis of exclusion that can present with such variable symptoms, a comprehensive, systems-based evaluation should be performed to provide a timely and accurate diagnosis in symptomatic patients with eosinophilia.

63) INFECTIVE ENDOCARDITIS IN SETTING OF DISSEMINATED SALMONELLOSIS

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Abstract: Salmonella bacteremia can affect any organ in the body. Blood cultures should be drawn in patients with Salmonella gastroenteritis sick enough to require hospitalization.

Endocarditis is a serious complication of Salmonellosis that is not usually suspected in setting of gastrointestinal disease. Because of rarity such cases are infrequently reported. Thus there is limited data on clinical presentation, progression and optimal management of Salmonella endocarditis. This case report depicts a 63 years old Caucasian female who originally came with Salmonella gastroenteritis and bacteremia complicated by splenic abscesses. She was found to have Endocarditis. Medical management was pursued with 4 week course of IV Antibiotic therapy.

64) THINK AGAIN: LISTERIA MONOCYTOGENES MENINGITIS IN A PATIENT WITH SYSTEMIC LUPUS ERYTHEMATOSUS

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Introduction: Infections are a major cause of morbidity and mortality in patients with systemic lupus erythematosus (SLE). An increased susceptibility to infections exists in SLE patients, either related to immunosuppressive therapies or from disease-related immunological dysfunction. Central nervous system (CNS) infections in patients with SLE are rare, affecting approximately 1.4% of patients; however, a death rate of higher than 40% has been reported in this patient group.

Case Description: A 68-year-old female with history of SLE, ischemic cardiomyopathy, and chronic diarrhea presented with acute worsening of diarrhea and rectal pain over 3 days. She had been hospitalized the prior month for new choreiform movements and dysarthria believed to be secondary to CNS involvement of SLE; at the time, she had been taking prednisone 40 mg daily and mycophenolate 750 mg twice daily for immunosuppression. Notable labs included leukocytosis 11.9 K/mcL, sodium 128 mmol/L, bicarbonate 18 mmol/L, glucose 273 mg/dL, BUN 30 mg/dL, and creatinine 1.25 mg/dL. Patient developed altered mentation with high fevers shortly after admission and was emergently intubated, then transferred to ICU; CT head was negative and lumbar puncture (LP) was performed. Empiric IV cefepime, vancomycin, ampicillin, acyclovir and oral vancomycin were started. Cerebrospinal fluid (CSF) culture and rapid meningitis panel grew *Listeria monocytogenes*, as did initial blood cultures. Repeat blood and CSF cultures were negative, and she completed antibiotic treatment for 3 weeks. Patient clinically improved and was transitioned back to her home from skilled nursing facility.

Discussion: Due to their similar manifestations, CNS infections and neuropsychiatric systemic lupus erythematosus (NPSLE) may be difficult to distinguish. The most common pathogens involved in CNS infections in SLE patients include *M. tuberculosis*, *C. neoformans*, and *L. monocytogenes*. A transient gastroenteritis may precede Listeria meningitis, providing an important clue for accurate diagnosis and therapy. In order to mitigate high mortality, clinicians must have clinical suspicion for meningitis and respond with early diagnosis and treatment.

65) NITROUS OXIDE ABUSE PRESENTING AS FUNCTIONAL VITAMIN B12 DEFICIENCY

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Introduction: Nitrous oxide is used in anesthesia, dental and obstetrical practices. The gas is also inhaled recreationally because of its ability to induce euphoria and reduce anxiety. There have been several reports of disabling neurological consequences due to prolonged nitrous oxide use, resulting in functional inactivation of vitamin B12. Common neurological presentations of nitrous oxide toxicity include paresthesias and gait disturbance.

Case Description: A 24-year-old female presented with 4 days of ascending lower extremity weakness and paresthesias in a stocking-glove distribution. Symptoms began in bilateral feet and progressed to the trunk, and she sustained a fall at home secondary to gait instability as well as leg weakness. Patient reported huffing nitrous oxide from "whippet" canisters every other weekend during the previous 9 months. On physical exam she was noted to have instability in gait, symmetrical loss of flexor and extensor strength in lower extremities, absent ankle reflexes and absent proprioception in the toes. CT head was negative and labs were mostly unremarkable except for WBC count 11.9 K/mcL (4.2-11) and magnesium level 1.6 md/dL (1.7-2.4). Neurology recommended high dose methylprednisolone for suspicion of transverse myelitis. MRI of brain and cervical/thoracic spine showed no abnormalities and patient underwent lumbar puncture with negative CSF culture, cell count, MS panel, and meningitis/encephalitis panel. Subsequent lab studies were notable for negative ANA, serum vitamin D 21.2 ng/mL (30-100), vitamin B12 242 pg/mL (211-911), and methylmalonic acid 25,626 nmol/L (79-376). Treatment with high dose intramuscular cyanocobalamin injections was initiated, and her ambulation gradually improved over the next several days.

Discussion: Patients with nitrous oxide-induced neurological dysfunction may have normal vitamin B12 levels. In these situations, a functional deficiency can be diagnosed by measuring methlymalonic acid and homocysteine (substrates of reactions catalyzed by vitamin B12). Nitrous oxide is believed to inactivate and deplete vitamin B12, and high dose replacement has been shown to improve neurological symptoms.

66) ALMOST BLEEDING TO DEATH FROM CUT K2

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Introduction: There has been a major increase in synthetic marijuana use in recent times as it considered a "safe" alternative to cannabinoid use. Unfortunately, as the composition is unknown, so are the side effects. We are presenting this case to bring awareness for one of the many life threatening side effects of synthetic marijuana use.

Case Description: Our patient is a 41 year old female presenting with hematuria, menorrhagia, and spontaneous bruising. She endorsed use of 'K2' synthetic marijuana (SM) four days prior to onset of symptoms. She denied any medications or history of coagulopathy. Initial labs revealed hemoglobin 4.2 g/dL (baseline: 10 g/dL), PTT: 175 seconds (25-35 seconds), and undetectable INR. CT abdomen did not show hemorrhage. With recent K2 use and signs and symptoms consistent with coagulopathy, our concern was for contaminated K2. Anticoagulant poisoning panel was positive for brodifacoum. She was treated with FFP, PRBCs, oral and IV vitamin K. INR improved to 1.7, PTT 45. Patient was discharged on Vitamin K 10 mg three times daily.

Discussion: The incidence of SM contamination leading to life threatening coagulopathy has only recently been identified. Little is known regarding the composition of SM or mechanism leading to coagulopathy. However, brodifacoum has been implicated as the culprit. This compound acts by inhibiting vitamin K epoxide reductase and is 100x more potent than warfarin. Half-ife is between 20-150 days and symptoms can present up to three months after use. Presenting symptoms can include epistaxis, melena, menorrhagia, and spontaneous intracranial hemorrhage. Labs will reveal elevated INR, prolonged PTT, and acute anemia. An anticoagulant poisoning panel is necessary to confirm presence of toxins. Treatment includes FFP or prothrombin complex concentrate with vitamin K. Duration of therapy is undetermined, but may be more than 6 months of oral vitamin K.

Conclusion: Maintain a high index of suspicion for brodifacoum poisoning with new onset coagulopathy. Treatment include FFP, repeated, high dose vitamin K supplementation while simultaneously contacting poison control. Patient education is of upmost importance.

67) PANCYTOPENIA IN PATIENT WITH AUTOIMMUNE POLYGLANDULAR SYNDROME TYPE 1

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Background: Autoimmune Polyglandular Syndrome Type 1 (APS1) is a rare autoimmune polyendocrine syndrome defined as having at least two of three conditions in a person with mutations in the autoimmune regular (AIRE) gene: chronic mucocutaneous candidiasis, hypoparathyroidism, and adrenocortical insufficiency. Treatment of APS1 is directed at supplementation of disease specific hormones.

Case: A 23-year-old woman PMH significant for APS1 presented with two week hx of night sweats, fevers, generalized fatigue, and weight loss. Two days prior to admission the patient had returned from Belgium where she had been living for 10 months. Patient denied sick contacts but reported extensive travel throughout Europe working with refugees in Romania, Slovakia, and Bulgaria. On admission temperature was 102.5 F, BP 89/50, HR 111, and labs were concerning for pancytopenia (WBC 1.7, H/H 9.4/29, platelets 36), transaminitis, and elevated ESR and CRP. The patient did not have a rash and denied arthralgias or myalgias. Broad-spectrum antibiotics and stress dose steroids were started. Endocrine, ID, rheumatology, hepatology, and hematology were consulted given concern for neutropenic fever, autoimmune hepatitis flare vs. drug induced hepatitis, and viral myelosuppresion. Infectious workup including parvovirus, CMV, EBV, and adenovirus was negative. Rheumatologic workup with ANA screen was also negative. Hepatology recommended stopping 6MP and MMF given concern that they were contributing to her leukopenia. Hematology performed bone marrow biopsy that showed hemophagocytosis. Patient met the criteria for Hemophagocytic Lymphohistiocytosis (HLH) given bone marrow findings, fever, splenomegaly, cytopenia, and ferritin > 500. Dexamethasone was started per HLH protocol. Patient's fevers resolved and her pancytopenia improved. The day before discharge her brucella IgM returned positive, IgG negative. ID said given that the patient improved without appropriate brucella treatment, the positive brucella IgM was likely a false positive. The underlying cause of the HLH was never definitively confirmed but thought to be secondary to an infectious etiology over drug induced (6MP) or autoimmune (APS1).

Conclusion: APS1 is a rare disorder in which immune cell dysfunction results in multiple autoimmunities and predisposes patients to recurrent infections. In a patient with APS1 presenting with neutropenic fever, the differential must be broad and encompass infectious, autoimmune, rheumatologic, genetic, hematologic, and drug related pathologies.

68) IMPELLA 5 AS A BRIDGE TO CLINICAL DECISION MAKING

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Introduction: Mortality from cardiogenic shock remains a significant clinical challenge. Short term mechanical circulatory support devices have the potential to improve outcomes. The Impella 5.0 is currently FDA approved for up to 10 days. We report our institutional experience with the Impella 5.0 as a means to improve survival and expand treatment options in patients presenting with severely decompensated end stage heart failure (HF) and cardiogenic shock.

Methods: A retrospective review was performed on all consecutive patients supported with Impella 5.0 from August 2017 to February 2018 at Froedtert and the Medical College of Wisconsin.

Results: A total of 19 Impella 5.0 devices were implanted in men and women as a "bridge to decision" regarding the potential for recovery vs. evaluation for long-term ventricular assist device (VAD). Eleven patients presented in cardiogenic shock and 8 in severely decompensated HF. All implantations were performed via axillary approach. The average number of pressors was 0.9 (range 0-3 pressors). The average norepinephrine dose was 0.15 mcg (range 0.04 – 0.33 mcg) and epinephrine 0.10 mcg (range 0.06 - 0.15 mcg). After Impella implantation, patients were on pressors for an average of 1.4 days (range 0 – 4 days). The average length of Impella duration was 14 days (range 0 – 36 days). There were no major adverse complications from prolonged Impella support. No devices malfunctioned and there were no reported strokes or major bleeding events. Two (11%) patients had hemolysis requiring device removal on days 6 and 18 respectively. Thrombocytopenia requiring more than 10 units of platelets occurred in 2 patients (11%). Seven patients (37%) died and twelve (63%) survived. Of those that survived seven (37%) regained their ejection fraction (EF) and five (26%) had a VAD placed. Of the seven (37%) that died, four were not VAD candidates (for psychosocial reasons) so decided on hospice/withdrawal of care and the other three died of worsening HF and rupture of a chronic abdominal aortic aneurysm.

Conclusions: Prolonged hemodynamic support with Impella 5.0 is a safe and viable option for patients presenting with decompensated HF and cardiogenic shock as a bridge to decision strategy. Use of Impella support for more than 10 days was safe and did not result in major adverse events. This type of strategy allows for thorough evaluation for viable VAD candidates and offers potential for cardiac recovery in a sub-group of patients.

69) A RARE PRESENTATION OF NECROTIZING COMMUNITY-ACQUIRED PNEUMONIA

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Introduction: *Pseudomonas aeruginosa* is a rare but potentially rapidly fatal etiology of necrotizing pneumonia. This case report aims to increase awareness of *Pseudomonas* as a causative agent of right upper lobe cavitary lesions in those with a history of smoking, alcohol use, and obstructive lung disease.

Case: A 62-year-old male with a history of asthma, CAD, PE, alcohol and tobacco abuse, CKD, T2DM, and HTN, presented with a three-week history of productive cough, dyspnea on exertion, and chest pressure. He complained of frequent white sputum with occasional flecks of blood. He denied fevers, but noted fatigue, night sweats, and a 15-20lb unintentional weight loss over the past nine months. On presentation, vital signs were notable for afebrile, normal heart rate, oxygen saturations of 93-100% on room air, and poorly controlled HTN. He was not in respiratory distress and his lung fields were clear to auscultation with egophony in the RUL. He had a normal leukocyte count (10.5 x 103/mL). A chest X-ray and CT PE (obtained due to history of prior PE) revealed consolidation in the right upper lobe with a large apical cavitary lesion, not present on prior imaging two months ago during a pre-operative assessment for penile prosthesis revision. Cardiac enzymes and hepatic function tests were unremarkable. HIV, bacterial, and fungal tests were all negative. Three separate sputum samples were collected, which showed gram negative rods, few to moderate neutrophils, and rare epithelial cells. Sputum samples were also negative for acid-fast bacilli. He was empirically started on ceftriaxone, clindamycin, and vancomycin in the ED and continued ampicillin/sulbactum after admission. Pulmonology and infectious disease were consulted. Two days later, sputum cultures came back positive for *Pseudomonas aeruginosa*. Therapy was switched to IV pipercillin/tazobactum and ciprofloxacin and narrowed to oral ciprofloxacin when sensitivities were returned. The patient continued to improve clinically and was discharged on day seven with five additional weeks of oral antibiotics.

Discussion: Community-acquired pseudomonas lung infection that progresses to necrotizing pneumonia is a rare presentation with only 20 documented case reports since 1966. On review of the literature, the majority of the cases reported a cavitary lesion in the RUL as was present in this patient. Common risk factors included a history of smoking, alcohol use, and obstructive lung disease. Unlike other causes of community-acquired pneumonia, the course can be insidious and can progress to respiratory failure and even death despite prolonged antibiotic treatment. Workup initially centered around identifying common causes of cavitary pulmonary lesions, including *M. tuberculosis, S. aureus, S. pneumo, Klebsiella*, and fungal infections. While these common causes usually come to mind when presented with an upper lobe cavitary lesion, physicians should also consider *Pseudomonas aeruginosa* in their differential.

70) UNIQUE CAE OF COCAINE INDUCED TMA AND RENAL INFARCTION MIMICKING ATYPICAL HUS

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Introduction: Cocaine use has known association with fatal medical complications, including myocardial ischemia, cerebrovascular accident, and kidney injury secondary to ischemia, rhabdomyolisis, and malignant hypertension. However, cocaine induced Thrombotic microangiopathy (TMA) and renal infarction is a rare entity that has not been described in the literature except in few case reports.

Case Presentation: A 24-year-old female with a past medical history of alcohol and cocaine abuse was admitted with alcoholic induced acute pancreatitis and acute anuric renal injury. She developed microangiopathic hemolytic anemia, thrombocytopenia, and neurologic symptoms including headache and blurry vision which were concerning for TMA versus atypical hemolytic uremic syndrome (HUS). Her labs showed creatinine 7 mg/dL with a baseline of 0.7–0.9 mg/Dl, thrombocytopenia (PLT 40), schistocytes on peripheral smear, a drop in hemoglobin (from 9 to 5.6 g/Dl), with elevated total bilirubin 3.2 g/dL and LDH 3400. She was started on plasma exchange and IV mehtylprednisone for presumptive diagnosis of Thrombotic thrombocytopenic purpura (TTP). ADAMTS13 level was 86 which is normal, therefore TTP was ruled out. Complement level was low shifting the diagnosis toward atypical HUS and the patient was started on Eculizumab. Further workup include immunologic work-up including ANA, CCP, RF, CRP, C3/C4, myeloperoxidase AB, and proteinase-3 AB which were all unremarkable. Lupus anticoagulant was positive. Finally the patient underwent kidney biopsy once her platelet count improved and the final report showed evidence of ATN, ischemic necrosis, focal interstitial hemorrhage, and infarcts most likely due to cocaine induced TMA.

Conclusion: Cocaine induced renal TMA should be suspected in patients with heavy use of cocaine who presents with a new onset Acute Kidney Injury. There is also emerging evidence that cocaine can activate complements and causes hypo-complementemia seen in our patient.

71) ALTERED MENTAL STATUS CAUSED BY WEST NILE ENCEPHALITIS

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Introduction: Many patients are admitted to the hospital for altered mental status, and the differential diagnosis for such chief complaints is very broad. Therefore, it is important to search widely for potential causes, including rare causes such as West Nile Virus.

Case Description: A 55 year old man with past medical history significant for atrial fibrillation, chronic heart failure, hypertension, type 2 diabetes mellitus, chronic kidney disease, and alcohol, tobacco and illicit drug use presented to the ED with one day history of altered mental status, confusion, hallucinations and incoherent speech. He was discharged from an outside hospital the day before presenting to our ED for alcohol withdrawal, fever, and headache. Vitals upon presentation were significant for temperature of 98.3, blood pressure of 180/126 and pulse of 115. Physical exam showed an alert, but agitated man with bilateral hand tremors, dry mucous membranes, and irregular heart rate. Lab values were significant for a bicarbonate of 20, BUN of 28, creatinine of 2.38, glucose of 246, and albumin of 3.2. Urine toxicology screen was positive for benzodiazepines, and urine analysis showed elevated glucose, ketones, protein, and blood with hyaline casts. CT of the head only showed chronic ischemic microvascular disease, and chest X-ray was unremarkable. ECG showed atrial fibrillation. The patient was treated with supportive measures for the first three days. On the fourth hospital day, the patient was transferred to the ICU for IV anti-hypertensive medications and a lumbar puncture was performed showing RBC of 12, WBC of 14, glucose of 124 and protein of 54. Further investigation showed positive West Nile IgG and IgM in the cerebrospinal fluid. The patient was treated with supportive measures including hydration, anti-emetics, seizure prophylaxis and pain control and was successfully discharged day 14 of hospitalization with improved mental status.

Discussion: This case demonstrates the importance of ruling out multiple diagnoses when presented with a patient with altered mental status. West Nile Virus only presents as neuro-invasive disease in 0.4-0.6% of those infected. Risk factors include diabetes, hypertension, chronic kidney disease, alcohol use and male sex, which were all present in this patient. Although alcohol withdrawal may have been the most likely diagnosis for this patient's altered mental status and may be what the patient initially presented with, it is important to further investigate a patient's differential as there may be other explanations of the patient's chief problem.

72) MONOPLEGIA WITH WEST NILE ENCEPHALITIS

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Introduction: West Nile virus is now the leading cause of nationally acquired arbovirus disease in the US. Manifestations range from asymptomatic, to fevers, to neuroinvasive disease, which can have permanent and devastating complications.

Case: A 65 year old with history of HFrEF, CAD, and Crohn's disease presented with fevers, headache, and one day of progressive right lower extremity weakness, ultimately resulting in flaccid paralysis. His exam was significant for fever to 102.9, neck stiffness, photosensitivity and paralysis of the right lower extremity with hypoactive reflexes. He had no lower extremity clonus or sensory deficits and his upper extremity neurologic exam was normal. Lumbar puncture revealed 107 nucleated cells with 10% neutrophils, elevated protein of 54, and normal glucose of 49. MRI of lumbar spine with contrast showed extensive smooth enhancement of leptomeninges, cauda equina and all lumbar visualized sacral nerve roots. MRA head and MRI of the right lower extremity did not show relevant abnormalities. Ultimately, the patient was found to have both serum and CSF West Nile Virus (WNV) IgM positive. He was first was treated with ceftriaxone, vancomycin, and acyclovir, which were subsequently discontinued with the positive WNV results. He was discharged with physical therapy to an acute rehabilitation facility with slow but gradual improvement of his right lower extremity weakness.

Discussion: Risk factors for neuroinvasive WNV disease include age, malignancy, organ transplant, and some genetic factors, of which our patient only had age. Clinical symptoms of neuroinvasive disease range from meningitis, encephalitis, flaccid paralysis or a mix of these. Paralysis, as seen with our patient, often is due to anterior horn lesions which can present with asymmetric weakness of limbs that rapidly progresses to paralysis, quadriplegia and respiratory failure. Other features that can be seen with WNV that were not present in this patient include hepatitis, pancreatitis, myocarditis, and rhabdomyolysis. Lab findings include CSF with elevated protein, pleocytosis of <500 cells, with lymphocytic predominance, though neutrophils are present in early infections. MRI may show meningeal involvement and instar-spinal abnormalities. EEGs may show generalized, continuous slowing, though this was not completed on our patient. Treatment is often supportive and aimed at rehabilitation with recovery typically occurring within the first six months of diagnosis. One third of patients fully recover, one third partially recover, and one third will show no improvement highlighting the severe complications that can result from this disease.

73) EVAN'S SYNDROME AS THE INITIAL FINDING IN SYSTEMIC LUPUS ERYTHEMATOSUS

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Introduction: Evan's Syndrome is an uncommon condition defined by immune thrombocytopenia (ITP), Coomb's positive autoimmune hemolytic anemia, with or without immune mediated neutropenia, that can be a manifestation of Systemic Lupus Erythematosus (SLE).

Case Presentation: A 24-year-old male with no past medical history presented to the ED for a "bump" in his mouth associated with bleeding and purpuric lesions to bilateral lower extremities, without other symptoms. He was noted to be profoundly thrombocytopenic with PLT of two-thousand and anemic with HGB of 11.9. Further workup revealed hemolysis (LDH 298, bilirubin 2.0, and haptoglobin < 8) with a positive Coombs antibody. Other causes of thrombocytopenia were excluded and he was given IVIG and high dose steroids for ITP with improvement. The patient was discharged with a steroid taper. Following completion of steroid taper he represented with a similar "bump" in his mouth, as well as platelets of one-thousand. He again had Coomb's positive hemolytic anemia and was treated with IVIG and steroids, with good response. At this time, C3 & C4 were reduced and autoimmune panel was sent and identified a homogeneous ANA titer of 1:640, positive double stranded DNA antibody, and positive SSA antibody. He denied any symptoms such has mucosal dryness, rash, Raynaud's, or arthralgia. He was seen in the Rheumatology clinic soon after discharge where his case of Evan's syndrome was felt to be the initial presentation for systemic lupus erythematosus (SLE). He was started on hydroxychloroquine, and is having ongoing discussion regarding the need for additional immunosuppressant agents.

Discussion: Evan's syndrome (ES) is recognized as a special variant of autoimmune hemolytic anemia and can be assocatied with autoimmunie conditions (SLE, ALPS), viral infections (hepatitis and HIV), common variable immune deficiency, malignancy (CLL, Non-Hodgkins Lymphoma), or allogenic stem cell transplant. ES is often resistant to standard treatments of ITP and can follow a chronic course. In relation to it's association with SLE, ES may be the initial presentation of SLE as in this case. First line treatments include high dose steroids, while second line treatments include further immuno-suppressants such as cyclophosphamide, azathioprine, rituximab, and treatment of the underlying disorder.

74) MULTIPLE MYELOMA WITH SOLITARY INTRAPARENCYMAL BRAIN METASTASIS

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BACKGROUND: Multiple myelomas (MM) are progressive and incurable neoplasia of the B cells characterized by monoclonal proliferation of immunoglobulin (Ig)-secreting plasma cells. Central nervous system (CNS) involvement is a rare complication of MM, and it can present as either an intraparenchymal or a leptomeningeal lesion. Nieuwenhuizen et al mention 109 cases of CNS multiple myeloma reported in the literature. Most of the tumors in the CNS are either skull or dural based, but MM based solely in the parenchymal sites are extremely rare.

CASE DESCRIPTION: A 68 year old female was diagnosed with IgG lambda plasma cell myeloma in September 2013. She was treated with Bortezomib, lenalidomide and dexamethasone. She eventually received autologous stem cell transplant in May 2014. She was doing well until her recent follow up with her oncologist in May 2018 when she complained of expressive aphasia. She was also having trouble ambulating. Her oncologist ordered a head CT which showed a left cerebral mass with extensive vasogenic edema. CT chest, abdomen and pelvis were unremarkable for any primary lesion. Patient was started on dexamethasone. She was then referred to our hospital. The admitting hospitalist ordered a MRI which showed a solitary enhancing mass in the left frontal lobe with extensive vasogenic edema and a midline shift of approximately 6mm. Patient was continued on dexamethasone which was started by her primary oncologist. She was seen by neurosurgery team. Patient underwent stereotactic brain biopsy on June 4, 2018. Pathology of the brain mass showed atypical plasma cell infiltrate consistent with metastasis of patient's previously diagnosed myeloma. She was positive for CD138 and Lambda and negative for Kappa. Our oncologist recommended PET scan and bone marrow aspiration and biopsy for staging. Both the test were negative. After discussion with neurooncologist and patient's primary oncologist, our oncologist decided to treat the solitary brain metastasis with radiation therapy. Patient was seen by her primary oncologist before starting the radiation therapy and she had significant improvement in her speech and gait with dexamethasone.

DISCUSSION: Myelomatous involvement of the CNS is an uncommon complication that portends a poor prognosis in patients with multiple myeloma. The risk of involvement of the central nervous system (CNS) is estimated to be approximately 1% in patients with established MM with overall survival (OS) reportedly < 6 months from the detection of CNS disease. Most of the chemotherapy drugs used for MM do not cross blood brain barrier. Brain radiation appears to be transiently effective. The optimal management of this challenging subset of MM remains unclear, because all approaches studied thus far have yielded unsatisfactory results.

75) MYCOPLASMA MUCOSITIS

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Mycoplasma pneumoniae is one of three Mycomplasma species known to commonly cause infection in humans. Respiratory symptoms are often the most common presentation, however patients may often present with extrapulmonary symptoms such as mucositis, skin rash, hemolysis, and joint involvement. When Mycoplasma infection is suspected, mycoplasma serology and NAAT is used for diagnosis and treatment can include antibiotics, systemic steroids, and occasionally IV immunoglobulin depending on the severity of symptoms.

Case Description: The patient is a 25 yo African American M who presented with chief complaint of oral, scrotal, and ocular ulcers. The patient began having symptoms of congestion and cough followed by the development of oral and ocular mucosal erosions. The symptoms eventually progressed to involve lesions to the skin of his trunk and extremities. Due to the pulmonary symptoms, the patient was initially thought to have pneumonia and was placed on antibiotic treatment which provided no resolution of symptoms. Rhematology was consulted due to suspicion of Behcet's and treatment was initiated. Still symptoms persisted. Eventually Dermatology was consulted and believed the patient to have mycoplasma-induced rash and mucositis. The patient was subsequently tested for mycoplasma serologies and initiated on empiric doxycycline treatment with continuation of IV steroids and cochicine treatment for suspected Behect's. The following day Mycoplasma IgM antibody came back positive which was confirmed by positive NAAT a few days later. The patient showed improvement of symptoms and was discharged on a 7 day course of doxycycline and 5 day course of oral steroids.

Discussion:

Here we present a case of Mycoplasma-induced mucositis in a 25 Y M. Mycoplasma pneumoniae most commonly infects school-aged children, military recruits, and college students. Patients who present with common respiratory symptoms are often are diagnosed with a viral upper respiratory tract infection. If a patient of this demographic presents with these symptoms and any additional extrapulmonary symptoms, Mycoplasma infection should be also be considered. Proper screening with mycoplasma serology should be done to determine the etiology of symptoms and guide appropriate treatment.

76) CRYPTOCOCCAL MENINGITIS

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Introduction: Cryptococcal meningitis is an opportunistic infection of the meninges. Dependent on the infectious syndrome, symptoms include, fever, fatigue, dry cough, headache, blurred vision, and confusion. Detection of cryptococcal antigen by culture of CSF, sputum, and urine provides definitive diagnosis.

Case Description: 58 yo M with no significant past medical history presented for evaluation of 2 weeks of frontal headache, intermittent nausea and vomiting, 20 lb weight loss over the past 2 months, and abdominal pain. In the ED, the patient was afebrile and CT of the abdomen, pelvis, and head were all unremarkable, however the decision for admission was made. The next day the patient developed neck stiffness and continued headache. A lumbar puncture was performed which showed an opening pressure of 23 mmHg. CSF came back positive for Cryptococcal Antigen with elevated CSF Protein at 108, RBC at 5531, WBC at 92, and low CSF glucose at 13. Cryptococcus Neoformans NAAT was positive and CSF cultures also came back positive. A 14 day induction course of Lipisomal Amphotericin B and Flucytosine TID were started with repeat LP and CSF fungal cultures planned upon completion of treatment course.

In addition to the recent diagnosis of Cryptococcal Meningitis, the patient was also discovered to have a high HIV RNA viral load and a CD4 count of 63, consistent with diagnosis of HIV. The patient received prophylactic antibiotics however due to the comorbid meningitis, ART was contraindicated at the time but planned for after clearance of the Cryptococcal infection. The patient remained in the hospital for treatment of cryptococcal meningitis until repeat LP showed normal opening pressure and CSF analysis. The patient was sent home on high dose fluconazole treatment for consolidation therapy with plan for maintenance low dose fluconazole therapy to follow.

Discussion: Here we present a case of Cryptococcal meningitis in a 58 Y M presenting with a 2 week history of new-onset headaches. Cryptococcal meningitis is considered an opportunistic infection that is commonly associated with untreated AIDS or undiagnosed HIV. When Cryptococcal meningitis is diagnosed in an otherwise healthy patient, undiagnosed HIV should be strongly considered.

77) DRUG INDUCED EOSINOPHILIC PNEUMONIA RELATED TO INTRAVENOUS DAPTOMYCIN THERAPY

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A 46-year-old man with a recent diagnosis of pyomyositis of the right deltoid and trapezius muscles with septic arthritis and osteomyelitis of the right acromioclavicular joint started on outpatient intravenous Daptomycin therapy re-presented to the emergency room with two days of dry cough, fevers to 101 F, night sweats, and chest tightness.

Inflammatory markers on this presentation increased from discharge of initial diagnosis: CRP 8.9 from 2.9 and ESR 47 from 24. Additionally, serum eosinophils were elevated to 610 which was marked from normal serum eosinophils at time of discharge for initial diagnosis. The white blood cell count was elevated, and the patient was tachycardic and febrile on admission so empiric therapy for hospital-acquired pneumonia was initiated. Daptomycin was discontinued given concern for drug induced lung injury. A CT scan on hospital day two revealed patchy ground glass and consolidative opacities involving the bilateral lower lobes and lung apices. A bronchoscopy was performed with broncho-alveolar lavage, which revealed 10% eosinophilia on cell count. The patient's dry cough and night sweats were slowly improving and fevers proceeded to defervesce in the hospital. BAL fungal and bacterial studies were negative. Given the patient's active infection, and symptom improvement with discontinuation of Daptomycin; the decision was made not to initiate steroid therapy.

Daptomycin is a known causative agent of eosinophilic pneumonia. The patient's presentation was consistent given his dry cough, fevers, and night sweats with correlative imaging studies and laboratory findings on serum and BAL. Discontinuation of offending drug, and consideration for glucocorticoid therapy based on severity of symptoms remains the mainstay of treatment.

78) PNEUMOCOCCAL MYCOTIC AORTIC ANEURYSM: AN UNLIKELY CAUSE OF ABDOMINAL PAIN

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Introduction: We present a patient with upper abdominal pain and fever, found to have pneumococcal bacteremia and mycotic aortic aneurysm.

Case Description: An 89-year-old male with a history of Waldenström macroglobulinemia on rituximab and remote gallstone pancreatitis status post cholecystectomy, presented with ten days of right upper quadrant abdominal pain, described as constant, sharp, worse with eating, and accompanied by anorexia and constipation. He denied nausea, vomiting, melena, hematochezia, jaundice, dysuria or hematuria, but reported several months of dry cough. Abdominal exam was unremarkable. He had bibasilar crackles on pulmonary exam. Labs were notable for WBC 18.6 (79% PMNs), normal lipase and liver function tests. Contrast-enhanced CT abdomen showed bibasilar pulmonary parenchymal disease, small bowel ileus, and a small area of localized inflammation around the distal thoracic esophagus and aorta. He then developed fever and was treated with antibiotics for a working diagnosis of community-acquired pneumonia complicated by ileus. Urinary pneumococcal antigen was positive, and blood cultures grew *Streptococcus pneumoniae*. Right upper quadrant ultrasound was unremarkable, and EGD revealed only gastritis, negative for *H. pylori*. Fever and leukocytosis resolved with antibiotics, but abdominal symptoms waxed and waned. On hospital day 11, he developed recurrent fever with worsening epigastric pain. Repeat CT showed a new large (6 cm) hiatal aortic aneurysm. He underwent urgent open vascular surgical resection and repair of a pneumococcal mycotic aortic aneurysm. The patient survived and is doing well two years later.

Discussion: Mycotic aneurysms are challenging to diagnose due to their diversity of manifesting signs and symptoms. *S. pneumoniae* is an uncommon causal pathogen, and positive outcomes are rare, with few case reports in the literature. This patient was at risk for complicated pneumococcal disease given his hematologic history. Diagnosis was delayed due to nonspecific findings on initial imaging and the coexisting diagnosis of pneumonia, although it inadequately explained his presenting symptoms. This diagnosis could be considered in bacteremic patients with abdominal pain in whom other evaluations are non-revealing.

79) A UNIQUE PRESENTATION OF VITAMIN B12 DEFICIENCY AND PANCYTOPENIA

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Pernicious anemia, a condition characterized by autoimmune destruction of parietal cells, is one of the most common causes of Vitamin B12 deficiency worldwide. In this report, we describe the case of a young woman who presented with severe pancytopenia and neurologic findings consistent with vitamin B12 deficiency secondary to pernicious anemia.

A 24-year-old female with a history of iron deficiency anemia presented to the hospital with marked weakness, abdominal pain, and upper extremity numbness and tingling for 1 month. Upon presentation, vital signs were stable except for heart rate of 110. Initial exam was normal except for slowed movements and generalized weakness. Admission labs revealed pancytopenia (WBC 3.1, PLT 114, RBC 2.6), hemoglobin of 6.5 g/dL, LDH of 3889, haptoglobin <10, and elevated total and direct bilirubin concerning for hemolysis. Fecal occult blood test was negative. Splenomegaly was identified on abdominal ultrasound. Extensive work up included peripheral blood smear, DIC panel, Coombs test, Vitamin B12, Folate, gastrin levels, and intrinsic factor and parietal cell antibodies. On day 2, Vitamin B12 injections were initiated, and by day 3 the patient was well enough for discharge home with a plan to continue Vitamin B12 injections and follow up with Hematology. Following discharge, anti-IF antibodies resulted positive, confirming pernicious anemia as the cause of Vitamin B12 deficiency and hemolysis.

Our patient presented with classic hematologic and neurologic findings consistent with pernicious anemia. Her case is interesting given her age and the severity of her symptoms. Her clinical presentation and laboratory results were concerning for hemolysis of unknown etiology. A negative direct Coomb's test made autoimmune hemolytic anemia less likely. Low vitamin B12 with pancytopenia raised suspicion for pernicious anemia, prompting administration of vitamin B12. Improvement of symptoms and cell counts with supplementation and subsequent identification of anti-IF antibodies confirmed a diagnosis of pernicious anemia with hemolysis.

80) MULTIMODAL INTERVENTION APPROACH REDUCES CATHETER ASSOCIATED URINARY TRACT INFECTIONS (CAUTI) IN A RURAL TERTIARY CARE CENTER

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Background: Catheter associated urinary tract infections (CAUTI) is the fourth most common hospital acquired infection. The Center of Disease Control and Prevention (CDC) defines a CAUTI as a urinary tract infection with an indwelling urinary catheter in place for >2 calendar days on the date of event, with day of device placement being Day 1, and an indwelling urinary catheter must have been in place on the date of event or the day before. Decreasing the CAUTI rates and catheter days are a challenge for acute care hospitals to improve the quality of care.

Interventions/methods: Standardized infection rates (SIR) for CAUTI were obtained before and after implantation of our multimodal intervention in 2015 and 2017 respectively. Our intervention included specific measures implemented to prevent CAUTI: (a) physician and nurse education, (b) modification of progress note templates to remind about Foley catheters in place, daily evaluation of the need for Foley, (c) established best practices for eliminating CAUTIs: limiting catheter use for appropriate indications, (d) advocating for alternative toileting options (e.g., urinals, condom catheters, measurement of diaper weight, intermittent catheterization) (e) promoting aseptic techniques for insertion and removal of catheters.

Results: The interventional study was performed in a 504 bed hospital. CAUTI prevention strategies were initiated in 2015 due to concerns over the increase in CAUTI incidence. Prior to implementation of measures, half yearly CAUTI event number was 13, urinary catheter days were 8782, no of infections predicted were 9.504 and SIR was of 1.368. With the effective implementation of prevention measures, the 2017 January through September CAUTI event number was 5, urinary catheter days were 9834, prediction infection numbers were 10.865, SIR was 0.46. CAUTI's SIR was reduced from 1.368 to 0.460, a 66% reduction over two years. Urinary catheter days were reduced from 8782 in the first half of calendar year 2015 to 6377 in the first half of calendar year 2017, a 27% reduction.

Conclusion: Incidence of CAUTI rates were significantly reduced with a team effort involving infection control, physician and nursing education, appropriate usage of urinary catheter, with modest reduction in urinary catheter days.

81) PROFOUND ACIDEMIA SECONDARY TO METFORMIN ASSOCIATED LACTIC ACIDOSIS

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Background: Metformin is a preferred oral hypoglycemic agent for the treatment of type2 Diabetes unless it is contraindicated or intolerant. Metformin is rarely associated with lactic acidosis (type B). The risk of metformin associated lactic acidosis (MALA) increases with abrupt decline in kidney function, underlying heart failure, chronic kidney disease stage IV, concomitant use of NSAIDS, ACE inhibitors and ARB.

Case presentation: 70 year old Caucasian female with Hypertension and Diabetes Mellitus type 2 presented to local hospital with nausea, abdominal pain, vomiting, and diarrhea for 2-3 days. She also noted decreased appetite, poor oral intake, fatigue, malaise. Her medications included glimepiride, valsartan, metformin, insulin, amlodipine. On examination, she was lethargic, alert and oriented with temp- 92.5F, HR-78/min, RR- 20/min, BP- 184/78mm Hg . Her labs revealed lactate- 13.5, potassium- 5.9mmol/L, anion gap – 36mmol/L, venous pH- 6.88, bicarbonate- 5mmol/L, VpCO2- 20mm Hg and creatinine- 11.8mg/dL. She was transferred to our facility for renal replacement therapy. En route, the patient developed agonal respirations, bradycardia and hypotension with nadir pH of 6.5. Supportive management with IV fluids, antibiotics, dopamine, atropine, bicarbonate was given before hospital transfer. Upon arrival, she was unresponsive on mechanical ventilation; pupils were dilated to 5-6mm with temp of 89F, HR- 73/min, BP- 100/50 mm Hg on 7mcg of dopamine. Labs revealed bicarbonate -3mmol/L, arterial pH- 6.65, lactate- 18.8mmol/L, WBC 26.4 x103/uL, creatinine- 9.7mg/dL, potassium 4.6mmol/L, anion gap-38mmol/L. Differentials at the time of presentations were septic shock, metformin associated lactic acidosis, ischemic bowel disease as well as acute renal failure, metabolic encephalopathy and multiorgan failure. CT abdomen and pelvis was normal with no evidence of ischemic bowel. Ejection fraction was normal on ECHO. Infectious workup was negative. She was started on supportive management with continuous renal replacement therapy and after 9 hrs, her neurological status improved and was following commands. On day 2 her metformin levels were- 5.7mcg/ml (therapeutic range 1-2mcg/ml). Following dialysis, she was back to her baseline and lactic acidosis was completely resolved on day 4.

Discussion: Metformin associated lactic acidosis (MALA) should be considered in all diabetes patients on metformin and lactic acidosis. Pathogenesis felt to be metformin accumulation secondary to metformin overdose or decline in renal function causing impaired mitochondrial function and inhibited oxygen consumption leading to increased lactate production. Extracorporeal removal with hemodialysis should be considered in patients with lactate >15mmol/L, pH<7.0, kidney injury, hemodynamic instability, decreased level of consciousness, liver failure.

82) MULTIPLE MYELOMA MASKING OTHER ETIOLOGIES OF DISEASE

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Introduction: Multiple myeloma (MM) can present with a wide range of symptoms. The classic prodrome includes hypercalcemia, renal impairment, anemia, and lytic bone lesions. Classic presentation of a disease state can make diagnosis by pattern recognition easy. However, it is important to maintain a broad differential to avoid anchoring bias.

Case Presentation: A 69 year old Caucasian male with history of chronic lumbago presented with complaints of worsened low back pain and weight loss of 25lbs in the past 1.5 months. Admission laboratory studies were remarkable for hemoglobin of 6.2, mean corpuscular volume 111, reticulocytes 1.78%, calcium of 13.0, and creatinine 2.19 (baseline 0.9). Initial evaluation was concerning for malignant process such as multiple myeloma (MM) based on the presence of anemia, hypercalcemia, kidney injury, and bone pain. Further testing revealed elevated serum protein at 9.6 (reference range 6.4-8.2), total urine protein 86.9 (ref range 0-11.8). PTH was 29.9 (18.4-80.1), and bone scan did not show lytic lesions. Hematology/oncology was consulted and bone marrow biopsy uncovered markedly increased plasma cells diffusely throughout the bone marrow with vast majority being atypical, confirming the diagnosis of MM.

Impact: Our case demonstrates the clinical importance of maintaining a broad differential when approaching cases. Despite our patient's apparent classical presentation, many of his metabolic derangements were of multifactorial etiology.

For example, our patient's hypercalcemia could have been solely attributed to MM, but further analysis showed that PTH was not sufficiently suppressed suggesting primary hyperparathyroidism as an additional etiology. If we had not investigated other causes outside MM, the primary hyperparathyroidism would have been left undiagnosed. The elevated creatinine on admission established renal impairment fitting the symptomology of MM, however MM was not the cause of his renal failure as it was discovered to be a post renal etiology and resolved with intervention. Bone lesions are typically lytic lesions, but it is important to note that our patient presented with long standing compression fractures which can be attributed to MM. In patients that present with apparently clear findings of MM, it would have been easy to make the diagnosis on presentation, However, we followed the protocol for the diagnostic work up of alternate etiologies which revealed contributing pathologies. This case reinforces that protocol guided diagnostic workup, treatment, and management is an essential part of modern day medicine.

83) BARRIERS TO NUTRITIOUS MEALS AMONG SENIORS IN BROWN COUNTY

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INTRODUCTION: Many seniors struggle to receive proper nutrition as they age. Factors such as living alone, declining health, and losing the ability to drive exacerbate this issue. Improper nutrition can increase the risk for serious illnesses. The ADRC of Brown County works to address some of these issues by providing one hot meal per week day to local seniors. Although one hot meal per weekday is tremendously beneficial for our local seniors, proper nutrition requires multiple healthy meals per day.

OBJECTIVE: This study aims to identify unknown barriers to eating multiple healthy meals per day in the senior population of Brown County and educate seniors on how to best receive proper nutrition despite such barriers.

METHODS: To achieve this, surveys were distributed to all ADRC of Brown County meal program participants, 60 years of age or older. The survey identified perceived barriers to healthy eating and compared responses between those seniors who are homebound and those who are not. A follow-up educational pamphlet was distributed.

RESULTS/CONCLUSIONS: The perceived barriers to eating multiple healthy meals per day include the physical inability to grocery shop, lack of appetite, and affordability. Congregate Dining Site Participants most often get their weekend meals from fast food or sit down restaurants. Therefore, although the Congregate Dining Site Participants have the physical ability to leave their homes, it does not result in the consumption of healthier meals on the weekends compared to those who are homebound.

84) RIGHT FLANK PAIN: WHAT'S MORE THAN A STONE AND A BUG?

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Introduction: Renal infarction (RI) is considered as rare condition; however, the incidence is underestimated due to the under diagnosis. Prompt diagnosis is crucial to prevent permanent loss of renal function.

Case Description: This case involved a 61-year-old African American female who presented with sudden onset of severe constant right flank pain and right upper quadrant abdominal pain. Her history was significant for uncontrolled

Type 2 diabetes, untreated hypertension, long standing smoking and intermittent use of marijuana. Patient had a blood pressure of 187/88 mmHg with otherwise normal vital signs upon presentation. She had moderate tenderness to palpation of the right upper quadrant of the abdomen, and significant right costovertebral angle tenderness. Initial lab work was unremarkable and her urine was positive for nitrites and no bacteria. Computer tomography of the abdomen without contrast and renal ultrasound ruled out nephrolithiasis or any other acute pathology of abdomen or pelvis. Patient continued to have severe right flank pain despite frequent use of intravenous narcotics and antibiotics. She had an unexplained creatinine elevation overnight. Repeated lactic acid remained negative. Renal doppler ultrasound revealed total occlusion of the right renal artery. The right kidney was deemed to be non-salvageable as it was out of the window for reperfusion. Extensive workup, including lipid panel, hypercoagulability, and cardiac tests was only remarkable for HDL level of 17 mg/dL, and HbA1c of 7.6. The infarction was considered due to atherosclerosis and patient was started on anticoagulation treatment.

Discussion: This case illustrates renal infarction as the rare cause of right flank pain with negative abdominal CT and renal ultrasound. Keeping the differential diagnosis in mind and knowing the proper radiological modality to perform, would help to recognize this condition promptly. Rapid diagnosis and institution of the appropriate therapy are essential for the prevention of permanent loss of renal function.

85) SUPPARATIVE THROMBOPHLEBITIS OF THE FACIAL VEIN: A VARIANT OF LEMIERRE'S SYNDROME

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Introduction: Lemierre's syndrome refers to infectious thrombophlebitis of the jugular vein most commonly caused by Fusobacterium Necrophorum. The thrombophlebitis is a serious condition that may lead to systemic complications if not treated promptly.

Case Description: This case involved a 33-year-old Hispanic female who presented with a 6-day history of fever, chills, nausea, vomiting and profound diarrhea. She received aggressive hydration and required pressors for persistent hypotension. Patient was started on broad spectrum antibiotics for sepsis of unknown origin. Her gastrointestinal symptoms resolved with above treatment; however, she continued to have fever and significant leukocytosis. Meanwhile, she complained of swelling of her right neck. Patient reported a history of dental abscess on the right side 3 months ago, but denied any current symptoms. Her initial blood cultures obtained in the ED grew Fusobacterium necrophorum. Although she did not have the typical history of pharyngitis, given the suspected submandibular lymph node and blood cultures growing Fusobacterium species, Lemierre's syndrome was suspected and neck/chest/abdomen/pelvis CTs were obtained to investigate the source of infection and possible systemic septic emboli. The CTs indicated purulent thrombophlebitis of the right facial vein and periapical abscess of the right maxillary incisor, where a branch of the right facial vein was draining from. In addition, there were cavitary lesions in the right upper lobe of lung, which were suspicious for septic emboli. It is considered, that she had a variant of Lemierre's syndrome. She was on intravenous metronidazole until her fever resolved and leukocytosis trended down. She continued oral Augmentin and amoxicillin for three weeks.

Discussion: This case illustrates a variant of Lemierre's syndrome that does not have preceding pharyngitis, involves the facial vein, and presents with prominent gastrointestinal symptoms. Recognizing atypical presentations of Lemierre's syndrome is essential as prompt treatment is the key to prevent serious systemic complications.

86) FRIGIDLY COLD: A CLASSIC PRESENTATION

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Introduction: The thyroid is an essential organ of the endocrine system and produces thyroid hormones which regulate multiple physiologic processes. Dysfunction can cause various clinical presentations with diagnosis relying heavily on laboratory testing. Hypothyroidism is a condition in which the thyroid fails to produce adequate amounts of thyroid hormone. This case highlights a severe form of hypothyroidism that is uncommonly encountered.

Case Description: A 64 year old woman with a history of arthritis and hypothyroidism presented to the hospital after two months duration of progressive weakness, excessive fatigue, and increased confusion. Vital signs showed bradycardia (30s), hypotension, and hypothermia with exam positive for altered mental status, garbled speech, dry mucous membranes, bradycardia, cool extremities, and non-pitting bilateral lower extremity edema. Pertinent labs revealed TSH of 127 and free T4 of 0. The family reported she had been unwilling to see a physician, and had not taken her levothyroxine for two years. Due to clinical instability, she was intubated and started on dobutamine infusion for bradycardia. She received a bolus of levothyroxine and hydrocortisone followed by daily intravenous levothyroxine. The patient showed improvement but failed extubation on two separate occasions leading to tracheostomy. One day following tracheostomy, she developed PEA arrest due to hypoxia and later passed away.

Discussion: Myxedema coma is a severe form of hypothyroidism, which is rarely seen due to availability of thyroid function tests. It is a medical emergency with a high mortality rate. Clinical presentation is highly variable but often includes altered mental status and hypothermia. Other findings can include bradycardia, periorbital and perioral swelling, macroglossia, hyporeflexia, and xeroderma. Early identification is paramount for improving outcomes. Patients should be managed in the ICU with attention to ventilation, hypotension, and hypothermia. Aggressive measures should be taken to identify precipitating factors, with early administration of intravenous steroids and thyroid hormones.

87) MASSIVE PERICARDIAL EFFUSION ASSOCIATED WITH HYPOTHYROIDISM

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Cardiovascular findings in hypothyroidism are often subtle. Hypothyroidism often presents as lethargy, cold intolerance, constipation, proximal muscle weakness, weight gain, decreased appetite, coarse dry skin, hair loss and non-pitting edema. Cardiovascular findings are usually mild, and include bradycardia, diastolic hypertension and narrow pulse pressure. Small pericardial effusions are seen in up to 30% of overtly hypothyroid patients. There are only a few case reports of hypothyroidism presenting as massive pericardial effusion. We report a case of 62-year-old female who presented with worsening dyspnea. She was found to have massive pericardial effusion with tamponade effect. Thyroid function analysis at initial presentation revealed raised thyroid-stimulating hormone and decreased thyroxine and triiodothyronine levels. She underwent urgent pericardiocentesis and had a complicated hospital course. Antithyroglobulin and anti-thyroperoxidase antibodies were significantly high. She was initiated on levothyroxine therapy. After extensive workup, negative for rheumatoid arthritis, lupus, infection, amyloidosis and malignancy, her pericardial effusion was attributed to have been caused by previously undiagnosed hashimoto's disease. However, she had no other symptoms and clinical signs suggestive of hypothyroidism. Follow up echocardiogram 4 months later showed complete resolution of the pericardial effusion with no recurrence.

88) A CASE OF THE RUNS: WHIPPLE'S DISEASE

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A 54-year-old male with developmental delay presented to the clinic with shortness of breath that had been progressively worsening for more than a month. He was pale and appeared lethargic. Physical exam was significant for tachycardia and bilateral pedal edema. The only symptom he reported was diarrhea 4-5 episodes/day that started about one month ago after returning from a camping trip. He denied having taken any antibiotics in the past 3 months. He was found to have a hemoglobin level of 5.3 g/dl, MCV 66 fl, MCH18 pg, RDW 25%, Platelets 314 k/ul, WBC k/ul 8.71. His ALKP was 160 u/l, albumin low at 2.2g/dl and CRP was elevated to 7.1 mg/dl. All other labs including liver function tests, electrolytes, creatinine, labs for hemolysis and thyroid function tests were normal. Iron studies and peripheral smear were suggestive of iron deficiency anemia. His family reported weight loss of greater than 40 lbs over the past 1 year. Later group home staff reported that he probably had diarrhea more than 3-4 days every week. Infectious work up including Blood cultures, stool cultures, C diff testing and stool for ova and parasites was negative. Malabsorption was suspected and upper GI endoscopy was ordered with Celiac disease being suspected. Preliminary duodenal biopsy was negative for celiac sprue and carcinoma. The patient underwent colonoscopy and small bowel capsule endoscopy which were unremarkable. Later final biopsy report revealed, focally active duodenitis, with histopathologic changes consistent with *T. whipplei* (Whipple's disease) with characteristic pattern of PAS staining. Confirmatory molecular (PCR) testing for *T. whipplei* also returned positive. He was treated with IV ceftriaxone for 2 weeks followed by trimethoprim-sulfamethoxazole course for 1 year.

Whipple's disease is a chronic systemic infectious disease caused by the bacterium *Tropheryma whipplei*. Gastrointestinal and general symptoms include marked diarrhea (with serious malabsorption), abdominal pain, prominent weight loss, and low-grade fever. Diagnosis is based on the clinical picture and small intestinal histology revealing foamy macrophages containing periodic-acid-Schiff- (PAS-) positive material. Long-term (up to one year) antibiotic therapy provides a favorable outcome in the vast majority of cases.

89) TED NUGENT'S FIRST DRAFT

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The American "pet" is changing. More and more people are keeping non-traditional animals as pets. This has led to increases in different types of animal bites. With this, there is a need for providers to have a general awareness of the danger that some of these bites can pose. This discussion will be looking into one specific type of these atypical bites from non-traditional pets – rat bite fever – in the context of a case of a 57-year-old female presenting with confusion, nausea, vomiting, hypotension, fever, and a new irregularly irregular rhythm. Work-up found her to have a bacteremia and after a lengthy work-up exploring possible infectious sources, the diagnosis of rat bite fever was made. This presentation will focus on some of the key aspects of this disease particularly the etiology, clinical presentation, and treatment.

90) ATYPICAL PNEUMONIA REVEALS PITFALLS OF MEDICAL DECISION MAKING

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Heuristics are mental shortcuts used to simplify complex problems. In medicine, these techniques can be an efficient strategy to provide a working diagnosis; however, it is not without faults. Medical professionals educated on heuristics could avoid some of the pitfalls of these decision-making strategies. Here we present a case of Legionella pneumonia misdiagnosed as ethanol-withdrawal and aspiration pneumonia.

A 65-year-old male with a history of daily alcohol abuse was brought to the emergency department having been found down at home. He had been recently treated at a neighboring institution for alcohol withdrawal and community-acquired pneumonia. On presentation, he had a 2-liter per minute oxygen requirement, and his exam was significant for rales, dyspnea, orientation only to self, and a court-ordered ankle bracelet. He was mildly hyponatremic (133 mmol/dl), had AST and ALT elevations of 281 and 108 [iU]/L, respectively, and an undetectable blood alcohol level. A chest x-ray revealed a left upper lobe infiltrate. He was initially treated for aspiration pneumonia thought to have occurred while intoxicated and monitored with the Clinical Institute Withdrawal Assessment for Alcohol (CIWA) protocol. The patient became more somnolent necessitating intubation and was started empirically on a 14-day course of azithromycin. He did not receive any benzodiazepines during his hospitalization. Several days later a urine legionella antigen obtained on admission returned positive and he was eventually discharged without complication.

An accurate history is difficult to collect in a patient with an altered mental status, hence providers often rely on their own biases. Anchoring bias is relying on an initial piece of information with disregard for subsequent information — such as presuming an alcohol-based etiology because of this patient's similar presentation at a neighboring institution. Availability bias, treating a disease the provider regularly encounters, further influences our heuristics. Our institution commonly treats alcohol withdrawal, aspiration pneumonias and other ethanol-based etiologies, while Legionnaires' disease is rarely encountered. We nearly missed this "textbook" case of legionella pneumonia because our heuristic method forced suspicion against the less probable diagnosis and also against the patient. Patients with a history of substance abuse are at risk for a widened breadth of pathology but also increased mistrust from medical professionals. Awareness of one's own biases and thought processes can reduce time to accurate diagnosis and prevent delays in care.

91) AN INCIENTAL FINDING OF TTP

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Introduction: Thrombotic thrombocytopenic purpura (TTP) is a rare thrombotic micro-angiopathy that classically presents with a pentad of microangiopathic hemolytic anemia, thrombocytopenic purpura, neurological dysfunction, fever and renal disease. Onset is usually acute to sub-acute. This pentad is only present in about 40% of cases which makes diagnosis of this rare condition challenging especially in cases with pre-existing conditions. Making the diagnosis of TTP is crucial as this condition has a 90% mortality rate if left untreated.

Case Description: 36 y/o F with a past medical history of bilateral pulmonary embolism, systemic lupus erythematosus, and bipolar disorder presented to ED with concerns of hypotension; however, that was quickly ruled out. Her lab work was consistent with acute kidney injury (creatinine of 2.5mg/dL), anemia (HGB of 7.0mg/dL), and thrombocytopenia (15k/mcL). The patient had no active complaints at the time of admission without fever and no overt signs of bleeding on physical exam. Neurological exam was normal. Patient appeared to be manic as she had pressured speech, difficulty concentrating and was hyperactive. Upon further investigation, she was found to have worsening of proteinuria with urine protein creatinine ratio of 14,639mPR/gCR. Other labs, including peripheral smear, LDH, Haptoglobin, Coombs test, d-dimer, fibrinogen, direct and indirect bilirubin, ADAMTS 13 activity and inhibitor were collected. Initial labs were consistent with hemolytic anemia. She was initially started on treatment for immune thrombocytopenic purpura (ITP) due to preexisting autoimmune disease, however that diagnosis was ruled out given negative Coombs test. About 4 days later, the ADAMTS 13 activity came back at <5% . She was subsequently started on plasma exchange therapy and clinically improved.

Conclusion: TTP is a rare diagnosis however it should remain on the differential diagnosis of a patient with anemia and thrombocytopenia until completely ruled out. Treatment should be started prior to known ADAMTS13 activity results as delaying treatment could be life-threatening.

92) A CATASTROPHIC SERIES OF EVENTS

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Introduction: Catastrophic Anti-phospholipid Syndrome (CAPS) is a rare life-threatening condition that occurs in <1% of patients with antiphospholipid syndrome, and carries a mortality rate greater than 50%. The condition involves widespread thrombotic disease with multi-organ failure.

Case Presentation: We present a 62yo female with a prior history of anti-cardiolipin antibody who initially presented to the ED for evaluation of back pain, was discharged with a presumptive diagnosis of musculoskeletal pain, and later admitted the next day after a fall. On presentation she had leukocytosis-WBC16.5, severe thrombocytopenia with PLT count of 21, rising troponin levels-3.8 as well as LFT elevations AST576, ALT280, AlkPhos166, and Acute Kidney Injury with Cr2.45. She was transferred to the ICU. On hospital day 2 the patient developed L sided weakness and MRI of the brain revealed subacute ischemia in the right parietal-periatrial white matter. The patient was treated, per current recommendations, with high dose steroids (methylprednisone1000mg for 3 days) and plasma exchange. Symptoms and labs continued to improve during admission. She was started on Rituximab, as this has been shown to rapidly improve severe thrombocytopenia associated with CAPS. She recovered with some focal deficits and was discharged to inpatient rehab

Discussion: CAPS is a rare condition, and a high level of suspicion is needed for diagnosis. Current literature suggests that the development of CAPS is caused by a combination of a gene mutation and an environmental trigger, often an infection, in patients with antiphospholipid-syndrome. Literature also suggests that a genetic predisposition may account for why so few people with antiphospholipid syndrome develop CAPS. Our patient was interesting as we were unable to determine an actual precipitating factor that led to CAPS. Patient fulfilled criteria for definite CAPS with symptoms presenting within 1 week, involvement of at least 3 organ systems, positive anticardiolipin antibody and histo-pathologic evidence of small vessel occlusion.

93) CRYPTOCOCCAL MENINGITIS IN A NON-HIV PATIENT WITH ALCOHOLIC CIRRHOSIS

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Introduction: Cryptococcosis is an invasive fungal infection due to *Cryptococcus neoformans* or *Cryptococcus gattii* that has become increasingly prevalent in immunocompromised patients worldwide. While many cases continue to occur in the setting of advanced HIV, it is being increasingly diagnosed in HIV-negative populations including solid organ transplant recipients, organ failure syndromes, and patients receiving immunosuppressive agents. Cirrhosis of the liver is one of the organ failure syndromes associated with higher incidence of Cryptococcosis. Cryptococcosis in patients with cirrhosis is associated with a high mortality rate and rapid progression to death.

Case: This is a case of a 31-year-old female with a past medical history significant for alcoholic cirrhosis who presented to the ED with progressively worsening headache in the past month. Associated symptoms included neck stiffness, nausea, vomiting, blurry vision, and fatigue. She was being evaluated for a liver transplant by hepatology. Reported noncompliance with medications which include Spironolactone and Lasix. Obtained LP showing decreased glucose with elevated protein and white blood count. Spinal fluid was positive for Cryptococcal antigen. Patient tested negative for HIV.

Discussion: This is an interesting care where a non-HIV patient presented with cryptococcal meningitis in the setting of alcoholic liver cirrhosis. Presentation is similar to bacterial or viral meningitis and can be easily overlooked. It is important to evaluate for fungal infections in the setting of liver disease. Early recognition is imperative in order to start appropriate therapy and avoid development of cryptococcomas and progression of disease which are associated with high mortality.

94) GENERALIZED WEAKNESS IN A YOUNG FEMALE

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Introduction: Myositis is the inflammation of muscle defined by pain, tenderness, weakness and/or swelling. The more common causes of myositis include infection, autoimmune, and endocrine etiologies. In our case, we report a young female who was diagnosed with myositis and rhabdomyolysis attributed to Coxsackie B infection.

Case: A 20 year-old female with a past medical history of PCOS and Raynaud phenomenon presented from a referring hospital with several weeks of progressive myalgias and weakness. Neurological exam was significant for marked symmetric proximal muscle weakness and generalized muscle tenderness. On admission, laboratory studies were significant for a CK of 20,186, AST/ALT of 825/261 respectively, normal bilirubin and ALP, and normal creatinine. Inflammatory markers were within normal limits. Aggressive fluid resuscitation provided no improvement in symptoms. Coxsackie B type 3 AB titer was 1:320. Studies for additional Coxsackie viruses, enteroviruses, CMV, EBV and HIV were negative. ANA, Anti-LKM, Anti-SM AB and AB panels for dermatomyositis and polymyositis were negative. An echocardiogram was done which showed no myocardial abnormalities. She was started on steroid burst and IVIG. Muscle biopsy was performed, which revealed numerous necrotic fibers without inflammation; pathology consistent with acute necrotizing myopathy with possible toxic, viral, or immune-mediated causes. The patient's pain and weakness improved, as did her CK and transaminases. Due to the profound response to this therapy and + viral titers, the leading diagnosis was viral-induced autoimmune necrotizing myopathy. Full remission without permanent end-organ consequence was attributed to early immunosuppression.

Discussion: Although rare, myositis is a known manifestation of many different viral infections. Examples in literature have described symptoms from myalgias to rhabdomyolysis. CK levels are variably elevated, with values ranging from two to more than 20 times the upper limit of normal. The pathogenesis is thought be due to direct viral invasion into muscle, however, may also be related to immunologic processes as viral effects on muscle are not commonly demonstrated on biopsy. Muscle biopsy is not required for diagnosis, but can be performed to rule out other causes of myositis including autoimmune etiologies. The disease course is typically self-limiting, and supportive care alongside early aggressive treatment with corticosteroids and IVIG may improve outcomes.

95) "IS AN OLD FOE MAKING A COMEBACK?"

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Introduction: In USA, the incidence of syphilis was declining for the past several years, recently because of HIV infection, the rate of primary and secondary syphilis is gradually increasing. HIV and syphilis affects similar patient groups and co-infection is common. Syphilis may present with non-typical features in the HIV patient.

Case Description: A 58-year-old African American female presented with unintentional weight loss, generalized body rash involving palms and soles associated with hair loss of two months duration. She has history of multiple sexual partners, unprotected sex and prostitution. Skin examination showed widespread papulonodular and ulcerated lesions. The lesions were wide spread, involving palms and soles. She also has thin, fragile scalp hair and scalp hair loss without genital ulceration; other system examination was benign. Patient found to be reactive for HIV antigen with HIV-1 genotype. CD4 count was 126. Rapid plasma reagin was 1:128. Treponema Pallidum antibody was reactive.

Discussion: Generally, syphilis presents in HIV infected patients similar to general population yet with some difference. Diagnosis is based on serologic test and microbiology. For serology, both non treponemal antibody test, and specific treponemal antibody test can be used. Secondary syphilis in patient with HIV has varied skin presentation, which can mimic cutaneous lymphoma, mycobacterial infection, bacillary angiomatosis, fungal infections or Kaposi's sarcoma. In our patient, she was having diffused maculopapular rash, involving palms and soles, significant hair loss, positive serology, and skin finding. She was treated for secondary syphilis with benzathine penicillin. In newly diagnosed HIV, patients should be screened for other sexually transmitted infections, including syphilis.

96) GLIOBLASTOMA MULTIFORME – WAY OUTSIDE THE BLOOD BRAIN BARRIER

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Introduction: Brain tumors represent approximately 2% of all cancers. Glioblastoma multiforme (GBM) is one of the most common and aggressive primary brain tumors in adults. The prognosis remains poor with median survival averaging 14.6 months. Extracranial metastasis (ECM) of GBM is extremely rare and occurs in <2% of all GBMs. Normally the brain is immunologically and anatomically separated from the body by the blood brain barrier and the absence of intracranial lymphatic channels. The prognosis is particularly poor in this subset, with an average time of 1.5 months from metastasis to mortality. The risk factors predisposing to GBM ECM are still not well understood. We present a rare case of GBM with multi-system metastasis.

Case: 53-year-old male presented with several months of headaches, left-sided weakness and personality changes. MRI revealed a right temporoparietal mass. He underwent subtotal resection with pathology revealing GBM. He was treated with concurrent chemoradiation with temozolomide. Five months later, he developed right temple soft tissue swelling and MRI showed a new right periauricular soft tissue mass. Biopsy revealed GBM. One week later, he developed severe new onset back pain and imaging demonstrated a sclerotic lesion at T12. Biopsy again showed GBM. He was started on bevacizumab and completed radiation to T-spine and right periauricular area. Follow-up CT spine, 1 week later, revealed widely metastatic disease throughout the thoracic spine, ribs, and sternum. Patient was transitioned to home comfort care and passed away 9.5 months after diagnosis. Patient had consented to a whole-body autopsy, which also revealed additional ECM to the lung pleura and parenchyma, liver and lymphatic system.

Discussion: Extracranial metastases are an extremely rare complication of Glioblastoma multiforme. The mechanism of spread in GBM ECM is still unclear, but several theories of spread include iatrogenic, leptomeningeal, intramedullary dissemination and breaks in protective structures in the brain. It is possible in this case that tumor cells were seeded during the initial resection of the tumor. It is important for physicians to be aware that these manifestations can occur and be cognizant of the rapid progression in which ECM spreads. Physicians should have a low threshold to obtain further imaging in GBM patients complaining of bone or back pain or presenting with anemia, thrombocytopenia or hypercalcemia. Further studies of case reports could help stratify risks and therapies for GBM ECM.

97) IS IT HLH??

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Introduction: A markedly elevated serum ferritin level has been associated with inflammatory conditions, such as hemophagocytic lymphohistiocytosis (HLH)/macrophage activation syndrome (MAS) or adult-onset Still's disease (AOSD). However, hyperferritinemia can be caused by a variety of conditions. We present a unique case of significantly elevated ferritin and transaminitis that was likely secondary to a viral etiology.

Case: Patient is a 47-year-old female with history of chronic migraines who presented to an outside hospital with abdominal pain and fever. She had a fever of 102°F on admission. Otherwise vitals were stable. Labs were significant for elevated LFTs (max: AST 652, ALT 358, ALP 396, T. bili 1.7, GGT 390) and thrombocytopenia (platelets 124). Blood cultures were negative. Extensive work-up, including CXR, RUQ ultrasound with dopplers, CT abdomen/pelvis with contrast, and HIDA scan were unremarkable. She was empirically treated with zosyn with resolution of fevers in 48 hours. Iron panel revealed normal iron level and a ferritin level of 182,910. She was transferred for further evaluation due to concern for HLH. Ferritin and LFTs downtrended and platelets increased without intervention. Hematology was consulted and did not feel patient had HLH or a blood-related disorder based on her rapidly improving labs. Hepatology and the Infectious Disease service thought patient's presentation was more consistent with a viral etiology, though work up, including hepatitis panel, HIV, EBV, CMV, HSV1 were unremarkable. Patient was hospitalized for seven days. On day of discharge, her serum ferritin was 4930 and she had normal LFTs. She was discharged with close follow-up with hepatology.

Discussion: Markedly elevated serum ferritin has typically been thought to occur in few conditions, including AOSD, MAS and HLH. However, recent literature shows that in the adult cohort, ferritin levels >50,000 μg/L were not specific for HLH and seen most often in patients with renal failure, hepatocellular injury, infections and hematologic malignancies. Furthermore, studies have shown that there is no ferritin value above which ferritin is specific for HLH/MAS in adults. Although a higher serum ferritin (particularly >50K μg/L) increases the probability of a patient being diagnosed with HLH, the majority of adults with this degree of ferritin elevation do not have HLH. It is important for physicians to maintain a broad differential in evaluating patients who present with hyperferritinemia as the treatment will differ greatly based on etiology. Further research is still required to find a more reliable marker for diagnosing HLH in adults.

98) ACUTE PANCREATITIS IN SETTING OF DKA AND HYPERTRIGLYCERIDEMIA

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Introduction: Pancreatitis is a commonly seen problem in the hospital setting. Many patients present with acute epigastric abdominal pain that may or may not radiate to the back. Workup generally consists of making the diagnosis with elevated lipase levels or imaging, and then determining the exact cause of the acute inflammation. Most commonly, clinicians look towards gall stones and alcohol use, and perhaps less commonly, hypertriglyceridemia, and medications. This case study will introduce a patient with a slightly more complex etiology.

Case Presentation: A 28 year old male with no known past medical history presents with acute onset epigastric abdominal pain after a weekend of binge drinking, and using cocaine. He complained of associated nausea and vomiting. He also endorsed increased thirst over the last 1 month. He noted drinking water until he was full, but still felt thirsty. On his admission, notable lab findings were a lipase of 6499, blood glucose of 397, anion gap of 22, and a triglyceride level of 3122. CT abdomen had findings consistent with moderate pancreatitis and no evidence of gallstones.

Conclusion: Pancreatitis is a well-known disease and can be common in the inpatient setting. Although there are many common causes for pancreatitis, our patient had a slightly more complex event of inciting factors leading to his presentation. He had a history of binge drinking, presented with hypertriglyceridemia, and was also noted to be in DKA. There are not many studies outlining acute pancreatitis in the setting of severe dyslipidemia and DKA occurring concomitantly.

99) PHEOCHROMOCYTOMA – THE GREAT MASQUERADER

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Case Description: 68-year-old man presented to primary care physician after falling in the shower at home and was found to be hypotensive. His past medical history was significant for hypertension (HTN), hyperlipidemia, type 2 diabetes mellitus, coronary artery disease and obstructive sleep apnea. He was found to have intermittent elevated home blood pressures for at least one month, along with sweating, posterior neck pain, headaches, and multiple night time awakenings to urinate. He was admitted to the hospital with a systolic blood pressure of 304 documented earlier that day. During hospitalization, he had HTN and orthostatic hypotension. Laboratory studies included normal TSH, T3, T4, aldosterone, renin, plasma metanephrine, and 24-hour urine metanephrine. However, plasma normetanephrine was 1,528 pg/mL (elevated) and 24-hour urine normetanephrine was 4,331 ug/L (elevated). Magnetic Resonance Imaging (MRI) of the abdomen and pelvis was pertinent for 2.8 cm right adrenal lesion, consistent with the clinical suspicion of pheochromocytoma. The patient was treated with doxazosin for alpha blockade, clonidine with taper, and plan for atenolol after appropriate alpha blockade was reached prior to surgical resection of the tumor.

Discussion: A pheochromocytoma is a rare catecholamine-secreting tumor derived from chromaffin cells of the adrenal medulla. The prevalence is about 0.1% in patients with HTN. These tumors may be sporadic or part of genetic syndromes. The majority of cases are sporadic presentations. The clinical presentation can be highly variable and has been called the "great masquerader," however, the classic triad of symptoms consists of episodic headaches, sweating, and tachycardia. HTN is also a dominant sign and can be either episodic or sustained. The most specific and sensitive diagnostic test for the tumor is the determination of plasma or urinary metanephrines. The tumor can be located by computed tomography, MRI, or metaiodobenzylguanidine scintigraphy. Treatment requires resection of the tumor. Prolonged catecholamine excess can lead to multi-system damage, such as heart failure, pulmonary edema, arrhythmias, and intracranial hemorrhage. About 10% of tumors are malignant either at first operation or during follow-up. Recurrences and malignancy are more frequent in cases with large or extra adrenal tumors. Patients, especially those with familial or extra adrenal tumors, should be followed-up indefinitely.

100) AGGRESSIVE CHRONIC LYMPHOCYTIC LEUKEMIA (CLL) IN A YOUNG PATIENT WITH RAPIDLY PROGRESSING DIFFUSE LYMPHADENOPATHY

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50-year-old male who is a recent immigrant from Nigeria with no past medical history presented with diffuse lymphadenopathy that started about 4 months ago. He first noticed swelling on the right side of his neck, followed by the left side, and later in the axilla and inguinal regions. About 6 weeks after his first symptoms, he noted bilateral and symmetrical feet swelling and numbness. He contacted his primary care physician back in Nigeria who shipped him Augmentin, diclofenac, and furosemide for possible infection, inflammation, and edema, respectively. However, due to the persistence and progression of his symptoms, he presented to our hospital. At the time of presentation, he had significant diffuse lymphadenopathy involving the cervical, axillary (measuring up to 5 cm in diameter), and inguinal lymph nodes (up to 9 cm in diameter). Laboratory work up showed leukocytosis (white count of 28,000 with 78% lymphocytes), anemia, and thrombocytopenia. Computed tomography showed extensive lymphadenopathy of the chest, abdomen, and pelvis, splenomegaly, and bilateral hydronephrosis likely secondary to extensive bilateral pelvic lymphadenopathy. Flow cytometry confirmed the diagnosis of chronic lymphocytic leukemia (CLL) with trisomy 12 and CD 5+ status. Bone marrow biopsy showed no evidence of transformation to large cell lymphoma (Richter transformation). Patient was also found to have concomitant chronic Hepatitis B infection. He was started on tenofovir for Hepatitis B and on fludarabine, cyclophosphamide, and rituximab for CLL.

Discussion: CLL is the most common form of adult leukemia, accounting for 10% of hematologic malignancies. Patients with CLL present at a median age of 70 years and are usually asymptomatic at presentation. This disease is less frequent in younger patients and is usually more aggressive to this population. There are no definitive causative factors associated with CLL, although it is more common among first-degree relatives.

101) THE METAMORPHOSIS OF THE URINARY CATHETER BAG

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Case Presentation: A 74 yo male presented with worsening pressure ulcers on his buttocks. Past medical history is significant for gastric lymphoma status post resection with resultant vitamin B12 deficiency, causing bilateral lower extremity loss of function as well as neurogenic bladder necessitating a suprapubic catheter. He has been bedridden for at least two years with repeated hospitalizations for pressure ulcers. Of note, his suprapubic catheter is changed monthly at urology clinic. On initial assessment, his catheter tubing and bag were noted to be purple. The purple discoloration was initially attributed to the design of the tubing and bag. Further discussion with the patient revealed that the tubing and bag are clear to begin with but turn purple over the course of the month. The discoloration begins at the bottom of the bag and gradually extends into the tubing. The patient reported the color change began at least six months prior; however, there is no mention of the discolored tubing or bag in any of his monthly urology visit notes or during any of his hospitalizations. Although the patient was asymptomatic, a urinalysis and urine culture were performed to further elucidate the etiology of the discoloration. Urinalysis was significant for yellow, cloudy urine with blood, leukocyte esterase, WBCs, pH of 8.0, bacteria, calcium oxalate crystals and triple phosphate crystals. A urine culture grew 60,000 cfu/mL *Proteus mirabilis*, 60,000 cfu/mL *Escherichia coli* and 60,000 cfu/mL *Morganella morganii*. This patient had been chronically colonized with these bacteria dating back at least three years. For that reason, in addition to the fact that he was afebrile and asymptomatic, he was not treated for a UTI. The diagnosis of purple urine bag syndrome was made on the basis of alkaline urine and positive urine culture for *Proteus mirabilis, Escherichia coli*, and *Morganella morganii* in a patient with a chronic suprapubic catheter.

Discussion: Purple Urine Bag Syndrome is rare but can be seen in patients with chronic urinary catheters. There is no clear consensus on the pathophysiology but it is thought that bacteria convert a product of tryptophan metabolism into indigo in an alkaline environment. Spontaneous resolution of purple discoloration has been documented, and it is not necessary to treat an asymptomatic patient. For that reason, ordering a urinalysis and urine culture in an asymptomatic patient is not necessary if the etiology of the discoloration has been documented.

Conclusions: Here we present a case of Purple Urine Bag Syndrome in a man with a chronic suprapubic catheter without prior documentation of discoloration in his medical records. The purpose of this case report is to stress the importance of detailed medical records to avoid ordering unnecessary tests.

102) PERCEPTIONS OF INTERNAL MEDICINE RESIDENTS ON WRITING AND PRESENTING CASE REPORTS

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Background: Scholarly activity is seen as a vital part of graduate medical education, which is evident by the ACGME requirement that all residents participate in scholarly activity during residency. Internal medicine residency programs are challenged to expose residents to a myriad of scholarly activities while also preparing them for rigorous clinical careers and specialty training. Writing and presenting case reports provides opportunity for scholarly activity. This study assesses internal medicine residents' perceived benefits, challenges, and barriers to writing and presenting case reports.

Methods: A Qualtrics survey was emailed to a total 125 internal medicine residents at the Medical College of Wisconsin. The survey questionnaire aimed to asses if: residents have presented a case report, their perceived barriers to and factors facilitating case report writing, and potential benefits of case report writing. Responses were obtained on a 5-point Likert scale. Data was analyzed as respective frequencies and percentages. The comparison of the responses between those who have and have not presented case reports previously was performed using Fischer exact test. All analyses were performed using SAS 9.4.

Results: Forty-one residents (33%) completed the survey. Fifty-nine percent said they have not previously presented a case report. Ninety-five percent believed that finding an interesting case was an important factor in facilitating writing a case report, while 81% perceived finding a good mentor as equally important. Perceived barriers to case report writing included: lack of time (59%), lack of training in case report writing (56%), and lack of a mentor (54%). Taken together, lack of proper training and/or lack of a mentor were perceived as a barrier by 88% of those who have not presented versus 65% of those who have previously presented case reports (p=0.13).

Conclusions: Our survey-based study on internal medicine residents showed the majority have not presented case reports. While residents believed case reports provide multiple educational values, the perceive barriers to accomplishing this included lack of time, proper training, and an experienced mentor. Our findings suggest that additional institutional resources should be dedicated for designing a curriculum that promotes clinical case based scholarly activity, structured mentorship, and protected time allocated for scholarly activity.

103) CARDIAC EVALUATION IN A YOUNG FEMALE WITH RARE IDIOPATHIC MAIN PULMONARY ARTERY DILATION

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Background: Pulmonary artery (PA) dilation is usually a coincidental finding during imaging studies for dyspnea and/or angina and is a diameter of greater than 29mm or 27mm for males and females respectively. Pulmonary hypertension secondary to multiple underlying etiologies is the commonest cause of PA dilation. However, in rare cases of PA dilation as in this patient, the Laplace law which states that "intravascular pressure is directly proportional to wall tension and radius" sometimes does not hold. In such cases, careful evaluation for other possible causes is warranted.

Case Description: A 22 year old female with no significant cardiopulmonary history presented to the emergency room with a 5 day history of cough, difficulty breathing, chest pain and hemoptysis following upper respiratory prodromal symptoms. She had a normal S1 with single S2 with no wide split, no parasternal heave, faint systolic murmur at the cardiac base. Systemic examination was normal. She had a computed tomographic angiogram which showed no pulmonary embolism but revealed a dilation of the main PA with no dilation of the right and left main stem PAs increasing the suspicion for pulmonary hypertension or a post-stenotic dilatation. Follow up echocardiograms confirmed that the main pulmonary artery was enlarged at its distal portion measuring 42mm whereas it measured 30mm proximally. Right atrium and ventricle were normal in size and function. The right ventricular outflow tract was enlarged and measured 27mm but the branch pulmonary arteries were of normal size. The echocardiograms were unable to adequately calculate the pulmonary artery systolic pressure. Both aortic and pulmonic valves were structurally normal and without stenosis/regurgitation. A right heart catheterization with shunt runs revealed no significant pulmonary hypertension and no significant step up in oxygen saturation during the shunt run without left to right shunt.

Discussion: It is unusual to find dilation of the main PA without associated causes of pulmonary hypertension and/or turbulence in blood flow including left to right shunting, hereditary hemolytic telangiectasia, arteriovenous malformation, infectious causes such as syphilis and tuberculosis, connective tissue diseases, rheumatologic or vasculitis diseases. To the extent of reasonable evaluation, our patient did not have any obvious cause why this may have occurred. There was no stenotic area proximal to the dilation. She had no history of congenital heart disease. She was managed medically and her symptoms improved.

Conclusions: Initial evaluation for symptomatic patients such as ours should include CT chest and echocardiogram if dilation is confirmed. Furthermore, abnormalities in right ventricular size or function or pulmonary hypertension/right ventricular systolic pressure on echocardiogram should be confirmed with a right heart catheterization that is essential to clinch the diagnosis.

104) BICALUTAMIDE INDUCED HEPATOCELLULAR INJURY

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Introduction: Bicalutamide is an anti-androgen commonly used for the treatment of advanced prostate cancer. Liver toxicity is a rare, but severe, side effect that requires regular evaluation of liver function and close clinical monitoring.

Case Presentation: A 65-year-old male with a past medical history significant for recently diagnosed prostate cancer treated with bicalutamide and congestive heart failure presented with shortness of breath. He had a two-day history of shortness of breath, a new oxygen requirement, vascular congestion on imaging, and an elevated BNP to 7,378 consistent with decompensated heart failure. On admission, his INR was found to be 2.1 despite no anticoagulant medications which prompted further evaluation of liver function. His hepatic function panel was significant for elevated transaminases (AST=1799, ALT=1501) which was greater than a 100-fold increase from labs obtained six days before admission (AST=17, ALT=12). He denied any abdominal distention or pain, pale stools, or dark urine. On physical exam he was not jaundiced, did not have scleral icterus, and his abdomen was non-tender and non-distended. He did not display hepatomegaly. Further laboratory evaluation ruled out Hepatitis A, B, and C as potential etiologies and a right upper quadrant ultrasound displayed a normal sized liver without focal lesions. Upon chart review it was found that six days ago he began anti-androgen therapy with bicalutamide 50 mg daily for his prostate cancer. Review of the literature revealed a 6% risk of elevations in aminotransferases with bicalutamide therapy and case reports detailing cases of fulminant hepatitis caused by bicalutamide treatment leading to death. Bicalutamide was discontinued and over the next two days LFTs showed improvement without further intervention (1 day after discontinuation AST=782, ALT=1,265.) He was discharged 2 days after discontinuation with his AST and ALT improved to 446 and 905 respectively and his INR improved to 1.2. The patient was followed closely in the outpatient setting and was found to have continued improvement at 7 days post discontinuation (AST=28, ALT=168). The patient's ALT and AST had returned to baseline when rechecked 29 days after discontinuation of bicalutamide (AST=19, ALT=14, INR=1.1.)

Discussion: This case illustrates the potentially serious risk of hepatotoxicity with the use of bicalutamide for prostate cancer. The use of anti-androgens is the one of the leading treatments for prostate cancer and has been associated with hepatocellular injury ranging from transient LFT elevations to fulminant hepatitis leading to mortality. While undergoing treatment with bicalutamide it is crucial to monitor liver function through routine LFTs and to have a higher level of clinical suspicion if signs of liver dysfunction arise.

105) DERMATOMYOSITIS PRESENTING WITH DYSPHAGIA

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INTRODUCTION: Dermatomyositis is an inflammatory condition characterized by distinctive skin findings and skeletal muscle disease. Inflammatory myopathy usually presents as symmetric proximal muscle weakness; less frequently, weakness of pharyngeal muscles leads to dysphagia. A high index of suspicion is critical with atypical presentations to assess for systemic involvement and potential malignancy.

CASE PRESENTATION: A 79-year-old male presented with worsening dysphagia over three weeks. He noted a 10-pound weight loss secondary to poor intake. Physical exam was notable for an erythematous, scaly confluence of plaques across his upper torso and bilateral upper extremities proximally and over the MCP joints bilaterally. Strength was 4/5 on shoulder abduction bilaterally. MRI brain was negative for intracranial pathology. A swallow study showed severe delay and weakness in the pharyngeal phase. Neither laryngoscopy nor EGD showed evidence of structural abnormalities, ulcerations, or esophagitis. Further workup revealed elevated ESR and CRP; complement levels, CK and aldolase, and autoimmune panels were normal. Skin biopsy showed features consistent with lupus erythematosus. EMG revealed proximal muscle weakness; therefore, muscle biopsy was obtained with findings of active and chronic inflammatory myopathy with features of dermatomyositis.

DISCUSSION: Dermatomyositis is a disease marked by muscle inflammation and characteristic skin findings. Most commonly, it presents with symmetric proximal muscle weakness; like this patient, a unique few present with predominantly pharyngeal muscle weakness. Skin findings known as a heliotrope rash and Gottron's papules are pathognomonic findings, though often not present at time of initial evaluation. Diagnosis can be made on clinical signs and symptoms alone in patients with typical presentations. Supportive diagnostic tests include elevated inflammatory markers, muscle enzymes, and autoantibodies. Muscle biopsy reveals perifascicular inflammation and muscle fiber necrosis. Skin biopsy demonstrates perivascular lymphocytes, and epidermal atrophy. Treatment starts with systemic steroids; in resistant disease, immunosuppressive drugs may be utilized. After diagnosis, evaluation for systemic disease requires chest X-ray for pulmonary symptoms, echocardiogram for cardiac signs, and esophageal motility studies for dysphagia. Further, evaluation for malignancy is critical, as the incidence of cancers is increased six-fold compared to non-affected counterparts in the first year after diagnosis.

106) UNUSUAL PRESENTATION OF POLYCYSTIC KIDNEY DISEASE

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Polycystic kidney disease (PKD) is a hereditary condition of severe cyst formation in the kidneys and other organs. Presentation can be variable making diagnosis difficult and important for monitoring of long-term complications such as hypertension and renal failure.

A 65-year-old Caucasian female presented with worsening right upper quadrant (RUQ) abdominal pain for a few weeks that was dull, intermittent, and throbbing. On presentation, her vitals were stable. Abdominal examination revealed a soft, non-distended abdomen with fullness at the (RUQ). It was not rigid but there was guarding and positive Murphy's sign on palpation. On laboratory exam, her CBC and BMP were unremarkable. She had mild elevation of liver enzymes and direct bilirubin. Urinalysis was significant for urobilinogen, protein, and RBCs. Serologies for hepatitis A, B, and C were nonreactive. ANA antibodies were negative and the iron panel was within normal range.

A RUQ ultrasound was obtained which showed numerous large hepatic cysts and one right renal cyst. The CT of the abdomen and pelvis was subsequently ordered which showed numerous hepatic cysts causing a mass effect on the porta hepatis, narrowing the common bile duct and portal vein. Also revealed were multiple kidney cysts and a normal appearing gallbladder. Based on these findings, the patient was diagnosed with PKD. Her pain was managed with narcotics, but her liver enzymes continued to increase. Surgical consultation was obtained and she underwent a laparoscopic liver cyst fenestration and cholecystectomy. Post-operatively, her liver enzymes progressively improved and total bilirubin normalized. She was discharged on post-op day two in improved condition with plans for follow-up to further evaluate her newly diagnosed PKD, including a brain MRI to evaluate for berry aneurysms and genetic counseling.

Although our patient's abdominal pain, elevated LFTs, and jaundice are consistent with a diagnosis of PKD, her lack of associated symptoms, her lack of family history and delayed development of cysts is atypical. However, recognition of this disease is critical for management of potential sequela including renal failure and complications such as berry aneurysms. Equally important is to offer counseling to screen family members.

107) THAT FEELING WHEN YOUR HEART'S HARD WORK IS NOT ENOUGH: HIGH OUTPUT FAILURE

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Introduction: High output heart failure (HOHF) can be caused by chronic conditions, including severe anemia, hypoxia, obesity, acutely by septic shock, and hemorrhage. Basically, the heart is normal and there's no "pump" failure. The main problem is the underlying decrease in systemic vascular resistance ,that drives low blood pressure, and activation of neurohormonal mechanisms, favoring salt and water retention.

Case Description: A 58-year-old male presents after developing shortness-of-breath at rest for two days. He reported diet and medications compliance. Patient noticed to have a lower extremity edema. He was hypotensive with BP 83/57. Chest x-ray revealed vascular congestion with cephalization. BNP was found to be elevated. EKG and troponin were unremarkable. 2D echocardiography revealed hyper-dynamic left ventricular systolic function with ejection fraction of 84 %.

Patient has been admitted five times over the past four months with similar presentations. Following each admission, he underwent aggressive diuresis and subsequently discharged home on Frusemide, conferring a diagnosis of diastolic heart failure. It was noted, that patient had those frequent admissions after developing chronic anemia, as low as Hgb of 6.8 mg/dl. He had rick factors for HOF including anemia, chronic hypoxemia secondary to tobacco abuse, and obesity. His estimated cardiac output was 7.6 L/min and estimated mixed venous oxygen saturation was 10.7%, all speaks for HOHF. Patient cautiously diuresed. Hematology was involved to address the anemia.

Conclusion: This case illustrates patients with HOHF are overlooked and sometimes aggressively diuresed, which could be deleterious. It is recommended that HOHF patients should be diuresed cautiously because preload is needed to avoid functional outflow obstruction. Management should focus on treating the underlying causes.

108) SCREENING FOR HYPERTHYROIDISM IN PATIENTS WITH COMPLEX MEDICAL HISTORIES

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Introduction: Hyperthyroidism can present with a variety of symptoms through effects on multiple organ systems. Rare but serious complications include heart failure, hepatic dysfunction, and pancytopenia. In patients with complex medical histories, screening for thyroid function may easily be overlooked in lieu of work up for other causes of these signs and symptoms.

Case: The patient is a 49-year-old African American female with a past medical history of Burkitt's lymphoma status post chemotherapy currently in remission who presented with 3-4 weeks of worsening nausea and vomiting. She endorsed decreased oral intake and had lost 20 pounds. On exam, patient exhibited a systolic murmur and was tachycardic and tender to palpation on the right abdomen. Labs showed pancytopenia, elevated AST/ALT/bilirubin, and elevated BNP. Hematology-oncology, pulmonary, and infectious disease specialists followed with extensive work up for differentials from bacterial pneumonia to lymphoma relapse which returned unremarkable. On day 6 of admission, thyroid studies showed decreased TSH (<0.005) and elevated free T3/T4 (>32.6/>7.77). TSH receptor antibody and thyroid stimulating immunoglobulin were also elevated (29.72, >500), indicating Graves' disease. On repeat history and physical, patient had diffuse goiter, dysphagia, and a family history of thyroid disease. The patient was discontinued on antibiotics and started on atenolol with a subsequent improvement of symptoms and lab findings. Upon discharge patient was started on methimazole and followed up with endocrinology, scheduling for a thyroidectomy.

Discussion: In retrospect, hyperthyroidism explained many of the patient's symptoms. Hemodynamic changes including increased heart rate, myocardial contractility, and preload as well as decreased systemic vascular resistance and afterload result in a high output cardiac state. Her ensuing heart failure likely caused passive hepatic congestion and increased central venous pressure that led to elevated LFTs and right abdominal pain. While the causes for pancytopenia in hyperthyroidism are not well understood, cases have been reported. For patients with complex medical histories such as malignancy and immunosuppression, it is important to conduct an extensive work up to detect serious causes of illness. However, it is just as important to maintain a broad differential in order to check for more benign and curable causes. It is also essential to combat possible cognitive biases in clinical decision making, such as anchoring and confirmation biases wherein one focuses on validating specific beliefs about the patient's illness and fail to make adjustments for other possibilities. Given that thyroid disturbances can manifest with a wide range signs and symptoms, and thyroid screenings tests are relatively cost effective and expedient, such tests should always be considered in the early investigation of symptoms that fit the clinical syndrome.

109) UNIQUE PRESENTATION OF MYASTHENIA GRAVIS

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Case: The patient is a 73 year old male with a history of psoriasis and bell's palsy, who presented to the ED with complaints of acute onset left sided tongue/throat weakness and difficulty speaking. The ED performed a full stroke workup including EKG, CT angiogram head/neck, and MRI; all of which were unremarkable. The patient was discharged home.

He saw his PCP on days 1 and 3 post ED visit, with complaints of ongoing waxing and waning and progressively worsening of the same symptoms. The throat weakness had become bilateral and he developed a slight right sided facial droop, as well as one episode of diplopia when looking left. The weakness had progressed to the point where he had difficulty swallowing solids/liquids and handling his secretions. On exam, the PCP noted erythema/rash in a V1 distribution on the right. There was concern the symptoms were due to shingles and he was admitted for hydration therapy and IV antivirals.

On admission, labs revealed normal or essentially normal CBC, CMP, Mg, TSH, T3, and T4. Speech therapy recommended NPO due to aspiration risk. Throughout hospital course he developed severe dysarthria, complete paralysis of his pharyngeal muscles, orthopnea and progressive weakness of the bilateral upper extremities. Neurology was consulted and extensive work up revealed a positive *anti-MuSK, anti-AChR * and EMG studies showing and 18% decrement on repetitive stimulation, ultimately leading to the diagnosis of Myasthenia Gravis. The patient was started on plasma exchange, prednisone, and pyridostigmine.

Discussion: Myasthenia Gravis (MG) is the most common neuromuscular transmission disorder. It is an autoimmune disease with autoantibodies directed against the post synaptic neuromuscular junction receptors. MG is characterized by fluctuating weakness in various ocular, bulbar, limb and respiratory muscles. The diagnostic approach is based off clinical suspicion confirmed via serological or EMG testing. Treatment of MG involves treating symptoms with acetylcholinesterase inhibitors, and immunomodulators to treat the underlying problem. This patient was unique in that the presentation had minimal ocular complaints and his disease was primarily bulbar in nature. It has showed that MG should be considered in all patients who present with dysarthria or dysphagia.

110) ST ELEVATION MYOCARDIAL INFARCTION AND ACUTE AORTIC OCCLUSION

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Introduction: Acute aortic occlusion (AAO) is a rare vascular emergency with a high mortality rate. Myocardial infarctions from a left main coronary artery (LMCA) occlusion is also a fatal condition. We present a 74-year-old patient who presented with shortness of breath and bilateral lower extremity weakness.

Case Description: A 74-year-old male with hypertension presented with shortness of breath and sudden onset of bilateral lower extremity weakness and paresthesia. The patient was brought to the emergency room saturating at 94% on 80% BiPAP. He was able to wiggle his toes but not raise his legs off the bed. His legs were cool to touch. Labs showed a WBC of 22.4 x103/uL, troponin of 5480ng/L, BNP 416pg/nL, CK 369U/L. ABG showed a pH of 7.13, pCO2 52, HCO3 17 and pO2 83. EKG showed ST elevations in the anterior leads. Chest X-ray demonstrated right upper lobe infiltrate. The patient continued to desaturate and he was intubated. He was thought to be in septic shock from a pneumonia. A heparin drip and broad spectrum antibiotics were started. Norepinephrine was started for hypotension. An MRI of the spine was obtained to rule out cauda equina syndrome. An echocardiogram was obtained that demonstrated a severe decreased ejection fraction of 12%. The patient's legs began to become mottled and additional vasopressors were added. His dorsalis pedis pulses had no identifiable signal and femoral arteries had a weak monophasic signal by doppler. The patient underwent cardiac catheterization, found to have 99% occlusion of the LMCA and on aortogram, there was complete occlusion of the infrarenal aorta. The LMCA was successfully stented. He was emergently taken to the operating room for right axillobifemoral bypass with grafts. Postoperatively continuous renal replacement therapy was required. It was established there was a poor outcome and the family withdrew care.

Discussion: ST elevation myocardial infarctions of the LMCA can already be fatal. When combined with an AAO, mortality is high even with early interventions. A patient who presents with bilateral extremity weakness, AAO should be included in a patient's differential. This complicated case of multiple lab abnormalities can obscure a clinical picture and lead to astray diagnoses. We present an unusual presentation of an AAO with a STEMI.

111) MYASTHENIA CAN MASQUARADE AS MULTIPLE COMORBIDITIES

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Myasthenia gravis is an antibody mediated neuromuscular junction disease whose predominant symptom is ocular muscle weakness and to a lesser extent, generalized weakness. Myasthenia crisis is a rare life threatening manifestation characterized by respiratory failure. A prompt clinical diagnosis must be made, as there are no timely confirmatory tests, to begin immediate treatment with plasmapheresis or IVIG.

A 77-year old woman with PMH significant for hypothyroidism, DM II, diastolic heart failure, and a remote history of ocular myasthenia gravis was admitted for progressive general weakness. She was diagnosed with statin myopathy, CK 680 units/L, and undertreated hypothyroidism, TSH 18.33 uIU/mL. Her statin was discontinued and levothyroxine increased. Four weeks later she presented again but with worsening general weakness in addition to profound dyspnea on exertion and new hypoxic respiratory failure on 2L O2/min nasal cannula. There were no focal neurologic deficits. Chest X-ray showed crowded bronchovascular markings and hypo-inflated lungs . Troponins peaked at 0.772 ng/mL and EKG was without ST/T wave abnormalities or bundle branch block. Her initial diagnosis was an NSTEMI with dyspnea as an anginal equivalent but an echocardiogram without wall motion abnormalities reduced suspicion for cardiac ischemia. Myxedema coma was considered with a high TSH of 13.7 uIU/mL but was ruled out with a normal free T4. Myopathy was ruled out with a normal CK. Additional history from family noted diminished PO intake from intermittent choking that was suspicious for bulbar weakness. This detail when paired with the rest of her presentation lead to the diagnosis of myasthenic crisis. She was intubated for impending respiratory failure and plasmapheresis initiated but discontinued due to hypotension so IVIG was given instead. Her respiratory failure and weakness gradually resolved over the next few days. Acetylcholine receptor antibody titers that resulted days later showed high levels of binding antibody at 64.9 nmol/L. A coronary angiogram performed later in her hospital course observed no significant atherosclerosis, suggesting her troponin leak was demand ischemia.

Myasthenia crisis is a rare manifestation of a chronic and often limited disease whose features can mimic many acute illnesses. This diagnosis can be challenging especially in the elderly population who have many chronic comorbidities. There are no confirming lab tests for Myasthenia crisis since antibody titers do not reflect the severity of disease. A thorough clinical interview, physical exam, and review of past medical history are the keys to prompt diagnosis and life saving treatment.

112) MYASTHENIA MASQUARADE

Charles Yoo, MD
Medical College of Wisconsin, Milwaukee, WI

Myasthenia gravis is an antibody mediated neuromuscular junction disease whose predominant symptom is ocular muscle weakness and to a lesser extent generalized weakness. Myasthenia crisis is a rare life-threatening manifestation characterized by respiratory failure. A prompt clinical diagnosis must be made, as there are no timely confirmatory tests, in order to start treatment immediately with plasmapheresis or IVIG.

A 77-year old woman with PMH significant for hypothyroidism, DM II, diastolic heart failure, and a remote history of ocular myasthenia gravis was admitted for generalized weakness two weeks after returning from India. She was diagnosed with statin myopathy, CK 680 units/L, and undertreated hypothyroidism, TSH 18.33 uIU/mL, and so her statin was discontinued and levothyroxine increased. Four weeks later she presented with profound dyspnea on exertion, generalized weakness, and new hypoxic respiratory failure requiring 2L of oxygen/min. There were no focal neurologic deficits. Chest X-ray showed crowded bronchovascular markings due to hypoinflated lungs. Troponins peaked at 0.772 ng/mL and EKG was without ST/T wave abnormalities or bundle branch block. Initial diagnosis was an NSTEMI with dyspnea as an anginal equivalent but an echocardiogram without wall motion abnormalities reduced suspicion for cardiac ischemia. Myxedema coma was considered with a high TSH of 13.7 uIU/mL but was ruled out with a normal free T4. Myopathy was ruled out with a normal CK. Further history from family noted diminished PO intake from intermittent choking that was suspicious for dysphagia secondary to bulbar weakness. This history paired with observed muscle weakness and respiratory failure lead to the diagnosis of myasthenic crisis. She was intubated for impending respiratory failure and plasmapheresis was initiated but discontinued due to hypotension so IVIG was given instead. Her respiratory failure and weakness gradually resolved over the next few days. Antibody tests that resulted days later showed high titers of Acetylcholine receptor binding antibody at 64.9 nmol/L. A coronary angiogram performed during her hospital course observed no significant atherosclerosis indicating her troponin leak was likely demand ischemia.

Myasthenia crisis is a rare manifestation whose features can mimic the exacerbation of many chronic diseases. This diagnosis can be challenging especially in the elderly population who have many chronic comorbidities. There are no confirming lab tests for Myasthenia crisis since antibody titers do not reflect the severity of disease. A thorough clinical interview, physical exam, and review of past medical history are the keys to prompt diagnosis and life saving treatment.

113) EPSTEIN-BARR VIRUS-MEDIATED COLD AGGLUTININ DISEASE, HEMORRHAGIC GASTRITIS, AND HEPATITIS

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Cold agglutinin disease is a form of autoimmune hemolytic anemia characterized by autoantibodies directed against erythrocytes with a thermal amplitude that may be just below body temperature. This condition can pose a diagnostic challenge secondary to the infrequency at which it is seen in clinical practice.

A 25-year-old man is admitted after developing small-volume hematemesis and abdominal pain. These sympoms were associated with several days of dark-discoloration of urine and intermittent numbness of the distal fingers bilaterally. The patient also reported a new sexual contact in the last month. Physical examination demonstrated moderate jaundice and livedo reticularis on the bilateral hands. EGD demonstrated several areas of hemorrhagic gastritis and esophagitis with no ulcerations and with biopsy negative for *H. pylori*. Initial evaluation demonstrated moderate anemia and thrombocytopenia with hemoglobin of 10.8 g/dL and platelets of 97 K/uL, respectively; total bilirubin was elevated at 6.9 mg/dL and transaminases were elevated with ALT of 222 U/L and AST of 192 U/L; INR was elevated at 1.2; urinalysis demonstrated 2+ hemoglobin with urobilogen of 4.0 mg/dL; inflammatory markers were notable for an elevated CRP of 10.8 mg/dL and undetectable ESR. There was an undetectable haptoglobin and a markedly elevated LDH of 1,253 U/L with no significant erythrocyte aggregation or other abnormal findings seen on peripheral blood smear; a direct Coombs test was positive. These findings were, after additional investigation, determined to be secondary to Epstein-Barr Virus-mediated cold agglutinin disease and hepatitis with positive monospot and EBV log DNA of 4.84, positive cold agglutinins with a titer of 1:128, and elevated IgM of 412 mg/dL. The finding of hemorrhagic gastritis was, additionally, attributed to EBV viremia with no characteristic offending medications or toxins based on history. Throughout this inpatient course, his hemoglobin and platelets concentrations gradually increased with associated decreases of his transaminases and bilirubin with supportive care alone. The patient was discharged on ranitidine with close outpatient follow-up.

Cold agglutinin disease is a form of hemolytic anemia often triggered by infection. Steroids are not of benefit in this disease, although severe cases may be treated with plasmapheresis. Treating the underlying infection and supportive care usually result in long-term remission of the cold agglutinin disease.


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