2020 ABSTRACT COMPETITION
SATURDAY, OCTOBER 10, 2020 via ZOOM

Resident Abstracts

CATEGORIES ACCEPTED:

Basic Research
Clinical Research
Clinical Vignette
Quality Improvement/Patient Safety
High Value Cost-Conscious Care
Resident Abstract 1

Category Submitting for: Clinical Vignette

Abstract Title MEDIASTINAL GRANULOMA SECONDARY TO HISTOPLASMOSIS: A RARE CAUSE OF DYSPHAGIA IN IMMUNOCOMPETENT PATIENT

Abstract Text

Introduction
Histoplasmosis is highly endemic in Ohio and Mississippi river valleys. While it is usually asymptomatic in immunocompetent individuals, severe disseminated infection is mostly seen in patients with impaired cellular immunity. Mediastinal granuloma related to histoplasmosis causing dysphagia is rare, especially in immunocompetent patients.

Case Description/Methods
A previously healthy 40-year-old male presented with pleuritic-type chest pain and solid-food dysphagia for two months. He was active in military with travel to multiple countries in the Middle East prior to his illness. On exam, he appeared well-nourished, with no palpable lymphadenopathy. Labs showed WBC count 11.3 ×10³/μL, negative HIV testing. Chest computed tomography (CT) with showed a 4.5 x 2.3 cm complex, partially cystic-appearing septated lesion, with peripheral solid component in the subcarinal region, inseparable from the esophagus. Esophagogastroduodenoscopy (EGD) and endoscopic ultrasound (EUS) showed extrinsic compression on mid-esophagus and a 20 x 50 mm thinly-septated, bi-lobed, hypoechoic structure with some internal debris in subcarinal region, with no calcifications. Patient underwent thoracotomy with excision of multiple purulent matted periesophageal lymph nodes. Biopsies showed reactive lymph nodes with necrotizing and non-necrotizing granulomas with scatter ed yeast suggestive of histoplasmosis. Considering location of the mass and its symptomatic nature, patient was treated with itraconazole for two months with complete resolution of symptoms.

Discussion
Gastrointestinal manifestations of histoplasmosis are recognized clinically in around 10% of cases. Esophageal involvement is rare and can be secondary to extrinsic compression by enlarged lymph nodes or as part of disseminated process. Rarely, dysphagia can be the initial
presenting symptom of mediastinal granuloma. CT is helpful in detecting well-defined nodes with central calcification. EUS typical features include matted mediastinal lymph nodes or a mass, with possible calcifications. Mediastinal granuloma secondary to histoplasmosis causing dysphagia is rare, especially in immunocompetent individuals. Detailed history, CT scan, EGD and EUS can help aid the diagnosis. Treatment is usually dictated by patient’s presentation and immune status, and may include anti-fungal therapy, surgery or combination.
A new abstract was submitted for the Nebraska Chapter on Saturday, September 19, 2020 - 00:36:

Resident Abstract 2

**Category Submitting for:** Clinical Vignette

**Abstract Title** Paradoxical Embolism Causing ST-Elevation Myocardial Infarction in Multiple Coronary Distributions

**Abstract Text**
Paradoxical Embolism Causing ST-Elevation Myocardial Infarction in Multiple Coronary Distributions

While ischemic stroke is the most well known complication of paradoxical embolism, paradoxical embolism causing ST-elevation myocardial infarction (STEMI) is a rare and likely under-diagnosed entity, reported to cause about 0.67% of all STEMIs. Investigation and recognition of this condition and consideration of patent foramen ovale (PFO) closure are crucial to prevention of potential future events.

A previously healthy 46-year-old male presented with sudden-onset chest pain following an 8-hour drive. Physical exam findings were relatively unremarkable. Electrocardiogram revealed anterolateral ST elevations (V2-5). Labs were notable for elevated Troponin-I of 1.69 ng/mL. Emergent cardiac catheterization revealed occlusion of both the distal left anterior descending and distal second diagonal coronary arteries without evidence of atherosclerotic coronary artery disease. The occlusions were opened using a Dottering technique with an angioplasty balloon. No stents were deployed given no evidence of mural pathology. Given occlusion of multiple coronary arteries without evidence of atherosclerosis, an embolic etiology was considered. Agitated saline bubble study was positive on transthoracic echocardiography. Transesophageal echocardiography confirmed a patent foramen ovale. A 2-week ambulatory rhythm monitor and
Hypercoagulability panel were unremarkable. PFO closure was performed successfully with a 25 mm Amplatzer PFO Occluder (Abbott, St. Paul, MN). Dual antiplatelet therapy was continued for 1 year after the myocardial infarction followed by aspirin 81 mg monotherapy. The patient has had no subsequent events.

This case emphasizes that acute myocardial infarction (MI) may result from paradoxical embolism via PFO and that a high index of suspicion is warranted in patients presenting with acute MI without coronary atherosclerosis. In addition to echocardiographic imaging for PFO, a comprehensive work-up for other conditions predisposing to venous thrombosis and embolism should be conducted, including hypercoagulability testing and ambulatory rhythm monitoring. PFO closure has been shown to decrease the risk of recurrent cryptogenic stroke, and while the current literature on acute coronary syndrome (ACS) in the setting of PFO is limited, in addition to ACS management, we would strongly recommend considering PFO closure for secondary prevention.
A new abstract was submitted for the Nebraska Chapter on Saturday, September 19, 2020 - 00:10:

Resident Abstract 3

**Category Submitting for:** Clinical Vignette

**Abstract Title** Taking over the territory: A case of superdominant right coronary artery

**Abstract Text**

A 76-year-old female with history of hypertension, diabetes mellitus type 2, and hyperlipidemia was admitted with a diagnosis of small bowel obstruction and managed conservatively. During her hospitalization, she complained of chest pressure. Initial evaluation included a physical examination and Electrocardiogram, which were unremarkable. Troponin I peaked at 3.94 ng/ml (≤0.04 ng/ml). Her Echocardiogram demonstrated apical segment akinesis with an ejection fraction (EF) of 30%. Coronary angiogram showed no angiographic evidence of coronary artery disease. However, the left circumflex artery (LCX) artery did not originate from the left main coronary artery. Rather, the super-dominant right coronary artery (RCA) had a large posterolateral branch, which occupied the atrio-ventricular groove and supplied the lateral wall of the myocardium (Figure 1 and Figure 2). She was initiated on guideline directed medical therapy for cardiomyopathy.

Coronary artery anomalies are a diverse group of congenital disorders, with a reported incidence of 0.6 – 1.3%. (1) Congenital absence of the LCX is an extremely rare anomaly, with a frequency of only 0.003% in all patients who undergo coronary angiography. (1) With absence of LCX, the lateral wall of the left ventricle is mostly supplied by a super-dominant RCA (90% of the time) or occasionally by a multiple diagonal branch of LAD. (2, 3) On literature review, there are no isolated cases of a super-dominant RCA, and all the reported cases are associated with an absent LCX. Considered to be a benign anomaly, a few cases have reported its association with systolic click syndrome, ischemic changes in the zone of hypoperfusion, heart failure, and syncope. (3, 4) Congenital absence of LCX is a very rare. Coronary angiogram or coronary CTA are used for definitive diagnosis.
A new abstract was submitted for the Nebraska Chapter on Saturday, September 19, 2020 - 00:58:

Resident Abstract 4

Category Submitting for: Clinical Vignette

Abstract Title Celiac Disease as Risk Factor for Recurrent Sepsis

Abstract Text
Introduction
Celiac disease is a chronic small bowel disorder associated with recurrent diarrhea and abdominal pain, triggered by gluten ingestion and characterized histologically by mucosal inflammation, villous atrophy, and crypt hyperplasia. It is found in approximately 0.7-1% of the US population, particularly in individuals with other autoimmune syndromes though it is under-diagnosed in the general population.

Sepsis is a life-threatening inflammatory condition typically characterized by hypotension, tachycardia, hypo/hyperthermia, leukocytosis, and elevations in inflammatory markers. Incidence of sepsis in the US is rising, accounting for 1.6 million cases annually from 1979-2000, and worldwide a significant proportion of sepsis-associated deaths occur in relation to underlying injury/non-communicable disease.

Case:
A 66 year old male with past medical history of T2DM, atrial fibrillation, and hypertension presents from OSH with septic shock secondary to enterococcal bacteremia. One month prior to his admission he was admitted at an OSH for enterococcus bacteremia requiring ICU-level hospitalization with an additional visit to outside emergency department for symptoms related to his bacteremia one week later. On admission he was treated aggressively with IV fluids and antibiotics. His admission was complicated by development of atrial fibrillation with rapid ventricular rate along with repeated episodes of delirium and altered mental status. Further careful history revealed a years-long history of recurrent episodic diarrhea and persistent rash on his chest and hamstrings that had not been previously evaluated. Testing revealed positive tissue transglutaminase antibody suggestive of celiac disease. Further workup with colonoscopy was deferred due to safety concerns with the use of anesthesia in this patient. He was discharged on IV ampicillin and ceftriaxone for possible endocarditis continued through a PICC line for three weeks.
Discussion and Conclusion:
Celiac disease has been associated with increased risk of other autoimmune diseases, pancreatitis, and infections- TB, influenza, C. difficile, and particularly pneumococcal infection. Patients with celiac disease have demonstrated increased risk of sepsis stemming from these conditions. In previous studies this increased risk was found solely in individuals diagnosed with celiac disease in adulthood. The causative link between celiac disease and increased risk of sepsis has not been definitely established, although proposed explanations have included hyposplenism, increased mucosal permeability, and altered composition of the intestinal glycocalyx.
This case illustrates the potential for undiagnosed celiac disease in patients with recurrent sepsis admissions. For this 66-year-old male, his repeated admissions of enterococcal bacteremia were exacerbated by and possibly due to his undiagnosed celiac disease. As celiac disease is under-diagnosed in the United States, it should be kept in mind as a potential complicating factor during hospital admissions for sepsis, particularly as incidence of sepsis continues to rise.
A new abstract was submitted for the Nebraska Chapter on Friday, September 18, 2020 - 22:32:

Resident Abstract 5

**Category Submitting for:** Clinical Vignette

**Abstract Title** Complicated MSSA Bacteremia Following an Influenza Vaccination Site Infection

**Abstract Text**

Introduction:
A healthy 75-year-old male suffers from multiple complications of MSSA bacteremia as a result of infected site from influenza vaccine. This case highlights the need for further investigation of vaccine administration techniques. Providers must recognize potential complications from parenteral medications to minimize complications.

Case Presentation:
A 75-year-old male presented with acute altered mental status, dysarthria, and agitation after two weeks of intermittent fevers. On initial evaluation, he was disoriented with in comprehensible speech. He was moving all extremities with equally reactive pupils. Laboratory findings were significant for WBC of 18,000 and procalcitonin of 2.25ng/mL. Head CT revealed a hemorrhagic infarction of the left parietal lobe. Blood cultures grew methicillin sensitive staphylococcus aureus. Mental status improved and was nearly back to baseline after three days of antibiotics and non-operative stroke management.

Patient was found to have a 1cm x 3cm area of fluctuance over his deltoid muscle with associated tenderness and erythema where he had received his influenza vaccine four weeks prior. This fluid collection was drained; cultures grew MSSA. Persistently positive blood cultures prompted further investigation of potential sources.

MRI of the spine and left wrist due to mild pain and stiffness showed T11-T12 osteomyelitis with epidural abscess and ulnar joint effusion. These were drained operatively and cultures grew MSSA. A TEE was done for new murmur which showed a 1.4cm x 0.4cm vegetation on his mitral valve. He was treated with an extended course of cefazolin with outpatient follow-up with cardiothoracic surgery.

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The patient had a medical history of hyperlipidemia; no history of intravenous drug use.

Discussion:
This patient’s bacteremia with seeding in his spine, wrist, and mitral valve is thought to be secondary to skin infection from influenza vaccine. His initial presentation with hemorrhagic stroke is attributed to septic embolization.

While cellulitis is the most common infectious event after administration of vaccines, case reports have identified bacteremia, osteomyelitis, meningitis, and necrotizing fasciitis secondary to parenteral injections. The majority of these infections are due to Staphylococcus aureus. There have been case reports of transmission of infectious agents such as MRSA and mycobacterium linked to a single vaccine administrator.

There are varying standards for skin disinfection guidelines prior to injections. Current WHO recommendations do not require cleaning the injection site unless the skin is visibly soiled or dirty. OSHA guidelines do not require gloves to be worn during vaccine administration. CDC guidelines recommend standard hand hygiene but do not have standards for patient skin sterilization. Small studies have shown varying effectiveness in disinfection rates using isopropyl alcohol and other sterilization techniques. Further research regarding sterilization technique and associated patient outcomes is warranted. Clinicians should consider parenteral injections as a potential source of infection in patients with cellulitis and complicated infections.
A new abstract was submitted for the Nebraska Chapter on Friday, September 18, 2020 - 21:32:

Resident Abstract 6

**Category Submitting for:** Clinical Vignette

**Abstract Title** Doxy Alone Can't Fix This

**Abstract Text**

Case Presentation

A 41 year-old man presented with five days of worsening fever, body aches, sore throat, shortness of breath, and productive cough with accompanying pleuritic pain. He was hypotensive and tachycardic. He had increased work of breathing with diminished lung sounds in right lung field. Although there was no significant leukocytosis, procalcitonin was elevated to 9.46 ng/mL. CT imaging revealed a mass-like 5.2 cm cavitary lesion in right upper lobe in addition to multifocal pneumonia. COVID testing three days prior was negative via nasopharyngeal PCR swab and there were no sick contacts. He had history of asthma, gastric bypass, and was most recently HIV negative on PrEP. Infectious work up was negative. He was treated empirically with Vancomycin and Cefepime, and later transitioned to intravenous Ceftriaxone and oral Metronidazole before discharge.

Nine days following his initial presentation, he was found to be hypotensive with diminished left lung sounds despite lack of symptoms. There was a new leukocytosis and chest x-ray was notable for large left hydropneumothorax with tension. Following chest tube placement, two liters of purulent material were drained. CT demonstrated multifocal pneumonia, unchanged cavitary lesion in right lung, and significant subcutaneous emphysema. He received doses of Vancomycin, Piperacillin/Tazobactam, Cefepime, Metronidazole and Ceftriaxone during this hospitalization.

Bacterial pleural fluid cultures grew Mycoplasma Hominis prompting addition of Doxycycline. With lack of clinical improvement, patient was taken to OR for VATS and required three separate procedures to debride necrotic tissue including serratus and intercostal muscles. Patient was discharged on a six-week course of oral levofloxacin and metronidazole with M. Hominis susceptibility testing still pending at an outside lab. Follow up imaging demonstrated resolution of hydropneumothorax.

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Discussion
The progression of symptoms in cavitary pneumonia can be a major distinguishing point between acute infectious etiologies, chronic infections, and non-infectious etiologies. Bacterial lung abscesses are more often acute in presentation and often polymicrobial. Streptococcus sp. and klebsiella pneumonia are the most common, but staphylococcus aureus and pseudomonas aeruginosa coverage are important considerations depending on patient risk factors.
While Mycoplasma pneumonia is a commonly known pathogen, the other mycoplasma species are rarely pathogenic. Mycoplasma sp are unique in their small size and lack of cell wall, which makes them naturally resistant to antibiotics that target peptidoglycan synthesis including beta-lactams and cephalosporins. They are usually susceptible to tetracyclines, fluoroquinolones, and sometimes macrolides. M. Hominis is a rare causative agent for respiratory infection in immunocompromised patients and has only been documented in a small number of immunocompetent patients. Many reported cases demonstrate poor outcomes unless there is timely identification of M. Hominis and switch to effective antimicrobial therapy. M. Hominis has also been implicated in rare cases of mediastinitis, meningitis, joint infection, and endocarditis.
A new abstract was submitted for the Nebraska Chapter on Friday, September 18, 2020 - 23:05:

Resident Abstract 7

**Category Submitting for:** Clinical Vignette

**Abstract Title** Hypertension in Pregnancy: Not Just Preeclampsia

**Abstract Text**

Lupus Nephritis is a well-known and likely under-recognized complication of Systemic Lupus Erythematosus (SLE). Distinguishing active disease from preeclampsia during pregnancy presents a substantial challenge if clinical suspicion is not present. A 31-year-old G5P4 with a history of SLE on Azathioprine/Hydroxychloroquine/Prednisone presented to the hospital at 28w3d after persistent hypertension to 180/100 mmHg. She exhibited anasarca, and was found to have a stable pericardial effusion without pericardial tamponade. Initial workup revealed proteinuria, creatinine of 2.31 (baseline 0.5), hyperkalemia (5.6), Hypoalbuminemia (1.4), and anemia (5.6 g/dL). Concern was present for preeclampsia, developing HELLP syndrome, and/or potential development of lupus nephritis. Additional work-up revealed C3 and C4 levels were both low, while anti-double stranded DNA levels were high. Thus, a diagnosis of SLE flare with suspected lupus nephritis was made. Her hospital course was complicated by continued hypertension in spite of three anti-hypertensives and active diuresis, and continued decline in renal function. On hospital day 12 at 30w1d the patient went into preterm labor and delivered a liveborn male without any apparent signs of neonatal lupus. She was started on Mycophenolate and PCP prophylaxis on the day of delivery, and a renal biopsy was performed the next day. Renal biopsy revealed signs of class V lupus nephritis. Despite early delivery, initiation of Mycophenolate, and continued aggressive diuresis, renal function continued to deteriorate, and the patient was ultimately started on hemodialysis for acute renal failure.

This particular case demonstrates the complexities of differentiating between medical and obstetrical complications when such processes share symptom overlap. Treatment for pre-Continued on next page-
eclampsia and lupus nephritis differ vastly, and consideration for teratogenicity of medical therapy is of the utmost importance. Ultimately, delaying delivery may worsen renal outcomes for the mother, and failure to accurately identify active lupus may result in complications for mother and child. Collaboration amongst specialties should occur early to promote the best possible outcomes.
A new abstract was submitted for the Nebraska Chapter on Friday, September 18, 2020 - 23:22:

Resident Abstract 8

**Category Submitting for:** Clinical Vignette

**Abstract Title** Blue toe syndrome- associated pain management: a case report for literature and review

**Abstract Text**
Achieving adequate pain control is one of the most common tasks Internists are faced with daily. We present the case of a middle-aged woman who presented to our hospital with blue toe syndrome of unknown etiology. The patient presented with constant severe pain not amenable to a multimodal analgesic regimen. In this case report, we analyze the opioid and non-opioid adjuvant medications employed in treating this complex pain. Notably, Tapentadol a centrally acting opioid analgesic was used for pain control. Up-titrating Gabapentin caused a noticeable relief of pain likely due to its neuropathic component.
A new abstract was submitted for the Nebraska Chapter on Friday, September 18, 2020 - 23:32:

Resident Abstract 9

**Category Submitting for:** Clinical Vignette

**Abstract Title** If left untreated, Hepatitis C may get on your nerves.

**Abstract Text**

**Background:**
Neurological manifestations of hepatitis C include pure-sensory axonopathy, mononeuritis multiplex, or distal sensory or motor polyneuropathy. Here we present a case of polyneuropathy in a patient with untreated Hepatitis C.

**Case:**
A 53 year old African American man with a 29 year history of untreated Hepatitis C presented with acute exacerbation of pain, numbness and new onset weakness of bilateral hands that began insidiously and progressed gradually over ten days till it stabilized. He had had chronic intermittent distal bilateral lower extremity paresthesia and pain for 8 years previously. Pain was initially thought to be neuropathic, however it was unresponsive to treatment with gabapentin. His past medical history was significant for rheumatic fever, alcohol abuse, group A streptococcal bacteremia, legionella pneumonia, chronic erectile dysfunction, coronary artery disease and decompensated heart failure. Examination showed bilateral intrinsic weakness of the muscles innervated by the ulnar nerve worse on the left; right sided weakness of muscles innervated by the median nerve and bilateral loss to pinprick sensation in the area of distribution of the ulnar nerve. Reduced sensation to pinprick, decreased deep tendon reflexes and thermal pain were noted in bilateral lower extremities. Multiple skin ulcerations were also noticed on both lower limbs. Clinical and EMG findings ruled out muscular pathologies and were suggestive of bilateral ulnar and right median neuropathy with the overall picture of mixed fiber polyneuropathy. Hemoglobin A1c, vitamin B12 and TSH were within normal limits. Polyneuropathy panel, Sjogren syndrome workup, ANCA, complement, syphilis and HIV screens were all negative. Serum cryoglobulins came back positive, leading to the diagnosis of Hepatitis C induced cryoglobulinemia resulting in polyneuropathy. He was referred to infectious

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diseases clinic, where he was started on sofosbuvir/velpatasvir for treatment of hepatitis C.

Discussion:
In patients with long standing Hepatitis C virus infection presenting with neuropathic symptoms, thorough evaluation for polyneuropathy, mononeuritis multiplex or multiple mononeuropathy by EMG and serum cryoglobulin levels should be considered. Presence of serum cryoglobulin is a prognostic marker for severity of neuropathy in hepatitis C. Duration of hepatitis C has a strong correlation with HCV polyneuropathy. Early treatment may prevent further progression of symptoms.
A new abstract was submitted for the Nebraska Chapter on Friday, September 18, 2020 - 21:08:

Resident Abstract 10

**Category Submitting for:** Clinical Vignette

**Abstract Title** Cryoglobulinemias: A Unique Cause of Glomerulonephritis

**Abstract Text**

*Introduction:*
Membranoproliferative glomerulonephritis is the inflammation of glomerulus found on kidney biopsy which can affect patients of all ages. Symptoms can range from acute kidney injury to end stage kidney disease. Patients may come in with complaints of bloody urine, peripheral edema, or acute onset hypertension. Since diagnosis can only be made by kidney biopsy, a high clinical suspicion should be kept. Herein, we present a rare etiology of immune-complex glomerulonephritis is type II cryoglobulinemia associated glomerulonephritis.

*Case Presentation:*
A 55-year-old male with past medical history of aortic stenosis with valve replacement, seizures, hypertension, atrial fibrillation was admitted with 1-month history of low-grade fever, fatigue and AKI. He had facial swelling and was tachycardiac. Original workup showed elevated Cr, proteinuria and hematuria. During the hospitalization he had acute worsening of Cr from 2.13 to 5.83, necessitating further workup to evaluate the cause of AKI. Nephrology was consulted. Further lab testing revealed low complement levels, positive Rheumatoid Factor, negative P-ANCA, abnormal serum free light-chain ratio, and positive serum cryoglobulin. Kidney biopsy was performed which confirmed the diagnosis of immune complex mediated glomerulonephritis. Integrating the biopsy results with his positive RF and abnormal serum free light-chain ratio, patient was diagnosed with type II cryoglobulinemia. Patient was started on high-dose glucocorticoids and cyclophosphamide and improved and discharged in stable condition.

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Discussion:
Type II cryoglobulinemia is a syndrome associated with cryoprecipitates that include immunoglobulins and complement components. This diagnosis should be considered in all types of glomerulonephritis. Cryoglobulinemias are differentiated by their immunoglobulin composition and are sorted into Types I, II and III. Type II cryoglobulinemia is unique in that it includes both monoclonal IgM with rheumatoid factor and polyclonal immunoglobulin. Its estimated that membranoproliferative glomerulonephritis is seen in around 20-30% of patients with cryoglobulinemia. Most patients will present with Meltzer's triad: purpura, arthralgias and weakness. Patients who are diagnosed with cryoglobulinemias receive high dose glucocorticoids, cyclophosphamide and rituximab for treatment. Plasmapheresis is occasionally used, but only for organ threatening disease. Knowing the presentation of cryoglobulinemia associated immune-complex glomerulonephritis is important in treating patients with worsening kidney functioning in the acute care setting to prevent further complications and dialysis.
A new abstract was submitted for the Nebraska Chapter on Friday, September 18, 2020 - 21:05:

Resident Abstract 11

**Category Submitting for:** Clinical Vignette

**Abstract Title** A Case of Invasive Aspergillosis in a Patient with Neutropenic Fevers

**Abstract Text**

Introduction: Aspergillus fumigatus is an opportunistic fungal pathogen that is known to cause invasive disease in immunocompromised individuals. Invasive disease often presents with pneumonia and septicemia. We present a unique case of invasive aspergillosis presenting as pneumonia and neutropenic fever in a patient with myelodysplastic syndrome.

Case: A 64-year-old male with a history of alcohol use, type 2 diabetes mellitus, and pancytopenia secondary to myelodysplastic syndrome presented with acute respiratory failure requiring intubation. A workup for possible fungal infection was negative, including β-D-glucan and aspergillus galactomannan. Other causes for neutropenic fever were investigated. Due to a high suspicion of fungal infection in the setting of an immunocompromised state, the patient was placed on voriconazole. An EKG showed QT-prolongation, and the patient was subsequently switched to micafungin. One week later, a chest CT showed new nodular infiltrates in the right middle lobe, and a repeat aspergillus galactomannan antigen test was positive. He was subsequently placed on isovuconazonium for broader mold-type fungal coverage in addition to micafungin.

Discussion: There is growing resistance of Aspergillus to traditional antifungal therapies. The FKS1 gene mutation is known to confer resistance to echinocandins such as Micafungin, which was an antifungal used to treat this patient. Voriconazole has been frequently used to treat Aspergillosis. However the CDC estimates that up to 19% of Aspergillus infections are resistant to azole antifungals. β-D-glucan testing has only an 80% and 63% sensitivity and specificity, respectively, according to a meta-analysis among patients with hematologic malignancy or solid organ tumors. A meta-analysis of Aspergillus galactomannan testing demonstrated a sensitivity and specificity of 71% and 89%, respectively. The moderate sensitivity of these tests should make providers suspicious for false negative testing, especially in the setting of neutropenic

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fevers and high clinical suspicion of Aspergillosis in immunocompromised patients.

Conclusion: This immunocompromised patient presented with invasive Aspergillus septicemia despite receiving appropriate antifungal coverage for the duration of his hospital admission and previously negative testing with β-D-glucan and Aspergillus galactomannan tests. This indicates either low sensitivity of fungal antigen tests or potential resistance of Aspergillus fumigatus to traditional antifungal therapy. We recommend a high clinical suspicion for Aspergillus infection in immunocompromised patients with frequent testing and antifungal coverage with wariness for the possibility of antifungal resistance and only moderate sensitivity of our current testing strategies.
A new abstract was submitted for the Nebraska Chapter on Friday, September 18, 2020 - 20:38:

Resident Abstract 12

**Category Submitting for:** Clinical Vignette

**Abstract Title** Vitamin B12 Deficiency Manifesting as Pseudo-Thrombotic Thrombocytopenic Purpura

**Abstract Text**

Vitamin B12 Deficiency Manifesting as Pseudo-Thrombotic Thrombocytopenic Purpura
Maureen Choman, Bernadette Lamb, Nathan Anderson
Department of Internal Medicine, University of Nebraska Medical Center, Omaha, NE

Case description: A 44 year-old man with a history of known alcoholic cirrhosis presented with decreased appetite, weakness, dark stools and one episode of hematemesis. He was found to have macrocytic anemia with a hemoglobin of 4.1, platelet count of 26 and bilirubin of 2.5. He was managed initially with concern for variceal bleed, although he had no further episodes of hematemesis, remained hemodynamically stable and upper endoscopy showed no active bleeding. Additional evaluation revealed schistocytes on peripheral smear, elevated lactate dehydrogenase and undetectable haptoglobin, raising concern for a microangiopathic hemolytic anemia. Vitamin B12 returned undetectably low and following intramuscular replacement and red blood cell resuscitation, the patient’s hemoglobin stabilized and he was discharged. His anti-parietal antibody was positive confirming a diagnosis of pernicious anemia.

Discussion: The differential diagnosis of microangiopathic hemolytic anemia is a critical consideration for the general internist as its etiology may be life-threatening. Vitamin B12 deficiency is prevalent in the general population and commonly presents with macrocytic anemia and both peripheral and central neurologic complaints, although anemia to this degree is rare. The exact mechanism for vitamin B12 deficiency causing pseudo-thrombotic thrombocytopenic purpura (TTP) is unknown, but it is believed to be related to high homocysteine levels. Elevated homocysteine levels cause endothelial dysfunction which leads to vasoconstriction, platelet aggregation with activation of the coagulation system and subsequent erythrocyte fragmentation. Recognition of vitamin B12 deficiency in this context is critical as simple supplementation is an effective management strategy and unnecessary, more invasive interventions like plasma exchange therapy may be avoided.

Conclusion: Severe vitamin B12 deficiency can present similar to TTP and should remain in the differential for the hospitalist evaluating microangiopathic hemolytic anemia as this dramatically affects management.
A new abstract was submitted for the Nebraska Chapter on Friday, September 18, 2020 - 20:37:

Resident Abstract 13

**Category Submitting for:** Clinical Vignette

**Abstract Title** Myotonic Dystrophy and Transient Ischemic Dilation on LexiScan

**Abstract Text**
Myotonic Dystrophy and Transient Ischemic Dilation on LexiScan

Transient Ischemic Dilation (TID) on lexiscan is a strong indicator of diffuse subendocardial ischemia and warrants further work-up, and often intervention, via left heart catheterization (LHC). Myotonic dystrophies have well documented cardiac sequelae in the form of various arrhythmias, but their effects on perfusion studies have not been documented. We present a patient with a known history of familial myotonic dystrophy, where TID on lexiscan resulted in especially discordant findings on LHC.

A 55 year old woman presented to the hospital for elective total hysterectomy and bilateral salpingo-oophorectomy for endometrial cancer. Following the procedure she was admitted to the ICU with low blood pressures not requiring pressor support, and was extubated 2 days later. Heart rates continued to be low, at which point cardiology was consulted and metoprolol was discontinued. On postoperative day 5 a rapid response was called for asymptomatic tachycardia, and she was shown to be in atrial fibrillation on EKG. After initial spontaneous resolution, the patient once again went into atrial fibrillation with rapid ventricular response, at which point IV amiodarone was initiated. Lexiscan performed the next day revealed TID of the left ventricle, suggesting severe diffuse coronary artery disease, and a small sized area of ischemia in the apical-anterior wall segment. Left heart catheterization performed 5 days later revealed a right dominant system with no stenosis of the left main coronary artery, left anterior descending (LAD), left circumflex, 30-40% of the mid right coronary artery and 70% stenosis of the first diagonal branch of the LAD. No stent placement was required and an ICD was placed prior to discharge.

Myotonic dystrophies are a group of progressive autosomal dominant disorders arising from tri and tetrancleotide repeats, their predisposition for the development of cardiac arrhythmias is a well documented complication in DM patients, and is something to be cognizant of when providing care. This presentation of DM is significant not only for spontaneous arrhythmias, but also especially discordant findings between lexiscan and LHC. While the association between TID and severe diffuse coronary artery disease is well documented, the exact pathophysiology behind the finding is unknown. This case suggests that in patients with myotonic dystrophy, TID on lexiscan may be less specific for diffuse coronary artery disease than in the general population.
A new abstract was submitted for the Nebraska Chapter on Friday, September 18, 2020 - 19:50:

Resident Abstract 14

**Category Submitting for:** Clinical Vignette

**Abstract Title** Acute Presentation of a Neuromuscular Disease in the Setting of Sepsis

**Abstract Text**

Case presentation:

A 75-year-old female with past medical history significant for diabetes mellitus and hypertension presented with three-day onset of dysphagia to both liquids and solids, fatigable chewing and dysarthria, left-sided ptosis, and mild shortness of breath. Vital signs were for significant for tachycardia and tachypnea. Physical examination revealed severe dysarthria and bilateral ptosis. The ptosis was aggravated with sustained upward gaze and improved with ice-packing test. Facial sensation remained intact however bilateral facial weakness was noted. Motor exam demonstrated 4+ proximal muscle weakness in both upper and lower extremities. Code stroke was activated; CT head and CTA head and neck were negative for acute bleeding or large vessel occlusion. Laboratory workup was remarkable for urinary tract infection and bacteremia, with both urine and blood cultures that grew Escherichia coli. Further laboratory investigation revealed a mild leukocytosis, ESR slightly elevated to 29, and elevated CRP of 134. MRI brain did not show any acute process. TEE showed normal ejection fraction without vegetations or wall motion abnormality. The patient was admitted to the ICU for close monitoring of respiratory status. The patient did not require any supplemental oxygen or intubation during the admission. With the new presentation of fatigable dysarthria, dysphagia, ptosis alleviated with ice-packing test, and proximal muscle weakness, the patient was diagnosed with Acute Myasthenic Crisis in the setting of sepsis due to urinary tract infection.

**Treatment:**

She completed a seven-day course of Rocephin and a five-day course of intravenous immunoglobulin for myasthenic crisis. She was administered a short course of Prednisone on day seven of admission and began treatment with Pyridostigmine on day nine after complete

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resolution of the dysphagia. CT-chest ruled tout the presence of a thymoma.

Discussion:

Myasthenic crisis is known to be triggered by several factors, one of which being concomitant infection. This patients’ underlying infection likely acted as the precipitant to the acute crisis, resulting in the presentation of bulbar, respiratory, facial, and ocular muscle weakness. Studies have shown that up to twenty percent of cases of myasthenic crisis present as the initial manifestation of Myasthenia Gravis, which is demonstrated by this patient. Thus, physicians should keep in mind that sepsis can be an initial presentation of an underlying disease and acute myasthenia crisis can have varying presentations including stroke like symptoms.
A Complicated Case of Aortic Bioprosthetic Regurgitation

Despite their numerous advantages over Mechanical Heart Valves, structural deterioration of Bioprosthetic Heart valves remains a cumbersome complication after several years of implantation. Depending on the severity, this can be life threatening. We present a challenging case of severe Aortic Bioprosthetic Valve Regurgitation that presented with multiple complications.

A 78 year old male with history of thoracic aortic aneurysm with root dilation, treated with total aortic root replacement, aortic bioprosthesis, and mitral valve repair 10 years ago, presented with new onset Chest pain. On examination Prominent diastolic murmur was noted with positive Corrigan's pulse, and signs of fluid overload. Patient had elevated troponin, ischemic EKG changes. Transthoracic echocardiography showed mild to moderate Aortic Regurgitation (AR). Patient underwent Left Heart Catha with Stent to Obtuse Marginal 2 artery. We decided to further evaluate aortic regurgitation with transesophageal echocardiogram (TEE). Severe AR was noted on TEE. Furthermore, a vegetation was noted on non-coronary aortic cusp. Subsequent Blood Cultures were 1/2 positive for MSSA. Patient had progressive deterioration with worsening Heart Failure, Pulmonary edema and Renal failure. Patient had poor response to diuresis. He was started on Antibiotics. Patient was evaluated by Infectious Disease, Cardiothoracic Surgery and Structural Cardiology. He was deemed very high risk for Surgery. After extensive discussion with the family Valve in Valve TAVR was planned due to rapid clinical deterioration, despite patient having suspicion for active endocarditis. During pre-op CT evaluation patient coded in the Radiology with ROSC achieved after a few minutes of CPR, however needed to be started on epinephrine drip and needed Hemodialysis prior to the procedure. Fortunately, patient’s second set of Blood Cultures stayed negative. He underwent successful Valve in Valve TAVR. Patient did well after the procedure. He completed long term

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Antibiotics course and was able to come off Hemodialysis a month after discharge.

Bioprosthetic valve deterioration is a clinically important long term complication of Aortic Valve replacement. Management varies depending on the severity of the regurgitation and presence of symptoms. Management includes regular Echocardiographic follow up, medical therapy, and surgical/TAVR approach in severe symptomatic cases. This case highlights the dilemma between taking a conservative approach in severe AR in the presence of active infection versus intervening given patient's rapid clinical decline. The Structural Cardiology team opted to proceed with Valve in Valve TAVR despite patient having active infection due to his rapid deterioration. this also highlights importance of Valve in Valve TAVR in these patient population that are prohibitive risk for Surgery due to their co-morbidities.
Abstract Title: Clinically Significant Ileus in Patients with Acute Pancreatitis is Associated with Severity of Pancreatitis, Not Volume of Fluid

Abstract Text

Background: The recovery of gastrointestinal functions is an important determinant of course of acute pancreatitis and the timing of hospital discharge. Here, we evaluated association between fluid resuscitation volume and opioid medical use with clinically significant ileus development in patients with acute pancreatitis.

Methods: Consecutive adults admitted with acute pancreatitis between Jan 2014 and Dec 2019 to our academic and two community hospital were included. The Bedside Index for Severe Acute Pancreatitis (BISAP) and systemic inflammatory response syndrome (SIRS) were used to predict severity of pancreatitis based on their readily availability and ease in use. Severity of pancreatitis was determined based on the Revised Atlanta classification. Fluid resuscitation volume was collected as administered on day 1 and 2. Opioid dose was calculated as morphine dose equivalent in first 48 hours. Clinically significant ileus was determined based on treating physician’s assessment with radiological diagnosis.

Results: A total of 441 unique patients were included in the study. Among them, 49 (11%) patients developed clinically significant ileus. Demographics of patients with or without ileus are were similar between the two groups. On univariate analysis, the presence of SIRS syndrome (<0.001), a >3 BISAP score (p<0.001) and severity of pancreatitis (p<0.001) were associated with ileus, fluid resuscitation volume and cumulative opioid dose on day 1-2 on the other hand, were not. However, Ileus development was associated with increased length of hospital stay and admission to intensive care unit.

Conclusion: Our observation show that ileus development is associated with severity of acute pancreatitis, not with fluid resuscitation volume or opioid pain medication dose.
Abstract Title  Acute hepatotoxicity secondary to immunosuppressive therapy for Autoimmune Hepatitis

Abstract Text
Introduction:
Autoimmune hepatitis (AIH) is characterized by hypergammaglobulinemia and circulating autoantibodies, resulting in chronic hepatic inflammation and immune-mediated destruction of hepatocytes. It is a rare condition, with annual incidence of 1 in 200,000 cases in the United States. AIH predominantly affects females of all ages and ethnic groups. The treatment of AIH includes glucocorticoids and immunosuppressants, such as Azathioprine (AZA) or 6-mercaptopurine (6-MP).

Case:
A 50 year old female presented to the emergency department with two weeks of abdominal pain associated with rigors, jaundice, anorexia, nausea without vomiting, acholia, and choluria. She has a past medical history of compensated cirrhosis secondary to non-alcoholic steatohepatitis (NASH), autoimmune hepatitis, and morbid obesity. The diagnosis of AIH and NASH were made on liver biopsy nine years prior to this admission. After diagnosis, treatment was initiated with prednisone and AZA. Due to medication side effects, she was switched from AZA to 6-MP 75 mg daily and budesonide 9 mg daily. Following an AIH flare, her dose of budesonide and 6-MP were increased to 9 mg and 100 mg, respectively. Her weight increased to 333 lbs (BMI 55 kg/m2), so she was referred for bariatric surgery and underwent a laparoscopic sleeve gastrectomy, successfully losing 150 lbs after her procedure. Three months prior to presentation, her dose of 6-MP had been increased 150 mg daily.

On arrival, she was noted to be jaundiced and icteric; she was tender to palpation in the left upper quadrant. Laboratory testing revealed and AST of 117, ALT 113, Alkaline Phosphatase

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116, and Total Bilirubin 3.8, amylase 53, lipase 57. She was admitted for evaluation of biliary duct obstruction. An abdominal ultrasound noted mild hepatic steatosis and liver surface nodularity, but no evidence of biliary duct dilatation. Given the lack of findings on abdominal imaging and persistent liver chemistry abnormalities, additional testing was performed. Thiopurine methyltransferase and 6-thioguanine levels were normal, but, 6-methylmercaptopurine levels were markedly elevated at 14,674 (reference range <5,700). It was determined that her elevated LFTs were a result of hepatotoxicity secondary to her 6-MP. Her abdominal pain, nausea, and vomiting spontaneously resolved and she was discharged.

Discussion:
In patients with moderate to severe AIH who are at increased risks for adverse effects of glucocorticoids, combination therapy with steroids and immunosuppressant agents should be considered. As observed in our patient, budesonide may be considered as the second line agent. Clinicians should be aware that the drug metabolites 6-thioguanine and 6-methylmercaptopurine can be measured to assess for drug toxicity or therapy adherence. In addition, dose adjustment should be considered in patients with new abnormalities in liver chemistry or significant weight loss, such as from bariatric surgery.
Abstract Text
Tuberculous meningitis (TBM) manifests as a sub-acute meningitis. We present a case of optic nerve involvement in TBM, a rare manifestation with diagnostic and clinical significance. A 33-year-old man from Nepal and migrated to the United States about 6 years ago with a history of positive TB quantiferon test but could not complete latent mycobacterium tuberculosis (MTB) treatment due to elevated liver enzymes. He initially presented to the hospital with the complains of progressive weight loss, persistent occipital headache, confusion and slurred speech. Physical exam was consistent with hyperreflexia, mild left-sided ptosis with Left abducens palsy and no visual field deficit. Initial MRI of the brain revealed multiple small contrast enhancing lesions throughout the bilateral cerebral hemispheres and inferior cerebellum areas with adjacent vasogenic edema and basilar meningitis pattern. Meanwhile CT Chest had bilateral centrilobular nodules. Cerebrospinal fluid (CSF) analysis showed neutrophilic pleocytosis, high protein and very low glucose. He was started on isoniazid (with pyridoxine), rifampin, ethambutol, and pyrazinamide along with high dose dexamethasone taper for suspected TBM. Later, MTB DNA probe from Bronchioalveolar lavage (BAL) and CSF reported positive and he grew pan-susceptible isolate of MTB on both samples. Brain MRI after 6 months of therapy revealed radiologic worsening of brain lesions. At that point, moxifloxacin 400 mg daily and linezolid 600 mg twice daily were added to the regimen along with oral dexamethasone taper and pyrazinamide 1000 mg daily was resumed, as he had clinical progression on first line therapy. After additional 6 months of this enhanced regimen, he started experiencing blurring of vision. Initially, it was presumed to be secondary to optic neuritis associated with linezolid and it was held. His vision did not improve despite holding linezolid and eye exam revealed left homonymous hemianopia. Repeat MRI of the brain confirmed extensive changes of basilar meningitis completely enveloping the optic chiasm. MRI of the orbit did not show involvement of the orbit. Given the progression of disease on current doses of anti-
tubercular therapy, medication levels were obtained which were sub-therapeutic. Hence, further increased the doses to isoniazid 1000 mg, rifampin 1200 mg, pyrazinamide 2000 mg and moxifloxacin 600 mg daily in divided doses to ensure adequate levels and CNS penetration. There was gradual improvement in visual symptoms on monthly follow up visits. Up to 12 months of enhanced therapy is planned.

TBM associated inflammatory infiltrate and adhesions can lead to cranial nerve palsies (particularly II, III, IV, VI, VII, and VIII) along with constriction of the internal carotid artery as well as obstruction of CSF flow leading to hydrocephalus. Physicians should remain cognizant of new onset visual field deficits in TBM cases, as it will require more aggressive clinical monitoring and dose adjustments for the anti tuberculous regimen.
Introduction:

To err is human; to report is being responsible. Physicians are still far behind on reporting medical errors they see on a day-to-day basis [1]. Recognizing this, the Accreditation Council for Graduate Medical Education requires trainees to know about event reporting systems and report events [2]. While 96.7% of residents knew how to report an event, only 18.2% had reported a near miss/close call event [2].

There have been several studies to improve event reporting. Program-specific educational sessions and individual feedback had mixed or no improvement in reporting [3-5]. A direct cash bonus or retirement benefit increased reporting [6, 7].

At UNMC, trainees get feedback inconsistently. We hypothesized by giving directed feedback to residency programs we could increase event reporting and comfort with reporting.

Methods:

Four residency programs were identified; Internal Medicine (IM), Family Medicine (FM), Anesthesia, and OB/Gyn. Program directors for each residency approved, and IRB exemption was obtained. IM & FM residents (Group 1) were given feedback in a monthly email regarding their event reports, and a Patient Safety staff member held twice-monthly meetings. OB/Gyn & Anesthesia residents (Group 2) were given feedback in a monthly email. All other residencies were treated as control with standard treatment. The intervention was completed over six months. Surveys gathering attitudes towards reporting were collected. Data regarding event
reporting rates and types of events were collected in a de-identified manner.

Results:

Group 1 and 2 had an increased level of reporting compared to the control in all time periods, but the median event reporting rate did not significantly increase over the intervention or post-intervention period. There was an increase in residents who received feedback on their event report (17% to 62%, p-value 0.0087). Overall, resident comfort in filling out an event report increased from a mean of 3.3 to 4 on a 5 point scale (p-value 0.0055).

Conclusion:

While medical trainees are witnessing preventable medical errors, they are not reporting them. Previous efforts to increase event reporting rates with education programs, explicit expectations for reporting, feedback, and financial incentives have seen variable changes in event reporting rates. These are felt to not be generalizable to other programs. We developed a standardized feedback program with in-person and electronic feedback over a six month pilot period, assessed residents' attitudes at the beginning and end of the pilot period, and actively tracked safety event reports. Our intervention did not demonstrate a significant increase in event reporting over time but did increase feedback to residents and overall comfort with reporting in the future. Increases may have been masked by groups already being likely reporters. Next steps include potential interventions in lower baseline groups and further automaticity in event report feedback.
A new abstract was submitted for the Nebraska Chapter on Friday, September 18, 2020 - 12:06:

Resident Abstract 20

Category Submitting for: Clinical Vignette

Abstract Title Unex-spleen-ed Syncope Following Colonoscopy

Abstract Text
Colonoscopy is the most common endoscopic procedure and is performed for the diagnosis and treatment of colorectal disorders. It is generally considered to be a safe procedure, with adverse events occurring in less than 1% of total cases. The most common adverse events associated with the procedure include hemorrhage and colonic perforation.

A 60-year-old female presented for elective colonoscopy. No intraprocedural complications were noted. Following the procedure, she suffered a syncopal episode with associated lightheadedness, dizziness, and diaphoresis in the recovery unit. Head CT demonstrated no intracranial hemorrhage or mass effect. She was admitted for observation overnight, which was complicated by worsening abdominal pain, left shoulder pain, and hypotension with significant acute blood loss anemia.

Abdominal CT angiography showed massive left perisplenic and paracolic gutter hematoma with hemoperitoneum contiguous with a large subcapsular splenic hematoma. She underwent proximal splenic artery coil embolization with Interventional Radiology. Her hemoglobin continued to downtrend and repeat CT abdomen and pelvis showed active contrast extravasation suggestive of active bleeding with multifocal gas foci within a perisplenic hematoma concerning for developing abscess or perforation. The patient was taken to the operating room for exploratory laparotomy and underwent splenectomy, lysis of adhesions, and right-sided chest tube placement for pneumothorax.

Clinical recovery was achieved over a 13-day hospitalization. She will now require additional vaccination for asplenia. Splenic injury following colonoscopy is an exceedingly rare complication, reportedly occurring in less than 0.045% of cases. Most affected patients are female (71%). Symptom presentation may occur immediately following the procedure or up to several days later, with the most common symptom being abdominal pain. However, referred left shoulder pain (Kehr’s sign) and hypotension have also been previously described. Emergent splenectomy is often required, though splenic artery embolization has been successfully performed. The procedural factors

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associated with splenic injury include looping of the instrument and traction on the splenocolic
ligament. Patient-specific risk factors include prior abdominal surgery, adhesions between the
colon and spleen, presence of a large polyp or mass at the splenic flexure, endometriosis,
abdominal inflammation, infection, and anticoagulant use. However, it can occur in patients
without these described risk factors. The patient in this case had a history of appendectomy
and right salpingo-oophorectomy. Splenic injury is likely an underreported and underrecognized
complication of colonoscopy. Rates of splenic complications are likely to increase due to the
rising number of colonoscopies and those being performed by a broader selection of providers.
Providers should have a low threshold for evaluation for hemoperitoneum secondary to splenic
injury in patients presenting with abdominal pain, hypotension, and syncope following
endoscopic evaluation.
A new abstract was submitted for the Nebraska Chapter on Friday, September 18, 2020 - 12:04:

Resident Abstract 21

**Category Submitting for:** Clinical Vignette

**Abstract Title** COVID-19 and the Liver

**Abstract Text**
Presentation: A 56 year-old woman presented to clinic with oliguria. Prior to presentation, patient reported three days of reduced urine output despite drinking large amounts of water as well as cough, neck pain, and facial swelling. Metabolic panel testing in clinic revealed hepatic injury with aspartate aminotransferase (AST) 8270 U/L, alanine aminotransferase (ALT) 1170 U/L, alkaline phosphatase (AlkP) 208 U/L, total bilirubin 6.3 mg/dL, with direct bilirubin 2.5 mg/dL. Patient was asked to visit ER the next day where further testing revealed improved liver function tests (LFT) compared to previous day, elevated INR of 1.8, undetectable salicylate level, and serum acetaminophen level of 18 ug/mL. Despite utilizing a video-interpreter, patient communication was difficult. However, her family members worked in a meat packing facility known to have a COVID19 outbreak. On physical exam, she appeared fatigued, but her exam was otherwise unremarkable.

Hospital course: Treatment with N-acetylcysteine was initiated, but later discontinued when acetaminophen level rapidly decreased and family denied heavy usage or attempt at overdose. Family denied alcohol or illicit drug use. SARS-COV-2 by RT-PCR was positive. Testing for viral hepatitis including serology for hepatitis A, B, C, cytomegalovirus, Epstein-Barr virus, herpes simplex virus, human immunodeficiency virus, varicella zoster virus, were negative for acute infection. Anti-DsDNA was negative. Acute inflammatory markers including CRP, LDH, and ferritin were significantly elevated and trended down during hospitalization. CT scan of abdomen revealed hepatic steatosis. Abdominal ultrasound showed fatty liver of normal size without ascites, normal biliary structure without cholelithiasis, and doppler imaging showed patent hepatic and portal vasculature. Patient was treated with intravenous fluids and electrolytes replenishment, with rapid improvement of patient’s clinical status and liver function tests. After four days, patient was discharged asymptomatic with significantly improved LFTs. At 3 week follow up, LFTs remained normal.

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Discussion: Although morbidity and mortality associated with COVID-19 occurs primarily due to respiratory failure, SARS-COV-2 infection is a systemic infection with diffuse inflammation and multiple organ involvement. COVID-19 epidemiology has shown elevations in liver function tests usually 2 – 3 x normal. In our case, the patient had a fulminant hepatitis associated with COVID19 infection. Tests for acute viral causes of hepatitis were negative. Patient did have a low positive Hep C antibody, but had undetectable viral load. Patient improved with conservative management, hence liver biopsy was not conducted. Further data is needed to assess pattern of liver injuries and liver enzymes elevation as well as long term implications. The exact spectrum of organ involvement and symptomatology remains unclear, though certain strains of SARS-COV-2 may be exhibiting tropism towards hepatocytes. In a patient presenting with hepatitis without any clear etiology, testing for SARS-COV-2 irrespective of symptomatology or pulmonary involvement should be considered.
A new abstract was submitted for the Nebraska Chapter on Friday, September 18, 2020 - 11:53:

Resident Abstract 22

**Category Submitting for:** Clinical Vignette

**Abstract Title** Not all ground glass opacities are caused by SARS-Cov-2: A case of pulmonary histoplasmosis mimicking COVID-19 pneumonia

**Abstract Text**
It is important to remember that not all diffuse ground glass opacities are COVID-19, even at the height of the pandemic. Here we present a case of a young, healthy, immunocompetent patient who presented with fever, shortness of breath and a diffuse radiographic pattern all suggestive of COVID-19 which was later found to be due to pulmonary histoplasmosis.

30 year old man with history of HTN, GERD and OSA presented to the ER on with complaints of cough, fever (Tmax of 104), chills, and generalized body aches with associated chest tightness and shortness of breath for the past four days. Reported oxygen saturation at home ranged from 88 - 90%. Patient was tested for COVID one day prior to his ER visit. Pertinent social history included remote travel via car to Michigan, having a babysitter whose parents tested positive for COVID but tested negative for COVID herself and a history of cleaning chimneys. On arrival, HR was 112/minute, RR of 18/minute and sating at 92% on RA. CBC and CMP were within normal limits. D Dimer was 1.1. CXR showed diffuse bilateral interstitial and alveolar infiltrates. CT angiogram was done which showed diffuse bilateral ground glass, nodular opacities with a pattern consistent with COVID along with mediastinal and hilar lymphadenopathy (image 1). Sputum culture, urine pneumococcal and legionella antigen, pro calcitonin and respiratory pathogen screen resulted negative. He tested negative for SARS-Cov-2 twice by this time. Given unexplained hypoxemia, bronchoscopy was performed with transbronchial biopsies of lymph nodes. Bronchoalveolar lavage (BAL) and biopsies both showed yeast forms consistent with histoplasmosis. Lymph node aspirate biopsies were negative for malignancy and for lymphoproliferative diseases. Histoplasma Ab Mycelial CF and Histoplasma Ab Yeast CF were 1:16 and 1:64 respectively. Patient was started and discharged on itraconazole and oral prednisone. He was readmitted 2 days later with complaints of fever and worsening shortness of breath requiring 10 L via NC upon presentation with a WBC count of 13.6. Patient was retested

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for COVID which came back negative. CT chest showed increasing opacities with interval
depression of previous infiltrates. Amphotericin B was added which was to be continued for 2
weeks in conjunction with steroids and plans to continue itraconazole for 3 months.

In light of the recent pandemic, it is very important not to forget about other infectious etiology of
acute hypoxic respiratory failure especially endemic mycoses like histoplasmosis, pneumocystis
pneumonia, cmv pneumonia and non-infectious etiologies like malignancies. Although quick
identification and management of COVID is important, other potential causes should also be
sought out to avoid delays in treating the actual cause.
A new abstract was submitted for the Nebraska Chapter on Friday, September 18, 2020 - 13:42:

Resident Abstract 23

**Category Submitting for:** Clinical Vignette

**Abstract Title** New-onset autoimmune progesterone dermatitis after in vitro fertilization

**Abstract Text**

Introduction: Autoimmune progesterone dermatitis (AIPD) is a rare hypersensitivity reaction to progesterone typically characterized by cyclical symptoms ranging from multiple skin rashes including delayed urticaria to anaphylaxis and corresponds to the luteal phase of the menstrual cycle. Few case reports have noted AIPD due to exogenous progesterone through infertility treatments. We present a unique case of new-onset AIPD secondary to in vitro fertilization (IVF) treatment.

Case Description: A 35-year old woman with a history of infertility presented to clinic for suspected urticaria in the setting of recent IVF attempts and no previous history of rashes associated with menstrual cycle. She recounted a diffuse, pruritic, and raised rash consistent with urticaria on her bilateral arms, legs, and hands that appeared 7 days following her first round of IVF. Her first IVF treatment consisted of intramuscular progesterone in sesame oil. Her rash was subsequently treated with antihistamines and oral steroids and resolved in 2 weeks. After a second round of IVF containing progesterone in ethylate oil, a different rash appeared 7 days following her treatment. She had a maculopapular rash with some sloughing primarily centered on her trunk with distal spread to her limbs as well. She also developed pruritus and mild swelling on her lips and scalp but denied anaphylactic symptoms. This was treated again with systemic steroids and she recovered similarly to her previous episode. During this time, she was on multiple immunosuppressants, including hydroxychloroquine, tacrolimus, and naltrexone, indicated for her IVF. Skin biopsy showed severe inflammation concerning for drug reaction. Autoimmune work up, thyroid studies, and unique food allergy alpha-gal were unremarkable. Allergy skin testing to ethyl oleate, polyethylene glycol, sesame oil, polysorbate, and progesterone have been scheduled and are not yet resulted.

Discussion: AIPD in the setting of multiple IVF treatments is a barrier to women attempting to conceive. Women are exposed to high doses of progesterone during fertility treatments, and little is reported in scientific literature regarding new-onset AIPD secondary to high amounts of exogenous progesterone in IVF. A 10-step protocol for progesterone desensitization was developed by Prieto-Garcia et. al to achieve viable pregnancies in women with AIPD. It is important for a patient with AIPD secondary to fertility treatments to be evaluated by an allergist to help improve symptoms and achieve a successful pregnancy through progesterone desensitization.
A new abstract was submitted for the Nebraska Chapter on Friday, September 18, 2020 - 15:17:

Resident Abstract 24

**Category Submitting for**: Clinical Vignette

**Abstract Title** COVID-19 disguising as a COPD exacerbation in a patient with COPD and pulmonary sarcoidosis.

**Abstract Text**

Ten months into the Covid-19 pandemic and the medical community is still baffled by the myriad of its clinical presentations, unpredictable nature of its progression, with its mortality rate, ranges between two and twenty percent depending upon patient's demographic factors, and other comorbidities.

A 63-year-old Caucasian male, with a past medical history that is significant for pulmonary sarcoidosis on steroids, COPD, heart failure, pulmonary embolism, type-2 diabetes mellitus, hypertension, hyperlipidemia, recent hospitalization for COPD exacerbation, and bacteremia with negative COVID test less than a month prior to presenting with a five-day history of increasing dyspnea, a slight increase in productive cough. On arrival, He was afebrile, tachypneic, with audible wheezes. His SpO2 was 91-percent on room air. He was self-quarantined at home denying any contact with sick individuals.

CXR and CTPA were negative for any newly acute findings. Procalcitonin and WBC were normal. The provisional diagnosis made was acute COPD exacerbation versus sarcoidosis flare. He received intravenous methyl-prednisone and BiPap therapy. He initially showed a noticeable improvement with short-acting Duo-Neb as well as aggressive pulmonary hygiene. His O2 requirement dropped to 1L via nasal-cannula (NC) by the forth-day post-admission. His home prednisone was increased to 60 mg, which corresponds to the effective dose managing his sarcoidosis flare in the past, and he received sulfamethoxazole-trimethoprim for Pneumocystis prophylaxis. Mycophenolate as a long-term term steroid-sparing alternative was considered.

Suddenly, on the 5th-day, he had respiratory decompensation. Other possible causes, such as
vocal cord dysfunction, sleep apnea, and exacerbation of heart failure were ruled out. On the 8th-day, Broad-spectrum antibiotics were started after procalcitonin jumped to 0.13 and continuous increase in O2 requirement to High flow NC. A COVID swab was collected on the 9th-day, prior to bronchoscopy, after CXR showed developing bilateral interstitial opacities. Following a positive COVID test result. He received dexamethasone, Remdesivir, and convalescent plasma. Unfortunately, his condition continued to expeditiously decline. He was transitioned to BiPAP 100-percent FiO2 on the 16th-day, followed by intubation the next day and Bronchoscopy to clear mucus plugging. antifungal was added to his extensive antibiotic regimen after BAL was positive for candida, aspergillus, and pseudomonas. Despite the above-mentioned therapeutic measures, he ended up with refractory type-2 acute hypoxic respiratory failure. His family opted for compassionate extubating and comfort care. Sadly he passed away on the 17th-day post-admission.

This case illustrates an example of SARS-CoV-2 presentation in immunocompromised patients and those having chronic respiratory conditions known for frequent exacerbations; that can obscure the universally recognized clinical features of Covid19. It delineates the need to have high clinical suspicion, which will increase the odds of early detection and access to therapeutic measures, acclaimed for being mostly beneficial when given at the early course of the disease.
A new abstract was submitted for the Nebraska Chapter on Thursday, September 17, 2020 - 11:49:

Resident Abstract 25

**Category Submitting for:** Clinical Vignette

**Abstract Title** Bacteria in the blood, gas in the bones

**Abstract Text**

Introduction: Emphysematous osteomyelitis (EO) is associated with gram-negative or anaerobic bacteria. The infection exhibits characteristic imaging findings and is important to recognize as it often requires prolonged antibiotic therapy and surgical intervention. This case is designed to assist physicians in recognizing a rare manifestation of infection.

Case Presentation: A 47-year-old male presented with a two-week history of non-productive cough, night sweats and anterior chest wall pain. He further endorsed 15lbs of unintentional weight loss, exacerbated by the presence of painful oral ulcers.

On initial presentation, his temperature was 38.4°C; he was tachycardic and appeared cachectic. He had diffuse white plaques on the buccal mucosa and tongue. The anterolateral chest wall was tender to palpation, along with diffuse abdominal tenderness. The remainder of his physical exam, including pulmonary evaluation, was unremarkable.

His labs showed a WBC count of 14.8 (4.0-11.0 x10^3/uL). Pulmonary tuberculosis was ruled out, along with negative HIV screening. He had pyuria and bacteriuria on urinalysis, with culture growing >100,000 col/mL of Escherichia coli. Blood cultures similarly grew E. coli. Chest x-ray showed hazy opacification in medial left lower lobe, suspicious for developing pneumonia, and follow-up computerized tomography (CT) of the chest revealed intramedullary gas in the right 5th and left 6th ribs without cortical destruction, accompanied by adjacent chest wall and pleural thickening. He underwent subsequent biopsy of the right 5th rib; pathology demonstrated primarily blood without significant amount of bone. However, the cultures did grow E. coli.

He was diagnosed with emphysematous osteomyelitis secondary to E. coli bloodstream infection. Initial antibiotic treatment with ceftriaxone was subsequently transitioned to oral

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levofloxacin at discharge for 6 weeks of total therapy, but he was lost to follow up.

Discussion: Emphysematous osteomyelitis (EO) was first described in 1981. Prior case reports have described a predominance in immunosuppressed patients with documented involvement of the femur, pelvis and vertebral bodies. To our knowledge, this is the first case identifying rib involvement. On imaging, EO appears as clusters of three or more irregularly irregular-sized distinct foci of intramedullary gas, giving it a characteristic “pumice stone” appearance, accompanied by the unique finding of the combination of intraosseous and extraosseous gas without any cortical destruction.

Anaerobes or the Enterobacteriaceae family are primarily responsible, and while there are cases of contiguous spread, most infections are introduced hematogenously. In addition to targeted antibiotic therapy, surgical intervention is beneficial. Duration of therapy has not been defined and tends to rely on the clinical scenario. However, a six-week minimum appears reasonable as with other forms of osteomyelitis.
A new abstract was submitted for the Nebraska Chapter on Thursday, September 17, 2020 - 10:43:

Resident Abstract 26

Category Submitting for: Clinical Vignette

Abstract Title A Slow and Steady Case of Osteomyelitis, the Turtle is to Blame.

Abstract Text
Often included at the end of the differential list, certain species of Non-tuberculous Mycobacteria (NTM) are known to cause soft tissue infections and osteomyelitis. In such cases a thorough social history is the key to making the diagnosis.

A 39-year-old man presented to the emergency department with a painful, swollen, and erythematous 3rd finger of his left hand. The patient sustained a ground level mechanical fall a few weeks prior to presentation. Initial X-rays and MRI confirmed a fracture of the proximal phalanx and extensive surrounding soft tissue induration suggesting both osteomyelitis and cellulitis. The patient underwent bedside incision and drainage. Culture of the purulent fluid showed no growth and he was discharged on Augmentin for empirical treatment of osteomyelitis.

He returned the next day due to worsening pain that was extending up his arm. Work up showed an elevated CRP at 35.3 μg/mL and elevated ESR at 69.0 mm/hr. He was admitted and started on intravenous vancomycin and piperacillin/tazobactam. Cultures from the initial incision and drainage showed rare gram-positive rods that failed to grow on the culture. He underwent surgical irrigation and debridement of left 3rd finger. No purulent fluid was noted intraoperatively. Bone and swab cultures were collected. The patient was discharged on intravenous ceftriaxone and oral doxycycline for six weeks. Final cultures isolated no organism and he completed the antibiotic regimen. At follow up appointments with Orthopedic Surgery, he reported residual left finger pain but stated overall improvement. Two months after his initial presentation the patient returned to the ED with similar complaints of left 3rd finger pain and swelling. Repeat imaging was highly suggestive of ongoing osteomyelitis. During this hospitalization it was discovered that the patient owned a bearded dragon and a turtle and that he commonly cleaned their enclosures with his bare hands. A second surgical debridement was performed. Both acid fast bacilli and fungal cultures were obtained in addition to routine cultures. These cultures eventually grew Mycobacterium chelonae (M.chelonae). He underwent amputation of the left 3rd finger through the metacarpophalangeal joint. Pathology showed residual chronic osteomyelitis and he was started on azithromycin, omadacycline, and imipenem for treatment of M.chelonae osteomyelitis.

Persistent osteomyelitis that fails to respond to broad spectrum antimicrobial therapy should raise suspicion for NTM osteomyelitis. Osteomyelitis due to NTM can occur within weeks of trauma, has an indolent course and persistent pain might be the only presenting symptom. Diagnosis is made by imaging and culture. Treatment involves surgical debridement followed by combination therapy with a macrolide. This case demonstrates the importance of good history taking and acts as a reminder to return to the patient for more information when the clinical picture is askew.
A new abstract was submitted for the Nebraska Chapter on Thursday, September 17, 2020 - 12:02:

Resident Abstract 27

Category Submitting for: Research

Abstract Title  Trimetazidine Reduces Contrast-Induced Nephropathy in Renal Insufficiency Patients Undergoing Coronary Angiography & Angioplasty

Abstract Text
Introduction: Contrast-induced nephropathy (CIN) is a serious complication of diagnostic and interventional angiography procedures, especially for higher risk patient populations undergoing coronary angiography. The incidence of CIN ranges from 1.6-2.3% in diagnostic interventions, to as high as 50% in high risk patients undergoing coronary intervention. This Systematic Review and Meta-Analysis assesses the utility of Trimetazidine (TMZ) to prevent contrast induced nephropathy (CIN) in patients with renal insufficiency undergoing coronary angiography and angioplasty.

Materials and Methods: This meta-analysis was formulated and reported according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines. A search of databases was conducted by two researchers independently for clinical trials, comparing hydration plus TMZ versus conventional hydration alone for the prevention of CIN through January 2020. Trimetazidine (TMZ) is a fatty acid oxidation inhibitor with cardio-protective effects used in some countries as an anti-anginal medication. All patients had renal insufficiency (defined as GFR< 89 mL/min/1.73 m2). The outcome of interest was the incidence of CI-AKI. The Odds Ratio (OR) was estimated with 95% confidence interval (CI). Heterogeneity was reported with the I2 statistic, using a fixed-effects model, and >50% of I2 was considered to be statistically significant.

Results: Eleven studies (1,611 patients) met the inclusion/exclusion criteria: 797 patients comprised the TMZ plus hydration group and the remaining 814 patients comprised the control (hydration only) group. Heterogeneity was low, I2=0%, P=0.84, and the heterogeneity of each study was also low. The incidence of CIN in the TMZ plus hydration group was 6.6% (53/797), while the incidence of CIN in the control (Hydration only) group was 20% (165/814). Pooled analysis of all studies showed TMZ reduced incidence of CIN compared to saline hydration alone (Odds Ratio: 0.30, 95% confidence interval [CI]: 0.21, 0.42, P<0.0001).

Conclusion: Combined data from 11 randomized control trials showed that TMZ added to standard normal saline hydration consistently reduced the incidence of CIN by 3-fold in high risk patients with renal insufficiency undergoing angioplasty or angiography.
A new abstract was submitted for the Nebraska Chapter on Thursday, September 17, 2020 - 21:22:

Resident Abstract 28

**Category Submitting for:** Clinical Vignette

**Abstract Title** Radial Artery Perforation: A Rare Complication of Left Heart Catheterization

**Abstract Text**
Radial Artery Perforation: A Rare Complication of Left Heart Catheterization

**Introduction:**
Trans-radial catheterization (TRC) is a widely accepted approach for coronary angiography in most parts of the world due to its lower incidence of procedure related complications compared to Trans-femoral approach. However, although rare, herein we present a case of radial artery perforation.

**Case Presentation:**
67 years old female with past medical history of essential hypertension, coronary artery disease, obesity, dyslipidemia, and unstable angina was undergoing a coronary angiogram when right radial artery injury occurred secondary to vasospasm. There was difficulty in advancing a 6 French catheter and when removed, a sheath injection demonstrated a patent artery with extravasation of contrast into the arm. She developed petechial rash of the right arm, swelling and numbness of the thumb and index finger. There were no signs of compartment syndrome. Vital signs remained stable during the procedure.

**Treatment:**
Patient had a sphygmomanometer cuff placed on the right arm and inflated above the systolic pressure to occlude blood flow. Sheath was removed, and a Vasc-Band was applied over the right radial artery. She had complete resolution of symptoms of radial artery perforation, including resolution of hematoma, numbness and tingling at one month follow up.

**Discussion:**
Radial artery perforation is an uncommon complication of TRC, comprising about 0.1%–1.0% of

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cases. It often presents with pain and spasm at the site of perforation, and can progress to a hematoma, pseudoaneurysm, and in rare cases arteriovenous fistula formation. It can potentially progress to severe unrecognized compartment syndrome requiring emergency fasciotomy. Previously described risk factors for RAP include female gender, short height, hypertension, excessive anticoagulation and aggressive wire manipulation, some of which were also present in our patient (female sex, Hypertension).

The management of RAP depends on the patient’s hemostatic response and includes conservative methods like mechanical compression (manually or with sphygmomanometer cuff) and reversal of anti-coagulation. Prolonged balloon inflation can be used as the next step if hemostasis is not achieved with conservative management. Open repair by endovascular surgery or covered (Polytetrafluoroethylene) stents, are used when all the previous methods have failed.
A new abstract was submitted for the Nebraska Chapter on Thursday, September 17, 2020 - 22:22:

Resident Abstract 29

**Category Submitting for:** Clinical Vignette

**Abstract Title** Spring Break: boats, blood clots, and Lemierre's syndrome

**Abstract Text**
Intro. Lemierre's syndrome is a rare sequela of bacterial infections which is typically not at the top of most physician's differential. However, it carries a mortality rate of about five to eighteen percent. This, along with its preference for young healthy people, makes Lemierre's syndrome an important disease to remember in the setting of bacteremia.

Case Report. A 20-year old male presented with complaints of fever/chills, shortness of breath, and right lower extremity pain for one-week duration. He had a history of sore throat, myalgias, and fever for three days two weeks ago and a cut to the lower leg two weeks before that. He had no significant past medical history. On physical exam, he was hypotensive, tachycardic with a temperature of 100.2°F, and SpO2 of 78%. His right thigh was tender to palpation. He was emergently intubated. Laboratory studies revealed leukocytosis, elevated creatinine, lactic acidosis, thrombocytopenia, mildly elevated AST, hyperbilirubinemia, and hypoalbuminemia. Blood cultures showed growth of Fusobacterium necrophorum. An abscess was found in the right vastus intermedius with myonecrosis as well as a right knee effusion. He received Vancomycin and Piperacillin-Tazobactam; the latter was replaced with Meropenem and Doxycycline. Knee fluid analysis showed a high amount of RBCs, WBCs, culture negative. During his hospital course, his respiratory status worsened. Chest CT showed diffuse bilateral lung consolidations with right lower lobe cavitation. He also had swelling in his neck. Neck duplex ultrasonography showed IJV thrombosis. After one week of admission, the patient had shown clinical improvement and was extubated. An MRI showed myositis, myonecrosis, and femoral osteomyelitis. Ultrasound of the right leg showed a DVT, which prompted anticoagulation with Apixaban. He received a total of 33 days of antibiotics. On follow up, MRI showed progression of acute myositis. He received IV Piperacillin-Tazobactam and Daptomycin. Subsequent X-ray and MRI of the right femur showed improvement. At the latest clinical

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encounter, the patient reported an activity level back to baseline.

Discussion. In the era of antibiotics, Lemierre's syndrome has become a rarer sequela of bacterial infections but can still have deadly consequences. The bacteria, which initially causes pharyngitis, spreads into the carotid sheath and forms a septic emboli. The diagnosis is made by a clinical picture revealing a combination of sore throat, bacteremia, and internal jugular vein thrombosis. In our case, the diagnosis was made after an ultrasound of the neck was ordered due to swelling of the neck. The preferred diagnostic test would be a contrast CT neck showing a clot in the internal jugular vein. Due to beta-lactamase producing bacteria, the preferred antibiotic regimen includes ampicillin-sulbactam or piperacillin-tazobactam. Anti-coagulation is not always necessary. This decision should be made based on clot size.
A new abstract was submitted for the Nebraska Chapter on Friday, September 18, 2020 - 05:57:

Resident Abstract 30

**Category Submitting for:** Clinical Vignette

**Abstract Title** Fever in a returning traveler: A case of Dengue fever

**Abstract Text**

Dengue is a leading cause of acute febrile illness among travelers returning from Latin America, the Caribbean, and Southeast Asia. Even in patients with compatible clinical presentation, serology might be falsely negative during initial stages of illness and may lead to missed diagnosis of disease. This case highlights the importance of obtaining repeat serology if the initial test was negative early in the disease course. A 27-year-old woman contacted her primary care provider due to two days of fever, chills, nausea, diarrhea, “bone pain”, and “sun burn rash” over her extremities. The patient recently returned from a trip to Thailand. Initial work up indicated leukopenia of 1.9K/UL, thrombocytopenia of 83K/UL, transaminitis with aspartate aminotransferase levels (AST) at 109U/l and alanine aminotransferase (ALT) of 71U/l, and a negative Dengue IgM and IgG, titer 1 IV and 0.88 IV respectively. She was prescribed ciprofloxacin and diphenoxyl ate-atropine for empirical treatment of gastroenteritis initially. Rash worsened while on ciprofloxacin and oral diphenhydramine was started for symptoms relief.

Follow up in primary care clinic showed progression of rash from the patient’s head to bilateral lower extremities. She continued to be febrile and was admitted to a local hospital for further testing and observation. Repeat Dengue IgG and IgM was performed five days after initial negative testing indicating positive titers: IgM 38.12 IV and IgG 3.32 IV confirming the diagnosis of Dengue fever. IV hydration, antipyretics, daily complete blood counts, liver function tests, and complete metabolic panels were conducted. Transaminases stabilized with AST at 93U/l, ALT at 88U/l, hemoglobin at 12.1gm/dl, and thrombocytopenia improved with platelets rising from 36K/UL to 61K/UL by day of discharge without signs of hemorrhagic or shock conversion of Dengue fever. Follow up with Infectious Disease clinic four weeks after initial presentation showed resolution of fever, rash, leukopenia, thrombocytopenia, and transaminitis. This case illustrates the need to maintain a high index of suspicion for Dengue fever in cases of fever with rash in a returning traveler if they were in an endemic area within two weeks of symptom onset. IgM against Dengue virus can be detected with ELISA >4 days after fever onset and can be negative prior to this period. Patients presenting in the first week after fever onset should receive RT-PCR or NS1 testing in addition to IgM. In patients presenting >1 week after fever onset, IgM test alone is most useful. It is imperative that a thorough history be conducted and in the presence of high clinical suspicion of Dengue fever, negative initial testing should not dissuade the possibility of diagnosis. Repeat testing for confirmation is warranted in these clinical settings.
A new abstract was submitted for the Nebraska Chapter on Tuesday, September 15, 2020 - 23:42:

Resident Abstract 31

**Category Submitting for:** Clinical Vignette

**Abstract Title** Atypical chest pain as an unusual presentation of Implantable Cardioverter-Defibrillator (ICD)

**Abstract Text**

**Background:** Implantable cardioverter-defibrillators (ICDs) remain the mainstay intervention for prevention of sudden cardiac death. Commonly reported complications of ICDs are wound infection, lead displacement, lead fracture, pneumothorax, hemothorax and uncommonly, lead perforation. Here we present a rare case of atypical chest pain after displacement of ICD.

**Case:** A 54-year-old caucasian male with past medical history of heart failure with reduced ejection fraction (30-35%) and S-ICD placement for ventricular tachycardia (VT) presented to the emergency department with acute asthma exacerbation complicated by sudden onset, severe (8/10) left sided chest pain. Vitals were normal; EKG showed no ischemic changes. Troponin, D-Dimer and pro-BNP levels were within normal limits. Despite symptomatic import emend of asthma, patient continued to have frequent, episodic sub-sternal, left sided chest pain, 9/10 in intensity, radiating to the back. Patient was diaphoretic, tachypneic, dyspneic, hypoxic and had non sustained VT. Pain improved on sublingual nitroglycerin and morphine, and patient was started on heparin drip as per Acute Coronary Syndrome (ACS) protocol. However, EKG remain unchanged with each episode, as did troponin levels. CTPA was done which ruled out medical emergencies including pulmonary embolism and aortic dissection. Upon detailed examination, it was noticed that there was displacement of patient’s ICD from left anterior axillary line to his back, corresponding to the site of pain, with tenderness elicited over the area. CXR confirmed displacement of ICD. Subsequently, ICD was explanted which resulted in resolution of the chest pain. New VVI ICD was implanted thereafter.

**Discussion:** ICDs are used for secondary prevention in patients with an episode of sustained ventricular arrhythmia, for primary prevention in patients with risk factors for sudden cardiac death, and for cardiac resynchronization therapy (CRT) in patients with dyssynchronous ventricles. ICDs monitor rhythms to detect life threatening ventricular tachyarrhythmias and provide therapy in the form of anti tachycardia pacing (ATP). Even though ICD placement is invasive, it is safe but still can complicates in less then 1% of the population who underwent ICD placement.

**Conclusion:** Displacement of ICD is an uncommon cause of atypical chest pain that may be considered in patients with ICD in situ in the absence of other explanations for intractable chest pain once emergent conditions including ACS, PE, aortic dissection and tension pneumothorax have been ruled out.
A new abstract was submitted for the Nebraska Chapter on Wednesday, September 16, 2020 - 16:04:

**Resident Abstract 32**

**Category Submitting for:** Clinical Vignette

**Abstract Title** The Forgotten and missed during COVID-19

**Abstract Text**
A 30 year-old man with history of low back pain treated with hydrocodone-acetaminophen presented with a several day history of rib pain with malaise, pleuritic chest pain, and shortness of breath without fevers during the COVID-19 pandemic. He denied having any known sick contacts. He had hypotension, tachycardia, tachypnea. The JVP was not elevated, but there were crackles in the bilateral lung fields. Labs revealed neutrophilic predominant leukocytosis of 16.8, platelets 11, hemoglobin 10.4, blood urea nitrogen 106, creatinine 4.05, aspartate aminotransferase 70, alanine aminotransferase 22, alkaline phosphatase 437, lactic acid 2.3. Computed tomography chest showed nodular and ground-glass opacities within both lung lobes consistent with multifocal pneumonia and mild bilateral pleural effusions. Computed tomography chest angiogram did not show evidence of pulmonary embolism but did show multiple pulmonary nodules with associated cavitation. Blood culture returned positive with *Fusobacterium necrophorum*.

On further questioning, he denied recent dental pain, ear pain, sore throat, neck pain, or recent dental procedures. An upper extremity ultrasound did not show echogenic material in the jugular or subclavian veins.

With multifocal ground-glass opacities initially observed on imaging, there was concern for COVID-19 infection. The patient did not tolerate nasopharyngeal testing, and as a result was kept in isolation with airborne precaution. He was discharged with a 14-day course of IV ceftriaxone 2g/day and metronidazole 500mg TID. Unfortunately, he returned to the hospital several days later with increased shortness of breath and chest discomfort. Repeat chest imaging showed scattered nodular opacities and worsening bilateral effusions with atelectasis. Thoracentesis was performed and the fluid was characterized as exudative. The patient required placement of several pigtail catheters near the left lung base to drain the effusions and

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pulmonary cavitary lesions. With continued antibiotics and drain placement, the patient improved and was discharged with a prolonged course of metronidazole 500mg TID.

Fusobacterium necrophorum bacteremia usually originates from a prior dental infection. If it spreads along oral tissue planes it can cause internal jugular vein septic thrombophlebitis; also known as Lemierre’s syndrome. The bacteria causes platelet aggregation, destruction of red and white blood cells, and bacterial toxins which can have a direct toxic effect on the liver. Our patient did not endorse a history of sore throat, dental pain, or ear pain, which has been noted in prior case reports of Fusobacterium necrophorum bacteremia but is unusual. Patients may note a remote history of sore throat which may precede their presentation by several months. Additionally, it is important to remember pain involving the fifth cranial nerve can be very nonspecific and does not always allow for localization. Lastly, NSAIDs or other pain medications can mask dental-related pain or sore throat symptoms patients may have.
A new abstract was submitted for the Nebraska Chapter on Wednesday, September 16, 2020 – 17:52:

Resident Abstract 33

**Category Submitting for:** Clinical Vignette

**Abstract Title** Appendicitis Causing Plaque Rupture - An Unusual STEMI Presentation of Appendicitis

**Abstract Text**
Appendicitis Causing Plaque Rupture - An Unusual STEMI Presentation of Appendicitis

**Introduction:**
Acute surgical abdomen is noticed to show EKG changes. This case highlights the relation between acute systemic event triggering plaque rupture, leading to STEMI

**Case description:**
Our patient is a 65-year-old Caucasian male with a past medical history of NSTEMI, 10 years back with status post DES in RCA and hypertension. He presented to us from the outside emergency department (ER) for an emergent PCI (percutaneous coronary intervention) after being found to have inferior STEMI (ST elevation myocardial infarction) and elevated troponin of 14.0. He presented to the ER with generalized abdominal pain, diaphoresis and sob. He required 5 L of O2 (oxygen) on initial presentation. Patient had leukocytosis at 15.9k/ul. His EKG showed significant ST elevations in inferior leads. Patient was taken emergently to the catheterization lab. Total occlusion of the dominant RCA (right coronary artery) was seen in proximal to mid segment. Collateral circulation was noted from the left coronary system. DES (drug eluting stent) of 2.75 x 24 mm was placed in acutely occluded proximal to mid RCA. Patient was started on DAPT (dual anti-platelet therapy): Aspirin and Ticagrelor. Post PCI, the EKG STEMI findings have resolved. Patient complained of abdominal pain still persisting and worsening. His leucocyte count is up to 24.6k/ul, lactic acid is 1.7mmol/ and blood cultures were drawn which later showed no growth. Abdominal CT showed severe appendicitis. He is continued on the DAPT. He was found to have a gangrenous appendix which was removed.

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Post appendectomy, his pain was relieved, his leucocyte count trended down and was discharged the next day.

Discussion:
Appendicitis is systemic inflammatory process. Cytokines and interleukins like IL-1, IL-6, IFN- and TNF- are studied to be significantly higher in serum of patients with appendicitis. These inflammatory markers are also significantly elevated in STEMI patients. Animal studies have shown that pro inflammatory cytokines can cause plaque instability leading to rupture. These inflammatory markers like IL-1, IL-6, IFN- and TNF- promote apoptosis and are studied to enlarge the lipid core, inhibit plaque stabilizing factors and matrix metalloproteinases. These factors ultimately can cause plaque rupture. This could serve as a hypothesis as why an inflammatory process such as appendicitis could lead to a STEMI.
Resident Abstract 34

**Category Submitting for:** Clinical Vignette

**Abstract Title** Lymphadenopathy, night sweats, and fever in a 35 year old; a diagnosis not TB overlooked

**Abstract Text**
A 35-year-old man presented with 3-week history of fever, night sweats, and prominent lymphadenopathy. He also described 10-pound weight loss over the past two months. On physical examination, patient had left cervical chain nontender lymphadenopathy with largest node being 3cm over the sternocleidomastoid. Computed tomography of the neck revealed prominent lymphadenopathy in the cervical chain with central cystic changes or necrosis. The patient had significant elevation of erythrocyte sedimentation rate of 71 mm/hr, a positive interferon gamma release assay, negative HIV panel, and positive blood cultures for Micrococcus sp., a likely contaminant. On further questioning, the patient had described recent immigration from India within the last 4 months, as well as recent rat bite on his left hand. Cervical lymph node biopsy demonstrated acid fast staining bacteria identified as Mycobacterium tuberculosis. The patient was treated with rifampin, isoniazid, ethambutol, and pyrazinamide. He initially had good response to therapy with subsequent cervical lymph node enlargement and sinus tract formation. He was continued on tuberculosis therapy in addition to steroids with resolution of his symptoms.

In the US general patient population, the constellation of several weeks of fever, night sweats, lymphadenopathy, and weight loss in a 30-year-old is most concerning for lymphoma; however this is not the case in all parts of the world. Tuberculous lymphadenitis is one of the most common extrapulmonary manifestations of tuberculosis. Most cases of tuberculosis lymphadenitis in developed countries occur in immigrants from TB endemic countries as demonstrated by our case. Tuberculosis lymphadenitis is typically due to reactivation of disease. Patients typically present with unilateral lymphadenopathy (most commonly cervical lymphadenopathy). Patients may also develop constitutional symptoms as our patient did. If active pulmonary disease is ruled out, airborne isolation is unnecessary. Tuberculous lymphadenitis is treated with rifampin, isoniazid, ethambutol, and pyrazinamide for two months followed by four additional months of rifampin and isoniazid. A paradoxical reaction with increased lymphadenopathy occurs in ~20% of patients and does not indicate treatment failure, but rather immune response to dying mycobacteria. Addition of steroids can help alleviate the paradoxical reaction. In order to accurately treat patients in our diverse nation, it is important to keep global infections such as tuberculosis and its many manifestations on the differential.
A new abstract was submitted for the Nebraska Chapter on Wednesday, September 16, 2020 - 22:50:

Resident Abstract 35

**Category Submitting for:** Research

**Abstract Title** Restricted Versus Liberal Oxygen Treatment For Patients' with A on C Respiratory Failure with Hypercapnia Requiring Noninvasive Ventilation

**Abstract Text**
Outcome of Restricted Versus Liberal Oxygen Treatment For Patients' with Acute on Chronic Respiratory Failure with Hypercapnia Requiring Noninvasive Ventilation

**Purpose**
To describe the current practice and outcome of oxygen treatment for patients with acute on chronic respiratory failure with hypercapnia (ACRFH) who are admitted to the ICU requiring non-invasive ventilation (NIV).

**Methods**
In this retrospective study; identified patients admitted to mixed (surgical and medical) 54-bed ICU with ACRFH. Data retrieved: demographics, underlying etiology for ACRFH, ABGs, complications and mortality. We calculated the mean and SD when appropriate. P value less than 0.05 was considered significant.

**Results**
205 patients included in the study. Patients’ mean age 67.1 (SD - 13.5) years, 94 (46%) were female, and 143 (70%) had underlying COPD. There were 42 patients (20%) initially within the restricted range of oxygen treatment (PO2 <63). 188 patients (91%) had a follow up ABG while on NIV; 33 (18%) of those patients remained or moved to the restricted range of oxygen treatment. There was no significant difference in PCO2 between the two groups 71.3 mmHg versus 74.8 mmHg on admission respectively. The group of patients who remained or moved to restricted oxygen treatment range had lower PCO2 compared to the liberal oxygen group: 63.91 mmHg vs 69.94 mmHg. There was no difference in NIV pressure, duration of treatment, or rate of those requiring intubation. Mortality and intubation rates were not independently related to the oxygen treatment group.

**Conclusion**
Restricted oxygen treatment during NIV is associated with lower PCO2 in patients with ACRFH.

**Clinical Implications**
Prospective study needed to identify the proper dose of oxygen while treating patients with ACRFH.
A new abstract was submitted for the Nebraska Chapter on Wednesday, September 16, 2020 - 22:23:

Resident Abstract 36

**Category Submitting for:** Clinical Vignette

**Abstract Title** Acute Sensory-Motor Axonal Neuropathy in a 57-year-old male presenting with paresthesia and distal muscle weakness.

**Abstract Text**

**Introduction**

Guillain-Barré Syndrome (GBS) is a relatively uncommon post-infectious immune-mediated monophasic neurologic disorder with an incidence of 0.5-2/100,000 characterized by demyelination of spinal nerve roots and peripheral nerves, with variable recovery and resolution. Diagnosis is made by clinical evaluation, cerebrospinal fluid (CSF) analysis, and nerve conduction studies. CSF albuminocytological dissociation is a classic finding. Immunoglobulin (IVIG) administration or plasmapheresis with supportive care is the standard treatment. Variants of GBS are classified by the type of nerve fiber involved, mode of injury, and presence of altered consciousness. Although GBS presents as acute flaccid paralytic polyradiculoneuropathy, these variants often do not fulfill diagnostic criteria for classical GBS. The case we describe is of a 57-year old male presenting with sensory features followed by symmetrical ascending paralysis, diagnosed as Acute Sensory-Motor Axonal Neuropathy (ASMAN).

**Case Presentation**

A 57-year old male with no significant past medical history presented to the emergency department with the complaint of bilateral lower extremity numbness and tingling for two days, which was gradual in onset, progressive, and associated with the weakness for one day. He denied recent gastrointestinal or respiratory illness.

On clinical examination, the patient was afebrile, alert, and oriented to time, place, and person. Blood pressure (120/75 mmHg), pulse rate (82/minute), and respiratory rate (20/minute) were normal. The neurologic examination demonstrated generalized hyporeflexia, decreased power (3/5) in the lower limbs bilaterally, and gait ataxia. Despite unremarkable initial laboratory

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workup, a diagnosis of lower motor neuron paraparesis was made. The Magnetic Resonance Imaging helped rule out a cerebrovascular accident. During his hospital course, his lower extremity weakness worsened and was followed by sensations of numbness and tingling in the bilateral upper extremities. Further workup showed albuminocytologic dissociation on CSF analysis concerning for GBS. IVIG and supportive care were initiated with subsequent improvement in his neurologic symptoms. Physical and occupational therapy support aided discharge after 2 months when he was able to walk with assistance.

Discussion

ASMAN is a recently described subtype of GBS characterized by acute onset of sensory symptoms with loss of deep tendon reflexes and distal weakness. Electrophysiologic studies show mildly reduced nerve conduction velocities in axons, combined with a marked reduction of muscle action and sensory nerve action potentials. Patients with AMSAN typically experience severe symptoms over a short time period, also experiencing prolonged and incomplete recovery despite treatment with immunomodulatory drugs. The therapy of IVIG or plasmapheresis is based on that of the acute inflammatory demyelinating polyradiculoneuropathy. The benefit of steroids remains ambiguous. Although rare, inflammatory polyradiculoneuropathy should be considered in the differential diagnosis of acute flaccid quadriparesis with length-dependent sensory dysfunction.
A new abstract was submitted for the Nebraska Chapter on Thursday, September 17, 2020 - 09:49:

Resident Abstract 37

**Category Submitting for:** Clinical Vignette

**Abstract Title** Chronic Cough: When To Think Aspergillosis?

**Abstract Text**
Chronic Cough: When To Consider Aspergillosis?
Chronic cough is a common problem encountered by internists. Etiologies include GERD, post nasal drip, ACE inhibitor cough, asthma, and COPD. However, specific patient populations and clinical scenarios require broader reasoning, including chronic infections such as chronic aspergillosis and allergic bronchopulmonary aspergillosis (ABPA). Although chronic aspergillosis is an uncommon cause of chronic cough, particularly in immunocompetent patients, identification is crucial as the treatment course differs from other causes of chronic cough.

A 73 year old male with a history of DM, SVT, HTN, pulmonary nodules, OSA, asthma-COPD overlap syndrome, and pulmonary aspergilloma status post right upper lobectomy in the 1970’s presented to the clinic for evaluation of chronic cough in 2020. The chronic cough had persisted for multiple years but worsened over the past 5 months and now produces a rubbery “plug” every few days. The patient denied any fevers, chills, sinus congestion, rhinorrhea, chest pain, leg swelling, dyspnea on exertion, dyspepsia, or weight loss. He had no known history of chronic granulomatous disease, glucocorticoid use, or prolonged neutropenia. The patient had been without therapy for COPD/asthma overlap for many years until two months prior to presentation when he began using budesonide-formoterol BID and albuterol due to worsening of his chronic cough. Sputum culture grew Aspergillus and the patient provided photos of the “plugs” resembling bronchial casts. Chest x-ray revealed interstitial thickening and bronchiectasis in the left upper lobe. Chest CT scan revealed left upper lobe multifocal opacities consistent with pneumonia and antecedent granulomatous disease, but did not reveal any cavitary lesions or nodules suggesting against cavitary aspergillosis. Aspergillus antibodies were negative and total IgE was normal which ruled out ABPA. Despite negative Aspergillus antibodies clinical history, imaging, sputum culture results, and laboratory findings supported a

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diagnosis of chronic aspergillosis rather than ABPA or cavitary aspergillosis and antifungal therapy was initiated.

This case illustrates when to broaden differential of chronic cough and when to have a high degree of suspicion for aspergillosis and the need to distinguish between chronic aspergillosis and ABPA as ABPA would require treatment with a prolonged course of steroids.

A new abstract was submitted for the Nebraska Chapter on Monday, September 7, 2020 - 21:58:

Resident Abstract 38

**Category Submitting for:** Clinical Vignette

**Abstract Title** COVID-19 concomitant infection with infective endocarditis and pyogenic abscess: case report and literature review

**Abstract Text**

Introduction: Coronavirus disease (COVID-19) is a global pandemic and public health crisis that has caused over 900,000 deaths to date. Although our understanding of the virus has grown, cardiac complications remains poorly understood. Here we present an unusual case of a COVID-19 patient who presented with concomitant bacterial endocarditis and associated pyogenic abscess.

Case: A 62-year-old African American male with history of aortic and mitral stenosis, presented to the ED with exertional SOB, abdominal pain, low-grade fever (99.5F), night sweats, and weight loss. Patient lost 15lbs in the past month with recent travel history to South Africa. Patient denied nausea/vomiting, chest pain, PND, orthopnea, peripheral edema, IV drug abuse, or recent dental procedures. Apart from HR of 115, vitals were within normal limits. Physical exam showed tenderness in the left upper quadrant, bilateral rhonchi, pan-systolic murmur in the aortic area, and splinter hemorrhage in the index and middle fingers of both hands. Laboratory investigations showed Hgb 10 g/dl, WBC 21,300 cells/mm³, alkaline phosphatase 278U/L, total bilirubin 2.6 mg/dl (direct 2.0 mg/dl), procalcitonin 2.51ng/ml, CRP 316 mg/l, ESR 95 min/hr, Lactic acid 3.5 mmol/L, LDH 600 U/L, and Ferritin 359 ng/ml. Chest CT scan showed extensive bilateral consolidations consistent with multifocal pneumonia. Abdominal CT scan showed a thick-walled 7.7x5x6.4cm diameter consolidation in the left liver lobe. Echocardiogram showed 1.02x0.88cm density at the posterior mitral valve leaflet suggestive of vegetation, confirmed later by TEE. Patient was started on broad spectrum antibiotics; subsequent blood cultures on day 3 showed Eikenella corrodens and Streptococcus anginosus growth. COVID-19 PCR testing returned positive for infection. Patient was switched to 500mg qd Azithromycin, 500mg tid Metronidazole, 4.5g qid Piperacillin/Tazobactam, and 2.5mg qd lisinopril. Dexamethasone and Remdesivir were held due to normal oxygen saturation. Interventional

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radiology drained abscess. Patient improved over 2 weeks and was discharged with follow-up. Discussion: Cardiac involvement in COVID-19 can vary in severity and presentation with ACS, myocarditis, cardiac arrhythmia, and endocarditis being cited in the literature. In patients with co-morbidities including age >60 years, obesity, hypertension, and diabetes, symptoms can have worsening severity. According to Shang et al., the ability of the SARS-CoV-2 to enter by target ACE2 epithelial receptors is hypothesized to explain its diverse organ involvement. Systemic inflammatory response by cytokines are also thought to be involved. ACEI and ARBs have shown promising results in cardiovascular patients possibly due to the ACE2 epithelial receptor involvement. Although countless case reports have described COVID-19, few examples of bacterial endocarditis have been seen and no others describing pyogenic abscess besides the one herein described. Conclusion: The mechanism of cardiac complications including bacterial endocarditis in COVID-19 patients remains poorly understood, subsequent studies will be helpful in elucidating the association and proper management of patients.
A new abstract was submitted for the Nebraska Chapter on Wednesday, September 9, 2020 - 21:31:

Resident Abstract 39

Category Submitting for: Clinical Vignette

Abstract Title Rhabdomyolysis and renal failure following Cardiopulmonary Resuscitation: A Case Report

Abstract Text
Introduction
Rhabdomyolysis is a condition where there is damage of skeletal muscle, causing myoglobin leak into the circulation. Very few reports of rhabdomyolysis causing renal failure following cardioversion have been described. This report describes a case of myoglobinuric renal failure with following cardioversion requiring hemodialysis.

Case presentation
A 69-year-old female with a history of hypertension, paroxysmal atrial fibrillation and chronic kidney disease with baseline creatinine of 1.3-1.9 was found unresponsive at home and EMS was called. En-route to the ER, initial rhythm showed ventricular fibrillation. The patient received CPR for 45 minutes; direct current cardioversion counter shocks of 300 - 360 J were given to restore effective cardiac rhythm. After the third attempt of defibrillation she converted to sinus rhythm. She was then transferred to the intensive care unit and was put on mechanical ventilation. Cardiac catheterization could not be performed due to her critical condition. Echocardiography of the heart did show motion wall abnormality of basal, mid anterior and inferior wall suggestive of underlying myocardial infarction. On the second day of her hospitalization, she started becoming oliguric without hypotension and her creatinine started rising up causing acute kidney injury. Her UA showed no RBCs, pyuria or bacteria, but did show some granular casts. A renal ultrasound was negative for obstruction, and her fractional sodium was found to be 4.5%. The patient's CK level peaked at 6380 u/l (normal range 26-192 u/l), myoglobin was >20,000 ng/ml (normal range 9-83 ng/ml) and myocardial bound (MB) isoenzyme of CK was 4.5ng/ml (normal range 0-3.6ng/ml). Plasma creatinine increased to 5.71mg/dl. She was started on hemodialysis. However, her kidneys continued to show minimal renal recovery and patient was discharged to a long-term care facility with the plan for long term hemodialysis.

Summary
Rhabdomyolysis can lead to the development of acute kidney injury (AKI), caused by the release of myoglobin from the muscle. Very few reports have been described rhabdomyolysis after cardioversion. The rise in creatine kinase, CK-MB and myoglobin after cardioversion and peak level strongly with the total energy delivered and peak energy. Our case illustrates that cardioversion delivered for the treatment for refractory ventricular fibrillation can cause muscle damage causing myoglobinuric renal failure.
A new abstract was submitted for the Nebraska Chapter on Sunday, September 13, 2020 - 13:51:

Resident Abstract 40

**Category Submitting for:** Clinical Vignette

**Abstract Title** A Rare Case of Anti Synthetase Syndrome Presenting As Rapidly Progressing Interstitial Lung Disease

**Abstract Text**
A Rare Case of Antisynthetase Syndrome Presenting As Rapidly Progressive Interstitial Lung Disease

Introduction:
Anti-synthetase syndrome is a rare connective tissue disorder characterized by antibodies directed against aminoacyl tRNA synthetase (ARS) with a constellation of findings that may include interstitial lung disease, myositis, Raynaud's phenomenon, and arthritis. We present the case of a newly diagnosed anti-synthetase syndrome presenting with rapidly progressing interstitial lung disease.

Case description:
A 67-year-old caucasian female with a past medical history significant for heart failure and esophageal dysmotility presented to the hospital with complaints of cough and dyspnea that progressively worsened over the last 4 months. She was unsuccessfully treated multiple times with outpatient antibiotic therapy in the last 4 months prior to this admission. Chest x-ray on presentation showed opacification of bilateral mid and lower lung fields and a CT chest with contrast done showed diffuse bilateral patchy ground-glass opacities associated with confluent

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airspace densities. Treatment was initiated with Broad-spectrum antibiotics and high dose corticosteroids. The patient's respiratory status rapidly declined within 2 days of hospitalization to respiratory failure requiring mechanical ventilation. Laboratory tests were pertinent for mildly elevated CK at 332, ESR 55, and CRP 77.5. Immunology work-up revealed positive ANA, anti-Jo 1, and anti-RNP. Initial A muscle biopsy was done which showed myofiber atrophy with perimysial inflammation consistent with immune-related myopathy. She was diagnosed with Antisythetase syndrome based on her histological findings and autoantibodies. Due to her critical status and rapid decline, the patient was started on pulse steroids with 1000 mg IV methylprednisone to be given for 3 days followed by maintenance mycophenolate, to which she responded well.

Discussion:
Anti synthetase syndrome is a rare autoimmune disorder. The pathophysiology is not entirely understood but it is presumed that a possible viral infection or genetic predisposition might have triggered the formation of ARS which lead to the clinical syndrome. Among the 8 identified ARS, anti-Jo 1 is the most common and has a better prognosis than the other. Prednisone at the dose of 1 mg/kg/day followed by tapering doses showed promising results. In more severe cases, pulse dose steroids and steroid-sparing immunosuppressive therapy like calcineurin inhibitors, cyclophosphamide, mycophenolate mofetil, and rituximab have been tried with good success. In refractory cases, IVIG has been used.
A new abstract was submitted for the Nebraska Chapter on Sunday, September 13, 2020 - 16:09:

Resident Abstract 41

**Category Submitting for:** Clinical Vignette

**Abstract Title** Bevacizumab - induced Nephropathy

**Abstract Text**

**Introduction**

Bevacizumab is a VEGF inhibitor used to treat diabetic retinopathy. It has been reported to be associated with worsening proteinuria and hypertension. We present a case of a patient (on intravitreal bevacizumab) hospitalized with worsening renal function, hypertension and nephrotic-range proteinuria in the setting of tight glycemic control.

**Case Description**

A 41 year old male with a past medical history of CKD stage 3, Diabetes Type 2, Diabetic Retinopathy and Hypertension presented to the ED with shortness of breath for about a month. He reported lower extremity swelling for about a year that was refractory to lasix 80 mg bid. He reported about 60 lb weight gain in about a year. He had never seen a Nephrologist. His family history was significant for ESRD in his father. Physical exam was remarkable for bilateral lower extremity pitting edema and lung crackles. His BP in ED was 219/117 that improved on applying nitro patch. Labs revealed normal troponin, pro BNP 5759 pg/ml, d-dimer 0.72 mg/L and creatinine 2.15 mg/dl (baseline around 1.5 - 1.9 mg/dl). CXR showed bibasilar opacities and Lung V/Q scan showed low probability of pulmonary embolism. Renal ultrasound was unremarkable. His home medications were furosemide 80 mg bid and metoprolol 100 mg bid. He had been getting intravitreal bevacizumab injection for diabetic retinopathy for a bout a year. He was given IV Lasix with improvement in shortness of breath. His BP remained high. He was started on nicardipine drip and then switched to amlodipine, carvedilol and oral hydralazine with various adjustments in dosages.

Urinalysis revealed proteinuria (over 1000 mg/dl), microscopic hematuria and granular casts.

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HbA1c was 5.9% (improved from 8.3% prior). Further work up revealed normal C3 and C4 levels, negative ANA, c-ANCA and p-ANCA, unremarkable SPEP, and PLA2R antibody, negative serum cryoglobulin, elevated kappa-lambda ratio of 2.66, urine immunofixation negative for monoclonal protein, urine protein/creatinine ratio of 11.2. Given microscopic hematuria and substantial proteinuria, patient underwent left kidney biopsy that showed diabetic nephropathy and scattered IgA deposits (possibly due to bevacizumab).

BP remained difficult to control. He was discharged on bumex 2 mg bid, amlodipine 10 mg, carvedilol 25 mg bid and hydralazine 100 mg tid and furosemide and metoprolol were discontinued. His creatinine remained between 2.15 to 2.76 mg/dl in the hospital. He was not started on ACEi/ARB due to decline in GFR. He was scheduled to follow-up with Nephrology in clinic for revaluation.

Discussion

Our patient had nephrotic-range proteinuria with HbA1c of 5.9%.

This case illustrates that intravitreal bevacizumab injection may lead to worsening proteinuria, renal function and hypertension. Both physicians and patients should be aware of this possibility and alternative therapies for diabetic retinopathy should be considered when possible.
A new abstract was submitted for the Nebraska Chapter on Tuesday, September 15, 2020 - 17:24:

Resident Abstract 42

**Category Submitting for:** Clinical Vignette

**Abstract Title** Ethylene Glycol Poisoning with Near-Normal Osmolal Gap - A Diagnostic Challenge

**Abstract Text**

**Introduction:**

Ethylene glycol poisoning is classically associated with a high anion gap metabolic acidosis (HAGMA). Neurological and gastrointestinal symptoms predominate early while renal failure and death occur if not diagnosed and treated promptly. The diagnosis is usually suggested by HAGMA and an elevated serum osmolal gap in the setting of a suspected ingestion. Rarely, the serum osmolal gap may be close to normal which can delay the diagnosis or lead to a misdiagnosis. We report a case of ethylene glycol ingestion with near-normal serum osmolal gap.

**Case Description:**

An 85-year-old man with a past medical history of Dementia presented to the Emergency Department with altered mental status, restlessness and elevated creatinine of 1.4 mg/dl (baseline 1.2mg/dl). History was difficult to obtain. Vital signs were normal and the physical exam was remarkable only for altered mental status. CT scan of the head did not reveal any acute abnormality. Laboratory workup revealed HAGMA (anion gap = 21 mEq/L, arterial blood pH = 7.26, serum bicarbonate = 9.3 mmol/L, lactic acid = 2.2 mmol/L) with a near-normal serum

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osmolal gap (12 mOsm/kg). Urinalysis, urine drug screen, blood ethanol, beta-hydroxybutyrate, acetaminophen and salicylate levels were normal.

Given a high clinical suspicion for toxic alcohol ingestion, the patient was treated with IV fluids and fomepizole. Over the next few days, his mental status improved, and repeat laboratory workup demonstrated correction of the anion and serum osmolal gaps. Additional history obtained later from his family increased the suspicion for toxic alcohol ingestion. Ethylene glycol level, a send out lab, eventually resulted at an elevated level.

Discussion:

In our case, the ethylene glycol level was mildly elevated at 3.1 mg/dl indicating that either there was a substantial ingestion and the ethylene glycol had substantially metabolized already or it was a mild ingestion. As the result was not readily available, we opted to start fomepizole based on high clinical suspicion. This decision was also further supported by the substantial metabolic acidosis. The patient’s rapid improvement in mental status and avoidance of hemodialysis also justified our early use of fomepizole.

The workup for a HAGMA should include evaluation of the serum osmolal gap, particularly in the setting of a suspected toxic alcohol ingestion. Although uncommon, toxic alcohol ingestion can present with a normal or near-normal osmolal gap as this case illustrates. Accordingly, the absence of an elevated osmolal gap should not discourage treatment for toxic alcohol ingestion when the clinical suspicion is sufficiently high.
A new abstract was submitted for the Nebraska Chapter on Friday, September 18, 2020 - 21:14:

Resident Abstract 43

**Category Submitting for:** Clinical Vignette

**Abstract Title** Troponin leak: A puzzle

**Abstract Text**

Introduction: Elevated troponin in patients hospitalized with non-cardiac systemic illnesses is commonly attributed to demand ischemia. Clinicians should be careful not to always dismiss elevated troponin in such cases as type 2 myocardial ischemia (MI). We present a case of incidental type 1 MI in a patient admitted for pneumococcal pneumonia.

Case presentation: A 79-year-old female admitted with complaints of generalized weakness with initial workup showing leukocytosis, elevated procalcitonin, and a right lower lobe consolidation on chest X-Ray. Initial troponin was 0.1 ng/mL, which then peaked at 10 ng/mL. No ischemic changes were seen on electrocardiogram, and she remained without chest pain or shortness of breath.

Treatment: The patient underwent coronary angiography revealing 90% mid-LAD occlusion with successful drug-eluting stent placement in mid LAD. The patient was then started on goal-directed medical therapy for heart failure, as she was found to have an ejection fraction of 30%.

Discussion: Demand ischemia with elevated troponin is common in the setting of infection and sepsis, especially in those with a previous history of coronary artery disease. However, we should consider the possibility of type 1 MI in the setting of localized infection, even in those patients without significant risk factors. Corrales-Medina et al. reported a higher incidence of cardiac events in patients with pneumonia who were also found to have advanced age, history of prior cardiac arrhythmias, respiratory rate >30 per minute, BUN >29 mg/dL, sodium < 130 mmol/L, hematocrit < 30%, and those with new pleural effusions. These signs and symptoms associated with a significant troponin elevation in a patient with acute infection should raise suspicion for a type 1 MI and may warrant a more in-depth investigation. Pneumonia from any cause has been associated with acute coronary syndromes, with the highest risk being during acute infection. Musher et al. found a rate of myocardial infarction of 7 to 8% among patients who were hospitalized for pneumococcal pneumonia. Other infections like influenza, acute bronchitis, and urinary tract infections have been also associated with increased risk for adverse cardiac events. These infectious processes continue to place patients at an increased morbidity and mortality risk for up to 10 years after an acute infection, compared to the normal population. This is believed to be a downstream effect seen from the activation of the inflammatory pathway via interleukins and catecholamines.
A new abstract was submitted for the Nebraska Chapter on Friday, September 18, 2020 - 21:21:

Resident Abstract 44

**Category Submitting for:** Clinical Vignette

**Abstract Title** Atypical pelvic abscess as a complication of necrotizing pancreatitis: A case report

**Abstract Text**

**Introduction:**
Acute pancreatitis is an acute inflammatory process which can further be characterized by the presence of pancreatic glandular necrosis in the severe form. Necrotizing acute pancreatitis can involve both pancreatic and peri-pancreatic tissue, that are typically sterile, but can eventually become infected. Walled-off necrosis is often treated endoscopically in patients that are symptomatic or refractory to medical management. We present a rare infectious complication of necrotizing pancreatitis – pelvic abscess.

**Case:**
A 58 year old male presented to the emergency department with progressively worsening abdominal pain associated with bilateral lower extremity weakness and fatigue 72 hours after a cystgastrostomy stent removal. He has a past medical history of necrotizing pancreatitis of unknown etiology. The diagnosis of necrotizing pancreatitis was diagnosed one month prior to presentation following his second episode of acute pancreatitis in two months. He underwent endoscopic ultrasound at the time of diagnosis with cystgastrostomy stent placement; one week later, he underwent a repeat esophagogastroduodenoscopy (EGD) with necrosectomy. His stent was removed after his necrotic collection resolved.

In the emergency room, his pain was located in the left upper and lower quadrants with diffuse tenderness to palpation despite narcotic analgesia. Laboratory testing included a lipase of 21 [15-60 U/L] He was admitted for evaluation of abdominal pain. A computed tomography (CT) scan of the abdomen and pelvis suggested two previously defined fluid collections within the pancreatic tail, smaller to unchanged in size, as well a new 3.2 x 3.2 cm fluid collection inferior

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to the pancreas and an 8.7 x 9.5 cm gas filled pelvic fluid collection concerning for colonic fistulation. The patient underwent CT-guided drainage of his pelvic collection by interventional radiology. A subsequent magnetic resonance cholangiopancreatography (MRCP) demonstrated multiple abdominal fluid collections suggestive of walled off necrosis/abscess formation; one collection in the left upper quadrant suggested a fistulous connection with the small bowel. Fluid analysis revealed Streptococcus viridans and coagulase-negative Staphylococcus species, with low amylase, lipase, and triglyceride levels. It was determined that his pelvic abscess was secondary to necrotizing pancreatic fluid accumulation along the paracolic gutters and retroperitoneum into the pelvis. His abdominal pain improved following drainage and administration of antibiotics, and he was discharged.

Discussion:
Necrotizing pancreatitis is associated with a high mortality rate. Clinicians should be aware that peri-pancreatic tissue necrosis and fluid collection has the potential to extend into other parts of the body by fascial planes, resulting in abscess formation. Complex fluid collections can recur in up to 30% of patients with necrotizing pancreatitis. It is important to identify the characteristics of complex collections, as they may require intensive management and/or multiple invasive procedures to treat.