

2020 ACP Colorado Chapter Meeting

February 6, 2020 thru February 9, 2020

Broadmoor Hotel. Colorado Springs. Colorado



RESIDENT ABSTRACTS

PRESENTED:
FEBRUARY 6, 2020 THRU
FEBRUARY 9, 2020

Name: Evan Cherry, MD

Presentation Type: Oral Presentation

Residency Program: University of Colorado School of Medicine

Additional Authors: Juan Lessing, MD, FACP

Abstract Title: Missed Connection: Imperfect Role of Imaging in Diagnosis of Esophagopleural Fistula

Abstract Information:

A 37 yo man presented with sudden food impaction. After endoscopic disimpaction, CT chest with oral contrast revealed perforation at the GE junction, extraluminal contrast in the Left paraesophageal space, and pneumomediastinum. After emergent endoscopic esophageal stenting, repeat CT chest demonstrated an unexplained Left pleural effusion without contrast enhancement, for which a percutaneous thoracostomy tube was placed. Plain esophagram did not demonstrate stent leak.

Medical management consisted of strict NPO, intravenous fluids, IV PPI, broad-spectrum antibiotics, and thoracostomy drainage with enzymatic disruption of loculated fluid. After 5 days, repeat plain esophagram was negative for esophagopleural fistula. CT chest demonstrated decreased Left effusion volume, but interval development of pneumohydrothorax. Video-Assisted Thoracic Surgery decortication was converted to open thoracotomy due to discovery of vegetable matter in the pleural space. His post-operative recovery was uneventful and the esophageal stent was endoscopically removed one month after thoracostomy. He has returned to work and travelling with his wife.

Discussion:

This case highlights limitations of advanced imaging and implications for nonsurgical management of esophageal perforation.

Esophageal rupture is a surgical emergency, though nonoperative management is emerging as a viable alternative to high-morbidity and high-mortality surgical interventions. Initial management includes NPO, hemodynamic monitoring, surgical evacuation of extra-mediastinal leaks, esophageal stenting, and broad-spectrum antibiotics. (PMID: 16427833, PMID: 25791945)

Chest CT is the preferred imaging modality for stratifying pleural space infections, though esophagopleural fistulas may not be apparent on imaging, especially within the first 24 hours, and can occur irrespective of radiographic mediastinitis (PMID: 3490162, PMID: 20133295, PMID: 28575240). An esophagopleural fistula was not demonstrated on the initial CT prior to esophageal stenting, nor in subsequent CTs and esophagrams. However, vegetable discovered in the pleural space during VATS indicated definite transesophageal migration of food into the pleural space. The esophageal rupture was not contained to the mediastinum and could not be successfully managed nonoperatively.

Understanding the limitations of radiology in diagnosing an esophagopleural fistula is essential to considering this diagnosis. Diagnostic performance of CT scan for esophageal perforation is imperfect for detecting leakage of contrast into pleural space (Sensitivity 55.6%, Specificity 96.3%, PPV 71%, NPV 92.9%, +LR 15, -LR 0.46) or detecting an over esophageal fistula (Sensitivity 11.1%, Specificity 100%, PPV 100%, NPV 87.1%, +LR N/A, -LR 0.89) (PMID: 24748526). In retrospect, a higher clinical suspicion was warranted for esophagopleural fistula as a cause for the effusion. Surgical intervention was delayed because of negative imaging. Meanwhile, the patient clinically deteriorated. Eventual development of hydropneumothorax necessitated decortication. His complete recovery validated surgical intervention for pleural effusion with distal esophageal perforation.

Name: Rachel Chung, DO **Presentation Type:** Oral Presentation

Residency Program: Sky Ridge Medical Center

Additional Authors: P. Jenkins, P. Judiscak, S. Moreland, D. Scherbak

Abstract Title: Inappropriate antibiotic use in the emergency department and the 2016 Surviving Sepsis Campaign Guidelines

Abstract Information:

Introduction:

The IDSA did not endorse the 2016 Surviving Sepsis Campaign guidelines. One of their concerns was that the one hour window from the recognition of sepsis to antibiotic therapy might lead to inappropriate antibiotic use. The aim of this study is to test the hypothesis that the 2016 Surviving Sepsis Guidelines have led to an increase in inappropriate antibiotics prescribed in the ER.

Materials and Methods:

We conducted a retrospective cohort study of all inpatients aged 18 to 89 years of age admitted to Sky Ridge Medical Center between June 2016- January 2017 and January 2017- April 2019. We performed a power calculation using a G- power for Fischer's exact test, assuming power of 0.8, alpha of .05 and a two tailed test. In order to find a minimum 15% statistically significant difference in antibiotic prescribing between groups, we needed 2,562 patients. Patients were included if they fulfilled HCA accepted SIRS criteria for sepsis and received an antibiotic on day 0. Patients were excluded if they met criteria for septic shock, received a vasopressor, or were diagnosed with respiratory failure. The cohort was split into group A, seen in the ER prior to the publication of Surviving Sepsis Campaign 2016 Guidelines (June 2016-January 2017), and group B, seen after the guideline (January 2017- April 2019). An antibiotic was appropriate if SIRS criteria were fulfilled and there was an ICD-10 code of an infection linked to the visit. If an antibiotic was given without a diagnosis of infection, it was inappropriate. Fisher's Exact test was used for data analysis.

Results:

Of Group A (N= 1050), 25.42% of patients met SIRS criteria for sepsis but received an inappropriate antibiotic and 9.6% did not meet SIRS criteria and received an inappropriate antibiotic. Of group B (N=1512), 27.58% of patients met SIRS criteria and received an inappropriate antibiotic, and 8.0% did not meet SIRS criteria and received an inappropriate antibiotic. Fischer's exact test of the difference between the two groups fulfilling SIRS criteria was not statistically significant (p=0.54). Fischer's exact test of the difference between groups not fulfilling SIRS criteria was not statistically significant (p=0.56).

Conclusion:

These results provide evidence that the 2016 Surviving Sepsis Campaign guidelines did not significantly affect antibiotic practices in the ER. We found that 25-27% of patients received

inappropriate antibiotics in the ER. These findings are lower than previous studies of antibiotic prescription practices in the ER. The discrepancy is likely due to our loose definition of appropriate antibiotics. We plan to perform a much larger study with 130,000 patients over a larger time-frame and across multiple hospitals to improve statistical power.

Name: William Grigg, **Presentation Type:** Oral Presentation

Residency Program: Parkview Medical Center

Additional Authors: Dhamista Chaudhary, MD, Bhavani Suryadevara, MD

Abstract Title: What to do when WATCHMAN Fails?

Abstract Information:

Introduction:

For patients that are not candidates for long-term blood thinners until recently there has been little choice but risk vs reward discussion. With the introduction of WATCHMAN this no longer just a discussion but now a procedure. The problem arises when failure occurs, what are the next steps when a patient “breaks through” and WATCHMAN fails? This case highlights an 88-year-old male with a history of Atrial Fibrillation (Afib) s/p WATCHMAN device placement in the fall 2018; was on Xarelto for 2 years prior to watchman device placement. Previously, a TEE done 45 days post watchman device placement in Nebraska had shown a leak, so patient was continued on Eliquis for 6 months post implantation. Repeat TEE done after 6 months of implantation showed a well-functioning device so anticoagulation was stopped.

Case Description:

He presented to the hospital on 4/19/19 with focal neurological deficits characterized by RUE weakness, dysarthria and difficulty with comprehension. He was given tPA and transferred to ICU. His symptoms eventually resolved completely. Work-up in the form of CT-Head did not show any acute intracranial findings, MRI-Brain did not show any infarct and head and neck CTA were unremarkable. Cardiology was consulted to give recommendations on anticoagulation as the patient had a TIA despite watchman device. Cardiology started the patient on Eliquis, stopped the Plavix, continued ASA and performed TEE to reassess the device: TEE showed a device leak of 0.68 cm despite endothelialization of the device. It was likely that the device size was chosen wrong and it's conceivable that there was a clot in the left atrial appendage that went around the watchman device causing TIA. Cardiology recommended continuing Eliquis lifelong for secondary prevention of TIA/stroke.

Discussion:

This case illustrates the potential for continued anticoagulation and possible need for continued imaging in patients with WATCHMAN long after the recommended 45 days post procedure. This case also high lights the need for further investigation and the need for guidelines on what to do when a patient fails WATCHMAN and what and what the anticoagulation of choice should be.

Name: Jason John, MD

Presentation Type: Oral Presentation

Residency Program: University of Colorado Medical School

Additional Authors: Bill Quach, MD, Erin Bredenberg, MD

Abstract Title: More Than a Fluke

Abstract Information:

A 60-year-old African woman presented to the emergency department with three days of abdominal pain, nausea, and vomiting. She had recently returned from a ten-month trip through rural Ethiopia where she consumed unpasteurized milk and well water with exposure to goats, cats, and livestock. Her medical history was otherwise unremarkable.

On exam, she was tachycardic (110 bpm), hypotensive (53/34 mm Hg), and afebrile. Her exam was also notable for midline abdominal tenderness. Laboratory findings were notable for white blood cell count of $22 \times 10^9/L$ without eosinophils, hemoglobin of 13.3 g/dL, platelet count of $35 \times 10^9/L$, total bilirubin of 1.4 mg/dL, direct bilirubin of 0.6 mg/dL, alkaline phosphatase of 143 U/L, aspartate aminotransferase of 38 U/L, alanine aminotransferase of 40 U/L, and lactate of 8.3 mmol/L. Blood cultures on admission grew *Klebsiella pneumoniae*. Non-contrast CT of the abdomen was notable for hepatomegaly with cavitory lesions in the left lobe of the liver consistent with abscesses, pneumobilia with biliary ductal dilation, and a hydropic gallbladder. Percutaneous drainage of the gallbladder and left liver abscess was performed with frank pus aspirated from the abscess. MRCP revealed tubular filling defects in the dilated extrahepatic biliary ducts.

ERCP revealed a choledochoduodenal fistula with a foreign body versus parasite visible in the common bile duct. Balloon sweep of the bile duct yielded sludge followed by passage of a 13-inch roundworm. The worm was retrieved and sent to infectious disease, where it was identified as *Ascaris lumbricoides*.

Ascaris lumbricoides infection is relatively common and is estimated to affect up to 800 million people globally, primarily in Asia. Transmission occurs via contaminated water or soil with up to a 24-month incubation period in the intestines. Although eosinophilia is classically associated with parasitic infections, the absence of eosinophilia does not rule out *Ascaris* infection. Hepatic abscesses and bacteremia as a result of *Ascaris* infection have only been described in one other case report in the literature.

The patient improved markedly and rapidly after biliary decompression and was then treated with two doses of oral albendazole. She passed additional *Ascaris lumbricoides* in her stool during the hospitalization. She was discharged on oral Augmentin with plans for drain removal six weeks later. At the two-week follow-up, she was symptom-free and clinically improved.

Name: Amanda Johnson, MD

Presentation Type: Oral Presentation

Residency Program: University of Colorado School of Medicine

Additional Authors: Deepa Ramadurai, MD, Chad Selph, MD

Abstract Title: The crowned dens syndrome: a rare cause of neck pain and fever

Abstract Information:

Introduction: Crowned dens syndrome (CDS) is a rare cause of neck pain due to calcium pyrophosphate crystal deposition (CPPD) along the atlanto-axial articulation. The syndrome occurs most commonly in older patients and classically presents with acute to subacute neck pain, rigidity, and fever. Inflammatory markers are often elevated and diagnosis is made by characteristic computed tomography (CT) findings of calcium deposition around the transverse ligament of the atlas. In most cases, pain responds well to non-steroidal anti-inflammatory drugs (NSAIDs), colchicine, or steroids.

Case Report: A 76-year-old woman presented with one week of neck pain and fevers. Past medical history was significant for chronic kidney disease (CKD) and chronic hypomagnesemia. On exam, she was febrile to 38.4 degrees Celsius with pulse of 110 beats per minute and blood pressure of 130/80 mmHg. She was tender along the left trapezius muscle, with full active and passive range of motion of the neck, no nuchal rigidity, and a non-focal neurologic exam. Labs were notable for a white blood cell count of 17.5 k/uL and lactate of 1.2 mmol/L. Erythrocyte sedimentation rate (ESR) was significantly elevated at >80 mm/h and C-reactive protein (CRP) was 96.1 mg/L. Empiric antibiotics for meningitis were initiated. Computed tomography (CT) of the head was unremarkable and lumbar puncture revealed clear cerebrospinal fluid with protein of 40 mg/dL, 0 white blood cells, 0 red blood cells, and no xanthochromia. Closer review of a CT of the neck revealed calcium pyrophosphate deposition along the atlanto-axial joint, specifically the dens, diagnosing crowned dens syndrome. Antibiotics were discontinued and prednisone was initiated with prompt resolution of her neck pain.

Discussion: It is important to consider and recognize CDS, given the significantly different management compared to other entities that present similarly including meningitis and temporal arteritis.

While still relatively rare, the presence of CDS may be underrecognized. A recent retrospective study at the University of Kansas noted 60% of patients with known or probable CPPD that had undergone imaging had findings consistent with CDS. Furthermore, CDS has a higher predominance in older individuals, with a median age of onset around 71.4. Particularly notable is that incidence of chondrocalcinosis increases with age, reaching 50% by the age of 80. Many cases remain asymptomatic.

Our patient had additional risk factors for CPPD, which in turn, increase her risk of CDS. CPPD has been associated with CKD and disorders of magnesium. Hypomagnesemia specifically may lead to excess extracellular concentrations of inorganic phosphates and subsequent crystal formation, as magnesium is a cofactor for alkaline phosphatase, which

otherwise maintains phosphorus homeostasis.

Treatment of CDS with steroids, NSAIDs, or colchicine should invoke rapid resolution of symptoms, as seen in our patient. Nephrology continues to manage her CKD and hypomagnesemia.

Name: Ayal Levi, MD

Presentation Type: Oral Presentation

Residency Program: Saint Joseph Hospital

Additional Authors: Alexander W. Steinberg MD

Abstract Title: A Headache of Discordant Labwork

Abstract Information:

Introduction:

Cerebral Spinal Fluid (CSF) analysis remains the standard of care in confirming or rejecting suspected bacterial meningitis.^{1,2} Normal CSF findings in this infection are exceedingly rare in immunocompetent adults.^{2,3,4} Previous literature suggests that a CSF white blood cell count of $>100 \times 10^6$ cells/L is diagnostic of bacterial meningitis.⁴ We present a case of a symptomatic woman with normal CSF analysis but PCR positive for *Haemophilus influenzae* who responded to antibiotic treatment.

Case:

A thirty-seven-year-old previously healthy woman presented to the Emergency Department complaining of headache, increasing fatigue, neck pain, and low back pain that began the prior evening. She awoke the morning of admission with worsening neck stiffness, pain that radiated down her back, a severe headache, photophobia, nausea, and vomiting. There were discrepancies between her and her mother's report of her vaccination history. Physical exam was significant for occipital tenderness, limited range of motion in her neck, and positive Kernig's and Brudzinski's signs. Initial labs were unremarkable and CSF studies showed a normal glucose, protein, opening pressure, and only one nucleated cell. CSF PCR was, however, positive for *H. influenzae*. She was hospitalized for meningitis and started on intravenous Ceftriaxone. Blood and CSF cultures were negative. The patient had symptomatic improvement over the following days. A midline IV was placed and she was discharged on day five of hospitalization to complete her antibiotic course at home. She was seen in clinic following discharge and had made a complete recovery. CSF and blood cultures remained negative at fourteen days.

Discussion:

A recent review demonstrated 124 reported cases of bacterial meningitis without pleocytosis on CSF analysis, with seventeen cases due to *H. Influenza*.² Of these cases, twelve had initially positive CSF cultures; four had negative CSF cultures but positive blood cultures; one had negative CSF and blood cultures and the diagnosis was made by PCR.² Traditional CSF analysis has a positive predictive value ranging from 68% to 98% and a very high negative predictive value of 95% to 100%.^{4,5} The reported positive predictive value of CSF PCR ranges from 89% to 100%. PCR has a strong negative predictive value of nearly 100%.^{6,7,8} Despite the high reported negative predictive value of traditional CSF analysis, it has been established that bacterial meningitis can present with normal CSF, and therefore can delay proper treatment. Meningitis without CSF pleocytosis is rare but potentially fatal, since it presents an increased risk of going untreated.^{2,4,9,10} This case is a poignant reminder that each test result may be misleading and

delivering excellent care depends on integration of all of the data into a comprehensive clinical assessment. We believe that faced with these discordant results in the setting of possible morbid bacterial meningitis, an unknown vaccination history, and a classical clinical presentation, aggressive antibiotic treatment is non-negotiable. Therefore we continue to use PCR in addition to traditional CSF studies and culture data to diagnose and guide treatment in patients with suspected meningitis.

References:

1. Tunkel A, Hartman B, Kaplan S et al. Practice Guidelines for the Management of Bacterial Meningitis. *Clinical Infectious Diseases*. 2004;39(9):1267-1284. doi:10.1086/425368
2. Troendle M, Pettigrew A. A systematic review of cases of meningitis in the absence of cerebrospinal fluid pleocytosis on lumbar puncture. *BMC Infect Dis*. 2019;19(1). Doi:10.1186/s12879-019-4204-z
3. Hayward R, Shapiro M, Oye R. LABORATORY TESTING ON CEREBROSPINAL FLUID A Reappraisal. *The Lancet*. 1987;329(8523):1-4. doi:10.1016/s0140-6736(87)90698-2
4. Brouwer M, Thwaites G, Tunkel A, van de Beek D. Dilemmas in the diagnosis of acute community-acquired bacterial meningitis. *The Lancet*. 2012;380(9854):1684-1692. doi:10.1016/s0140-6736(12)61185-4
5. Manning L, Laman M, Mare T, Hwaiwhanje I, Siba P, Davis T. Accuracy of cerebrospinal leucocyte count, protein and culture for the diagnosis of acute bacterial meningitis: a comparative study using Bayesian latent class analysis. *Tropical Medicine & International Health*. 2014;19(12):1520-1524. doi:10.1111/tmi.12400
6. Tzanakaki G, Tsopanomichalou M, Kesanopoulos K et al. Simultaneous single-tube PCR assay for the detection of *Neisseria meningitidis*, *Haemophilus influenzae* type b and *Streptococcus pneumoniae*. *Clinical Microbiology and Infection*. 2005;11(5):386-390. doi:10.1111/j.1469-0691.2005.01109.x
7. Saravolatz L, Manzor O, VanderVelde N, Pawlak J, Belian B. Broad-Range Bacterial Polymerase Chain Reaction for Early Detection of Bacterial Meningitis. *Clinical Infectious Diseases*. 2003;36(1):40-45. doi:10.1086/345438
8. Chakrabarti P1, Das BK, Kapil A. Application of 16S rDNA based seminested PCR for diagnosis of acute bacterial meningitis. *Indian J Med Res*. 2009 Feb;129(2):182-8.
9. Harrison L, Trotter C, Ramsay M. Global epidemiology of meningococcal disease. *Vaccine*. 2009;27:B51-B63. doi:10.1016/j.vaccine.2009.04.063
10. Hase R, Hosokawa N, Yaegashi M, Muranaka K. Bacterial Meningitis in the Absence of Cerebrospinal Fluid Pleocytosis: A Case Report and Review of the Literature. *Canadian Journal of Infectious Diseases and Medical Microbiology*. 2014;25(5):249-251. doi:10.1155/2014/568169

Name: Kristina Barber, MD

Presentation Type: Poster Presentation

Residency Program: Saint Joseph Hospital

Additional Authors: Sagarika Shina

Abstract Title: ACUTE DEMYELINATING ENCEPHALOMYELITIS FOLLOWING MYOPLASMA MUCOSITIS

Abstract Information:

Introduction:

Acute demyelinating encephalomyelitis (ADEM) is a rare yet severe condition generally seen in pediatric patients following infection or vaccination. Here, we present a case of ADEM in a young female patient following multifocal *M. pneumoniae* infection.

Case Description:

An otherwise healthy 27-year-old female with a remote history of viral encephalitis presented with fevers, cough, headache, non-bleeding ulcers of her oral mucosa, and “foreign body” sensation of her eyes. She was diagnosed with mycoplasma pneumonia and mucositis based on respiratory viral panel and dermatology consultation respectively. Shortly after admission and initiation of doxycycline, she developed rapidly ascending paralysis requiring intubation. Initial neuraxial MRI showed extensive T2 hyperintensity of the corpus callosum, periventricular region, thalami, midbrain, pons, and anterior upper brain stem with many foci of enhancement and restricted diffusion and others with microhemorrhage and necrosis consistent with ADEM or hyperacute variant, acute hemorrhagic leukoencephalitis (AHLE). She was initially treated with IVIG and high dose steroids. Showing no improvement, she next received 5 days of PLEX followed by another 5 days of IVIG, still with little change. Repeat MRI 14 days later showed mild improvement in the enhancement and hemorrhagic appearance of the extensive lesions; however, the patient had no improvement in neurologic function.

Discussion:

ADEM is a rare inflammatory demyelinating condition seen mostly in the pediatric population. It is typically triggered by infection or vaccination and is generally monophasic and self-limited but can be severe. ADEM is diagnosed based on a constellation of clinical and MRI findings like those described for our patient. A hyperacute variant called AHLE shows similar MRI findings though with additional hemorrhagic features and fibrinoid necrosis of vessel walls on autopsy. In both conditions, an overwhelming inflammatory response leads to edema and perivascular infiltration of inflammatory cells that destroy myelin leading to

multifocal neurologic sequelae. There are a handful of reported cases of ADEM and AHLE following *M. pneumoniae* infection, generally in patients with only pulmonary involvement. While neurologic complications are known to exist with *M. pneumoniae*, they are thought to be caused by the unmodulated immune response rather than direct invasion of bacteria. To our knowledge, this is the first case reported of a patient with mycoplasma mucositis developing ADEM. Unfortunately, despite receiving the standard treatment for ADEM including high dose steroids, IVIG, PLEX, and antibiotics, our patient showed little improvement in her neurologic function. Since this is such a rare presentation of an already uncommon condition, prognostication remains incredibly difficult.

Name: Natalie Beck, MD

Presentation Type: Poster Presentation

Residency Program: University of Colorado Medical School

Additional Authors: Karen Marsden, MD; Juan Fernando Martinez, MD; Francisco La Rosa, MD

Abstract Title: Calcific Uremic Arteriopathy: A Painful Diagnosis to Miss

Abstract Information:

Introduction: Calcific uremic arteriopathy or "calciophylaxis" manifests as painful skin necrosis, most classically in patients with end-stage renal disease and carries a high mortality rate. We describe a case in which this diagnosis presented as penile necrosis.

Case: A 54 year-old man with a history of end-stage renal disease on peritoneal dialysis, secondary hyperparathyroidism taking ergocalciferol, calcitriol and cinacalcet, atrial fibrillation on warfarin and coronary artery disease with prior coronary stenting was transferred to our hospital with a penile ulcer concerning for necrosis.

One month prior he noted penile pruritis and progressively developed a blackened, tender area on the glans penis. He presented to care multiple times and was treated with antibiotics and antifungals without relief. He had no history of sexually-transmitted infections, had not had sexual intercourse in the past several years and denied any recent trauma or urologic procedures.

On admission, his vital signs were within normal range. Physical exam revealed a tender, black ulceration on the glans penis weeping serous fluid. Laboratory data was notable for hyperphosphatemia, uremia, and therapeutic INR; he had a negative treponemal antibody, HIV, and acute hepatitis viral panel. The initial admitting provider was concerned for primary penile neoplasm versus ischemia and pursued Doppler ultrasound which showed calcifications throughout both cavernosal arteries with absent arterial flow. The urology team was notified and prepared the patient for partial penectomy the following evening. The nephrology team was consulted to arrange peritoneal dialysis and were concerned for calcific uremic arteriopathy. When prompted, the patient had recalled undergoing treatment for this in the past with involvement of his anterior leg. He was started on phosphorus binders and increased cinacalcet dosing, but due to prior intolerance of sodium thiosulfate this medication was held.

After surgery the patient declined peritoneal dialysis due to post-operative discomfort and unfortunately the following day suffered a PEA arrest and could not be resuscitated. Laboratory data returned and was notable for an increased calcium-phosphate product and hyperkalemia. Surgical pathology of the penis resulted post-mortem, showing extensive necrosis with fibrosis and calcification of the blood vessel media consistent with calciophylaxis.

Discussion: Calcific uremic arteriopathy is a diagnosis not to be missed in a patient with end-stage renal disease with evidence of skin necrosis and should remain on the differential diagnosis as a cause of superficial ischemia. Risk factors include warfarin use, vitamin D

supplementation, and calcium-based phosphorus binders, some of which were being taken by this patient. Treatment includes administration of sodium thiosulfate, decrease in the calcium-phosphate product and removal of the offending agent(s). With symptoms present for one month, earlier diagnosis may have resulted in a better outcome for this patient, who unfortunately required penectomy and later suffered a cardiac arrest.

Name: James Fitman, DO

Presentation Type: Poster

Presentation

Residency Program: Sky Ridge Medical Center

Additional Authors:

Abstract Title: A Classic Case of the Not-So-Classic Acute Phlegmonous Gastritis

Abstract Information:

Introduction:

Acute phlegmonous gastritis (PG) is a rare clinical condition characterized by diffuse inflammation and thickening of the gastric wall secondary to infection. These infections are often severe, causing gastric wall necrosis and peritonitis resulting in high mortality. Much of the clinical information about PG to date revolves around case reports given the rarity and ill-defined causes and risk factors in patients who develop this condition. This is the case of a young 35-year-old male who was found to have acute phlegmonous gastritis at SJH.

Case Presentation:

A 35-year-old male with PMHx of obesity and hypertension presented to the ED for 2 days of acute onset of constant epigastric abdominal pain, nausea/vomiting, and diarrhea with melanic stools. On arrival, he was found to be in severe sepsis with a leukocytosis to 25,000 and significantly elevated lactate of 5.9. Non-contrast CT revealed marked gastric wall thickening without evidence of perforation. Sepsis protocol was initiated and Zosyn was started for antimicrobial therapy. Intravenous PPI therapy was started for suspected UGIB. An EGD was scheduled for the next day however the patient developed worsening sepsis and respiratory distress secondary to aggressive volume resuscitation. Blood cultures became positive for streptococcus pyrogens and the patient was transferred to the ICU with EGD placed on hold. The antibiotics were transitioned to Unasyn and clindamycin at the direction of infectious disease. Surgery followed as if he acutely decompensated, he would have to undergo gastrectomy. Fortunately, he was stabilized and improved markedly with resolution of abdominal pain and sepsis over the next few days. EGD was performed a few days later in the stay which revealed diffusely moderately erythematous mosaic patterned mucosa without bleeding. Endoscopic biopsy did reveal a second organism (*Enterobacter aerogenes*) but this was thought to be a contaminant species given such clinical improvement with current antimicrobial therapy. Ultimately, he completed Unasyn therapy and sent home to complete three months of oral PPI.

Discussion:

While acute phlegmonous gastritis is a rare disorder, it is important to maintain clinical suspicion of PG given the remarkable severity of the presentation with case reported mortality outcomes close to 50% even with appropriate antibiotic treatment. Unfortunately, this is rather difficult given the non-descript symptoms with which these patients present. PG is also particularly important given it can affect a wide range of ages, possibly causing the loss of years of life (as was the case of our 35-yr-old). Early detection is crucial given the potential morbidity of this condition; if antimicrobial therapy is ineffective, patients typically must undergo significant, life-altering procedures such as total gastrectomy. Further understanding of the pathogenesis and risk factors associated with development of PG are needed to help better identify patients with this disorder.

Name: Stephanie Franquemont, DO **Presentation Type:** Poster Presentation

Residency Program: Parkview Medical Center

Additional Authors: Ricki Kumar, DO; Teresa Braden, DO

Abstract Title: A social switch from a grim to hopeful prognosis

Abstract Information:

Introduction:

Social history is a required but often neglected portion of a patient's history. We present a case of a 60-year-old male who presented with gastrointestinal complaints and was ultimately found to have a mass that routine preventative screening may have prevented.

Case Description:

Patient is a 60-year-old male with past medical history of type 2 diabetes and anxiety presented to the emergency department with complaints of intractable nausea with episodes of vomiting and diarrhea. He also reported chronic diabetic foot ulcers and unintentional weight loss of approximately 40lbs over the last year. Social history on admission included past smoking history, occasional alcohol use, and no marijuana or illicit drug use. Physical exam revealed a thin, anxious man with epigastric tenderness and left supraclavicular lymphadenopathy. A CT abdomen/pelvis revealed a 4.8x7.5x4cm pancreatic mass as well as retroperitoneal lymphadenopathy. The patient had a family history of pancreatic adenocarcinoma in his mother and, knowing the grim prognosis of pancreatic adenocarcinoma, he was unsure if he would want treatment. He underwent an endoscopic biopsy of the pancreatic parenchymal mass. Pathology revealed diffuse large B-cell lymphoma (DLBCL). As DLBCL is an AIDS defining illness, a more thorough sexual history was discussed; the patient reported having unprotected sex with both men and women at least 10 years prior to presentation. Review of outpatient records showed that HIV testing had been ordered more than two years prior but the patient had never had the testing done. During this hospital stay, HIV testing was completed and found to be positive with a CD4 count of 119. Testing for hepatitis B and C were negative but syphilis was positive. The patient was started on antiretroviral therapy and chemotherapy. Unfortunately, the patient passed away approximately one month after diagnosis and initiation of treatment.

Discussion:

The risk of developing non-Hodgkin lymphoma, including DLBCL, is increased with HIV. It is one of the AIDS defining malignancies along with Kaposi sarcoma and cervical cancer^{1,2}. Diffuse large B-cell lymphoma has a good prognosis but the mortality increases with the degree of immunosuppression from AIDS². Recommendations for screening of HIV and other sexually transmitted infections in patients with high-risk sexual behaviors should be done but does not delineate the frequency with which repeat screening should be done³. Our patient had a distant history of high-risk sexual activity but his medical chart only indicated that he was not sexually active. Screening was not done (even when ordered years prior) as the patient did not feel it was necessary. A more robust social history may have led to further discussion of the recommended screening and earlier treatment that may have prevented development of an AIDS defining malignancy.

Name: Maksim Grachev, DO

Presentation Type: Poster Presentation

Residency Program: Parkview Medical Center

Additional Authors: Widian K. Jubair, MD; Jacob Mathew Jr., DO FACP; Irina Vancea, MD

Abstract Title: Severe Lower Extremity Weakness in Amiodarone Induced Thyrotoxicosis.

Abstract Information:

Introduction:

BRASH (bradycardia, renal failure, AV node block, shock, Hyperkalemia) syndrome is a relatively new term describing the onset of renal failure leading to hyperkalemia and accumulation of ingested atrioventricular (AV) nodal blocking agents which culminates in bradycardia and hypovolemic shock. This syndrome is treated with the correction of the underlying electrolyte derangement, often the hyperkalemia and cessation of the AV nodal blockers, along with chronotropic or inotropic support if necessary. While previously reported in patients with underlying chronic kidney disease, this case represents the first literature developing in a patient without known kidney disease. (Diribe & Le, 2019)

Case Description:

The patient is a 72-year-old male with a past medical history of coronary artery disease, heart failure with reduced ejection fraction, and atrial fibrillation rate controlled with metoprolol who presented to an outside facility with acute renal failure, hyperkalemia, hypotension and back pain. One week prior to his presentation, he was diagnosed with a urinary tract infection and prescribed trimethoprim/sulfamethoxazole. At home he had noticed his systolic blood pressures to be with 70s along with a profound feeling of fatigue and reduced urine output prompting his presentation to the ER. His hospitalization was complicated by profound renal failure with a serum creatinine of 11 from baseline of 0.8, oliguria, hyperkalemia, and hypotension responsive to intravenous (IV) crystalloid fluid resuscitation. Urgent dialysis was not deemed necessary, and he was stabilized with IV calcium, IV insulin with dextrose and Kayexalate in response to his hyperkalemia. Given the patient presented to our institution prior to completing his outpatient antibiotic, trimethoprim/sulfamethoxazole was held due to the patient's renal failure and transitioned to ceftriaxone. He gradually improved clinically, his blood pressure stabilized and his renal dysfunction resolved. He was subsequently discharged with close follow-up with nephrology, primary care.

Discussion:

The presentations of BRASH Syndrome revolves around a trigger inducing acute renal failure which leads to hyperkalemia and decreased renal excretion of AV nodal blocking agents which causes worsening bradycardia, hypotension, and shock. In our case, the trigger appeared to be the patient's antibiotic, Trimethoprim/Sulfamethoxazole, which caused an acute renal insult leading to the downstream effects described above. Ultimately, discontinuation of the offending agents, hemodynamic support and electrolyte management lead to resolution of the syndrome however pressor support may be required in patients not responding to conservative measures. Early recognition of BRASH syndrome

and recognizing the constellation of symptoms may lead to quickly discontinuing the offending agent allowing hemodynamic improvement without requiring dialysis or inotropic support.

Name: Danial Hankins, DO

Presentation Type: Poster Presentation

Residency Program: Saint Joseph Hospital

Additional Authors: Brian Daily, MD

Abstract Title: A Monster Arrhythmia

Abstract Information:

Introduction:

Energy drinks are increasingly popular in young adults and a rising cause of ED visits for arrhythmias and ACS, often when they are consumed in large quantities or in combination with alcohol. A systematic literature review (Goldfarb 2014) revealed 17 published cases of cardiovascular disorders caused by energy drink consumption including 4 cases of Afib, 2 cases of SVT, 1 case of TdP, 4 cases of Vfib, 4 cases of STEMI, and one cardiac arrest. We present a case of inpatient VT due to consumption of Monster energy drink.

Case Description:

- Overnight a rapid response was called by the cardiac telemetry station for a patient in sustained VT
- On arrival he was a 29 year old man who was alert, BP 104/63, HR 170, he was playing video games in no acute distress, and he complained of palpitations. He reported having recently consumed a Monster energy drink
- He was hospitalized for a CF exacerbation, and he reported a history of palpitations after consuming Monster energy drinks
- Rhythm strip showed a wide complex tachycardia (see rhythm strip below)
- A review of the EMR and his previous EKGs revealed no previous EKGs with delta waves
- We considered giving adenosine to differentiate VT and SVT. After reviewing his rhythm strip with the Critical Care and Cardiology attendings we concluded, based on Brugada criteria, that he was in VT (we will discuss this in our poster). We administered a 150mg bolus of amiodarone over 10 minutes, and 5 minutes into the bolus he converted to NSR
- He reported that he had 2 additional Monster energy drinks in his room and was planning on drinking them later that night; we declined his request.

Discussion:

Energy drinks are increasing in popularity, but their consumption is associated with ACS and deadly arrhythmias. They contain large amounts of caffeine, which is well established as an arrhythmogenic agent, but some research suggests energy drinks pose a greater risk of arrhythmias than would be expected by their caffeine content alone (Fletcher 2017).

Distinguishing VT from SVT can be difficult, and the 1991 Brugada criteria and 2007 Verecke algorithm can be helpful.

Name: Peter Hyson, MD

Presentation Type: Poster Presentation

Residency Program: University of Colorado Medical School

Additional Authors: M. Zipse MD, A. Charlton BA, T. Trujillo PharmD, C. Shakowski PharmD, R. Page PharmD, B. Fleeman MD, N. Mantini MD, B. Miller MD

Abstract Title: Uninterrupted Apixaban Increases Intraprocedural Heparin Requirement During Catheter Ablation of Atrial Fibrillation

Abstract Information:

Purpose: Therapeutic heparinization during catheter ablation of atrial fibrillation (AF) is critical to reducing the risk of periprocedural stroke. Patients on uninterrupted direct oral anticoagulants (DOACs) may require adjusted dosing of heparin compared to warfarin, and delays in reaching therapeutic ACTs due to heparin underdosing could increase potential for adverse events. We sought to evaluate the effects of Apixaban (A), Dabigatran (D), Rivaroxaban (R), and Warfarin (W) on intraprocedural heparin dosing in AF ablation.

Methods: 256 consecutive patients undergoing AF ablation on uninterrupted DOAC or W were identified. Procedural details with heparin dosing and ACTs were collected. Statistical analysis was performed utilizing one-way ANOVA with pairwise comparisons of results by the Tukey post hoc test.

Results: The mean age was 64, and 97/256 (38%) were female. AC therapy included: A (n=35), D(n=142), R(n=25), and W (n=54). Mean baseline ACT was lowest for A (147 s) and R (157 s), versus D (212 s) and W (209 s) ($p < 0.05$). High outlier baseline ACTs were observed in 5 D patients (445-582 s). Time to achieve target ACT (>350 s) was longest for A (130.5 min), compared to R (76.5 min), D (70.8 min), and W (71.3 min) ($p < 0.05$). Heparin required to achieve therapeutic ACT was highest in A (259.8 u/kg) compared to R (197.2 u/kg), D (165.4 u/kg), and W (184.2 u/kg) ($p < 0.05$); total intraprocedural heparin was also highest in A (352.8 u/kg) compared to R (313.5 u/kg), D (293.8 u/kg) and W (260.6 u/kg) ($p < 0.05$). 19 patients never achieved target ACT: A 8.6%, D 6.3%, R 0%, and W 7.5%.

Conclusions: In comparison with D, W, and R, use of A required more (>30 u/kg) intraprocedural heparin with delay in reaching target ACT. Use of R also showed nonsignificant trend toward higher time to therapeutic ACT and higher heparin use. This suggests a class effect of uninterrupted Xa-inhibitors on therapeutic AC with heparin which merits further study and DOAC-specific heparin dosing protocols.

Name: Shabnam Khoie, DO **Presentation Type:** Poster Presentation

Residency Program: Sky Ridge Medical Center

Additional Authors: Dmitriy Scherbak, DO

Abstract Title: Adrenocortical Carcinoma, A Stressful Diagnosis

Abstract Information:

Introduction:

Adrenocortical carcinoma (ACC) is a rare and biologically aggressive malignancy. The incidence of ACC is between 0.7-2 cases per million, accounting for 0.05-2% of all malignant tumors. Cortisol-producing carcinomas are the most common, with clinical features consistent with hypercortisolism: refractory hypertension, glucose intolerance, and central obesity.

We present a case of a 75-year-old male with a cortisol-secreting variant of adrenocortical carcinoma.

Case Description:

A 75 year-old male with a prior medical history significant for refractory hypertension on multiple anti-hypertensives including hydrochlorothiazide, type two diabetes mellitus, and untreated obstructive sleep apnea, presented to the emergency department with a blood pressure of 259/123 and in respiratory distress. His work up included a chest CTA, revealing a heterogeneous slightly lobulated, macroscopic fat-containing mass in the left adrenal gland, measuring 4cm. This was interval increase from 2.1cm five years ago; radiology favored this to represent either an adrenal myelolipoma or adenoma.

Nephrology was consulted for management of his hypertension and assessment of the adrenal mass. Total urine metanephrines were 505 mcg/24h and total urine catecholamines were <60 mcg/24h, inconsistent with pheochromocytoma. CTA abdomen/pelvis revealed no evidence of renal artery stenosis. His mild hypokalemia was attributed to thiazide use. At discharge, he started spironolactone in lieu of thiazides, but plasma-aldosterone-concentration to plasma-renin-activity [PAC/PRA] was pending. The ratio returned equivocal for primary hyperaldosteronism at 23.

A year later, he was hospitalized for methicillin-sensitive *Staphylococcus aureus* pneumonia, extended spectrum beta-lactamase (ESBL) *E. coli* pneumonia, and ESBL

E. coli bacteremia. The same left adrenal mass now measured 7.1cm x 5.8cm with extensive mediastinal and hilar lymphadenopathy. Pathology of four CT-guided adrenal biopsies was consistent with benign adrenocortical neoplasm, favor adenoma, with a caveat that mass resection would give a definitive diagnosis. Fine-needle aspiration of a sub-carinal lymph node revealed malignant cells consistent with metastatic adrenocortical carcinoma. Immunohistochemical evaluation stained positive for Synaptophysin, Melan-A, and GATA 3.

Urine cortisol levels returned at 2259.6 mcg/g, consistent with glucocorticoid hypersecretion. AM cortisol was 147mcg/dL and ACTH was 10pg/mL; dexamethasone suppression test was not performed as the patient transitioned to hospice.

Discussion:

Approximately 80% of adrenal incidentalomas will be non-functional; adrenocortical carcinoma accounts for less than 4% of functional tumors. When the mass measured 2.1cm, AM cortisol measured 19.9 mcg/dL. In the following five years, multiple CT scans denoted the incidentaloma as “an adenoma” or “myelolipoma”, despite its growth. Aldosterone was not elevated (<15ng/dL) during evaluation for primary hyperaldosteronism and aldosterone-renin ratio was equivocal. Spironolactone use may have masked the symptoms elevated mineralocorticoids, resulting from excessive cortisol.

At the time of diagnosis, his endocrine work up was likely clouded by septic shock necessitating stress-dose steroids. This patient had an elusive diagnosis, with a poor chance of detection, despite longitudinal findings.

Name: Rachel Kubowicz, MD

Presentation Type: Poster Presentation

Residency Program: Saint Joseph Hospital

Additional Authors: Brian Daily, MD

Abstract Title: A Difficult Diagnosis to Swallow

Abstract Information:

Introduction:

Dysphagia is usually caused by narrowing of the esophageal lumen due to strictures, webs, neoplasm, and infiltration. Another broad category is disorders of motility such as achalasia and oropharyngeal dysphagia after a stroke. Less common etiologies include infections, medications, autoimmune diseases, and neurologic disorders. We present a unique case of dysphagia.

Case Description:

- 52 year old woman presented with dysphagia.
- HPI: 3 month history of dysphagia to solids, 85 lb weight loss, fatigue, night sweats, mood disturbance.
- No significant PMH. Surgical history of cesarean section.
- Exam: tachycardia, conjunctival and general pallor, otherwise unremarkable.
- Labs: Hgb 4.5, Hematocrit 13.4, MCV 125, Ferritin 200, Total Iron 28, TIBC 238, 12% sat, Reticulocyte count 1.50 with reticulocyte index 0.19. TSH normal, LDH 1,825. Peripheral smear showed “anemia and marked anisocytosis consistent with acute and or chronic blood loss. Significant polychromasia noted. No significant schistocytes.”
- Imaging: Barium esophagram with Abnormal mucosa level of vallecula and possible base of tongue as well as significant aspiration. CT neck and direct pharyngoscopy with no correlating abnormality. CT chest abdomen pelvis with gallbladder wall thickening, diverticulosis, uterine fibroids. EGD with patchy atrophic gastritis in body and fundus (see path below). Colonoscopy with diverticulosis, no obvious mass or source of bleeding.
- Workup: Initial presentation of dysphagia, weight loss, and anemia was concerning for GI malignancy, however extensive workup for this was negative. On re-examination of her labs, particularly the MCV of 125 led us to focus on the etiology of her anemia. B12 level ordered at the time of admission came back several days into workup at <60 pg/ml (normal 193-986). Intrinsic Factor antibody was positive. On EGD gastric biopsy showed chronic atrophic gastritis (see path image below). Neuro exam repeated with intact cranial nerves, normal strength, sensation, proprioception, and gait.
- Treated with B12 injections, and high dose oral B12. On hospital follow up anemia had improved from discharge Hgb of 7.8 to 9.4. Patient reported more energy and slight improvement of dysphagia.

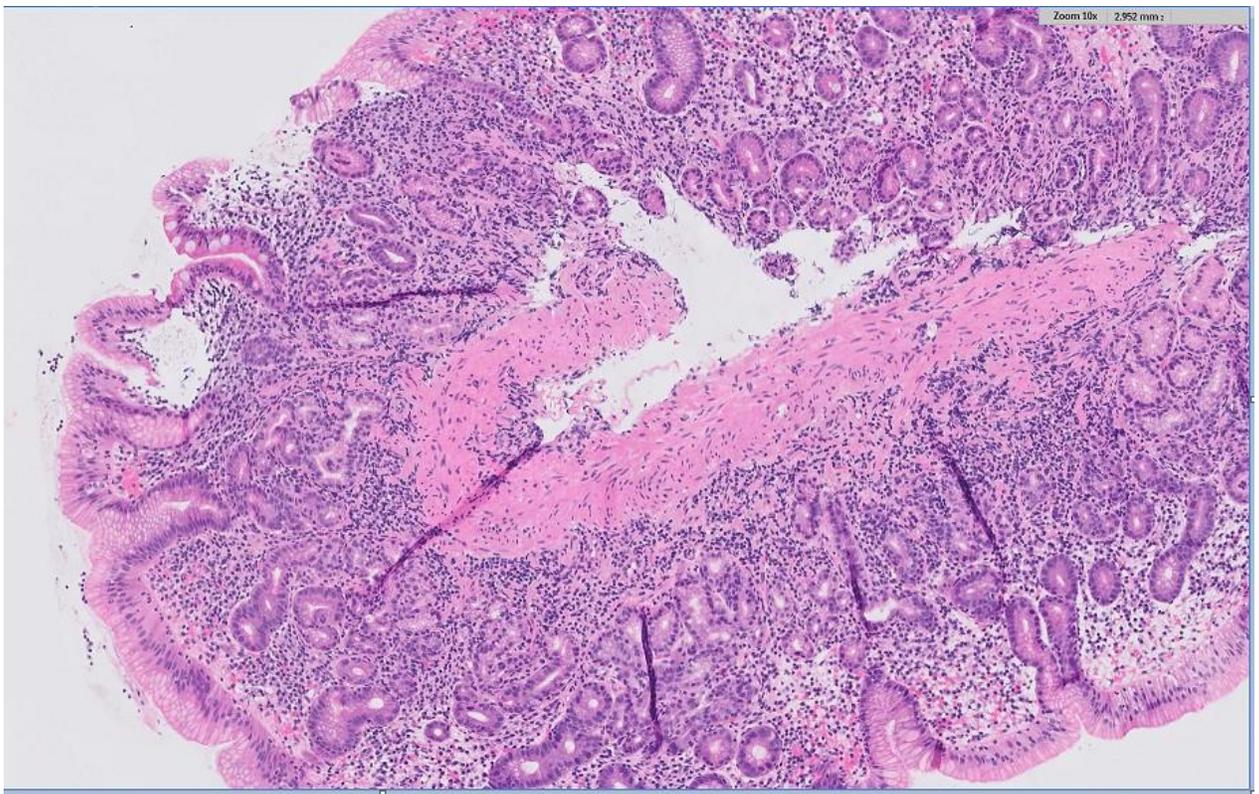
Discussion:

Pernicious anemia typically presents with symptomatic anemia. Well known neurologic effects of B12 deficiency include paresthesias, numbness, impaired position and vibration sense, ataxia, and weakness. Rarely, pernicious anemia can cause dysphagia which may be the chief complaint, as was the case with our patient. Pernicious anemia is diagnosed with serum antibodies to gastric parietal cells or intrinsic factor and elevated MMA, and if EGD is performed gastric biopsy will show chronic atrophic gastritis. Treatment is with oral or parenteral B12 supplementation. Patients respond very well to treatment with hematologic improvement within 2 months and neurologic improvement or resolution within 6 months. As such, vitamin B12 deficiency is an important cause of reversible neurologic symptoms. In patients with

megaloblastic anemia, it is important not to erroneously treat for B9 deficiency since the anemia would improve but the neurologic symptoms would continue to worsen.

References:

1. Reversible neurogenic dysphagia: A rare presentation of vitamin b₁₂ deficiency.
Edward JA, Bowman A, Heppe DB.
eNeurologicalSci. 2018;13:31–32. Published 2018 Nov 19. doi:10.1016/j.ensci.2018.11.005
2. Vitamin B12 Deficiency.
Stabler SP.
N Engl J Med. 2013 Jan 10;368(2):149-60. doi: 10.1056/NEJMcp1113996. Review.
No abstract available.
3. Dysphagia: Thinking outside the box.
Philpott H, Garg M, Tomic D, Balasubramanian S, Sweis R.
World J Gastroenterol. 2017;23(38):6942–6951. doi:10.3748/wjg.v23.i38.6942
4. Pernicious anemia.
Toh BH, van Driel IR, Gleeson PA.
N Engl J Med. 1997 Nov 13;337(20):1441-8. Review. No abstract available.



“Chronic inactive gastritis with focal intestinal metaplasia. Negative for H.pylori on immunostain.”

Name: Samara Levine, MD

Presentation Type: Poster Presentation

Residency Program: University of Colorado Medical Center

Additional Authors:

Abstract Title: Anal Mass and Diffuse Lymphadenopathy

Abstract Information:

Introduction: There is a broad differential for anal lymphadenopathy and anal masses, including lymphoma, squamous cell cancer of the anus, lymphogranuloma venereum, IBD and HPV infections. Primary and secondary anorectal syphilis also presents this way and recognition of more unusual manifestations of syphilis is important to avoid unnecessary diagnostic procedures and treatments.

Case Description: 58 year old male living with HIV, anal dysplasia, and hemorrhoids, presented to the ED with 2 week history of constipation, worsening lower abdominal cramping pain, and intermittent bright red blood per rectum. He had been off antiretroviral therapy and out of care for 1 year when he re-engaged in care 3 months prior. CT demonstrated anorectal wall thickening with prominent lymph nodes as well as perirectal and perianal anal fat stranding. Anal cancer was suspected, and patient was referred to IR for biopsy of inguinal lymph node as well as to anal dysplasia clinic for high resolution anoscopy after hospital discharge. Physical exam prior to anoscopy was notable for significant inguinal lymphadenopathy bilaterally, with left inguinal lymph node induration to 3cm, non-tender to palpation. Perianal exam with rubbery 1cm, smooth mass and digital anorectal exam with indurated lesion posteriorly. Diffuse macular erythematous rash was noted over torso and extremities, including palms and soles. Hgb 11.9, HIV viral load 66, and CD4 count was 529. Treponema antibody was reactive, RPR titer 1:128, previously negative in 11/2016. Anal biopsies, including that of the perianal mass, revealed low grade squamous intraepithelial lesions as well as areas of extensive inflammation and reactive changes. Follow up immunohistochemical staining for spirochetes was positive. This confirmed the diagnosis of anorectal syphilis. He received a intramuscular injection, with penicillin 2.4 million units per week, for 3 weeks. Follow-up exam 3 weeks later demonstrated decreased size of perianal lesion. Additionally, anal canal ulcerations had improved, demonstrated healing. The anal canal indurated area had resolved.

Discussion: There is an increasing incidence of syphilis in the United States and recognizing its diverse clinical manifestations is crucial for prompt treatment, especially given that primary and secondary syphilis are the most infectious stages. Primary anorectal syphilis presents as chancres, ulcerations, and diffuse pelvic lymphadenopathy. Presentations of secondary anorectal syphilis include condyloma lata, broad, smooth, gray to erythematous wart-like lesions, and mucous patches, which are shallow ulcers with overlying gray exudate. While syphilitic chancres are thought to be painless, anorectal ulcers can be painful. Anorectal syphilis often presents with diffuse lymphadenopathy, which can be mistaken for malignancy. Symptoms of anorectal syphilis

include variations in bowel habits, mucorrhea, hematochezia, tenesmus, itching and anal discharge. This case highlights the importance of investigating patient's risks and routinely performing STD screening

Name: Shannon Mazer, DO

Presentation Type: Poster Presentation

Residency Program: Sky Ridge Medical Center

Additional Authors: Oliver Garbo, DO, Paula Watts, DO, David Mohlman, DO

Abstract Title: Heat Exhaustion Causing Conduction System Abnormality; Phenocopy or Brugada Syndrome?

Abstract Information:

Introduction: Brugada syndrome (BrS) is an inherited channelopathy with risk for sudden cardiac death. Its diagnosis includes spontaneous or induced ST-segment elevation in the right precordial leads. In the absence of BrS, an acute condition associated with a Brugada electrocardiogram (ECG) pattern that resolves with resolution of the transient condition, has been termed Brugada ECG phenocopy (BrP). This is a case of a 61-year-old female presenting with fever and Type 1 Brugada pattern on ECG.

Case Description: A 61-year-old healthy female presented with fever, rigors, and diarrhea. Her symptoms began upon return from vacation, after spending time in 110°F heat. Upon presentation, her abnormal vitals included a temperature of 107.4°F and heart rate of 145. She was alert, with normal mental status. She had a papular erythematous rash to the trunk and extremities. Her exam was, otherwise, unrevealing. Her work-up included a lactate of 5.22 mmol/L, c-reactive protein 32.6 mg/dL, total creatine kinase 470 U/L, magnesium 1.5 mg/dL, white blood cell count $12.1 \times 10^9/L$, platelet count $130 \times 10^9/L$, and a troponin level $<0.015 \text{ ng/mL}$. The 12-lead ECG revealed a Brugada pattern. The patient denied a personal or family history of syncope, arrhythmia, or sudden cardiac death. No prior ECG's were available. With antipyretics, she became normothermic and by the following day, the ST-segment normalized.

Discussion: BrS is an inherited sodium channelopathy with decreased sodium channel density and conductance, as well as risk for ventricular arrhythmia. SCN5A mutation, most frequently implicated in BrS, causes diseased sodium channels that result in quicker entry and recovery from the fast inactivation state. Higher temperatures also cause a slowing of the sodium current by inducing sodium channels to enter the slow inactivation state. These changes produce ECG patterns representative of BrS. There have been reports of transient disease states that induce a Brugada pattern, without the diagnosis of BrS. The ECG normalizes upon resolution of the acute condition. Reported etiologies include metabolic conditions, mechanical compression, ischemia and others. This phenomenon is called BrP; a poorly understood mechanism of dysfunction distinct from BrS. It is not due to an inherited sodium channelopathy, therefore sodium channel blockade has no impact on the resting ECG. Several

criteria should be met to diagnose BrP. These include no personal history of syncope, palpitations, arrhythmia, agonal respirations, and no family history of unexplained syncope or sudden death. A provocative test should be attempted and if negative, a diagnosis of BrP could be made. This case remains unanswered. Is this BrS or BrP in this asymptomatic patient who declined further work-up by provocative testing, genetic analysis, or otherwise?

Name: Amanda Mika, DO **Presentation Type:** Poster Presentation

Residency Program: Saint Joseph Hospital

Additional Authors: Ayal Levi, MD

Abstract Title: Breast-Feeding the Baby to Barely Breathing: Toxic Shock secondary to Mastitis

Abstract Information:

Introduction:

In the first reported cases of toxic shock syndrome (TSS) in the 1970s, menstrual causes were the primary source. With the removal of specific hyper-absorbent tampons and subsequent public education and regulation, the ratio of menstrual cases to non-menstrual cases has declined, with surgical and post-partum wound infections now making up roughly 50% of all reported toxic shock cases^{1,2}. In this case, we describe a rare incidence of TSS secondary to mastitis.

Case:

A 29-year-old female three weeks post-partum after a spontaneous vaginal delivery presented with confusion and right breast pain. 48 hours prior to admission she experienced breast tenderness that was treated conservatively. The morning of admission she was disoriented and weak. She presented with a temperature of 104°F, pulse of 92, blood pressure of 103/77, and respiratory rate of 27. She was somnolent but arousable with an area of induration on the right breast that was tender on palpation and palmar erythema with complaints of palmar pruritus. Her labs showed a WBC of 14.22, and a lactate of 4.5. A head CT demonstrated no acute process. She received fluids and broad-spectrum antibiotics and transferred to the ICU. Approximately four hours after admission she developed a diffuse, blanching macular rash with petechiae across the upper extremities. Clindamycin was added to her antibiotic regimen. Repeat labs were consistent with mild DIC and revealed a troponin of 8. An echocardiogram revealed an ejection fraction of 35%. Two abscesses were aspirated in the right breast, and cultures grew methicillin sensitive *Staph aureus*. For 48 hours the patient remained on multiple vasopressors with a waxing and waning mental status. Her rash disappeared, she improved clinically and was discharged home. Within a week after discharge she experienced diffuse desquamation of her skin consistent with TSS.

Discussion:

The rate of streptococcal TSS occurs at about 3/100,000 per year, and staphylococcal TSS at 0.5/100,000 per year in the USA^{3,4}. The mortality rate of streptococcal TSS is roughly 50%, while staphylococcal TSS is roughly 5%^{2,3,5}. One review of 51 cases of non-menstrual TSS demonstrated mortality from non-menstrual TSS being significantly higher than menstrual TSS⁶. There have been a few case reports of TSS secondary to mastitis⁷⁻⁹. In our patient her initial symptoms to severe shock exacerbated by cardiomyopathy, a documented complication of TSS^{2,10}, occurred within 48 hours. Considering that non-menstrual TSS has overtaken menstrual TSS in the developed world^{1,2,11} and that incidences are likely under-reported^{2,3,4}, awareness of the different non-menstrual etiologies is important for the clinician. Given the danger posed and the rapid acuity of the illness, simply ruling out the presence of a longstanding tampon is not enough to ignore the threat of TSS.

References

1. (CDC) C. Reduced incidence of menstrual toxic-shock syndrome--United States, 1980-1990. - PubMed - NCBI. [Ncbi.nlm.nih.gov. https://www.ncbi.nlm.nih.gov/pubmed/2113167](https://www.ncbi.nlm.nih.gov/pubmed/2113167). Published 2019. Accessed September 14, 2019.
2. DeVries A, Leshner L, Schlievert P et al. Staphylococcal Toxic Shock Syndrome 2000–2006: Epidemiology, Clinical Features, and Molecular Characteristics. *PLoS ONE*. 2011;6(8):e22997. doi:10.1371/journal.pone.0022997
3. Smit M, Nyquist A, Todd J. Infectious Shock and Toxic Shock Syndrome Diagnoses in Hospitals, Colorado, USA. *Emerging Infect Dis*. 2013;19(11):1855-1858. doi:10.3201/eid1911.121547
4. Low D. Toxic Shock Syndrome. *Crit Care Clin*. 2013;29(3):651-675. doi:10.1016/j.ccc.2013.03.012
5. Schmitz M, Roux X, Huttner B, Pugin J. Streptococcal toxic shock syndrome in the intensive care unit. *Ann Intensive Care*. 2018;8(1). doi:10.1186/s13613-018-0438-y
6. Descloux E, Perpoint T, Ferry T et al. One in five mortality in non-menstrual toxic shock syndrome versus no mortality in menstrual cases in a balanced French series of 55 cases. *European Journal of Clinical Microbiology & Infectious Diseases*. 2007;27(1):37-43. doi:10.1007/s10096-007-0405-2
7. Demey H, Hautekeete M, Buytaert P, Bossaert L, Demey H. Mastitis and Toxic Shock Syndrome. *Acta Obstet Gynecol Scand*. 1989;68(1):87-88. doi:10.3109/00016348909087698
8. Fujiwara Y, Endo S. A Case of Toxic Shock Syndrome Secondary to Mastitis Caused by Methicillin-resistant *Staphylococcus aureus*. *Journal of the Japanese Association for Infectious Diseases*. 2001;75(10):898-903. doi:10.11150/kansenshogakuzasshi1970.75.898.

9. Moseson E, Win H, Green A. Baby Takes a Bite: Toxic Shock Syndrome from Mastitis. *Journal of Hospital Medicine*. 2010; 5 (suppl 1).

<https://www.shmabstracts.com/abstract/baby-takes-a-bite-toxic-shock-syndrome-from-mastitis/>.

10. Cian McDermott and Michael Sheridan, Staphylococcal Toxic Shock Syndrome Caused by Tampon Use. *Case Reports in Critical Care*. 2015:2015(640373). doi.org/10.1155/2015/640373.

11. Sharma H, Smith D, Turner C et al. Clinical and Molecular Epidemiology of Staphylococcal Toxic Shock Syndrome in the United Kingdom. *Emerging Infect Dis*. 2018;24(2).
doi:10.3201/eid2402.170606

Name: Patryk Purta, DO

Presentation Type: Poster Presentation

Residency Program: Sky Ridge Medical Center

Additional Authors: Dmitriy Scherbak, D.O.; Michael Gruber, M.D., Oliwer Dziadkowiec, Ph.D.

Abstract Title: Reverse Takotsubo- Like Cardiomyopathy and Pheochromocytoma

Abstract Information:

Introduction:

Pheochromocytoma is a rare catecholamine-producing tumor of chromaffin cells in the adrenal gland. Takotsubo Cardiomyopathy is stress induced cardiomyopathy typically with apical ballooning shape of the left ventricle on echocardiography due to apical akinesis. Rarely a reversed variant of Takotsubo cardiomyopathy in which the apex is spared while the base of the heart is akinetic. We present a case of a pheochromocytoma with reverse Takotsubo cardiomyopathy.

Case Description:

34-year-old female presented with lightheadedness, dizziness, flushing, near-syncope, dyspnea on exertion for 1 week. She also reported increasing panic attacks not relieved with home clonazepam. She had a heart rate of 166 and blood pressure of 58/34 that was fluid responsive. Her point of care troponin I was elevated to 1.96 (units), pro-B-type natriuretic peptide (BNP) of 10,773 (pg/mL), an electrocardiogram (ECG) showed no ischemic changes. A Computed Tomography Angiography (CTA) revealed a right adrenal mass measuring 3.9x2.7cm.

The patient was admitted to the intensive care unit for further management. She was found to have severe orthostatic hypotension. Transthoracic echocardiography showed an ejection fraction of 25-30% with severe global hypokinesis. Wall motion abnormalities included the left ventricular base and midportion, sparing the apex - consistent with reverse Takotsubo cardiomyopathy.

Systolic blood pressures remained consistently above 210 mmHg until the beta blocker was replaced with phenoxybenzamine. Pheochromocytoma was strongly suspected and soon confirmed. Plasma fractionated metanephrines and 24-hour urinary catecholamines and metanephrines were observed at greater than ten-fold the upper limits of normal.

She was eventually transferred to the University of Colorado Hospital for emergent resection of the pheochromocytoma. She underwent laparoscopic right adrenalectomy. Plasma metanephrines and catecholamines normalized post-surgery.

Discussion

Pheochromocytomas are catecholamine producing neuroendocrine tumors with an annual incidence approximately 0.8 per 100,000 person-years. The signs and symptoms include elevated heart rate, hypertension, orthostatic hypotension, palpitations, anxiety, headaches, and hyperglycemia. Pheochromocytoma is rarely associated with cardiomyopathy attributed to catecholamine excess that is similar to stress-induced (Takotsubo) cardiomyopathy. The true incidence of the two together is unknown due to the extreme rarity of such cases. The presence of an adrenal mass along with worsening hypertension secondary to unopposed alpha adrenergic vasoconstriction with beta blockade resulted in high suspicion for pheochromocytoma in this case.

The reverse Takotsubo-like cardiomyopathy observed on echocardiogram was likely a result of catecholamine-induced cardiotoxicity triggering apoptosis. Only 2.2% of patients with Takotsubo had the reverse variant which shows basal akinesis/hypokinesis with apical sparing with a female to male preference. Coronary artery disease is an important consideration in any variant of Takotsubo cardiomyopathy. In our case, a left heart catheterization was not conducted due to the patient's young age, echocardiogram findings and elevated catecholamine levels which made the case for reverse Takotsubos cardiomyopathy far more likely.

Name: Logan Reimer, DO

Presentation Type: Poster Presentation

Residency Program: Parkview Medical Center

Additional Authors: Ricki Kumar, DO; Meghan Timmerman, DO

Abstract Title: Appendicitis: Uncooked

Abstract Information:

Introduction:

Appendicitis is one of the most common causes of an acute abdomen, with an incidence of 233/100,00 with the most prevalent age group being in the 2nd decade of life and having a predisposition towards males¹. We present a case of a common disease process with an uncommon etiology.

Case Description:

An 18 year old male presented to the ER after evidence of colitis on CT scan at an outlying facility. His initial presentation was for vomiting, abdominal pain, diarrhea, and fatigue that started the night before coming to the hospital. He was admitted to the general surgery team where he was started on Piperacillin-Tazobactam and bowel rest for suspected infectious colitis. Gastroenterology was consulted for the colitis and evaluated the patient with stool studies. In the interim, a sepsis alert was called due to a decompensation in vital signs with T104.6, HR 122, and chills. The patient's stool studies had returned at this point and the patient was found to have Salmonella by PCR. The patient was transitioned to Ceftriaxone and oral Metronidazole. Surgery recommended continued non-operative treatment as difficult to distinguish right-sided colitis and appendicitis on repeat imaging while awaiting culture results. Culture results at 62 hours of the stool confirmed Salmonella. The patient continued to have mild pain and a third CT scan was obtained which showed an increasing in size fluid collection consistent with appendiceal abscess. CT-guided drain placement was performed with Salmonella growing on fluid culture. Patient was discharged on antibiotics with follow up with surgery for elective appendectomy and drain removal.

Discussion:

Non-typhoid Salmonella is a foodborne illness which usually manifests as a self-limiting gastroenteritis which presents as nausea, vomiting, diarrhea, and abdominal pain. In the case above, the patient initially presented with right sided infectious colitis secondary to Salmonella which directly invaded the appendix causing a purulent appendiceal abscess. The worsening abdominal rigidity and right lower quadrant abdominal pain in addition to no significant improvement with antibiotics was a key trigger in repeating a CT scan revealing appendicitis. Salmonella appendicitis is seen in only ~8% of Salmonella bacterial enteritis³. The proposed mechanisms of spread to the appendix are by direct invasion or via bloodstream as in cases with Salmonella bacteremia². In this case, the Salmonella infection developed an abscess within the appendix requiring drainage and ultimately appendectomy. Additionally, the month prior to admission the patient received a dose of Dupilumab which is a monoclonal antibody used for allergies such as eczema and nasal polyps. This case shows that with Salmonella infections

there should be a low threshold for repeat imaging if abdominal pain persists or worsens, as untreated appendicitis risks appendiceal rupture and severe complications.

Resources

1. Addiss DG, Shaffer N, Fowler BS, Tauxe RV. The epidemiology of appendicitis and appendectomy in the United States. *Am J Epidemiol.* 1990;132(5):910.
2. Siow Yun Wong, Samuel Kang Lian Lee, Chaozer Er, Navin Kuthiah, Appendicitis in nontyphoidal salmonella bacteraemia, *Oxford Medical Case Reports*, Volume 2018, Issue 11, November 2018
3. Stewart-Parker EP, Atta M, Doddi S. *BMJ Case Rep* Published online: October 04, 2019; doi:10.1136/bcr-2016- 216150

Name: Maeveen Riordan, MD

Presentation Type: Poster Presentation

Residency Program: University of Colorado Medical School

Additional Authors: Tyler Kaufman, MD

Abstract Title: This one kiss will break your heart

Abstract Information:

Case: A 71-year-old male presented with six months of progressive dyspnea and orthopnea. The patient recently emigrated from Bolivia and reported multiple episodes of heart palpitations, lightheadedness, and fatigue. He was diagnosed with atrial fibrillation six months prior to presentation and medications included Metoprolol and Aspirin. Vital signs were BP 136/88, HR 124, RR 18, and 97% on room air. He was warm and well perfused with elevated JVD, bibasilar crackles, and irregularly irregular rhythm without murmurs. Labs significant for troponin of 0.03, BNP 1580, and TSH 1.01. His ECG showed atrial fibrillation with a rapid ventricular rate. An echocardiogram showed an ejection fraction of 35% and global hypokinesis of the left ventricle with a dyskinetic apex. A cardiac CT showed a crescent shaped defect on the apical inferoseptal wall and minimal coronary artery disease. A Trypanosoma Cruzi IgG was positive, making the diagnosis of Chagas cardiomyopathy.

Discussion: Once considered a disease of rural Central and South America, Chagas disease is growing in prevalence in traditionally nonendemic areas due to changing global migration patterns. There are approximately 300,000 residents in the U.S. affected by Chagas disease. Given the extent of disease burden, the severity of illness, and the availability of treatment, the CDC has listed Chagas disease as one of five neglected parasitic infections to target for public health action.

Chagas disease is caused by infection with the protozoan Trypanosoma cruzi, and is transmitted through insect vectors ("kissing bugs"), blood transfusions, organ transplantation, or in-utero. The disease can present with varying degrees of heart failure, cardiac arrhythmias, atypical angina, or thromboembolism. Chagas cardiomyopathy is the most common complication of chronic infection and portends higher morbidity and mortality. It involves damage to the cardiac myocytes, endothelial cells, and conduction system by activation of the host immune response and direct by parasite burden. Chagas cardiomyopathy can be distinguished from other dilated cardiomyopathies by prominent fibrosis in the posterior and apical regions of the left ventricle and extensive damage to the electrical conduction system. Apical aneurysms are seen on echo in up to 60% of patients with Chagas cardiomyopathy. Cardiac MRI is the preferred imaging modality in characterizing the degree and distribution of myocardial fibrosis. Definitive diagnosis of chronic Chagas infection requires two serological tests aimed at T.Cruzi. Treatment is aimed at initiating patients on guideline therapy for heart failure and antitrypanosomal medications such as Benznidazole or Nifurtimox.

Conclusion: This case aims to increase physician knowledge over an increasingly prevalent infection and to highlight the cardiac complications of chronic Chagas infection.

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Presentation Type: Poster Presentation

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Abstract Title: Euglycemic Diabetic Ketoacidosis in a Patient Recently Initiated on a SGLT2-inhibitor

Abstract Information:

Case Presentation:

A 73 year-old man with history of type II diabetes and alcohol use disorder presented to emergency department with nausea, vomiting, and malaise since the morning of admission. He was started on canagliflozin two weeks prior to admission. His last alcohol-containing drink was one day prior to admission. Vitals were notable for tachycardia and physical exam was notable for dry mucous membranes and diffuse abdominal tenderness. Initial labs showed a mildly elevated glucose, severe hyponatremia and an anion gap metabolic acidosis (AGMA). He was triaged to the medicine floor. After gentle intravenous fluids, repeat labs showed worsening AGMA. Venous blood gas was consistent with a compensated metabolic acidosis and beta-hydroxybutyrate was elevated. The patient was diagnosed with euglycemic diabetic ketoacidosis (EuDKA) and was transferred to the medical intensive care unit for an insulin and dextrose drip. His AGMA resolved within 24 hours and the patient was transitioned to subcutaneous insulin.

Discussion:

EuDKA is a rare, but potential complication of SGLT2-inhibitors. The mechanism is thought to be due to inhibition of the SGLT2 transporter on the apical surface of the proximal tubule nephron, which reduces sodium reuptake and consequently depletes the substrate for the NA-K ATPase pump, leading to ketoacid metabolism. SGLT2-inhibitor use alone is thought to be insufficient to cause DKA; however, the risk may be increased when there is a “second hit” that increases fatty acid metabolism. In this case, this patient’s alcohol use likely increased his risk of EuDKA due to SGLT2-inhibitors. Euglycemia may lead to a delay diagnosis and treatment in EuDKA. Management of EuDKA is similar to hyperglycemic DKA in that both require admission to ICU for insulin drip, however, in EuDKA, dextrose is added at the initiation of insulin. SGLT2-inhibitors should be avoided in the future for this patient given his chronic alcohol use.