



# 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** Ecthyma Gangrenosum in an Immunocompromised Host

**INTRODUCTION:** Ecthyma gangrenosum (EG) cutaneous lesions are classically associated with *Pseudomonas aeruginosa* bacteremia. It is most commonly associated with immunocompromised states such as underlying malignancy or neutropenia [1].

**CASE PRESENTATION:** A 78-year-old female with active AML on chemotherapy presented with one week onset of a painful left groin necrotic lesion. Physical exam revealed fevers to 100.8, along with a 1 x 2 cm dry necrotic ulcer in the left groin with tender surrounding erythema. Laboratory findings were notable for WBC 12.6 (k/ul), Blasts 5.1 (k/ul), ANC 1000. Due to neutropenic fevers, blood cultures were drawn that grew 1 out of 2 positive *Pseudomonas aeruginosa*. The patient was empirically treated with IV Cefepime and Voriconazole, and later added Isavuconazonium. CT imaging of the left lower extremity was negative for fluid collection or gas in the subcutaneous tissue, and CTA chest exposed a 1.9 cm mass in right mid-lung field not seen on prior imaging. A punch biopsy of the necrotic lesion displayed epidermal necrosis with wound cultures also growing *P. aeruginosa*, and a diagnosis of EG was made. A biopsy of the new lung mass was negative for bacterial or fungal growth; flow cytome try was consistent with AML. Hospital course was complicated by massive hemoptysis and endobronchial clotting during IR biopsy of the lung mass. This required ICU transfer along with intubation, worsening absolute neutropenia and blast crisis, and eventual fatal outcome.

**DISCUSSION:** EG is often described as a cutaneous infection that develops from an infarcted macule into a black necrotic ulcer with surrounding erythema [2]. Classically, EG is observed in immunocompromised patients that developed *P.aeruginosa* septicemia. However, cases have been reported in immunocompetent, non-bacteremic patients as well [1]. Blood cultures along with punch biopsy with tissue cultures should be performed to confirm the presence of infection. Prompt initiation of antipseudomonal antimicrobials along with close monitoring of the patient is essential, as systemic infection with *P.aeruginosa* is often life-threatening.

**CONCLUSION:** *P. aeruginosa* bacteremia is commonly found in immunocompromised patients presenting with EG. It is crucial to begin empiric treatment with broad spectrum antipseudomonal agents once infection is suspected as outcomes are often fatal.

Reference #1 Vaiman, M., Lazarovitch, T., Heller, L., & Lotan, G. (2015). Ecthyma gangrenosum and ecthyma-like lesions: review article. *Eur J Clin Microbiology & Infect Dis*, 34(4), 633–639.

<https://doi.org/10.1007/s10096-014-2277-6>

Reference #2 Wu, D.C., Chan, W.W., Metelitsa, A.I. et al. (2011). *Pseudomonas* Skin Infection. *Am J Clin Dermatol* 12(3), 157–169. 10.2165/11539770-000000000-00000

## Resident Abstract 2

**Category Submitting for:** Clinical Vignette

**Abstract Title** Malaria Prophylaxis and Treatment: More than Meets the Eye

### Abstract Text

Introduction: Malaria is a protozoal infection caused by Plasmodium species. *P. falciparum* is the most prominent throughout Africa. Malaria can be diagnosed clinically in under resourced areas with a constellation of symptoms including fever, anemia, thrombocytopenia. Additional diagnostic techniques include light microscopy of peripheral smears, and molecular testing by histidine-rich protein 2 (HRP2) antigen or PCR. Treatment strategy has multifactorial considerations including local epidemiology, prophylactic treatment, and severity of infection.

Case: A 64-year-old female with past medical history of previous malarial infections and heart failure with reduced ejection fraction presented with 1 day of fever, severe headache, weakness, and fatigue. One month prior to presentation she returned to the U.S. from a 6-month trip to Central Africa. While there, she took one 3-day course of artemether-lumefantrine for malaria prophylaxis. Initial thick and thin smears resembled *P. vivax/ovale* and she was started on atovaquone-proguanil. She subsequently developed worsening headache, intermittent hypotension, and new onset acute kidney injury (AKI) with creatinine peak of 2.29 mg/dL. Repeat review of thick and thin smears resembled *P. malariae* with possible co-infection with *P. vivax/ovale*. Malaria therapy was then transitioned to artemether-lumefantrine while awaiting IV artesunate ordered from CDC. Her renal function stabilized within the next 24 hours with repeat BMP monitoring and fluids. Malaria PCR was obtained from an external reference laboratory, and PCR result after discharge was consistent with *P. ovale*. The patient was started on primaquine for eradication of liver hypnozoites after negative G6PD testing.

Discussion: Malaria treatment strategy is complex. Molecular diagnostic techniques aid in guiding therapy, but as in this case, peripheral smears are used to facilitate therapy while awaiting PCR confirmation as rapid antigen testing was not available. Empiric treatment is chosen based on most likely species, location of exposure and local resistance patterns, as well as receipt of prophylaxis. It is important to perform sequential smears to evaluate parasite burden and assess for potential change in therapy. Trends show that malarial infections seem to be decreasing throughout Africa with the introduction of artemisinin combination therapies (ACTs) which destroy gametocytes and lower the parasite load to decrease transmission. Though CDC recommends using a different antimalarial for treatment than was used for prophylaxis, in this case, artemether-lumefantrine was prescribed for broader coverage of possible co-infection given >3 weeks since initial treatment/prophylaxis. ACTs are an effective treatment with a low level of resistance in Africa. However, counterfeit products are sold widely, and inadvertent use of counterfeit antimalarials may increase future morbidity/mortality risk with subtherapeutic dosing and development of resistance. Prior prophylactic treatment should be discussed with the patient on admission, and determining where the medications were obtained is important, as this may impact initial treatment choices.

## Resident Abstract 3

**Category Submitting for:** Clinical Vignette

**Abstract Title** New onset left bundle branch block – activate the Cath lab, or not?

### Abstract Text

An 84 year old male was transferred to the hospital overnight for chronic obstructive pulmonary disease exacerbation and pneumonia with an extensive past medical history including: hypertension, atrial fibrillation, nonobstructive coronary artery disease (Cath 2016), heart failure with reduced ejection fraction (EF 25-30%), carotid artery stenosis status-post stent, abdominal aortic aneurysm, diabetes, prostate cancer and chronic kidney disease. In the morning, the team was called for EKG changes on telemetry; the patient was in atrial fibrillation with rapid ventricular response and new onset left bundle branch block (LBBB). The patient had no chest pain, but endorsed shortness of breath. He was afebrile, heart rate 110-130s with normal respiratory rate, saturating well on room air, blood pressure 130/70s. The question: activate the Cath lab, or not?

Patients with acute ischemic symptoms and new onset left bundle branch block pose a unique diagnostic challenge. These patients are less likely to have an acute culprit lesion on cardiac catheterization but are also a high risk patient population. The 2004 and 2007 American College of Cardiology (ACC) and the American Heart Association (AHA) STEMI guidelines recommended reperfusion therapy for patients with ischemic symptoms and new or presumed new LBBB. The 2012 European Society of Cardiology STEMI guidelines recommend early percutaneous coronary intervention for clinical presentation of STEMI and new LBBB. However, in 2013 the ACC/AHA STEMI guidelines removed the specific recommendation for reperfusion therapy for patients with new LBBB. This change may reduce the incidence of false activations of Cath lab and inappropriate fibrinolytic therapy. However, withholding appropriate reperfusion therapy in patients with true coronary artery occlusion has potential for increased morbidity and mortality.

Application of Sgarbossa's criteria is the most widely accepted tool to aid in diagnosis of MI in the presence of LBBB. Sgarbossa et al. identified three EKG criteria that may improve diagnosis of MI in patients with LBBB: ST-elevation of  $\geq 1$ mm and concordant with the QRS complex, ST-segment depression  $\geq 1$ mm in leads V1, V2, or V3, and ST elevation  $\geq 5$ mm and discordant with the QRS complex. Concordant changes on electrocardiogram are defined as the presence of more than one waveform change with the same polarity, or in the same direction. Sgarbossa's criteria are met if the is  $\geq 1$ mm ST segment elevation or depression that is concordant with QRS complex, this equates STEMI. If Sgarbossa's criteria are not met, serial cardiac biomarkers or echocardiogram is indicated.

In this case, cardiology was consulted and they recommended not activating the Cath lab. Troponin did not trend up and an echocardiogram revealed further reduction in ejection fraction, EF 15-20%, and a dyskinetic apex with best preserved function at the bases suggestive of stress induced cardiomyopathy.

## Resident Abstract 4

**Category Submitting for:** Clinical Vignette

**Abstract Title** Evaluation of Hematochezia: Many Factors(V) to consider

### **Abstract Text**

A 31-year-old female presented with a several day history hematochezia. She also noted bloating and new onset nausea with vomiting, but no diarrhea. She had a prior instance of abdominal pain one year prior. A colonoscopy was performed at that time and, per the patient, colitis of uncertain etiology was diagnosed. She denied using non-steroidal anti-inflammatory drugs; however, she had been taking oral-contraceptive pills for the last several years. She had no family history of bleeding or cancer. She worked as a flight-attendant and had flown several international flights however she denied ever leaving the plane during these flights.

On examination, abdomen was tender to palpation in both lower quadrants and there was splenomegaly. There were no peritoneal signs or cutaneous manifestations of liver disease. Colonoscopy revealed diffuse colitis with mucosal edema and congestion with prominence of the venous plexus and localized non-bleeding left colonic varices. Portal-vein thrombosis, and splenomegaly were found on US liver w/ Doppler. Diffuse portal hypertensive gastropathy and 1+/4 esophageal varices were found with EGD. Thrombophilia workup revealed the patient was heterozygous for Factor V Leiden mutation.

In the case described, the patient's rectal bleeding was caused by portal hypertension due to portal-vein thrombosis. The patient was found to have thrombophilia due to a Factor V Leiden heterogenous mutation, an autosomal dominant disorder and the most commonly inherited hypercoagulable state. Factor V Leiden mutation is a mutation in Factor V making it resistant to inactivation by Protein C. It affects approximately 5% of the population.

Studies have shown the Factor V Leiden mutation alone does not cause portal-vein thrombosis. Most often several prothrombotic factors including obesity, smoking, extrinsic compression from a tumor, cirrhosis, and elevated estrogen levels from medications or pregnancy combine to produce a portal-vein thrombosis. Our patient's OCP medication was stopped, and she was placed on lifelong anticoagulation.

Consideration of risk factors for thrombus includes the patient's activity level, evaluating for local damage to the vessel endothelium, and determining if there is a procoagulant state. Our patient had two pro-coagulant risk factors with her inherited condition, and she was taking oral contraceptive pills. Patients with Factor V Leiden mutation increase their relative risk of developing venous thromboembolism by a factor of ten when they also take oral contraceptive pills. Additionally, her history of international flights may have added an additional risk factor toward the development of venous thromboembolism.

The location of venous thromboembolism will vary and, although they are more likely to manifest as deep venous thromboembolism or as pulmonary embolism, first presentation can manifest as portal-vein

thrombosis. Physicians should consider portal hypertension and the development of portosystemic shunts as a cause of rectal bleeding in patients that have pro-coagulant risk factors.

## Resident Abstract 5

**Category Submitting for:** Clinical Vignette

**Abstract Title** Sarcoidosis as an Imitator of Primary Sclerosing Cholangitis

### Abstract Text

#### Introduction

Sarcoidosis is a multi-system, granulomatous disorder that can involve the liver in 50-95% of cases. Symptoms usually consist of pruritus, jaundice, scleral icterus, weight loss, and generalized abdominal symptoms. As few as 20 cases of sarcoidosis presenting as primary sclerosing cholangitis (PSC) have been reported. In these cases, the patients typically had a previous diagnosis of sarcoidosis before the development of biliary symptoms. Here we present a case differing from what has been classically described in the literature.

#### Case

A 34 y.o. male with past medical history of iritis presented to the clinic for the evaluation of pruritus, jaundice, and weight loss. Initial evaluation showed a cholestatic pattern. US abdomen showed biliary duct dilation, and further imaging with MRCP showed intrahepatic and extrahepatic biliary duct dilatation with narrowing near the confluence. ERCP showed beading of ducts bilaterally with a dominant stricture which was dilated and sphincterotomy performed. The patient's itching and jaundice resolved while his liver enzymes returned to normal. In the ensuing 2 months, the patient's symptoms returned. Repeat ERCP demonstrated multiple dominant strictures which were once again dilated. However, the patient's symptoms only moderately improved. Seemingly unrelated, the patient then had a sebaceous cyst removed with pathology showing noncaseating granulomas. Chest XR showed increased perihilar interstitial markings, and CT displayed findings consistent with pulmonary sarcoid. Br onchoscopy with biopsy was positive for sarcoidosis. The patient was started on prednisone and, within 2 months, the patient's symptoms and labs returned to normal.

#### Discussion

As this case illustrates, it is essential to understand the potential similarities in presentation between PSC and biliary sarcoidosis. PSC is a chronic hepatobiliary disorder that leads to fibrosis and obliteration of the intra and extrahepatic bile ducts. Analysis of explanted livers has shown that sarcoidosis may present with biliary tree involvement due to either granulomas in the ductal wall or fibrous adhesions in the surrounding lymph tissue. For this reason, the prototypical radiologic findings of PSC with "beads on a string" or "pruning" may be imitated by sarcoid.

In our case, the patient was planned to undergo repeated ERCP to alleviate his symptoms until he would be an eligible candidate for transplant. Liver biopsy failed to demonstrate sarcoidosis as the culprit of his liver dysfunction, and it wasn't until his cutaneous and pulmonary biopsy that sarcoidosis became the leading candidate for his presentation. With the appropriate therapy, his biliary symptoms resolved along with his cutaneous and pulmonary symptoms.

In summary, it is important to consider sarcoidosis when addressing a case of recurrent symptomatic PSC. Early identification of systemic sarcoid symptoms and confirmation with biopsy will allow patients to be started on appropriate steroid therapy, and prevent repeated invasive treatments for PSC.

## Resident Abstract 6

**Category Submitting for:** Clinical Vignette

**Abstract Title** Extradurellary Ependymoma: A Case Report

### **Abstract Text**

Extradurellary Ependymoma: A Case Report

#### Introduction:

Ependymomas are a group of glial tumors that typically arise within or adjacent to the ependymal lining of the ventricular system. Several histologic subtypes exist including papillary, cellular, clear cell, and myxopapillary. Of these the myxopapillary subtype is the least common, representing only 13% of all ependymomas. Myxopapillary ependymomas are thought to arise from the glia of the filum terminale, whereas other subtypes typically occur in the cervical spinal cord. Here we outline an interesting case of a 27-year-old female who presented with cauda equina syndrome and was eventually diagnosed with myxopapillary ependymoma. Her case was further complicated by the diagnosis of pancreatic gastrinoma only 1 year after.

#### Case presentation:

A 27-year-old female with no significant PMH presents with a 3-year history of progressive lower back pain, numbness involving her back, hips, bilateral legs, double vision, and rectal incontinence. MRI of the lumbar spine revealed a large 4.9 cm intradural extradurellary mass extending from the L4 pedicles to the endplate of L5 filling the spinal canal with leptomeningeal spread along nerve roots of cauda equina inferiorly. The patient was diagnosed with grade 1 myxopapillary ependymoma and underwent L3-L5 laminectomy with 90% debulking and completed 50 CGE of lumbar radiation over 30 fractions. Disease surveillance showed an interval decrease in size of the lesion and no progression or recurrence. A year later, the patient presented with a two-week history of abdominal pain, vomiting, nausea, persistent diarrhea, and a 25-pound weight loss. An EGD revealed gastritis with hemorrhage and multiple duodenal ulcers. Gastrin levels were found to be elevated at 883. A PET scan revealed an abnormal focus of somatostatin uptake in the third portion of the duodenum measuring 11 mm, compatible with gastrinoma. She eventually underwent gastrinoma resection complicated by pancreatic leak, intra-abdominal abscess, and pancreatitis.

#### Discussion:

Ependymomas most commonly occur in the posterior fossa, in contact with the fourth ventricle, or in the intradurellary spinal cord. They occasionally occur outside the posterior fossa, and very rarely outside the central nervous system. Ependymomas represent 60% of all glial spinal cord tumors, making them the most common intradurellary neoplasm in adults with a peak age at presentation between 30 and 40 years. In our case, the ependymoma is neither located in the fourth ventricle nor intradurellary, which makes it a rare occurrence. The myxopapillary subtype is also the least common. Initial management of these tumors consists of laminectomy with attempted complete surgical resection. Interestingly, in very



few individual case reports, Ependymomas are reported in patients with MEN1 Syndrome.

Conclusion: This case concludes that ependymomas are not only restricted to the brain and intramedullary spinal cord but can also present as an extramedullary spinal cord tumor along with MEN1 syndrome phenotype.

## Resident Abstract 7

**Category Submitting for:** Clinical Vignette

**Abstract Title** The Story of the Sticky STI

### **Abstract Text**

A 38 year-old woman from Sudan presented with acute altered mental status and fever of 39.2C. She was hypotensive, tachycardic, and had a lactate of 20. Initial evaluation did not reveal a focal source of infection – head, chest, and abdominal imaging were reassuring, UA was bland, and there was no evidence of skin or soft tissue infection. She was initiated on broad-spectrum antibiotics, intravenous fluids, and norepinephrine. At 26 hours, blood cultures grew *Neisseria gonorrhea*. Antibiotics were narrowed to ceftriaxone. Our patient stabilized and her mental status cleared. Patient endorsed ankle and foot pain. Orthopedics was consulted but did not have a concern for a septic joint. Patient was HIV negative. A possible mitral valve vegetation was seen on transthoracic echocardiogram. Transesophageal echocardiogram revealed no definitive vegetations but did reveal mitral valve leaflet thickening. Our patient was treated with four weeks of cef triaxone in the setting of possible gonococcal endocarditis.

### **Learning Points:**

1. Understand the epidemiology and presentation of disseminated *N. gonorrhea* infection
2. Recognize the relationship between *N. gonorrhea* bacteremia and infective endocarditis
3. Understand the association of mitral valve leaflet thickening with endocarditis

*N. gonorrhea* infections are commonly encountered by the internist. An estimated 600,000 cases occur each year, making it the second most common STI in the United States. Although most cases are localized to the urogenital system, disseminated infections occur in 1-3% of cases. Hematogenous spread of the bacteria occurs within 2-3 weeks of the inciting infection. The initial presentation of disseminated infections can be heterogenous, ranging from fevers and malaise to polyarthralgia, tenosynovitis, dermatitis, or rarely septic shock. Endocarditis is also a rare manifestation of disseminated gonococcal infections occurring in 1-2% of cases. The overall mortality rate of gonococcal endocarditis is 20%. The aortic valves are most affected and large vegetations are often seen on echocardiography. No discrete vegetations were identified in our case, but a thickened mitral valve leaflet was present. In the setting of questionable endocarditis, this finding was concerning for two reasons. First, we could not definitively rule out endocarditis as the cause of the valvular abnormality. Thickened mitral valves are most associated with non-bacterial thromboembolic endocarditis, but rarely can be seen with bacterial endocarditis. Secondly, the thickened mitral valves may have predisposed our patient to endocarditis. Thickened mitral valve leaflets are seen in over 90% of patients with rheumatic heart disease, a common childhood illness in Sudan. These valve deformities disrupt blood flow dynamics and are therefore a leading risk factor for endocarditis in developing countries. With these two factors in mind, as well as the high mortality risk of gonococcal endocarditis, we opted to treat our patient with an extended course of antibiotics.

## Resident Abstract 8

**Category Submitting for:** Clinical Vignette

**Abstract Title** Drug induced Hemolytic Anemia in patient with Spotted Fever Rickettsiosis

### **Abstract Text**

Spotted Fever Rickettsiosis (SFR) is an uncommon illness in Nebraska but requires prompt diagnosis and treatment. Improper treatment results in severe illness and complications including drug induced hemolytic anemia (DIHA).

A 32-year-old man with no past medical history presented to the emergency room complaining of an insect bite on his left deltoid obtained 4 days ago. Shortly after the bite, he developed localized erythema, fevers, and malaise. Rash then spread inward to his torso. Patient was initially diagnosed with cellulitis and treated with trimethoprim-sulfamethoxazole (TMP-SMX). Patient returned three days later complaining that his symptoms had gotten worse. He also reported yellowish discoloration of his skin. On exam, he was noted to have scleral icterus and a petechial rash on his left arm spreading down towards his chest. Labs were notable for white blood cell (WBC) count 18.1 K/uL, hemoglobin 10.6 g/dl, lactate dehydrogenase 750 U/L, haptoglobin <8 mg/dl, aspartate transaminase 115 U/L, alanine transaminase 149 U/L, total bilirubin 13.5 mg/dl, direct bilirubin 7.9 mg/dl. Repeat WBC count a day later increased to 26.1 K/uL and hemoglobin dropped to 4.6 g/dl. Direct antiglobulin test performed and was positive. Given high suspicion of DIHA, TMP-SMX was discontinued and patient was started on 80mg prednisone. Patient placed on broad spectrum antibiotics initially for suspicion of sepsis. However, with no improvement suspicion was raised for SFR. Patient was started on oral doxycycline. His symptoms improved and was discharged home on a seven-day course of 100mg doxycycline twice per day. Rocky Mountain Spotted Fever IgM Enzyme immunoassay test was obtained and later found positive at 1.64 index.

In 2010 the CDC coined the term SFR due to the inability to differentiate Rickettsia species on serological tests. Most recognized of the group is Rocky Mountain spotted fever. SFR are the most reported tick-borne diseases in the state of Nebraska. The Nebraska Department of Health and Human Services reports an increase in the median of cases over the last 5 years from 14 cases per year up to 26 annually on average. Symptoms typically start out nonspecific such as fever, headache, and gastrointestinal complaints. Rash is a common finding but may be absent in 10% of cases. Labs may reflect leukocytosis or leukopenia, thrombocytopenia, anemia, or elevated liver enzymes. The standard serologic test for diagnosis of SFR is the indirect immunofluorescence antibody (IFA) assay for immunoglobulin G (IgG) using R. rickettsii antigen. Doxycycline is the drug of choice and is recommended in patients of all ages, including children less than 8 years of age. Delay in treatment may result in mortality, therefore treatment is required prior to confirmatory testing. This case illustrates importance of prompt diagnosis of SFR, treatment and adverse effects of misdiagnosis.

## Resident Abstract 9

**Category Submitting for:** Clinical Vignette

**Abstract Title** Menacing Meningitis, Fulminant Disease in a Derelict Male.

### **Abstract Text**

Invasive meningococcal disease without meningitis has become so rare in the United States that it can be considered a forgotten diagnosis. Dissemination of *Neisseria meningitidis* can lead to unique and fatal complications including Disseminated Intravascular Coagulation (DIC) and adrenal infarction/insufficiency. We present a case of a 45-year-old homeless man who was found down by emergency medical service personnel. On arrival to ER he was hypotensive with a blood pressure of 78/61mm Hg, and hypothermic with a temperature of 91.6 °F. His initial Glasgow Coma Scale score was 3. On exam, he was noted to have cold and clammy extremities with mottled dusky skin and a right foot wound. He was intubated for airway protection and placed on mechanical ventilation. Therapy with intravenous vancomycin, piperacillin/tazobactam, clindamycin and vasopressors was started. Laboratory evaluation revealed a white blood cell (WBC) count of 23.9 k/ uL (reference range, 4-11), and elevated levels of creatinine kinase (14,566 U/L), AST (1,899 U/L), ALT (741 U/L), creatinine (3.2 mg/dl), procalcitonin (59.4 ng/ml), and c-reactive protein (82.1 mg/L). His INR was 1.3 and his PTT was elevated at 28.8 with a D-dimer level over 10,000 mcg/ml. Blood cultures were obtained and lumbar puncture was performed in ER. Cerebrospinal fluid (CSF) was clear, with a WBC count of 3 leukocytes per mm<sup>3</sup>. Meningitis/encephalitis panel performed on CSF did not detect any pathogens. A computed tomography (CT) scan of his head did not show any acute intracranial abnormalities. A CT abdomen revealed bilateral lower lobe consolidations. Initial blood cultures and cultures of tracheal aspirate showed no growth. Transthoracic echocardiogram was performed which revealed a right ventricle thrombus for which heparin infusion was initiated. The patient continued to deteriorate, he developed multiorgan failure including renal failure requiring renal replacement therapy. Repeat endotracheal tube aspirate was obtained which grew *Neisseria meningitidis* (*N.meningitidis*). His antibiotic regimen was switched to Ceftriaxone. Despite these efforts the patient eventually succumbed to the infection and expired.

This case highlights the forgotten manifestation of *N.meningitidis*, that of pneumonia complicated by invasive meningococcal disease in the absence of meningococcemia or meningitis. Primary pneumonia occurs only 5–10% of patients with meningococcal disease, most often caused by serogroup Y. Occurrence of invasive meningococcal diseases secondary to pneumonia has been reported in the literature. The overall case fatality rate of invasive meningococcal disease is 10% despite antibiotics and aggressive supportive therapy even in developed countries. Prompt recognition of this manifestation of *N. meningitidis* is important for early antibiotic treatment and to avoid potentially dangerous complications of invasive meningococcal disease.

## Resident Abstract 10

**Category Submitting for:** Clinical Vignette

**Abstract Title** A Plume of Foul Dust: An Unusual Case of Pneumonia

### **Abstract Text**

**Introduction:** Community-acquired pneumonia (CAP) is a common diagnosis for internists. Common causes include viruses and typical bacteria such as *Streptococcus pneumoniae*. However, it is important to identify cases with unique exposures or unusual presentations to reach the correct diagnosis and treatment.

**Case:** A 58-year-old man presented with two weeks of fevers, dry cough, and worsening dyspnea. He was tachycardic, tachypneic, febrile, and required 50L/min with 50% FiO<sub>2</sub> via high flow nasal cannula. Labs were notable for a white blood cell count of 14,000/ $\mu$ L, platelet count of 148,000/ $\mu$ L, aspartate aminotransferase of 142 U/L, alanine aminotransferase of 74 U/L, and procalcitonin of 2.65 ng/ml. Computed tomography of the chest showed multifocal consolidation greatest in the right lower lobe with hilar lymphadenopathy and splenomegaly. Initial empiric antimicrobials included vancomycin and cefepime, which were rapidly de-escalated to ceftriaxone and azithromycin for CAP. Sputum culture, respiratory viral panel, and urine streptococcal and legionella antigens were negative. After several days without improvement, azithromycin was switched to doxycycline, as symptoms began after exploring old buildings with dead pigeons and aerosolized avian excrement. Further work-up including histoplasma antigens, galactomannan, cryptococcal antigen, and (1-3)-beta-D-glucan was unrevealing. Bronchoalveolar lavage with biopsy revealed neutrophil-predominant white blood cell elevations, negative cultures, and histopathology with acute inflammation and evidence of organizing lung injury. Chlamydia psittaci antibody IgG returned elevated at 1:512, confirming a diagnosis of psittacosis. He was treated with doxycycline for ten days and after significant improvement, discharged on supplemental oxygen.

**Discussion:** Psittacosis is rare, accounting for an estimated one percent of CAP cases. Birds are the primary reservoir for the causative organism, *Chlamydia psittaci*, and the organism is shed in avian feces, urine, and respiratory secretions; when these vehicles dry, they can become airborne as droplet nuclei. Humans are usually infected when they inhale these droplets. Psittacosis has higher prevalence in middle age and in males. Common symptoms include fever, headache, and dry cough with symptom severity ranging from asymptomatic infection to death. The differential diagnosis for pneumonia with bird exposure includes psittacosis, Q fever, *Mycobacterium avium*, influenza, histoplasmosis, cryptococcus, and hypersensitivity pneumonitis. Diagnosis is made with serologic testing as culture is difficult and discouraged, as it is highly contagious to lab personnel. Doxycycline is the preferred treatment of choice, and in patients with appropriate exposure history, it should be included in the empiric CAP antibiotic regimen.

**Conclusion:** While rare, psittacosis pneumonia should remain on the internist's differential when there is a history of bird exposure in a patient presenting with CAP. This case emphasizes the importance of obtaining a thorough exposure history, maintaining a broad differential diagnosis, and using appropriate atypical coverage for CAP.

## Resident Abstract 11

**Category Submitting for:** Clinical Vignette

**Abstract Title** Tularemia Peritonitis: A Rare Presentation of a Rare Disease

**Abstract Text**

Tularemia Peritonitis: A Rare Presentation of a Rare Disease

Introduction: Tularemia is an infectious disease caused by the bacterium *Francisella tularensis*, a naturally occurring pathogen most commonly found in rabbits and small rodents. Disease manifestations differ based on the portal of entry: Humans are most often infected via the skin, which typically results in lymphadenopathy with or without overlying skin ulceration. Though tularemia has a wide range of uncommon presentations, peritonitis is exceptionally rare.

Case Description: A 66-year-old male with end-stage renal disease on peritoneal dialysis presented in May to the emergency department with abdominal pain, fever, and malaise. He reported recent removal of two engorged ticks from his lower abdomen several days prior. Physical exam revealed a bulge in his left groin, without overlying rash or ulceration. The patient met SIRS criteria for sepsis, with fevers reaching 102.3 F and tachycardia. Spontaneous bacterial peritonitis was suspected; blood and peritoneal fluid cultures were drawn. The patient was empirically treated with IV ceftriaxone, which resulted in fever resolution and symptom alleviation by hospital day three. Oral doxycycline was prescribed for a two-week course of continued empiric therapy. No organisms were isolated from his cultures.

His symptoms quickly returned and the patient visited an urgent care clinic twice before returning to the ED one month post-discharge. Peritoneal fluid analysis revealed a neutrophil count of 168 cells/uL, solidifying a diagnosis of bacterial peritonitis. Broad spectrum empiric coverage was started with intraperitoneal vancomycin and ceftazidime, given as an initial loading dose and then as maintenance doses via his nightly peritoneal dialysis exchanges. The patient had marked clinical improvement and he was again able to discharge after three days. Nine days following discharge, *Francisella tularensis* was isolated from peritoneal fluid cultures. Glandular tularemia was diagnosed; his antibiotic regimen was changed to a three-week course of intraperitoneal gentamicin and oral doxycycline, resulting in lasting symptom resolution and no further recurrences.

Discussion: This case illustrates the importance of a detailed history in the setting of infection with an unidentified organism. Tularemia is difficult to culture, making clinical clues such as a potential tick vector important for broadening the differential and guiding empiric management—though, as our case demonstrates, even the clinical presentation can be misleading. Our literature search revealed only two instances of tularemia presenting as peritonitis—neither of which were associated with a tick vector. Furthermore, tularemia in a peritoneal dialysis patient is also exceedingly rare, likely making this a novel situation. The recognition of tularemia is important, as treatment with aminoglycosides is often curative and these antibiotics are rarely (if ever) employed empirically.

## Resident Abstract 12

**Category Submitting for:** Research

**Abstract Title** Cardiac MRI and intracoronary OCT in patients with a clinical diagnosis Of MINOCA (MI with non-obstructive Coronary Arteries)

**Abstract Text**

Background: Myocardial Infarction with Non-Obstructive Coronary arteries (MINOCA) is defined as acute Myocardial Infarction (MI) despite the absence of severe coronary artery stenosis on angiography (i.e., stenosis severity <50%) and no specific alternative diagnosis (e.g., sepsis, pulmonary embolism). MINOCA remains a serious diagnostic and therapeutic challenge given the various possible underlying etiologies. Cardiac Magnetic Resonance Imaging (CMR) can distinguish between acute myocarditis (AM), MI, and Takotsubo cardiomyopathy (TCM) which are some of the commonest causes of MINOCA. Hence, The European Society of Cardiology (ESC) recommend CMR in all MINOCA patients with an unclear etiology. Intracoronary Optical coherence tomography (OCT) is a high-resolution imaging modality that allows for assessment of the integrity of the atheroma. We aimed to study the efficacy of CMR and OCT in detecting the etiology in MINOCA patients, which has the potential to guide medical therapy.

Methods: A systematic search was made in PubMed, Medline and Cochrane database. Search terms used: Myocardial infarction, Coronary angiography, Normal coronary arteries, CMR, and OCT. Inclusion criteria was fulfilled by 18 studies. Meta-analysis and meta-regression was performed with 14 studies due to increased heterogeneity.

Results: A total of 2474 patients were included in the meta-analysis. The mean age of all the patients was 51.5 and 56.4% were men. CMR was able to establish diagnosis in 74% of the patients; 30.4% had AM, 21.3% had true MI and 12% had TCM. Upon metaregression the demographic variables, cardiovascular risk factors, troponin levels and C-reactive protein levels did not have any association with AM or MI. A diagnosis was established in 85-100% cases by incorporation of OCT with CMR, which was better at finding the etiology than CMR or OCT individually.

Conclusion: CMR is integral in finding the cause of MINOCA. Coupling coronary OCT and CMR is better than either technique alone at identifying the etiology.

## Resident Abstract 13

**Category Submitting for:** Clinical Vignette

**Abstract Title** Improve your practice: recognize calciphylaxis!

### **Abstract Text**

#### Case Description:

A 68-year-old female was admitted with encephalopathy after missing dialysis. Her history included ESRD, HIV/AIDS, Type 2 Diabetes, heart failure, and depression. She also complained of severe right leg pain. She had recently been admitted for this and it was treated as cellulitis. At admission the lesion was erythematous, indurated, warm to the touch, and exquisitely painful. A similar but smaller lesion was present on her left calf.

Soft tissue ultrasound of the lesion showed shadowing within the subcutaneous tissue suggesting calcification. Biopsy showed interstitial calcification surrounding small blood vessels consistent with calciphylaxis. Notably, the patient's encephalopathy improved with resuming dialysis.

#### Discussion:

A retrospective study found that 73% of patients eventually diagnosed with calciphylaxis were initially misdiagnosed.<sup>1</sup> Many etiologies of soft tissue inflammation, such as cellulitis, peripheral artery disease, or vasculitis can have significant overlap in physical appearance. Calciphylaxis can present as livedo reticularis, plaques, nodules, or ulcers. Distinguishing physical exam characteristics of calciphylaxis are pain out of proportion to exam and a characteristic induration similar to but distinct from peripheral edema.<sup>2</sup>

Understanding calciphylaxis's pathogenesis provides a framework to understand its risk factors. Persistently elevated calcium, phosphorus, and PTH result in smooth muscle cells within the media layer of blood vessel walls becoming osteoblast-like and producing hydroxyapatite crystals. Fibrointimal hyperplasia also develops and leads to decreased nutrient diffusion and an increased risk of small vessel thrombosis.<sup>2,3</sup>

Risk factors include ESRD and concomitant hyperphosphatemia, hypercalcemia, and hyperparathyroidism. Factors that increase the risk of thrombosis include obesity, female sex, and an underlying hypercoagulopathy. Hyperglycemia and warfarin use are additional risk factors. Our patient closely fit this risk profile: she had electrolyte derangement as a result of poor adherence to dialysis regimen. Obesity, type 2 diabetes, and female sex also increased her risk.

In the right clinical context, diagnosis can be made without biopsy. Biopsy is contra-indicated because of poor wound healing. Bone scans can be used as a non-invasive diagnostic alternative.<sup>2</sup>

The mainstay of treatment is slowing progression and preventing formation of additional lesions by



correcting risk factors. Specific interventions can include increased duration and frequency of dialysis, hemodialysis instead of peritoneal dialysis, calcium and phosphorus binding medications, and medical or surgical parathyroidectomy.

Sodium thiosulfate, as an infusion or intralesional injection, is thought to be efficacious because it acts as a calcium chelator and antioxidant. Vitamin K is an inhibitor of carboxylation; its supplementation is an area of study. 2 A recent case series described complete resolution without reoccurrence when diagnosed promptly and treated with an interdisciplinary team.<sup>4</sup>

Disease course:

Our patient had inconsistent follow-up and missed multiple consecutive dialysis treatments. When seen three weeks after discharge, the affected area had more than tripled in size.

## Resident Abstract 14

**Category Submitting for:** Clinical Vignette

**Abstract Title** When Antibiotics Go Bad

**Abstract Text**

An 82-year-old female presented to the Emergency Department with dysphagia, confusion, and lethargy. Labs were remarkable for neutropenia (WBC 0.9, ANC 0) and anemia (hemoglobin 9). Head CT did not show any signs of acute stroke.

Additional history includes a diagnosis of culture negative, presumed bacterial meningitis about four months prior. She was treated with vancomycin and ceftriaxone, and was discharged after clinically improving. Two months later, she presented with headaches. MRI showed three rim enhancing lesions including the left anterior temporal lobe and left cerebellum. There was also evidence of left sphenoid sinus opacification with dehiscence. She underwent sinus washout with ENT, followed by aspiration of the abscess by Neurosurgery. Sinus cultures returned positive for *Pseudomonas aeruginosa* so she was started on cefepime. A few days later, she became lethargic. She was found to be in non-convulsive status epilepticus, suspected to be from cefepime neurotoxicity. She was transitioned to meropenem with plans for prolonged antibiotic therapy on an outpatient basis. Six weeks later, upon routine lab monitoring while on meropenem it was noted she became leukopenic. There was concern for meropenem induced leukopenia. Due to the limited antibiotic options to treat *Pseudomonas* with appropriate blood-brain barrier penetration, cefepime was resumed for final few weeks of therapy. One week later, she presented as above with dysphagia and altered mental status. It was quickly apparent the most likely etiology of this patient's encephalopathy was cefepime use. She had not completed therapy for her intracranial abscesses and was therefore started on levofloxacin after thorough discussion with pharmacy. Given leukopenia, she was also started on immunocompromised host prophylaxis with acyclovir and fluconazole. Per Infectious Disease recommendation, G-CSF supplementation was started, and leukopenia resolved after three days. Repeat MRI showed no residual abscess, so antimicrobials were ultimately stopped. Unfortunately, she then developed diarrhea and was ultimately diagnosed with *Clostridioides difficile* diarrhea. Diarrhea resolved after initiation of oral vancomycin.

This patient had numerous complications from antibiotics including cefepime induced neurotoxicity, meropenem induced leukopenia, and *Clostridioides difficile* diarrhea. The management of this case is interesting, as there are few antibiotics that provide adequate coverage for *Pseudomonas* in the cerebrospinal fluid. Cefepime has often taken the place of piperacillin/tazobactam and is used in conjunction with vancomycin in the initial treatment of patients with sepsis before cultures and susceptibilities have resulted. Cefepime induced neurotoxicity is an underrecognized phenomenon which is important to differentiate from alternative etiologies of encephalopathy in hospitalized patients, particularly delirium. Antibiotic use is ubiquitous in hospitalized patients; recognizing adverse reactions from antibiotics is imperative in patient care.

## Resident Abstract 15

**Category Submitting for:** Clinical Vignette

**Abstract Title** Simple Ankle Fractures? Think Again.

**Abstract Text**

Simple Ankle Fractures? Think Again.

Case Discussion:

A 57 year-old female presented to the emergency department with lower extremity pain found to have bilateral fibular fractures after syncopal episodes. She had a past medical history notable for pulmonary hypertension, obstructive sleep apnea compliant with CPAP, pulmonary embolism (PE) on anticoagulation, tobacco use, and epilepsy. She had multiple syncopal episodes at home over the last several months. Of note, seizures had been well controlled, with last seizure five months prior. Orthopedic consultation recommended surgical intervention for fracture stabilization. However, preoperative risk assessment revealed recent echocardiogram demonstrating severe pulmonary hypertension with pulmonary artery systolic pressures (PASP) >50mmHg. Further, recent outside CT scan imaging documented a pulmonary embolus. Formal pulmonary hypertension workup was recommended prior to surgery. A V/Q scan was consistent with substantial bilateral pulmonary artery clot burden, and subsequent right heart catheterization demonstrated PASPs of 105/35. Surgical intervention was deferred, as the patient represented a high-risk surgical candidate given PASPs greater than systemic pressures.

She was subsequently treated for her pulmonary hypertension with Macitenaten and Riociguat while ankle fractures were managed non-operatively. Her interval increase in PA clot burden while on Eliquis prompted further investigation. Review of her medications demonstrated induction reaction between carbamazepine for epilepsy and Eliquis resulting in sub-therapeutic levels of anticoagulation. Additional malignancy workup resulted in CT evidence of large bowel thickening, consistent with possible malignancy.

Discussion:

This case highlights several important points that are applicable to the general internist and hospitalist. First, syncope evaluation has numerous etiologies requiring a thorough history and exam; syncope secondary to pulmonary hypertension is a relatively uncommon etiology as it represents advanced disease state with poor prognosis as noted by Rosenkranz et al., 2020 in a review of Systemic Consequences of Pulmonary Hypertension and Right Heart Failure. This case also underscores the importance of performing a thorough preoperative assessment and risk stratification prior to surgery. Lastly, it represents a challenging scenario of progressive thrombosis with adverse clinical outcome in a patient treated

“appropriately” with direct oral anticoagulant (DOAC) therapy. DOACs provide a patient-friendly alternative to vitamin-K dependent clotting factor antagonism with Warfarin, however specific drug-drug interactions and altered pharmacokinetics must be considered in patient selection. In the presented case, sub-therapeutic levels of anticoagulation in the setting of carbamazepine use, resulted in relative failure of DOAC to achieve desired clinical goal, with resultant chronic thromboembolic pulmonary hypertension (CTEPH). Therapeutic drug selection must be made with the disease state, patient physiology, and co-existing pharmaceutical therapies understood and respected.

## Resident Abstract 16

**Category Submitting for:** Clinical Vignette

**Abstract Title** A case of *Pneumocystis jirovecii* not following prophylaxis guidelines

**Abstract Text**

In immunocompromised patients not related to HIV or malignancy, guidelines for *Pneumocystis jirovecii* pneumonia (PCP) prophylaxis suggest prophylaxis for patients on daily doses of prednisone 20 mg or greater for one month or longer duration. We present a case of a 71-year-old male with history of ulcerative colitis and recent diagnosis of bullous pemphigoid who had been treated with prednisone taper along with immunosuppressive therapy that developed dyspnea on exertion, fever, and cough that was found to have PCP pneumonia while on low-dose prednisone (10 mg daily).

A 71-year-old male with past medical history of ulcerative colitis, diabetes mellitus type II, hypertension, hyperlipidemia, chronic kidney disease stage 2, obstructive sleep apnea was diagnosed 6 months ago with bullous pemphigoid on his lower extremities and was treated with prednisone along with adjustments to his ulcerative colitis regimen to treat both his ulcerative colitis and bullous pemphigoid. He had been recently switched to mesalamine due to recent AKI and elevated LFT's that he developed while previously on methotrexate. 6 months after his bullous pemphigoid diagnosis, he then presented for hospital admission due to a 3-week history of cough, dyspnea on exertion, fever and found to have mild hypoxia in the emergency department. Initial work-up showed lactic acid, troponin, pro-BNP, and procalcitonin within normal range. CBC was overall unremarkable and CMP with stable chronic kidney disease, stable electrolytes and normal LFT's. A CXR showed borderline cardiomegaly and bilateral interstitial prominences that could suggest viral illness. He was requiring 2 L by nasal cannula for supplemental oxygen. There was initial concern this could be related to mesalamine pneumotoxicity as an etiology outside of infectious causes. A CT chest without contrast demonstrated ground glass opacities and multiple small pulmonary nodules bilaterally. An echocardiogram showed preserved ejection fraction. Pulmonology was then consulted, and a bronchoscopy was performed, which BAL studies ultimately confirmed PCP pneumonia by PCR testing. He was then treated with trimethoprim-sulfamethoxazole with resolution of his hypoxia and improvement in his cough and dyspnea.

This case demonstrates the importance of keeping a broad differential while undergoing an infectious evaluation in an immunocompromised patient without HIV. While guidelines suggest PCP pneumonia prophylaxis is not indicated in a patient that receives less than 20 mg of prednisone daily, this case demonstrates that not all patients may follow the guidelines, which is important when building a differential diagnosis list for an immunocompromised patient not due to HIV or malignancy.

## Resident Abstract 17

**Category Submitting for:** Clinical Vignette

**Abstract Title** Left Main Coronary Artery Vasospasm: a misdiagnosed case of severe coronary artery disease

**Abstract Text**

Introduction: Left main coronary artery (LMCA) vasospasm is extremely rare with only a few cases reported in the literature. LMCA spasm can cause demand-supply mismatch that can mimic coronary artery disease (CAD). Coronary artery spasm (CAS) can be challenging to diagnose because it has a wide range of presentations from silent ischemia, chest pain to sudden cardiac death. We describe a patient who had isolated LMCA stenosis which was misdiagnosed with severe left main disease requiring surgical referral. CT angiography unmasked spasm as the underlying cause and prevented unnecessary surgery.

Case description: A 55-year-old woman with a history of hypothyroidism presented with exertional retrosternal chest pain radiating to the neck and left arm. Vital signs were stable and physical exam was unremarkable. Chest x-ray was normal and ECG revealed T-wave inversions in the inferior leads. Laboratory testing demonstrated normal levels of cardiac troponin. Lipid profile was normal and HbA1C was 5.4%. Echocardiography revealed a left ventricular ejection fraction (LVEF) of 60-65% without regional wall motion abnormalities. The patient underwent nuclear stress testing which was not completed due to a severe reaction to the regadenoson. Rest images were normal. The plan was to perform left heart catheterization (LHC) with coronary angiogram which revealed 80% stenosis of the distal portion of LMCA. Patient was referred for surgery, however given the high suspicion of LMCA spasm in the setting of no CAD risk factors and lack of even mild disease in the other coronaries, the decision was made to obtain a CT coronary angiography which showed normal LMCA without stenosis. Repeated LHC showed normal LMCA, which supported the diagnosis of CAS.

Discussion: CAS can angiographically mimic CAD potentially leading to misdiagnosis and, in cases of LMCA stenosis, inappropriate referral for surgery. CT angiography is a non-invasive tool that can unmask LMCA vasospasm and help prevent unnecessary surgery in such cases. Physicians should have a high suspicion for coronary spasm especially in patients with atypical presentations who lack CAD risk factors and have no coronary atherosclerosis in the unspammed coronary arteries.

## Resident Abstract 18

**Category Submitting for:** Clinical Vignette

**Abstract Title** Serratia: From Military Blunder to Virulent Problem

### **Abstract Text**

*Serratia marcescens* is an uncommon cause of bacteremia. As a rare cause of gram-negative rod cardiac infections (9%), it is an even rarer etiology of cardiac implantable electronic device (CIED) infections. With recurrent bacteremia and a cardiac device, a transesophageal echocardiogram is the main diagnostic test that should be pursued.

An 82-year-old man with history of prior TIA, hypertension, HFpEF (60-65%), CAD with prior PCI, prostate cancer, well controlled diabetes, pacemaker insertion with revision a few months prior, and recent treatment for *Serratia* bacteremia, presented for the second time in two months with neurological symptoms. Five weeks prior, he had been hospitalized for blurry vision and a right visual field deficit. At that time, the initial work-up was unremarkable except for CTA head/neck showed possible left occipital lobe infarct and 68% stenosis of left ICA. Neurology recommended anti-platelet therapy. Trans-thoracic echocardiography was notable for chronic valvular changes. On day 7, the patient had a new fever to 101.5F. CT abdomen, chest x-ray, and urinalysis were unremarkable. Procalcitonin was 0.797. Blood cultures were collected, and he was empirically started on IV vancomycin and IV cefepime. The following day, *Serratia marcescens* bacteremia was confirmed. He was treated with 3 days of IV cefepime with clearance of bacteremia on repeat blood cultures. He was discharged with PO trimethoprim-sulfamethoxazole for a total of 10 days.

Five weeks later, the patient was readmitted for feeling unwell and progressive global weakness. CT head showed remote left, and not previously noted, right cortical infarcts. On day 2, he had repeat fever. Overall fever work-up was only remarkable for blood cultures again growing *S. marcescens*; thus IV Cefepime was started. The work-up for the source of recurrent *Serratia* bacteremia was unrevealing until a transesophageal echocardiogram was completed on day 5 and showed a vegetation on the right atrial pacer lead. Cardiology and cardiothoracic surgery consultants ultimately determined that removal of the cardiac device was too high risk for this patient. Ongoing IV cefepime was continued with plans for at least 6 weeks with possible need for further suppressive therapy.

This case illustrates a relatively rare case of a gram-negative bacteremia secondary to a CIED infection. While guidelines specifically recommend transesophageal echocardiography for persistent bacteremia with *Staphylococcus* species, it is less frequently needed with gram-negative organism bacteremia. Due to increased virulence, there should be strong consideration with two gram-negative organisms: *P. aeruginosa* and *S. marcescens*. Recognition of possible cardiac device infection regardless of organism is critical to making an appropriate treatment plan for any patient with bacteremia.

## Resident Abstract 19

**Category Submitting for:** Clinical Vignette

**Abstract Title** Rapidly progressing *Streptococcus anginosus* mediastinitis with a masquerading deep tissue neck infection

### **Abstract Text**

*Streptococcus anginosus* group (SAG) species remain an uncommon etiology in descending necrotizing mediastinitis, however incidence has been increasing over the last decade. We present a case of rapidly progressing SAG infection causing esophageal edema, retropharyngeal abscess, bilateral pleural empyema, and pericardial involvement despite treating appropriately with antibiotics and surgical debridement.

A 51-year-old male with no previous medical history presented to the emergency room with acute shortness of breath and right sided pleuritic chest pain. Prior to arrival, he experienced worsening sore throat, fever, and vomiting for 10 days. On presentation, pertinent vitals revealed temperature of 37.1 C, pulse rate 92 beats/min, respiratory rate 46 breaths/min with 97% oxygen saturation. On physical exam, he was noted to have oropharyngeal erythema, cervical adenopathy, increased respiratory effort without overt dental issues. Laboratory evaluation showed a white cell count of  $33.6 \times 10^9/L$  (reference range,  $4.0-12.0 \times 10^9/L$ ), serum lactate 1.7 mmol/L (reference range, 0.4-2.0 mmol/L), and serum procalcitonin 20.9 (reference range,  $<0.05 \text{ ng/mL}$ ). Sputum cultures, blood cultures and a respiratory pathogen panel were unremarkable. Intravenous (IV) vancomycin and piperacillin/tazobactam were initiated for broad coverage given concern of sepsis. CT scan of the chest revealed a small loculated left pneumothorax, pneumomediastinum and identified esophageal wall thickening with extraluminal fluid which raised concern for possible esophageal perforation. However, esophagogastroduodenoscopy ruled out perforation. On day of admission, due to continued clinical deterioration, hypotension and respiratory failure, patient was transferred to critical care unit for vasopressor requirement and invasive mechanical ventilation. Subsequent imaging showed a significant left hydropneumothorax and a new right loculated pleural effusion with unchanged pattern of pneumomediastinum. Left-sided chest tube was inserted which revealed purulent fluid with pH 6.83, nucleated cell count 5,141/uL, LDH  $>4,000 \text{ UL}$  and protein 4.2 gm/dL followed by a right-sided chest tube revealing similar fluid analysis. On second day of hospitalization, due to worsening hemodynamic instability, a CT neck was obtained which revealed a 10 x 1.3 cm retropharyngeal abscess. Patient was taken emergently for surgical debridement of retropharyngeal abscess and right lung thoracotomy. Cultures from the operation room isolated *Streptococcus anginosus* and *Peptostreptococcus micros* in the pleural fluid. Based on the culture data, antimicrobials were changed to IV ampicillin-sulbactam, but patient stayed in the critical care unit despite getting multiple surgical interventions, when his hospital stay was further complicated by pericardial effusion.

Identification of SAG mediastinitis requires an aggressive surgical approach since it tends to travel past facial planes and is difficult to treat solely with antimicrobials. Physicians should remain cognizant when clinical recovery is slow since it can raise concern for progressing abscess foci which may lead to prolonged hospitalization and increased mortality.

## Resident Abstract 20

**Category Submitting for:** Clinical Vignette

**Abstract Title** Gut to Heart: A Rare Case of Bacterial Myocarditis

### **Abstract Text**

Salmonella species are an uncommon cause of myocarditis. We present a case of young immunocompetent man with Salmonella bacteremia with myocarditis.

30-year-old man presented to an outside hospital with a four-day history of chills, worsening headache, diarrhea, nausea, and vomiting. In the ED he was noted to have a fever of 103F, mild tachycardia (100-110), normal blood pressure, and elevated troponin 1.12 (normal <0.04). Exam was otherwise normal. Empiric ceftriaxone and azithromycin were initiated for presumed community acquired pneumonia. Initial blood cultures grew Enterobacteriaceae species within 24 hours of collection. The patient insisted on leaving against medical advice. He was transitioned to oral levofloxacin.

Two days later he returned to the outside hospital for evaluation of new onset substernal “burning” and pain associated with cough, and dyspnea. Laboratory evaluation showed a white cell count of 4.2 (reference range, 3.8-10.5 x 10<sup>9</sup>/L), elevated CRP 2.6 mg/dL (normal <1.0) and a troponin of 1.12 ng/mL (normal <0.04). COVID-19 PCR was negative. CT angiogram of the chest and abdomen did not show any acute pathology. Anticipating further cardiac investigation, he was transferred to the tertiary facility.

Once there, work up showed absolute neutrophil count (ANC) of 1500 cells/mm<sup>3</sup>, platelet count of 110 x 10<sup>3</sup>/uL (150-450 x 10<sup>3</sup>/uL), and an up-trending troponin (1.12 to 4.27 ng/mL). Chest pain and dyspnea had improved but he continued to have a non-productive cough. Additional infectious disease work-up included a negative HIV screen, West Nile, and Lyme serology. Procalcitonin was 0.4 ng/mL (normal <0.05). Blood cultures that were collected at the outside facility prior to transfer grew Salmonella paratyphi that was susceptible to ampicillin, trimethoprim-sulfamethoxazole, ceftriaxone, and ciprofloxacin.

Given his persistent cardiac symptoms, despite a normal EKG, transesophageal echocardiogram was performed and showed no signs of endocarditis. A Cardiac MRI demonstrated myocardial edema and delayed enhancement near the apex findings that are concerning myocarditis. He was treated with IV ceftriaxone as inpatient and transitioned to oral cefixime at discharge to complete a course of 14 days.

Salmonella typhi and paratyphi species are the most frequently reported species causing salmonella associated myocarditis but other non-typhoid salmonella (NTS) are also infrequently reported. A high suspicion and low threshold for ECG and troponin, in patients with NTS-infections might reveal more patients with subclinical myocarditis. Cardiac MRI is proven to be a safer, noninvasive diagnostic tool compared to traditional endomyocardial biopsy. Ceftriaxone and fluoroquinolones are preferred drugs of choice. Prompt recognition and initiation of antimicrobial therapy may prevent future morbidity in myocarditis related to bacterial infection.



## Resident Abstract 21

**Category Submitting for:** Clinical Vignette

**Abstract Title** Recurrence of Gastrointestinal Stromal Tumor After Surgical Resection

**Abstract Text**

Gastrointestinal stromal tumor (GIST) is the most common mesenchymal tumor of the gastrointestinal tract. It is most frequently located in the stomach, and the first-line curative treatment is surgical resection. However, there is still a significant rate of recurrence after surgery. This report presents a case of a 76-year-old female who had a recurrence of GIST in her liver 12 years after surgical resection of the primary GIST in her stomach. 5 years after the surgery, CT abdomen did not detect any new tumors. 10 years after the surgery, abdominal ultrasound only showed a liver lesion that biopsy revealed to be NASH. The recurrent GIST was found outside of the standard 5-year imaging follow-up of surgical resection. We argue that follow-up imaging guidelines should be extended past 5 years post-surgery.

## Resident Abstract 22

**Category Submitting for:** Clinical Vignette

**Abstract Title** An Abnormal Presentation of Renal Cell Carcinoma

### **Abstract Text**

#### Introduction

Only 0.2-0.7% of metastatic cancers will affect the stomach. Renal cell carcinoma (RCC) most commonly metastasizes to the lung, bone, and liver. With metastatic RCC to the GI tract, the duodenum is the most frequent location whereas the rectum and stomach are much less frequent, occurring in only 0.2-0.7% of cases. In these patients, 81.8% will present with GI bleeding as their primary symptom. Because it usually spreads via peritoneal seeding, presentation can be delayed up to 20 years. For this reason, the majority of cases with mets from RCC have a prior diagnosis of RCC. Here we present a differing case where a gastric metastasis was the primary finding.

#### Case

A 62-year-old male with a history of colonic polyposis presented for evaluation of 1 week of tarry stools and weight loss. He was found to have acute anemia, so he was referred for EGD. Upper endoscopy revealed a large, ulcerated, non-circumferential mass on the greater curvature of the stomach and a smaller lesion on the lesser curvature. Biopsies were taken of both masses and sent to pathology. Histologic evaluation showed renal cell carcinoma, clear cell type with high nuclear grade. Imaging with CT abdomen/pelvis showed a large right renal mass consistent with renal cell carcinoma. Patient proceeded to have a radical right nephrectomy with pathology confirming the diagnosis of grade 4 clear cell renal cell carcinoma. He was then started on immunotherapy with ipilimumab and nivolumab. It was initially recommended that the patient have subtotal gastrectomy, but repeat scans showed no change in metastatic gastric tumor burden. Subsequently, the patient developed metastasis to the lungs and brain in the ensuing 8 months. The largest brain mass was 2.8cm in the right temporal lobe and associated with midline shift, so he was started on palliative measures.

#### Discussion

In order to make the diagnosis of RCC in patients with a gastric tumor, EGD with a biopsy of the mass is essential. Studies are conflicting regarding the utilization of gross appearance on diagnosing these tumors. When the diagnosis is made, the median survival ranges from 3-6 months, negatively affected by the presence of additional metastatic sites, development of mets in less than 6.3 years from nephrectomy, and grossly protruding tumor.

When a gastric site of metastatic RCC is found, surgical resection is the currently recommended management. One study suggested that 31.8% of metastatic gastric tumors from RCC are potentially resectable. If surgery isn't an option, molecular agents such as sorafenib and sunitinib could be used. As our case shows, RCC with multiple sites of metastasis often leads to increasing morbidity and mortality. Although his gastric tumor responded to molecular therapy, additional sites of spread lead to a worsening prognosis.

## Resident Abstract 23

**Category Submitting for:** Clinical Vignette

**Abstract Title** Pseudogout: Paraneoplastic presentation of Myelodysplastic syndrome (MDS)

### Abstract Text

#### Abstract

Myelodysplastic syndrome (MDS) is often associated with a wide variety of paraneoplastic syndromes but there are very few reported cases of pseudogout as a paraneoplastic manifestation. We present a 79-year-old male who was admitted with sudden onset pain and swelling involving his right knee. The patient also had chronic pancytopenia and we diagnosed him with MDS. The synovial fluid analysis confirmed pseudogout with CPPD crystals. Each of the episodes above responded very well to intravenous and oral steroids. The patient further developed a new onset of myalgias and arthralgias with increased CK, aldolase, and myoglobin raising suspicion for inflammatory myositis but further testing could not be performed due to the patient's refusal for treatment.

#### Introduction

Myelodysplastic syndrome (MDS) has been associated with autoimmune paraneoplastic manifestations, such as vasculitis, IBD, neuropathies, and seronegative inflammatory arthritis. Discussed below is a case of pseudogout manifesting as an auto-immune paraneoplastic syndrome of MDS

#### Case report

A 79-year-old male with a past medical history of hypertension, hyperlipidemia, CAD s/p PCI, and DVT was admitted with chief complaints of right knee pain and chronic worsening pancytopenia. Labs on admission were consistent for pancytopenia. Bone Marrow biopsy confirmed the diagnosis of MDS with 70% hypercellular marrow and 6% blasts. Pan CT scans showed no evidence of metastatic disease. Knee x-rays were obtained showing bilateral effusions with no fractures. Synovial fluid analysis showed CPPD crystals with synovial WBC of 17,000 with no organisms, suggestive of crystal-induced arthritis most likely pseudogout and less likely an infectious process. Lab work for PTH, TSH, Magnesium, Phosphorous, iron, and calcium was unremarkable. The patient was started on intravenous and oral steroids and responded well.

Further, the patient started having diffuse myalgias and arthralgias with no acute changes in imaging and labs. Due to immense pain on ambulation and movement, the patient refused physical therapy and was chronically bed-bound. Further investigation showed an elevated aldolase, CK, and myoglobin suggesting a component of inflammatory myopathy. The patient refused further treatment and investigation and was converted to comfort measures.

#### Discussion

Myelodysplastic syndrome (MDS) is a group of hematological malignancies with clonal hematopoiesis and one or more cytopenias and. Paraneoplastic syndromes happen when T cells mistakenly attack normal cells in the nervous system as a response to malignancy. There are reports describing a variety of paraneoplastic phenomena associated with myelodysplastic syndrome. Pseudogout is associated with excessive cartilage pyrophosphate production, leading to local CPP supersaturation and crystal formation

or deposition. Autoimmune paraneoplastic syndrome and administration of granulocyte colony-stimulating factor (G-CSF) in MDS patients for neutropenia are the two causes reported to manifest pseudogout in MDS.