Persistent Symptomatology: A Case of Celiac Disease with underlying Crohn’s Disease

Introduction
Gastrointestinal tract diseases can vary among symptomatology therefore necessitating investigation with serology, imaging, and sometimes endoscopy. Celiac disease (CeD) is a condition characterized by hypersensitivity to gluten with patients presenting with steatorrhea, abdominal pain and flatulence. While in inflammatory bowel disease (IBD), there is immune activation with recurrent inflammation to the intestines. Similar to CeD, IBD retains some clinical manifestations notably digestive symptoms, abdominal pain, diarrhea and nutrient malabsorption. Due to alignment of their presentations, it’s noteworthy to consider both conditions when attributing a diagnosis. We present a case of a 30-year-old male with celiac disease whom remained symptomatic with dietary compliance; but was later on found to have underlying Crohn’s disease.

Case Report
A 30-year-old male with history of rectal fistula, anxiety, and CeD on gluten-free diet presented to the ER with 1 year duration of diffuse abdominal pain, nausea, and vomiting. Patient endorses daily symptoms despite his compliance with his gluten-free diet. He denies any diarrhea or loose bowel movement but has experienced unintentional weight loss of 50kg /1 year. CT abdomen and pelvis obtained on presentation showed small bowel obstruction (SBO) with normal inflammatory markers. Previous colonoscopy with biopsies revealed nonspecific chronic inflammation in the terminal ileum. Patient was made n.p.o. with NG to LIS and started on Solu-Medrol for possible Crohn’s disease flare. SBO failed to improve with conservative measures, prompting exploratory laparotomy with ileocecectomy and anastomosis on Day 12 of hospitalization. Pathology report from the resected segment displayed chronic active ileitis with deep fissures, constriction, and serosal adhesion consistent with Crohn’s disease. Postoperatively the patient made a full recovery and TPN was discontinued.

Discussion
Both celiac disease and IBD have varying pathophysiological mechanisms with genetic and environmental predispositions prompting research advancements to determine amongst these conditions. Studies have found a bidirectional causal relationship between the CeD and IBD alluding to the notion that celiac disease is a risk factor for IBD and vice versa. The prevalence of IBD was found to be nine times higher in CeD than the general population while patients with IBD experienced only moderate increased risk for CeD. Given the overlap of symptomatology between the two aforementioned conditions (CeD and IBD) along with shared genetic patterns; a detailed history, physical exam and workup are paramount in accurately diagnosing and promptly treating the underlying disease. It is crucial to educate the patient regarding their symptoms and encourage them to seek professional advice, especially in the case like this one where the patient continues to have celiac-like symptoms despite a strict gluten-free diet. This should prompt further investigation especially in the light of abnormal terminal ileum biopsy in the past.

Conclusion
This case underscores the challenge of diagnosing gastrointestinal conditions due to symptoms overlap and the necessity of a comprehensive approach, including endoscopy. Notably, not all patients show elevated serology and/or inflammatory markers. The lack of adequate response to standard treatments prompts reevaluation of the diagnosis or exploration of additional causes to prevent irreversible and maybe fatal complications.
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A rare case of gastroenteritis: Aeromonas Hydrophila

Introduction:
While watery diarrhea can be caused by a wide range of etiologies, gastroenteritis is on the top of the differential diagnosis. We discuss a rare case of Aeromonas-associated gastroenteritis.

Case presentation: An 88-year-old woman with history of stage II colon cancer s/p right hemicolectomy and CLL, presented with diarrhea and nausea for four days. The patient recalled eating a "Beef sandwich" one week prior symptom onset. Labs revealed WBC of 47,000 with >70% lymphocytes. CT abdomen/pelvis showed newly enlarged axillary mesenteric and pelvic lymph nodes but no evidence of colitis. Differential diagnosis included infectious etiologies vs CLL, less likely microscopic colitis, drug-induced or inflammatory etiologies. Patient had positive lactoferrin, negative Clostridium difficile and positive stool culture for Aeromonas hydrophila. Patient was started on ceftriaxone with improvement of her symptoms. The patient was discharged after 1 week of hospitalization.

Discussion:
The differential diagnosis of diarrhea is vast. A comprehensive history, physical exam and work up is crucial to identify the underlying cause. Gastroenteritis is caused by inflammation of the stomach and intestines. The typical manifestations include nausea, vomiting, diarrhea and abdominal pain. Many pathogens have been implicated. While some organisms are more prevalent, the clinician must have a high level of suspicion and investigate less common causes to correctly identify the culprit pathogen and treat accordingly. Aeromonas hydrophila is a gram negative facultative anaerobic coccobacillus, which has been isolated from various sources, such as produce, aquatic environments, domesticated pets, and fishes. It can cause gastroenteritis, septicemia and cellulitis. Reported cases showed that Aeromonas primarily affects immunocompromised patients and those with inflammatory bowel disease. Stool culture is necessary in the work up of diarrhea as not all organisms are detected by PCR and some cases of gastroenteritis may not reveal signs of inflammation on CT as observed in the presented case.

Treatment includes a third-generation cephalosporin or a fluoroquinolone, such as Ciprofloxacin.

Conclusion: This case draws attention to less common pathogens. It underscores the need for a thorough evaluation and diagnostic approach to avoid undertreatment and ultimately improve the quality of patient care.
Atrial septal aneurysm leading to Ischemic stroke: A case report and literature review

Background:

Stroke is the second most common cause of death and disability in the world. 13.7 million new cases of stroke were noted in 2016, out of which around 87% were secondary to ischemic strokes. Atrial septal aneurysm (ASA) is a localized saccular deformity of the atrial septum, associated with ischemic stroke independently or in association with other atrial septal defects (ASD). The incidence of stroke in the population with ASD is around 10%. In these patients, the presence of ASA is an important predictor of recurrent stroke.

Case Report:

We present a case of ischemic stroke in a 44 years old lady, presented with sudden onset right-sided body weakness, expressive aphasia and non-specific confusion for one hour, with an initial NIH score of 7. CT Angiogram revealed occlusion of the M3 branch of the left middle cerebral artery in the left lateral frontal lobe. Code stroke was called and the patient was given TNK after which her right side weakness and aphasia resolved. Transthoracic Echo with bubble study showed ASA with positive bubble study.

Conclusion:

Lone ASA or ASA with concomitant ASD is associated with increased risk of recurrent stroke in younger patients especially those without significant risk factor for strokes. Patients with ASA and concomitant ASD are at high risk for recurrent ischemic stroke and should be kept under surveillance with continued medical therapy. We present a case of ischemic stroke caused by ASA and review of current literature and case reports documenting cases with similar presentations.
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**Navigating Colorectal Cancer Screening: Insights from a Quality Improvement Study**

Background: Colorectal cancer ranks as the second leading cause of cancer-related deaths in the United States, disproportionately affecting individuals aged 65-74. While screening recommendations have shown efficacy in reducing mortality, there exists a concerning rise in incidence among adults aged 45-49. This quality improvement study addresses the need for improved screening compliance, particularly among the age group of 50-75, to mitigate the impact of colorectal cancer.

Methods: A quality improvement study was conducted targeting average-risk individuals aged 50-75 in an academic internal medicine clinic. Patient outreach and education, facilitated through virtual visits, aimed to improve colorectal cancer screening compliance. Secondary goals included assessing the performance of academic internal medicine practitioners in colon cancer screening, enhancing patient education on screening modalities, and promoting awareness of the benefits of age-appropriate screening.

Results: Of the 1258 identified patients deficient in colorectal cancer screening, 215 were chosen as the initial study population. A total of 36 orders were placed for patients. There was a slight preference for non-invasive screening during the initial outreach phase of the study. Despite challenges in patient outreach, 30.5% of orders were completed, with a 31.3% completion rate for colonoscopy and a 30% completion rate for non-invasive methods like FIT and Cologuard.

Conclusion: The study reveals similar patient preferences for invasive and non-invasive screening, as well as similar completion rates for both methods. Factors influencing completion include cost, privacy, time commitment, and perceived risks. Future studies could expand the age range and explore diverse outreach methods, incorporating patient portals or dedicated websites. Despite limitations, our study highlights the challenges and opportunities in enhancing colorectal cancer screening rates. The need for diverse outreach strategies is emphasized, with virtual visits/phone calls emerging as potentially effective tools. Patient education, personalized outreach, and consideration of patient preferences play pivotal roles in improving screening rates. The study’s insights can potentially inform strategies not only for colorectal cancer but also for other medical illnesses, contributing to reduced mortality and improved overall health.
Delayed Brain Abscess Formation Due to Streptococcus Pneumoniae Following Craniotomy in an Immunocompetent Young Adult: A Case Report

Bacterial meningitis presents a life-threatening scenario requiring immediate and comprehensive management, as highlighted in the challenging case of a 23-year-old male who arrived at the Emergency Department (ED) unresponsive, exhibiting fever, headache, and seizures. His medical history included a traumatic brain injury from a previous motor vehicle accident, with subsequent frontal craniotomy in Colombia. The patient's symptoms, including a recent tonic-clonic seizure, raised concerns for acute bacterial meningitis and prompted diagnostic and therapeutic interventions to address the intricacies of this condition and its potential intracranial complications.

He was unresponsive upon admission, subsequently intubated and sedated, and transferred to ICU, after a lumbar puncture confirmed bacterial meningitis. Treatment involved Decadron, Ceftriaxone, cefepime, and vancomycin. Preceding the lumbar puncture, a CT head revealed post-op changes from a frontal craniotomy with bilateral encephalomalacia, ruling out hemorrhage. Neurology consultation and EEG findings excluded ongoing seizures, while MRI indicated bifrontal cerebritis with a possible abscess. Neurosurgery, concerned about a potential abscess, added Flagyl to the antibiotic regimen. Blood cultures identified Streptococcus pneumoniae. EVD placement addressed ventriculitis, leading to clinical improvement, successful extubation, and subsequent identification of a CSF leak. ENT consultation, confirmed by a CT cisternogram, diagnosed defects in the cribriform plate and sphenoid bone, prompting surgical intervention with right ethmoidectomy and mucosal grafting. Pathology revealed a necrotizing ulcer, and a follow-up MRI showed resolution of cerebritis. The patient completed a 4-week course of Rocephin, with plans for repeat imaging and a Prevnar 20 vaccine before discharge.

In a retrospective study spanning 1991 to 2005, involving 2111 neurosurgical procedures, the incidence of postoperative central nervous system infection (PCNSI) was less than 1%, irrespective of factors like cerebrospinal fluid leakage and diabetes. Targeted prophylaxis against pathogens, especially Staphylococcus aureus and Propionibacterium acnes, is crucial in high-risk cases. Another study examined 44 patients with cerebral abscesses post-brain surgery, emphasizing their rare and severe nature. Tumor resection (61%) was common, with Staphylococcus aureus in 41% of cases. The median duration between surgery and abscess was 1.5 months, underscoring the need for postoperative vigilance. To address the risk of postoperative cerebrospinal fluid leakage, proposed strategies include autologous bone plate placement. A modified approach in cases with disrupted barriers demonstrated success in 183 patients, cautioning against surgical injury, especially anticipating postoperative irradiation, reflecting ongoing efforts towards refined neurosurgical practices. This case underscores the atypical nature of his complications, diagnostic complexities, and multifaceted management of bacterial meningitis, including the rarity of cerebritis and the development of a CSF leak this long after a neurosurgical procedure. It emphasizes the importance of interdisciplinary collaboration and prompt recognition in managing not only the common but also the atypical clinical presentations of this life-threatening condition. Further investigations are required to understand the long-term implications of intracranial complications in patients with bacterial meningitis.
Idiopathic Communicating Hydrocephalus: Not all Ventricles are Created Equally

Hydrocephalus occurs when there is abnormal accumulation of cerebrospinal fluid in the ventricles, or spaces within the brain. This can be due to either overproduction, obstruction, or problems with reabsorption of CSF fluid. There are two classes of NPH, idiopathic or primary NPH, where identifiable cause is found, and symptomatic (secondary) NPH. In adults, idiopathic NPH is the most common, a type of communicating hydrocephalus. Characteristically, NPH presents with the triad of gait ataxia, cognitive impairment, and incontinence. However, nonspecific complaints such as headaches, decreased attention and concentration, and impaired executive function, may also be present. This array of symptoms can make it difficult to parse out whether a patient needs a more extensive workup. We present a case of idiopathic communicating hydrocephalus in an otherwise healthy 30 year old male whose chief complaint was refractory headache of one week duration.

The patient was a 30 year-old-male with PMH of Lumbar and Sacral Fractures secondary to motor vehicle accident (MVA) 5 years ago presenting for one week of progressively worsening headache. Headache was described as both sharp and dull/throbbing with varying locations (frontal, temporal, occipital) and severity and did not seem to have any provocative or palliative factors. Patient endorsed associated disruption of coordination and balance during this timeframe as well as one episode of associated emesis since onset of symptoms. He reports he has had headaches in the past, but never this severe and persistent.

Vital signs were normal on admission, and detailed neurological examinations, including gait testing, were unremarkable. The emergency department performed a non-contrast CT head due to uncertainty of the origin of the patients’ headache, which showed massive communicating hydrocephalus (see image 1) without midline shift, mass, fracture, or bleeding. By this time, the patient’s headache was starting to improve with medication. Neurosurgery was consulted and recommended a ventriculoperitoneal (VP) shunt. After the VP shunt was placed (see Image 2), the patient’s headache resolved and he was successfully discharged with close outpatient follow-up.

The overall global prevalence of hydrocephalus is roughly 85 per 100,000 individuals, with a bimodal distribution occurring in the pediatric and elderly populations. Gait disturbance generally is the first and most common symptom to develop. MRI is the imaging modality of choice and the classic finding of NPH on imaging is ventriculomegaly out of proportion to sulci enlargement without gross obstruction to CSF flow. Ventriculomegaly is defined by a modified Evans ratio of greater than 0.3. This is calculated by dividing the maximum diameter of the frontal horns of the lateral ventricles by the width of the cranial cavity at the same level. The treatment for NPH is placement of a ventricular shunt, most commonly a VP shunt.
Malignancy-Associated Thrombotic Thrombocytopenic Purpura: A Case of Diagnostic Challenges and Therapeutic Strategies

Thrombotic Thrombocytopenic Purpura (TTP) is a rare and life-threatening condition that is imperative to recognize early as even with therapy the estimated mortality is 10 - 20%. The presented case is of a young woman with metastatic breast cancer who acquired TTP.

47-year-old woman with recently diagnosed angioinvasive ductal carcinoma three weeks prior presented with rapidly worsening weakness, fatigue, tachycardia, dyspnea, intermittent chills, and decreased appetite. Though lethargic, she remained alert and oriented during examination, displaying tenderness solely in the right axilla due to a recent core needle biopsy, with no other notable skin lesions or abnormalities. Her labs showed critical thrombocytopenia (22,000 platelets), a hemoglobin level of 12.9, and elevated nucleated red blood cells, atypical lymphocytes, metamyelocytes, myelocytes, basophils, and monocytes. Schistocytes were observed, alongside acute kidney injury, high anion gap metabolic acidosis, increased LDH and bilirubin, and low haptoglobin levels.

Peripheral smear analysis reduced suspicion for TTP due to few scattered schistocytes. As platelets continued to decline, accompanied by worsening lactic and respiratory acidosis, anemia and further deterioration in renal function, the possibility of TTP was reevaluated. Plasmapheresis was initiated following identification of high-density schistocytes with peripheral smear, concerning for TTP. Despite this, her condition deteriorated rapidly, culminating in respiratory failure, intubation, and subsequent cardiac arrest. Though return of spontaneous circulation (ROSC) was achieved initially, she ultimately succumbed. Postmortem ADAMTS13 levels were low (40.7%), suggestive of acquired TTP.

In cancer patients there are two primary etiologies of TTP: 1) chemotherapy, 2) malignancy itself. In the case of malignancy driven TTP it is suspected that bone marrow invasion and secondary myelofibrosis play crucial roles.

In the presented case, labs were suggestive of bone marrow invasion and nuclear medicine bone scan showed increased radiotracer uptake in the proximal humeri, distal femora, right sacrum and proximal left tibia. Microangiopathic hemolytic anemia, acute renal failure, thrombocytopenia, and schistocytes on peripheral smear raised concern for TTP and plans for plasmapheresis were made.

Plasmapheresis remains the cornerstone of therapy for TTP, however, despite intervention the mortality for cancer patients who develop TTP remains high. One study reports 75% mortality for cancer patients who had not previously received chemotherapy. These patients were treated with plasmapheresis alone, and had high rates of TTP recurrence with average survival of 1 month. The single surviving patient was treated with chemotherapy and several rounds of plasmapheresis, and survived 31 months with complete remission of TTP.

TTP remains diagnostically challenging due to ADAMSTS13 levels requiring several days to result. It requires high clinical suspicion due to high mortality even with treatment. The PLASMIC score and peripheral smear can assist with risk stratification. However, several cases report TTP was present despite absence of schistocytes on peripheral smear, while other cases report presence of schistocytes without TTP. Therefore, the entirety of the clinical picture is imperative when evaluating patients for TTP, particularly in cancer patients where symptoms of metastatic disease and TTP may overlap.
Central venous catheter (CVC) placement is commonly performed in the intensive care unit (ICU) to deliver vasopressor support to hemodynamically unstable patients. The most common location for placement is in the internal jugular (IJ) vein with other sites usually less preferred. This case highlights a rare occurrence of a left IJ vein CVC placement terminating in a segmental pulmonary vein, which indicated an anomalous pulmonary venous return.

A 75 year-old female with a history of insulin dependent diabetes, hypertension, and substance use disorder was brought to the hospital for encephalopathy. On arrival, she was hemodynamically unstable and in respiratory distress. Labs indicated severe diabetic ketoacidosis, likely as a result of not taking insulin. Given her obtunded state, she was admitted to the ICU and intubated due to worsening tachypnea. The patient required pressor support despite aggressive intravenous fluid resuscitation, indicating the need for a CVC. A left IJ triple lumen CVC was placed under ultrasound guidance using the over-the-wire technique. Location of guidewire in the vein was confirmed with ultrasound before removal. All three lumens were checked for blood draw back and were flushed before suturing the CVC in place. After the procedure, a chest X-Ray (CXR) was obtained to confirm placement before use, which showed the CVC kinked over the aortic arch with the tip projecting cephalad over the left fourth rib. There was concern that the CVC was incorrectly placed in an artery so blood gasses were ordered from both the CVC and the existing arterial line. Unfortunately, the CVC was then unable to draw back blood, so a stat computed tomography angiography (CTA) study was obtained. The CTA showed the CVC going through a small vein at the level of the aortopulmonary window, with the tip terminating in a small segmental pulmonary vein within the superior left upper lobe. After confirming a non-arterial placement, the CVC was withdrawn without problem and another triple lumen CVC was placed in the femoral vein without complication.

Clinical diagnosis of isolated instances of partial anomalous venous drainage is rare. Partial anomalous pulmonary venous drainage is a congenital defect found in 10% of patients with atrial septal defects and is an incidental finding in 0.7% of routine autopsies. Most commonly, the right upper lobe pulmonary vein drains directly into the superior vena cava or right atrium. Further imaging, including echocardiography, cardiac catheterization, and angiography can help define the anatomy of partial anomalous venous drainage. If the left to right shunt involves less than 50% of the pulmonary blood flow, the patients are usually asymptomatic and further workup is not needed. Shunts involving greater than 50% of pulmonary blood flow can cause right atrial and ventricular dilatation leading to the development of dysrhythmias, right-sided heart failure, and pulmonary hypertension. This case highlights the importance of following-up CVC placement with a CXR to confirm correct placement and demonstrates a rare occurrence of abnormal pulmonary vasculature that was incidentally discovered after a routine ICU procedure.
Mycoplasma Pneumoniae Presenting with Cavitary Lung Lesions: A Case Report

Introduction:
Mycoplasma pneumoniae is a common cause of atypical community-acquired pneumonia (CAP), responsible for up to 30% of cases [1]. It typically causes mild, self-limited illness. However, in rare cases it can escalate into severe manifestations like cavitary lesions [2]. We present a case of M. pneumoniae pneumonia with multiple cavitary lesions refractory to broad-spectrum antibacterial and antifungal treatment.

Case Presentation:
A 36-year-old woman with pertinent medical history of alcohol and tobacco use disorder presented for one week of worsening respiratory symptoms and fever. Exam showed tachycardia, tachypnea, and fever. Imaging revealed lung masses and cavitary lesions. She was admitted for atypical pneumonia and started on IV antibiotics. Despite escalating treatment, she deteriorated, requiring intensive care for hypoxemic respiratory failure. Serial imaging showed enlarging lung cavities. Bronchoalveolar lavage cultures were negative but pneumococcal antigen urine was positive. After an extensive viral, fungal, and bacterial infectious workup, ultimately Mycoplasma IgM was positive. With antibiotic adjustment including azithromycin, she clinically improved. Patient was eventually discharged in stable condition and sent home on a 9-day PO steroid taper and oral antibiotic therapy.

Discussion:
Mycoplasma pneumoniae is a common bacterial cause of upper and lower respiratory tract infections, infecting 1% of the United States population annually. It can progress to community acquired pneumonia, which is typically atypical in nature with patients experiencing mild systemic symptoms. This report illustrates a rare presentation of severe M. pneumoniae CAP causing cavitary lesions refractory to broad-spectrum antibacterial and antifungal therapy.

Mycoplasma pneumoniae is a short rod with no cell wall, which makes the pathogen not visible on Gram Stain. It is not routinely cultured because of the special culture media it requires and the length of time required for growth (7-21 days) [3]. M. pneumoniae can also not be differentiated from other causes of atypical pneumonia such as Chlamydia and Leigenalla through clinical or radiological findings. If required, PCR can be used for rapid diagnosis [4]. Cold agglutinin test can also be carried out to support clinical diagnosis of M. pneumoniae.

Our patient was found to have a positive M. pneumoniae IgM, which is indicative of acute infection. Despite the positive pneumococcal urine antigen, no growth resulted from the bronchoalveolar lavage samples that were cultured, making streptococcus pneumoniae the unlikely causative organism. Mycoplasma PCR was also performed and the result was negative. However, results may have been affected by the patient being started on antibiotics promptly in the emergency department due to meeting sepsis criteria. Hence, antibiotics were initiated before samples for PCR were collected. Overall, this would indicate that mycoplasma pneumoniae was the most likely cause of the infectious process.

M. pneumoniae should be considered as a potential etiology of cavitary pneumonia after common bacterial and fungal etiologies have been reasonably excluded. Macrolide therapy led to clinical improvement in this patient, highlighting the importance of empiric therapy for atypical organisms in severe CAP even with negative cultures.
Detection of Air Embolism on Transesophageal Echocardiography during Placement of a Left Atrial Appendage Occluder Device.

“Percutaneous left atrial appendage (LAA) closure is a routinely performed procedure to reduce the risk of stroke in patients suffering from atrial fibrillation, when oral anticoagulation is deemed high risk due to bleeding complications. Among the complications (pulmonary artery perforation, pericardial effusion) air embolism is rare. However, TEE visualization of air embolism is rare and only described in a single prior case report.

A 71-year-old female with a past medical history of hypertension, hyperlipidemia, atrial fibrillation, underwent an elective left atrial appendage occlusion with the Amulet device. The catheter was inserted into the right femoral vein and advanced to the left atrium following an interatrial septal puncture under TEE guidance. During preparation of the amulet device, a 14 French sheath was used. After establishing proper sheath positioning using contrast injection through a pigtail catheter, ST segment elevations were noted on the cardiac monitor which coincided with a transient distal septum and ventricular apex hyper echogenicity on TEE. There was also new hypokinesis of the basal inferior wall. Coronary air embolism was suspected. Urgent coronary angiography demonstrated air trapping in the right coronary artery and air embolization distal to a high-grade stenosis in the proximal right coronary artery. Multiple balloon inflations without stent placement were performed that restored flow. A drug eluting stent was placed in the proximal RCA, The patient was then transferred to the intensive care unit for further observation where she recovered without further sequelae.

There has been concern about the type of sheath used to introduce the amulet device into the heart and the risk of air embolism forming. This has accumulated in a type of sheath Called the Amplatzer sheath, made by Abbot, to be recalled. The steerable delivery sheath used to insert the company’s Amplatzer Amulet implant through the skin has the potential to create an air embolism—which, as it travels through the vessels to different organs, can cause sudden reductions in blood flow to the heart or consequences as serious as stroke and death. In our scenario, air entered the system despite careful catheter flushing. It appeared as though having to perform multiple transeptal punctures in order to establish adequate positioning for the amulet may have introduced air into the left atrium inadvertently. Fortunately, the presence of an experienced cardiologist performing TEE allowed rapid detection of the air embolism.

TEE detection of air embolism during Amulet procedure is a rare finding. The early diagnosis and intervention for our patient led to a favorable outcome. In addition to refinements in Amulet techniques, further studies using TEE as a diagnostic tool are needed to diagnose air embolism. The type of sheath used may increase the risk of air embolism formation, even with careful and proper catheter technique. Furthermore, Clinicians should be cognizant of sheath recalls and use sheaths associated with decreased air embolism risk.
Puzzling Pancreatitis: Unraveling the Unusual Connection to Paxlovid

Introduction:

The increased utilization of Paxlovid during the COVID-19 pandemic has coincided with a notable uptick in cases of acute pancreatitis, prompting exploration of a potential link. This case report delves into the details of a distinctive case involving a 67-year-old female who developed pancreatitis two days after initiating Paxlovid treatment.

Case Description:

Patient was a 67 year old female with no known past medical history, that initially presented to the hospital due to a new onset cough, fevers, chills for 2 days. Subsequent testing revealed that the patient was COVID-19 positive. She was subsequently started on Paxlovid and anticipated for discharge next day. On the second day of treatment, the patient began to complain about new-onset severe abdominal pain radiating to the back which was associated with nausea and vomiting. Lab testing revealed a lipase level of 962, and she was diagnosed with pancreatitis and etiology was worked-up. The patient had acceptable alcohol use, with about 5-6 drinks a week. An abdominal ultrasound did not reveal any gallstones. Lipid panel was also within normal limits. Over the course of the patient’s hospital stay, she improved very quickly and was discharged in stable condition.

Discussion:

The surge in Paxlovid usage during the COVID-19 pandemic has coincided with an increased incidence of acute pancreatitis among these patients, suggesting a potential correlation. Paxlovid, a combination drug of protease inhibitors Nirmatrelvir and Ritonavir, acts by inhibiting proteases involved in viral replication. Notably, existing literature highlights a suggested link between Ritonavir and acute pancreatitis, with some few proposed mechanisms, including protease inhibitor-induced hypertriglyceridemia, idiosyncratic delayed immunologic or T-cell mediated response, or mitochondrial toxicity. As this patient’s triglycerides were within normal range, the latter proposed mechanism seems more plausible. Another hypothesized explanation points to the abundance of ACE2 receptors in the gastrointestinal tract, providing an easily accessible attachment site for the virus, potentially leading to pancreatitis. Although one could argue that the pancreatitis episode may be attributed to the COVID-19 infection itself, the timing of the pancreatitis in relation to the initiation of Paxlovid in our case raises the likelihood that the drug served as the primary inciting factor.

Conclusion:

Evaluating drug-induced pancreatitis becomes particularly challenging in cases with multiple risk factors. Hence, clinicians must exercise heightened awareness when prescribing such medications to such patients. Given the limited number of reported cases linking Paxlovid to pancreatitis, additional studies are imperative to establish and strengthen this association. Such studies may further broaden the limitations and contraindications of this medication.
Introduction: About 40% of the adult U.S. population uses over-the-counter antacids. Among them, bismuth subsalicylate is the most common, comprising 60% of the market. Each tablespoon (15 ml) of bismuth sub-salicylate contains 130 mg of salicylate, or 230 mg per recommended dose. Original Pepto-Bismol is sold in sizes ranging from 118 ml to 473 ml. One large bottle of bismuth sub-salicylate roughly contains 4 grams of salicylate. It requires only one and a half large bottles of bismuth sub-salicylate, 6.5 g, to cause acute salicylate toxicity. Salicylate disrupts the mitochondrial oxidative phosphorylation, inhibiting Tricarboxylic Acid Cycle and mitochondrial ATP production leading to lactic acidosis and accumulation of ketones causing increased anion gap metabolic acidosis.

Objective: demonstrate how bismuth sub-salicylate overuse/misuse can precipitate salicylate toxicity.

Case: A 64-year-old male presented to the emergency department with a complaint of new-onset, progressively worsening nausea, bilateral tinnitus, and an episode of melanotic stools. He reported taking one-and-a-half bottles of bismuth subsalicylate (estimated dose of 700 ml) to self-medicate for nausea, after which he developed tinnitus and the nausea worsened. He further reported taking two naproxen-sodium tablets per day for 2 months to address post-exercise muscle pain. The physical exam was significant for decreased bilateral hearing and a black tongue. Initial labs showed salicylate levels of 34.0 mg/dL. Venous blood gas showed a pH of 7.39, PCO2 of 39mmHg, PO2 of 44mmHg, HCO3 of 23 mmol/L. He was started on a bicarbonate drip to alkalinize the urine. Symptoms improved. There was no indication for dialysis. Follow-up labs showed salicylate levels of 20.1 mg/dL; venous blood gas pH of 7.52, PCO2 of 32mmHg, PO2 of 65mmHg, HCO3 of 25mmol/L.

Conclusion: overdose of bismuth sub-salicylate can cause salicylate toxicity, requiring appropriate treatment.
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Introduction:

Sudden Cardiac Death (SCD) is a complex, multi-faceted phenomenon that can strike at any stage of life. On presentation, the physician must collectively save the patient’s life, prevent recurrence, discover the etiology, and explain it to family members who are likely traumatized by such an event. SCD is defined as cardiac arrest and inability for the heart to perfuse the body within one hour of onset of symptoms. When a patient is only in their third decade of life with no risk factors for heart disease, uncommon etiologies need to be worked up. This is a case about a cardiac arrest in a 22-year-old male that we believe was due to Short QT Syndrome.

Summary:

A 22 year-old male with no significant medical history presented to the ER one morning after a cardiac arrest with CPR in progress. EMS was called to the home and found his friends performing CPR. It was reported that he smoked some marijuana right before the event but did not use any other drugs. The patient’s father endorsed several family members with “heart problems” in early adulthood. He was in V-fib arrest for 60 minutes and was shocked multiple times before regaining circulation. Labs were significant for a potassium of 2.8. A post-arrest ECG showed a short QT of 330ms with no ischemic changes and an ECHO showed an Ejection Fraction of 40%. Due to loss of neurological function, the patient was intubated and admitted to the ICU. After two weeks, he was able to receive a feeding tube and tracheostomy and move out of the ICU. Electrophysiology evaluated the patient and believed his hypokalemia shortened his already short QT enough to cause the near-fatal arrhythmia. He was started on metoprolol for heart rate control, and recommended strict electrolyte control. Family was recommended for further evaluation and prevention. He was discharged in stable condition with a feeding tube and tracheostomy, and moved to a Long Term Acute Care Hospital for further recovery.

Discussion:

Short QT Syndrome is one of the rarer forms of Sudden Cardiac Death, with a prevalence of 0.05% (or 45/100,000) in the adult population. It is defined as having a QT interval under 360ms. Incidence of cardiac arrest is 0.4% in patients with no previous arrest. It is passed on in an autosomal dominant fashion, and more prevalent in males. It is important to quickly resuscitate such patients, consult specialists to help diagnose the etiology, and keep the disease under control. Additionally, it is emphasized to the family to be worked up for cardiac abnormalities, hopefully preventing other family members from suffering such a tragic event.
That's Not the Smile that the Patient was Expecting!

Introduction:

Internal carotid artery dissection (ICAD) represents an important cause of cerebrovascular accidents in young and middle-aged patients [1]. About 25% of strokes in patients under the age of 45 are related to cervicocephalic artery dissections [2-3]. The most common causes include trauma and connective tissue disorders.

Case presentation:

A 47-year-old female with a medical history of type 2 diabetes and hyperlipidemia presented to the Emergency Department with complaints of sudden onset dysarthria and left sided facial droop. These symptoms persisted for less than 5 minutes. On admission, the blood pressure was recorded at 156/112, and initial lab results were unremarkable. The National Institutes of Health Stroke Scale (NIHSS) score at that time was 0.

CT angiography of the head and neck revealed left carotid dissection with thrombus. The patient was promptly initiated on Integrihin, and aspirin and Plavix were administered. Subsequent cerebral angiography confirmed left carotid dissection/ulcerated plaque with luminal thrombus, leading to the placement of a carotid stent. However, the patient experienced a recurrence of dysarthria after the carotid stent placement. Imaging studies, including CT head and neck and CT angiography, indicated no significant vessel occlusion or stent thrombosis.

Subsequent labs showed hemoglobin A1c of 6% and a LDL level of 95; an echocardiogram with a bubble study was unremarkable. The patient was discharged on dual antiplatelet therapy and speech therapy.

Prior to discharge, the patient mentioned that she had a dental appointment for a root canal of her left maxillary molar. She stated that it went longer than expected. During that time, she had her head extended and in right rotation, which put a strain on her neck, according to the patient. This may have been the etiology for the development of this patient’s carotid dissection.

Discussion:

This case demonstrates an unusual manifestation of internal carotid artery dissection (ICAD) that emerged subsequently to prolonged neck extension during dental procedures and possibly as a result of direct trauma from a lidocaine injection. Siwiec and Solomon [4] described a patient with a bilateral ICA dissection after removal of a right tooth abscess. Cerrato et al [5] and De Santis et al [6] described a woman with a severe left periodontal infection, suffering from a left ICA dissection after mandibular third molar extraction. The authors hypothesized that mechanical injury of the dental extraction and the periodontal infection might represent the causative factors. [5-6] Microbial agents and an indirect inflammatory and immunological host response, with activation of cytokines and proteases, could induce excessive extracellular matrix degradation and thus weaken the vessel wall. [7] These add credence to the notion that internists should consider inquiring about recent dental surgeries when admitting their stroke patients.
A case of Streptococcus sanguinis endocarditis mimicking ANCA-associated vasculitis

Introduction:
The presentation of bacterial endocarditis may vary and mimic rheumatic disease. ANCA-positive bacterial endocarditis constitutes a special subgroup. ANCA positivity was detected in 18-43% of infectious endocarditis (IE), with 79% c-ANCA/PR3, 11% ANCA/MPO and 8% double positive. Here we present a male with a vasculitic rash later diagnosed with streptococcus sanguinis IE. He has positive c-ANCA and biopsy evidence of leukocytoclastic vasculitis (LCV).

Case:
A 50-year old male presented with rash. Medical history significant for ESRD 2/2 FSGS, chronic diastolic heart failure, aortic insufficiency and essential hypertension.

He initially developed a rash on left hand 2 days prior to presentation spreading to the upper left arm. He noticed continued spread to bilaterally lower extremities with mild itchiness. He denies recent vaccinations, new medications, or dental procedures. He started hemodialysis a month prior by left arm AV graft.

ROS: Intermittent sharp burning chest pain for a month alleviated by leaning forward (he finished 5-day prednisone without improvement). Dark urine and fatigue. Otherwise negative.

Physical exam on presentation: blood pressure 131/55, HR 103, RR 22, afebrile, 100% saturation on room air. Petechiae and purpura with central scabbing over the left arm, bilateral feet, cheeks and nose tip. Non-pitting edema on left lower leg and left arm.

Pertinent labs: WBC 7.84 K/mcL, hemoglobin 8.2 g/dL, platelet 154 K/mcL. Urinalysis showed moderate blood with 20 RBC/HPF, low C4 (10 mg/dL), elevated CRP (38 mg/L).

Autoimmune vasculitis was initially suspected with plan to initiate steroids. Extensive rheumatic workup only positive for c-ANCA (1: 80). However, TTE completed on day 2 to evaluate pericardial effusion showed worse aortic regurgitation, small to medium pericardial effusion and a new multilobulated mass attached to the aortic valve later confirmed by TEE. Blood culture obtained grew Streptococcus sanguinis. Ceftriaxone was started on day 3.

On day 9, he underwent aortic valve replacement surgery. Repeat blood cultures were negative. Patient was discharged on day 17 to complete a 6-week course of ceftriaxone, with resolution of edema and near resolution of rash.

Discussion:
SBE may present with positive ANCA and LCV. In a study among 110 IE, biopsy-proved cutaneous LCV was found in 4. Reversely, retrospective study of 138 biopsy-proven cutaneous LCV, IE was identified as the culprit in 3.

A few clues in our patient may help guide us towards SBE. His risk factors include aortic valve insufficiency, recent hemodialysis initiation and recent fistulogram for AV graft malfunction. Part of his picture represents an overlap between rheumatic disease and IE including pericardial effusion, hematuria, fatigue, intermittent fever and LCV.

Conclusion:
Patients with SBE may present with LCV and test positive for ANCA. Empirical immunosuppression may improve ANCA vasculitis while causing fatal progression in SBE. Therefore, early recognition of infection in ANCA positive endocarditis is crucial. Careful review of history with high suspicion in patients with risk factors may help clinicians avoid extensive workup and initiate antibiotics earlier.
Rapidly Progressive Stenosis: Mild to Wild in One Year
Introduction: Cardiovascular disease is the number one cause of mortality in patients with CKD. As CKD progresses to ESRD, it is common practice to perform a cardiac catheterization prior to renal transplant surgery. This is a case of rapidly progressive CAD found at time of transplant despite screening one year prior to transplant.

Case description: A 67 year old male with the past medical history of ESRD on hemodialysis, DM2, HTN, PVD, CAD, TIA, and hyperparathyroidism s/p parathyroidectomy presented to the hospital for a renal transplant. There were no intra-operative complications. His postoperative renal transplant course was complicated by NSTEMI with chest pain radiating to the left arm that did not respond to nitroglycerin drip. Given his clinical picture he underwent emergent left heart coronary angiography. He was found to have rapid progression of coronary artery disease compared to his preoperative catheterization one year prior. His mid left anterior descending lesion progressed from 50% to 80%, a new 90% stenosis ostial left circumflex lesion, and known proximal right coronary stenosis progressed from 40% to 70%. CTS was consulted for multivessel disease, but deemed too high risk given recent transplant and high STS score. The hospital course was further complicated by acute blood loss anemia from moderate hemorrhage around the transplanted kidney while on heparin and clopidogrel. Heparin was discontinued, and he was transfused with 3 units of packed red blood cells. Patient was stabilized then underwent successful cardiac catheterization with PCI to the left circumflex and LAD.

Discussion: Atherosclerosis starts in our late teens and progresses over many decades. It occurs with endothelial damage, lipid formation, and fibrous cap formation. In the 1980s accelerated atherosclerosis was initially described in patients that had heart transplant, coronary artery bypass grafting (CABG), and percutaneous transluminal coronary angioplasty (PTCA). These patients had significant endothelial damage with very fast platelet aggregation and thrombus formation. ESRD patients have many risk factors for accelerated atherosclerosis including endothelial dysfunction, inflammation, oxidative stress, enhanced platelet activation, and vascular calcification. All of these factors contribute to rapidly progressive CAD. Therefore, in patients with ESRD on hemodialysis, age-adjusted cardiovascular mortality is 10-20 times higher than in the general public. Due to high risk of cardiovascular disease, it is a common strategy to screen for CAD prior to renal transplant surgery. Common forms of screening include EKGs, echocardiograms, and cardiac catheterization. Since cardiac catheterization is the gold standard to evaluate the coronary arteries; it is a common practice for high risk patients to undergo a cardiac catheterization prior to renal transplant surgery. However, this common strategy of preoperative cardiac catheterization failed due to rapidly accelerated atherosclerosis and escalation to therapeutic cardiac catheterization still had to be performed post-transplant.

Conclusion: Rapidly progressive atherosclerosis is seen in ESRD patients; therefore, even with pre-operative cardiac cath, rapidly CAD may require post-transplant intervention.

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**Resident Poster # 017**  
**Category:** Clinical Vignette

**Residency Program:** Ascension St. John  
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**Recurrent Right-Sided Pleural Effusions secondary to pleuroperitoneal leaks from Peritoneal Fluid**

**Introduction:** Pleural effusion is a rare but notable complication of peritoneal dialysis, with various etiological factors attributed to its pathogenesis, including pleuroperitoneal defects and high peritoneal-pleural pressures. The onset time of pleural effusions in relation to the initiation of peritoneal dialysis may serve as a crucial diagnostic marker for the underlying pathology of this condition. Notably, early onset of pleural effusions may indicate the presence of diaphragmatic defects. In this regard, the presented case report emphasizes the significance of identifying peritoneal leaks as the underlying cause of pleural effusion in dialysis patients.

**Results:** A 64-year-old female with end-stage renal disease on peritoneal dialysis who presented with dyspnea. The patient had been undergoing peritoneal dialysis for approximately two months prior to presentation. On examination, the patient was found to be tachypneic and tachycardic. She reported experiencing shortness of breath symptoms for three days prior to admission, which had worsened significantly over the past day. Chest X-ray imaging revealed a large pleural effusion on the right side. An ultrasound-guided thoracentesis was performed with the removal of 2200 ml of light straw-colored transudative fluid. However, the patient experienced re-accumulation of pleural fluid on the right side two days after the thoracentesis procedure. Therefore, the patient underwent video-assisted thoracoscopic surgery with pleurodesis, where 1000 ml of peritoneal fluid was removed from the right chest. No evidence of malignancy was found and the lung was in good shape. Furthermore, the peritoneal dialysis catheter was removed and a hemodialysis catheter was placed. The patient was eventually switched to hemodialysis. A follow up chest X-ray done one month later after the initiation of hemodialysis showed no accumulation of pleural fluid.

**Conclusions:** Pleuroperitoneal leak represents an infrequent complication of peritoneal dialysis, which involves the escape of dialysate from the peritoneal cavity into the pleural space. The etiology of this condition can be traced back to various factors, including the pleuroperitoneal pressure gradient, diaphragmatic defects, and diaphragmatic muscular hypotonia. Notably, most cases of pleural effusions occur on the right side.

**Clinical implications:** It is imperative for clinicians to consider the possibility of pleuroperitoneal leaks in patients undergoing peritoneal dialysis who present with recurrent unilateral pleural effusions. Early recognition of this condition is crucial to facilitate timely and appropriate management, thereby preventing any further deterioration in respiratory function.

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**Resident Poster # 018**  
**Category:** Clinical Vignette

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You aren’t You When You are Hypoglycemic”, Anterior Hypopituitarism In Setting of Post COVID

Introduction:
Hypopituitarism is the deficiency of one or more of the hormones of the anterior or posterior pituitary gland. It presents as symptoms of deficiency of the hormone that is deficient, such as low cortisol can present as postural hypotension, fatigue, anorexia, weight loss, decreased libido, eosinophilia, and hypoglycemia. Hypopituitarism can result from tumor compression of normal pituitary cells, traumatic brain injury, medications, cranial surgery, or radiation therapy. Our case is a patient post-COVID with a history of pituitary tumor resection.

Case Report:
An 86 y/o male with pmhx of bipolar, A-fib, hypertension, and obesity, presented to the ED after a syncopal episode. Patient was found to be tachycardic and given a fluid bolus with improvement. He was admitted for further workup. Patient was found to have a fasting glucose of 43. A1c was checked to see if the patient was newly diabetic, but came back normal. His fasting glucose the next morning was 31. It was suspected to be the cause of his syncope. Endocrinology was consulted and a cortisol and growth hormone level were ordered off the labs with the fasting glucose. These resulted as low with inappropriate response to hypoglycemia. Further labs of ACTH, AM cortisol, LH, IGF, FSH, Testosterone, TSH, T4, and prolactin were ordered. The family confirmed that the patient did have a pituitary tumor back in 2006 that was removed with no recurrence. Patient was also hospitalized the month prior for a COVID infection. The lab results showed a low ACTH, low AM cortisol, low LH and FSH, low testosterone, and low IGF. TSH was normal with a low T4. These all pointed towards anterior hypopituitarism hence the patient was started on hydrocortisone and levothyroxine. Patient had previously been requiring D5LR at 75cc/hr with POC glucose q4hr. MRI was obtained to rule out tumor recurrence, however was negative. After the steroids were started, he was weaned off the fluids. Patient’s fasting sugars responded to the hydrocortisone. Patient was encouraged to eat small frequent meals and a snack before bed to keep his sugars stable. Patient was discharged to a subacute rehab and will follow up with Endocrinology outpatient for steroid and levothyroxine management.

Discussion:
Multiple cases have been reported of the pituitary gland being affected post-COVID. It is important to keep this in mind when a patient presents to the hospital with hormone deficiency symptoms. The hypothalamus and pituitary glands are targets for SARS-CoV-2 due to the expression of ACE-2 receptors on the surface of their cells. Long-COVID symptoms include tiredness, headache, dyspnea, myalgia, impaired concentration, memory impairment, depression, anxiety disorder, insomnia, palpitations, diabetes, and thromboembolism. These symptoms could be due to hypopituitarism and COVIDs effect on the pituitary gland. It is important for prompt treatment of hormonal deficiencies, especially in the setting of low cortisol and thyroid deficiency.

References:

Left Atrial Septal Pouch as Nidus for Recurrent Cardioembolic Stroke

Background: Stroke patients require a thorough evaluation, including all possible embolic sources. Suspect atherosclerotic disease and atrial fibrillation along with thrombosis from the heart. However, a thrombotic source that may remain unknown to many is an atrial septal pouch. This structure, which is formed by the incomplete fusion of the septum primum and septum secundum, may represent a significant thrombotic nidus.

Case: A 55 year old female with a medical history of diabetes mellitus, coronary artery disease and hyperlipidemia presented to the emergency department for evaluation of diplopia. Initial CT imaging was negative. On the following day the patient began to experience right sided paresthesia and MRI imaging was subsequently obtained and noted acute infarction of the left thalamus and cuneus. On day three of admission the patient began to experience acute dysarthria and L-sided facial droop. Repeat CT imaging done at that time was negative for any new infarcts. Transthoracic echocardiogram (TTE) with contrast was negative for patent foramen ovale or atrial septal defect. Transesophageal echocardiogram (TEE) noted a patent foramen ovale, left atrial septal pouch and echodense mass suggestive of thrombus within the septal pouch. The patient was started on IV heparin, which would later be transitioned to rivaroxaban on discharge.

Discussion/Decision Making: Due to patient’s recurrent stroke symptoms, a thrombo-embolic source was highly suspected. TEE was pursued for further visualization of the left atrial appendage and atrial septum. TEE imaging was a crucial tool in order diagnose the patient’s source of thrombus and thus cause of stroke. Due to a lack of evidence, we decided with the patient that a conservative strategy consisting of oral anticoagulation would be best.

Conclusion: The left atrial septal pouch represents another, less known nidus for cardioembolic stroke. In patients with suspected embolic stroke a thorough workup for cardiac source should be performed. TEE with the use of agitated saline is considered the gold standard for diagnosis. In regards to treatment, no guidelines exist to steer towards an invasive exclusion versus medical management with anticoagulation.
Unleashing the Thunder: A Case of Electrical Storm

Introduction:
Electrical storm is a life-threatening syndrome that is defined by 3 or more sustained episodes of ventricular tachycardia, ventricular fibrillation, or appropriate shocks from an implantable cardioverter-defibrillator (ICD) within 24 hours. Electrical storm can manifest itself during acute myocardial infarction and in patients who have structural heart disease, or an inherited arrhythmic syndrome. Today we present a case of electrical storm in a patient with a known history of infiltrative cardiomyopathy due to sarcoidosis.

Case Report:
A 60-year-old woman with a medical history significant for atrial fibrillation on Eliquis, infiltrative cardiomyopathy due to sarcoidosis with an ICD in place, hyperlipidemia, and hypertension presented to the emergency department after receiving a shock from her defibrillator.

The patient reported a sudden onset of uneasiness in her upper epigastric region, escalating until the defibrillator shock prompted her visit to the emergency department. On arrival, she experienced six rounds of sustained monomorphic ventricular tachycardia, remaining hemodynamically stable but expressing discomfort characterized by heaviness and esophageal contractions. Intravenous administration of amiodarone, lidocaine, and metoprolol tartrate initially alleviated symptoms. She was subsequently transferred to the cardiovascular intensive care unit (CVICU).

In the CVICU, the patient continued to experience episodes of sustained ventricular tachycardia. She was started on a combination of a lidocaine drip, esmolol drip, and continued on amiodarone. This regimen successfully restored normal sinus rhythm. Upon discharge, she was prescribed oral amiodarone (400 mg twice daily), toprol-XL (75 mg twice daily), and mexiletine (150 mg three times daily).

Discussion:
The origin of electrical storms spans a spectrum, from myocardial ischemia to infiltrative cardiomyopathy, as observed in our patient. Decoding the morphology of the QRS complex serves as a diagnostic clue, with monomorphic ventricular tachycardias typically linked to structural heart diseases, while polymorphic ventricular tachycardias are associated with myocardial ischemia. Symptoms may manifest as palpitations, dizziness, and syncope.

Early intervention necessitates the identification and correction of underlying issues such as ischemia, electrolyte imbalances, or other contributing factors. Antiarrhythmic therapy, primarily by amiodarone and β-blockers constitutes the initial line of management for most patients.

Conclusion:
Electrical storms can cause rapid clinical deterioration in patients. Quick identification of the condition and initiation of treatment is crucial for a good prognosis. Timely intervention is imperative not only for the immediate stabilization of the patient but also for steering the course towards a favorable outcome. The urgency arises from the fact that an electrical storm is not merely a symptom but rather a manifestation of an underlying condition. To improve the prognosis, it is essential to delve into the root cause or inciting event that triggered the electrical storm. In essence, the clinical management of electrical storm necessitates a dual focus – the immediate quelling of the acute arrhythmic storm and a comprehensive exploration of the causative factors.
A Rare Case of Computer Duster Inhalant Abuse Complicated by Thoracic Syringomyelia

Introduction: Computer duster is a widely available aerosolized can used for cleaning and contains Nitrous Oxide, Difluoroethane, Alkyl Nitrates and various other chemicals. Nitrous Oxide and amyl nitrates have become commonly abused insufflated drugs recently. Nitrous oxide irreversibly oxidizes the cobalt ion of cobalamin (Vitamin B12) depleting the body’s vitamin B12 altering myelin formation. Nitrous oxide use is not common, but can be associated with the formation of syringomyelia.

Case: A 23-year-old female with a history of psychiatric disorders presented with severe bilateral lower extremity pain for two weeks. The pain is 10/10 in intensity, sharp and achy in nature, feeling like “shin-splints” radiating down her legs. It is steady and exacerbated by ambulation affecting ADLs and light touch triggering a crippling pain. She was seen at several other hospitals and was diagnosed with Gout and was given Decadron and Indomethacin which did not relieve the pain. Interestingly, the pain is alleviated by inhaling computer duster. Upon further questioning, she mentions that she has been inhaling two cans of computer duster daily for the past two years. The pain is also worse when the effect of the duster subsides. On presentation, the patient was afebrile, tachypneic and tachycardic. Physical exam was notable for extreme tenderness to light touch and decreased strength of the bilateral lower extremities. Labs showed hemoglobin of 10.7 g/dl, platelet count of 415 K/mcL, Ferritin of 44 ng/mL, iron saturation of 7.4%, folate of 7.3 ng/mL, Vitamin B12 of 231 and methylmalonic acid level was elevated. Syphilis, HIV and ANA screen are negative. MRI of the cervical, thoracic, and lumbar spine with and without contrast revealed a thoracic spinal cord syrinx measuring up to 3mm in diameter spanning from C7-T1 to approximately T9-T10. As well as a T11-T12 small disc herniation with mild spinal canal stenosis and some degenerative changes with mild to moderate foraminal narrowing at C3-C4. The patient was promptly started on Vitamin B12 injections along with IV Decadron 10 mg q6hrs. She reported relief in symptoms after 3 days and then discharged with vitamin B12 supplements and a Medrol dose pack.

Discussion: Severe bilateral lower extremity pain and decreased strength experienced by the patient are likely caused by syringomyelia resulting from inhaling Nitrous Oxide in a computer duster. Nitrous Oxide abuse led to Vitamin B12 depletion and consequently myelin damage, which contributed to the development of the spinal cord syrinx. This case highlights the potential dangers of uncontrolled products sold over the counter leading to abuse. Timely recognition of Nitrous Oxide inhalant abuse and its associated neurological complications such as syringomyelia is crucial for appropriate intervention and management to prevent further harm and improve patient outcomes. This case also highlights the importance of history taking as internists.
A Case of Sparganosis in Breast Tissue

Introduction:

Sparganosis is a parasitic infection, caused by Spirometra tapeworm larvae, which usually presents in humans as a subcutaneous nodule. Infection in the United States is rare, with the majority of cases reported in east and southeast Asia. As of 1996, approximately 62 cases of sparganosis were reported in the United States. We highlight the importance of awareness of parasitic infections in the differential diagnosis of an enlarging breast mass.

Case:

A 69-year-old male with hypothyroidism presented for a right breast lump. He noticed it three years ago, and since then it has varied in size, growing and shrinking unpredictably. He stated it had become increasingly pruritic over the last 12 months and endorsed occasional tenderness. He denied nipple discharge. He denied ingestion of uncooked seafood, raw frogs, tadpoles, snakes, or residence in Southeast Asia. However, he endorsed that as a child he played with tadpoles and frogs. He lived in rural Arkansas and drank well water until adolescence. Vitals were unremarkable and exam showed a firm, tender right chest wall lump without induration or drainage. Complete blood count showed a normal white blood cell count without eosinophilia. Ultrasound and mammogram showed a palpable mass measuring 1.2 x 0.6 x 0.9 cm suspicious for an epidermal inclusion cyst. Core needle biopsy showed an organizing cyst wall with foreign body reaction to an organism compatible with sparganosis and negative for malignancy. Excisional biopsy showed focal larval organisms compatible with spargana. Four lymph nodes were also biopsied and found to be reactive, negative for malignancy. When evaluated two weeks after excision he stated his symptoms resolved.

Discussion:

Sparganosis is a zoonotic infection caused by ingestion of larvae, or spargana, of tapeworms in the genus Spirometra. Humans can remain asymptomatic for years before developing pain at the site of infection. Spargana may invade the central nervous system, orbits, genitourinary, pleural cavity, or subcutaneous tissues. Visceral and/or proliferative sparganosis invades multiple organs and may have devastating consequences. Human beings are considered the second intermediate host of the larvae, usually contracting the infection via consumption of water or meat infected by Spirometra. In our patient, the consumption of well water as an adolescent may have been the source of his infection. Sparganosis only manifests as breast disease in less than two percent of cases. The patient’s ultrasound and mammogram results were interpreted as a possible epidermal inclusion cyst, which only requires biopsy if the lesion is symptomatic or greater than 2 cm and palpable. This highlights the importance of awareness for other conditions that may mimic epidermal inclusion cyst type findings, such as parasitic infections, as without a biopsy this condition may have gone untreated.
Residency Program: Ascension St. John  
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**Thyrotoxicosis Precipitated by Pembrolizumab Initiation**

**Introduction**

Pembrolizumab, or Keytruda, is one of many immune checkpoint inhibitors used in the treatment of multiple different types of cancer, including triple-negative breast cancer. Keytruda works as an anti-programmed death-ligand 1 (PD-L1) antibody, which helps to boost the body’s immune response against cancer cells. Although it has made great strides in the treatment of certain cancers, the medications in this class have been associated with various immune related adverse events (irAEs). The incidence of irAEs has been found to be approximately 57-79.5% of patients on Keytruda (1). Of these patients, up to 10.1% may experience hypothyroidism and up to 6.5% may experience hyperthyroidism, according to the KEYNOTE-006 phase III trial (2).

**Case Presentation**

Here we present a case of a 59-year-old female patient with significant autoimmune history including lupus and rheumatoid arthritis who presented with thyrotoxicosis in the setting of recent Pembrolizumab initiation for breast cancer. The patient’s initial symptoms began about three weeks after medication initiation and included profound fatigue, heat intolerance, palpitations, and loose stool. Family history was significant for Graves’ disease in her daughter and hypothyroidism present in her mother. Labs were significant for decreased thyroid stimulating hormone (TSH) along with elevated Free T4 and T3 levels. Thyroid ultrasound revealed mild increase in size with diffuse heterogeneous hypoechoic echotexture and diffuse hyperemia, potentially concerning for Graves’ disease as opposed to thyroiditis due to the increased vascularity. Although thyroperoxidase and thyroglobulin antibodies were both normal, a radioactive uptake scan is pending to definitively confirm the diagnosis. In any case, treatment has been initiated with propranolol and dexamethasone daily with improvement of symptoms.

**Discussion**

Quick identification of an irAE, cessation of the offending agent, and initiation of symptomatic treatment is important to uphold a patient’s quality of life. Although many of these patients may present with cytopenias and a clinical picture concerning for an infectious source due to their immunocompromised state, it is important for a clinician to maintain this less common but significant adverse event at the forefront of his/her mind. Personal medical history and family history significant for autoimmune processes can also be helpful aspects that aid a physician in making the diagnosis of these lesser common side effects.
A Novel Case of Reverse Takotsubo Cardiomyopathy Following Gastroenteritis

Takotsubo syndrome (TTS) also known as stress cardiomyopathy is a condition that causes a temporary change in the heart’s contractility. Generally, involves apical akinesis, basal hypercontractility, and regional wall motion abnormalities beyond single vascular distribution. Resembles acute coronary syndrome (ACS) and is hypothesized as triggered by emotional or physical stress, often affecting postmenopausal women. Reverse Takotsubo Syndrome r-TTS is a rare variant of TTS and is characterized by apical hyperkinesis and basal segment depression. We present the case of a 72-year-old male with a history of prostate cancer, diabetes mellitus, hypertension, and hyperlipidemia who developed r-TTS following symptoms of gastroenteritis. Vitals upon arrival showed a heart rate of 81 bpm, blood pressure 164/94 mmHg, and respiratory rate of 16. The patient presented with fatigue, nausea, vomiting, subjective fever/chills, and abdominal pain. Initial assessments in the emergency department revealed leukocytosis (22.92k), elevated D-dimer (5270), and elevated Troponin levels (0.11) with a repeat troponin of (0.11). EKG showed sinus rhythm, with no ST-segment elevation or depression. Furthermore, CK was elevated (309), along with a lactate of as (1.8). Chest x-ray and CTA chest were unremarkable ruling out pulmonary embolism. Patient was initially managed for suspected sepsis with empiric antibiotics. Further investigation for elevated troponin was ordered to rule out coronary artery disease. Echocardiography revealed severe regional hypokinesis of the basal-mid anterolateral wall and akinesis of the basal-mid anterior along with the inferolateral wall. Moderate regurgitation of the mitral valve was noted with an estimated ejection fraction of 35-40%. These findings were consistent with r-TTS. Subsequent left heart catheterization ruled out ischemic cardiomyopathy, and the patient was initiated on GDMT therapy. When comparing TTS with r-TTS, elevated plasma catecholamine levels are observed in both, implicating sympathetic nervous system over-activity as a possible common etiology. Attempts to discern differences between TTS and r-TTS were attempted, revealing some distinctions. r-TTS patients are notably younger and more likely to have greater stress response as trigger. However, arguments suggesting that TTS and r-TTS may represent a temporal evolution of the same disease have been put forth as well. Instances of patients developing r-TTS several weeks to months after a typical TTS episode support this notion. Additionally, reverse TTS is noted to also occur more commonly in patients with severe brain damage; subarachnoid hemorrhage. This case emphasizes a rare occurrence of r-TTS post gastroenteritis. Per literature review only a handful of cases appear of patients suffering from TTS post gastroenteritis, and no cases are recorded of patients with r-TTS. This unique presentation may assist in discerning whether TTS is fundamentally different from r-TTS.
Isolated Aquagenic Pruritus: When Water Becomes Harmful

Introduction
Aquagenic pruritus (AP), an extremely rare disorder, is characterized by intense itching induced by contact with any form of water; it is often accompanied by uncomfortable sensations like prickling, tingling, burning, or stinging without any skin rash. Symptoms generally occur after a latent period of up to 15 minutes, and then disappear within one to two hours after exposure. AP has been commonly associated with polycythemia vera and other myelodysplastic syndromes, and rarely with lactose intolerance, hepatitis C, juvenile xanthogranuloma, and T-cell non-Hodgkin’s lymphoma. Currently, there is no effective treatment and symptoms recur sporadically, causing significant physical, psychological, and social distress.

Case Description
A 26-year-old African American male with hereditary elliptocytosis presented with a two-year history of generalized itching when in contact with pool water, sweat, shower, and rain. He states that the itching lasts at least 10-20 minutes after exposure, and is located on the torso, back, upper arms, and thighs, with an associated “pins and needles” sensation. He denies any rash, photosensitivity or exposure to chemicals or drugs. The patient works as a government employee and is exposed to fire pits and fumes, for which he uses a face mask. In-depth CBC, CMP, auto-immune, inflammatory, infectious, and nutritional workup was negative. Moisturizers and antihistamines were tried without improvement. He was then referred to an allergist; histamine, tryptase, IgE, complement, C1Q binding assay, and skin prick testing were within normal limits, amongst other labs. After relapsing with various treatment methods including nerve stimulation, a regimen combining levocetirizine and omalizumab provided intermittent relief. The patient was also advised to avoid dense water areas such as beaches or pools, use lukewarm water when showering, drying well with cotton towels, and using anti-itch lotion. Additionally, he experienced severe social isolation and depression from the symptoms, and was prescribed an SSRI along with cognitive behavioral therapy. Currently, the patient is in remission and following up closely for further work up.

Discussion
Aquagenic pruritus is a very rare condition with little known information. It is a diagnosis of exclusion and workup must rule out associated disorders. Patients with AP should be evaluated for the presence of JAK2 mutations to identify myeloproliferative neoplasms, in particular polycythemia vera, which can precede AP by many years. Although the exact pathophysiology is unknown, pharmacological studies have shown correlation with local release of acetylcholine in the skin, mast cell degranulation, and elevated histamine concentrations. Treatment is supportive, with antihistamines being the mainstay therapy. Other options include adding bicarbonate to water, topical hyoscine, and UV light therapy. Avoidance of water is not recommended; however, limiting jetstreams, temperature extremes, and swimming may be beneficial. Several case studies have assessed the effect of various treatments such as beta blockers, calamine, psychotherapy, and SSRIs with mixed results. The lack of definitive treatment options can be physically and mentally distressing for both patients and physicians. A collaborative effort with close follow up and cognizant lifestyle changes can help ensure optimal results for this challenging disorder.
From Collapse to Recovery: Thiamine Intervention in Cardiac Beriberi.

Introduction:
Thiamine deficiency, historically known as Beriberi, poses diagnostic dilemmas due to its potential occurrence without overt malnutrition or alcohol dependence. This case report explores the intricate dynamics of cardio-circulatory collapse coupled with pericardial effusion, unveiling an underlying thiamine deficiency.

Case Description:
A 68-year-old male with a medical history of hypothyroidism, heart failure, and chronic mild pericardial effusion presented with worsening dyspnea, leg edema, and hypotension. The patient's physical evaluation disclosed notable findings, including marked obesity, distant heart sounds, an audible S4 sound, and swelling in the lower extremities. Laboratory results revealed chronic anemia. Brain natriuretic peptide (BNP) was mildly elevated at 286 pg/mL, accompanied by severe hypoalbuminemia (1.6 g/dL) and low pre-albumin levels (6 mg/Dl). The coagulation panel and cardiac ischemia workup were within normal limits.

The patient underwent surgical pericardial effusion drainage with a pericardiocentesis drain. Despite the intervention, the patient developed hypotension unresponsive to fluid resuscitation, prompting a septic workup and initiation of vasopressors. We initiated thiamine replacement due to the patient’s risk factors, including significant weight loss, frequent hospitalizations with parenteral nutrition, and chronic inflammation, as well as refractory hypotension, resulting in rapid and dramatic restoration of hemodynamics and resolution of effusions. We provided intravenous thiamine 100 mg three times daily according to the guidelines.

Discussion:
Thiamine deficiency can manifest in diverse clinical presentations, complicating timely diagnosis. Its deficiency disrupts adenosine triphosphate (ATP) production, which leads to adenosine accumulation. This accumulation causes direct vasomotor depression and reduced systemic vascular resistance. It eventually leads to hypotension and cardiovascular collapse unless thiamine is replaced. The challenges in diagnosing thiamine deficiency highlight the importance of a comprehensive clinical assessment and a high index of suspicion, particularly in critical care settings where rapid diagnostic tests may be lacking. This case contributes to the growing body of evidence emphasizing the potential for thiamine deficiency in patients without explicit risk factors.

Clinical Significance:
The presented case underscores the importance of recognizing thiamine deficiency in patients with refractory cardio-circulatory collapse. Timely initiation of thiamine replacement therapy proved pivotal in reversing the hemodynamic compromise and resolving pericardial effusion. This report advocates for increased awareness among clinicians, urging them to consider thiamine deficiency as a potential etiology in critical patients, even in the absence of overt malnutrition or alcohol dependence.
AIDS Associated Cryptosporidial and CMV Cholangiopathy

Introduction

AIDS-associated cholangiopathy is a biliary tract condition seen in AIDS patients who are severely immunosuppressed, contributing to significant mortality in this population even in developed countries with access to HAART therapy.

Case Description

We discuss a thirty-six-year-old HIV positive male, non-compliant with HAART therapy, who presented with a one year history of weight loss, persistent fatigue and chronic diarrhea, which had worsened significantly in the past few weeks. Routine laboratory studies on presentation indicated elevated liver enzymes and alkaline phosphatase, a CD4 count of two, and a high HIV RNA count of 8.8 million. Imaging via CT abdomen and pelvis and ultrasound abdomen both displayed thickening and edema in the gallbladder with evidence of gallstones, raising concerns of acalculous cholecystitis. The patient subsequently decompensated, requiring intravenous vasopressors to maintain hemodynamic stability, broad-spectrum antibiotics, and resumption of antiretroviral therapy. Biliary fluid drainage was performed, and Cryptosporidium and cytomegalovirus (CMV) were detected via PCR testing. The diagnosis of AIDS cholangiopathy was established, however the patient’s diarrhea worsened upon the introduction of tube feeds. Despite ongoing antimicrobial treatment, the patient developed a fever of 101.4°F, became asystolic and subsequently passed away.

Clinical Significance

This case highlights the diagnostic, management and therapeutic challenges of AIDS cholangiopathy, and also underscores the importance of thorough investigation into even mild or intermittent diarrhea and abnormal liver function tests in all HIV-infected patients, particularly in the severely immunosuppressed patients.

Discussion

AIDS cholangiopathy should be considered in AIDS patients with diarrhea and abnormal liver function tests, irrespective of age, due to its associated morbidity across all age groups. Laboratory investigations often reveal markedly elevated alkaline phosphatase, gamma-glutamyltransferase, and mild to moderate liver enzyme elevations as hallmark findings of AIDS cholangiopathy. Ultrasonography is the first line screening modality of AIDS cholangiopathy. Cryptosporidium parvum is the most common infectious etiology of AIDS cholangiopathy, and can be identified by PCR antigen testing of the stool or acid fast staining of the stool specimens.

Early detection of HIV infection and the prompt initiation and adherence to Highly Active Antiretroviral Therapy (HAART), which helps with maintaining a normal CD4 count and a low HIV viral load through HAART therapy, thereby significantly reducing the risk of developing AIDS cholangiopathy in HIV patients.
Splenic Infarct Following Roux-en-Y Gastric Bypass Surgery

Introduction:
Roux-en-Y gastric bypass (RYGB) is a standard surgical procedure used to treat morbid obesity. Although RYGB is generally considered safe, it's essential to remain vigilant in identifying and addressing rare complications such as splenic infarction. Splenic infarction is a primarily silent condition, and because many cases go undiagnosed, the exact incidence of the condition is obscure and likely underestimated. Similarly, the incidence and severity of these rare conditions in the postoperative period for bariatric surgeries are still unknown.

Case presentation:
A 31-year-old female with morbid obesity underwent RYGB six weeks before presentation with acute abdominal pain, nausea, and a 15 lb weight loss since surgery. The pain, located in the middle of the abdomen and radiating to the back and the shoulder, had progressively worsened overnight. The patient denied alleviation of pain with bowel movements and reported no vomiting. Vitals were stable. Patient labs revealed chronic microcytic anemia; other parameters, including WBC, PLT, amylase, lipase, and electrolytes, were unremarkable. A CT abdomen/pelvis showed small bowel dilatation involving proximal small bowel loops with postsurgical changes, indicative of small bowel obstruction. Additionally, a small wedge-shaped hypodensity within the spleen was suggestive of a small splenic infarct.

Discussion:
Splenic infarction is a rare and serious complication associated with hematological disorders, trauma, and vascular interventions, particularly after laparoscopic procedures and the use of energy vessel sealing devices. Excessive use of tissue sealing systems can elevate the risk of splenic injury. Prevention involves avoiding frequent mobilization of connective tissue around vascular structures. Diagnosis is often incidental through CT scans, and while most cases are asymptomatic, complications like hemorrhage, rupture, or abscess may require surgical intervention. A nonoperative approach is considered, with surgery recommended for persistent symptoms or complications. Patients undergoing bariatric surgery should be alert to sudden abdominal pain, especially if radiating from the left upper quadrant to the left shoulder. Uncomplicated cases typically resolve within 7-14 days. Treatment varies based on underlying conditions, with options ranging from careful monitoring to surgical assessment, depending on the severity of symptoms and complications.

Clinical significance:
This case highlights the need for increased awareness of potential vascular complications, such as splenic infarction, in patients undergoing RYGB. Timely recognition and intervention are crucial for optimal patient outcomes. Surgeons should exercise caution, particularly in the use of tissue sealing systems, to prevent unnecessary mobilization of connective tissue around vascular structures. Further research is required to determine the incidence and severity of visceral infarction in patients undergoing bariatric surgeries.
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The Silent Michigan Epidemic: Unraveling Neurological Changes Posed By Recreational Nitrous Oxide Abuse

Introduction
Nitrous oxide (N2O), traditionally considered a benign, odorless gas with analgesic and anesthetic properties, has seen a rise in abuse among young individuals in the United States. This abuse can lead to clinically significant neurological symptoms, including peripheral neuropathy and subacute combined degeneration of the spinal cord due to functional B12 deficiency induced by N2O. This case series examines four patients with N2O toxicity, and the functional recovery observed during subsequent follow-up visits after appropriate management. We will explore their motivation for abusing N2O, their sources of access, and why its use is on the rise.

Cases Description:
The first two cases involve young females under 30 years old with a history of recreational nitrous oxide use for two months and a year, respectively. They presented with acute onset ascending bilateral upper and lower extremity weakness and numbness, one with concurrent urinary incontinence. Physical examinations revealed reduced motor strength and sensation of light touch and vibration in all extremities. Initial workup showed decreased Vitamin B12 levels, unremarkable CSF studies, and cervical MRI findings consistent with subacute combined degeneration of the spinal cord. Both patients received four weeks of vitamin B12 supplementation, resulting in near-complete functional recovery.

The next two cases involve adults in their 20s with approximately six-month history of recreational nitrous oxide use, presenting with bilateral upper and lower extremity numbness and paresthesia’s. Physical examinations revealed diminished light touch, pin prick and vibration sense in all extremities along with absent reflexes in bilateral lower extremities. Both had decreased serum vitamin B12 levels of <200 pg/ml, unremarkable CSF studies, and MRI findings. Treatment varied, with one patient receiving duloxetine and B12 injections, and the other managed with gabapentin and B12 injections. Both were instructed to complete a four-week course of B12 supplemental IM injections. One of the patients who was not able to complete a course of 4 weeks, did not have improvement in his symptoms on a follow-up phone call discussion.

Clinical significance:
Nitrous oxide oxidizes cobalt atom, inactivating methionine synthetase. This disruption impairs the production of methionine from homocysteine, which is needed during DNA synthesis. Subsequently, Methyl cobalamin cannot facilitate the production of S-adenosylmethionine interrupting myelin sheath formation as seen in subacute combined degeneration.

Discussion
This case series reveals a worrying emergence of recreational nitrous oxide use in Michigan especially after the Covid-19 Pandemic. Nitrous oxide is not categorized as a controlled substance by the United states federal Law and despite the state of Michigan passing legislation such as the 2022 Senate Bill 996 to regulate use in 2018, nitrous oxide cartridges and cannisters are easily accessible for purchase in large quantities from convenience stores, vape shops and Amazon in the form of metal cannisters disguised as propellant to make whipped cream and advertised as a “harmless” gas. This false perception of a “safe-high” and the short-lasting euphoric effects of N2O result in youngsters using multiple cartridges at once with the potential risk of significant neurological morbidity.
Navigating Recurrent Acute In-Stent-thrombosis: Insights from a Single Admission Case Study

In-stent thrombosis (ISR) is one of the dreaded complications of percutaneous coronary angioplasty. Despite marked reductions in ISR since the transition from bare metal stents to drug-eluting stents, ISR still occurs at a rate of 1% - 2% per year. We present the case of a 64-year-old Caucasian male who was admitted for staged angioplasty after a left heart catheterization had shown he had significant stenosis to the LAD and mid ramus intermedius. He had DES placed to the mid-LAD and to the mid-ramus intermedius. Despite being placed on dual antiplatelet therapy after the stent placement, 6 hours later he complained of chest pain and EKG showed new ST elevations in leads 1, AVl, and V2-V6. Repeat LHC showed thrombosis to recently placed LAD and mid ramus-intermedius stents so a new set of stents was placed and the patient was loaded with eptifibatide and then restarted on dual antiplatelet therapy after procedure.

However, only 48 hours later the patient started complaining of chest pain again. EKG showed new ischemic changes and a high sensitivity troponin of 5000. He was taken to the catheterization and LHC once again late stenosis of the previously stented LAD and mid-ramus intermedius. He underwent percutaneous coronary angioplasty to these stents. Post- LHC he was started on prasugrel and aspirin instead of the 2 other times when he was on clopidogrel and aspirin then ticagrelor and aspirin.

In addition to the dual antiplatelet therapy, he was also started on a Factor X inhibitor, rivaroxaban. Our hypercoagulability investigations did not yield any positive result, however we found out the patient was on testosterone replacement.

This case is very unique in the fact that we had early re-stenosis of a drug-eluting stent and it did not only occur once, it occurred twice. One of the questions raised in this unique case is, when you have patients who have early restenosis of their stents, what is the preferred anti-platelet agent, and is a Factor Xa inhibitor indicated in this scenario? In the case of our patient, we went for prasugrel + aspirin + rivaroxaban and we did not have any re-stenosis.
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Instent thrombosis 20 years after stent placement

Stent thrombosis is not very common but it is a serious complication of percutaneous coronary intervention (PCI). Here in, we present a case of 55 years old who presented with acute chest pain, was found to have non ST elevation myocardial infarction due to a thrombotic occlusion of prior drug eluting stent placed 20 years ago.

A 55 years old male presented to the emergency department with one day history of mid-chest retrosternal pain that started while playing hockey, associated with nausea and diaphoresis. On initial evaluation, vital signs were stable, and patient was afebrile. Cardiac examination was normal with regular rate and rhythm with no evidence of murmur, gallop or rub. physical examination was unremarkable. Blood studies revealed leukocytosis with white count of 10.3. Comprehensive metabolic panel revealed creatinine of 1.60 along with normal liver function tests. Cardiac troponin was 0.04 and started trending up to reach 2.02. The initial EKG revealed normal sinus rhythm with no ST or T wave abnormalities. Patient continued to have chest pain during hospitalization, along with rising in troponin level, patient was taken urgently to the cath lab and he was found to have totally occluded LAD with TIMI flow 0. Patient was found to have thrombosis of the mid LAD stent which extended to the proximal LAD. Patient`s ejection fraction was around 30%. He underwent successful vascularization of the LAD with balloon angioplasty and manual thrombectomy with retrieval of thrombus.

Coronary stent thrombosis is rare but it is associated with a very high risk of mortality or death [1]. It is classified as acute which occurs within 24 hours post stent placement, subacute which occurs within 1 to 30 days, late that occurs in 1 to 12 months and very late which occurs beyond one year [2]. Very late stent thrombosis is the rarest to happen as it occurs in 0.4-0.6% in patients who have sirolimus drug eluting stent [3]. The cause of stent thrombosis generally is either related to the patient, procedure or to the stent [4]. Whereas similar to our case, in very late stent thrombosis it is mainly attributed to malposition or fracture of the stent, de novo plaque rupture or abnormal re-endothelialization [5-8].

Multiple case reports were published in the past for in-stent thrombosis, where the longest to happen was 9.5 years after stent placement [9]. For our knowledge, our case is by far the longest reported interval to happen after 20 years of drug eluting stent placement. This occurrence of stent thrombosis after this long period of time will continue to question the time interval of how long patient should stay on dual antiplatelet therapy [11]. Where our patient was discharged on dual antiplatelet therapy for one year and then he continued to be on aspirin until his recent presentation.
Myxedema Coma Induced Heart Failure with Reduced Ejection Fraction: Unraveling the Complex Interplay

Introduction

Myxedema coma is a rare condition with a high mortality rate. It poses the risk of cardiovascular complications, typically affects elderly females, and is often triggered by an underlying infection or systemic illness in the setting of preexisting severe hypothyroidism. Patients may exhibit multiorgan dysfunction with altered mentation ranging from disorientation and lethargy to obtundation and coma. Other physical exam findings may include dry scaly skin, non-pitting edema, delayed reflexes, bradycardia, and hypothermia. Prompt diagnosis and early treatment are crucial to reducing mortality rates associated with this severe condition.

Case description

A 74-year-old female with no known past medical history except for smoking who had not sought any medical attention for the past 30 years was brought to the ED for increased weakness and fatigue over the past month, which worsened three days before presentation. She had altered mentation, hypothermia (84.8°F), and sinus bradycardia with 1st-degree AV block. TSH 42.44, Free T4<0.4, TPO Ab 81, thyroglobulin Ab <20, Cortisol 14.6, ACTH 20, Cr 1.7, BUN 46, BNP 648, and high-sensitivity troponin 19. Head CT showed no acute intracranial process. Furthermore, she was found to have severe sepsis secondary to bilateral lower extremity cellulitis and was started on broad-spectrum antibiotics. With evidence of myxedema coma, the patient was intubated, transferred to CCU, rewarmed, and treated with IV hydrocortisone and IV levothyroxine. TTE revealed a normal-sized LV with LVEF 15% and severe global hypokinesis.

Due to AKI, she was not a good candidate for a left heart catheterization. She underwent a myocardial SPECT stress test, which was negative for stress-induced ischemia, suggestive of non-ischemic cardiomyopathy. The patient was initiated on maximally tolerated guideline-directed medical therapy for heart failure.

Follow-up labs revealed TSH 7.68, T4 0.8, and T3< 1.5, which improved to 2 in the following day. A month later, her functional status was NYHA class III and repeated TTE revealed LVEF to 40%, mild global hypokinesis, and grade II diastolic dysfunction.

Clinical significance

Myxedema coma is rare, with a yearly incidence of 0.2-1.08/million, yet a life-threatening emergency with a high mortality rate of 30-60%, necessitating a comprehensive understanding of its signs and symptoms for timely diagnosis and appropriate treatment. Pathophysiological mechanisms involve impaired myocardial contractility, reduced cardiac output, and altered vascular resistance. The cardiac myocyte lacks significant deiodinase activity and thus depends on T3 from the bloodstream. Cross-sectional studies suggest that nearly 30% of individuals suffering from congestive heart failure (CHF) exhibit reduced T3 levels, which serves as a robust indicator for both all-cause and cardiovascular mortality. This case underscores the importance of considering myxedema coma in the differential diagnosis of obtundation and symptoms of cardiovascular compromise.

Discussion

Thyroid disease is common, affecting 9-15% of adult females; however, overt hypothyroidism impacts almost 3% of adult females. The cardiovascular presentation of myxedema coma is often serious and poses a potential threat to life. Nonetheless, the development of overt CHF is a rare phenomenon. In most instances, identifying and addressing the underlying thyroid disorder leads to reversible cardiovascular changes. Thus, early recognition is crucial to facilitate timely intervention and optimize patient outcomes.
Unveiling a No-Flow Enigma: West Nile Virus and the Intriguing Threat of Hypercoagulability

West Nile Virus (WNV) is a single-stranded RNA virus, a part of the Flaviviridae family. It is transmitted to humans by mosquito bites. Clinical presentations range from asymptomatic to severe infections such as encephalitis. A rarely reported outcome of WNV infection is hypercoagulability. We present the case of a 72-year-old male patient who presented with a complaint of altered mental status (AMS), secondary to WNV infection, and an incidentally found superior sagittal sinus thrombosis.

Our patient has a history of hypertension, type 1 diabetes, and bipolar disorder. He was found by his family to be minimally responsive and was brought to the emergency department. On presentation, his vital signs were within normal limits, and due to his AMS, he was promptly initiated on antibiotics for suspected meningitis, together with anticonvulsants for possible status epilepticus. He was admitted to the ICU for encephalopathy. Workup included a complete blood count (CBC) showing mild leukocytosis, and a complete metabolic panel (CMP), which was noncontributory. Lactic acid level was within normal limits, urine drug screen was positive only for Cannabinoids. Electroencephalogram (EEG) ruled out seizures. Initial brain imaging included a computed tomography (CT), and subsequently a magnetic resonance imaging (MRI) both demonstrating age-related changes and small prior ischemic events. His antibiotics were de-escalated, and his antiseizure therapy was discontinued.

During his hospitalization, this patient demonstrated some clinical improvement with spontaneous eye opening, but his mentation remained altered. Lumbar puncture (LP) and repeat brain imaging were ordered. On day seven of hospitalization, a brain magnetic resonance angiogram (MRA) uncovered a non-filling defect of the anterior portion of the superior sagittal sinus. This was confirmed with a repeat MRI, and computed tomography angiogram (CTA) to be an anterior superior sagittal sinus thrombosis. The cerebrospinal fluid (CSF) analysis showed 11 WBC (0% granulocyte, 92% lymphocyte), 3 RBC, glucose 87, and protein 66, pointing towards aseptic meningitis. The patient was started on a heparin drip per neurosurgery recommendations and was placed on antiviral therapy with IV acyclovir pending the finalization of CSF analysis. The meningitis panel result was negative for common viral pathogens including HSV. West Nile Virus IgM and IgG serologies were positive in both serum and CSF analysis making a diagnosis of West Nile infection very likely.

On further questioning, the family didn’t report any personal or family history of hypercoagulable disorders. Hematology was consulted recommending switching to oral anticoagulation, to pursue further hypercoagulable workup outpatient. Due to continued altered mentation, the patient required a PEG tube for feeding, but the family decided to place him on hospice care.

Previous case reports that linked West Nile to hypercoagulable states have been published. One case describes a cryptogenic stroke in a healthy 57-year-old man diagnosed with WNV infection, and another case of purpura fulminans in a 42-year-old African-American patient. The pathophysiology of hypercoagulable states with WNV infection is hypothesized to be endothelial dysfunction, but the exact link is yet to be established.
**Tylenol Tangle: A Case of 5-Oxoproline Toxicity from Unintentional Acetaminophen Overdose**

**Introduction:**

MUDPILES is a well-known mnemonic to describe the causes of anion gap metabolic acidosis, however novel forms of anion gap acidoses have been elucidated as causes not encompassed by this popular memory aid. Acetaminophen toxicity is widely known to cause hepatic injury and renal injury producing decreased glomerular filtration rate, hypokalemia, and hypophosphatemia but its deleterious effects on renal physiology are less understood. High anion gap metabolic acidosis due to metabolites from chronic acetaminophen consumption is rare and underdiagnosed, making epidemiology and prevalence relatively unknown.

**Case Description:**

A 41-year-old Caucasian female with past medical history of right hemicolectomy for perforation and sepsis, endometriosis, and Graves’ disease post thyroidectomy presented complaining of generalized myalgias, arthralgias, and fatigue. She complained of pain radiating from the pelvis to the chest with intense nausea and had been unable to eat a full meal for approximately six weeks. Home medications prior to admission consisted of levothyroxine 88 µg daily and up to 4g of acetaminophen daily. Physical exam revealed an emaciated woman with dry mucous membranes, 4/5 strength in all extremities, diffuse abdominal tenderness to palpation, hypertension, and tachycardia. Laboratory testing revealed hypokalemia, elevated creatinine, an elevated anion gap of 23 mmol/L with concurrent metabolic acidosis with a venous pH of 7.09, and normal thyroid function. Organic acid urine testing was pursued due to the otherwise unexplained anion gap metabolic acidosis, chronic acetaminophen ingestion and the clinical picture fitting an emaciated female with intense nausea and vomiting. It revealed 5-oxoproline markedly elevated at 989 mmol/mol creatinine highly suggestive of acetaminophen toxicity. Treatment was initiated with bicarbonate drip and n-acetylcysteine. Her intense pain and nausea subsided with correction of the anion gap metabolic acidosis. She was stabilized and discharged with instructions to discontinue acetaminophen.

**Discussion:**

Detoxification of acetaminophen is accomplished by glutathione and rare mutations of glutathione synthetase and 5-oxoprolinase have been described as causes of 5-oxoprolinuria. Additionally, chronic consumption of acetaminophen has been established as a rare but increasingly recognized cause of 5-oxoprolinuria high anion gap metabolic acidosis in specific patient populations including chronically ill malnourished women. This causes depletion of glutathione and reduces negative feedback on γ-glutamyl-cysteine synthetase producing increased γ-glutamyl-cysteine and thus increased levels of 5-oxoproline. Prevalence is exceedingly rare and is currently unknown and in the cases of 5-oxoprolinase and glutathione synthetase deficiencies prevenances are <1/1,000,000. Treatment is simple with administration of sodium bicarbonate acutely, cessation of acetaminophen, and vitamins E and C for free radical scavenging. Increased recognition of and testing for 5-oxoproline anion gap metabolic acidosis could be a potential life-saving measure in hospitalized patients. Testing for 5-oxoproline metabolite is not widely available. Increased knowledge of and testing for 5-oxoprolinuria could be beneficial for survival of patients experiencing high anion gap metabolic acidosis due to chronic acetaminophen ingestion.
A Rare Case of Hypomyopathic Dermatomyositis

Dermatomyositis (DM) is both a complex and rare inflammatory myopathy affecting 1 in every 100,000 individuals, most commonly in women ages 40-60. Classically, DM presents with progressive proximal muscle weakness in conjunction with characteristic dermatological findings, which are highly disabling. However, it is important to note that the timeline of these symptoms may present differently in a subset of patients. I will present a very rare case of hypomyopathic dermatomyositis in a young 26 year old female.

This is the case of a 25 year old female with unremarkable past medical history that presented to the rheumatology clinic for a positive ANA result. She mentioned that the ANA was ordered after a thyroid nodule was found by her PCP, and since then, she started having some noticeable fatigue. Additionally, she has had both weight and hair loss. On further prompting, the patient noted a skin rash on her hands, nodular in nature on a few of her metacarpal and proximal interphalangeal joints. She confirmed that she has had a red rash across her cheeks before and that her PCP believes she likely has Lupus. She has no family history of cancers however she does have an uncle with Lupus. The physical exam was positive for mechanic’s hands, erythematous scaly papules over 2 knuckles, periungual erythema and an erythematous upper back. The patient was asked to complete some rheumatological labs, which came back exhibiting an ANA of 1:1280, elevated TPO and iron deficient anemia. TSH, ESR were normal. DsDNA, ENA, Anti-smith, Anti-Jo1, alpha-1 antitrypsin, ssDNA, SSA, SSB were all negative. There were multiple differentials at the time ranging from autoimmune thyroiditis, dermatomyositis, anti-synthetase syndrome, SLE and others. After 6 months, the patient re-presented to the clinic due to worsening symptoms, including muscle weakness such as difficulty gripping objects and some intermittent numbness in the hands. EMG demonstrated electrodiagnostic evidence of myositis present proximally and distally. Right thigh biopsy was performed and sent for analysis showing myositis. Additional labs completed showed CK of 5719, Aldolase of 36.6, AST 239, ALT 129. Anti-centromere, Anti-chromatin, Anti-Ribosomal P, Anti SCL 70, and Anti RNP antibodies came back negative. Patient was started on a high dose steroid taper, and placed on immunosuppressive medications including Methotrexate and Plaquenil. Due to the association of DM with cancer, appropriate cancer screening was performed as well. Once her disease was more stabilized, the patient was referred for low grade physical therapy. She was officially diagnosed with Hypomyopathic DM, due to the late presentation of muscle symptoms, stirring away from the classical presentation of the disease.

This case demonstrates the importance of a concrete history and physical. The scattered time-line of cutaneous and muscle symptoms and the plethora of symptoms in the following case is infrequently encountered, making it a formidable diagnostic challenge. Recognizing this uncommon inflammatory myopathy is critical in initiating early appropriate therapy, due to its debilitating symptoms and cardiovascular, pulmonary and/or oncologic complications if left untreated.
From MRSA infection following chest tube removal to genetic testing: An incidental finding of a rare genetic multi-system disorder

Birt-Hogg-Dube Syndrome (BHDS) is a rarely diagnosed genetic disorder affecting three primary organ systems (Lung, Renal and Skin) that can be laborious to distinguish from other diseases. A 48-year-old female was hospitalized with a spontaneous pneumothorax (SP) after 5 days of new-onset exertional dyspnea. The SP was treated with a chest tube and cardiothoracic surgery (CTS) was consulted. Due to the presence of air leak, she needed to be hospitalized for 5 days for resolution of the pneumothorax. Chest tube was removed on hospital day 5 with a post-removal chest x-ray showing left lower lobe infiltrate atelectasis with blunting of the left pleural recess. Following the x-ray, the patient was discharged home. Six days following discharge the patient noted redness and drainage at the chest tube site and was prescribed cefalexin. Two days later, the patient had a chest X-ray which showed collection/abscess and with worsening redness and drainage at the chest tube site, the patient went to the emergency room. The patient was readmitted 11 days after previous discharge with an elevated WBC but negative infectious workup and MRSA nasal swab was negative. Chest CT showed air in fluid containing cavitory lesion, a surrounding consolidation presumed to be pneumonia and atelectasis in addition to a small left parapneumonic effusion. A trace finding of approximately 10 cystic lesions with no discernible walls are noted consistent with emphysematous changes. CTS determined that the abscess did not need to be drained. Would cultures however were positive for MRSA, so the patient was discharged home on IV vancomycin for 6 weeks.

Chest CT obtained one month after discharge showed the abscess has resolved with minimal residual scarring/subsegmental atelectasis in the lung, however it was noted nonspecific thin-walled lung cysts throughout both lung fields unchanged from prior studies. Differential diagnostic considerations at that time included BHDS and lymphangiomomatosis (LAM). The patient is a lifetime non-smoker, is adopted with no information about her biological family. VEGF-D was negative for LAM. The patient was referred to dermatology for evaluation and biopsy of skin. Screening for FLCN gene that was sent to Mayo Clinic was positive and histopathology of the skin biopsy from the forehead was consistent with fibrofolliculomas, both of which are consistent with the diagnosis of BHDS. BHDS is an exceptionally rare autosomal dominant condition that has approximately 200 families identified worldwide with this condition to date.1 The patient being an adopted child and no clear traceable family history, the initial diagnosis was uncertain. The presence of multiple cysts on CT chest imaging did prompt various differentials including LAM since the patient was a female. However, with genetic testing, imaging and biopsy, a diagnosis of BHDS was conclusively made. The patient will have lifelong surveillance for renal tumors and the patients’ children may undergo genetic testing and counseling at their discretion. With the incredible rarity of BHDS and its AD inheritance pattern, this paper provides a unique opportunity for the combination of clinicopathologic, radiologic and genetic findings leading to a diagnosis.
Introduction:

Daily oral antiretroviral therapy (ART) is the standard of care for treatment of HIV. It is composed of three drugs, taken once daily, which reliably achieves durable viral suppression when taken with strict compliance. Cabotegravir and rilpivirine have been approved as a two-drug long-acting injectable antiretroviral therapy (LAIART). The current indicated use of LAIART is for maintenance therapy in patients who have already achieved virologic suppression (HIV-1 RNA <50 copies/mL) on stable ART for at least 3 months without previous treatment failures. LAIART is not presently approved as a first-line induction ART. This new LAIART represents a convenient treatment modality in patients who prefer once monthly or bimonthly (every 2 months) injections in clinic, as opposed to daily oral therapy. There are currently no large studies demonstrating efficacy in patients with high viral loads. Recent reports are emerging suggesting they may also induce durable viral suppression. Control of HIV viral load not only improves patient mortality and quality of life, but also carries public health benefits for the prevention of the spread of HIV.

Case Presentation:

A 24-year-old male who achieved prompt viral suppression with the unindicated use of LAI-ART. The patient had congenitally acquired HIV infection, and neurocognitive deficit. While under the care of his guardian (grandmother), he was virologically controlled with Elvitegravir / Cobicistat / Emtricitabine / Tenofovir, the daily intake of which she was supervising (CD4 at 561 cells/mL, HIV undetected). When the patient turned 18, he left home, and was lost to follow up with our infectious disease clinic. His family later re-assumed guardianship at 23 years of age. He re-established care in the infectious disease clinic at their behest and was found to have developed AIDS. His CD4 cell count of 89 cell/μL, and an HIV1 viral load at 2,670,000 copies/μL. His previous enteral therapy was re-initiated. The patient initially reported compliance (VL down to 25,139 cells/μL) but within 4 months, his CD4 cell count had decreased to 35 cells/μL and his HIV1 viral load had increased to 394,946 copies/μL. Multiple attempts at garnering compliance failed. It was decided through joint decision making that initiating LAI-ART with Cabotegravir and rilpirivine as monthly IM therapy would increase the patient’s adherence to therapy and may improve viral control. The patient was himself agreeable and over the next 6 months presented to the clinic for monthly injections. Subsequently, his CD4 cell count improved to 93 cells/μL, and viral load was detectable but less than 20 copies/μL. 6 months after achieving suppression, the patient continued to have an undetectable viral load.

Discussion:

HIV infection is often co-morbid with many social barriers that prevent the effectiveness of oral therapies to induce remission. LAIART overcomes many of these issues, but there are currently no recommendations for this indication. Case reports suggest that LAIART may be a useful alternative therapy and further studies to explore the effectiveness of LAIART for inducing durable viral suppression should be pursued.
Is that a tumor? Endobronchial Actinomyces, a rare case of an anaerobic pulmonary infection

Introduction:
Bronchiectasis is a disease of dysregulated inflammation leading to bronchial dilation. The resulting autoimmune dysregulation can cause many issues with airway clearance and mucus plugging. It can present with exacerbations due to an infectious insult like exacerbations of Chronic Obstructive Pulmonary Disease. These patients frequently present with exacerbations and antibiotic treatments are often tailored to individual patients based on previous cultures symptom severity. Actinomyces is an anaerobic bacterium that is difficult to diagnose when associated with pneumonia because it is uniquely difficult to culture. Endobronchial Actinomyces is a unique disease pathophysiology that results from this actinomyces pulmonary infection which results in the growth of obstructing granular masses within the bronchi.

Case Presentation:
A 79F with a history of asthma and bronchiectasis presented to the Emergency Department with dyspnea and fevers. Her symptoms had been slowly progressing for 1 week before presentation with increasing sputum production, coughing, and wheezing. She was afebrile but was unable to saturate above 86% on pulse oximetry without supplemental oxygen. Her immediate labs were significant for a leukocytosis of 18K/mcL. Her lactic acid level was normal at 1.4 mmol/L, but her C-Reactive Protein was 231.5 mg/L. Providers were immediately concerned for community acquired pneumonia, but a chest X ray did not evidence such a process. She had met only one of the four SIRS criteria; hence she was not given a fluid bolus. She had not previously presented with bronchiectasis exacerbations, so she was empirically started on Ceftriaxone and Doxycycline and admitted for supplemental Oxygen. She would remain vitally stable but would have up trending leukocytosis to 23K/mcL concurrent with significant clinical improvement of her dyspnea. Her Antibiotic regimen was escalated to Piperacillin-Tazobactam and Vancomycin. Her sputum culture would be negative with negative PCR for viral and bacterial organisms. Despite broadened coverage, her leukocytosis would continue to increase to 30K/mcL. The patient was clinically improving despite her worsened laboratory values. The patient would additionally have a Computed Tomography (CT) of the chest that showed evidence of mucus plugging. The Pulmonology team was consulted for bronchoscopy with suction of her CT identified plug. Bronchoscopy would demonstrate the left sided distal irregular and granular mass, which could not be suctioned. It shared the appearance of a tumor and was associated with wide-spread bronchial hyperemia. A bronchial wash was taken for culture and the patient was once again changed to Levofloxacin monotherapy. Her bronchial culture would grow actinomyces which was susceptible to penicillin and the patient was discharged on Penicillin G IV infusions for a total therapy of 6 weeks.

Discussion:
Actinomyces is a rare infection for which there is no recommended empiric therapy. It is typically associated with abscesses and other surgical emergencies given its anaerobic nature. Without culture and sensitivity data to guide providers, Penicillin G at high doses for 6 weeks is the recommended therapy. Endobronchial actinomyces is a rare but recognized disease process. Providers initially thought this mass was a tumor, but the identification of actinomyces on culture guaranteed the diagnosis.
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**SARS-CoV-2 Infection and Major Adverse Cardiac and Cerebrovascular Events in HFpEF Patients: A Population-Based Analysis of Predictors and Disparities**

**BACKGROUND:** Studies have shown a short-term and long-term impact of SARS-CoV-2 infection on heart failure patients; however, large-scale data focusing on heart failure with preserved ejection fraction (HFpEF) remains limited. Therefore, to address this knowledge gap, we aimed to evaluate the impact of SARS-CoV-2 infection on an increasingly recognized but largely understudied HFpEF population and associated sex-based disparities in risk factors for observed major adverse cardiac and cerebrovascular events (MACCE) in the US population.

**METHODS:** We queried 2020’s National Inpatient Sample and identified admissions among HFpEF patients with SARS-CoV-2 infections using relevant ICD-10 codes. The primary endpoint was the MACCE, consisting of all-cause mortality, acute myocardial infarction, cardiac arrest, or acute ischemic stroke. We evaluated the prevalence and predictors of MACCE for the overall HFpEF cohort with SARS-CoV-2 infection and assessed the gender-specific risk of MACCE in male and female populations. To control for potential confounders, multivariate regression was performed. A two-tailed p<0.05 was considered statistically significant.

**RESULTS:** There were 31,960 inpatient MACCE (29.1%) among the 109,750 HFpEF + SARS-CoV-2 admissions in 2020, with 15315 males and 16645 females. Males experienced a higher rate of MACCE than females (31.1% vs. 27.5%, p<0.001). Elderly patients (>=65) had a higher adjusted risk of MACCE (OR: 1.47 [95%CI: 1.33-1.62]) than those aged 45-64, and males had a higher risk of MACCE (1.20 [1.12-1.28]) than females (p<0.001). Hispanics, Asian/Pacific Islanders, and Native Americans were significantly more likely to develop MACCE than Whites.

Furthermore, patients with prior CABG (1.15 [1.02-1.30]), cancer (1.24 1.08-1.42), or chronic kidney disease (1.15 [1.08-1.23]) (All p<0.05) had a higher adjusted risk of MACCE. Subgroup analysis revealed that hyperlipidemia (1.31 [1.18-1.44]), obesity (1.13 [1.01-1.27]), tobacco use disorder (1.37 [1.23-1.53]), prior stroke/TIA (1.30 [1.10-1.53]), prior VTE (1.47 [1.20-1.80]), alcohol abuse (1.53 [1.10-2.12]), depression (1.27 [1.09-1.48]), and valvular disease (1.50 [1.17-1.92]) (All p<0.05) were all predictors of MACCE in the male subset. Hyperlipidemia (1.24 [1.14-1.35]), tobacco use disorder (1.25 [1.13-1.39]), prior stroke/TIA (1.19 [1.04-1.37]), prior VTE (1.33 [1.13-1.57]), and depression (1.29 [1.14-1.45]) (All p<0.05) were significant predictors of MACCE in the female subset.

**CONCLUSION:** Risk factors for MACCE included male gender and the elderly age group, as well as a history of CABG, cancer, CKD, hyperlipidemia, obesity, tobacco use, stroke/TIA, prior VTE, alcohol misuse, depression, and valvular disease.

**CLINICAL IMPLICATIONS:** Patients over the age of 65 and men are at a higher risk of MACCE and should be closely monitored and aggressively treated. Additionally, physicians should be aware of and address the MACCE risk factors identified in this study.
Pheochromocytoma recurrence; is lifelong surveillance necessary?

Introduction

Pheochromocytomas are rare neuroendocrine tumors originating from the adrenal glands. They are typically identified during work-up of resistant hypertension usually associated with episodes of hyperhidrosis and headache. Untreated, pheochromocytoma can result in hypertensive crises leading to strokes, malignant hypertension, myocardial ischemia, and heart failure which could often be lethal. Surgical resection is mostly curative. Surveillance after surgery is needed to detect recurrence. Studies have shown that the mean time to recurrence following a resected sporadic pheochromocytoma is about 49.4 months with a recurrence rate of approximately 3% (1). It is possible that recurrence may present with metastatic disease. However, there are no specific recommendations regarding the duration for surveillance after resection.

Case Presentation

A 47-year-old African American female underwent right sided adrenalectomy for pheochromocytoma. During the first two years of postoperative follow up, she was asymptomatic, and her catecholamine levels were normal. Four years after initial surgery, her blood pressure was elevated during an office visit with primary care physician. Workup revealed elevated plasma metanephrines level by 30-fold and she was referred to our clinic. On evaluation, there was concern for pheochromocytoma recurrence. A computed tomography (CT) scan of the abdomen showed a mass extending into the porta hepatis region, suggestive of recurrent pheochromocytoma. A PET CT per adrenal protocol confirmed an ill-defined somatostatin receptor 2 positive soft tissue mass in the right retroperitoneum surrounding the superior pole of the right kidney, which extended anteriorly to the porta hepatitis, and postero-medially to the perihepatic space in addition to multiple nodular soft tissue densities with increased radiotracer uptake in the right paracolic gutter suggestive of metastatic deposits. To decide the next best step for care, she was referred to a tertiary care center for opinion. After a tumor board review, she was offered four options which included experimental therapy with Belzutifan vs chemotherapy with capecitabine and temozolomide vs lutetium dotatate vs no intervention. She eventually was started on peptide receptor radionuclide therapy (PRRT) using lutetium dotatate.

Conclusion

Pheochromocytoma can be a fatal disease if not identified and treated promptly. Patients can become completely free of disease if treated early and long-term surveillance is necessary to identify recurrence. The European Society of Endocrinology (ESE) recommends a follow-up duration of at least 10 years after surgery (2). Whereas the American Association of Clinical Endocrinologists and Endocrine Surgeons (AACE/AAES) and the Endocrine Society (ES) provide no definitive recommendations regarding this matter (2). We conclude that further studies should be done to identify data about follow-up duration and high-risk features of malignancy after resection of sporadic pheochromocytomas. Given the data we have now, we agree with the European guidelines that follow-up duration of at least 10 years after resection of pheochromocytoma is needed, however we feel that follow-up should be lifelong with yearly serum metanephrines level especially in high-risk patients.
Mind Games - Uncovering the Stealthy Foe of Cryptococcal Meningitis

INTRODUCTION

Cryptococcus neoformans meningitis is well known in literature as a concern for HIV/AIDS patients, but our case uniquely presents a case of a patient that is not immunocompromised. Due to this, data is lacking for incidence and burden in this population worldwide. This case will specifically demonstrate the importance of early consideration of C. neoformans to initiate management and prevent mortality especially in the immunocompetent state in a non-HIV individual.

CASE PRESENTATION

69-year-old male with untreated prostate cancer presented to the emergency room with increasing confusion and involuntary muscle movements of the upper and lower extremities. Three weeks prior to presentation, presented due to fall and difficulty walking. Initially was treated for disequilibrium disorder with meclizine after other causes ruled out. Despite this his weakness and confusion progressed, on examination the patient exhibited writhing motions of bilateral upper and lower extremities, cervical and truncal areas and involuntary tongue movements with hallucinations. Laboratory studies showed leukocytosis. MRI Brain showed no evidence of abnormal enhancement. At this time, the patient’s symptomology could not be explained, which prompted a lumbar puncture (LP) followed by treatment with dexamethasone and ceftriaxone. Unfortunately, opening pressures were not obtained. Upon CSF analysis meningitis/encephalitis CSF PCR was positive for Cryptococcus neoformans and cryptococcal antigen titer of 1:640. Blood cultures and fungus cultures positive for Cryptococcus neoformans. HIV antibody/antigen negative. As a result he was started on Amphotericin B, which improved his mentation and involuntary movements.

The treatment course included complete Amphotericin B and Flucytosine for 2 weeks followed by Fluconazole 800 mg daily for 8 weeks and then Fluconazole 200 mg daily for one year. He was discharged to inpatient rehabilitation, which he successfully completed prior to going home.

Conclusion

Cryptococcal meningitis is rare but causes significant morbidity and mortality. The clinical presentation is non-specific, that can develop over several weeks to months. This can ultimately delay diagnosis and contribute to mortality. However, there should be a high suspicion for C. neoformans in immunocompetent patients without HIV. The management of cryptococcus meningoencephalitis includes three phases – induction, consolidation and maintenance. The induction phase consists of Amphotericin B and Flucytosine for at least two weeks. Consolidation is with Fluconazole 800 mg daily for 8 weeks and then maintenance with Fluconazole 200 – 400 mg for 1 year. The patient’s history was significant for prostate cancer with metastasis to the lungs not bone and has not undergone treatment thus solid tumor with no immunosuppression. Its important to note he was exhibiting symptoms before dexamethasone initiation regarding immunosuppression. He presented with non-specific symptoms that were worsening, yet he did not have any new exposures. Altered mental status creates a broad differential diagnosis list, one of which includes meningitis. A high degree of clinical suspicion led to a lumbar puncture which ultimately saved this patient’s life. This is a reminder of the importance that although C. neoformans is usually associated with HIV, it’s important to consider in non-HIV individuals.
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The Renal Riddle: A Hypertensive Emergency Complicated by Page Kidney

Introduction:
Page Kidney, or page phenomenon, is a rare condition where external compression of the kidney due to an encapsulated subcapsular hematoma leads to renin-dependent hypertension and decreased renal perfusion. Here, we present a case of resistant hypertension complicated by page phenomenon.

Clinical description:
A 38-year-old African American man with a history of hypertension was admitted for three weeks in an outlying facility due to hypertensive emergency and acute renal failure. He underwent hemodialysis and a renal biopsy, which revealed severe arteriosclerosis, acute tubular necrosis with significant interstitial fibrosis, tubular atrophy, and glomerulosclerosis. He subsequently developed left flank pain, for which he came into the ED and was found to have blood pressure (BP) of 230/120 mmHg, creatinine of 6.9 (previously at 3), and significant proteinuria on urinalysis. He underwent a CTA of the abdomen/pelvis that revealed a large subcapsular hematoma of the left kidney. Duplex renal artery ultrasound demonstrated a peak systolic renal artery/aortic velocity ratio of 0.72, ruling out renal artery stenosis. Aldosterone to Direct Renin ratio was 0.7, ruling out hyperaldosteronism. Serologies for ANA, ANCA, and C3/C4 were negative.

Suspicion arose for page kidney as the subcapsular hematoma would cause compression of the renal parenchyma. Interventional Radiology attempted ultrasound-guided aspiration with 8 mL being aspirated with difficulty due to the nature of the hematoma. No interventions were pursued by urology and vascular surgery.

The initial BP regimen included IV labetalol and hydralazine pushes, subsequently transitioning to a regimen of Aliskiren 150 mg daily, Aldactone 50 mg daily, Minoxidil 10 mg daily, Doxazosin 8 mg daily, Labetalol 300 mg three times daily (TID), Hydralazine 100 mg TID, and Clonidine 0.2 mg TID. The patient was unresponsive to Nifedipine and Hydrochlorothiazide, which were discontinued. The regimen was developed in an additive manner. All drugs were gradually up-titrated to maximally tolerated doses and frequency with a goal BP of below 140/90.

The Direct renin level was normal at 45.9. However, this was misleading since multiple factors can affect the level, such as the patient being on labetalol/clonidine and having a low JG cell reserve, given the setting of severe tubular atrophy with fibrosis.

Discussion:
Post-renal injury hypertension is rare. In one review of referrals to a general medical clinic, only 10 out of 17,410 cases were identified [0.06%]. Page kidney is treatable and diagnosed from imaging studies. It should be considered in young patients in the setting of abdominal or iatrogenic trauma who rapidly develop resistant hypertension after the causative event.

Management entails renal decompression by evacuation of hematoma by interventional/surgical methods, which were not possible in this case due to the chronic nature of the hematoma that underwent capsulation with a fibro-collagenous shell not amenable to percutaneous aspiration.

Conclusion:
This case highlights how resistant hypertension is multifactorial and to approach its management with a multidisciplinary team. To optimize patient outcomes, we should tailor the regimen for every patient and have an aggressive approach to meet adequate BP parameters to avoid complications like Stroke, Hypertensive encephalopathy, retinopathy, and cardiomyopathy.
A Case of Relapsing Malaria from Inadequate Primaquine dosing

Malaria still remains extremely prevalent around the world, with significant morbidity and mortality. The following case looks at a patient that was treated months prior for P. Vivax with the appropriate regimen yet still had recurrence of disease.

A 39 yo M with no significant pmh presented to the hospital with ongoing fevers. He stated that around 5 days prior to his ED presentation, he started to have cyclical fevers, chills, and body aches. Of note the patient was in Papa New Guinea from summer of 2022 to December of 2022 working as a missionary pilot. He stated he contracted malaria in October of 2022 and was treated for 3 days, but had it again in December and treated again. Due to his malaria history the patient underwent a malaria rapid screen which was positive. Confirmatory results with thick and thin smears were positive for P.Vivax. The patient was started on Artemether-lumefantrine with plans for Primaquine following. The patient did eventually receive records from Papa New Guinea which confirmed being given artemether-lumefantrine for 3 days and Primaquine for 14 days in December. However, reviewing his regimen he had received a lower dose of primaquine (3.5mg/kg total over 14 days). Patient clinically improved the following day after admission and was discharged with Coartrem and high dose Primaquine (7g/kg total) over 23 days for relapsing P.Vivax. G6PD enzyme activity showed normal levels.

P.Vivax and Ovale are unique malaria species which have an additional liver stage known as the hypnozoite stage which allows for the infection to lie dormant for weeks to months and then represent later on. Primaquine is one medication that is available to target this hypnozoite stage. The World Health Organization (WHO) recommends to use a higher dose of 7 mg/kg (0.5 mg/kg per day) especially for the tropical, frequent-relapsing P. vivax prevalent in East Asia and Oceania. In most other regions, the WHO recommends lower dose primaquine (3.5 mg/kg, administered as 0.25 mg/kg/day over 14 days) considering that G6PD enzyme screening might not be available and on the basis of the historically low incidence of adverse events when used in patients without previous G6PD testing. Nonetheless, primaquine does not remain universally effective. Over the last decade, clinicians around the world have complained of more frequent failures of primaquine therapy. This is why the CDC (2015) changed its recommendation to high dose primaquine for P. vivax from all regions of the world as long as no evidence of G6PD deficiency. Chamma-Siqueira NN et al (NEJM, 2022) nicely showed that a higher dose primaquine dosing had greater efficacy than the standard dosing in preventing recurrence. Standard dosing for primaquine is 30mg (max dose daily) for 14 days in patients who are <70 kg. But as per CDC recommendations, dosing can be adjusted to 6mg/kg when above patients weight is above 70kg and thus duration extends as well.

This case highlights the importance of correct dosing of Primaquine when considering treatment of P. Vivax or P. Ovale.
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Congenital LQTS Unmasked By Postpartum Cardiac Arrest
Introduction: Women with long QT syndrome (LQTS) are four times more likely to suffer arrhythmias postpartum than age-matched controls. (1, 2) Despite this, without documentation of cardiac symptoms and a personal or family history of arrhythmia, electrocardiograms (ECG) are rarely performed during the perinatal period. (1, 2) Hence, LQTS remains underdiagnosed unless symptoms arise.

Case: We present a 23-year-old female with history of preeclampsia who was two months postpartum when she presented following an out-of-hospital sudden cardiac arrest, requiring multiple rounds of CPR, defibrillation, and subsequent intubation. Post-resuscitation ECG showed sinus bradycardia with prolonged corrected QT of 479 ms (ECG 1). She was started on amiodarone and lidocaine, and was eventually extubated, given uneventful telemetry monitoring. On day 2 of hospitalization, the patient had another episode of sustained pulseless polymorphic ventricular tachycardia requiring brief CPR, which spontaneously converted after intravenous magnesium. She was subsequently reintubated and deeply sedated to decrease sympathetic tone. Repeat ECG showed further prolongation of QTc at 645 ms (ECG 2). Cardiac MRI demonstrated preserved biventricular size and function, normal valvular anatomy, and absence of late gadolinium enhancement. A subcutaneous implantable cardioverter-defibrillator (ICD) was placed for secondary prevention. Genetic testing revealed a pathogenic variant of KCNH2 mutation consistent with hereditary LQT2.

Discussion: Congenital LQTS is a channelopathy caused by loss- or gain-of-function mutations in the myocyte ion channel proteins. The loss-of-function KCNH2 mutation associated with LQT2, diagnosed in this patient, is associated with decreased current across the rapid acting potassium channel (IKr). This change prolongs phase 3 of the cardiac action potential, resulting in lengthening of the QTc interval and increased risk of torsades de pointes and cardiac arrest. More than 17 genes have been associated with subtypes of LQTS. However, greater than 80% of cases are subtypes 1-3. (3-5)

Hormonal changes in the postpartum period and increased life stressors of caring for a newborn make women with LQTS increasingly high risk for cardiac events. Interestingly, this increased risk is seen almost exclusively in LQT2. (5, 8) Progesterone has demonstrated antiarrhythmic properties in contrast to estrogen with proarrhythmic elements. Literature suggests a rapid decrease in the progesterone postpartum is implicated in the increased risk of cardiac events. (6-8) Additionally, increased prolactin, oxytocin, estrogen, and a decreased heart rate have demonstrated QT-prolonging effects. (1) This likely explains why females with LQT2 remain at much higher risk than males even later into adulthood.

Treatment of LQTS perinatally involves beta-blocker therapy. Maternal use of these medications is associated with decreased newborn birth weight and hypoglycemia but not with increased risk of miscarriage, spontaneous abortion, or fetal malformation. (6-8)

Conclusion: The above case highlights the importance of early identification and risk mitigation of QT-related events in postpartum females. Once LQTS is identified, uninterrupted beta blocker therapy can be used, and is safe during pregnancy and postpartum.
Streptococcus pneumoniae neck abscess in a patient with Waldenstrom's Macroglobulinemia

Patients with Waldenstrom's macroglobulinemia/lymphoplasmacytic lymphoma are at increased risk of sinopulmonary infections, pyelonephritis, and septicemia. The underlying etiology is assumed to be immune dysregulation. Patient's with waldenstrom's macroglobulinemia have been shown to have somatic mutations in the "V", "D" and "J/H' segments of immunoglobulin gene. These mutations can affect affinity maturation and formation of memory B cells. Subsequently patient's with Waldenstrom's macroglobulinemia also have a suboptimal response to vaccination against Staphylococcus aureus, Streptococcus pneumoniae and Varicella, similar to those with multiple myeloma. Severe infections with gram positive organisms causing multifocal osteomyelitis, pleural effusion and sepsis have also been reported with waldenstrom's macroglobulinemia.

64-year-old female with past medical history of recently diagnosed Waldenström’s macroglobulinemia/chemical plasmacytic lymphoma presented to the emergency department with complaints of pain and swelling in the neck. Around 3 weeks prior to presentation, patient noted a swelling in the left side of the neck after she accidentally punctured the neck of her skin with her fingernail. She went to the ER but workup was negative for acute infection. Contrast-enhanced CT scan of the neck showed multiple enlarged bilateral cervical lymph nodes. Around 14 days after the the incident, patient started developing painful swellings in the neck. The pain was sharp in nature, 10/10 in intensity localized to the area of swelling. In the ER, patient was tachycardic and afebrile. Examination revealed a visible 3x3 cm swelling in the neck, with overlying redness, warmth and tenderness to palpation. Lab investigations were significant for lymphocytic leukocytosis with WBC of 31.5 (lymphocyte %; 24.8, neutrophil% 6.1). Contrast-enhanced CT scan of the neck 5x5 centimeter ring-enhancing lesion in the subclavicular space at the junction of the proximal 2/3rd and distal 1/3rd of the clavicle. Patient underwent US guided drainage of abscess with drain placement. Abscess fluid cultures were positive for Streptococcus pneumoniae. Patient had history of vaccination with PCV 13 around 18 years ago. Patient was treated with IV ceftriaxone followed by oral cephalexin for total of 14 days. She also underwent repeat vaccination with PCV, meningococcal and hemophilus influenzae vaccine after resolution of infection. Of note, patient's previous PET scan showed marked splenomegaly with increased FDG intake.

While neutropenia has been known as a cause of recurrent infection in B cell neoplasms, the cause of recurrent infection in patients with a normal neutrophil count remains elusive. The theoretical explanation of immune dysregulation due to genetic mutations have not been demonstrated in vivo. Moreover, abscess formation in a vaccinated individual highlights the decreased ability of patient's with waldenstrom's macroglobulinemia to mount an effective antibody response. Splenic involvement in patients with plasma/ B cell disorders further increases chances of infection possibly secondary to alteration in antibody opsonisation. Defective opsonisation is a known complication of multiple myeloma; as a result patients with multiple myeloma are predisposed to sepsis with streptococcal pneumoniae, klebsiellas pneumoniae and other encapsulated organisms. It is possible that a similar mechanism predisposes patient's with waldenstrom's macroglobulinemia to infections with encapsulated organisms. It is important to ensure resolution of localised infection to prevent bacteremia.
A rare case of primary sclerosing pneumocytoma in a healthy female

Introduction: Pulmonary sclerosing pneumocytoma is a rare group of benign pulmonary neoplasms that usually affects older adults over 50 years of age. This neoplasm is more commonly seen in women, with female to male ratio of 5:1 and is predominantly seen in Asian ethnicities. These tumors originate from type II alveolar pneumocytes and are hence classified as adenoma under the 2021 World Health Organization (WHO) classification of tumors. They account for 3-5 percent of total benign lung tumors. One of the striking aspects of PSP is its clinical presentation, or rather the lack thereof in many cases. It is often discovered incidentally during routine chest imaging, such as chest X-rays or CT scans, due to its asymptomatic nature. Moreover, it is essential to consider the potential for malignant transformation, as a subset of PSP cases have exhibited aggressive behavior.

Case Description: A 73-year-old female was seen in her cardiologist’s office for a routine visit. The patient had a past medical history significant for hypertension, hyperlipidemia, and coronary artery disease diagnosed on CT imaging. The patient was incidentally also found to have a lung nodule in her right middle lobe. In the office, the patient was completely asymptomatic without any chest pain, cough, shortness of breath, recent weight loss, loss of appetite, fevers, or night sweats. The physical exam was within normal limits. The patient had no history of smoking nicotine or any other drugs, worked as a chef in the food service industry, and denies any occupational metal or fume exposures. CT showed marginated, noncalcified pulmonary nodule in the right middle lobe measuring 2.6*2.4 cm. There was no evidence of hilar/mediastinal lymphadenopathy, other nodules, septal thickening, or interstitial lung disease. The patient had a chest CT 4 years prior, on which this nodule measured 1.6*1.6 cm. The patient was referred to pulmonologist and was recommended for a PET scan, which revealed a 2.8*2.9 cm well-circumscribed mildly hypermetabolic lesion in the with SUV of 2.68. CT-guided biopsy revealed pulmonary sclerosing pneumocytoma with hematoxylin and eosin staining showing classic cuboidal surface cells and round stromal cells. Immunohistochemistry was positive for AE 13, TTF-1 and EMA; the tumor was negative for Napsin and ERG. The patient was referred to see oncology and thoracic surgery. Her case was discussed at thoracic tumor board and was recommended video assisted thoracic surgery guided right middle lobectomy. The patient opted for active surveillance instead, with repeat imaging in 4 months and regular flow up with oncology, pulmonology, and surgery.

Discussion: Although benign, these tumors have high potential for proliferation, local recurrence and metastasis to lymph nodes, pleura, and bones. Pathologically four different patterns have been described, namely papillary, solid, hemorrhagic, and sclerotic. Molecular alterations that have been most commonly described include mutations in the AKT-1, beta-catenin, BRAF genes. The prognosis of PSP is excellent due to its benign nature. First-line treatment modality is usually surgical resection. Interestingly, in a retrospective in South Korea, no difference was observed in all-cause mortality between surgical management and active surveillance.
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Uterine Leiomyoma: Rare Cause Of Pulmonary Embolism
Pulmonary embolism is a life-threatening condition that most commonly occurs due to the embolization of deep venous thrombus in the lower limbs, arising from a constellation of risk factors that affect the flow, stasis, or coagulability of blood. Stasis is an important mechanism and is the primary factor in venous obstruction and long-haul flights. We present a case of a young woman who experienced pulmonary embolism due to venous compression in the pelvis from a massive leiomyoma.

A 30-year-old female presented to the emergency department with dyspnea and palpitations lasting for a duration of 2 days. She had a past medical history of recently provoked deep vein thrombosis, poorly compliant with Apixaban, uterine leiomyoma with menorrhagia, iron deficiency anemia, and Factor VII deficiency. Tachycardia was noted at presentation, along with a palpable, nontender mass in the hypogastric region of the abdomen. Due to a high Wells score, a CTA PE was performed, revealing a large saddle pulmonary embolism with an RV:LV ratio of 2. Echocardiography demonstrated an enlarged IVC with less than 50% respiratory variation. She underwent PE thrombectomy and was initiated on IV Heparin. Lower extremity venous Doppler revealed acute DVT in the left distal femoral vein. A CT venogram of the abdomen and pelvis showed an enlarged uterus with leiomyomas, the largest one measuring 13.7x10.2cm. The leiomyoma was compressing the common iliac vessels and inferior vena cava. She was not considered a candidate for venous stenting due to the risk of stent stenosis from leiomyoma compression. Moreover, she was contemplating myomectomy, which could relieve the obstruction. While an IVC filter could decrease the risk of future pulmonary embolism, especially from DVT in the lower limbs, it was not performed as the IVC was dilated which increases the risk of dislodgement. Anticoagulation was switched to Apixaban, and she was discharged from the hospital with plans to discuss myomectomy for uterine leiomyoma.

While Factor VII deficiency could potentially result in thrombosis in a small proportion of patients due to the overexpression of other clotting factors, she had associated heavy menstrual bleeding with normal levels of other clotting factors. This suggests that Factor VII deficiency is unlikely to be one of the causes of VTE in this patient.

This case illustrates the risk posed by venous obstruction in the development of deep vein thrombosis and subsequent pulmonary embolism. Timely intervention for pelvic masses can prevent potentially life-threatening pulmonary embolism.
Navigating Clinical Uncertainty: A Convergence of HIV, Positive ANA, and Genetic Cardiomyopathy

We present a case involving a 36-year-old male with a complex medical history, including hypertension, hyperlipidemia, cerebrovascular accident (CVA), and Hypertrophic Obstructive Cardiomyopathy (HOCM). The patient presented with a week-long history of chest, lower extremity, and back pain, tingling, and a history of recurrent falls. The initial presentation, combined with his history of ICD placement in 2019, led to investigations for Acute Coronary Syndrome (ACS) and ICD malfunction. The patient's initial laboratory workup, which included a comprehensive drug screen, was unremarkable except for mild normocytic anemia and elevated protein levels. CT scans of the head and spine and X-rays of the limbs were normal. The cardiac workup confirmed HOCM on echocardiography.

Elevated protein and gamma globulins were found as part of the emergency department workup. The broad differential diagnosis at this point included multiple myeloma, given the patient's history of muscle and bone pain, and chronic infections such as bacterial endocarditis, Lyme disease, HIV, and syphilis. However, his blood cultures remained consistently negative, and the gammopathy was found to be polyclonal. Ultimately, the patient was diagnosed as HIV-positive. Additional findings included evidence of prior Hepatitis B Virus (HBV) infection and a recent primary Epstein-Barr Virus (EBV) infection with no active symptoms of an infection.

Complicating the case further, during the gammopathy workup, the patient was found to have an Erythrocyte Sedimentation Rate (ESR) of 111 mm/hr (with normal CRP), positive ANA with titers 1:640, positive anti-dsDNA, false-positive syphilis, and positive antiphospholipid syndrome antibodies. These findings suggested a potential diagnosis of Systemic Lupus Erythematosus (SLE). However, after further questioning and examination by our hospital's rheumatologists, the patient did not meet the criteria for SLE diagnosis as he lacked any clinical signs of the disease, such as arthritis, nephritis, or serositis. Close follow-up was recommended to monitor for development of disease. The patient's management was complicated by his inability to provide accurate identification or contact information for relatives, thus hindering comprehensive healthcare provision. Unfortunately, our infectious disease specialists did not initiate antiretroviral treatment due to fears of poor outpatient follow-up.

In this complex case, the patient's reluctance to disclose information resulted in significant challenges in managing and initiating antiretroviral therapy for HIV-1. His unique combination of conditions required a holistic, multidisciplinary approach, reinforcing the importance of a wide-ranging differential diagnosis in patients with diverse symptoms. This case underscores the need for innovative approaches in managing patients with complicated, overlapping diagnoses, especially when these are further complicated by issues related to poor follow-up or compliance.
Pleural Epithelioid Hemangioendothelioma: An Ultra Rare Etiology for Recurrent Pleural Effusion

Introduction

Epithelioid hemangioendothelioma (EHE) is an extremely rare sarcoma of vascular origin. EHE has an incidence of less than 0.1 per 100,000 patients per year. It can involve multiple organs; pleural involvement is relatively rare and under-reported in the medical literature. We present a unique case of pleural EHE presenting with recurrent pleural effusion, severe pleuritis, scarring, and trapped lung syndrome.

Case presentation

A 54-year-old Caucasian female was referred to the pulmonary clinic for two months of progressive dyspnea, fatigue, and right pleuritic chest pain. Initial chest x-ray showed a large right pleural effusion. Thoracentesis identified exudative lymphocyte predominant pleural fluid with normal pH, high protein, high lactate dehydrogenase, normal glucose, negative infectious workup, and fluid cytology negative for malignancy. Chest computed tomography scan showed pleural calcifications, nodular thickening of the pleura, and a 1.8 cm right paraesophageal lymph node. She underwent esophagogastroduodenoscopy (EGD) with endoscopic ultrasound-guided lymph node biopsy that was unrevealing. In two months, the patient underwent five thoracenteses with negative cytology. To address the recurrent pleural effusions, she underwent video-assisted thoracoscopic surgery, which showed chronically inflamed right parietal pleura with pleural thickening and fibrosis (Figure). A thick fibrous scar partially entrapped the right lower lobe. Pleural biopsies revealed calmodulin binding transcription activator 1 (CAMTA-1) positive epithelioid cells with prominent intracytoplasmic vacuoles resembling signet ring cells, confirming the diagnosis of EHE.

Discussion

The pleura is an infrequent primary site of EHE, with roughly 10% of cases having primary pleural involvement. Its delayed presentation, difficulty establishing a tissue diagnosis, and aggressive nature make pleural EHE associated with the poorest prognosis compared to EHE involving other sites. In addition to recurrent pleural effusions, it can present with pleural thickening, hemoptysis if lung involvement exists, or lung entrapment from severe pleural inflammation and scarring, as illustrated in our patient. Surgical resection of pleural EHE is often not feasible due to metastasis at presentation and tumor friability. Symptomatic management with pleurodesis versus indwelling pleural catheter therapy is often warranted to prevent recurrent pleural effusion. There is a lack of consensus on system therapy for metastatic disease. However, agents like bevacizumab, anthracyclines, or ifosfamide are commonly used. Molecular medicine targeting the CAMTA-1 downstream signaling pathway is currently under investigation.
Non-Immune Hemolytic Anemia in the Setting of Alcohol-Associated Hepatitis: Zieve’s Syndrome

Introduction:
Zieve’s syndrome describes a triad of non-immune hemolytic anemia, jaundice, and transient hyperlipidemia found in patients with alcohol-use disorder/alcohol-associated liver disease. This case involves a 39-year-old female with suspected Zieve’s syndrome in the setting of severe alcohol-associated hepatitis.

Case Description:
A 39-year-old female with alcohol-use disorder presented to the emergency department with complaints of jaundice, lethargy, and confusion. On initial presentation, patient was anemic with a hemoglobin of 7.8 g/dL (12.1-15.0 g/dL), ALP of 235 U/L (33-120 U/L), AST of 116 U/L (<35 U/L), ALT of 41 (<37 U/L) total bilirubin of 24.2 mg/dL (<1.2 mg/dL), direct bilirubin of >15 mg/dL (<0.4 mg/dL). Her blood alcohol level was negative. She was admitted due to concerns for alcohol-associated hepatitis. Her hospital course was complicated by frequent declines in hemoglobin requiring a total of six transfusions of packed red blood cells. No overt bleeding such as hematemesis or hematochezia was ever evident during these declines. Further hematologic workup revealed a normal glucose 6 phosphate dehydrogenase (G6PD), normal pyruvate kinase, elevated plasma hemoglobin of 150 mg/dL (<30 mg/dL), decreased haptoglobin of <8 mg/dL (40-250 mg/dL), and elevated lactate dehydrogenase (LDH) of 595 (100-240 U/L), elevated reticulocyte count of 213 bil/L (21-100 bil/L). Direct Coombs Test was negative. The combination of these labs illustrated a picture of non-immune hemolytic anemia. Patient’s course was further complicated by a persistent rise in her total bilirubin, eventually peaking at 30.6 mg/dL, and ascites which required paracentesis and fluid studies was consistent with spontaneous bacterial peritonitis (SBP). Given the presence of SBP, corticosteroid therapy was not initiated for alcohol-associated hepatitis. Lipid panel remained within normal limits on two separate occasions. Vitamin E level was <2.5 mg/L (5.5 – 17.0 mg/L). During hospitalization, she tested positive for Influenza A H1N1, developed acute hypoxic respiratory failure, was intubated, and was transferred to the intensive care unit and later extubated. Her mental status during her hospital stay was frequently waxing and waning and never fully recovered. The patient was eventually discharged home with hospice and her hemoglobin levels remained stable after supportive blood transfusions during her stay.

Discussion:
Zieve’s syndrome is an underdiagnosed cause of anemia in patients with alcohol-associated liver disease. There are multiple proposed mechanisms to explain the pathophysiology behind the red cell lysis seen in this disorder. One leading hypothesis suggests the red cell membrane becomes unstable due to vitamin E deficiency leading to disordered pyruvate kinase. Importance of early recognition of this syndrome is essential as treatment revolves around strict abstinence from alcohol.
Introduction: Cushing’s disease, arising from a pituitary macroadenoma, is marked by excessive cortisol production. Labs usually signify overproduction of adrenocorticotropic hormone (ACTH), leading to elevated cortisol levels. While cortisol is essential for various physiological functions, its excess can result in various symptoms and complications associated with Cushing’s disease. Notably, individuals with this condition may experience an association with compression fractures, particularly in the vertebrae, due to weakened bones. Prolonged exposure to elevated cortisol levels contributes to the development of osteoporosis, characterized by weakened bone density, making fractures more likely. These compression fractures can manifest as localized or radiating back pain, a gradual loss of height over time, and kyphosis.

Case: 43 year old female with pertinent past medical history of HFpEF, HTN, OSA, T2DM on insulin and morbid obesity who presented to the emergency room with complaints of intractable low back pain. CT of the abdomen and pelvis demonstrated age- indeterminate mild compression fractures of T8, T9, as well as T11 through L3, favored to be remote. She worked with physical therapy and was only able to sit up with her legs off the side of the bed, unable to walk. Patient was found to have hypercortisolemia on lab work. Her cortisol and ACTH was 29.1 and 313 respectively. MRI pituitary was completed and revealed a pituitary macroadenoma measuring greatest diameter of 3.3 cm. Patient was without visual symptoms but symptomatic given elevated ACTH, cortisol, glucose, multiple compression fractures, and overall clinical picture consistent with Cushing disease. The patient denied any facial plethora changes, denies history of anxiety/depression, prolonged steroid therapy, or use of megestrol acetate. The patient denied tobacco use, admits to social alcohol use stated as 2-3 glasses of wine on 3 out of 7 days weekly and denies illicit drug use. The patient denied any personal or family history of adrenal or autoimmune disease.

Discussion: Patient was found to have Cushings disease with a 3.3 cm pituitary macroadenoma. Given this information, patient needed a complete pituitary hormonal profile to evaluate for hypopituitarism, including free T4, free T3, prolactin, IGF-1. She was found to have secondary hypothyroidism secondary to pituitary macroadenoma and was started on 75 mcg of levothyroxine for replacement.

Neurosurgery was consulted for resection of pituitary mass and patient underwent a trans- sphenoidal tumor resection.

Conclusion: In summary, Cushing’s disease which is often triggered by a pituitary macroadenoma can present in various ways. It is important to consider it in younger females with compression fractures. This patient was also obese and in this population there is a low risk of osteoporosis. Thus, in this population, when there is a presence of fractures it is important to consider other causes such as cushings disease. Ultimately, a comprehensive approach is essential to address both endocrine and skeletal abnormalities in these clinical scenarios.
Hydralazine Associated Neutrophilic Dermatosis: A Unique Case Observing DILE, ANCA Vasculitis, and Sweet Syndrome

Introduction: This case report highlights a 73-year-old male with biopsy-proven neutrophilic dermatosis with concern for Sweet Syndrome in the context of suspected hydralazine-induced systemic lupus erythematosus and autoimmune markers that initially pointed towards hydralazine-induced ANCA vasculitis.

Case Presentation: The patient is a 73-year-old male with a past medical history significant for chronic kidney disease, urothelial cancer in 2021, renal cancer in 2013, and stage 1A left lung cancer. The patient additionally underwent workup for low white count and splenomegaly in months leading up to hospitalization, with bone marrow biopsy and workup negative for myelodysplastic syndrome or malignancy. Patient initially presented with acute COVID pneumonitis and received treatment with steroids and IV remdesivir. Admission was complicated by acute onset pancytopenia. Presentation also significant for a diffuse purpuric rash on his extremities, which was present for eight months since starting hydralazine and worsened with recent COVID infection. Laboratory evaluation revealed an elevated ANA titer, anti-histone antibodies, positive ANCA, myeloperoxidase antibodies, and low C3/C4. This raised concern for drug-induced lupus erythematosus and hydralazine-induced ANCA vasculitis. Hydralazine was discontinued and the patient was placed on darbepoetin, with improvement in pancytopenia. The patient was subsequently discharged.

The patient was readmitted several days later with worsening pancytopenia, acute kidney failure, respiratory distress, and hemorrhagic bullae in addition to worsening purpuric maculopapular rash over extremities, face, and chest/back. Labs revealed elevated PT/aPTT, urinalysis with hematuria and proteinuria, elevated CRP, and normal ESR. No infection was found during the workup. Dermatology was consulted and numerous skin biopsies, which showed neutrophilic dermatosis characterized by marked dermal interstitial and perivascular neutrophilic infiltrate with karyorrhexis, basophilic debris, and edema with neutrophilic inflammation extending beyond perivascular areas. New CT imaging showing continued splenomegaly with new onset ascites found to be transudative in nature.

Management and Outcome: The patient was treated with pulse dose steroids and transitioned to oral steroids as well as Cellcept with improvement in pancytopenia and rash/skin lesions without the formation of new lesions. Continued investigation for etiologies, including malignancy workup, is ongoing.

Discussion: Neutrophilic dermatosis is commonly associated with Sweet Syndrome, a rare condition that features cutaneous eruption of erythematous & violaceous tender papules and plaques with occasional hemorrhagic bullous formation. The patient presented with similar findings, however, this case is unique as the patient did not exhibit the classic findings of fevers or leukocytosis before rash onset. This condition is associated with malignancies, infections, drugs, and autoimmune diseases. Several cases have documented hydralazine as a cause for Sweet Syndrome. Noncirrhotic portal HTN is a component of underlying hydralazine-induced DILE.

Conclusion: The case demonstrates a diagnostic challenge and raises questions regarding the association of DILE and neutrophilic dermatosis in this patient. We aim to contribute to the body of knowledge surrounding neutrophilic dermatosis and its potential associations. Through this case report, we seek to enhance clinicians’ awareness of the diagnostic challenge posed by neutrophilic dermatosis within the context of drug-induced lupus, providing insights into its clinical course and implications for patient care.
Euglycemic DKA: commonly missed diagnosis

Introduction
Sodium-glucose cotransporter-2 inhibitors, the latest anti-diabetic medications with recognized cardiovascular and renal benefits, have seen increased usage. However, a slight but noteworthy rise in the risk of diabetic ketoacidosis (DKA) is associated with their use. Notably, a considerable subset of DKA cases related to SGLT2 inhibitors manifests with initially normal or mildly elevated serum glucose, posing a diagnostic challenge. Within this spectrum, euglycemic diabetic ketoacidosis stands out, characterized by euglycemia (<200 mg/dl), heightened plasma ketones, and severe anion gap metabolic acidosis. Representing a rare clinical entity, euglycemic DKA accounts for approximately 2.6% to 3.2% of total DKA admissions.

Case
A 70-year-old female with a medical history of hyperlipidemia and type 2 diabetes mellitus presented to the hospital with dizziness that commenced a day before admission upon waking. Orthostatic vitals were normal, and neurological examinations, including the Dix-Halpike maneuver, Romberg sign, and cerebellar signs, showed no abnormalities. Relevant laboratory results indicated a hemoglobin level of 13.2 g/dL, blood glucose of 118 mg/dL, serum bicarbonate of 17, and an anion gap of 15. Imaging studies, including a CT head and MRI brain, excluded acute stroke, and a CT angiogram of the neck with IV contrast revealed no aneurysm or evidence of branch occlusion. The EKG demonstrated a normal sinus rhythm. Administration of a 500 mL normal saline bolus resulted in gradual symptom improvement. Initially attributed to dehydration, the persistent anion-gap metabolic acidosis prompted further investigation.

The patient was prescribed metformin 1000 mg twice daily and dapagliflozin 10 mg once daily for diabetes management. However, she took dapagliflozin intermittently due to concerns about potential weight loss. Subsequent inquiry revealed she took dapagliflozin a day before experiencing dizziness. Laboratory findings included an HbA1c of 7.8% and an elevated serum beta-hydroxybutyrate of 5.6 mmol/L, with a normal lactic acid level. Dapagliflozin was discontinued, presuming its role in euglycemic DKA and dehydration. The endocrinology service recommended the continuation of metformin at a dose of 1000 mg twice daily while discontinuing dapagliflozin. Follow-up in the endocrinology clinic after 2 months was advised.

Discussion:
Sodium-glucose cotransporter-2 inhibitors, novel anti-diabetic medications, pose an elevated risk of DKA. The diagnostic complexity arises from the fact that many patients with euglycemic DKA exhibit normal or minimally increased serum glucose levels, challenging early identification. This case underscores the critical role of heightened clinical suspicion in diagnosing euglycemic DKA, emphasizing its atypical presentation and infrequent occurrence. Clinicians managing patients on SGLT2 inhibitors should maintain vigilance for euglycemic DKA, particularly when confronted with nonspecific symptoms and unexplained metabolic acidosis.
Adenovirus and Herpes Simplex Virus 1 co-infection leading to acute respiratory distress syndrome (ARDS) in a latent mycobacterium tuberculosis, immunocompetent, patient

Introduction:
Adenovirus and Herpes Simplex Virus 1 (HSV-1) typically cause self-limiting upper and lower respiratory tract infections. However, in immunocompromised individuals, severe complications such as pneumonia can arise, leading to acute respiratory distress syndrome (ARDS). Management typically requires prompt intubation and mechanical ventilation for survival. This case report details a rare occurrence of community-acquired adenovirus and HSV-1 pneumonia in an immunocompetent adult, resulting in ARDS.

Clinical Vignette:
A 40-year-old Hispanic female, without a known medical history, presents to the hospital with left-sided chest pain, shortness of breath, and hemoptysis lasting one day. The pain, rated 10/10, began on the left chest side and radiated to the center back, exacerbated by deep breaths and upper body movement. Over-the-counter ibuprofen provided no relief, prompting hospital evaluation. In the ED, vital signs revealed a temperature of 98.2°F, heart rate of 116, respiratory rate of 27, blood pressure of 135/91, and SpO2 of 95% on 6L nasal cannula.

Initial labs were remarkable for sodium 133, potassium 3.4, chloride 99, BUN 14, creatinine 0.66, lactate 1.4, troponin 6, WBC 13.7, hemoglobin 12.7, hematocrit 38.4, and platelet 280. Urinalysis was unremarkable. COVID test was negative. CT Angiogram Chest revealed severe multifocal pneumonia but no pulmonary embolus. Before transferring to medicine services, the patient received ceftriaxone, azithromycin, and vancomycin. Overnight, the patient’s condition worsened, necessitating Vapotherm oxygen therapy and eventually BiPAP. Imaging and blood gases confirmed moderate ARDS, with ABG showing pH 7.46, pCO2 36.4, PO2 49, and HCO3 25.7.

ICU was consulted for continuous BiPAP use. She continued to worsen and was intubated overnight. She was receiving lung protective ventilation and paralytics and was put in a prone position. Labwork in the ICU indicated a positive Quantiferon antibody, suggestive of latent TB. A bronchoscopy with BAL on Day 4 revealed adenovirus antibody, HSV-1 antibody, and aspergillus antigen-antibody. Negative results came from Acid Fast bacillus stain, fungal Beta-D-Glucan, and Aspergillus Galactomannan. Rheumatological workup was remarkable for mildly positive ANA and SS-A antibodies. Double-stranded DNA Antibody, C3 antibody, C4 Antibody, P-ANCA, and C-ANCA antibody were all negative. She received high doses of IV Sol-u-medrol and was tapered down to prednisone.

The patient responded well and was successfully extubated on the 10th day. She was transferred back to the general medicine floor.

Discussion/Conclusion:
This case underscores the rare progression of community-acquired adenovirus pneumonia to ARDS in immunocompetent adults. It emphasizes the complexity and severity of viral co-infections and latent tuberculosis in complex clinical presentations. Timely diagnostics and interdisciplinary management, involving antiviral, antimicrobial, and immunosuppressive agents, contributed to the successful outcome. Clinicians should remain vigilant for atypical presentations and collaborate across specialties for optimal patient care.
A post-transplantation puzzle: a case of recurrent fevers and lymphadenopathy

Lymphadenopathy, often first noted on a physical exam, can create a diagnostic dilemma when an obvious cause is not apparent from the medical history. Imaging studies such as ultrasound are frequently performed for further lymph node characterization, or to distinguish between local and generalized involvement. Diagnosis can ultimately require a biopsy to reveal specific pathologic features or staining patterns. Lymphadenopathy often coexists with non-specific symptoms such as fevers, myalgias, and fatigue. The differential diagnosis can usually be grouped into infectious, cancerous, lymphoproliferative, and immunologic causes. In the setting of immunosuppression, as in post-transplant recipients, a high index of suspicion must be kept for rare or unusual disease processes, especially if the initial work-up is non-revealing.

A 32-year-old male with a history of ESRD secondary to FSGS, s/p kidney transplant 10 years prior (maintained on tacrolimus and mycophenolate), with recent transplant rejection requiring reinitiation of dialysis, presented to the hospital due to worsening fatigue, fevers, shortness of breath, and episode of syncope. He had recently been discharged after an extended hospitalization for similar presenting symptoms. Routine lab work-up had shown a significantly elevated white blood cell count (neutrophil-predominant), anemia (thought to be mainly from renal disease), and thrombocytopenia, but were otherwise unrevealing. Patient’s vitals were significant for labile hypotensive blood pressure and recurrent fevers to the point of rigor, poorly responsive to antipyretics. Patient’s exam was notable for a generalized pruritic maculopapular rash that was not responsive to topical steroids. Chest imaging revealed diffuse patchy airway disease thought to initially represent multifocal pneumonia, though with multiple enlarged thoracic lymph nodes. Abdominal imaging revealed hepatosplenomegaly. He was empirically started on broad spectrum IV antibiotics, without improvement. An extended infectious diagnostic panel was performed, which returned negative for bacterial, viral, and fungal pathogens. Bronchoalveolar lavage was likewise unrevealing. A bone marrow biopsy was negative for malignancy. In the absence of an identifiable infectious cause, and history of prolonged immunosuppression, the presumptive diagnosis was thought to be post-transplant lymphoproliferative disease. A lymph node biopsy was performed to confirm this diagnosis, which instead revealed a plasmablastic pattern of histologic features consistent with HHV-8 associated multicentric Castleman disease. Staining for latency-associated nuclear antigen (LANA-1) returned positive, confirming HHV-8 infection. Skin biopsy of the lesions was consistent with a neutrophilic dermatosis, not suggestive of Kaposi sarcoma (an angioproliferative disorder also mediated by HHV-8).

Castleman disease represents a rare disease of lymph nodes on the spectrum of lymphoproliferative disease and cancer. Each of the three subtypes have their own clinical features, treatments, and outcomes. Treatment depends on factors such as presence of multi-organ dysfunction, performance status, or coexistence of Kaposi sarcoma. HIV represents an important risk factor that should be tested and concomitantly treated. Transfer to a tertiary center was ultimately deemed to be in the patient’s best interests, to begin rituximab-based or IL-6 inhibitor therapy. As illustrated in this case, rare diseases may require extensive testing and pose unique challenges. A structured diagnostic framework and the consultation of various specialists may be essential in obtaining the correct diagnosis.
Presentation of Neuroleptic Malignant Syndrome in the setting of a Levodopa, Carbidopa, and Entacapone Triple Pump

Neuroleptic malignant syndrome is a life threatening syndrome characterized by muscle rigidity, autonomic dysfunction, hyperthermia, and altered mental consciousness. It is caused by medications that affect the dopaminergic pathways resulting in a reduction in central dopaminergic activity due to D2 dopamine receptor blockade in the nigrostriatal, hypothalamic, and mesolimbic/cortical pathways. Typically this is caused by antipsychotics, antiemetics and withdrawal from Parkinson’s disease medications. But what about withdrawal from an levodopa, carbidopa, entacapone triple pump?

This is a case of a 76-year-old man with a 12-year history of Parkinson’s disease who presented to the hospital with diffuse muscle rigidity, altered mental status, temperature of 38.2 degrees celsius, new onset hypertension, tachycardia, and urinary retention of 1.2L. Per his daughter, the patient was found on the couch, diffusely stiff, soiled in his urine, and disoriented to family members. At baseline, he has mild gait instability, right upper extremity rigidity with a tremor. On presentation, he was combative with healthcare personnel. The patient’s medication regimen includes carbidopa-levodopa, and ropinirole. Upon further history taking, we learned that he was participating in a trial involving the triple pump, although his pump was halted 4 weeks ago for unclear reasons. While on the pump, his Parksonian symptoms were well controlled. Per family, once the patient was off the pump, he experienced worsening tremors, right upper extremity rigidity and multiple falls secondary to worsening gait instability. In the emergency room, he was given 2 mg of Ativan and approximately one hour later, his symptoms improved.

This patient transitioned from his triple pump to oral carbidopa-levodopa 4 weeks prior to presentation. His presentation with tachycardia, hyperthermia, diffuse rigidity, altered mental status and hypertension, were consistent with neuroleptic malignant syndrome (NMS). This leads us to reinforce the importance of thorough history taking and understanding of pathophysiology as it connects to pharmacology. In this case, we found that the patient had adequate control of his Parksonian symptoms while he was on triple pump therapy and his symptoms returned in a worsened state than his baseline, weeks following discontinuation. The timing of withdrawal from the pump to his presentation of NMS is approximately 4 weeks. Our clinical suspicion for NMS was reassured after marked improvement of presenting symptoms after benzodiazepine administration, which is known as a second line therapeutic for NMS. We know that carbidopa and entacapone inhibit peripheral breakdown of L-dopa by inhibiting DOPA decarboxylase and catechol-O-methyltransferase, respectively. Our suspicion is that receiving this type of therapy through a continuous pump may be a risk factor for NMS and withdrawal once discontinued. Despite transitioning to oral therapy, our patient experienced symptoms of NMS. This makes us question that oral therapy may not have adequate equivalent dosing compared to the pump, resulting in decrease levels of dopamine in the nigrostriatal, hypothalamic, and mesolimbic/cortical pathway resulting in NMS 4 weeks later. Ultimately, this patient was started on bromocriptine and he was almost back to his baseline the following morning, further confirming our diagnosis of NMS.
Losing Sight: A Case of Multifocal Glioblastoma Multiforme

Glioblastoma Multiforme (GBM) is the most aggressive and lethal primary brain tumor. Unfortunately, this deadly malignancy is relatively common, with an incidence rate of 5 people diagnosed per 100,000 in the United States. GBM is associated with significant morbidity and mortality, with a median survival of only 15 months. A rare subset of GBM is multifocal GBM, which manifests as multiple distinct lesions that develop simultaneously. Multifocal GBM has significant radiological findings that are similar to brain metastases, which often lead to misdiagnosis in the initial stages of a patient’s care. Multifocal GBM is broadly not understood due to its rarity, as it is only seen in approximately 30% of cases of GBM.

A 58-year-old Caucasian man with a non-significant past medical history presented to the hospital due to acute confusion and left-sided vision loss that started that morning. Earlier in the morning, he was driving to work and made multiple uncharacteristic errors, such as pulling into the wrong driveway and leaving the lights on. Also, he reports feeling frustrated and disoriented, which has never happened before. On admission, CT Head with contrast was significant for multiple mixed and cystic solid masses in the right cerebral hemisphere, with the largest mass measuring up to 2.8cm in diameter. He was started on high-dose steroids and closely monitored with frequent neurological exams to monitor for worsening symptoms. During his hospitalization, an MRI head confirmed the findings that were seen on his CT head, which was concerning for metastatic disease with an unknown primary. He had an EGD, colonoscopy, and CT chest/abdomen/pelvis, which all came back negative. Despite minor improvements in his vision, neurosurgery felt resection of the largest brain mass was needed for symptom relief and diagnosis. He had a right occipital stereotactic craniotomy with resection of the right occipital brain mass. Biopsies of the mass obtained during the resection were positive for IDH wildtype tumor that is consistent with high-grade Multifocal Glioblastoma Multiforme. While little is understood about this rare subset of GBM, there is a consensus that the standard of care for treatment consists of maximum safe resection followed by adjuvant concomitant radio-chemotherapy and maintenance chemotherapy. Despite recent advancements in the field of oncology, the prognosis for multifocal GBM is extremely poor, with many patients' prognoses being less than 15 months.
Bupropion induced stuttering

Stuttering is a multifactorial speech disorder defined by frequent prolongations, repetitions, or blocks of spoken sounds, excessive physical tension during speech production and/or syllables. It affects about 1% of the adult population. Other coexisting symptoms may include facial grimacing, tremors of muscles involved in speech, and eye blinks as well as avoidance of words or situations which exacerbate stuttering episodes. It is classified as a disorder when it is severe enough to disturb the fluency of speech. It is developmental but can be acquired in adulthood secondary to neurologic events such as a head injury, tumor, stroke, or certain medications.

This case discusses bupropion as one of the drugs that has been rarely reported to cause stuttering. It is a norepinephrine and dopamine reuptake inhibitor commonly used for depression and smoking cessation. It is favored for its better side effect profile, such as libido and weight gain compared to other antidepressants.

A 41-year-old man with a history of anxiety, depression and ADHD who presented to the ED with 2 days of acute onset stuttering and bilateral hand tremor. The tremor was with intention and worse on the right than the left. On the day of admission, he noticed tightness of the muscles of his neck. He denied any previous history of either symptom. He takes fluoxetine for depression and about 3 weeks ago bupropion was added due to suicidal ideation. It is worth pointing out that the patient takes lisdexamfetamine as needed a couple times a week to help improve his attentiveness at work.

It has been hypothesized that stuttering is likely due to abnormal abundance of cerebral dopamine activity. Dopamine acts as an inhibitor of striatal metabolism and leads to striatal hypometabolism. Some studies revealed striatal hypometabolism seen on PET imaging in patients who stutter, which can be explained by the presence of the hyperdopaminergic state. The FDA has not yet approved of a treatment for stuttering, but pharmacological trials on medications that lower dopamine activity have shown improvement of stuttering.

Despite the favorable side effect profile of bupropion, it is important for both medical providers and patients to be informed of the possibility of developing stuttering after starting this medication or in some cases increasing its dose. This will help in maintaining the patient’s trust in their healthcare providers in case of developing this terrifying speech impairment. It is believed that bupropion ability to increase dopamine in the prefrontal cortex is the main cause of stuttering.

In this case, it is unclear whether the combination of all of his medications contributed to his presentations, mainly bupropion and lisdexamfetamine. The commonality between those two medications is the increase of dopamine in the extra-neuronal space. This will obviously lead to a problematic outcome given our understanding of the role of dopamine in the pathogenesis of stuttering. Although only few cases have been reported on bupropion and its link for causing stuttering, it is important to be vigilant when prescribing it.
Unusual case of Salmonella Enteritidis causing septic shock, aspiration pneumonia and acute renal failure in an immunocompetent patient

Nontyphoidal Salmonella is the leading cause of bacterial diarrhea worldwide with outbreaks being relatively common. Bacteremia is a serious, but uncommon manifestation of salmonella infection with 1% of infections progressing to the bloodstream. Immunosuppression, HIV, malignancy, diabetes are the most common risk factors for developing bacteremia. Salmonella bacteremia has a high predilection for seeding the heart and great vessels with endarteritis of the aorta and endocarditis approaching 25%, and a lower likelihood (5%) for causing UTI, pneumonia, osteomyelitis, or meningitis. This is the case of a 66-year-old female who presented to the emergency department with 5 days of intractable vomiting and non-bloody diarrhea. She was found to have acute renal failure, small bowel obstruction, and salmonella gastroenteritis. The patient was started on ciprofloxacin, NG tube was placed and dialysis was planned. After 2 days of antibiotics and bowel decompression without relief, the patient was noted to acutely desaturate and become altered after lying flat for additional bowel imaging. The patient required intubation with stool-like vomit being visualized in the airway. Post intubation imaging showed multifocal opacities bilaterally. Blood cultures at this time and subsequent respiratory cultures produced nontyphoidal salmonella. The patient required pressor support and antibiotic coverage was initially broadened to meropenem then de-escalated to ceftriaxone with culture susceptibilities. A follow up transthoracic echocardiogram showed no evidence of cardiac vegetations. The patient required 12 days of ventilator and pressor support, and a total of 14 days of ICU care. The patient had complete resolution of her salmonellosis bacteremia, pneumonia, and gastroenteritis and was able to be discharged home. Nontyphoidal salmonellosis is the leading cause of foodborne illness and mortality due to foodborne pathogens in the US. Transmission can also occur through contact with certain animals such as turtles, iguanas and exposure to pet food and treats. However, common sources of infection and were we think our patient was infected is from unpasteurized dairy products, poultry or eggs. Even though this is typically a mild infection some are at risk for severe disease or have risk factors for complications. Therefore, it is recommended to consider antibiotic therapy in patients that are over 50 years of age, patients with prosthetic valves, malignancy, severe atherosclerosis or immunocompromised. Due to increasing rates of resistance amongst nontyphoidal Salmonella for patients with known bacteremia 2 antibiotics can be started until susceptibilities are obtained. In our case we increased to meropenem and then de-escalated coverage after susceptibilities were available. Our patient was negative for HIV and her immunoglobulins were within normal limits. She did have known history of diabetes and very poor functional status which we think contributed to the severity of her infection.
Kratom Induced Cardiac Arrest

Introduction:

Mitragyna speciosa also known as kratom is a Southeast Asian medicinal plant within the coffee family which contains the indole alkaloid Mitragynine. Mitragynine is noted to have mild opioid activity with mu, deltaoid and kappa receptor agonism. Traditionally, used as an herb for its psychoactive properties, there are also claims of multiple non-FDA approved medical benefits such as a pain reliever, treatment of diarrhea, fever and diabetes. Kratom has recently become commonly available and is used as a recreational drug and a cheaper alternative to opioids. The FDA has issued multiple warnings against the use of kratom noting the lack of approved medical use and similar risk of abuse, addiction and death as other opioids. Kratom has been associated with serious side effects including seizures, liver damage, and withdrawal symptoms.

Case Presentation:

This is the case of a 47-year-old female who presented to the emergency department after being found pulseless at home by EMS personnel. Return of spontaneous circulation was performed after 15 minutes of CPR. In the ED the patient was noted to myoclonic jerking and 1 episode of witnessed seizure. After loading with Levetiracetam, midazolam and propofol to abort the seizure, the patient had EEG read as electrocerebral silence with occasional myoclonus. The patient was placed on Arctic Sun for cooling to 34C. Gas chromatography coupled mass spectrometry revealed mitragynine, oxycodone metabolites and hydrocodone metabolites. After rewarming, the patient was found to have absent brainstem reflexes when examined by an intensivist and neurologist on two separate occasions, and unchanged EEG findings. A diagnosis of brain death was made, and the shared decision was made for organ donation.

Discussion:

This case represents a severe complication of kratom abuse highlighting the need for closer regulation on the drugs use and safety. Current regulations surrounding kratom use vary widely between states with multiple having outright bans, some with kratom protection laws permitting use and distribution, and others with regulation on who the drug can be marketed towards and sold to. There have been multiple reports of kratom laced with other opioids increasing the risk of adverse events with usage. Our patient presented with both oxycodone and hydrocodone metabolites in addition to the mitragynine suggesting a laced dose may have contributed to her cardiac arrest.
Drug-Induced Liver Injury Secondary to Immunotherapy with Pembrolizumab: A Rarely Fatal Complication

Introduction: Immune checkpoint inhibitors (ICI’s) are a novel class of chemotherapy medications that include cytotoxic T-lymphocyte-associated protein 4 (CTLA-4) inhibitors and programmed cell death protein 1 (PD-1) inhibitors. Commonly used for immunotherapy in a variety of advanced stage malignancies, CTLA-4 inhibitors are more associated with drug-induced liver injury (DILI). Here, we present a rare occurrence of severe DILI secondary to a commonly used PD-1 inhibitor, pembrolizumab, ultimately leading to a fatal outcome.

The Case: A 62-year-old male with a history of relapsed Hodgkin’s lymphoma on salvage chemotherapy was transferred to our facility for acute liver injury noted by transaminitis, hyperbilirubinemia, and coagulopathy. His last dose of pembrolizumab was roughly one month prior to presentation and acute causes of liver failure such as autoimmune, viral, or infection had returned negative. Given the risk of drug-induced liver toxicity, he was started on steroids promptly. Due to worsening of liver enzymes, hyperbilirubinemia, and progression to grade IV hepatotoxicity, mycophenolate mofetil was added with steroid dose reduced due to possible steroid-induced cholestasis. Liver ultrasound showed cirrhotic morphology suspected from steatohepatitis and subsequent MRCP showed acute vs chronic cholecystitis, cirrhotic liver, and non-dilated biliary tree. He ultimately underwent liver biopsy which showed portal and lobular inflammation consisting of mostly lymphocytes with some plasma cells and few neutrophils and macrophages along with hepatocyte injury with occasional acidophil bodies and hepatocellular cholestasis, overall consistent with moderate hepatitis supporting a diagnosis of checkpoint inhibitor treatment induced liver injury. Ultimately, despite appropriate treatment, his liver failure progressed to multi-organ involvement and he was then terminally weaned in the ICU per family’s wishes.

Discussion and Conclusions:

Cases of DILI secondary to PD-1 inhibitor use are a rare occurrence and fatal outcomes occur in less than 1% of scenarios. Oftentimes, cessation of ICI use or appropriate therapy with steroids is sufficient for resolution of symptoms, however as presented above, patients can often decompensate quickly despite therapy. Some theories regarding DILI secondary to ICI use include predisposition with a history of steatohepatitis as well as an autoimmune process against hepatocytes due to inhibition of T-cell negative regulators. With rising use of ICI’s due to their utility as an oncological chemotherapy, clinicians must remain vigilant to diagnose potential serious adverse effects and complications early and initiate treatment.

While most studies find no association between liver disease and DILI secondary to ICIs, one reports an increased incidence with a previous history of steatohepatitis such as with our patient. Mechanisms of DILI secondary to ICIs have been proposed, including a possible autoimmune reaction against hepatocytes due to ICI’s inhibition of T-cell’s negative regulators. Histopathology of liver biopsy has also been shown to be useful, with findings such as lymphocytic infiltration and hepatic necrosis aiding in clinical diagnosis. Although ICIs can have some serious and potentially fatal complications such as in this case their utility as an oncologic intervention cannot be understated, however it is important for clinicians to remain aware of this complication’s presentation, diagnosis, and management to improve patient outcomes.
Inferior Vena Cava Syndrome with Dyspnea and Edema in a Patient with Metastatic Esophageal Adenocarcinoma

Introduction:

Inferior Vena Cava Syndrome (IVCS) is a rare condition often resulting from thrombosis or external compression. This syndrome can manifest as dyspnea on exertion and hypoxemia, and its diagnosis is challenging, typically considered only after excluding more common causes.

Case Description:

A 74-year-old male with poorly differentiated esophageal adenocarcinoma metastasized to thoracic and abdominal lymphadenopathy presented with dyspnea on exertion that persisted for over a month. He experienced no respiratory distress when resting or any chest pain. He reported worsening lower extremity swelling extending up to the scrotum. Chest X-ray revealed mild pulmonary vascular congestion. High-sensitivity troponin levels were unremarkable at 8 and 10, while BNP was significantly elevated at 1010. Physical examination noted an S4 heart sound and pitting edema in the lower extremities extending to the scrotal region. CT PE (Pulmonary Embolism) did not show any pulmonary embolism but showed lymphadenopathy, mild pulmonary vascular congestion, and small bilateral pleural effusions. Transthoracic echocardiography (TTE) showed a left ventricular ejection fraction (LVEF) of 55-60%, with normal right ventricular and diastolic function. The patient showed initial improvement with Lasix but developed severe orthostatic hypotension, causing discontinuation of diuretics. During the hospital stay, he reported back pain and given concerns for metastasis, a CT of the lumbar spine was performed, revealing multiple large necrotic lymph nodal masses in the pelvis and retroperitoneum, invading the right iliacus muscle, and severely compressing the vena cava at the aortic bifurcation. Oncology was consulted for potential adjustments to his cancer treatment. He was discharged with compression stockings and was qualified for home oxygen therapy at 2L during exertion, with plans for follow-up regarding his cancer progression.

Discussion:

Inferior Vena Cava Syndrome (IVCS) is an under-recognized cause of edema and dyspnea in cancer patients. In this case, lymph node enlargement led to vena cava compression at the aortic bifurcation, reducing venous return to the right atrium and causing downstream back pressure, evidenced by lower extremity and scrotal swelling. This reduced venous return and elevated venous pressure resulted in tissue fluid accumulation, and contributed to his heart strain and respiratory symptoms. The elevated BNP level in this patient could reflect cardiac strain secondary to increased venous pressure, despite the echocardiogram being negative, which there have been cases where a normal echocardiogram can miss diastolic dysfunction. The case shows the importance of considering IVCS in the differential diagnosis of dyspnea and edema in cancer patients, particularly when cardiac and pulmonary etiologies are excluded as in this case. It also highlights the complexity between cancer progression, treatment side effects, and secondary complications such as IVCS. The management of such cases requires balancing oncologic treatment with symptom management and supportive care. In this case, the treatment would be to reduce the size of the lymphadenopathy, reducing the obstruction.
Disseminated Mycobacterium Abscessus with skin nodules and and septic arthritis following systemic immunosuppression in a patient with suspected primary immunodeficiency misdiagnosed with atypical sarcoidosis

Introduction:

Nontuberculous mycobacteria (NTM) are environmental pathogens found in water and soil which seldom cause disease in normal hosts but can present with a variety of clinical syndromes in immunocompromised patients. Mycobacterium abscesses is a rapidly growing NTM associated with a variety of infections ranging from pulmonary disease to cutaneous infection and dissemination in susceptible hosts. Here we present a patient who had been profoundly immunosuppressed by treatment for sarcoidosis not fitting typical diagnostic criteria and subsequently developed M.abscesses septicemia with dissemination to skin and joints.

Case Discussion:

A 21-year-old gentleman presented with fevers, painful skin nodules, and 35 pound weight loss of 12 months duration. He had been diagnosed with sarcoidosis based on retroperitoneal lymph node biopsy showing lymphoplasmacytic necrotizing inflammation suggestive of atypical granuloma which was never sent for culture. He worsened on several courses of steroids then 2 doses of infliximab. His father had died at age 26 due to pneumonia and had also been diagnosed with sarcoidosis but had never received a biopsy. Exam was significant for fever, tachycardia, micrognathia, atypical facies, and cachexia. There was diffuse palpable lymphadenopathy, multiple purpuric nodular lesions of the hands/fingers, hepatospelenomegaly, and a right knee effusion. Labs showed pancytopenia and blood cultures had M. abscesses in both standard and lysis-centrifugation bottles. Bone marrow biopsy was hypocellular (10-15%) with depressed hematopoiesis without granulomas or lymphohistiocytosis. HIV screening and an extensive rheumatology work up was nonrevealing. Knee arthrocentesis had 6200 nucleated cells (80% lymphocytes) with acid-fast bacilli on staining. He declined skin biopsy. Due to reticuloendothelial involvement, abnormal facies and family history of early death due to infection, infectious diseases strongly suspected that disseminated NTM infection due to an inherited immunodeficiency and infliximab. Further immunosuppression was held and he was treated with a four drug regimen of amikacin, azithromycin, tigecycline, and imipenem. By day 14 of treatment fevers and skin nodules resolved. By day 22 cell counts had improved and he was discharged with plans for 6-12 months of therapy and follow up with infectious diseases.

Discussion: The prior diagnosis of sarcoid in this case had been made without typical findings of hilar lymphadenopathy or granuloma, and worsening after steroids had prompted more profound immunosuppression rather than a re-thinking of the underlying diagnosis. In this case it is unclear whether the immunosuppressive medication or a concomitant genetic primary immunodeficiency had predisposed the patient to disseminated NTM as the Mendelian Susceptibility to Mycobacterial Disease Panel was pending at this time of submission. Disseminated NTM infection is uncommon in persons who are not infected with HIV but is seen in patients on immunomodulatory medications used to treat oncologic and auto-immune conditions. Disseminated M abscessus infections have at least one of the following characteristics: involvement of >1 organ, of >2 groups of lymph nodes, or positive blood culture, which were all met in our patient. Certain M. abscesses subspecies contain an inducible "erm" gene which causes macrolide resistance, therefore macrolide combined with parenteral medications (amikacin, cefoxitin, or imipenem) for serious infections is recommended.
Aortic Valve Vegetation Due to Nonbacterial Thrombotic Endocarditis in a Patient with Antiphospholipid Antibody Syndrome

Introduction
Nonbacterial thrombotic endocarditis (NBTE) is a rare condition characterized by the development of sterile thrombotic vegetation(s) on cardiac valves that is most commonly associated with advanced malignancy and chronic inflammatory diseases including systemic lupus erythematosus (SLE) and antiphospholipid antibody syndrome (APLAS). NBTE is most often found postmortem with rates in autopsy series approximately 1.2 percent. We presented a case of nonbacterial thrombotic endocarditis with aortic valve vegetation in a patient with antiphospholipid antibody syndrome who developed an acute cerebrovascular event after an asymptomatic period.

Case Report
A 47-year-old man with a history of triple positive antiphospholipid syndrome (APLAS) was transferred to the hospital due to abnormal findings on transthoracic echocardiography (TTE). On exam, an early diastolic murmur in the left sternal border suggestive of aortic regurgitation was noticed. No abnormal lesions were noticed on the fingers or hands. Lab investigations revealed normal hemogram and chemistries. He was positive for lupus anticoagulant (LAC). His levels of anti-β2-glycoprotein (anti-β2GP) antibody and anticardiolipin (aCL) antibodies were abnormally elevated. Transesophageal echocardiography (TEE) showed a 0.61 cm x 1.2 cm highly mobile mass on the ventricular surface of the left coronary cusp with aortic insufficiency. The patient has been on warfarin therapy for the treatment of antiphospholipid antibody syndrome (APLAS) which was switched to rivaroxaban due to dietary restrictions and difficulty with therapy monitoring. He was then switched from rivaroxaban to Dabigatran due to the failure of therapeutic anticoagulation with rivaroxaban. Repeat TTE in two months demonstrated resolution of aortic valve mass with mild aortic regurgitation. Four months later, the patient was admitted to the hospital with an ischemic stroke. TTE was performed and did not show any vegetation, ventricular thrombi, or masses. After five days of bridging with heparin, the patient was transitioned to warfarin and discharged home with close therapy monitoring.

Discussion
Aortic valve vegetation is a clinically intriguing and infrequently encountered manifestation of NBTE. NBTE is a rare condition that refers to a spectrum of noninfectious lesions of the heart valves in the absence of bloodstream bacterial infection, ranging from microscopic platelet aggregates to large vegetations, and is often diagnosed postmortem. However, the condition carries a high clinical significance due to its association with a high prevalence of valvular heart disease, leading to an increased risk of thromboembolic events, particularly cerebrovascular events. This case is unusual in that it was diagnosed in an asymptomatic patient while on anticoagulation therapy, as most cases are discovered incidentally postmortem and are not well described in the literature. Furthermore, most of the previous cases of NBTE with aortic valve vegetations have been reported in patients with underlying malignancy, however, data regarding nonbacterial thrombotic aortic valve endocarditis in patients with primary APLAS is limited in the literature. The proper management of APLAS and prevention of thromboembolic events remains extremely challenging. Patients should be closely followed up for potential complications of the disease. Further research and documentation of diverse cases will refine our insights, ultimately enhancing the management strategies for patients with NBTE.
The Rising Prevalence of Aortic Disease in Women with SLE: A Growing Concern and its Consequences

Introduction:
Despite a significant decline in the all-cause mortality rates in systemic lupus erythematosus (SLE) over the past twenty years, the risk of death due to cardiovascular disease remains a leading cause of mortality in SLE. A rare but deadly complication of SLE is the development of an aortic dissection. We report a case of a young woman presenting with an acute aortic dissection.

Case Description:
A 25-year-old female with SLE diagnosed at 17, uncontrolled hypertension, frequent SLE flares, and ESRD due to nephritis presented with chest discomfort and dyspnea. An echocardiogram from two weeks prior to presentation showed a mildly dilated ascending aorta, a stable mild-to-moderate pericardial effusion, and left ventricular hypertrophy. CTA abdomen and pelvis revealed a Stanford type A aortic dissection extending past the inferior mesenteric artery and terminating above the aortic bifurcation. Her ascending aorta and hemiarch were repaired using the frozen elephant trunk procedure. Post-op, she developed recurring loculated pericardial effusions with associated cardiac tamponade and multiple cardiac arrests with successful ROSC, eventually resulting in anoxic brain injury.

Discussion:
Aortic aneurysms and dissection in SLE patients stem from various pathological changes, encompassing atherosclerosis, vasculitis, and cystic medial necrosis. In our case, the patient exhibited a Type A dissection that extended downward to the aortic bifurcation. The data available suggests that vasculitis-induced pathology might have played a role in thoracic aortitis, leading to an acute dissection. This dissection likely progressed to the abdominal aorta due to underlying chronic atherosclerotic changes. Notably, premature atherosclerosis in SLE correlates with the duration of the disease, its activity and damage, corticosteroid use, hypertension, and chronic kidney disease. Atherosclerotic vascular events including aortic aneurysms and dissections are uncommon in early-stage SLE but become more prevalent in later stages. Given the rarity of vascular complications in SLE, routine screening may not be cost-effective. However, optimizing modifiable risk factors can potentially facilitate the earlier diagnosis of cardiovascular complications, thereby improving overall outcomes in SLE patients.
A Rare Case of Left-Ventricular Non-Compaction Cardiomyopathy

Left-ventricular non-compaction cardiomyopathy (LVNCCM) is a rare and not very well understood cardiac condition. It is theorized to be congenital and has been categorized as an unclassified cardiomyopathy. Often, patients present with non-specific cardiac symptoms but unique features are noted on echocardiography. An array of life-threatening complications may follow and will be discussed. A case of LVNCCM in an elderly male with no past medical history who presented with acute onset HFrEF is presented to bring attention to the lack of literature and guidelines on diagnosis and management of this disease.

A 68-year-old male who denies any medical history or current medication use presents with a two-week history of shortness of breath associated with chest pain, dry cough, orthopnea, and PND which is elicited by exertion and relieved by rest. Electrocardiogram is significant for right axis deviation and T-wave inversions in the inferolateral leads with no indication of acute ischemia. Chest radiograph revealed pulmonary congestion and transthoracic echocardiography showed severely decreased left ventricular systolic function with ejection fraction of 15-20% and global hypokinesis with minor regionality. Significantly, prominent left ventricular trabeculations suggestive of LVNCCM were also seen. The patient was taken for coronary angiography which revealed only mild non-obstructive coronary artery disease. The patient was treated with guideline directed medical therapy for acute HFrEF secondary to a new diagnosis of LVNCCM. Cardiac magnetic resonance imaging is pending.

This case sheds light on a rare cause of heart failure, occurring in 3-4% of patients with the diagnosis (6). LVNCCM was thought to be a congenital cause of cardiomyopathy due to a lack of compaction of the myocardium during intrauterine development. However, the disease may be improperly named as embryologic cardiac imaging suggests that the trabecular layer may rather be abnormally thickened and the process of compaction is minimal (4). Nonetheless, dyspnea is the most common complaint amongst other non-specific cardiac symptoms like chest pain or palpitations. There is no gold standard of diagnosis but the most widely accepted and specific method is by meeting four of four Jenni criteria on 2-D transthoracic echocardiography. Criteria include a >2:1 ratio of epicardial to endocardial layers, a compacted LV wall thickness ≤8.1 mm, trabeculations in the LV and possibly RV, and doppler flow in these recesses (3). It is important to note that LVNCCM must be differentiated from other conditions that lead to cardiac remodeling and trabeculations; for example, in athletes, pregnant, or hypertensive patients with left ventricular hypertrophy or aortic stenosis (2). With increased availability and visualization, cardiac MRI should also be considered to aid in diagnosis (1). Complications of LVNCCM include arrhythmias, ventricular failure, thromboembolism, and sudden cardiac death. With a reduced EF, GDMT is often implemented in treatment plan. Holter monitoring, genetic testing, ICD placement, and anticoagulation are recommended to patients on an individual basis (5). There are no guidelines in diagnosis, management, or treatment of this underreported and often incidentally discovered disease. More evidence must be gathered to provide evidence-based recommendations to patients with LVNCCM to prevent its deleterious complications.
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Successful Resolution of MSSA Bacteremia Complicated by Infective Endocarditis in a Patient with Malignancy: The Efficacy of Combined IV Nafcillin and Ceftaroline Therapy

Infective endocarditis is a dangerous sequela of bacteremia that has a high rate of mortality without prompt and effective treatment. Surgical intervention to remove heart valve vegetations is indicated for bacteremia that persists for 5-7 days, left heart infections with certain species including Staph aureus, and embolic events despite antibiotic therapy. However, many patients have comorbid conditions that increase the risk of surgery to unacceptable levels. Combination therapy with nafcillin and ceftaroline has been shown to be successful in treating persistent MSSA bacteremia, but there are no reports of its use in cancer patients. Here we present a case of MSSA bacteremia and infective endocarditis with indications for surgery that resolved following nafcillin and ceftaroline combination therapy.

A 49-year-old female patient with history of developmental delays on active treatment for stage IA triple-negative breast cancer with docetaxel and cyclophosphamide presented with symptoms of fever, cough, and red, watery eyes. After drawing blood cultures, empiric antibiotics with cefepime and vancomycin were started for suspected bacterial infection. Initial blood cultures grew methicillin-sensitive Staphylococcus aureus, and antibiotic therapy was narrowed to cefazolin. Port-a-cath was considered to be the most likely source of infection and was removed. On further examination, the patient had small conjunctival hemorrhages in both eyes which increased concern for septic embolism from infective endocarditis. Transesophageal echocardiogram confirmed the presence of a 0.5x0.5cm vegetation on the mitral valve. MRI of the brain showed multiple tiny acute ischemic infarcts concerning septic emboli. Antibiotics were switched to nafcillin for better CNS penetrance. Despite effective antibiotic therapy, blood cultures collected over the first 7 days persisted in growing MSSA. Cardiothoracic surgery evaluated the patient for excision and replacement of infected mitral valve. Intervention was indicated, however the patient’s mother and decision-maker was concerned about the high risk of complications from an invasive procedure due to the patient’s poor performance status. After discussion with family and infectious disease, ceftaroline was added in combination with nafcillin. After initiating combination therapy, repeat blood cultures remained negative throughout admission. After repeat blood cultures had remained negative for 72 hours, Ceftaroline was discontinued after a total of 7 days of combination treatment. Repeat echocardiogram did not demonstrate any remaining vegetation. She was discharged to a skilled nursing facility with PICC line in place to continue nafcillin for a total of 52 days. She did not have any recurrence of this infection following discharge.

This case illustrates the novel combination therapy with nafcillin and ceftaroline in patients with MSSA infective endocarditis who may be poor surgical candidates for valve vegetation removal. There are limited reports in literature on the successful outcomes of this novel therapy. Notably, this case illustrates its successful employment in a patient with malignancy. As such, we aim to characterize the trajectory of recovery with this combination and increase its consideration as a standard of treatment of persistent MSSA bacteremia with endocarditis, particularly in patients who may not tolerate surgical intervention.
Rare Case of Atraumatic Loculated Pericardial Effusion Leading to Cardiac Tamponade

Pericardial effusion is the accumulation of fluid in the pericardial sac surrounding the heart. While pericardial effusions can be incidental asymptotic findings, they may progress to cardiac tamponade in a subset of cases (7-10%), causing hemodynamic compromise that can be fatal. Early stratification and appropriate management are essential. In this case, we present an even rarer occurrence—an atraumatic loculated pericardial effusion (LPE) leading to cardiac tamponade.

A 58-year-old male with chronic hypoxic respiratory failure on mechanical ventilation, cerebrovascular accident, peripheral vascular disease, presented from the nursing home to the intensive care unit with acute-on-chronic hypoxic respiratory failure. Upon presentation, vitals were normotensive with a normal heart rate. Initial white blood cell count showed a leukocytosis at 11.5 K/CUMM. A CT thorax revealed multifocal pneumonia. Respiratory culture grew Pseudomonas and ESBL Klebsiella pneumonia. Urinalysis was unremarkable, and blood cultures were negative. Despite initiating culture-susceptible antibiotics, the clinical status progressively worsened, leading to hemodynamic instability requiring three vaspressors, continuous renal replacement therapy, and increased mechanical ventilator support. The patient became hypothermic and bradycardic, with worsening leukocytosis of 33 K/CUMM, lactic acidosis of 13 gm/dL, and troponinemia of 394 ng/L. A transthoracic echocardiogram (TTE) revealed a moderate to large loculated pericardial effusion (LPE) along the right ventricle-free wall, with fibrinous exudate suggestive of chronic pericardial effusion. Given concern for tamponade, cardiology performed pericardiocentesis, removing 200 mL of bloody fluid. Blood pressure initially improved following the procedure; however, he later hemodynamically unstable. Repeat bedside TTE showed worsening LPE with complete collapse of the right ventricle suggestive of tamponade. Pericardial fluid analysis was negative for mycobacterium, anaerobic, and fungal growth. ANA serology and ANCA panel were within normal range. As a poor surgical candidate, the patient’s family declined a pericardial window or repeat pericardiocentesis and elected for hospice care.

LPE comprises roughly 15% of all effusions and rarely progresses to cardiac tamponade. It occurs more frequently in patients with previous instrumentation or trauma due to the formation of adhesions bridging the parietal and visceral pericardium. However, this was not the case in our patient, who was found to have an atraumatic LPE. Etiologies include infection, malignancy, and rheumatologic disorders. In our patient, once the loculations were discovered, a complete workup was not possible due to the transition to hospice care. According to the literature review, even with a complete diagnostic evaluation, it can be difficult to identify an inciting event. To our best knowledge, there is only one other case of cardiac tamponade caused by non-instrumental hemorrhagic pericardial loculations. The pathogenesis of cardiac tamponade from loculated effusions results from localized chamber compression, with even a small volume compromising cardiac output. Once tamponade is diagnosed, urgent pericardiocentesis should be pursued; however, it can be challenging to perform given septations. Effusions with rapid fluid re-accumulation may require pericardial drain and/or surgical pericardial drainage with a pericardial window. This case highlights the rarity and the necessity of timely pericardial procedures for accurate diagnosis and optimal management, which should be promptly considered given the risk for cardiac tamponade.
“CADASIL Unveiled: A Rare Genetic Disorder Mimicking Migraine Symptoms”

Introduction:

Cerebral Autosomal Dominant Arteriopathy with Subcortical Infarcts and Leukoencephalopathy (CADASIL) is an inherited neurological disorder characterised by symptoms such as migraines, transient ischemic attacks (TIAs), lacunar infarcts, vascular dementia, and psychiatric conditions like depression. The disease also encompasses cognitive impairment, apathy, and premature onset small vessel ischemic disease. We present the clinical ark of the patient leading to the diagnosis and its progression.

Case-

Our patient was a 33-year-old male with a history of obsessive-compulsive disorder and acute stress disorder. Patient first presented with a severe headache with aura and right upper extremity weakness, which later went on to be diagnosed as migraine. Recurrent migraine attacks prompted him to take Excedrin for relief; it continued until a motor vehicle accident in 2019, where he underwent imaging which showed multiple foci of abnormal T2/flair signal in the brain’s white matter. Demyelinating disease was in differentials. He was prescribed Aspirin and Plavix for secondary stroke prevention however, he discontinued it, citing it was responsible for the increased frequency of headaches. He later presented with RUE weakness and was found to have scattered lacunar and subcortical ischemic strokes consistent with CADASIL. MRI showed a left lacunar stroke (centrum semiovale). MR Angio showed no carotid or vertebral stenosis. Further, the workup led to a positive NOTCH3 gene mutation, diagnosing him with CADASIL. Patient was given genetic counselling and was discharged with outpatient neurology follow-up.

Discussion -

(CADASIL) is an inherited neurological disorder characterised by symptoms such as migraines, transient ischemic attacks (TIAs), lacunar infarcts, vascular dementia, and psychiatric conditions like depression. Migraine is the earliest feature of the disease, reported in more than 55% of the Caucasian population. More than 80% of them had aura(3) with a mean age of onset at 30 and/or TIA/stroke with onset after 40. (4), and 10% also had encephalopathy with a mean age of onset at 42. 20-40% with underlying diseases have psychiatric symptoms (5), which can significantly affect the quality of life of the patients. The clinical phenotype of the disease is variable with no known reasons. Brain MRI with bilateral anterior temporal pole T2-hyperintensities highly suggests CADASIL, but GOM (granular osmiophilic material ) is the neuropathological hallmark. Vascular risk factors, including smoking and hypertension, should be controlled. Most patients are treated symptomatically, but definitive treatment needs further research. The median age of death was significantly lower in men (61.7-67.6) as compared to women (67.6-73.9) (6). Our case aims to contribute to the need for early diagnosis of individuals with CADASIL, shedding light on the myriad of symptoms as described. Headaches are highly nonspecific, so it is essential to broaden the differentials, obtain detailed family history, and employ MRI sooner rather than later to prevent delayed diagnosis. Additionally, it emphasizes the significance of genetic workup and counseling, as it could play a crucial role in slowing and preventing comorbidities earlier in families at high risk.
Heparin-Triggered Inferior Epigastric Artery Bleeding Leading to a Remarkable Rectus Sheath Hematoma

Rectus sheath hematoma represents a rare clinical entity, exhibiting an incidence rate of 1.5-2%. The etiology of this hematoma remains largely elusive, contributing to its frequent misdiagnosis or underdiagnosis among physicians due to its infrequency. Trauma to the superior epigastric artery is promptly tamponaded by the rectus sheath. Conversely, injury to the inferior epigastric artery poses challenges in control, attributed to the absence of a posterior sheath.

The presented case involved a 73-year-old female with a myriad of comorbidities, including arthritis, hypertension, hyperlipidemia, diabetes mellitus, asthma, and sickle cell trait. Following admission for encephalopathy with concurrent COVID-19 infection, the patient's management included dexamethasone, supplemental oxygen, and subcutaneous heparin for DVT prophylaxis. Notably, she developed abdominal pain during her hospital stay, and a CT Abdo/pelvis revealed a 6.8 cm rectus abdominis hematoma, suspected to be related to subcutaneous heparin injections. Despite the absence of acute surgical indications as noted by the general surgery team, the patient's clinical course took a critical turn with worsening encephalopathy, leukocytosis, elevated creatinine, and hematologic abnormalities with a drop in hemoglobin from 11-8. A septic workup was initiated, and empirical broad-spectrum antibiotics were administered. Subsequently, the patient deteriorated into shock, necessitating transfer to the MICU for further management. CTA Abdo/pelvis revealed an interval increase in the rectus sheath hematoma size to 12.4 cm, accompanied by contrast blushing indicative of active bleeding from the inferior epigastric artery. Additionally, hematoma and diffuse subcutaneous soft tissue suggested an extension from the rectus sheath hematoma. Interventional radiology was promptly consulted for emergent embolization, resulting in hemodynamic stabilization and subsequent improvement in hemoglobin levels.

The case underscores the importance of recognizing and promptly managing rectus sheath hematoma, particularly in patients with complex medical histories. The atypical course of this patient with potential sepsis and active bleeding, leading to shock underscores the multifaceted nature of this condition necessitating a multidisciplinary approach to optimal patient care.
A Rare Case of 1,25-(OH)2 Vitamin D-mediated Hypercalcemia in the setting of recently diagnosed HIV

Introduction: Hypercalcemia in patients with human immunodeficiency virus (HIV) has been previously reported, however 1,25-(OH)2 vitamin D-mediated hypercalcemia is rarely described with HIV-related infections or malignancies. We describe a case of 1,25-(OH)2 vitamin D-mediated hypercalcemia in the setting of recently diagnosed HIV.

Case Description: A 35 year old male with a past medical history significant for HIV (diagnosed 5 months prior to presentation), disseminated mycobacterium avium complex (MAC) and cytomegalovirus (CMV) retinitis presented to the hospital with chief complaint of generalized weakness, productive cough and fevers for one week. On presentation, he was afebrile, saturating at 98% on room air. On physical examination he had generalized weakness and diffuse bilateral rales on lung auscultation.

Lab work was significant for chronic pancytopenia and elevated albumin corrected calcium at 13.5 mg/dl with ionized calcium of 1.56 mM. Alkaline phosphatase was also elevated at 1647 U/L, total protein elevated at 9.2 g/dl. CD4 cell count was 57 cells/uL, and HIV viral load <30 million copies/mL. Remaining serum chemistry panel was unremarkable.

Chest x-ray was done demonstrating multifocal pneumonia. Given his history of HIV there were concerns for tuberculosis- three acid-fast bacilli sputum cultures were obtained, all negative. Acid fast bacillus culture for MAC was positive. Patient was already being treated for existing MAC pneumonia and his home antibiotic regimen was restarted: rifabutin, moxifloxacin, ethambutol, and azithromycin. His home HIV regimen was also resumed: emtricitabine/tenofovir and dolutegravir, in addition to dapsone for pneumocystis jirovecii prophylaxis.

Regarding his hypercalcemia, parathyroid hormone was undetectable. Serum PTH-related protein was <2 pmol/L. Serum 25-OH Vitamin D was low, 17.5 Ng/mL. Serum 1-25-(OH)2 Vitamin D was elevated, 87.1 pg/mL. Abdominal computed tomography demonstrated diffuse lymph node enlargement and hepatosplenomegaly. Initial differential for hypercalcemia included granulomatous disease, infectious etiology, monoclonal gammopathy of unknown significance, and malignancy. Infectious disease, nephrology, and hematology were consulted. Bone scan was done given elevated alkaline phosphatase, however was negative for lytic lesions. Bone marrow biopsy was deferred to outpatient setting given active MAC infection.

Patient was started on normal saline at 150 cc/hr and received one dose of pamidronate 30 mg IV. His serum calcium had reduced to 8.5 by time of discharge and alkaline phosphatase levels trended down.

Given the elevated 1-25-(OH)2 Vitamin D levels, the consensus was that his hypercalcemia is likely due to granulomatous disease versus lymphoma. He is now scheduled for lymph node biopsy for further assessment.

Discussion: 1,25-(OH)2 vitamin D-mediated hypercalcemia is uncommon in patients with newly diagnosed HIV. In this patient, it was likely due to granulomatous disease versus an underlying lymphoma. This case emphasizes the importance of evaluation of hypercalcemia in HIV patients given their high risk for developing rapid dynamic changes in mineral hemostasis. Management of moderate hypercalcemia includes aggressive fluid resuscitation in addition to bisphosphonate. Bone marrow and lymph node biopsy are beneficial tools in determining the etiology of 1,25-(OH)2 vitamin D-mediated hypercalcemia.
Introduction: Over the last decade, the introduction of targeted molecular therapy has become widely used in the treatment of specific cancers. Ibrutinib (brand name Imbruvica) is one such medication that works by inhibiting B cell proliferation by binding the Bruton's tyrosine kinase protein and preventing downstream signaling of this pathway. Although rare, withdrawal side effects have been reported in a small subset of patients using targeted molecular therapies and here we present a case of a patient with chronic lymphocytic leukemia who developed abnormal symptoms post medication cessation.

Case Description: Patient is a 59-year-old female with known medical history of chronic lymphocytic leukemia, diagnosed in 2020 following cervical lymph node biopsy, for which she has been treated with Ibrutinib 420mg daily. She presented to our emergency department with the acute onset of a severe headache, with associated fatigue, fever, and chills. To note, three days prior to presentation, patient had abruptly discontinued treatment with Ibrutinib at the recommendation of her hematologist as she had been experiencing mild hemoptysis and vaginal bleeding. All infectious work-up, including a chest x-ray and sputum studies were unrevealing. Her initial labs resulted well within normal limits. Given her recent bleeding, further evaluation with abdominal, pelvic, and trans-vaginal ultrasounds were unremarkable for any abscesses or other foci of infection. Over the course of her hospitalization, patient continued to clinically deteriorate and ultimately developed persistent fevers with a T max of 39.5 and worsening neck stiffness her headache. Further evaluation with inflammatory markers was also negative. After lengthy discussion with the patient, the decision was made to re-introduce Ibrutinib. She experienced marked improvement in her headache and fevers the following day.

Discussion: Targeted molecular therapies are becoming widely used in the treatment of certain malignancies as they are designed to act on specific receptor pathways all while minimizing generalized systemic side effects. Ibrutinib, which is used in B cell-specific blood cancers, is one such treatment that is becoming more commonly used to treat patients with chronic lymphocytic leukemia, especially if they have already tried and failed a different first-line treatment. Generally, the cessation of Ibrutinib and other similar medications has been linked to disease progression. However, in upwards of 20% of patients, withdrawal symptoms have been reported with abrupt cessation of medication use, such as in our patients. Although toxicity and the need for toxicity-related dose adjustments have been well described in the literature, withdrawal from the use of these medications is much less known. Furthermore, the mechanism of action as to why withdrawal symptoms occur also has yet to be determined, owing to the reactivation of the immune systems and subsequent activation of cytokines. Nonetheless, this case highlights the importance of being able to recognize withdrawal symptoms and manage patients appropriately.
A Rare Duo: An Atypical Case of Esophageal Adenocarcinoma in a patient with Lynch & Hereditary Breast and Ovarian Syndrome

Introduction: DNA mismatch repair enzymes are essential to maintain the integrity of DNA and prevent formation of cancer. Mutations in certain genes (MLH1, MSH2, MSH6, PMS2, EPCAM), and in (BRCA 1 & BRCA 2) are associated with Lynch and Hereditary Breast and Ovarian Syndrome (HBOC) respectively. These enzyme deficiencies increase the risk of gastrointestinal and genitourinary malignancies such as the traditionally associated colorectal cancer (Lynch) and breast and ovarian cancer (HBOC). Only 1% of esophageal adenocarcinoma is attributed to Lynch syndrome and less than 1% is due to HBOC. Simultaneous mutations in genes associated with Lynch and in BRCA 1 or BRCA 2 are incredibly rare and can pose a significantly increased risk of malignancy.

Case Presentation: A 36-year-old male without gastroesophageal reflux disease or extensive tobacco smoking history presents to the clinic with progressive dysphagia to solids along with a thirty pound weight loss over a course of three months. Upper GI endoscopy revealed a distal friable esophageal mass with surrounding erosion. The mass was biopsied, and pathology was consistent with invasive poorly differentiated stage IV esophageal adenocarcinoma; staging PET scan revealed mediastinal, left infra-hilar, and retroperitoneal lymphadenopathy along with a proximal stomach mass. Family history was significant for male breast cancer in his paternal grandfather. Genomic testing via liquid biopsy using GUARDANT360 showed high microsatellite instability (MSI-H) and high fraction of BRCA 2 mutation (c677G>A pathogenic variant). Germine genetic testing was performed on white blood cells and indicated the presence of MLH1 mutation (c518delG pathogenic variant). Together, these genetic markers were consistent with Lynch and hereditary breast-ovarian cancer syndrome. The patient was initiated on standard of care palliative systemic treatment with chemotherapy (FOLFOX) and immunotherapy (Nivolumab). He completed 4 months of therapy after which repeat PET scan showed marked reduction in proximal stomach mass as well as resolution of FDG-avid lymphadenopathy. The patient was then transitioned to maintenance nivolumab and oral capecitabine at approximately 6 months from his initial diagnosis.

Discussion: Co-occurrence of Lynch and HBOC syndrome is an extraordinary phenomenon making this case the first of its kind to be reported in literature. It would be interesting to know if both promoted the tumorigenesis of this esophageal cancer. Genomic testing of the patient's family members is highly advised as the presence of Lynch or HBOC would necessitate extensive cancer surveillance; however, our patient has no siblings or offspring. This case also highlights the importance of treatment response prediction with platinum-based agents in the presence of BRCA 2 mutation and to immunotherapy in the presence of high microsatellite instability. In the instance the cancer is found to be platinum resistant, studies have found cancers with biallelic BRCA mutations to be sensitive to poly ADP ribose-polymerase inhibitors (PARPi). Currently, we are not certain if this patient will remain in long-term remission but will continue to monitor his progress.
Hiding in Plain Sight: An Unusual Case of Shone Complex

Introduction: Shone complex (SC) is a rare congenital heart disease involving the coexistence of four left ventricular (LV) inflow and outflow obstructions including a parachute mitral valve, supravalvular mitral ring, subvalvular aortic stenosis (AS), and aortic coarctation. Incomplete forms involve at least one LV inflow and one outflow obstruction. Previous data suggests that complete and partial SC makes up only 0.67% of all congenital heart diseases and is commonly diagnosed in infancy due to significant congestive heart failure. The commonly associated anomalies include mitral stenosis, parachute mitral valve, aortic coarctation, subvalvular AS, and bicuspid aortic valve. We present a unique case of delayed diagnosis of partial SC with multiple rare associations including supravalvular AS, patent ductus arteriosus (PDA), and an aberrant right subclavian artery.

Case Description: A 46-year-old male a medical history of multiple congenital heart anomalies and permanent atrial fibrillation s/p biventricular pacemaker placement presented for a TEE to evaluate for LV systolic function and aortopathies to plan replacement of his pacemaker vs ICD placement. He was born with congestive heart failure and found to have aortic coarctation, patent ductus arteriosus (PDA), bicuspid aortic valve with subvalvular stenosis, and an aberrant retroesophageal right subclavian artery. Cardiac catheterization at 14 months demonstrated a severe AS and severe pulmonary hypertension which prompted an aortic valvulotomy and ligation of his PDA. At 14 years old, echocardiography showed residual moderate AS, enlarged LV with moderate concentric hypertrophy with mildly depressed LV function. Although asymptomatic, the coarctation was repaired due to declining LV function. Despite interventions to the LV outflow tract, he developed a dilated LV at 21 years old and had his first episode of atrial fibrillation one year later. Due to persistent atrial fibrillation despite multiple interventions, he underwent AV nodal ablation with pacemaker implantation. He never developed symptoms of congestive heart failure. At 46 years old, TEE revealed normal LV systolic function, a severely dilated right ventricle with preserved systolic function, sclerotic bicuspid aortic leaflets with a mean gradient of 37.6 mmHg and a valve area of 0.8 cm² suggesting borderline severe AS, narrowing of the sinotubular aortic valve junction consistent with supravalvular stenosis, and a posteriorly displaced mitral orifice with a parachute mitral valve and a single papillary muscle without mitral stenosis. The multiple LV inflow and outflow obstructions revealed the diagnosis of partial Shone complex.

Discussion: The discovery of a parachute mitral valve led to the diagnosis by defining an LV inflow obstruction. Supravalvular AS is exceedingly rare compared to the typical subvalvular AS in SC. Mitral stenosis is the most common anomaly found in SC and its absence is unusual despite our patient having a parachute mitral valve. The lack of mitral stenosis may have allowed his asymptomatic cardiomyopathy, delaying his diagnosis. Common complications of SC include pulmonary hypertension, atrial arrhythmia, congestive heart failure, and worsening valvular stenosis requiring multiple surgical interventions. Although diagnosis can be challenging, it is important to recognize patients with SC to properly manage the associated cardiac complications of this fascinating congenital disease.
A Rare Case of Candida Pneumonia in the Intensive Care Unit

Introduction:

Pneumonia is a common respiratory tract disease and is one of the leading causes of mortality. Candida species rarely cause pneumonia, and candida pneumonia can be challenging to diagnose. C. glabrata is typically a nonpathogenic candida species and is present within normal respiratory flora. In this case report, C. glabrata pneumonia was diagnosed in a patient who is immunocompromised.

Case description:

A 64-year-old female with a history of lupus, scleroderma, thyroid cancer s/p resection, dilated esophagus, nicotine dependence, hyperlipidemia, hypothyroidism, and GERD initially presented to the ED due to shortness of breath and hemoptysis and was treated with azithromycin without improvement. Her CT findings were consistent with mucus plugging consistent with aspiration and post obstructive pneumonia on the left. Multifocal consolidations were observed bilaterally along with distended esophagus. Due to increased oxygen requirements, she was intubated and transferred to the ICU. Flexible bronchoscopy was performed which revealed mucus and debris in the left lung as well as a blood clot which was successfully removed. EGD was also performed which revealed a dilated esophagus and abnormal esophageal motility, LA grade A reflux esophagitis with no bleeding. Biopsies were obtained from the esophagus which showed squamous mucosa with mild squamous acanthosis and only rare lymphocyte exocytosis, otherwise without eosinophilic exocytosis or other specific histopathological changes.

Respiratory cultures were positive for scant growth Candida glabrata and scant growth of Acinetobacter baumannt. Fungal cultures showed moderate growth of Candida glabrata. Acid-fast smear and culture from bronchoscopy were negative. A flavus, fumigatus, and niger were negative. She remained hypoxic with minimal change in left lung infiltrates and underwent bronchoscopy again with removal of debris. Her antibiotic coverage was broadened. Her hospital course was complicated by spontaneous pneumothorax for which a chest tube was placed. Chest X ray showed a cavitary lesion in the left upper lobe concerning for necrosis prompting transfer to an outside facility for cardiothoracic surgery evaluation for possible VATS/pleurodesis.

Discussion and Conclusions:

Candida represents an opportunistic pathogen. To distinguish pathogenic candida from normal components of microflora, it can be helpful to obtain a tissue diagnosis instead of relying on its presence in sputum alone. In one study of 140 patients with pulmonary fungal infections, 4 patients were found to have C. glabrata. Candida pneumonia does not have a pathognomonic sign on radiology, but CT findings commonly include multiple nodules and air-space consolidation and multiple nodules, which can be surrounded by discrete areas of ground-glass opacity (CT halo sign). In our case consolidation and cavitation were demonstrated on the CT in the left upper lobe.

Fungal pneumonia can be challenging to diagnose and has a high mortality. Candida glabrata is less commonly observed than Candida albicans and can be challenging to treat as it has a higher minimum inhibitory concentration to fluconazole than other species. Successful treatment is possible with voriconazole, which has demonstrated efficacy in reducing tissue fungal load.
A Rare Case of Pulmonary Valve Endocarditis Without Typical Risk Factors in the Intensive Care Unit

Introduction: Right-sided endocarditis, which is comprised of endocarditis of either the tricuspid or pulmonic valve, accounts for approximately five to ten percent of all cases of infective endocarditis. Diagnosis can be challenging given the presence of nonspecific complaints but recognition of the diagnosis and prompt treatment are required to optimize patient outcomes.

Case: A 58-year-old woman without significant past medical history presented to the Emergency Department for one week of left-sided sciatica pain. Social history was negative for alcohol, drug, or cigarette use. Vitals signs were remarkable for tachycardia with heart rate of 102 and tachypnea with respiratory rate in the low 20s. Her blood pressure was 90s/60s. Laboratory workup revealed mild leukocytosis with white blood cell count of 11.2. The patient remained hypotensive despite fluid resuscitation and diastolic blood pressure was as low as the 40s and subsequently developed fever of 103.3F. Vasopressor support was required, blood cultures were taken, and broad spectrum antibiotics were started. Blood cultures grew MRSA. Additional questioning of family revealed the patient had recent history of incision and drainage of axillary abscess. Due to respiratory status decline CT chest was obtained showing what were likely septic emboli. Transthoracic echocardiogram was performed which was negative but due to high clinical suspicion, transesophageal echocardiogram was completed which revealed pulmonic valve vegetation 1.7 x 1.3 cm in size on the ventricular aspect of the valve. The patient was transferred to a tertiary facility for cardiothoracic surgery evaluation. Repeat echocardiogram was performed which did not re-demonstrate the endocarditis. She was seen by cardiothoracic surgery and surgical intervention was not recommended. The patient was discharged on oral linezolid.

Discussion:

The diagnosis of tricuspid valve endocarditis is based upon the modified Duke criteria, which have an estimated sensitivity and specificity between 70 and 80 percent. Treatment of pulmonary valve endocarditis involves antibiotics targeted towards the implicated pathogen. The need for operative intervention depends on the size of the vegetation, presence of recurrent septic pulmonary emboli, presence of a highly resistant organism, persistent bacteremia despite appropriate antimicrobial therapy, and/or complications such as heart block or paravalvular abscess. Risk factors for right-sided endocarditis include injection drug use; presence of a cardiac implantable electronic device; presence of an intravascular device such as a central line, intra-aortic balloon pump, or ventricular assist device; or the presence of an underlying right-sided cardiac anomaly. Staphylococcus aureus has been found to be implicated in approximately 50 to 70% of the cases of right-sided endocarditis as was the case in this patient. This patient’s vegetation was 1.7 x 1.3 cm in size and lacked high risk features that would necessitate surgical intervention. One study of mortality in tricuspid valve endocarditis determined mortality rates in patients with vegetations <1 cm, 1.1 to 2 cm, and >2 cm were 0, 3, and 33 percent respectively, putting this patient in the medium risk category. Ultimately, this case provides an example of a patient with atypical presentation of right-sided endocarditis of the pulmonic valve in the absence of typical risk factors.
Arrhythmia Induced or unmasked by medications and illness? A case report

Introduction: Adding multiple medications during hospitalization might induce or precipitate serious health issues. Therefore, physicians should add medications carefully and be vigilant of any potential side effects.

Case reports: 54-year-old female with a past medical history of migraine, irritable bowel syndrome, Iron deficiency anemia, and subacutie shoulder pain due to subacromial bursitis who presented to Garden City Hospital on 1/14/2024 due to a three-day history of headache, nausea, vomiting, and abdominal pain. Also, she reports odyphagia. Otherwise, the review of system negative. The preliminary lab results were significant for hemoglobin of 5.7. The patient was admitted for further evaluation. Three units of blood transfused. Gastroenterology and surgery were consulted due to suspected GI bleeding. CT abdomen, EGD and colonoscopy ordered. The patient started on antiemetics, PPI, and migraine cocktail.

Rapid response was called multiple times due to different types of arrhythmias in this patient who is without any known heart pathologies. First, on 1/ 15/2023 around 7 am, because the patient’s heart rate dropped to late 20s to early 30s bpm which resolved spontaneously. EKG

On 1/16/2023, after the EGD was done, the patient’s heart rate went to 140s-160 bpm on Telemetry. EKG was ordered and showed sinus rhythm with PVCs and prolonged QTc 512. Then patient’s heart rate went down to 70s-80s bpm spontaneously. Telemetry was re-reviewed thoroughly by the cardiology team and showed bigeminy with runs of A. fib with RVR. Subsequently CTA coronary ordered and showed clear coronary arteries with calcium score of 0. Electrolytes were within the normal limits.

On 1/17/2024, while the patient was in the pre-op area, waiting to have colonoscopy done, at 4:10 pm, telemetry showed tachycardia, suspected torsade de point spotted on telemetry. She was given 20 mg magnesium IV and 20 mg magnesium push ordered. Then Telemetry showed V-tach. She then went into bradycardia. ICU consulted due to V-tach and torsade de Pointe episode. At 5:30 pm, another Rapid response was called because the patient was in torsade. Magnesium was 3.72 from the previous rapid. The patient was shocked with 200 J and came back with A fib with CVR. Cardiology updated and then initiated transfer to Henry Ford Hospital for EP study. Again, at 6:30 pm, another rapid response was called for torsade, the patient was pulseless. One round of CPR was done then the patient was in V fib. She was shocked. ROSC achieved. The patient was transferred to ICU. Initially started on Amiodarone drip. Then cardiology discontinued Amiodarone drip and all medications that can cause QTc prolongation and started the patient on Lidocaine drip. Then the patient was transferred on 1/18/2024 to HFH where the patient was started on Isoproterenol.

Conclusion: carefully adding and revising medication during hospitalization is critical, especially in patients with risk factors.
Dermatological Adverse effects of cortisol pump: A Case Report

Introduction:
Dermatological adverse effects are not uncommon with the use of adhesives used in insulin infusion pump and continuous glucose monitoring devices (CGM). Synthetic cortisol is the cornerstone in management of chronic adrenal insufficiency. Advances in management range from immediate release hydrocortisone tablets to once a day tablets. Insulin pumps have been used to deliver hydrocortisone as Continuous Subcutaneous Hydrocortisone Infusion (CHSI) and this therapy is showing promising beneficial effects in mitigating fatigue, improving sleep and reducing over use of corticosteroids. However it has its own side effects. We entail a case of a CHSI user who developed a nodular skin infection leading to sepsis requiring in-hospital management and bolus hydrocortisone need.

Case description
45 year old female with medical history significant for secondary adrenal insufficiency, atopic dermatitis and type 2 diabetes on insulin on continuous subcutaneous hydrocortisone infusion via cortisol pump, cetirizine and basal insulin was admitted due to worsening swelling and redness at the pump site that she noticed growing from a pea size to cotton ball size in three days prior to admission. She had fever and chills for 2 days and her labs were remarkable for leukocytosis, thrombocytosis and elevated CRP. She was treated with appropriate antibiotics, crystalloids for sepsis and General surgery consultation was sought. She underwent incision and drainage for the abscess and required bolus dosing with hydrocortisone 50 mg every 6 hours for the first 24 hours. Cortisol pump was removed and she was discharged home on oral antibiotics according to wound culture results and gradual steroid taper.

Discussion
Exogenous steroids fail to faithfully recreate the effects of physiologic cortisol. Hydrocortisone, whether oral or intramuscular, cannot mimic variations in cortisol levels attributable to activity and the circadian rhythm. Absorption is further complicated by exogenous administration’s susceptibility to interactions with other drugs, food, and an individual’s gastrointestinal motility. These artefacts of administration result in the absence of a pre-awakening surge and create supraphysiological peaks, predisposing to obesity, depression, hypertension, dyslipidemia, and cardiovascular events.

Infusion pumps allow finer control over cortisol delivery improving mood, fatigue, energy and Quality of life. Wider use of it can lead to reduced hospitalizations and morbidity. However clinicians need to be aware about the serious adverse events related to adhesive medical devices like insulin pump, cortisol pump and CGM in order to prevent it from happening and ensure compliance to therapy. Adhesives of infusion pump is the main causative agent for superficial dermatitis, steroid use in any form can predispose to skin infection due to impaired immunity and dermal atrophy. Atopy is found to have significant association with dermatological side effects like eczema, allergic dermatitis and wound infection.

Conclusion:
Frequent checking and changing of the cortisol pump site with proper cleaning are important points to be discussed with the users to create awareness. Infections should not be managed lightly and patients should visit their PCP promptly.
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**Insulin Resistance Spawns Hypertriglyceridemia-induced Acute Pancreatitis and Diabetic Ketoacidosis: A Narrative Review**

**Introduction:** Individuals with obesity and type 2 diabetes mellitus (T2DM) exhibit defective insulin-stimulated glucose uptake accompanied by impaired insulin suppression of hepatic glucose output, referred to as “insulin resistance (IR)”. Hypertriglyceridemia (HTG) is a common form of dyslipidemia closely associated with IR, representing valuable clinical abnormalities of the metabolic syndrome. Firstly described in 1988, “the metabolic syndrome” functions as a link between IR, dyslipidemia, T2DM and hypertension. These four components collectively contribute to an increased risk of atherosclerotic cardiovascular disease (ASCVD). Traditionally, the risk of HTG to ASCVD focuses on low-density lipoprotein and decreased high-density lipoprotein. Here we presented two cases describing severe HTG in poorly controlled T2DM which provoked acute pancreatitis and diabetic ketoacidosis (DKA). The manifestations were dramatically different from “the metabolic syndrome” and represent an acute crisis of lipid and glucose deregulation.

**Case 1:** A 48-year-old female presented with a 3-day epigastric pain. She had a history of T2DM, Hypertension, and Obstructive Sleep Apnea. She is a nonsmoker, has no alcohol, no significant family history, and exercises regularly. Initial TG was 3841 (mg/dL), HbA1c (10.1%), lipase (693 u/L), anion gap (AG, 18.6 u/L) with positive serum ketones. Abdomen Computed Tomography (CT) showed pancreatitis and hepatic steatosis. Case 2: A 32-year-old male with T2DM came with 2-day abdominal pain. He was hospitalized 9 months ago for Hypertriglyceridemia–induced acute pancreatitis (HTGAP, TG 650mg/dL and lipase 392 u/L). After discharge, he was non-compliant with the medications. He is a non-smoker and non-drinker. His both parents were diagnosed with T2DM in the 40s. His TG was 5359 (mg/dL), lipase (601 u/L), HbA1c (12%), anion gap (AG, 17.8 u/L) with positive serum ketones. Abdomen CT showed AP and hepatic steatosis. Both patients were admitted to the intensive care unit receiving continuous insulin and Lactated Ringer’s solution infusion. The DKA’s were resolved in 24 hours and TGs level gradually came down to less than 1000(mg/dL) on days 5 to 6. Both patients were smoothly discharged with Fenofibrate, Atorvastatin and Insulin regimens.

**Discussion:** This clinical scenario represents an acute crisis of lipid and glucose deregulation (Acute Metabolic Crisis Syndrome). It highlights the remarkable significance of IR to intertwine the deranged lipid metabolism in white adipose tissue with hepatic glucose metabolism and defective pancreatic endocrine and exocrine functions, which ultimately generates HTGAP and DKA. We present a working model of intertwined unregulated lipolysis from HTG and IR triggering HTGAP and DKA. IR and lipolysis create a two-way feedback between white adipose tissue and the liver. It then produces pancreatic injury with pancreatic self-hydrolysis leading to HTGAP, which results in insulin deficiency and triggers counter-regulatory hormone further worsening hyperglycemia and lipolysis, ultimately causing the downward spiral of HTGAP-DKA. Acute management of this acute metabolic crisis involves fluid resuscitation, continuous insulin infusion, nutrition management, and prevention of complications. Chronic management includes non-pharmacological measures and medications such as Fibrates, Statin, Omega-3 FAs and Metformin. Further investigation involves developing clinically applicable assessment of IR, lipolysis, and stratified functional and pathological diagnosis for IR- lipolysis cycle.
Right thalamic abscess in a Caucasian man with rheumatoid arthritis, an intriguing case report.

Introduction:
A brain abscess is a localized collection of pus within the brain tissue. Approximately 1500-2000 cases are presented annually in the United States. A thalamic brain abscess is a deep-seated intraparenchymal lesion that has been reported in a few of the cases in the literature.

Case description:
Herein, we present the case of a 57-year-old male patient with a two-year history of rheumatoid arthritis who was under treatment with methotrexate and etanercept for one and half years, presented to our hospital with sudden onset headache, nausea, vomiting, and fever for 5 days. Brain magnetic resonance imaging (MRI) illustrated the right thalamic intracranial abscess. The right stereotactic brain biopsy was performed, and cultures were obtained which isolated Streptococcus intermedius. He was treated successfully with ceftriaxone and metronidazole for eight weeks with no neurological sequelae.

Discussion/Conclusion:
Patients with immunocompromised states have a higher incidence of acquiring a thalamic abscess. The most frequent pathogens are potentially streptococcus and staphylococcus. Most of the abscesses are spread from contiguous sites, such as dental infections, mastoiditis, orbital cellulitis and otitis media; or hematogenous route. Common odontogenic sources include streptococcus, Prevotella, Haemophilus, and Fusobacterium. The most common presentation is headache, nuchal rigidity, seizures, cranial nerve deficits, altered mentation, fever, vomiting, and focal neurological deficits such as hemiparesis or aphasia. Thalamic abscess carries a high morbidity and mortality. Therefore, early recognition and intervention are paramount to prevent long-term sequelae.
A Clinical Case of Icteric Leptospirosis in an Urban Setting

Introduction: Leptospirosis, caused by the gram-negative spirochete, Leptospira, is prevalent in tropical locales. It is significantly less common in more temperate areas. Infection can present with complex clinical features ranging from a mild subclinical infection to Weil disease, characterized by multiorgan involvement, most commonly kidney and liver failure. In this report, we detail a patient admitted to a large urban hospital for management of leptospirosis, emphasizing the need for empiric therapy with strong clinical suspicion.

Case: We present a 49-year-old man with no medical history who transferred to our hospital following a two-day stay at an outside facility. One week prior, he developed abdominal pain, fatigue, irregular bowel movements, and notable jaundice. He also reported shortness of breath, abdominal fullness, swelling of the arms and feet, and intermittent visual changes. Examination revealed jaundice, scleral icterus, bilateral lower extremity edema, and petechiae on the upper extremities. He was afebrile and hypertensive. Laboratory results revealed leukocytosis (21.6 K/ul), absolute neutrophil count (14.77), thrombocytopenia (Plt 53 K/ul), acute kidney injury (Creatinine 6.88 mg/dL) and acute liver injury with total bilirubin of 19.1 mg/dL, direct bilirubin of 16.4 mg/dL, alkaline phosphatase 81 IU/L, AST 53 IU/L, ALT 53 IU/L. Other testing including lipase, acute hepatitis panel, EBV/CMV, and autoimmune liver panel were unremarkable. CTAP revealed mild splenomegaly and MRI demonstrated hepatic steatosis without cirrhosis and two indeterminate liver lesions, likely benign hemangiomas.

Common causes of acute biliary and hepatic etiologies were ruled out. He reported insignificant alcohol use, no supplement, analgesic or illicit drug use. The patient reported a recent camping trip in Northern Michigan, involving camping, fishing, a swim in the Great Lakes, and canoeing in wooded areas along a river. He denied tick exposure or rash but mentioned a recent rat outbreak in his neighborhood. Despite no direct contact with rats, his dog killed two, and he disposed of them using a plastic bag.

Despite the absence of certain classic signs like conjunctival suffusion, the patient exhibited a constellation of symptoms and possible exposure consistent with leptospirosis. IV Doxycycline was initiated for high suspicion of icteric Leptospirosis while awaiting results, leading to significant improvement in symptoms, hyperbilirubinemia, and creatinine. Post-discharge, results returned positive for anti-leptospirosis IgM, which confirmed the diagnosis.

Discussion: Early symptom recognition is crucial, especially in patients with multiorgan involvement and specific exposures. Most cases are mild, with only 10% progressing to severe forms (Weil). Leptospira transmission involves contact with contaminated soil, food, or water from urine of infected animals, predominantly rodents, entering through skin or mucous membranes. High suspicion is essential for prompt therapy to prevent further sequelae. This case emphasizes the significance of thorough history-taking, particularly in non-tropical urban settings where conditions like leptospirosis may be overlooked. Despite modern medicine’s reliance on diagnostic testing, a comprehensive history, physical examination, and basic lab analysis were adequate to clinch a diagnosis and initiate therapy. Focus remained on optimizing patient care with empiric antibiotics and a nuanced treatment approach.
**Medical treatment of STEMI in patients with SCAD opposed to PCI**

**Background:**

SCAD, holds a significant clinical importance as a notable cause of ST Elevation Myocardial Infarction (STEMI), most likely affecting young to middle-aged women around the childbearing age. An accurate reporting of SCAD likely necessitates a low index of suspicion and more familiarity of its angiographic variants. Myocardial Infarction with Non-Obstructive coronary arteries (MINOCA) is a subgroup of which SCAD is imbedded within. The gold standard treatment for STEMI is Percutaneous Intervention, however it is contraindicated in SCAD patients.

**Case presentation:**

The following case describes a patient with no significant past medical history besides controlled hypertension, who presented with chest pain and was found to have a dissection of the Left Main coronary extending to Left Anterior Descending coronary. A 49-year-old female with unremarkable medical history, presented with sudden onset of mid-sternal chest pain radiating to her neck. The initial EKG showed ST elevations in V2-V4 with negative troponin.

Bedside Echocardiogram confirmed an Ejection Fraction of 43%, with severe hypokinesis of the mid-distal septal and anterior wall. The patient urgently underwent cardiac catheterization which revealed spontaneous dissection in Left Main coronary extending to Left Anterior Descending coronary with a hematoma compressing the Left Main lumen. Impella was placed for ventricular support, the procedure was complicated with large flow-limiting dissection extending from the common iliac artery into the Common Femoral Artery, which was fixed with two overlapping stents. Our patient was started on Aspirin 81mg, Clopidogrel 75mg and Metoprolol 25mg, her chest pain has improved, and she was subsequently discharged.

**Conclusion:**

Patients with STEMI are usually treated with an urgent PCI, However, stenting the culprit vessel is contraindicated in patients with an overlapping SCAD, as this could further propagate the dissection. Beta-blockers are often started as they are commonly agreed to decrease the recurrence of SCAD, which is estimated to be around 10%.
Rare Sequela of Chronic Sinusitis: Simultaneous Orbital Cellulitis, Subdural Empyema, and Endocarditis

Abstract:
Chronic sinusitis is a common condition among primary care patients, it generally yields a good prognosis; However, locoregional and distant complications can occur as a result of extension of the infection beyond the sinuses which can be fatal. [1].

Infections from the paranasal sinuses can spread to the cavernous sinus via pterygoid plexus, leading to further spread to other sinuses via communicating veins. Spreading of the infection to the orbit can cause a range of ocular complications, most commonly orbital cellulitis, while spreading of the infection to the brain may cause cranial complications from subdural empyema to brain abscess [2]. We present a case of a 19-year-old patient with chronic rhinosinusitis which led to oculo-orbital, cranioencephalic, and cardiac complications.

Case:
A 19-year-old male with history of allergic rhinitis and sinusitis presented for right eye swelling. He was in his usual state of health until he developed a headache, body aches, tearing of right eye and fever. Physical exam showed significant right eye proptosis, erythema, and pain with all eye movements. Initial CT imaging revealed right orbital cellulitis with subperiosteal abscess compressing the superior rectus. MRI brain revealed right-sided sinusitis with right orbit abscess and frontal empyema without dental anomalies. Nasal endoscopy revealed right-sided hypertrophic turbinites with pus draining from middle meatus. Patient was taken to OR requiring multispecialty intervention for right endoscopic frontal sinusotomy, total ethmoidectomy, and sphenoidotomy, followed by right orbitotomy with abscess drainage.

Cultures grew Streptococcus Anginosis, antibiotics were deescalated to ceftriaxone and metronidazole. Trans-Thoracic Echocardiography (TTE) followed by Trans-Esophageal Echocardiography (TEE) were performed showing Infective Endocarditis of pulmonic valve. Patient initially improved with antibiotics and remained afebrile. However, several days after initial surgical abscess drainage, the patient developed worsening headache with left-side weakness and foot drop. STAT Brain MRI showed increased size of subdural empyema with mass effect on right frontal lobe. He underwent right frontal craniotomy and empyema evacuation. Post-operatively, the patient improved and was eventually discharged on IV antibiotics. Six weeks later, the patient returned to work with resolution of all symptoms and no residual deficits.

Discussion:
Rhinosinusitis is the inflammation of the mucosa of nasal cavity and paranasal sinuses. Acute rhinosinusitis is usually viral; However, chronic rhinosinusitis is mostly bacterial [1]. Staphylococcus aureus, gram-negative bacteria, and anaerobes are the leading causes of chronic sinusitis, however Streptococcus Viridans has been isolated from chronic sinusitis patients and is the leading cause of sinusitis complications. Sinusitis infection can spread causing serious orbital and cranial complications, leading to loss of vision, serious neurological sequela, and death.

This case describes the rare, potential life-threatening complications of sinusitis infection spreading through the danger area of the face and stresses the importance of rapid multidisciplinary management of the condition.

References:

Mycotic pseudo-aneurysm of the left coronary cusp in MSSA bacteremia

Introduction:
Staphylococcus aureus bacteremia is a common cause of bacteremia and is associated with a mortality rate of 13.3% at two weeks and 27% at three months. Complications include infective endocarditis, metastatic infections, CNS seeding, pulmonary issues, and septic thrombophlebitis. Mycotic coronary aneurysms, though rare, may occur, with very few documented cases involving coronary arteries.

Case description:
A 73-year-old man with a past medical history significant for CAD (s/p CABG x3 and PCI), HTN, HLD, T2DM, who initially presented to an outside hospital due to worsening right hip and left shoulder pain. Of note, the patient had a steroid injection of his right hip a month prior to presentation. He underwent CT imaging that was notable for soft tissue mass at the left sternoclavicular joint that was suspicious for an abscess. Hip arthrocentesis and culture revealed Methicillin Sensitive Staph Aureus (MSSA) as the culprit. The patient was started on appropriate antibiotics. Patient simultaneously had a troponin elevation (900) with ST segment depression, that was later diagnosed as a type II NSTEMI secondary to sepsis. At this point the patient was transferred to our hospital for further management.

The patient underwent incision and drainage of the right hip, right shoulder, and left sternoclavicular joint. Intraoperative cultures grew gram positive cocci in clusters. Patient was switched to nafcillin. Throughout the hospital admission, the patient developed shortness of breath for which a CTPE was ultimately ordered to rule out PE. It demonstrated a 3.4 x 1.6 x 1.7 cm mycotic pseudoaneurysm arising off the left coronary cusp with adjacent hematoma. This was a new finding compared to a prior study.

TEE was then done which showed pseudoaneurysm of the aortic root consistent with an aortic root abscess, as well as new mild aortic regurgitation, severe tricuspid and mitral regurgitation. Subsequent CTA coronary showed enlargement of the pseudoaneurysm with worsening contrast extravasation into the pericardium. While awaiting cardiothoracic surgery intervention, the patient went into cardiac arrest and expired.

Discussion
Infective endocarditis, a complication of MSSA, was seen in our patient with a further complication of a mycotic aneurysm of the cusp of his left coronary artery. CT angiography is the primary diagnostic tool. Management of MSSA bacteremia involves antibiotics and source control. Surgical intervention is required for coronary aneurysms that are larger than 1-2 cm, as seen in our patient. They are excised and the distal coronary artery is revascularized to avoid complications like tamponade or sudden death. Revascularization of the distal coronary artery is crucial to prevent complications such as tamponade or sudden death.

Conclusion
In conclusion, there are life-threatening complications associated with MSSA bacteremia with rare occurrence of a mycotic coronary aneurysm. Despite diligent efforts in diagnosis and intervention, the unfortunate outcome highlights the importance of heightened awareness, prompt management, and ongoing advancements in addressing challenges.
A Case Presentation of M. Kansasii Resembling Lung Malignancy

Background: Nontuberculous mycobacterial (NTM) pathogens are opportunistic pathogens with increasing prevalence since the HIV pandemic. They are indistinguishable from that of Mycobacterium tuberculosis and thus require microbiological confirmation with three different AFB smears. These pathogens are typically not transmitted person-to-person. M. kansasii is the second most common NTM infection and usually affects patients with underlying lung disease or those immunocompromised. M kansasii infection occurs throughout the United States, with the highest incidence in the Midwest and the Southwest making up only 3% of all clinical isolates of NTM.

Case Presentation: A 57-year-old female with severe emphysematous lung disease and a 43 pack-year history presented with worsening shortness of breath and pleuritic chest pain. Despite multiple hospitalizations in the past year and treatment for bacterial pneumonia, her symptoms persisted. Initial CT findings showed a left upper lobe (LUL) cavitary lesion and multiple pulmonary nodules. A subsequent respiratory culture grew Mycobacterium kansasii, initially disregarded as a contaminant given interval improvement in symptoms and resolution of sputum production. A month later, the patient presented again with cough and shortness of breath and was afebrile, and without leukocytosis. Repeat CT scans showed progression of the LUL cavitary lesions and bilateral pulmonary nodules and an incidental pulmonary embolism. A PET scan revealed significant FDG uptake, raising concerns for possible malignancy. She was transferred to our hospital for a high-risk lung biopsy. On admission to our hospital, she was vitally stable, with an unremarkable physical exam with normal lung sounds and unremarkable labs. While undergoing further evaluation, three AFB smears and sputum cultures confirmed M. kansasii. Meeting the 2020 ATS/IDSA/ERS/ESCMID diagnostic criteria for NTM infection, she was started on Rifampin, Isoniazid, and Ethambutol. However, she developed asymptomatic elevated transaminases due to Isoniazid-induced liver injury and was switched to Azithromycin. The patient showed symptomatic improvement on this revised regimen.

Discussion: In the United States, M. kansasii is the second most common cause of NTM with tap water being its only known environmental source. Cases have been described in the literature of M. kansasii, however, usually occurring in patients with active HIV infections or those on immunosuppressant medications such as TNF-alpha inhibitors. However, in our case, it was reported in an immunocompetent patient with severe lung disease identified as an important risk factor for developing NTM lung disease.

Conclusion: This case highlights the diagnostic complexity of M. kansasii infections, particularly in immunocompetent patients with pre-existing lung disease. It highlights the necessity for thorough microbiological confirmation to differentiate it from similar presentations, like tuberculosis. Additionally, the case illustrates the importance of adaptable treatment strategies, considering potential drug-related side effects, to effectively manage NTM infections.
A Curious Case of a Syphilitic Ulcer

Introduction:

Syphilis is a sexually transmitted disease caused by the spirochete Treponema pallidum. Often referred to as the great mimicker, it can affect a multitude of organ systems and can present with a wide array of clinical symptoms, leading to high rates of misdiagnosis.

Case:

A case of a 28-year-old male previously untreated for syphilis presented with progressively excruciating throat pain that began ten days prior, associated with decreased oral intake. His physical exam was notable for cervical lymphadenopathy, a large right erythematous necrotic ulcer on the posterior oropharynx, and hyperpigmented macules on the palms. His labs demonstrated neutrophilic leukocytosis, acute kidney injury, and starvation ketosis. CT imaging revealed an ulcerative necrotic lesion at the base of the right tongue. Regarding his social history, the patient engaged in oral and anoreceptive sex with male and female partners with inconsistent protection usage.

A 4th-generation HIV test was ordered, which was reactive. Furthermore, in light of his prior history of syphilis, serologies were obtained with an RPR 1:4 from 1:128, one year prior, during which the patient wasn’t treated. He was started on treatment for HIV with Biktarvy. Regarding the necrotic ulcer, our differential was narrowed to infectious etiologies such as syphilis, tuberculosis, gonorrhea, chlamydia, and histoplasma vs. malignant etiologies such as lymphoma or Kaposi sarcoma. Chlamydia, gonorrhea, and Histoplasma cultures were all negative. Due to its friable nature, a biopsy was deferred, and the patient was treated presumptively with doxycycline for a total of 28 days for late latent syphilis with symptomatic resolution.

Discussion:

The differential for a necrotic oral ulcer can be extensive; moreover, as our patient had a new diagnosis of HIV, it made the differential even broader. Oropharyngeal manifestations of syphilis are rare and can occur at any stage. In 62.8% of patients, it occurs in the secondary stage and can lead to deep, painful ulceration. In contrast, primary syphilis presents as a painless papule at the site of inoculation, which later progresses to a chancre. Our case was unique as our patient was in the late latent stage of the disease and presented with severe mucocutaneous manifestations with a concomitant new diagnosis of HIV. Syphilis represents a substantial global health burden, particularly between the ages of 15-49, wherein 6 million new cases are diagnosed each year. This patient presented to an emergency room 1-year prior requesting HIV testing and was denied; he had a viral load of >6 million at the current presentation. Prioritizing timely HIV and STD testing can help mitigate complications of these diseases.

Conclusion:

Our case highlights the importance of having a high index of suspicion of syphilis, particularly in an immunocompromised host who presents with new-onset oral ulceration. Furthermore, it also opens up a wider discussion of the need to have greater access to HIV and STD testing.
Complete Radiological Response of HER2+ Breast Cancer with Active Brain Metastases to Trastuzumab Deruxtecan

Introduction: Novel therapeutics for patients with human epidermal growth factor receptor 2 positive (HER2+) breast cancer offer significantly improved outcomes for those with historically poor prognoses. Studies indicate that 30 to 50% of patients with HER2+ metastatic breast cancer will develop brain metastases. The management of breast cancer with central nervous system (CNS) metastases is contingent upon many factors including breast cancer subtypes, size and location of tumors, and patient characteristics. Treatment modalities for breast cancer with CNS metastases include surgical resection, radiation therapy, and/or systemic therapy. However, blood-brain barrier drug penetration remains a significant barrier in systemic therapy. Despite significant strides in anti-HER2 therapies, the optimal permutation of these treatments remain unclear.

Case Description: An 84-year-old woman presented with a diagnosis of de novo bilateral breast adenocarcinoma with metastases to the mediastinum, lungs, and liver. Liver biopsy was positive for over-expression of the HER2 oncogene protein (3+). Two months following diagnosis, she was started on paclitaxel, trastuzumab, and pertuzumab. Positron emission tomography scans at three and six months after treatment initiation showed complete response. However, ten months after treatment initiation, MRI of the brain revealed new, multiple, small nodular enhancing lesions within the bilateral cerebellar hemispheres. Several treatment options including whole brain radiation and systemic therapy with regimens including tucatinib, capecitabine, trastuzumab emtansine, or Trastuzumab deruxtecan (T-DXd) were discussed. Due to her asymptomatic state, whole-brain radiation was not preferred in avoidance of potential neurocognitive decline associated with said radiation. Given the patient’s concerns regarding medication adherence, she opted for T-DXd. Imaging three and six months following initiation of T-DXd demonstrated resolution of the bilateral cerebellar hemisphere lesions.

Discussion: A standardized approach to managing HER2+ breast cancer with brain metastases is rarely universally applicable. Standard of care includes a combination of pertuzumab, trastuzumab, and a taxane. Patients may remain on first-line therapy after development of brain metastases if there is controlled extra-cranial disease and if brain metastases are localized and controllable with radiation.

Rapid progression of disease necessitates consideration of an alternative anti-HER2 therapy. Selection of second-line therapy typically involves options such as tucatinib, capecitabine, trastuzumab emtansine, and T-DXd. Clinical trials such as HER2CLIMB noted that adding tucatinib to trastuzumab and capecitabine led to improved outcomes. Although T-DXd has demonstrated superior overall survival and responses compared to trastuzumab emtansine, there is no standardized approach newly diagnosed brain metastases and/or CNS disease progression after receiving local therapy.

The DESTINY-Breast03 trial exclusively included patients without CNS disease or those with stable CNS metastasis. The phase 2 TUXEDO-1 trial evaluated the efficacy and safety of patients with HER2+ metastatic breast cancer and active brain metastases. In this context, T-DXd has exhibited a noteworthy intracranial response rate of active brain metastases originating from HER2+ breast cancer.

Conclusion: Although there are no standardized treatments for HER2+ breast cancer with new or active brain metastases, T-DXd has demonstrated promising. Ongoing clinical trials are imperative to refine our understanding and guide the development of more effective strategies for managing HER2+ breast cancer, particularly those with CNS involvement.
A rare case of hemophagocytic lymphohistiocytosis (HLH) in a patient with chronic lymphocytic inflammation with pontine perivascular enhancement responsive to steroids (CLIPPERS)

CLIPPERS is a syndrome of pontine and cerebellar encephalomyelitis of unclear pathogenesis characterized by CD3+ reactive T-lymphocyte infiltration of the perivascular white matter and parenchyma, along with CD20+ B-lymphocytes, activated microglia and CD68+ histiocyte. HLH describes a spectrum of diseases with the shared features of natural killer (NK) and cytotoxic T lymphocyte (CTL) deficiencies, leading to impaired cytotoxic downregulation of macrophage response. Here, we describe a rare case of a patient with CLIPPERS who presented to the hospital with a 4-month long history of high grade fevers, chills, malaise, headaches, and splenomegaly who was subsequently diagnosed with HLH, suggesting a possible overlap syndrome between these rare immunologic syndromes.

A 53 year-old Caucasian male with a known diagnosis of cirrhosis and CLIPPERS disease for 8 years on chronic steroids presented to the hospital with a 4-month history of high grade fevers to a maximum temperature of 104°F, chills, malaise, headaches, and progressive abdominal distention with splenomegaly. Over the preceding 4 months the patient had undergone extensive evaluation to elucidate the cause of his fevers without success.

Upon admission, infectious workup was negative for bacterial, viral, and common fungal pathogens. Serologic evaluation notably revealed that ferritin was elevated at 27,862 ng/mL, triglycerides were 472 mg/dL, and fibrinogen was 76 mg/dL. CBC revealed leukopenia of 2.6 K/uL, anemia of 12.2 g/dL, and thrombocytopenia to 78 K/uL. Peripheral smear revealed normocytic normochromic anemia with slight anisopoikilocytosis including slight burr cells and ovalocytosis, thrombocytopenia with occasional macrothrombocytes, absolute lymphopenia and occasional apoptotic neutrophils, and no evidence of dysplastic or neoplastic cells with cytometry immunophenotyping. Bone marrow biopsy at this time revealed occasional hemophagocytosis. Interestingly, CA 19-9 and CA-125 were both newly elevated in the patient, though PET-CT performed approximately a month after admission revealed no concern for solid tumor disease.

Based on these findings, our patient was diagnosed with secondary hematophagocytic lymphohistiocytosis. He was started on etoposide and decadron per the HLH-94 regimen. He improved clinically and was discharged from the hospital.

While complete pathophysiologic description of both diseases remains elusive, individually they may in fact represent a spectra of immunomodulatory disorders with similar clinical presentations, rather than two contained pathologic entities, with the possibility of some arcane immunologic overlap between the two disorders. This case highlights the significant gaps in our knowledge about the immunologic dysregulation involved in CLIPPERS and HLH. Therapeutic advancement will only be possible as we continue to unravel the complexities of the immune system as they relate to these and other disorders. Further research is hindered by the relative rarity of both disorders individually, and especially together. We sincerely hope that this case will enhance clinical awareness and provide insight to future investigators.
Mystical Minds: The Enigmatic Encounter of Acute Encephalopathy and Multiple Myeloma

Multiple myeloma-induced encephalopathy is a rare neurological complication. Although the precise pathophysiology remains unclear, it is often attributed to the effects of abnormal proteins produced by myeloma cells, leading to neurotoxicity. Early recognition and prompt management, typically involving treatment of the underlying multiple myeloma, is crucial for improving neurological outcomes in affected patients. We present a case of acute onset encephalopathy in a previously healthy female found to have multiple myeloma.

A 57-year-old female presented to the emergency department with a chief complaint of weakness and fatigue. She was admitted for hypercalcemia and acute kidney injury. However, she became lethargic and altered requiring intubation. She was treated for pneumonia and extubated yet remained encephalopathic. She was non-verbal and unable to follow commands and thus treated broadly for meningitis. Work-up for acute encephalopathy was initiated and remained largely unremarkable. Ammonia levels were initially noted to be only mildly elevated. A lumbar puncture obtained was pertinent for elevated lactic acid. She was ultimately found to have an elevated IgA level and borderline elevated serum viscosity level. CT head showed numerous aggressive lucencies within the calvarium with destructive lesions concerning for osseous metastatic disease or multiple myeloma. Bone marrow biopsy led to the diagnosis of IgA lambda myeloma. MRI head revealed numerous small foci of chronic microbleeds in the peripheral gray-white matter junction. She received steroids with no improvement. The patient remained persistently encephalopathic and received lactulose for non-cirrhotic hyperammonemia-induced encephalopathy, potentially secondary to multiple myeloma, but her symptoms were not resolved. Due to the persistence of her encephalopathy of unknown etiology, she underwent plasmapheresis for suspected hyperviscosity syndrome with a 50% reduction in IgA levels following three treatments. Her encephalopathy did not improve thus she was started on bortezomib chemotherapy and steroids during admission. About one week after her first cycle of chemotherapy, and about 3 months from initial presentation, the patient’s encephalopathy resolved as she became more responsive, alert, and orientated.

This case demonstrates a challenging presentation of acute onset encephalopathy of unknown origin, requiring extensive diagnostics and an interdisciplinary approach. It illustrates the importance of maintaining broad and evolving differentials. It is difficult to determine the cause of her encephalopathy, however, given the response to plasmapheresis and chemotherapy, it is reasonable to believe that multiple myeloma and hyperviscosity contributed to this presentation. It is hypothesized that this patient’s encephalopathy was likely secondary to subacute hyperviscosity limiting adequate brain perfusion evidenced by lactic acidosis in the cerebrospinal fluid and MRI findings of diffuse micro bleeds. There are rare cases of non-cirrhotic hyperammonemia-induced encephalopathy secondary to multiple myeloma, however, such cases often present with greater elevations in ammonia, which was not the case in this patient. Nonetheless, with adequate recovery in her mental status following chemotherapy, her treatment was continued.
Posterior ST elevation Myocardial Infarction from Left Ventricular Thrombus Embolization

Background: Left ventricular thrombus is a well-documented complication of patient’s with reduced ejection fraction. The literature supports anticoagulation of left ventricular thrombus to reduce the risk of embolization. Cerebral emboli remain the most common complication from left ventricular thrombus, however, rare complications including coronary, splenic, renal, and limb embolism leading to acute ischemia are less commonly documented sequelae. We present a case of a young man with a posterior STEMI secondary to coronary embolism of left circumflex from left ventricular thrombus.

Case presentation: A 29 year old man with a past medical history remarkable for heart failure with improved ejection fraction presented to our institution’s emergency department with chest pain, diaphoresis, and shortness of breath that had been ongoing for a 3 hours. On physical examination, he was afebrile, regular heart rate, and tachypneic. He appeared uncomfortable and diaphoretic with trace pitting edema.

Initial laboratory workup revealed a high sensitivity troponin value trend of 28, 32, and 130 which peaked at >20,000 within 10 hours of presentation. Brain-natriuretic peptide was 1,567. Hemoglobin was 6 from a normal baseline. EKG demonstrated ST depression in V2-V4 consistent with a posterior STEMI.

Given the laboratory and EKG findings, acute coronary syndrome was suspected. However, due to concern for bleeding with abnormal hemoglobin as well as history of normal coronary arteries on cardiac catheterization 2 years prior, the decision was made not to move forward with coronary angiogram with or without percutaneous coronary intervention. He was admitted to the intensive care unit for further monitoring. His repeated hemoglobin was within normal limits. He was started on dual anti platelet therapy and low intensity heparin. Echocardiogram demonstrated ejection fraction of 8% (severely reduced), large fixed apical thrombus 2.9 cm in size, Grade III diastolic dysfunction, severely dilated left and right atrium, pulmonary artery pressure of 41 mmHg. When compared to echocardiogram 2 years prior, the ejection fraction was reduced and there was a new apical thrombus present.

Following stabilization of anemia, 3 days after presentation, patient underwent coronary angiogram which was remarkable for mid left circumflex 90% stenosis, 2nd obtuse marginal 71% stenosis, 3rd obtuse marginal 90% stenosis, posterior left ventricular artery 99% stenosis. Findings were consistent with thrombotic embolization likely secondary to left ventricular thrombus into the distal left circumflex, posterior left ventricular artery, and 2nd obtuse marginal branch. Given the late findings and lack of current symptoms, the decision was made to treat medically with anticoagulation.

Clinical implications: Clinicians should be aware of the cardiac risks of left ventricular thrombus and keep left ventricular thrombo-embolism on the differential for patients presenting with a ST elevation myocardial infarction. In our case, a particularly high level of suspicion was warranted given patient’s normal coronary arteries prior and new left ventricular thrombus on echocardiogram. Additionally, our case highlights the importance of identifying patient’s with risk factors to develop left ventricular thrombus, those with reduced ejection fraction, and echocardiographic screening.
Left ventricular noncompaction: A rare cause of cardiomyopathy

Background: Left ventricular non-compaction (LVNC), sometimes referred to as spongy myocardium or hypertribeculation syndrome, is a rare myocardial disorder and possible cause of cardiomyopathy. While different echocardiographic criteria for LVNC are used, LVNC is generally defined by the presence of a thickened left ventricular wall with two distinct layers, one thin and one thickened with significant trabeculations in which doppler demonstrates flow. Cardiac MRI is also used in diagnosing LVNC, though no gold-standard diagnostic guidelines are agreed upon. Whether LVNC is a distinct cardiomyopathy versus a phenotype or shared morphologic trait is also debated. Furthermore, left ventricular hypertribeculation can been seen as a result of remodeling in athletes and pregnant patients, or hypertrophied left ventricles from any etiology, mimicking LVNC, and further complicating diagnosis.

Case: A 24-year-old active, healthy male presented to medical attention with exertional chest pain. He has a family history of sudden cardiac death, including in first degree relatives in their 20s. Echocardiogram showed ejection fraction 45% with increase trabeculation in the left ventricle apex, suggesting noncompaction. Cardiac MRI confirmed increased trabeculation at the cardiac apex with approximate noncompacted: compacted myocardial ratio of 3:1, suggesting LVNC, and LVEF of 43%. Left heart catheterization showed no coronary artery disease. Although exercise stress testing revealed no ischemia, there was evidence of impaired exercise capacity. His peak VO2 was 31.4 ml/kg/min which is 67% of predicted age. He was managed with a beta blocker and ARB, later switched to an ARNI, at maximum tolerated doses. He remained without symptoms of heart failure. Echocardiogram after about 6 months of therapy showed improvement in ejection fraction to 57%.

Discussion: This case highlights a rare and incompletely understood cardiomyopathy or left ventricle phenotype. Whether by echocardiogram, cardiac MRI, or other modalities, diagnostic criteria are debated, and many are thought to now overestimate LVNC. LVNC is managed according to its presenting complication, which when present, most commonly include cardiomyopathy, cardiac arrythmias, and systemic embolic events. While LVNC can certainly be pathologic, LVNC can also result from physiologic remodeling in response to certain left ventricular stressors. With unclear diagnostic criteria and variance between pathologic and physiologic LVNC, there is a risk for over-treatment or over-monitoring.
Acute Rheumatic Fever in a Lifelong Michigan Resident

Acute rheumatic fever is rare in the United States, but it can still occur in certain marginalized populations. It is important to recognize and diagnose this condition because of its progression to rheumatic heart disease.

We present a case of a 31-year-old male who presented to the emergency department with symptoms of chills, night sweats, decreased appetite, and fatigue for 1 week. He reported no sick contacts, no recent travel, and had always lived in Michigan. Upon examination, the patient was febrile (>38.5°C), tachycardic, and hypotensive. The physical exam revealed posterior oropharyngeal erythema and a grade I/VI systolic murmur best appreciated at the tricuspid listening post. Lab tests showed leukocytosis to 17.3, troponins elevated to 565, BNP elevated to 1409, erythrocyte sedimentation rate (ESR) elevated to 68, and c-reactive protein (CRP) elevated to 24.1. An EKG showed non-specific ST segment and T wave changes, but no signs of ischemia. Due to oropharyngeal erythema, a rapid streptococcal antigen test was performed and was positive, so the patient was treated with penicillin. A transthoracic echocardiogram (TTE) was done and showed an ejection fraction of 50% with mild global hypokinesis, mildly reduced right ventricular systolic function, moderate mitral regurgitation, moderate to severe tricuspid regurgitation, and a small pericardial effusion. A cardiac MRI was done which showed pericardial effusion and signs suggestive of myocarditis. Given positive rapid streptococcal antigen test, TTE, and cardiac MRI findings, streptolysin-O antibodies were drawn and were noted to be elevated. Based on elevated streptolysin-O antibodies, findings of myocarditis, fever >38.5 C, elevated ESR and CRP, the patient met the revised Jones Criteria set by the American College of Cardiology, for diagnosis of acute rheumatic fever and associated rheumatic heart disease. Treatment was initiated with penicillin and the patient was recommended to get prophylactic monthly penicillin injections until the age of 41. On discharge, close follow-up with infectious disease and cardiology was arranged.

Diagnosing acute rheumatic fever in the United States requires a high index of suspicion due to its low prevalence. The diagnosis can be missed in favor of more common infectious etiologies or causes of troponin elevation. This patient likely had prior episodes of undiagnosed acute rheumatic fever leading to the underlying cardiac sequelae. This case illustrates the importance of early diagnosis and treatment of acute rheumatic fever to prevent progression to rheumatic heart disease.
A Curious Case of Recurrent Candida Vaginitis

Background: Recurrent vulvovaginal candidiasis (RVVC) is defined by three or more episodes of symptomatic infection within one year. Pathology of relapse is not well understood but it is hypothesized that certain intrinsic factors such as endogenous vaginal flora or altered immune response and extrinsic factors such as antibiotic use, sexual activity, or previous bacterial vaginosis infection contribute to the disease process. Vaginal cultures should be obtained to test for non-albicans Candida species. Initial treatment for RVVC includes 10-14 days of induction therapy (topical or oral fluconazole) followed by low dose fluconazole for 6 months.

Objective: Understand role of culture and sensitivities in recurrent Candida vulvovaginitis

Case Report: 30-year-old healthy female with a past medical history of bacterial vaginosis and RVVC presents to Gynecology clinic for increased vaginal discharge and burning sensation for several days. For the past 4 months, patient has presented multiple times for similar symptoms and repeat vaginal cultures revealed Candida albicans infection. She was treated with repeated rounds of treatment including fluconazole, miconazole and tetraconazole with only temporary relief in symptoms for a few days. Physical exam is significant for cottage cheese-like vaginal discharge with associated redness and irritation of the introitus and no evidence of cervical motion tenderness.

Decision-making: Patient was referred to the Infectious Disease clinic for further evaluation. She tested negative for syphilis, gonorrhea, and chlamydia. Ultimately her vaginosis screen revealed presence of yeast on wet mount. Vaginal fluid culture with sensitivities were obtained the demonstrated Fluconazole resistant Candida albicans. Patient was initiated on voriconazole therapy for treatment of fluconazole resistant candida vulvovaginitis. However, due to lack of response, she was subsequently initiated on nystatin therapy and referred to a specialty vaginitis clinic.

Conclusion: We present a case of fluconazole resistant Candida albicans in a woman with RVVC. Clinicians should maintain a high index of suspicion in individuals with refractory RVVC despite appropriate azole treatment. There is an increase in rates of azole-resistance which can make RVVC difficult to manage. Resistance may develop in individuals with repeated or prolonged use of fluconazole and recent bacterial vaginosis infection, as seen in this patient. Therefore, vaginal culture with sensitivities should be obtained early on in these individuals. Patients can be managed with dosing azoles based on minimum inhibitor concentration (MIC) or alternative therapies including nystatin and ibrexafungerp.
Staphylococcus aureus infection-related glomerulonephritis with dominant IgA and C3 deposition: case report and review of literature

Two distinct types should be considered when evaluating bacterial infections and the development of glomerulonephritis (GN): postinfectious GN, which arises weeks after infection resolution and is mostly Staphylococcus infection-mediated GN (SIAGN), and GN associated with current infection, mainly immunoglobulin A (IgA)-dominant SIAGN.

IgA primarily exists in mucous secretions and plays a critical role in establishing the microbiome, tolerating benign environmental pathogens, and locally defending against infections. One of the main causes of renal failure and glomerulonephritis is IgA nephropathy. Hematuria and renal failure result from immune-mediated basement membrane damage. Several environmental and genetic variables affect an individual’s susceptibility to IgA nephropathy. A kidney biopsy is usually diagnostic for IgA nephropathy.

We present a 71-year-old male with a past medical history of essential hypertension and atrial fibrillation on Xarelto who was admitted for acute kidney injury and hematuria. The patient developed left lower extremity cellulitis and was receiving antibiotics. Labs showed creatinine of 6.5 mg/dL with a basal serum creatinine of 1.1 mg/dL, C3 of 107 mg/dL, C4 of 41 mg/dL, many urine RBCs and proteinuria, and staph aureus isolation in the urine culture. Therefore, a CT-guided kidney biopsy was obtained and showed focal proliferative and exudative glomerulonephritis with IgA and C3 co-dominant deposits, mostly IgA nephropathy, given clinical correlation. Glomeruli immunofluorescence revealed diffuse global granular mesangial staining for IgA, C3, kappa, and lambda. He underwent a brief course of dialysis and was treated with a pulse dose of steroids in the hospital, followed by a slow taper. A combination of beta-blockers and calcium channel blockers was used to manage blood pressure. At the last follow-up, his serum creatinine value was stable at 1.6 mg/dL.

Appropriate supportive care and treatments that lower blood pressure, reduce lifestyle-associated risks, and reduce proteinuria help reduce kidney injury, which is a key component of therapy. Blood pressure control with dual RAS inhibitors has been used in treating glomerulonephritis disease; however, the beneficence of using RAS inhibitors in IgA nephropathy is unclear. The usefulness of immunosuppression is still controversial, given that corticosteroid side effects significantly increase with diminished GFR. There is little evidence to support the use of immunosuppressive medications other than corticosteroids, except in patients of Asian origin, where mycophenolate mofetil is commonly used.

When evaluating acute kidney injury or glomerulonephritis in the setting of concomitant infection, a high suspicion for this unusual disease should be raised, and a histological examination confirmation should be performed.
A Rare Case of Mantle Cell Lymphoma Involving the Gallbladder

CASE DESCRIPTION

This case report details the presentation and management of a 78-year-old male with a past medical history, including monoclonal gammopathy of undetermined significance (MGUS), coronary artery disease, chronic kidney disease, hyperlipidemia, and type 2 diabetes mellitus, who presented to the emergency department with 6 months of abdominal pain, weight loss, and abnormal laboratory findings, including a corrected calcium level of 14.6. The initial symptoms prompted further investigation, CT abdomen showed splenomegaly, multiple enlarged lymph nodes visualized in lower thorax, abdomen and pelvis as well as groin consistent with lymphoma. EGD showed one non-bleeding cratered ulcer in the duodenal bulb, the lesion was 25 mm in largest dimension. Biopsies were taken, and pathological diagnosis was duodenal mucosa with extensive atypical lymphoid infiltrate, consistent with mantle cell lymphoma, classic morphology, proliferation index of 30-40% by MIB1 immunostain. Ultrasound showed Cholelithiasis with a prominent gallbladder wall, and a solid mass measuring 2.5 x 1.7 cm in the gallbladder without evidence of biliary ductal dilation. PET CT showed Multiple enlarged lymph nodes in the neck, thorax, abdomen, groin, and splenomegaly consistent with lymphoma. Immunohistochemical stains showed the atypical lymphoid cells are positive for CD20, PAX5, CD5, BCL-1, SOX11, CD43, and BCL-2. In summary, the above findings are diagnostic of mantle cell lymphoma, classic morphology, and 30-40% proliferation index by MIB1. It was negative for TP53 targeted regions. Flow cytometry was positive for a population of CD5+/CD200-neg monotypic B-cells (46.5% of total events and 83.9% of total lymphocytes). In conjunction with the patient's recently diagnosed mantle cell lymphoma (AS238545), the immunophenotype is compatible with peripheral blood involvement by mantle cell lymphoma. Thus revealing lymphoma involvement of the duodenum, multiple lymphadenopathy, gallbladder, and peripheral blood. The hypercalcemia is due to increased hydroxylation of vitamin D secondary to lymphoma. Given the patient's age and comorbidities, high-intensity chemotherapy was deemed unsuitable. The proposed treatment plan involved rituximab/lenalidomide with the potential consideration of Bruton's tyrosine kinase (BTK) inhibitor as a second-line option. The patient received pamidronate for hypercalcemia, showing improvement with hydration. Monthly outpatient bisphosphonate was recommended.

DISCUSSION

Mantle cell lymphoma frequently involves the gastrointestinal tract and further involvement of the biliary system is exceedingly rare. There are no standard guidelines but in patients presenting with gastrointestinal symptoms, endoscopy must be pursued, and biopsies must be taken even in the regions of normal mucosa to exclude mantle cell lymphoma. Generally, advanced symptomatic patients undergo chemoimmunotherapy. Common immunotherapy regimens include R-CHOP (Rituximab, cyclophosphamide, doxorubicin, vincristine, prednisolone), R-DHAP, BR, and R-BAC. Reporting atypical presentations of mantle cell lymphoma can assist in the diagnosis and management of such patients.
A Rare Case of Spontaneous Recurrent Pneumoperitoneum Attributed to Jejunal Diverticular Micro-perforation

INTRODUCTION: While most pneumoperitoneum cases arise from visceral perforations necessitating emergent surgery, approximately 5%-15% result from non-surgical causes. Among these, small bowel diverticular micro-perforations are notably rare, with jejunoileal diverticulosis occurring in only 1%-2% of cases posing significant diagnostic and management challenges.

CASE: We present the case of an 86-year-old female with a medical history of GERD, chronic idiopathic constipation and a surgical history of colectomy and AAA repair. She presented to the ED with acute abdominal pain and bloating without associated emesis or diarrhea. Her vital signs were stable and the examination revealed generalized abdominal tenderness and significant tympany. Laboratory tests were unremarkable. A CT scan of the abdomen and pelvis showed jejunal pneumoperitoneum without evidence of perforation. Given the benign nature of her symptoms, conservative management was initiated. She had two readmissions within the same year for similar symptoms and received similar management each time. Later, she underwent an outpatient Barium small-bowel follow-through which revealed extensive jejunoileal diverticula throughout the entire length. Micro-perforations in these diverticula, likely caused by underlying untreated constipation were suspected to be the cause of her recurrent episodes. She was prescribed a regular laxative regimen to manage the constipation and prevent further episodes.

DISCUSSION: Spontaneous pneumoperitoneum, also known as misleading, idiopathic or non-surgical pneumoperitoneum (NSP), is a challenging diagnosis often overlooked. Micro-perforations from jejunal diverticula, while rare, should be considered when jejunal pneumoperitoneum is detected. Barium studies can prove beneficial for establishing this diagnosis. The presence of pneumoperitoneum without signs of peritonitis must raise high degree of clinical suspicion for NSP. Conservative management involving serial abdominal exams and prophylactic antibiotics is the recommended approach. Addressing preventable factors like constipation is pivotal in reducing recurrent presentations and improving patient outcomes. This case highlights the importance of suspecting NSP in patients with pneumoperitoneum but no evidence of perforation, thereby avoiding unnecessary surgical interventions and focusing on tailored conservative management strategies.
**I Can Hear You But Can’t See You. A Case of Endophthalmitis**

Introduction: Endophthalmitis is an eye infection that results from bacterial, viral, fungal, or parasitic infection. Most cases are exogenous and occur after penetrating eye trauma, following eye surgery, or as an extension of corneal disease. An increasing number of instances arise after intraocular injections with anti-vascular endothelial growth factor (VEGF) medications. Other infections are acquired endogenously from hematogenous seeding from another location.

Case: This abstract highlights a case of endophthalmitis diagnosed in an 85-year-old female who presented to our community hospital to be evaluated for concerns of three days of progressively worsening fatigue and weakness. Evaluation in the ED revealed that she was septic with no apparent source of infection. She was then admitted overnight for treatment with antibiotics. Throughout the night, her vision continued to deteriorate to a point that she could hardly see. The provider caring for her performed a physical exam of her eyes the following day. A physical examination of her eyes revealed erythema of bilateral conjunctiva and yellowish-green drainage from her eyes. Her blood cultures collected on presentation to the Emergency Department were positive for group G streptococcus. Given these findings, there was increasing suspicion that the patient had endophthalmitis, and this may have been the source of the sepsis. Given our community hospital’s lack of ophthalmology services, arrangements were made to transfer the patient to a tertiary care facility with in-house ophthalmology services. A diagnosis of endophthalmitis was made at the tertiary care facility. The patient received an anterior chamber tap and intravitreal injection of vancomycin, ceftazidime, and dexamethasone bilaterally. Cultures collected from the left eye grew streptococcus G. She was then taken to the Operating room (OR) for anterior chamber (AC) washout and intravitreal injections with vancomycin, ceftazidime, and dexamethasone. She was discharged to a subacute rehab facility after she regained some of her vision.

Discussion: Endophthalmitis is an uncommon but sight-threatening eye condition that can occur at any age and in either sex via the injection of the eyes by pathogens spread through the bloodstream. Our patient had poor eyesight from underlying macular degeneration, but she was still fully functional. Other etiology, like strokes, was not as high on the differential as she lacked any new focal neurological deficits. With the patient exhibiting sepsis with no apparent source of infection and now an acute worsening of her vision and physical exam findings worrisome for an eye infection, endophthalmitis was the working differential. This led to an escalation of care by transferring the patient to a tertiary care facility with ophthalmology care.

Conclusion: Acute endophthalmitis is a medical emergency. The most critical treatment component is the intravitreal injection of antibiotics and vitrectomy in severe cases. Systemic antibiotics should be utilized in cases of endogenous bacterial and fungal endophthalmitis. Repeated intravitreal injections may be necessary if there is no response to systemic antibiotic therapy. Prompt treatment is needed to avoid complete blindness and full vision restoration.
A rare cause of vitamin B12 deficiency

Presentation:
A 31-year-old male with a history of polysubstance use and bipolar disorder presented with a 1-week history of ambulatory difficulty, trouble holding objects, loss of balance, and mild paresthesias in the fingers and toes. The physical examination further revealed slow fine coordinated movements (finger tapping) bilaterally, significant dysmetria on finger to nose and heel to shin, unsteady gait, asymmetric bilateral upper and lower extremity weakness, and no achilles reflex bilaterally. Blood work shows hemoglobin of 13.3 with MCV 93.2, Vitamin b-12 level <150 pg/ml (232 - 1245 pg/mL), homocysteine >44umol/L (3-14umol/L), and methylmalonic acid 1.22umol/L (<0.4umol/L), UDS only positive for benzodiazepines. MRI spine shows vague and symmetric T2 signal hyperintensity within the dorsal columns of the spinal cord as noted in the cervical spine that is in keeping with underlying subacute combined degeneration.

Detailed history, workup, and imaging further ruled out autoimmune disease, malabsorption, infection, trauma, vascular disease, and metabolic causes of vitamin b-12 deficiency as well as any other possible synergic cause of presentation. Upon interviewing multiple times on the latter days of admission, the patient did admit that he was using the whippet pretty heavily or at least daily before presentation to the hospital. That is the inhalation of nitrous oxide.

The patient was treated with a high dose of IV vitamin B12 and was discharged from the hospital, on out-patient follow-up after a year the patient recovered completely with abstinence from NO inhalation, B12 supplements, and physiotherapy.

Discussion
Subacute combined degeneration of the dorsal (posterior) and lateral columns (white matter) of the spinal cord due to demyelination is the classic neurologic presentation of vitamin B12 deficiency that commonly presents with progressive weakness, ataxia, and paresthesias. Identifying the etiologies of vitamin B12 can sometimes be challenging for an internist. Among the rare causes of vitamin B12, NO poisoning is one of the established causes. Neurologic symptoms associated with nitrous oxide use have been attributed to vitamin B12 deficiency. Through oxidation, nitrous oxide inactivates vitamin B12.

The neurological symptoms of vitamin B12 deficiency are unspecific and can be irreversible. Early detection is therefore important, using the most sensitive and specific markers available. Individuals can present with increased levels of homocysteine and methylmalonic acid in addition to low vitamin B12 levels. Classic MRI findings associated with nitrous oxide-induced vitamin B12 deficiency include T2 hyperintensity in the posterior columns, usually over several vertebral segments, with or without involvement of the lateral corticospinal tracts. These findings represent myelin sheath degeneration. Treatment should be with IV vitamin b12 supplement and response is mainly monitored with symptomatic improvement however can also be monitored with serum levels of vitamin b12, homocysteine, and methylmalonic acid levels.

Conclusion
There is no available test to screen for nitrous oxide use. Therefore it is vital to get a detailed history regarding illicit drug use in every patient and specifically about nitrous oxide use in those presenting with neurologic complaints. Identifying the cause and earlier treatment of vitamin B12 deficiency is very important to prevent irreversible loss and possible complete recovery.
Temporal Trends in Alcoholic Cardiomyopathy-Related Mortality Rates from 2000 to 2020 - An Analysis of Epidemiology Disparities in the United States

Background: Alcoholic cardiomyopathy (ACM) is one of the major causes of mortality in the United States. This study examines ACM-related mortality (ACMRM) trends from 2000 to 2020, using age-adjusted mortality rates (AAMR) to assess disparities among key demographic and geographic subsets.

Methods: We conducted an extensive cross-sectional analysis using the Centers for Disease Control and Prevention Wide-Ranging Online Data for Epidemiological Research database. AAMR rates per 1,000,000 were calculated, and annual percent changes with 95% confidence intervals were determined. Using Joinpoint regression analysis, we assessed changes in the overall trend and within specific demographic (age, gender, race, urban/rural) and regional groups.

Results: From 2000 to 2020, 12,961 deaths were attributed to ACM. High-risk groups included men, African Americans, individuals aged 55-74, and rural residents. AAMR for ACMRM increased from 1.50 per 1,000,000 in 2000 to 2.22 in 2020. The overall trend was stable from 2000 to 2005. Subsequently, it peaked with AAMR of 2.50 in 2007 before declining to 1.64 in 2011, and has steadily risen over the past decade.

Conclusion: This study highlights a rising trend in ACM-related mortality in the United States from 2000 to 2020, emphasizing the highest-risk groups. Targeted interventions are essential to mitigate the burden of ACMRM.
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Frigid Resolve: Self-Induced Target Temperature Management in Cardiac Arrest Patient - A Case Study

The role of Targeted temperature management (TTM) in post-cardiac arrest care is controversial. The underlying principle of TTM lies in its ability to attenuate brain injury by decelerating cerebral metabolism at a rate of 6 to 7% per 1°C, cerebral blood flow, intracranial pressure, cellular apoptosis, and oxidative stress. The Practice Changing Trials in 2002 reported the beneficial role of hypothermia in mitigating neurological recovery and was posited to reshape intensive care standards, however, subsequent investigations with larger sample sizes challenged the superiority of hypothermia over normothermia in out-of-hospital cardiac arrest patients. The timing of TTM initiation from hospital admission significantly impacts clinical outcomes, as shown in a study where early TTM (within 122 minutes) led to increased survival to hospital discharge and better neurological outcomes, particularly in cases of shockable rhythms. We report the case of an out-of-hospital cardiac arrest in low ambient temperature with favorable neurological recovery.

A 59-year-old male with a significant medical history including alcohol use disorder and hypertension, was discovered unresponsive in the snow during subzero temperatures of -1 °F by a bystander who found him pulseless and commenced CPR. On arrival of EMS, he was found to be in Pulseless Electrical Activity (PEA), given epinephrine, converting to Ventricular Fibrillation (VF), and was defibrillated with Return of Spontaneous Circulation (ROSC) before losing pulses again. He was intubated in the field, administered amiodarone and CPR carried out using a Lucas Machine en route to the hospital.

On arrival to the hospital, he was in refractory VF and received procainamide, lidocaine, and additional defibrillation per the ACLS protocol. His initial body temperature was recorded as 25°C. A Bair Hugger device and warmed intravenous fluids were initiated, and a left-sided resuscitative thoracostomy was completed for warm fluid lavage. Despite extensive rewarming efforts and continued ACLS for 45 minutes without ROSC, and bedside echo confirming cardiac standstill, ACLS was aborted. Unexpectedly several minutes later an organized rhythm was noted on the cardiac monitor. Bedside ultrasound confirmed weak cardiac activity, leading to the resumption of rewarming efforts and vasopressor support. He was transferred to the ICU for post arrest care. After achieving a body temperature of 32.1°C, the patient was transitioned from aggressive rewarming to targeted temperature management (TTM) protocol with a target temperature of 32-34°C.

Computed Tomography (CT) did not show any evidence of anoxic brain injury but did note a grade 3 liver laceration. Over the course of the next several days, he developed multiorgan failure including ischemic hepatitis and renal failure requiring the initiation of continuous renal replacement therapy. The patient was ultimately weaned off sedation and followed commands, tolerating spontaneous breathing trials, and was ultimately extubated 6 days after his cardiac arrest. He had recovery of his neurological function with preserved speech and mobility.

Our case demonstrates the remarkable neurological outcomes in an out of hospital cardiac arrest with an unknown downtime in the setting of hypothermia as a surrogate for TTM.
Rare Listeria Rhombencephalitis with Brain Abscess: Diagnostic Challenges and Successful Management in a Complex Case

Introduction

Listeria monocytogenes is an opportunistic infection that can lead to listeriosis manifesting as meningoencephalitis, rhombencephalitis, and brain abscess. We present a rare case of listeria rhombencephalitis with brain abscess, of which only a few cases have been reported so far with a high mortality rate.

Case

A 46-year-old male with a history of atypical meningioma at the pineal cistern presented with slurred speech and nausea. The patient underwent subtotal resection one year prior, followed by radiation and ventriculoperitoneal (VP) shunt placement six months prior to presentation. He was initially treated with Decadron and Keppra for suspected radiation necrosis based on the MRI brain findings of hyperintense lesions in bilateral thalamic regions. The patient had an episode of fever initially and blood cultures were negative. Therefore, empiric antibiotics were held. Patient continued to experience worsening confusion, dysarthria, right hemiparesis, and persistent fever, followed by progressive lethargy requiring intubation. Repeat MRI brain revealed enhancing lesions consistent with abscesses in the bilateral thalami as well as in the cerebellum and midbrain. The patient’s family later revealed his consumption of soft cheese several days before the presentation. Repeat blood cultures and CSF cultures from the VP shunt were positive for listeria monocytogenes. He was treated with ampicillin for 8 weeks and gentamicin for 3 weeks, clearing CSF cultures on day 5. Repeat MRI brain showed improvement in brain lesions. The patient underwent externalization of VP shunt. Subsequent MRIs brain showed improvement in the ring-enhancing lesions. Despite requiring a tracheostomy and PEG tube, the patient clinically improved and was discharged to an LTAC facility.

Discussion

Listeria monocytogenes is the main cause of rhombencephalitis, an inflammatory brain stem and cerebellum disease. Neurolisteriosis, presenting as a brain abscess, comprises only 2% of cases, with even rarer occurrences in the brainstem. Listeria rhombencephalitis is observed in 17% of patients, yielding a 51% mortality rate despite proper antibiotic therapy due to the challenging diagnosis and rapid disease progression. Common risk factors include ages <2 and >50, malignancies, immunosuppression, AIDS, and pregnancy. Rhombencephalitis manifests with a non-specific prodrome of fever, neck stiffness, and headache, followed by cranial nerve palsies. CSF studies remain the mainstay of diagnosis, demonstrating pleocytosis, high protein, and low glucose, though CSF can be normal. Polymerase chain reaction (PCR) and enzyme-linked immunoassay (ELISA) are equally sensitive. Next-generation sequencing can also aid in the rapid diagnosis of listeria rhombencephalitis because blood and CSF cultures tend to be positive in only 61% and 11% of cases, respectively. MRI brain reveals leptomeningeal enhancement. Treatment consists of ampicillin in combination with aminoglycosides (usually gentamicin). Rifampin is also used, given its ability to cross the blood-brain barrier. Treatment duration can be up to 6 to 8 weeks. Listeria rhombencephalitis should be suspected in immunocompromised hosts who present with neurologic deficits, particularly in those with exposure to unpasteurized dairy products. CSF studies and MRI brain should be pursued promptly, and empiric antibiotics should be started immediately in patients with high clinical suspicion to prevent mortality.
**A rare case of coccidioidomycosis-induced acute pericarditis**

Coccidioidomycosis is a fungal infection caused by Coccidioides Immittis and Coccidioides Posadasii, acquired by inhalation of fungal spores in the air. It is observed in the Southwest United States specifically in California, Texas, Arizona, New Mexico, Nevada, Utah, and Central and South America. This a case of a young immunocompetent adult who initially presented with acute pericarditis and was subsequently diagnosed with Coccidioidomycosis.

A 36-year-old male patient presented to the ED for evaluation of intermittent sub-sternal chest pain lasting 1-2 hours radiating to his back. Chest pain was worse with deep inspiration and lying flat, and improved with sitting up. The patient also had fever, chills, diaphoresis, and myalgias. The patient is a truck driver and recently returned from Texas.

Vital signs included a temperature of 101.9 F, Pulse 124 BPM, and Respiratory Rate 26. Labs were notable for leukocytosis of 14.2, Troponin peaked at 528. Electrocardiogram showed sinus tachycardia with possible pericarditis and diffuse ST segment elevation in leads I, II, aVF, V4-V6, and PR depression in leads I, II, and aVF. Chest X-ray showed right lower lobe consolidation. The echocardiogram showed an ejection fraction of 41%, with hypokinesis of the entire left ventricle wall. CT chest dissection without contrast showed bulky right hilar and mediastinal lymphadenopathy. Infectious disease workup was negative including respiratory virus panel, fungal studies, and two negative TB PCR Tests.

The patient was initiated on Unasyn and Doxycycline for pneumonia, and colchicine and indomethacin for acute pericarditis. He showed significant improvement regarding his symptoms.

The patient underwent bronchoscopy with FNA of one of the mediastinal lymph nodes that showed no immunophenotypic evidence of B-lymphoid neoplasia. Cytopathology showed atypical lymphoid tissue and granulomatous inflammation, however fungal stains were negative. PET-CT scan showed hyper-metabolic right lower lobe opacity with multiple hyper-metabolic right hilar and hyper-metabolic mediastinal lymph nodes.

After discharge, the patient tested positive for Coccidioides IgM, then seroconverted to IgG. Repeat antibody testing with complement fixation showed negative antigens, however seroconversion was noted. The results were consistent with true infection with Coccidioides. A repeat echocardiogram showed an ejection fraction of 59%, with no evidence of regional wall motion abnormality or pericardial effusion.

The patient reported improvement of symptoms with resolution of chest pain, fevers, fatigue, and body aches. Antifungal therapy was deferred at the time as it did not prevent further dissemination, and given the patient’s marked clinical improvement, routine monitoring was only warranted.

Coccidioidomycosis-induced pericarditis is extremely rare with only two reported cases in the USA from the literature review within the last 10 years. Adding to its rarity, diagnosis of coccidioides infection can be challenging. Relying solely on one diagnostic modality may result in a missed diagnosis. In our case, despite the absence of positive antigens in complement fixation testing, the detection of Coccidioides IgM antibodies followed by Seroconversion to IgG strongly supported the diagnosis. Our case also underscores the importance of maintaining broad differentials and considering fungal infections as a potential cause of acute pericarditis in patients originating from endemic areas.
A rare case of streptococcus pyogenes meningitis in a healthy female after a lumbar epidural steroid injection

Introduction:

Streptococcus pyogenes (GAS) is an atypical and rarely reported cause of bacterial meningitis, especially in adults. Streptococcus pyogenes is well known for causing a spectrum of diseases ranging from tonsillopharyngitis to severe invasive diseases like necrotizing fasciitis and STSS, however, rarely causing bacterial meningitis. Although rare, GAS meningitis is associated with a high case fatality rate and can result in severe neurological complications. Streptococcus pyogenes is universally susceptible to all beta lactam antibiotics, with no published reports of resistance to date. Therefore, majority of individuals respond to penicillin G as a single agent. We present a rare case of Streptococcus pyogenes causing meningitis in an adult following Lumbar Epidural Steroid injection.

Case presentation:

A 66 y.o. Caucasian female with a history of arthritis, hypertension, hyperlipidemia was admitted with headache and rigors for 5 days. A week prior, patient received a Lumbar epidural steroid injection for chronic back pain. Two days later, patient developed new onset headache that she initially attributed to the injection. In next few days headache intensified and was also associated with nausea, neck pain, dizziness and photophobia.

Patient was in acute distress and toxic appearing, however alert and oriented. Patient was febrile at 103.1°F and HR 117 bpm. Physical examination revealed positive Nuchal rigidity, Brudzinski’s and Kerning sign. Otherwise, no focal neurologic signs were noted. Laboratory tests showed leukocytosis of 23.9, CT head without IV contrast showed no acute intracranial hemorrhage or mass effect. Ctx lumber spine without IV Contrast was negative for any infection. Patient was empirically treated with antibiotics including ceftriaxone, vancomycin, ampicillin, acyclovir and dexamethasone. Lumbar Puncture with CSF analysis was compatible with bacterial meningitis. CSF Gram stain showed gram positive cocci in pairs and subsequently grew many streptococcus pyogenes (Group A). Blood cultures were negative. Antibiotics were de-escalated to IV Penicillin G for 14 days. Patient responded well to treatment with remission of fever, resolution of meningeal signs and symptoms, normalization of leukocyte count. Patient was discharged home to complete 14-day course of IV Penicillin G via Midline. Patient did not develop any neurological complications.

Discussion:

The incidence of acute bacterial meningitis is 5–10/100,000 persons per year in high income countries, resulting in 15,000–25,000 cases in the US annually. Most common pathogens include Streptococcus pneumoniae (72%) and Neisseria meningitidis (11%) in people older than 16 years. Escherichia coli and Streptococcus agalactiae cause about 35% of cases of early-onset neonatal meningitis. GAS accounts for less than 1% of cases of bacterial meningitis.

Our case was unique because GAS meningitis is extremely rare in healthy adults. Previously reported GAS meningitis cases are limited to children, elderly and immunocompromised people, often associated with recent upper respiratory tract infections. This emphasizes that GAS can cause meningitis in healthy adults without recognizable foci of infection. Adding an extra layer to rarity, our case developed GAS meningitis after receiving lumbar epidural steroid injection showcasing the importance of unconventional routes of spread of infection.
A report of Pleural Tuberculosis in a recent immigrant: a Diagnostic Dilemma

Introduction:

Pleural tuberculosis (TB) poses a significant diagnostic challenge due to its diverse clinical presentation and limitations in available diagnostic tools, accounting for approximately 20% of extrapulmonary cases in the United States. With TB associated pleural effusion thought to be as a result of hypersensitivity response to mycobacterial antigens rather than acute infectious process in the pleural space, further hindering diagnosis. We explore the intricacies of identifying pleural TB by highlighting the unique challenges encountered by health providers to help refine diagnostic strategies and improve patient outcomes in the ongoing global effort against tuberculosis.

Case presentation:

We present the case of a 20-year-old male, a recent immigrant from South America with no significant medical history, who sought medical attention due to a 1-week history of fevers, chills, night sweats, dry cough, and right-sided pleuritic chest pain.

Upon presentation, the patient displayed low-grade fevers and a small right-sided pleural effusion with adjacent infiltrates on chest X-ray. CT imaging failed to identify overt parenchymal consolidation, Ghon's complex or pleural thickening. Initial empiric coverage for community-acquired pneumonia (CAP) was initiated based on pleural fluid studies consistent with an uncomplicated exudative effusion, which later showed no growth on cultures. Subsequent imaging days later revealed worsening of the effusion with the development of a loculated component. Due to the patient's recent travel history, interferon-gamma release assay was ordered which returned positive. Patient was placed on airborne precautions however respiratory cultures were unable to be obtained due to lack of phlegm production. Bronchoscopy with bronchoalveolar lavage yielded negative results, including both cultures and Mycobacterium tuberculosis (MTB) PCR. IV antibiotics were discontinued pending further TB workup, with Video-assisted thoracic surgery subsequently performed which revealed pleural thickening with a diffusely studded appearance and firm adhesions of the right lower lobe to adjacent pleura and diaphragm. Decortication was deferred at that time due to the absence of a definitive diagnosis, with pleural biopsies obtained later demonstrating fibrinous pleuritis with non-necrotizing granulomas, however failing to demonstrate acid-fast bacilli. After a prolonged hospital stay and extensive dialog between a multidisciplinary team and the Health department, the patient was initiated on RIPE therapy (Rifampin + Isoniazid + Pyrazinamide + Ethambutol) for empiric treatment of acute pleural TB.

Conclusion:

Given the low microbial burden present in MTB infections, this case emphasizes the diagnostic difficulties associated with pleural tuberculosis. Despite inconclusive findings and diagnostic uncertainty, a cautious clinical approach guided by a multidisciplinary team resulted in initiating empiric RIPE therapy, hence underscoring the essential need for ongoing studies for improving diagnostic approaches for evaluating this insidious condition.
Hereditary hypomagnesemia and recurrent nephrolithiasis

Introduction: The body’s most common intracellular divalent cation, magnesium, is a co-factor or activator in nearly 800 enzymatic reactions. Despite its abundance, the electrolyte is deficient in around 12% of hospitalized patients and occasionally, a gene mutation affecting its metabolism is responsible. This case report brings to light, a rare hereditary defect causing renal magnesium loss, its unconventional presentation and diagnostic challenges.

Case description: A middle-aged female of Danish descent presented with episodes of palpitations and recurrent kidney stones since the age of 35 years. Her vitals were stable, ECG noted QRS widening and lab results showed severely low serum magnesium (< 1 mg/dL), normal serum calcium, serum potassium and serum creatinine. A renal ultrasound and CT abdomen & pelvis demonstrated multiple bilateral non-obstructing 2-5 mm renal calculi. There was no history of smoking and significant alcohol or illicit drug use. Most causes of hypomagnesemia were ruled out. Hence, a 24-hour urine magnesium was obtained which confirmed increased magnesium excretion (> 140 mg). A closer look at her family history revealed several affected male and female relatives, some incidentally diagnosed, others with mild weakness and many who suffered sudden cardiac deaths. Data on consanguinity was not available. At this point, hereditary etiologies for renal magnesium wasting were considered. Eventually, genetic analysis performed in the family established the diagnosis of 11q23 mutation. The magnesium replacement subsequently required for this patient was 3 g/day to maintain her serum magnesium levels between 1.2 and 1.7 mg/dL.

Discussion: A mutation of the FXYD2 gene on 11q23 causes impaired Na-K-ATPase functioning, leading to insufficient transapical membrane potential, and thereby reducing magnesium absorption. It was previously documented as early as 1987, in 1 Belgian and 2 Dutch families with autosomal dominant inheritance and a similar spectrum of presentation ranging from no symptoms to tetany and convulsions. In this case, it was evident that many individuals remained predominantly unaware of their magnesium deficiency. Genetics, environment and lifestyle may have also influenced the magnitude of symptoms. Interestingly, this patient’s presentation was offset by recurrent nephrolithiasis, which is more commonly associated with HOMG3 (CLDN16) and HOMG5 (CLDN19) due to high urine calcium. Probable explanations, as evidenced by past studies, could be broadly related to decreased urinary and intestinal binding of magnesium with oxalate causing hyperoxaluria, stable monohydrate calcium oxalate formation and citrate re-absorption. Although there was no haplotype analysis, this pedigree from Denmark is likely being recorded for the first time.

Conclusion: Magnesium is a highly essential element and any derangement in its metabolism can lead to catastrophic consequences. Hereditary defects causing hypomagnesemia are extremely rare and often undiagnosed. Given the strong likelihood of discovering more similar gene defects, there is scope for broadening our understanding of magnesium homeostasis. A good history and more importantly, awareness of these disorders can significantly aid in the diagnosis and timely management.
Medullary thyroid carcinoma presenting with severe hypokalemia

Introduction: Medullary thyroid carcinoma (MTC) accounts for about 5-7% of all thyroid malignancies. 70% of most medullary thyroid carcinoma are sporadic in nature. The typical age at initial presentation ranges between 41 and 55 years. Aside from signs and symptoms of Cushing's disease, other clinical features included generalized weakness, exertional dyspnea, distal paresthesia, galactorrhea, back or hip pain and pathological fractures with metastases to the neck, mediastinal lymph node, liver, bones and adrenals.

The objective of this case is to bring attention to the symptomatic ectopic ACTH or CRH in the setting of medullary thyroid carcinoma.

Case: Thirty eight-year-old male, with past medical history of hypertension treated with lisinopril, presented to the hospital due to left upper extremity swelling, redness and pain. On arrival, labs were significant for hypokalemia, 2.6 mmol/L and hyperglycemia at 166 mg/dL with normal kidney function. There was no reported history of vomiting, diarrhea or decreased PO intake. He was diagnosed with acute cellulitis and started on appropriate antibiotics. At the same time, aggressive replacement of potassium resulted in only minimal improvement and recurrence of severe hypokalemia prompting further work up. Urine electrolyte panel results were suggestive of renal potassium wasting defect. Aldosterone level was 5 ng/dL and direct renin level was 9.4 pg/ml. Random cortisol level was 69.2 microgram/dl. Dexamethasone suppression test was done which showed elevated cortisol levels confirming ectopic ACTH. Brain MRI revealed no pituitary lesions. Due to lack of improvement in the left arm swelling, the patient underwent CT scan with IV contrast to look for possible obstruction which showed mass in the thyroid region with abdominal lymph nodes, and multiple hepatic lesion. Liver biopsy was done which was consistent with medullary thyroid carcinoma. Hematology oncology service was consulted. Patient was started on metyrapone with improvement of cortisol levels.

Discussion: The diagnosis of ectopic ACTH production secondary to MTC is based on the presence of hypercortisolism not suppressed by high cortisol, absence of pituitary adenoma, and Cushing syndrome symptom. Tertiary hypercortisolism mostly presents with rapid onset of resistant hyperkalemia similar to the presentation of this case. MTC can spread to lymph nodes, and to the lungs, liver and brain via blood. Treatment for ACTH production includes adrenolytic or suppression of steroidogenesis. In extreme cases bilateral adrenalectomy may be needed. Tyrosine kinase inhibitors are used for chemotherapy for MTC. Immunotherapy was found to significantly decrease cortisol and ACTH levels. Due to the high resistance rates of MTC to tyrosine kinase inhibitors, screening for recurrence is essential. Other substances associated with MTC include production of corticotrophin-releasing factor (CRF), serotonin, somatostatin and substance P occurring typically in the presence of distant metastases. Ectopic ACTH, must be considered in the differential diagnosis, especially in the setting of thyroid neoplasm such as MTC, while investigating for severe refractory hypokalemia.
Fatal cardiac tamponade following cardiac ablation procedure.

Introduction: A catheter ablation procedure is an established treatment for symptomatic atrial fibrillation. There are several complications associated with atrial fibrillation ablation, among them cardiac tamponade is a rare yet serious one. It is one of the major causes of mortality associated with surgical procedures.

Case presentation: A 60 year old male with a past medical history of end stage renal disease on hemodialysis, hypotension on midodrine, atrial fibrillation status post ablation a week prior, cerebrovascular accident and anemia presented to the emergency department with a chief complaint of weakness, nausea and vomiting for the past few days. He reported dizziness and a few syncopal episodes. He also reported being confused for the past few days. He denied chest pain, dyspnea and a history of alcohol use.

On arrival at the emergency department, the patient’s blood pressure was 97/65. He was tachycardic at 106. Laboratory evaluation revealed significantly elevated aspartate transaminase at 1370 and alanine transaminase at 705. Gamma-glutamyl transferase was elevated to 60. Troponins were mildly elevated and peaked at 0.360. Electrocardiogram revealed sinus tachycardia. Chest x ray showed low lung volumes with a left retrocardiac opacity and cardiomegaly. The patient had mild right upper quadrant tenderness on exam. Abdominal ultrasound revealed shrunken liver with heterogeneous hepatic echotexture and nodular hepatic contour suggesting cirrhosis. Initial diagnoses included hepatic encephalopathy and he was started on lactulose.

Given the patient's hypotension requiring vasopressor support, elevated troponins and a recent cardiac procedure, an echocardiogram was done, and it revealed an ejection fraction of 55-60%; grade I diastolic dysfunction. It also showed a large posterior pericardial effusion adjacent to the left ventricle. Tamponade was suspected based on worsening hypotension, and the patient was taken for an emergent pericardial window. Intraoperatively, a perforation of the right ventricle was found with a large area of surrounding necrotic and ischemic tissue. There was a significant hemorrhage with opening of pericardium, which could not be controlled due to the large area of non-viable cardiac tissue. The patient went into cardiac arrest with pulseless electrical activity. Open cardiac massage was performed but return of spontaneous circulation could not be achieved and the patient was declared deceased.

Discussion: Pericardial effusion with tamponade requiring intervention is the complication of cardiac ablation associated with highest mortality. The clinical findings in tamponade include hypotension, increased jugular venous pressure, muffled heart sounds, also known as Beck’s triad. However, these may not be reliable indicators for diagnosis. An echocardiogram should be done when tamponade is suspected. The echocardiographic findings of tamponade include a pericardial effusion, diastolic right ventricular collapse, systolic right atrial collapse, a plethoric inferior vena cava with minimal respiratory variation, and an exaggerated respiratory cycle change in mitral and tricuspid valve inflow velocities. A small pericardial effusion can be treated conservatively, but a true tamponade requires immediate medical attention. The treatment for tamponade involves drainage of the effusion with needle pericardiocentesis. In some cases, a catheter drainage or surgical pericardial window may be required to prevent reaccumulation of the effusion.
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**Leadless Pacemaker Dislodgement - A Rare Complication**

**Introduction**

The leadless pacemaker (LPM) is much smaller than a conventional pacemaker which requires no chest incision or subcutaneous generator pocket. LPM has lower rates of complications which are coupled with cosmetic benefits. LPM provides only single-chamber ventricular pacing and lacks defibrillation capacity, hence used for permanent atrial fibrillation with bradycardia or bradycardia-tachycardia syndrome or when infrequent pacing is required. We present a rare complication of dislodgement and successful retrieval of LPM.

**Case Report**

A 90-year-old man with a past medical history of hypertension, coronary artery disease with coronary artery bypass graft, atrial fibrillation and osteoporosis presented to the emergency department with visual changes and confusion. Computed tomography (CT) head and CT angiography of head and neck was remarkable for left occipital lobe ischemic stroke with posterior cerebral artery stenosis for which he was started on dual antiplatelet therapy. He was noted to have atrial flutter with evidence of bradycardia while in the emergency department. He underwent LPM (Micra, Medtronic) placement for sick sinus syndrome with atrial flutter along with episodes of bradycardia. Post-procedure he became hypotensive requiring significant vasopressor support. A large right groin hematoma with ecchymosis was noted. Right leg angiogram showed pseudoaneurysm/arteriovenous fistula at the right sapheno-femoral artery (SFA)/femoral vein for which a covered stent was placed in the right SFA. A day after Micra placement, imaging revealed dislodgement of the LPM in the left pulmonary artery. It was successfully retrieved. Five days later, he underwent placement of a dual-chamber permanent pacemaker implantation.

**Discussion**

Pacemaker dislodgement is a rare complication with incidence of leadless pacemaker dislodgement significantly lower than lead dislodgement with conventional pacemakers. Some reports suggest a 0.13% incidence of Micra dislodgement. Reasons for dislodgement are not well understood. Benefits of retrieval of dislodged pacemakers need further studies. Retrieval of the device after dislodgement being a challenging procedure. One case report showed the dislodged device to cause nonsustained right ventricular tachycardia. Another case report suggested that a dislodged leadless pacemaker might be harmless if it is wedged in a stable manner, but there is also the potential for the device to fall back into the right ventricle if it is not wedged in a pulmonary artery.

**Conclusion**

Dislodgement of a LPM (Micra) into the pulmonary artery is a rarity. Retrieving the device can be challenging given the lack of support in the right ventricular outflow tract. Further studies are needed to better elaborate on LMP retrieval associated pros and cons.
Cardiac-Related death in US Adult Cancer patients: A SEER Based Study

Introduction

Heart disease and cancer rank as the top leading cause of death in the United States and globally and remain the primary barrier to increasing life expectancy. Previous studies have suggested cardiac death in cancer patients can be primarily attributed to cardiotoxicity associated with various anticancer treatments, including chemotherapy, radiotherapy, endocrine therapy, or surgery. We report cancer-specific causes of mortality attributed to heart disease in the USA between 2001-2020 in adults 20 years or older.

Methods

Surveillance, Epidemiology, and End Results (SEER) Program with Incidence-Based Mortality Research Limited Field Data from 22 registries with a subset from November 2022 (2000-2020) was used to identify the incidence of cardiovascular related deaths in cancer-specific mortality from 2001 until 2020 among adult patients 20 years or older. SEER*Stat 8.4.3 was used to obtain rates per 100,000 for the cause of death for different age groups above 19 years of age for common malignancies.

Results

We identified a total of 3,117,595 all cancer specific mortality cases. There were 959,294 cancer deaths from lung and bronchus cancer, 384,556 from colorectal cancer, 291,914 from breast cancer, 246,943 from pancreatic cancer, 233,710 from prostate cancer, 154,397 from lymphoma, 122,983 from the urinary bladder cancer, 119,971 from liver cancer, 109,883 from stomach cancer, 101,049 from kidney and renal pelvis cancer, 89,310 from ovarian cancer, 87,921 from brain cancer, 74,995 from esophageal cancer, 65,945 from melanoma, 33,655 from cervical cancer, 17,217 from thyroid cancer, 17,117 from small intestine cancer and 6,735 from bones and joint cancer.

The heart disease contributing to mortality among cancer patients were 49.18% in prostate cancer, 35.15% in urinary bladder cancer, 34.16% in melanoma, 30.75% in thyroid cancer, 25.23% in breast cancer, 20.68% in kidney and renal pelvis cancer, 19.33% in colorectal cancer, 17.89% in lymphoma, 14% in small, intestinal cancer, 7.18% in stomach cancer, 7.16% in cervical cancer, 6.77% in bones and joint cancer, 5.63% is esophageal cancer, 5.32% in lung and bronchus cancer, 4.09% and ovarian cancer, 3.91% in liver cancer, 2.52% in brain cancer and 2.42% in pancreatic cancer.

Conclusion

By comparing the cardiovascular mortality among 18 different cancers, it can be concluded that prostate cancer has the highest cardiac mortality rate (49.18%) followed by bladder cancer (35.15%) and melanoma (34.16%). Most of the chemotherapeutic agents used in cancer treatment are cardiotoxic, which are thought to increase the cardiovascular related mortality in cancer patients. Literature showed the use of androgen deprivation therapy (ADT) in patients undergoing radical prostatectomy for localized prostate cancer as a possible reason. ADT is thought to be related to its role in promoting atherosclerosis, dyslipidemia, adiposity, and insulin resistance.
A case of pneumothorax secondary to iatrogenic esophageal perforation in a 96 years old male

Introduction:

Nasogastric tube has a key role in the management of hospitalized patients, particularly the critically ill. Significant complications include pneumothorax, subcutaneous emphysema, and respiratory failure. Risk factors for complications include altered mental status, absence of a gag reflex, presence of an endotracheal tube, supine position, and head and neck surgery. We will present a case of iatrogenic pneumothorax secondary to esophageal perforation.

Case presentation:

A 96 year old female with a past medical history of coronary artery disease, hypertension, aortic stenosis, gastroesophageal reflux disease, prior occipital lobe cerebrovascular accident with PEG tube dependence presented to the ED with a PEG tube malfunction, surrounding skin excoriation and pus. PEG tube was successfully removed and the site was sutured under endoscopic guidance. During manual nasogastric feeding tube placement, an iatrogenic esophageal perforation was noted distal to the upper esophageal sphincter muscle that was not amenable to endoscopic closure. Subsequently the patient developed subcutaneous emphysema. Subsequent CT scan noted a large pneumomediastinum extending diffusely into the neck, chest wall, face including the orbit and even peritoneal cavity and left pneumothorax. Trauma surgery was consulted and the patient underwent chest tube insertion with a surgical intervention planned later for perforation repair. After discussion with the family, they decided to change patient code to comfort care only and do not proceed with any intervention.

Discussion:

Iatrogenic pneumothorax is a rare but potential complication of nasogastric tube insertion. Clinicians need to be aware of this risk, especially in unconscious or obtunded patients. Early detection and management are crucial to prevent further complications.
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A case of Platypnea-orthodeoxia syndrome secondary to PFO with reversal of symptoms with Intervention

Introduction:

Platypnea-orthodeoxia syndrome (POS) is a medical condition causing dyspnea, where patients experience breathlessness when standing or sitting, which is relieved by lying down. This is the opposite of orthopnea. POS is characterized by decreased blood oxygen saturation upon changing from a supine to an upright position.

Case Presentation

A 79-year-old female with a significant past medical history of deep Vein Thrombosis/pulmonary embolism on warfarin, a history of breast cancer status post lumpectomy and chemotherapy, hypertension, and CKD presented to the hospital from a living facility with the symptoms of persistent hypoxemia and platypnea-orthodeoxia syndrome. The results of the physical examination and laboratory tests were normal and did not reveal any notable abnormalities or concerns. An echocardiogram revealed severe patent foramen ovale (PFO) with right to left shunt. PFO was closed using the GORE CARDIOFORM septal occluder. After the procedure, the patient’s oxygenation greatly improved, and her symptoms of POS disappeared. She was discharged two days later.

Discussion:

Platypnea-orthodeoxia syndrome (POS) is a rare condition characterized by difficulty breathing and low oxygen levels when standing upright. It is often underreported due to the challenge in diagnosis. POS is typically caused by the mixing of deoxygenated venous blood with arterial blood, facilitated by abnormalities in the heart or lungs. Two mechanisms, intracardiac and intrapulmonary, contribute to this condition. The intracardiac mechanism involves the transfer of deoxygenated blood between atria through defects like patent foramen ovale (PFO). Intrapulmonary mechanism occurs due to various pulmonary abnormalities. In this case, a patent foramen ovale on a bubble study and the improvement in symptoms and oxygen requirement after closing PFO indicate that platypnea-orthodeoxia syndrome was due to the PFO. It is common for around 25% of adults to have Patent Foramen Ovale (PFO). This condition happens when the foramen ovale, a small opening between the right and left atria, remains open despite the drop in pulmonary resistance and blood pressure on the right side of the heart after birth. The most well-established complication of PFO is cerebrovascular accident, but it has also been associated with other adverse neurological, embolic events, and hypoxemic symptoms such as POS. SCAI guideline suggests PFO closure in patients with POS if another explainable cause of POS is not found. In a study called "Percutaneous Intervention to Treat Platypnea-Orthodeoxia Syndrome," 52 patients were treated with percutaneous closure of an interatrial communication between January 1997 and July 2015. The study showed that patients with platypnea-orthodeoxia syndrome can be successfully treated with a percutaneous intervention, which may require different types of devices.

Clinical significance:

Platypnea-orthodeoxia is a rare condition that causes shortness of breath (dyspnea) and is often not recognized initially. This condition occurs due to a mismatch between the pulmonary and systemic blood flow, which leads to arterial desaturation in the upright position. Awareness of this condition is crucial to prevent delays in appropriate management. Early diagnosis and treatment can effectively alleviate symptoms and prevent further complications arising from this condition.
A case report of upper extremity dvt presenting as metastatic lymphoma

Introduction
Upper extremity DVT is defined as clot formation in veins such as the internal jugular and brachial veins. The etiology is not well understood, but can be primary (hypercoagulable states) or secondary. Classic presenting symptoms include arm and facial edema, shoulder or neck discomfort, and limb heaviness. Although less common than lower extremity DVT, it is important to recognize and promptly treat to avoid complications such as pulmonary embolism.

Case Description
A 48 year old female presented to the emergency department with chief complaints of shortness of breath, sore throat, left arm and neck swelling. Labs were remarkable for leukocytosis and elevated LDH. Physical examination showed left upper extremity tenderness and swelling. Initial CTA of the chest demonstrated prominent left neck and axillary lymph nodes, a small soft tissue lesion in the left breast and multiple focal areas of splenic hypodensities. Upper extremity doppler showed a DVT in the left internal jugular and subclavian veins. CT of the neck with contrast showed an infiltrating mass in the superior mediastinum extending into the left side of the neck with occlusion of the left subclavian and internal jugular veins. Given the patient’s initial imaging findings and clinical presentation, the concern for metastatic lymphoma was raised. Subsequent imaging including CT abdomen showed calcified splenic granulomas, ruling out splenic masses. CT venogram of the neck and repeat CTA of the chest revealed that the initial soft tissue abnormalities seen on CT were likely reactive, secondary to the thrombus. Breast ultrasound showed a benign left breast cyst and MRI of the chest showed no focal masses necessitating a biopsy. Patient started anticoagulation therapy, and on day 3 of admission, underwent successful mechanical thrombectomy and was discharged with outpatient follow-up.

Discussion
In reported cases of UEDVT, 38% of patients were found to have cancer. In this case, although initial imaging findings were concerning for a malignant process, repeat imaging was consistent with a reactive process secondary to an underlying thrombus. This case demonstrates the overlapping symptoms of upper extremity DVT and lymphoma, and highlights the importance of follow-up imaging in establishing the final diagnosis.

References
Abstract

Acute myeloid leukemia (AML) is a cancer of hematopoietic stem cells. Typical symptoms include manifestations of anemia, thrombocytopenia, and leukocytosis. We report a case of a 68-year-old patient who suffered a traumatic workplace injury and had persistent symptoms of shortness of breath, fatigue, and unresolved bruising. Multiple ambulatory care facilities attributed these symptoms to the injury above, but upon reevaluation, workup instead revealed a diagnosis of high-risk myelodysplastic syndrome rapidly evolving to AML.

Introduction

Acute myeloid leukemia is a cancer of hematopoietic stem cells. It can coexist as a spectrum of diseases arising from high-risk myelodysplastic syndromes or develop independently. Risk factors include exposure to benzene, ionizing radiation, alkylating agents, and Down syndrome. We report a case of a 68-year-old patient who presented to the emergency department with persistent symptoms of shortness of breath, fatigue, and unresolved bruising. Although the patient had a prior workplace injury potentially exacerbating these symptoms, diagnostic workup revealed an underlying myelodysplastic syndrome, which rapidly progressed to AML.

Case Report

A 68-year-old patient with a past medical history of hypertension, seizure disorder, and dementia presented to the emergency department with a chief complaint of generalized fatigue. He worked as a mechanic and suffered a traumatic injury three months prior. On physical examination, the patient had left-sided contusions and diffuse ecchymoses. The patient visited several urgent care facilities due to these symptoms, which were attributed to the prior injury.

Diagnostic workup included a complete blood count, a basic metabolic panel, an electrocardiogram, a CT angiogram of the chest, abdomen, and pelvis, and a chest x-ray. EKG and imaging studies demonstrated no acute process. CBC was significant for anemia, leukocytosis, and severe thrombocytopenia. These findings prompted hospital admission to perform a bone marrow biopsy. Aspirate showed bone marrow consistent with high-grade myelodysplastic syndrome, with blasts approaching almost 20%, borderline for acute myeloid leukemia.

Discussion

Acute myeloid leukemia is a cancer of hematopoietic stem cells. It is the most common type of leukemia in adults, accounting for roughly 80% of leukemias. The pathophysiology of AML is related to myeloblast cells proliferating to the point where the bone marrow cannot function, leading to symptoms of pancytopenia. Since AML often presents with nondescript fatigue or malaise, it is usually first detected on routine blood work. Confirmatory diagnosis can be made with bone marrow biopsy showing >20% myeloblasts.
Skin Lesions as a Harbinger: An Uncommon Presentation of Pyoderma Gangrenosum in New-Onset Ulcerative Colitis

Pyoderma Gangrenosum is an uncommon non-infectious neutrophilic dermatosis often associated with underlying immune-mediated conditions, notably, inflammatory bowel disease (IBD). Pyoderma gangrenosum remains a diagnosis of exclusion due to absence of definitive tests, leaving patients susceptible to diagnostic pitfalls and delayed treatment.

We present a 67-year old woman with hypothyroidism and hypertension who presented to the hospital for two non-healing abdominal wounds. She underwent a surgical wound debridement 3 weeks prior to presenting to the hospital. When she presented, she did not demonstrate any systemic signs of infection, however the dark heaped margins of her recently debrided abdominal wounds were concerning for necrotizing fasciitis, so general surgery was consulted. It was only after a second wound debridement and a negative infectious work-up, including negative surgical wound and blood cultures, that a possible non-infectious immune-mediated cause was considered. A punch biopsy was obtained from the surgical wound margin and sent along with the surgical tissue biopsy to pathology. While waiting for pathology results, the patient reported several bloody bowel movements not revealed during her initial presentation. Stool studies were sent and showed elevated fecal calprotectin and stool WBCs. Subsequently, the patient underwent colonoscopy which showed moderate to severe colitis and proctitis that was characterized by ulcerated, diffuse and chronic active colitis with crypt abscesses. Shortly thereafter, pathology results demonstrated neutrophilic dermatosis, most consistent with pyoderma gangrenosum.

She was newly diagnosed with ulcerative colitis (UC) complicated by a rare form presentation of abdominal wall pyoderma gangrenosum. The patient was started on a combination regimen of oral and suppository mesalamine along with a prescription for a steroid course and taper. On follow-up post-discharge from the hospital, her abdominal wounds were noted to decrease in size in response to her new medical therapy. The patient underwent several painful wound dressing changes and a protracted wound healing course that might have been lessened with a more thorough history at presentation and a punch biopsy sent to pathology prior to a second wound debridement.

This case highlights the significance of identifying PG in cases of subclinical UC, as it facilitates targeted treatment. Although PG is widely acknowledged, there is frequently a lack of timely identification and diagnosis. Physicians should have an understanding of this condition and actively take into account PG when evaluating patients with ulcers. This is crucial since timely and suitable therapy in the early stages of the disease can prevent the difficulties associated with extended systemic treatment, delayed wound healing, and scarring.
Impact of Sarcoidosis on In-Hospital Outcomes among Patients with Atrial Fibrillation: a Nationwide Readmissions Database Analysis

Introduction: Sarcoidosis is a disease that involves multi-organs, particularly the cardiovascular system. While cardiac sarcoidosis has been increasingly recognized, the impact of sarcoidosis on atrial fibrillation (AF) is not well-established.

Objective: This study aimed to analyze the impact of sarcoidosis on in-hospital outcomes among patients admitted for a primary diagnosis of AF.

Methods: Using the all-payer, nationally representative Nationwide Readmissions Database, our study included patients aged 18 years or older admitted for AF between 2017 and 2020. We stratified the cohort into two groups depending on the presence of sarcoidosis diagnosis. The in-hospital outcomes were assessed between the two groups via propensity score analysis.

Results: A total of 1,031 (0.27%) AF patients with sarcoidosis and 387,380 (99.73%) AF patients without sarcoidosis were identified in our analysis. Our propensity score analysis of 1031 (50%) patients with AF and sarcoidosis and 1031 (50%) patients with AF but without sarcoidosis revealed comparable outcomes in early mortality (1.55% vs 1.55%, \(p=1.000\)), prolonged hospital stay (9.51% vs 9.70%, \(p=0.874\)), non-home discharge (7.95% vs 9.89%, \(p=0.108\)) and 30-day readmission (13.29% vs 13.69%, \(p=0.797\)) between two groups. The cumulative cost of hospitalization was also similar in both groups ($12632.25 vs $12532.63, \(p=0.839\)). The in-hospital adverse event rates were comparable in both groups.

Conclusion: Sarcoidosis is not a risk factor for poorer in-hospital AF admission outcomes. These findings provide valuable insights into the effectiveness of current guidelines for AF management in patients with concomitant sarcoidosis and AF.
Acute Decompensated Heart Failure in a Patient with Erythrodermic Psoriasis

Introduction

Erythrodermic psoriasis (EP) is an acute life-threatening complication of psoriasis and one of the most severe and rare subtypes of EP. It is defined as erythema and scaling that comprise more than 80-90% of total body surface area. It can occur as the first sign of psoriasis, but more commonly develops as a complication of steroid withdrawal (known as the rebound withdrawal), drugs or an illness. This condition poses significant risks with multiorgan involvement that includes hemodynamic and metabolic disturbances, secondary infections, heart failure and even death, necessitating prompt hospitalization for fluid management and treatment of disease. Due to diffuse vasodilation, hypotension and peripheral edema, patients with EP can develop severe compensatory fluid retention, possibly explaining why these patients develop heart failure and pulmonary edema. The mortality rate ranges from 9%-64% and patients with EP have a 6.2% absolute risk of a cardiovascular event in a ten-year period. A retrospective single-center study of 225 patients with EP found that 11.1% of the patients had heart failure.

Case

Here we present a case of a 93 year old male with a past medical history of psoriasis who presented to the Emergency Department one month after being newly diagnosed with erythrodermic psoriasis. Physical examination at that time showed impressive very thick, silvery scaly plaques on erythematous bases of the scalp, cheeks, trunk and extremities with areas of excoriations due to itching, especially on the upper and lower extremities. There was additional tenderness with weeping of serous fluid and areas of superimposed impetiginization on his lower extremities. His nails were pitting, had evidence of onycholysis, leukonychia and splinter hemorrhages. Workup was notable for normal cardiac function and prerenal acute kidney injury (AKI) and was started on high dose oral prednisone and switched to topical steroids with good response to treatment. Although improved from a month prior, the patient still had severe widespread skin inflammation. He presented in acute respiratory distress and was found to have acute heart failure with reduced ejection fraction. He was intravascularly depleted as reflected by an elevated lactate (3.7) and prerenal AKI that was worse from previous. Repeat echocardiography showed new hypokinesis with segmental wall abnormalities and an ejection fraction of 30-35%. CT PE was remarkable for bilateral pleural effusions and pneumonitis. Patient was started on medical therapy for his heart failure and high dose oral steroids and topical steroids for his erythrodermic reaction. Of note, the patient incidentally tested positive for Covid.

Conclusion

In summary, this case highlights the importance of identifying the risk of heart failure in patients with psoriasis who present with erythroderma. Treatment of EP includes supportive care, topical steroids and sometimes systemic therapy. This case report also poses the question whether treatment with systemic steroids contributes to the development of heart failure in patients with erythrodermic psoriasis.
A Case of Hepatocellular Carcinoma in Hepatitis B and Hepatitis D Coinfection

Introduction: It is estimated that approximately 300 million individuals are chronically affected with the Hepatitis B virus (HBV). Coinfection with the hepatitis D virus (HDV) is estimated to be found in roughly 5% of patients who have HBV. HDV infection occurs when people become infected with both HBV and HDV. Prevalence of HDV infection in the US is around 100-150 thousand people. The annual incidence of HCC in patients with HBV can be as high as 2-3% in patients with liver cirrhosis. However HDV has not been proven to be directly associated with increased incidence of HCC. We present a case of a patient that was admitted due to decompensated liver cirrhosis and found to have a liver mass and positive serologies for both HBV and HDV.

Case
58 y.o. male with a past medical history of HTN who presented to the ED 1/12 with complaints of worsening swelling of both lower extremities up to the scrotum for the past 4 months, associated with abdominal distention, shortness of breath and moderate generalized itching. He also endorsed epistaxis and bleeding gums. His laboratory was significant for revealed hypokalemia, elevated liver enzymes, leukopenia with thrombocytopenia hypoalbuminemia, increased creatinine, hyperphosphatemia. Patient was diagnosed with liver cirrhosis, mild AKI.

CT an MRI scan revealed 2 masses (with possibility of hepatic hemangioma and HCC).

In addition, the patient was diagnosed with chronic hepatitis B and Hep D also positive. Biopsy of the liver mass resulted positive for well differentiated hepatocellular carcinoma.

<table>
<thead>
<tr>
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<th>Result</th>
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<tr>
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<td>Hepatitis B e Ab</td>
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<td>Hepatitis C Ab</td>
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<tr>
<td>Hepatitis D virus total</td>
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</table>

Discussion
HBV is the second most important environmental carcinogen that can predispose to tumors. It is related to as much as 80% of all HCCs in black african populations. HCC is currently the fifth most prevalent malignancy in men and eighth in women. The mortality of HCC is almost the same as its incidence, correlating to the fact this is a rapid progressive disease with a very poor prognosis. HDV is a virus that is known to cause worsen liver damage when compared to HBV alone but there is no direct association between HDV and potential for carcinoma. HDV is not known to be a carcinogenic virus.

Conclusion: The correlation between hepatitis B and HCC is well known. However, it is still not clear whether HCC can be a consequence of the cumulative effect of HBV and HDV, an effect of cirrhosis or if there is a direct oncogenic effect of HDV. More studies are needed to understand the oncogenic role of HDV.
Treatment Resistant Hypertension with Normal Kidney Function: A Manifestation of Bilateral Renal Artery Stenosis

Introduction

Renal artery stenosis (RAS) frequently presents in patients with peripheral artery disease (PAD), coronary artery disease (CAD) and severe retinopathy and is most commonly caused by atherosclerosis or fibromuscular dysplasia. Less common causes include vasculitis, infrarenal aneurysms, arterial dissection and thromboembolic disease. Common complications of RAS include decompensated heart failure syndromes, chronic kidney disease (CKD) and end-stage renal disease (ESRD). Risk factors for atherosclerosis include dyslipidemia, tobacco use, infection and increased homocysteine levels. Bilateral RAS merits extra attention because of its indolent, asymptomatic course and accelerated clinical contribution ischemic nephropathy. Consequently, patients with bilateral RAS are at a higher risk of renovascular disease and are important to identify as they are most likely to benefit from revascularization, usually by percutaneous angioplasty with stenting, in addition to medical management. Identifying factors include a recent or rapid progression of severe hypertension, the development of flash pulmonary edema, and a rapid rise in serum creatinine (which points to hemodynamic compromise of the kidneys). Risk factor reduction including secondary prevention of cardiovascular disease is important as patients with atherosclerotic renovascular disease are at increased risk of cardiovascular complications and adverse outcomes.

Case

Here we present a case of a 47 year old female who presented with intractable nausea and emesis consequent of gastroparesis and constipation and known unilateral RAS with stent placement, CAD with five-vessel coronary artery bypass grafts in 2019 (CABG), uncontrolled type 2 diabetes mellitus, tobacco dependence and previously well-controlled hypertension. Hospital course was complicated by persistently severe elevated blood pressure measurements that were treatment resistant to four antihypertensive medications. Abnormal flow velocities of bilateral renal arteries were found on US Renal Artery Doppler ultrasound and CT angiogram of the abdomen with Contrast showed high-grade right renal artery stenosis with a patent left renal artery stent without evidence of restenosis. Mild-moderate atherosclerotic disease was noted throughout abdominal vasculature and normal appearing kidneys. Patient was taken for right renal artery stent placement.

Summary

Our case highlights the importance of identifying secondary causes of hypertension in patients who are refractory to medical therapy. In the case of patients with severe atherosclerotic disease and treatment-resistant hypertension, it is important to identify the risk factors that predispose patients to RAS.
An unusual presentation of eosinophilic pneumonia induced by daptomycin

Introduction:

Eosinophilic Pneumonia (EP) is a rare respiratory illness that is caused by eosinophilic infiltration of the lung. One report estimates EP has an incidence of 9.1 per 100,000 person-years in select military populations, or 0.54 per 100,000 per year in general population. EP symptoms often present over 7 days, but symptoms may also progress up to over a month. The disease can rapidly progress to respiratory failure requiring respiratory ventilation. One of the most common etiologies of EP is medication side effect. There are multiple types of medications associated with this disease. Here we present a case of a patient with EP secondary to Daptomycin use.

Case:

Patient is a 77-year-old male with a past medical history of asthma, gastroesophageal reflux disease and chronic right foot osteomyelitis with distant smoking history. He presented to the emergency department for shortness of breath. The patient was admitted one month prior to current admission for chronic ulcer complicated by osteomyelitis of right foot and was discharged with a PICC for daptomycin and cefepime for 8 weeks. Patient reported within days of starting antibiotics experiencing symptoms of shortness of breath, cough with clear sputum and wheezing that had been progressive. On arrival he was hypoxic and had to be placed on high flow nasal cannula. A CT scan of his chest was remarkable for ground glass opacities and air-space consolidations with background of pleural effusions with evidence of superimposed pneumonitis. Initial blood differential showed absolute eosinophilia, differential showed progressively increasing eosinophilia over course of antibiotics. Given concern of Daptomycin induced eosinophilic pneumonia, this medication was discontinued and replaced for vancomycin. Daptomycin was replaced with vancomycin for the osteomyelitis. Pulmonary services were consulted, and empiric therapy for eosinophilic pneumonia was initiated with methylprednisolone 1.5 mg/kg. Bronchoscopy with bronchoalveolar lavage was not deemed appropriate prior to administration of steroids due to oxygen requirements and patient not able to provide sputum, improvement with steroids, and clinical improvement after holding the offending drug. Eosinophilia quickly resolved within about one day seen on CBC and sputum eosinophilia was also negative. Oxygen requirements gradually decreased to 3L and improving. Patient was discharged with oxygen and oral prednisone taper with close pulmonary follow-up with uninterrupted osteomyelitis antibiotic course.

Conclusions:

This case highlighted the importance of including eosinophilic pneumonia in differential in the setting of use of certain antibiotics and characteristic lab findings. The presumptive diagnosis of eosinophilic pneumonia in this case was suggested by imaging, a precise antibiotic use to symptom onset history, characteristic eosinophilia, and response to treatment. Patients risk factors considered included medication history, occupational history, history of asthma and environmental exposures. Interestingly, patient had only a distant smoking history. He had left shift attributed to the osteomyelitis after administration of steroids and experienced new-onset asymptomatic tachyarrhythmia thought to be secondary to QT prolongation associated with steroid use, which resolved as steroid was tapered. Patient had initial rapid decline but was able to recover without advancing airway needs due to timely suspicion and treatment of eosinophilic pneumonia.
A case of Unicuspid Aortic Valve presenting with Infective Endocarditis

Introduction
The normal anatomy of the aortic valve compromises three cusps, each of them separated by commissures. The most common anatomical variant is a bicuspid aortic valve (BAV) where only 2 cusps are present. An even rarer congenital variant is a unicuspid aortic valve (UAV).

The incidence of UAV is approximately 0.02 % in adults undergoing echocardiography and 4-5% in patients undergoing surgery for aortic valve replacement. It has a higher incidence in males with a male-to-female ratio of 4:1.

BAV is a well-established risk factor for infective endocarditis (IE) with a recent meta-analysis showing a 12-fold higher risk compared to the general population. The proposed pathophysiology is abnormal blood flow through the valve predisposes to bacterial seeding. In the case of UAV data is scarce, but the same principles can be applied given that it is also an abnormal anatomical variant.

Case presentation
This is the case of a 49-year-old man with a past medical history of psoriatic arthritis and gout with recent multiple tick bites, with the most recent lasting approximately 12 hours. After tick removal, no symptoms were noted. 5 weeks later, the patient started experiencing fatigue, headaches, and diffuse joint pain. He visited his PCP who prescribed a 10-day course of doxycycline. Lyme antibodies were negative, and symptoms subsided. After completion of antibiotics, symptoms recurred and he received a second course with doxycycline and a steroid taper. During the interim, he developed sudden onset, severe left-sided clavicular pain which radiated to the shoulder. He visited the ER where troponins were elevated and was transferred to a tertiary care center for NSTEMI.

Left heart CATH revealed normal coronaries. In the post-op the patient developed afib and routine blood cultures grew Strept mitis group. He underwent TEE/cardioversion which showed a UAV with severe aortic regurgitation and two small vegetations. He underwent valve replacement surgery and was discharged on IV ceftriaxone.

Discussion
By definition, a unicuspid aortic valve only has one leaflet. There are two types: the commissural in which there is a teardrop orifice attached to the aorta through a commissure. The acommissural is where there is only a center orifice.

The acommissural variant presents with symptoms of left-sided heart failure and severe aortic stenosis (AS) early in life. While the commissural type has a more progressive course presenting with symptoms of AS in early adulthood. UAV is associated with other disorders including patent ductus arteriosus, ventral septal defect, anomalous coronary artery, and ascending aortic dilation.

Management in children varies depending on associated conditions. In adults, valve replacement is the mainstay treatment. Guidelines determine BAV as an intermediate risk for IE and recommend against antibiotic prophylaxis. If a patient were to develop IE, treatment would be the same as a person with a tricuspid aortic valve.

Conclusion
UAV is a rare congenital variant of the aortic valve and data is scarce. Regardless of presentation, treatment usually involves valve replacement. In the setting of IE, data is lacking but the same principles for BAV could be applied.
Mediastinal Intrusion: A Case Report on the Unusual Course of Pancreatic Pseudocyst

A pancreatic pseudocyst, a recognized complication of both acute and chronic pancreatitis, refers to a confined accumulation of fluid abundant in pancreatic enzymes. Its development is attributed to the disruption of the pancreatic duct resulting from pancreatitis or trauma, leading to the leakage of pancreatic secretions. In instances where the disruption occurs at the posterior aspect of the pancreatic duct, the pancreatic secretions may extend into the mediastinum. However, the occurrence of mediastinal extension is rare.

31-year-old female, with a past medical history of alcohol use disorder and chronic pancreatitis, presented to the Emergency Department (ED) with complaints of chest and epigastric pain radiating to the back. Initial investigations, including EKG and troponin, yielded negative results, while an elevated lipase at 326 and a WBC count of 23.3 prompted further evaluation. A CT abdomen revealed severe fat stranding around the pancreas, decreased attenuation of the pancreatic tail suggesting necrotizing pancreatitis, and a 4 x 2.8 cm pancreatic tail pseudocyst. Despite appropriate management with analgesics and Ceftriaxone, the patient continued to experience unexplained chest pain, dysphagia and odynophagia which prompted a CT chest revealing posterior mediastinal abscesses as a direct extension of the pancreatic pseudocyst. EGD was negative for any abnormalities. Antibiotic coverage was broadened to Piperacillin-Tazobactam and Fluconazole. Further investigation, including MRCP, indicated necrotizing pancreatitis and a communicating tract between peripancreatic and mediastinal collections. Initially, surgery was deferred due to the high risk of ascending infection. However, persistent chest pain led to a repeat CT, disclosing an increased size of pancreatic pseudocyst to 9.6 x 7.6 cm and mediastinal collections inseparable from the thoracic esophagus. An endoscopic ultrasound guided cystgastrostomy was subsequently performed, resulting in significant symptom improvement. The patient was advised to undergo a repeat EGD for LAMS removal in 2-3 weeks, continue Zosyn and fluconazole for 4 weeks, followed by a repeat CT chest abdomen pelvis.

Pancreatic pseudocysts, common benign cystic lesions of the pancreas, typically arise in 5-15% of patients with peri-pancreatic fluid collections following acute pancreatitis. However, in approximately 20-22% of cases, these pseudocysts may extend to the mediastinum, pleura, peritoneal cavity, or the pelvis. Diagnosis primarily relies on abdominal ultrasound, followed by CT scan or MRI. Treatment options range from watchful waiting, which resolves about 65% of acute pseudocysts spontaneously, to interventions like drainage.

Indications for drainage include symptomatic, enlarging, infected pseudocysts, or those affecting adjacent organs. While surgery has been the traditional management, it carries a significant complication rate of 35 % and a mortality rate of 10 %. Nonsurgical approaches like Percutaneous puncture and aspiration guided by ultrasound or CT have been employed, yet aspiration alone proves ineffective, resulting in recurrence rates as high as 71%. Continuous percutaneous drainage with catheters can lower relapse rates but poses risks of complications ranging from 5% to 60%, including fistula formation, infection, and bleeding.

Endoscopic transmural drainage, specifically EUS-guided drainage, has emerged as a less invasive and potentially preferable first-line treatment due to its lower morbidity, faster recovery, and cost-effectiveness compared to surgical or percutaneous methods.
A Chilly Phenomenon: Submissive Pulmonary Embolism and Bilateral Deep Venous Thrombosis (DVT) in a Patient with Cold-Agglutinin Disease

Cold Agglutinin Disease (CAD) is a distinctive form of autoimmune hemolytic anemia characterized by the presence of cold-reacting autoantibodies, leading to hemolysis and agglutination of red blood cells at temperatures below the normal body range. Despite being a rare condition, CAD poses a significant risk of thrombotic events (TEs) in both venous and arterial systems. Existing literature has identified an association between various forms of hemolysis and an increased incidence of TEs, yet limited attention has been given to understanding this phenomenon in the context of CAD. In CAD, ongoing complement activation plays a pivotal role in mediating hemolysis, contributing to a range of clinical manifestations such as anemia, acrocyanosis, and fatigue. The potential morbidity and mortality associated with TEs in CAD patients underscore the need for a deeper exploration of this relationship.

We present a compelling case of a 90-year-old woman diagnosed with CAD, whose clinical course was complicated by the occurrence of a submassive pulmonary embolism and bilateral DVTs in the right gastrocnemius and left popliteal vein and was started on heparin. Cardiac and infectious workup was unremarkable. Her clinical course was complicated by worsening anemia without active bleeding, requiring recurrent transfusions of warmed pRBC and discontinuing heparin. Coincidentally, the patient was hospitalized during a deep freeze and a cold draft could be felt throughout the hospital despite their best heating efforts. Initially, the patient's recurrent need for daily transfusions was thought to be consequent to the cold, and additional heating in the form of a Bair hugger was added. Hemolytic workup was elevated at baseline without changes and peripheral smear was unremarkable. Despite these measures, the patient continued to require daily blood transfusions. Rituximab infusion was planned, however, she developed a transfusion-associated circulatory overload (TACO) during her sixth blood transfusion. While an exacerbation of autoimmune hemolytic anemia secondary to transfusion cannot be completely excluded, no underlying alloantibodies were identified on subsequent testing nor contamination of blood products identified. DIC was ruled out. Goals of care discussions led to the decision of transitioning the patient to comfort care/hospice measures, resulting in the discontinuation of treatment including rituximab and further blood transfusions.

Discussion

Our patient, without prior infection or hypercoagulable conditions, presented to our hospital. In Cold Agglutinin Disease (CAD), triggered by low temperatures, cold agglutinins causing RBC agglutination, leading to increased blood viscosity. During cardiopulmonary bypass cooling, reports indicate agglutinates forming, potentially contributing to venous thrombosis through reduced blood flow and stasis. This case emphasizes the critical importance of recognizing and addressing thrombotic risks in CAD patients, highlighting a less-explored aspect of the disease. Understanding this interplay is essential for improving the management and prevention of thrombotic complications in CAD.

In summary, our case highlights the crucial link between Cold Agglutinin Disease (CAD), temperature-induced red blood cell agglutination, and increased thrombotic risk. Recognizing and managing this connection is vital for optimizing care and preventing severe complications in CAD patients. Further research is needed to enhance our understanding and strategies for addressing thrombotic risks in CAD.
A Rare Case of E. Coli Endocarditis

Background
Infective endocarditis due to E. Coli is a rare etiology of endocarditis with a reported incidence of less than 1% of all native valve endocarditis. Urinary tract infection is known as the most common cause of endocarditis. Generally, E. Coli has a higher mortality rate compared to HACEK-group gram negative bacteria.

Case
A 83-year-old male with past medical history of Parkinson’s disease, hypertension, hyperlipidemia was admitted for complaints of fever and agitation. Approximately 1 hour after dinner patient became confused, somnolent, had generalized shakiness, and was warm to touch and brought to the ED for further evaluation. Upon arrival patient was febrile, tachycardic, tachypneic, and hypertensive. His initial labs showed a lactate of 9, anion gap 17, and bicarbonate of 21. Patient denied urinary symptoms or recent instrumentation, however, on further work up, laboratory investigations were significant for positive urine leukocyte esterase and nitrites, but negative urine cultures. He was started on Zosyn with persisting fevers and concern for sepsis. He eventually was diagnosed with E. Coli bacteremia, confirmed by blood cultures, and switched from Zosyn to Ceftriaxone due to sensitivities. A transesophageal echocardiogram was done to further evaluate the cause of E. Coli bacteremia that revealed a 4-mm filamentous mobile density attached to the ventricular side of non-coronary cusp of the aortic valve confirming the diagnosis of infective endocarditis. The patient was started on a 6-week course of intravenous ceftriaxone due to sensitivities. A repeat transesophageal echocardiogram after 7 weeks showed complete resolution of above-mentioned mobile density.

Decision-Making
Transesophageal echocardiogram was crucial in the management of the E. Coli bacteremia in the present case. The low incidence of E. Coli infective endocarditis has been attributed to the inability of this bacterium to adhere to the endocardium as well as the existence of antibodies to E. Coli in normal serum. Due to limited literature evidence, infective endocarditis in setting of E. Coli bacteremia, can pose diagnostic challenges due to anchoring bias resulting from the treatment of solely urinary tract sources, which although are most common, may not represent the entire spectrum of E. Coli infection presentations. Treatment duration of intravenous antibiotics vary significantly with infective endocarditis as compared to complicated urinary tract infections, and adequate therapy duration is critical for source control.

Conclusion
Urinary tract infection appears to be an important factor in the development of E. Coli endocarditis in this patient who had no specific cardiac risk factors. E. Coli endocarditis is more likely to effect native valves without valvulopathy rather than prosthetic valves. Proper antibacterial coverage is recommended for 6 weeks. Infective endocarditis resistant to antibiotic therapy might require surgical intervention. Persisting fevers and altered mental status in an individual with a urinary tract infection despite antibiotic treatment should be investigated for other serious infections like endocarditis.
Atypical Hemolytic Uremic Syndrome in a Young Adult with Complex Psychiatric History: A Diagnostic Challenge

Atypical hemolytic uremic syndrome (aHUS) is a rare, life-threatening condition characterized by the triad of microangiopathic hemolytic anemia, thrombocytopenia, and acute kidney injury, often without a preceding significant diarrheal illness. Its rarity and the broad differential diagnosis make aHUS a formidable challenge, particularly in patients with complex medical histories. We report a case of aHUS in a 26-year-old male, underscoring the diagnostic challenges and the critical role of early intervention.

A 26-year-old male with a complex history of bipolar disorder, autism, Attention-Deficit/Hyperactivity Disorder (ADHD), oppositional defiant disorder, and pervasive developmental disorder presented with symptoms of perianal bleeding, itching, and significant lethargy. Initial laboratory results were within normal limits, masking the imminent diagnosis of aHUS. However, within 4-5 days, a marked deterioration in his condition was observed. This was highlighted by a significant drop in platelet count to a nadir of 22, indicating severe thrombocytopenia, alongside an acute kidney injury with creatinine levels peaking at 5.52. Hemolysis was evident from an elevated lactate dehydrogenase level of 2248 and a critically low haptoglobin level of <6. Further complicating the clinical picture, the patient’s complement system exhibited substantial dysregulation: Protein S levels were alarmingly low at 15 (normal range: 63-140), Protein C levels were decreased to 30 (normal range: 73-180), total complement activity (CH50) was below 17 (normal: >41), C3 levels were reduced to 34 (normal range: 79-152), and C4 levels were undetectably low at less than 4 (normal range: 16-38), aligning with the diagnosis of aHUS. The presence of Shiga toxin-producing E. coli (STEC) introduced an additional layer of diagnostic complexity, blurring the lines between typical STEC-HUS and aHUS.

This case highlights the diagnostic complexities encountered in patients with aHUS, especially when psychiatric comorbidities obscure the clinical presentation. The absence of diarrheal prodrome and the patient’s unique psychiatric profile necessitated a comprehensive diagnostic approach, ultimately leading to the diagnosis of aHUS based on clinical and laboratory findings, including evidence of complement dysregulation. The successful management of this case with eculizumab, despite the initial diagnostic ambiguities, highlights the effectiveness of complement inhibition in aHUS, even in cases with atypical presentations. It also reinforces the importance of early diagnosis and intervention in aHUS management. This case contributes to the literature by illustrating the efficacy of complement inhibition in aHUS treatment and underscores the necessity of a multidisciplinary approach in managing patients with complex medical and psychiatric backgrounds. The case not only adds to the growing body of evidence supporting the use of complement inhibitors in aHUS but also serves as a reminder of the critical need for vigilance and adaptability in the face of diagnostic uncertainties.
Unusual Case of Cardiac Myxoma Mimicking Infective Endocarditis

Introduction:

Myxomas are the most common primary cardiac tumor with overall low incidence that usually arises from the interatrial septum and rarely from the mitral valve (MV). The clinical presentation of myxomas shares common systemic signs and symptoms with infective endocarditis (IE), rendering the latter a chief consideration in the differential diagnosis.

Case Presentation:

A 75-year-old female patient presented with a one-month history of fever, chest pain, and dyspnea. She had hypotension, fever, and elevated inflammatory markers. Electrocardiography revealed ST-elevation myocardial infarction and atrial fibrillation with rapid ventricular response, necessitating an emergent but successful synchronized cardioversion. Coronary angiography showed a thrombus in the left anterior descending (LAD) artery. To preserve LAD integrity, no percutaneous intervention was performed. Transthoracic/esophageal echocardiography (TTE/TEE) revealed a large, irregular, mobile MV mass (2.7x2.3 cm). IE was suspected, and intravenous (IV) antibiotics were initiated without improvement in infectious markers, and cultures remained consistently negative.

Discussion:

In view of the patient's high surgical risk (Society of Thoracic Surgeons operative risk score > 50% due to extensive comorbidities), she was deemed an unsuitable surgical candidate by the cardiothoracic team. Consequently, structural cardiology performed a transcatheter vacuum-assisted debulking procedure (>90% reduction) on the left MV mass by AngioVac device. Subsequent echocardiography confirmed the complete resolution of the mass with no remnants noted. The patient exhibited only marginal improvement. Pathology confirmed an atrial myxoma.

Conclusion:

The diagnostic challenge arises from the similarities between cardiac myxoma and IE, frequently leading to misdiagnosis. Typically, TTE/TEE serves as differentiating tools, but approximately 5% of myxomas originate from the MV, the site most susceptible to IE vegetation. While IE is primarily managed with IV antibiotics, the cornerstone of myxoma treatment lies in mass removal. Therefore, in cases of suspected IE that are unresponsive to antibiotics, the possibility of a myxoma should also be considered.
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**Diffuse large B-cell lymphoma presenting as a cardiac mass**

**Introduction:** Cardiac involvement as the initial presentation of malignant lymphoma is a rare occurrence. The most common type of cardiac lymphoma is diffuse large B-cell lymphoma (DLBCL) and often affects the right atrium. Cardiac lymphoma can either be mediastinal DLBCL invading the heart or primary cardiac lymphoma.

**Case Presentation:** An 86-year-old female with past medical history of hypertension and hypothyroidism presented to the ED with progressive shortness of breath. She reported worsening dyspnea over the past 8 weeks, which was associated with a dry cough. She denied any fever, weight loss or fatigue. Physical examination was unremarkable.

Labs revealed mild leukocytosis and elevated D-dimer. CXR showed a new right sided pleural effusion. CTA chest revealed a large mass like density within the anterior pericardium, measuring 8.5 x 5.2 x 9.4 cm, compressing the right atrium and right ventricle, and encasing the right coronary artery. There was also an area of a filling defect in the right atrium that could be mass invasion versus thrombus. The scan showed no PE but did show the right pleural effusion with collapse of the right middle and lower lobes. TTE showed a multilocular hypoechoic mass in the right atrium measuring 7 x 6.7 cm with some component invading into the wall of the right atrium. The patient underwent diagnostic and therapeutic thoracentesis. Pleural fluid cytology revealed diffuse large B-cell lymphoma, with positive stains for CD20, PAX5, CD10, BCL6 and Mum–1. FISH revealed abnormality of BCL2/18q (16%), negative for BCL6 rearrangement, MYC rearrangement, MYC/IGH rearrangement and IGH/BCL2 rearrangement.

**Staging PET scan showed large mediastinal mass involving right pericardium, focal uptake in left thyroid lobe, left skull base, musculature around proximal left femur.**

Chemotherapy was initiated with R-miniCHOP (rituximab, cyclophosphamide, doxorubicin, vincristine and prednisone) during her hospitalization due to the extent of disease on presentation. She tolerated the first round of chemotherapy and was discharged in stable condition with close Oncology follow-up. PET scan after 3 cycles of chemotherapy showed complete metabolic response to chemotherapy with resolution of previously noted hypermetabolic lesions within the neck, thyroid, pericardium, mediastinum, right atrium, musculature around the left proximal femur, consistent with Deauville score of 1.

**Discussion:** The differential diagnosis of a right atrial cardiac mass should include lymphoma. Mediastinal DLBCL with cardiac involvement may present with non-specific symptoms like dyspnea. TTE is usually the initial imaging test, and a tissue biopsy is required for definitive diagnosis. DLBCL is highly aggressive and carries a poor prognosis. Early diagnosis and treatment with standard chemotherapy are crucial for favorable outcomes.
An uncommon cause of B12 deficiency

Introduction: Nitrous oxide use is not often thought of as a cause of B12 deficiency. Diagnosis can be challenging in such patients and awareness is imperative for patients to receive appropriate treatment.

Case: A 43yo male with a history of polysubstance abuse, depression, ADHD, and possible seizure, presents with altered mental status. The patient was using nitrous oxide when he started to feel confused and called an ambulance. Over the past month, he has been ‘sniffing’ nitrous oxide canisters with increasing frequency. He also endorses having double vision and difficulty with balance. On exam, he is oriented x3, but has difficulty answering questions due to significant anxiety. He also has hyperreflexia in the bilateral upper and lower extremities and is intermittently tremulous. B12 level was found to be 113. Methylmalonic acid level was 3.24 and Homocysteine was >130. MRI brain and MRI C-spine were unremarkable, with no evidence of a demyelinating process. EEG showed no epileptiform abnormalities. The patient was treated with IM B12 injections. He did show some improvement in his mentation, but still endorsed difficulty concentrating and word-finding difficulty. Vitamin B12 levels normalized and the patient was discharged to inpatient rehab.

Discussion: Nitrous oxide use is a lesser known cause of B12 deficiency. This occurs when nitrous oxide binds directly to the cobalt atom of B12, inactivating it. When in this form, B12 can no longer convert homocysteine to methionine. When used over a prolonged period of time, this can lead to depletion of B12 stores and build up homocysteine. Diagnosis can be challenging for a number of reasons. Patients may be hesitant to admit to using nitrous oxide. The symptoms of B12 deficiency can be nonspecific. Also, the patient population using nitrous oxide more commonly has psychiatric comorbidities, which further obscures the underlying cause of nonspecific symptoms. This particularly was relevant to this case, as it was quite difficult to determine the patient's baseline functioning prior to using nitrous oxide. Awareness of the patient's substance use was vital to obtaining a diagnosis and initiating appropriate treatment.
Quality Review of False-Positive Cardiac Catheterization Lab Activations

Introduction

Cardiac catheterization is a procedure that mobilizes a significant amount of hospital resources and is not without risk to the patient. While most activations identify a culprit coronary artery occlusion, on occasion there is unnecessary mobilization for catheterization at every stage of hospitalization. This study's primary goal was to determine the rates of false activation for cardiac catheterization. The secondary goal was to provide education based on findings to reduce the risk of false activations.

Method

This retrospective study was based on review of emergent catheter lab activations within a single institution between September 2021 and July 2023. Cases were analyzed for patterns based on patient location at time of activation including pre-hospital and emergency department. EKG interpretations at time of catheter lab activation and associated culprit lesion were examined. False activations were defined as cardiac catheterizations with no identified culprit lesion. EKGs from false activations were then reviewed by a cardiology and emergency department provider not directly involved in the study to determine any patterns of EKG misinterpretation.

Results

180 catheter lab activations were reviewed with 60 deemed false activations. 20 of those cases were pre-hospital activations cancelled on arrival and so were excluded from the study. The remaining 40 patients underwent cardiac catheterization but had no culprit lesion found. Further review of EKGs by specialists suggested small errors in correct application of standard STEMI criteria, Sgarbossa criteria, and misunderstanding of STEMI equivalents.

Conclusions

These findings suggest a discrepancy in EKG interpretation for acute coronary ischemia within the emergency department. Amongst 160 patients, 25% underwent cardiac catheterization with no treatable coronary occlusion. Ongoing education for ED providers on EKG interpretation could reduce spurious use of hospital resources and reduce risk for patient complications.
Green Neutrophilic Inclusions: Really the "Green Crystals of Death"?
There are many types of inclusions that can be seen on blood smears that can aid in diagnosing a patient. Bright green neutrophilic inclusions are findings on a peripheral blood smear that pathologists deem a rare find but can indicate the rapid demise of a patient.

A 70-year-old female presented to the ED with recurrent abdominal pain. She was hospitalized 1 month prior to admission with a newly diagnosed pancreatic mass with good clinical response to biliary stenting via ERCP. Endoscopic biopsy was consistent with malignancy but not diagnostic for the specific type of cancer. Before she could be seen by Oncologic Surgery, she presented back to the hospital with pain, elevated alkaline phosphatase, AST, and ALT. She underwent repeat ERCP with stent exchange with initial clinical and laboratory improvement. Two days after the ERCP, she developed worsening pain, hypotension, and lactic acidosis requiring vasopressor support. She experienced progressive decline in mental status, acute kidney injury, and thrombocytopenia (dropping from 129 to 17 over 4 days). Due to worsening coagulation studies (PT/INR and aPTT) a manual differential was performed revealing normochronic and normocytic erythrocytes with mild anisocytosis with rare nucleated red blood cells. No schistocytes or rouleaux changes identified. Platelets were markedly reduced. Leukocytes were increased with mature neutrophils showing toxic granulation, vacuolization, and Dohle bodies. There were rare bright green-blue inclusions seen. Antimicrobials were expanded from ceftriaxone to piperillin/tazobactam due to desire to broaden intra-abdominal coverage. The Infectious Diseases team was contacted regarding the blue-green inclusions implying very significant poor prognosis. The arrival of these inclusions has a strong indication that the patient will expire within 48 hours of the result. The patient continued to rapidly decline, and she passed away on day 6 of admission. Post-mortem the patient had blood cultures turn positive for Enterococcus faecalis and a next-generation sequencing of whole blood revealed Escherichia coli.

This further illustrates the prognostic accuracy of the appearance of green inclusions within neutrophils. This finding has been described previously in multiorgan failure and septic shock. This has been most frequently described with Escherichia coli. This case additionally shows the value of a manual differential in diagnosis of systemic illness.
Fulminant Endocarditis in a Healthy Young Farmer - A Rare Case of Staphylococcus lugdunensis Endocarditis with Multiple Valve Destruction and Complete Heart Block.

Introduction:
Fulminant endocarditis is an exceptionally rare and potentially life-threatening condition, often posing diagnostic challenges due to its diverse manifestations. Staphylococcus lugdunensis (S. lugdunensis), a coagulase-negative staphylococcus (CoNS), has been identified as a causative agent of destructive infective endocarditis (IE) with various clinical presentations. Despite its rarity, the overall mortality associated with S. lugdunensis IE is alarmingly high, with only 17 reported cases worldwide, including 4 in the United States. This scarcity highlights the critical importance of heightened awareness within the medical community. We present a unique case of S. lugdunensis endocarditis, resulting in a ventricular septal defect and the destruction of aortic, tricuspid, and mitral valves.

Case Presentation:
A 32-year-old male, without significant medical history, presented with intermittent fevers, joint pain, and frothy urine. Initial assessments at urgent care revealed no notable findings. However, escalating symptoms over the next three days, including intense chest pain, shortness of breath, blurry vision, headache, myalgia, vomiting, and diarrhea, prompted an Emergency Department visit. The patient exhibited a fever of 102.6°F, tachycardia, and systolic blood pressure in the 150s. No history of intravenous drug use, bug bites, rashes, travel, or recent vaccinations was reported. Laboratory findings indicated an elevated white blood cell count of 12,100/mm3 and troponin of 78ng/L. Negative results for Lyme and Syphilis antibodies, influenza A/B, and COVID tests, coupled with unremarkable urinalysis and urine drug screen, ruled out common causes. An electrocardiogram revealed third-degree heart block, prompting an immediate cardiology consultation. Subsequent investigations, including a transesophageal echocardiogram, unveiled large-sized vegetations on the mitral and tricuspid valves, a bicuspid aortic valve with severe regurgitation, and a potential aortic ring abscess.

The case necessitated an urgent transfer to the Structural Heart Center, where extensive repairs, including valve replacements, intravalvular fibrous body reconstruction, aortic annular enlargement, and ventricular septal defect repair, were performed. Blood cultures later confirmed Staphylococcus lugdunensis. The patient was discharged on prolonged intravenous antibiotics via a PICC line, achieving a successful recovery.

Discussion:
This case challenges conventional perspectives on fulminant endocarditis, particularly in healthy individuals without traditional risk factors. Coagulase-negative Staphylococci (CoNS) are generally considered nonpathogenic commensals in individuals with a normal immune response. However, the paradigm shifted when Staphylococcus lugdunensis, a member of the CoNS group, was identified as a causative pathogen of infective endocarditis in 1988 by Freney et al. Unlike other CoNS, S. lugdunensis exhibits high virulence, adhering to vessel walls and cardiac valves through biofilm production and binding to von Willebrand factor. The pathogen can cause rapidly progressive endocarditis with valve and septal destruction, necessitating early identification and aggressive intervention for optimal outcomes. The rarity of S. lugdunensis endocarditis emphasizes the importance of recognizing its potential severity and considering it as a pathogen rather than a blood culture contaminant. This case underscores the need for heightened vigilance in diagnosing and managing fulminant endocarditis, particularly when caused by uncommon pathogens like S. lugdunensis. Early recognition and prompt intervention, including surgical measures, are crucial for improving patient outcomes in such challenging cases.
The Great Masquerader: 3 Cases of Bartonella in Renal Transplant Patients

Introduction
Bartonella infections present with a wide array of non-specific clinical manifestations including lymphadenopathy, fever, diarrhea, and hepatosplenomegaly. Literature regarding Bartonella infections in renal transplant recipients is limited. The following 3 cases demonstrate Bartonella infections following renal transplantation at a single center. Standard induction immunosuppression included Thymoglobulin and Solumedrol in all 3 patients.

Case 1
25-year-old female with a history of neonatal left renal vein thrombosis and secondary hypertensive nephrosclerosis leading to ESRD underwent a deceased donor kidney transplant (KDPI 17%. HLA-zero-antigen mismatch. Recipient cPRA 99%. CMV:D-/R+. EBV:D+/R+) with an uneventful course until 1 year later. Maintenance immunosuppression was MMF, Tacrolimus and prednisone. She presented with fevers, diarrhea, generalized fatigue and an unproductive cough who was found to have an AKI. Patient had adopted flea-infested kittens 4 months prior to admission. She reports both kitten and flea bites. Creatinine on admission was 1.5, peaked at 1.7 and returned to her baseline of 1.1-1.3. CT showed numerous ill-defined hypo-attenuating liver lesions. MRI showed diffuse nodular lesions throughout the liver with diffuse enhancement. Concern for PTLD was high. Karius was positive for both EBV and Bartonella. Bartonella Henselae IgG antibody 1: 128 henselae IgM antibody 1:256. She was treated with doxycycline 100 mg twice daily for 28 days. Post-treatment CT scan showed significant decrease in liver nodules.

Case 2
67-year-old male with a history of type 2 DM, CAD, hypertension and multifocal renal cell carcinoma status post bilateral nephrectomies underwent a successful deceased donor kidney transplant (HLA-5 mismatch. KDPI 53% Recipient cPRA 3%. CMV:D+/R-. EBV:R+) until 2 months later when he presented with fevers of unknown origin. He was discharged then re-admitted 2 weeks later for syncope and an AKI but remained afebrile during his second admission. Maintenance immunosuppression was with Myfortic and Envarsus. Creatinine on admission was 6.6, peaked to 7.7. Baseline creatinine 1.8-2. Renal biopsy findings consistent with C3 dominant infection associated glomerulonephritis with numerous neutrophils. Karius was sent for work-up of unknown origin, which was positive for Bartonella. He was positive for both Bartonella Henselae IgG > 1:1024 and Bartonella quintana IgG > 1:1024. TEE showed evidence of vegetation on the non-coronary leaflet of the aortic valve. Patient was treated with doxycycline 100 mg twice daily and rifampin 600 mg daily.

Case 3
41-year-old female with a history of hypertension, EBV viremia, MRSA cellulitis, and ESRD of unclear etiology underwent a successful second deceased donor renal transplant (KDPI 4%, HLA-1 mismatch. Recipient cPRA 100%. CMV:D+/R-. EBV:D+/R+) until 4 years later when she presented with fevers and pyelonephritis. Maintenance immunosuppression was Myfortic, Tacrolimus and chronic prednisone. She was found to have incidental portocaval lymphadenopathy and splenomegaly on CT. Bone marrow biopsy showed normocellular marrow and a PET scan was negative. Bartonella Henselae IgG peaked to > 1:1024 and IgM to > 1:256. She was successfully treated with Azithromycin 500 mg daily for 3 days.

Conclusion
Bartonella often presents in an obscure manner and should be considered in transplant patients presenting with fever of unknown origin and unexplained adenopathy.
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Myxedema Coma Presenting with New Onset Cardiomyopathy and Large Pericardial Effusion.

Introduction: Myxedema coma is a rare medical emergency and potentially fatal if not recognized early. Here we present a case of Myxedema coma presenting with newly diagnosed heart failure and large pericardial effusion.

Case presentation: A 66-year-old female with history of hypothyroidism and coronary artery disease who presented to the emergency department (ED) for significant fatigue, weakness, dyspnea, and mild confusion over the course of few weeks. The patient was not adherent to her medications for almost a year due to multiple social stressors. On presentation, her vital signs were within normal limits and body temperature was 36.5 Celsius. On physical examination, she was alert and oriented but slow to respond. Periorbital edema, bibasilar crackles and hyporeflexia were noted, otherwise normal heart sounds and no murmurs. Electrocardiogram (ECG) shows low voltage QRS complex. Laboratory evaluation revealed anemia with hemoglobin of 7 g/dl, severely reduced Ferritin to 4 ng/dl, significantly elevated thyroid stimulating hormone (TSH) of 350 uIU/ml and severely reduced free T4 level at <0.25 ng/L. Chest imaging reveals significant cardiomegaly for which echocardiogram was performed revealing newly reduced ejection fraction to 30% and large circumferential pericardial effusion without evidence of tamponade. She was treated with Intravenous levothyroxine and hydrocortisone with improvement of symptoms, and she was followed up with serial echocardiograms to ensure stability of effusion and fortunately she did not require intervention. She was started on goal directed medical therapy and discharged to follow up with cardiology and endocrinology outpatient.

Discussion: Myxedema coma is a rare and fatal disease that is mainly described in elderly women. Overt heart failure and ventricular dysfunction are rare cardiac complications in the absence of preexisting cardiac disease. Identifying the associations of these complications with such critical illness is essential and can alter the outcome of the disease.
Challenging Giant Cell Myocarditis: A Case Report and Clinical Insights

Background: In 1905, Saltykow reported a groundbreaking case of Giant Cell Myocarditis (GCM) in a young man with acute heart failure. GCM diagnosis was post-mortem until endomyocardial biopsy (EMB) was evolved as a diagnostic method in the 1970s. This case ignites the awareness about GCM’s significance and the need for early detection.

Case Presentation: A 59-year-old female with multiple comorbidities including neurofibromatosis type I presented with acute onset of dyspnea and generalized swelling consistent with acute on chronic heart failure exacerbation. In addition to medical therapy, workup included echocardiography revealing an ejection fraction of 20%, and coronary angiography which was normal. Therefore, cardiac MRI was performed which showed late gadolinium enhancement in multiple coronary artery territories, indicating myocardial inflammation. EMB was pursued which revealed multifocal inflammatory infiltrates with multinucleated giant cells consistent with GCM. The patient rapidly decompensated after and went into cardiogenic shock requiring mechanical circulatory support device. She was initiated on immunosuppressant with Prednisone and Mycophenolate mofetil; however, no improvement was noted, and she was deemed not a candidate for advanced heart therapies given her significant comorbidities. The patient unfortunately passed away after clinical deterioration.

Discussion: GCM is a very rare and fatal condition, usually with rapid progression, especially if not detected early. Cardiac transplantation is often needed within 5.5 months from diagnosis for most patients; however, recurrence of disease remains a concern. Immunosuppressive therapy may provide some benefit in extending survival rates, however, the most important factor that impacts survival is early diagnosis and initiation of therapy.

Conclusion: Timely diagnosis and a multidisciplinary approach involving immunosuppressive therapy, cardiac support, and consideration of advanced heart therapies are essential in the management of this disease. Further research is necessary to enhance our understanding of GCM’s pathogenesis and develop improved treatment options.
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Ibrahim El-Mair

The Comparison of Using Single Versus Two-Catheter Concept in Diagnostic Trans-Radial Coronary Angiography: A Single-Community Center Experience

Background: Coronary artery anomalies (CAAs) are congenital conditions characterized by abnormal origin or course of the 3 main coronary arteries. Most variations are benign; however, some may lead to myocardial ischemia and/or sudden cardiac arrest depending on where the artery travels.

Case: 66-year-old female presented with non-exertional syncope and hot flashes that occurred while having dinner. She was found hypotensive by EMS and was transferred to the emergency department. Workup revealed orthostatic hypotension, hypokalemia and lactic acidosis with normal troponin levels. EKG showed left axis deviation. An incidental finding of an anomalous origin of the RCA from the left coronary cusp was discovered on chest CT. An echocardiogram and nuclear MPI study were performed and revealed preserved systolic function and no perfusion defects, respectively. Due to negative ischemic workup, the patient was discharged with outpatient coronary CTA that revealed an anomalous RCA arising from the left sinus of Valsalva with an interarterial course.

Discussion: CAAs are discovered in about 1% of the population undergoing coronary angiography. It is usually an incidental asymptomatic finding but can be pathologic with presenting illnesses ranging from chest pain to sudden cardiac death if the artery has an interarterial course. Malignant CAAs with an interarterial course can manifest symptoms.

In our case, the patient presented with non-exertional syncope due to orthostatic hypotension and an anomalous RCA was incidentally discovered on imaging.

Conclusion: With increasing utilization of coronary angiography and CT imaging, more CAAs are being diagnosed, and more variants are being described. Knowledge of normal and variant anatomy is becoming increasingly vital. Classification of CAAs as either benign or malignant helps guide treatment.
A Case of Suspected Hypocomplementemic Urticarial Vasculitis (HUV) and Mini-review of Associated Conditions

Introduction: Urticarial vasculitis (UV) is an uncommon disease process that is defined by cutaneous, serologic, and systemic features. This case reviews the subtypes of UV, highlights its associations, histopathological characteristics, clinical manifestation, work-up, and treatment.

Case Presentation: A 50 year old Caucasian female with a history of nickel allergies, and ductal carcinoma in-remission that was referred to the dermatology clinic with a three-month history of intermittent angioedema and painful urticaria-like rash.

Physical examination was significant for periorbital edema, and angioedema of the lips. Areas of post-inflammatory hyperpigmentation (PIH) were visible over the torso and lower extremities as well.

Work-up revealed elevated ESR, decreased complement C1q and elevated C2, C3, and C4. Negative pertinent work-up included lack of systemic involvement including absence of leukocytosis, eosinophilia, neutrophilia, renal or hepatic involvement. Work-up for associated autoimmune and infectious work-up was also negative.

Patient achieved complete cutaneous remission with mycophenolate mofetil and dapsone daily; biopsy was not attained as her urticaria-like rash resolved, so our differential diagnosis of hypocomplementemic urticarial vasculitis (HUV) was never histologically confirmed.

Discussion: Histopathologically, UV is associated with the deposition of immune complexes within vessel walls, thereby causing damage and triggering inflammation via activation of the classical complement cascade. The inciting factor for these immune complexes is unknown in most occurrences of UV; however, studies reveal an association with autoimmune conditions, underlying malignancies, certain medications, and viruses.

The most significant histological findings of UV include leukocytoclasis and fibrinoid deposits. These findings are indicative of damage to the vessel walls. Immunofluorescent studies reveal depositions of immune complexes, fibrin, or complements in the basement wall membrane in the majority of patients.

Spectrum of Disease: Urticarial Vasculitis - 3 Distinct clinico-histological entities:
- Hypocomplementemic UV Syndrome (HUVS): Severe cases with systemic findings that include recurrent abdominal pain, myalgia, arthralgia, glomerulonephritis, and ocular manifestations. Complement levels are decreased.
- Hypocomplementemic UV (HUV): Limited to cutaneous findings without systemic symptoms. Complement levels are decreased.

Similar-presenting inflammatory conditions:
- Schnitzler’s Syndrome – Rare condition characterized by chronic urticaria, IgM monoclonal gammopathy, and indicators of systemic inflammation.
- Muckwell’s Syndrome – Rare hereditary autoinflammatory syndrome characterized by recurrent episodes of systemic inflammation resulting in urticarial-like rashes, arthralgia, fevers, and sensorineural hearing loss.

Work-up and differential
An array of work-up is necessitated if LCV is confirmed with biopsy and immunofluorescence to evaluate for systemic involvement and associated disease processes.
- CBC with differential – evaluate for leukocytosis, neutrophilia, and eosinophilia
- Renal panel, urinalysis and hepatic panel – evaluate for systemic involvement;
- Complement studies to classify UV
- Auto-immune work-up for auto-immune associated diseases
- Evaluation of underlying infections, malignancies, or drug-mediated reactions

Treatment: Urticarial vasculitis treatment depends on the degree of severity and systemic manifestations. Antihistamines or NSAIDs may be trialed for mild disease including NUV. Systemic manifestation necessitates the use of glucocorticoids; alternatives include dapsone, colchicine, and hydroxychloroquine.

For severe disease, methotrexate, azathioprine, mycophenolate mofetil, and biologic agents can be considered. Referral to appropriate specialties is recommended.
An unexpected escalation: A case of RSV myositis

Introduction:

Viral myositis and post-viral rhabdomyolysis have been previously described in the literature. In most cases, the pathophysiology is assumed to be immune-mediated rather than direct muscle injury. However, Respiratory syncytial virus (RSV) has rarely been the main culprit and has primarily been reported in the pediatric population, as benign acute childhood myositis (BACM) in winter months.

Case presentation:

A 64-year-old female with a past medical history significant for hypertension and benign hepatic cysts presented to the hospital for progressive leg swelling and pain worsening over two weeks. Prior to this, the patient and her husband had a recent viral upper respiratory tract infection. On arrival, vital signs were stable. The examination was only remarkable for mild left lower extremity tenderness and swelling at the ankle. The patient tested positive for RSV during respiratory viral panel testing. AST and ALT were elevated at 691U/L and 394 U/L, respectively (previously within normal limits) and CK was elevated at 12000 U/L. DVT, significant cardiac pathology, and pulmonary embolism were ruled out during the hospitalization. The patient was treated with supportive measures for presumed rhabdomyolysis predisposed by her recent viral infection and subsequently, was discharged home. However, the patient presented to the ED ten days later with severe debility. Previously independent, at this presentation she was unable to walk with evidence of significant muscle weakness, diffuse muscle pain and edema, and a CK level of 31000 U/L. Due to lack of clear etiology, she underwent diagnostic muscle biopsy which showed scattered necrotic and regenerating fibers indicative of an active myopathy with denervation atrophy, proposed to be compatible with immune-mediated necrotizing myopathy consistent with viral etiology. The patient was treated with high-dose steroids with a resultant decrease in CK levels and improvement in functional status. Currently, the patient is on a steroid taper and has enrolled in an inpatient rehabilitation.

Discussion:

The limited cases of RSV myositis previously reported have been in the pediatric population. Moreover, the initial presentation usually was that of severe muscle pain, weakness, and swelling. In our case, the initial presentation was mild unilateral point tenderness and trace unilateral edema which progressed to a completely debilitating picture. The use of steroids resulted in significant improvement in patient outcomes in most reported cases, like ours. Therefore, prompt recognition and management play a crucial part. As an increasing number of RSV cases present in the hospital, progressive myositis and related rhabdomyolysis should be considered as a potential complication. Additionally, this case among others highlights the importance of improving RSV vaccine compliance to prevent such complications, although, we need more data to further understand the impact of doing so.
Residency Program: Trinity Health Livonia
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Not every confusion is UTI

Introduction: Invasive Listeriosis is a treatable but potentially fatal disease if not recognized early. It is a relatively rare condition that mainly affects elderly patients and immunocompromised hosts. Here we present a case of missed Listeria bacteremia in a patient with polymyalgia rheumatica on chronic immunosuppressive therapy.

Case Presentation: Patient is a 76-year-old female with a past medical history of vulvar carcinoma with prior radiation therapy, polymyalgia rheumatica currently on leflunomide 20 mg and chronic prednisone who presented to the emergency department (ED) for fatigue, significant diarrhea, and poor appetite for few days. On presentation, her vital signs were within normal limits. Physical examination revealed decreased level of orientation, otherwise had benign cardiopulmonary and abdominal examination. Laboratory testing revealed hypokalemia, normal white blood cell counts (WBC), pyuria, and bacteriuria on urinalysis. Her symptoms were attributed to possible urinary tract infection. She was discharged from ED with Antibiotics and a tapering dose of prednisone to prevent secondary adrenal insufficiency. The patient was brought back to the hospital in 2 days with worsening confusion, fever, and diarrhea with vital signs indicating sepsis. Repeat laboratory testing was unchanged, with normal WBC. A more thorough Infectious workup was sent including blood and urine culture, and stool testing for C. difficile, bacteria and parasite, and patient was started on broad spectrum Antibiotics. During her stay, blood cultures grew Listeria monocytogenes, and antibiotic regimen was changed to Ampicillin for treatment of listeriosis.

Discussion: Listeriosis has a high mortality rate and should be considered as an important differential in a patient with presentation of confusion and diarrhea especially in high-risk populations; elderly patients, pregnant women, and immunocompromised individuals such as this patient. Early diagnosis and treatment of listeriosis is crucial and can prevent complications of meningitis, encephalitis, and endocarditis. This case emphasized the importance of history-taking and incorporating it into a meaningful differential diagnosis.
IVIG-Induced Aseptic Meningitis in an Adult Patient with Acute Inflammatory Demyelinating Polyneuropathy: A Case Report

Intravenous immunoglobulins (IVIG) therapy is used to treat various autoimmune, immunodeficiency, and inflammatory conditions. One of the rare, but serious, side effects is aseptic meningitis. In this case report, we present a 55-year-old female who experienced IVIG-induced aseptic meningitis for treatment of acute inflammatory demyelinating polyneuropathy (AIDP).

We present a 55-year-old female with multiple comorbidities who presented to the emergency department with a chief complaint of acute bilateral lower extremity weakness for 2 days associated with ascending paresthesia, tingling, and worsening chronic back pain. Additionally, she reported sore throat and intermittent fever in the last few weeks prior to presentation, however, she denied headache and neck stiffness. Physical examination showed tenderness overlying the thoracic spine, 4/5 strength in the bilateral lower extremities, but intact sensation.

Initial work up consisted of a comprehensive metabolic panel, complete blood count with differential, and viral panel including COVID-19 and Influenza testing, which all returned within normal limits. The patient underwent MRI with and without contrast of the entire spine which was within normal limits. A lumbar puncture (LP) was performed and was within normal limits as well. The patient was suspected to have AIDP for which she received treatment with IVIG. On day 4 of treatment, the patient was noted to have headache, neck stiffness, and chills, therefore the IVIG was stopped. A repeat lumbar puncture was performed revealing 129/mm3 total nucleated cells with 77% lymphocytes, 56/mm3 RBCs, glucose 52mg/dl, and protein 35mg/dl. Pathology review revealed small mature lymphocytes. An extensive infectious workup was performed including CSF PCR for Escherichia coli K1, H. influenzae, L. monocytogenes, N. meningitidis, S. agalactiae, S. pneumoniae, CMV, Enterovirus, HSV 1 and 2, HHV 6, VZV, and C. neoformans, which all returned negative. In addition, Gram stain and culture, and Lyme serologies were negative. With the return of the above findings, a diagnosis of aseptic meningitis induced by IVIG was made with the collaboration of a multi-disciplinary team which included neurology and infectious diseases. In addition to IVIG discontinuation, supportive care was started with adequate hydration and symptomatic management with improvement of her symptoms over the following days and she was subsequently discharged to a rehabilitation facility.

IVIG-induced aseptic meningitis is a known, but relatively rare, adverse effect that must be included in the differential if a patient develops symptoms consistent with meningitis during or after treatment. The clinical presentation of aseptic meningitis is very similar to other types of meningitis and work up should include lumbar puncture to rule out infectious etiology first and foremost. CSF analysis generally shows a neutrophil-predominance however this is not sensitive or specific. Symptoms usually occur within the first 48 hours after initiation of treatment, however in our case symptoms developed after 96 hours. Therefore, clinicians should have a high index of suspicion for such a complication throughout the treatment course as identification may warrant immediate discontinuation of IVIG treatment. In most cases, the symptoms are self-limited with conservative treatment, however complications may arise if not detected early.
From Outbreak to Outcomes: COVID-19 and the Subsequent Development of Takotsubo Cardiomyopathy

Background: As we slowly get accustomed to COVID-19 virus, many long-term cardiovascular complications of the infection and the vaccine come to light. One is stress cardiomyopathy, also known as Takotsubo Cardiomyopathy (TC). Almost 100 cases of TC were identified, 60% of which were following the infection. Here we present a case of late diagnosis of TC as a sequel to a COVID-19 infection.

Case presentation: A 61-year-old female with multiple comorbidities including endometrial and colon cancer in remission, presented with worsening dyspnea over several weeks after being diagnosed with COVID 19 infection 4 weeks prior to presentation. The patient’s vital signs and physical examination were benign; however, she was noted to have elevated high sensitivity troponin peaking at 200 ng/L. Cardiac evaluation was pursued, including echocardiography showing newly decreased ejection fraction at 30% with mid to distal wall hypokinesis and basal segment hypercontractility. Coronary angiography revealed normal coronary anatomy. Goal directed medical therapy was initiated with significant improvement of her symptoms and echocardiographic findings within 3 months. Of worth mentioning, the patient was fully vaccinated against COVID-19.

Discussion: TC is usually associated with emotional and/or physical stressor, however, multiple viral infections and recently COVID-19 infection, and even vaccination, have been reported as a cause. The underlying pathophysiology is thought to be due to exaggerated inflammatory response and cytokine release leading to myocardial toxicity and microvascular dysfunction. Despite the outstanding impact of vaccination on preventing COVID-19 pulmonary complications, this case demonstrates that patients are still at risk of cardiovascular complications.

Conclusion: As COVID-19 pandemic slowly transitions towards another flu-like infection, it is pertinent for physicians to be aware of various complications associated with the infection and underlying inflammation leading to increased hospitalization, morbidity, and mortality. COVID-19 infection as the etiology of TC should be considered in the absence of other stressors.
Purpose: Overall Survival of Multiple Myeloma has significantly improved in the past decade with the advent of multiple treatment modalities such as Proteasome Inhibitors, Immunomodulatory drugs, anti-CD 38 antibodies and CART therapy. We hereby address the substantial disparities that exist in CART trial access among various regions in the United States.

Methods: Data about the clinical trials are obtained from clinicalTrials.gov, the largest clinical trial registry database using the keywords Multiple Myeloma, MM, CAR-T.

Inclusion Criteria:
Only the studies which have one or more open sites in the United States are taken into consideration
All Interventional Multiple Myeloma CAR-T studies as of January 14, 2024
Including Active, recruiting, not recruiting and Completed Trials

Exclusion Criteria:
All terminated and observational trials.
After removing all the duplicates, a total of 74 trials are included.

Results: A total of 74 unique clinical trials distributed across 399 sites across the United States with 7431 patients enrolled or expected to enroll are included in the analysis. Among them, the majority of the trials were Phase I (34), followed by Phase II (22), Phase I/II (9), Phase III(5) and Not Available (3). Most of the trials were funded by Industry. Only, 9 trials across the US were funded by NIH, 52 trials were funded by Industry and 33 trials were funded by non-industry.

These 75 trials were distributed across 34 states, with an average of 7.98 trials per state (range 0–33).

Southern states have the most number of trial sites, 136 (34.1%) followed by North Eastern states 98 (24.6%), Mid-Western 94 (23.6%) and Western states 71 (17.8%). Seventeen states had no access to CART trials. These include Alaska, Delaware, Hawaii, Idaho, Louisiana, Maine, Mississippi, Montana, Nevada, New Mexico, North Dakota, Rhode Island, South Carolina, South Dakota, Vermont, West Virginia and Wyoming. The highest number of clinical trials is clustered in California, 33, followed by New York (31) and Massachusetts (27).Out of the 75 clinical trials, only 11 clinical trials are available in the state of Michigan, contributing a mere of 2.8%. Majority of these trials are in Urban regions,

Conclusion: Geographic disparities exist in clinical trial access. Patients in 17 states (32%) did not have any access to MM clinical trials. Participation in these trials are feasible only for people living in urban areas as most of the trials are in the urban regions, creating disparities as logistics and financial challenges become an issue for people living away from these regions. We hereby emphasize the need for necessitating the strategies to improve access to these trials, especially for marginalized population.

Newer antibodies T cell engagers may be one of the potential options in the patients at community settings. This is yet another area to be explored in the future.
Exploring The Role of Infectious Triggers in Causation of Sarcoidosis

INTRODUCTION: Sarcoidosis has complex pathogenesis, with multiple innate and external risk factors. Microbial organisms have been implicated as a trigger in those with genetic predisposition. This systematic review of case reports/series aims to determine patterns in pulmonary sarcoidosis which was diagnosed along with or post an infectious episode.

METHOD: Case reports/series published from January 2015 to September 2023 were searched through PubMed. Subsequently, articles were exported to Rayyan software. Each study was reviewed by two investigators based on inclusion and exclusion criteria. The data from these selected case studies of patients with pulmonary sarcoidosis diagnosed alongside or after an infection, were then collected and analyzed for clinical, laboratory and radiological abnormalities. The study was IRB exempt.

RESULT: Total 21 patients were included from 19 case reports and one case series. Mean age of the patients was 45 years, ranging from 12-65 years. Total 12 of them (57%) were male. Most common presenting symptoms were cough (47.6%) and dyspnea (43%). Comorbidities noted in the patients are Type II Diabetes Mellitus, Hypertension and Chronic Kidney disease (14% each). Stage III sarcoidosis was diagnosed in 33.3%, Stage II and concomitant extra-pulmonary involvement were seen in 28.5% each. The predominant infectious etiology recognized were Mycobacterium tuberculosis (38%), SARS-CoV-2 virus (24%) and Propionibacterium acnes (14%). 8 of 21 patients (38%) had abnormalities on chest radiography. On Computerized tomography of the chest, most common radiological abnormalities noted were nodular opacities (47.6%) and mediastinal lymphadenopathy (38%). ACE levels were abnormally elevated in seven patients and only one had hypercalcemia. Patients were treated with systemic steroids (61.9%) and methotrexate(14.2%), with clinical and radiological resolution noted in 52.3% of the study population.

CONCLUSION: Our systematic review focussed on clinical presentation, blood and radiological patterns, treatment administered in those who were diagnosed with sarcoidosis precipitated by an infection. It is important to be aware of such a complication in those who do not improve after appropriate antibiotic therapy for a recognized infectious etiology.
Concomitant Herpes Simplex Virus (HSV) 1 and Adenovirus ARDS in an immunocompetent patient – Innocent bystander or Vicious culprit

Introduction:

Acute respiratory distress syndrome (ARDS) is associated with a very high mortality (40 to 60%). Identification of the etiology and specific treatment, in addition to guideline-based ventilation strategies improves outcomes. We present an immunocompetent woman with Herpes Simplex Virus 1 (HSV1) and Adenovirus-related severe ARDS successfully managed with mechanical ventilation and early steroid therapy.

Case Presentation:

A 40-year-old healthy woman presented to the emergency department (ED) due to high-grade fever and chills, and after two days of fever, she developed cough with hemoptysis, dyspnea and pleuritic chest pain. In ED, she was afebrile, tachycardic (heart rate: 116/min), tachypneic (RR 24/min), with a blood pressure of 135/91 mmHg. She was hypoxic, requiring high flow nasal cannula (HNFC) oxygen at 40 L/min to maintain oxygen saturation > 90%. Chest x-ray showed diffuse bilateral interstitial opacities, suggesting pulmonary edema or atypical pneumonia. She had a PaO2/FiO2 of < 100 indicating severe ARDS. Given the history of fever, pulmonary infection was suspected. Echocardiogram revealed normal cardiac function with an ejection fraction of 55-60% ruling out pulmonary edema. In addition to bacterial pneumonia, viral pneumonia, acute eosinophilic pneumonia (AIP), and diffuse alveolar hemorrhage (DAH) were considered. The patient was started on Ceftriaxone, Azithromycin, and intravenous methylprednisolone. SARS-CoV-2, respiratory syncytial virus, Influenza A/B, IgM mycoplasma antibodies, blood/sputum culture, and urine Legionella antigen were negative. Workup for DAH revealed positive antinuclear antibody (1:360), but negative extractable nuclear antigens, antineutrophil cytoplasmic antibodies, and anti-glomerular basement membrane antibodies. The patient failed HFNC, requiring endotracheal intubation with mechanical ventilation within 48 hours of admission. Bronchoalveolar lavage showed a neutrophilic predominance (86%) and positive HSV1 PCR and Adenovirus PCR. A diagnosis of concomitant HSV1 and Adenovirus ARDS was made. She was continued on in intravenous steroids due to severe ARDS. The patient gradually improved and was extubated after 10 days. She is currently requiring 4 L/min oxygen.

Discussion:

HSV 1 PCR positivity is seen in about 16-65% of patients admitted to the ICU. Interpretation of positive test results should be done cautiously. HSV1 isolation from lower respiratory tract may be just viral shedding, and proof of invasive disease may need bronchoscopic biopsy to see cytopathic effects. Clearcut benefit from acyclovir treatment has not been shown. Adenovirus may also lead to severe ARDS, characterized by high fever and dyspnea with bilateral diffuse infiltrates and quick progression to ARDS. Non-invasive and mechanical ventilation may fail requiring extracorporeal membrane oxygenation (ECMO) in adenovirus ARDS and is associated with high mortality. Early cidofovir and ECMO have been promising in some reports. Steroids are given in ARDS for steroid responsive diseases (AIP, COVID-19), associated refractory septic shock, and early in moderate to severe ARDS (PaO2/FiO2 ratio < 200). We used steroid early due to severe ARDS. Coinfection with HSV1 and adenovirus leading to ARDS in an immunocompetent adult is exceedingly rare. We did not show proof of HSV1 invasive disease, but ours is a report of a successful outcome of adenovirus ARDS with mechanical ventilation and early initiation of steroid therapy.
2024 Michigan Chapter ACP and SHM Michigan Chapter Resident and Medical Student Day
Resident Posters

Resident Poster # 143  Category: Clinical Vignette

Residency Program: Trinity Health Oakland
Presenter: Rishab Prabhu
Additional Authors: Nikhil Vojjala, MD; Sangeetha Krishnamoorthy, MD; Ibrahim Azar, MD; Muhammad Nadeem Khan, MD; Shireesh Gadgeel, MD; Geetha Krishnamoorthy, MD FACP

Reno-Duodenal fistula: An unusual cause of polymicrobial sepsis in a lung cancer patient (Turning Tumor titbits into tumor trouble)

Introduction:

Sepsis in patients with underlying malignancies can be due to many causes such as infections due to neutropenia, pneumonia, and gastrointestinal infections. Fistula formation leading to sepsis is very rare. Factors associated with gastrointestinal fistulization as a possible cause of sepsis include recent radiation therapy, gastrointestinal surgery, and polymicrobial growth on blood cultures, especially gut microbiota.

Case description:

A 60-year-old man with hypertension, Hyperlipidemia, and non-small cell lung cancer presented to the emergency department (ED) with a history of fever with chills of one-day duration. Family members reported that the patient was dull appearing and had difficulty in word finding, for the past week. Of note, the patient’s non-small cell lung cancer initially responded to therapy, then became resistant to multiple lines of therapy including progression while on a clinical trial. The patient was then placed on Gemcitabine-based chemotherapy and underwent radiation therapy for right renal metastasis three months before presentation as a palliative intent. On presentation to the ED, he was febrile (103.6°F), hypotensive (blood pressure 70/50 mm Hg), tachycardic (121/min), and hypoxic saturating 87% on room air. Physical examination revealed an ill-appearing man with tachypnoea. Neurological examination showed that he was oriented only to a person. There was right costovertebral angle tenderness with a resonant percussion note in the lower posterior thorax. He was resuscitated with intravenous fluids, and oxygen, and started on antibiotics as per sepsis protocol. A computerized tomography (CT) scan of the chest with an angiogram showed multiple nodules with cavitation and no pulmonary embolism. The patient’s vital signs improved with fluid resuscitation and antibiotics initially. Blood cultures grew multiple atypical organisms both bacteria and fungi. Bacteria were Lactobacillus acidophilus, Streptococcus, and Leuconostoc mesenteroides, and fungi were Saccharomyces Cerevisiae and Candida Albicans. Given polymicrobial sepsis, the gut was suspected as the source. CT scan with contrast of the abdomen showed a right upper pole renal mass. Air was seen within the right renal parenchyma, with a fistula between the C-loop of the duodenum and the anterior surface of the kidney. Esophagogastroduodenoscopy confirmed the large fistulous opening in the duodenum and extending into the kidney. Endoscopic closure of the fistulous opening was attempted but was not possible due to adherence to the kidney. Trans-esophageal echocardiogram did not show any vegetation. A CT scan brain showed metastatic disease. Because of multiple medical comorbidities, poor performance status, and progressive disease, the patient and his family members requested hospice care.

Reno-duodenal fistula is a very rare and unusual cause of sepsis in patients with malignancy. Polymicrobial blood cultures prompted us to think about gut translocation. Early identification may make surgical intervention possible. Though in this patient, we were not able to control the source of sepsis, as a minimally invasive procedure, endoscopy should be done with an attempt to close the fistula, if possible, in poor surgical candidates. Poor performance status, diffuse metastatic disease, and recurrent hemodynamic instability excluded the surgical treatment options for this patient.
Unraveling the Bone-Weakening Effects: Tenofovir-Induced Severe Osteoporosis – A Case Study

Introduction: Tenofovir Disoproxil Fumarate (TDF) is one of the first-line antiretroviral regimens for the treatment of HIV infections with a rare side effect of bone toxicity. We describe a case of an HIV-positive man who had been using TDF for 9 years and presented to the emergency room with multiple fractures. The bone fracture investigation identified severe osteoporosis. TDF was discontinued, treatment was focused on controlling the bone disease through vitamin D and calcium supplementation which showed slow improvement in his bone mass density (BMD). This abstract provides a concise overview of the mechanisms underlying tenofovir-induced bone loss and management strategies.

Case Presentation: A 54 White M with history of Anxiety, Depression, and HIV on efavirenz-emtricitabine-tenofovir (ATRIPLA) 600-200-300 mg daily for 9 years, presented to the hospital with right-sided hip pain after a trivial fall. He was found to have a femoral neck fracture on X-ray, which was fixed with open reduction and internal fixation. The younger age of presentation prompted further evaluation with DEXA scan, which showed osteoporosis in the lumbar spine and the femoral neck. His PTH, Vitamin D, and Ca levels were normal. Other causes of osteoporosis were ruled out which suggested that osteoporosis could be secondary to HIV medication. His Atripla was then changed to abacavir, dolutegravir, and lamivudine (Triumeq) and was started on Fosamax, Vitamin D, and calcium supplements. After a brief period, he stopped taking medication due to fear of osteonecrosis, following which DEXA showed stable osteoporosis without improvement. Later, the patient was switched to Forteo for another year and was restarted on Fosamax weekly doses. Recent DEXA showed significant improvement in BMD in the spine and the hip and has been compliant with medications and monitoring with a DEXA scan every 24 months.

Discussion: Tenofovir is a nucleotide analog which represents the foundation of highly active antiretroviral therapy (HAART) and was granted approval in 2001 for HIV treatment. Despite its success, there have been clinical reports of tenofovir-associated side effects such as bone loss.

Data implicating TDF exposure in bone pathology are limited. Some prior research indicates simultaneous inhibition of osteoblast and stimulation of osteoclast activity, leading to bone loss. It can cause proximal renal tubular dysfunction leading to hypophosphatemia, and calcium abnormalities that can disrupt bone mineralization and cause hyperparathyroidism, further exacerbating bone loss. Tenofovir has been associated with mitochondrial toxicity, which can impair cellular function interfering with normal bone metabolism.

Tarantal et al. proposed that persistent prenatal and postnatal tenofovir can affect bone metabolism in some animals. Other studies suggested osteopenia and osteoporosis are more detected with TDF regimens than abacavir/lamivudine. Fractures mainly involved the femoral neck, in comparison to protease inhibitors, which involved lumbar spine. Tenofovir alafenamide, an improved tenofovir formulation, with less side effects and without compromising viral suppression.

Our case emphasizes the importance of physicians anticipating and appropriately managing the adverse effects of TDF on bone, calcium and vitamin D supplements, regular monitoring of BMD, lifestyle modifications, and consideration of alternative antiretrovirals with a more bone safety profile.
The Perfect Storm: Co-occurrence of Agranulocytosis and thyroid storm successfully managed with lithium

Introduction:

Thyroid storm is a medical emergency commonly precipitated by factors including surgery, sepsis, and stress. Recently there has been increased awareness of thyroid storm in the setting of COVID-19 illness. However, the literature is still limited to a few case reports. Methimazole is used frequently in the management of Graves’ disease. Thyroid storm is a mimicker of sepsis. Co-existent severe agranulocytosis and thyroid storm may be extremely rare but presents a therapeutic conundrum. We present a patient with thyroid storm with severe neutropenia precipitated by COVID-19 infection and methimazole use and managed successfully with lithium. Agranulocytosis precluded the use of methimazole and propylthiouracil.

Case presentation:

A 32-year-old woman, with Grave’s disease, controlled on methimazole and metoprolol presented to the Emergency Department (ED) with fever and palpitations of one-day duration. The patient was found to be COVID-19 positive 5 days prior and started on Molnupiravir for mild COVID-19 illness. On presentation to the ED, she was febrile (temperature: 102.3 F), tachycardic (136/min), and a respiratory rate of 22/min, with normal oxygen saturating on room air (98%). Physical examination revealed an anxious woman with fine tremors. Vascular bruit was heard over the thyroid gland. An electrocardiogram (ECG) showed sinus tachycardia. Laboratory workup revealed severe leukopenia with a White Blood Cell Count (WBC count) of 2100 cells/mm3 with an absolute neutrophil count of 63 cells/mm3 and hypokalaemia (3.3 mEq/L). Methimazole was stopped due to agranulocytosis. Differential diagnoses considered were bacterial sepsis (febrile neutropenia), progressive symptoms related to COVID-19 infection, and thyroid storm. Serum Thyroid Stimulating Hormone level was < 0.01 mclU/ml (Range: 0.45-5.33) with elevated T4 and T3 {10.4 pcg/ml (range: 2.1-4.1 pcg/ml) and 4.34 ng/dl (range: 0.61-1.24 ng/dl)}. Burch-Wartofsky Point Scale (BWPS) score was 60 points, highly suggestive of thyroid storm. The final diagnosis of Thyroid storm was made after ruling out all sources of sepsis, with negative cultures, and imaging. Precipitating factor identified was the COVID-19 infection. Co-existence of agranulocytosis prevents us from using propylthiouracil and methimazole for treatment of thyroid storm. The patient was started on non-selective beta blockers, intravenous fluids, potassium supplementation, and steroid therapy to suppress T4 to T3 conversion. Lithium was started given her neutropenia and thyroid storm. She responded to treatment with oral lithium and a non-selective beta blocker and was discharged home after the resolution of the thyroid storm. Agranulocytosis resolved, with WBC count increased to 8300 cells/mm3 after one week of follow-up.

Agranulocytosis and thyroid storm are an extremely rare combination and ours is first clinical vignette as per our knowledge. Lithium may be a suitable agent in such cases which counteracts thyroid storm and leukopenia through different mechanisms. Use of lithium should be done with expert consultation, since evidence is currently limited to case reports and since lithium can cause hyperthyroidism as well.

Learning points we highlight are:
1. COVID-19 has been reported as a precipitating factor for thyroid storm.
2. Co-existent thyroid storm and agranulocytosis are very rare, and lithium can be a useful option in such cases with expert consultation.
Hyponatremia with progressive neuropsychiatric symptoms prompts investigation for LGI1 (Leucine-Rich glioma inactivated-1) antibody encephalitis?

Introduction:
Limbic encephalitis (LE), a rare neurological disorder characterized by limbic system inflammation, presents with a wide range of symptoms, including subacute cognitive decline, short-term memory loss, faciobrachial dystonic seizures (FBDS) and behavioral abnormalities (1). Etiologies include infections, tumors, and autoimmune reactions. During the disorder, 60-80% of patients develop hyponatremia (1) that might delay the diagnosis of encephalitis. The etiology for hyponatremia remains unclear but is presumed secondary to SIADH. Herein, we report a man with progressively worsening dementia and hyponatremia due to autoimmune Leucine-Rich glioma inactivated-1 (LGI-1) encephalitis.

Case presentation:
A 67-year-old man with prior stroke, hypertension, coronary heart disease, and depression presented to the emergency department due to seizure-like activity. Patient was sleeping while he experienced sudden stiffness and jerking movements of both upper and lower limbs lasting for a minute. The patient appeared confused and was brought to the hospital.

For the past nine months patient has been experiencing multiple neuropsychiatric symptoms including vivid dreams, hallucinations, delusions, mild cognitive impairment with some gait imbalance and urinary incontinence. He had a prior admission due to new onset cognitive impairment. He was seen by neurology, and computerized tomography (CT) scan head, magnetic resonance imaging (MRI) brain and electroencephalogram were negative. Considering encephalitis, an autoimmune panel was ordered that was later positive for Leucine-Rich glioma inactivated 1 antibody (LGI-1 IgG). On this admission with worsening mentation and recurrent seizures episodes, neurology was reconsulted.

Laboratory investigations were significant for hyponatremia (130 mmol/L), serum osmolality was 264 Mosmol/K. Further workup showed urine sodium of 49 meq/L, urine osmolality of 470 mosmol/K and uric acid 2.8 mg/dL. Thyroid Stimulating Hormone and cortisol levels were normal. Due to euvolemic hypoosmolar hyponatremia, syndrome of inappropriate antidiuretic hormone (SIADH) was diagnosed. CT chest and abdomen was negative, Positron Emission Tomography scan of the body was normal, done in a search for primary malignancies. Repeat brain imaging was normal. SIADH was attributed to ongoing underlying LGI-1 encephalitis. The patient was started on levetiracetam 500 mg twice daily for seizure control with salt tablets and IV immunoglobulins (IVIG) for 5 days. There was improvement in psychiatric/behavioral symptoms and sodium trended up with fluid restriction and salt tablets. Post IVIG, the patient was started on tapering dose of oral steroid to reduce the risk of recurrent seizures, hyponatremia, and behavioral problems with advice to follow-up as outpatient with neurology.

Discussion:
LE has been considered a paraneoplastic phenomenon, most commonly in association with underlying lung cancers [2]. Recently it has become apparent that LE may occur in the absence of malignancy due to autoimmune etiology. In animal studies, LGI-1 is highly expressed in the hypothalamus, indicating a possible inflammatory response. ADH (antidiuretic hormone) secretion can be disrupted by this inflammatory reaction, resulting in hyponatremia. This clinical vignette raises awareness of anti-LGI1 autoimmune encephalitis in patients with rapidly progressive dementia with hyponatremia, highlighting the importance of early recognition and treatment with ongoing follow-up.
Shoulder effusion: not a crystal-clear diagnosis

Introduction:

Shoulder complaints are common in the primary care office. Pseudogout is an overlooked cause of acute monoarthritis of the shoulder. Presentation can be similar to other diagnoses, including septic arthritis, which can coexist with pseudogout.

Case Description:

An 83 year old male with a history of gout presented to the primary care clinic with acute onset right shoulder pain. Symptoms had been present for one week without trauma or inciting event. Surgical history was notable for remote right rotator cuff repair. On exam, he was afebrile and nontoxic. Shoulder exam showed tenderness to palpation at the acromioclavicular and glenohumeral joints, severely limited range of motion in all directions, and a shoulder joint effusion. Given lack of constitutional symptoms, inflammatory markers and x-rays were obtained to differentiate between infectious, inflammatory, mechanical, or malignant etiologies. Laboratory work-up demonstrated no leukocytosis, CRP 19.9, ESR 96. Shoulder x-ray showed greater tuberosity cystic changes and proximal migration of the humeral head consistent with rotator cuff pathology, mild glenohumeral arthritis. Given elevated inflammatory markers, he was sent to the ED. Arthrocentesis revealed synovial fluid with 20K WBCs, 97% neutrophils, and crystal exam revealed calcium pyrophosphate crystals. He was treated with colchicine, steroids, and discharged home. Synovial fluid culture subsequently grew gram positive cocci in clusters. He was readmitted and started on vancomycin. Cultures ultimately speciated to Staph lugdunensis. Orthopedics and Infectious Disease were consulted and he was discharged on colchicine and an extended course of linezolid with outpatient follow up. Subsequent shoulder ultrasound demonstrated complete supraspinatus and infraspinatus tears. Follow up is ongoing and he has had continued clinical improvement.

Discussion:

Shoulder pain is frequently encountered in primary care and has a broad differential. In this case, the absence of trauma and presence of an effusion was most consistent with acute monoarthritis. The differential for acute monoarthritis without trauma includes septic arthritis, crystalline arthritis, autoimmune disease, and malignancy. While the shoulder joint is less frequently affected by crystalline arthropathy, it should be considered in acute onset joint pain with inflammatory features. Symptoms such as significantly reduced range of motion, erythema, and effusion warrant further evaluation beyond shoulder x-ray. This should include inflammatory markers and arthrocentesis. In this case, while the synovial fluid count and visualization of calcium pyrophosphate crystals were consistent with inflammatory arthritis, it has been documented in the literature that approximately 5% of crystalline arthropathies have concomitant septic arthritis. While the patient’s positive fluid culture could have been a contaminant, treatment for presumed septic arthritis was warranted. Finally, crystalline arthropathy can occur more frequently in a diseased joint. Ultrasound revealed a complete rotator cuff tear, which likely predisposed to the flare.

Conclusion:

Crystalline arthropathy must be included in the differential of acute nontraumatic monoarthritis, especially with effusion, and evaluated with joint aspiration.
Platypnea-orthodeoxia syndrome: A unique type of hypoxia

Introduction: Platypnea-orthodeoxia syndrome (POS) is a unique clinical syndrome seen in specific types of hypoxia, categorized by the decrease in oxygen saturation by more than 5% or the decrease in arterial oxygen pressure by more than 4 mmHg when transitioning from recumbency to standing. The differential is limited to intracardiac or intrapulmonary shunts. Prompt identification is crucial as delayed closure can result in progressive hypoxia, heart failure, or neurologic complications.

Case presentation: A 68-year-old woman with a notable history of hypothyroidism, 10-15 pack-year smoking history, asbestos exposure, and recent diagnosis of presumed provoked deep vein thrombosis (DVT)/pulmonary embolism (PE) on apixaban, presented to pulmonology for a second opinion of a three month history of persistent and progressive hypoxia requiring 2-4L nasal cannula at home. She had previously had an echocardiogram and a CT chest at an outside hospital that were reportedly unremarkable. During her pulmonary function testing (PFT), which was remarkable only for persistent hypoxia, she was noted to require up to 15L nasal cannula to maintain saturations >90%, so she was sent to the emergency department for further evaluation. Initial evaluation, including VBG, CBC, CMP, TSH, ESR, BNP, and troponins as well as EKG were unremarkable. CTPE showed scattered bronchial thickening in the lung bases, but no evidence of clot.

The patient was admitted for further evaluation at which point she was noted to have worsening hypoxia and increased oxygen requirements when sitting or standing, consistent with POS syndrome. Further evaluation with high-resolution CT scan showed mild interstitial lung disease and deep venous ultrasound was negative for residual DVT. Notably, transthoracic echocardiogram revealed a large right to left shunt on bubble study. Cardiology was consulted, at which time cardiac MRI was ordered and showed a large sinus venous atrial septal defect (ASD) with associated partial anomalous pulmonary venous return (PAPVR) from right superior pulmonary veins to the superior vena cava (SVC) along with normal biventricular size and systolic function. She underwent diagnostic cardiac catheterization which revealed two right upper pulmonary veins returning to the posterior caudal SVC before subsequent drainage across the sinus venosus defect with right ventricle end diastolic pressure (RVEDP) of 5 mmHg and Qp:Qs of 1.4:1. She subsequently underwent transcatheter SVC stent placement to occlude the sinus venosus ASD. Repeat transthoracic echocardiogram showed a trivial right to left shunt by bubble study. Clinically the patient had immediate reduction in her oxygen requirements discharging on room air.

Conclusion: This case illustrates the importance of careful physical examination in patients with hypoxia as well as maintaining a broad differential diagnosis. Although this patient presented later in life with a rare diagnosis, careful history and a well-done bubble study were able to reveal a previously undiagnosed anomaly in a patient that was not following the typical course of hypoxia or recovery from a PE. This case highlights the rapid clinical improvement seen in POS following accurate diagnosis and prompt intervention.
Huge abscess collection following Hernia repair surgery caused by Trueperella Bernardiae: a case report

Surgical site infections affect a small percentage of patients, ranging from 0.5% to 3%, but can still cause significant morbidity and mortality after surgery. Trueperella Bernardine, a Gram-positive coccobacillus, is typically considered a contaminant found in the normal skin flora and oropharynx. However, it has become increasingly associated with infections in surgical wounds, prosthetics, and the bloodstream.

A 73-year-old female with a past medical history of hypertension, atrial fibrillation, and pulmonary embolism was admitted to the hospital following a mechanical fall. Fractures were ruled out during the initial evaluation. The patient experienced diffuse abdominal pain of gradual onset, increasing fatigue, nausea without vomiting, and reduced appetite over the week preceding the fall. Notably, she had undergone laparotomy, enterolysis, and ventral abdominal hernia repair with mesh placement one month earlier. The patient denied experiencing fever, chills, or sweats.

Upon initial examination, the patient was alert, oriented, and vitally stable. The abdominal assessment revealed asymmetric distension with bulging on the right side. Additionally, a well-healed midline abdominal incision was observed with no discharge or dehiscence. Mild diffuse tenderness was noted upon palpation.

Laboratory results showed a white blood cell count of 7.8, hemoglobin level of 8.6, creatinine of 1.18, and blood urea nitrogen of 44; other chemistry parameters were unremarkable. An abdominal and pelvic CT scan revealed an extensive fluid collection (40x22x18 cm) in the subcutaneous tissues overlying the anterior abdomen/pelvis, concerning for a hematoma or an infectious/inflammatory process.

In the emergency department, the patient's condition deteriorated as she became drowsy, and a copious purulent discharge was observed from her abdomen. Consequently, she was started on ceftriaxone, vancomycin, and metronidazole. The surgical team opted to proceed with urgent debridement and washout of the abdominal wall. Two days later, she was sent to the operation room for a second debridement and washout procedure. The older mesh had incorporated well into the abdominal wall, so it was kept in place. Four drains were left underneath the subcutaneous fat. Wound cultures obtained during the procedure grew Trueperella Bernardine. Antibiotics were switched to ampicillin-sulbactam. The patient experienced clinical improvement over the following days and was discharged after a total of 10 days of antibiotics. She was sent home on amoxicillin-clavulanic acid for an additional five days. She followed up with the surgery clinic one month later and reported significant improvement, denying pain, fever, or chills. She had minimal sanguineous drainage with occasional purulent drainage, which was attributed to a seroma rather than a deep tissue infection. As such, drains were removed, and wounds were closed.

This case highlights the need for greater awareness of Trueperella Bernardine as a causative organism in surgical wound infections. It also underscores the importance of further investigating this organism, as it may have more significant pathogenic potential than previously recognized.
Glomerulopathy in the setting of Morbid Obesity with controlled Diabetes

Introduction: Obesity is a very common and complex presentation in the US. Class 3 obesity is defined as a BMI of 40 or higher. The prevalence of obesity is rapidly increasing across the world and all sections of society. Obesity-related renal disorders have surged tenfold in recent years, with obesity causing increased glomerular filtration rate (GFR) and glomerulomegaly. In type 2 diabetes, heightened hyperfiltration leads to irreversible kidney damage and eases the progression to end-stage renal disease (ESRD). The initial presentation is nephrotic range proteinuria and can be distinguished from Focal segmental Glomerulosclerosis (FSGS) by glomerulomegaly and less severe foot process effacement band without a complete nephrotic syndrome. Further studies are needed to explain why patients with obesity-related glomerulopathy do not develop complete nephrotic syndrome.

Case Description: A 48-year-old male with a past medical history of type 2 diabetes mellitus, obesity class III, and associated chronic kidney disease stage 3, presented to the emergency department complaining of increased lower extremities bilateral swelling, exertional dyspnea, and reduced urine output over the last week. Upon examination, he exhibited a significantly marked bilateral pitting edema in the lower extremities. Vital signs on admission were within normal limits. However, his BMI measured 54.6 kg/m2. Laboratory investigations revealed elevated serum creatinine levels (1.9 mg/dL) and proteinuria (627 mg/dl). Glycosylated hemoglobin (HbA1c) was 7.8%, indicating optimal glycemic control. Abdominal ultrasound was suboptimal due to body habitus and ascites. Abdominopelvic CT scans showed atrophic kidneys. 24-hour urine collection confirmed the presence of significant proteinuria. A renal biopsy was done during admission which revealed glomerular hypertrophy, mesangial expansion, and evidence of focal segmental glomerulosclerosis. The patient was started on a renin-angiotensin-aldosterone blockade medication, SGLT 2 inhibitor, and was extensively counseled on weight loss and was referred to bariatric surgery.

Discussion: This case demonstrates the coexistence of obesity and diabetes which poses a significant risk for the development of obesity-induced nephropathy. Obesity contributes to glomerular and tubular injury through different mechanisms such as insulin resistance, chronic inflammation, lipotoxicity, and activation of the renin-angiotensin-aldosterone system, leading to increased glomerular filtration and proteinuria ultimately causing FSGS and severe renal damage. These factors, combined with the metabolic perturbations seen in diabetes, create a synergistic effect, accelerating the progression of renal damage.
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Comparative Efficacy of Spironolactone and Eplerenone in Chronic Heart Failure  

Introduction: Chronic heart failure is a prevalent clinical syndrome associated with significant morbidity and mortality, affecting approximately 6.5 million Americans over the age of 20. Mineralocorticoid receptor antagonists (MRAs) have been integral to HF therapy for the past 16 years, with spironolactone and eplerenone being the recommended options. This systematic review aims to contribute evidence-based insights into the comparative efficacy of these MRAs specifically Spironolactone and Eplerenone, in the treatment of chronic heart failure (HF). The primary endpoint evaluated is all-cause mortality, with secondary endpoints including left ventricular ejection fraction (LVEF) improvement, cardiovascular mortality, and HF-related hospitalization.  

Methods: A comprehensive search strategy was employed, utilizing PubMed to identify randomized controlled trials (RCTs) conducted from 2010 to 2023. The inclusion criteria encompassed RCTs with active treatment of spironolactone or eplerenone in adults (age > 18 years), without exclusions based on sex, race, sample size, follow-up duration, or language. Three relevant RCTs were included in the final analysis.  

Results: The included studies demonstrated consistent findings in favor of eplerenone over spironolactone. Nabati et al. (2021) (number of patients 85) reported greater improvements in LV systolic function and left atrial diameter with the addition of eplerenone (25 mg) to optimal HF therapy compared to spironolactone. Naser et al. (number of patients 142) (2023) highlighted favorable effects of eplerenone (25 mg) on cardiac remodeling parameters, along with a reduction in cardiovascular and all-cause mortality when compared with spironolactone. Zannad et al. (number of patients 2737) (2011) showed that eplerenone (up to 50 mg daily) significantly reduced the risk of death and hospitalization in patients with systolic heart failure.  

Discussion and Conclusion: Mineralocorticoid receptor antagonists, such as eplerenone and spironolactone, have proven benefits in improving survival and reducing morbidity in patients with HF and reduced ejection fraction. Eplerenone, as evidenced by the reviewed trials, demonstrates superiority over spironolactone concerning all-cause mortality, cardiovascular mortality, HF-related hospitalizations, and echocardiographic improvement of LVEF. The findings emphasize the clinical significance of choosing eplerenone as the preferred MRA in the management of chronic heart failure.  

References:  
Shagreen patch on the face in a patient with Tuberous sclerosis Complex - A Uncommon Site

Introduction:

Tuberous Sclerosis Complex (TSC) is a rare neurocutaneous disorder that includes multiple variations of skin changes that can occur throughout the body. The most common skin findings include Ash leaf marks which are often present from birth; facial angiofibroma’s which commonly occur around the cheeks and nose starting in childhood, cephalic plaques on the forehead that are raised and fibrous, Shagreen patches which are commonly found on the torso or lower back, and ungual fibromas around or under the nails. While these skin conditions can commonly be found in patients with TSC, it is rare to find Shagreen patches outside of the torso or lower back. Here, we present a case of a Shagreen patch found on the patient’s face.

Case Report:

A 53-year-old African American female with a past medical history significant for tuberous sclerosis, seizure disorder, schizophrenia, and cocaine use presented due to altered mental status and hypothermia, initially as a Jane Doe with an unknown history. Per history, she had been sitting in a pool of water outside her home for an undisclosed amount of time. On arrival at the emergency department, she was altered and unable to answer questions appropriately. During physical examination, she was noted to have multiple skin findings consistent with TSC – facial angiofibroma, confetti hypopigmented macules, and ash leaf macules on the torso and back. She had two hyperpigmented face plaques with irregular borders, and the surface increased skin markings and nodularity, consistent with the morphology of Shagreen patches. Due to the patient’s initial presentation, she underwent CT head, CT abdomen/pelvis, and CT lumbar spine, which were significant for multiple cystic lesions in the bilateral kidneys and multiple sclerotic lesions in the lumbosacral spine. Due to the confluence of renal and spinal lesions, there was initially concern for renal cell carcinoma, however further delineation with MRI confirmed that renal masses were consistent with angiolipomas, a known complication of TSC, and sclerotic bone lesions consistent with TSC. During hospitalization, the patient was also treated for schizoaffective disorder with risperidone and long acting paliperidone depot injection.

Discussion:

In this case, we describe common skin findings of Tuberous Sclerosis but in a rare distribution. Shagreen patches can often be found on the torso or back; however, in our patient, there were Shagreen patches found on the face, which helped diagnose Tuberous Sclerosis in the setting of additional skin findings and diffused organ and bony involvement. Understanding the morphology of classic TSC lesions, even in an uncommon location, is crucial in diagnosing TSC. Also, understanding the morphology of the Shagreen patch in the skin color is vital.
Introduction

Weeksella virosa is a rare cause of sepsis and septic shock. There are few case reports in literature, most of which indicate that it causes septic shock in immunocompromised and patient with multiple comorbidities. Weeksella virosa is an aerobic Gram-negative rod which grows on blood and chocolate agar, culture will produce cream-colored mucoid colonies. We describe a fatal case of septic shock due to weeksella virosa in a 69-year-old male.

Case description

A 69-year-old with past medical history of chronic Foley secondary to urinary retention, hypertension, hyperlipidemia, benign prostatic hyperplasia who was brought to the emergency department via EMS due to altered mental status. He had a history of chronic urinary retention after surgery in April of this year. On arrival he was completely obtunded with a GCS of less than 7. He was intubated, mechanically ventilated immediately. He had a distended abdomen with pus noted around the urethral meatus. The existing Foley’s was pulled out and it drained about 200 mL of frank purulent pus. A new Foley’s was put in and it drained about 1.5 L of reddish pus. On arrival vitals BP 70/50; HR 114. Initial labs were significant for white count of 36.8, potassium of 8.3, BUN of 240, creatinine 13.57. Blood and urine culture were sent. Initial blood gas showed 6.84/57.1/490. Initial lactic acid was 9.56. Initial UA showed more than 100 blood cells, 3+ bacteria, 3+ blood, 3+ protein. Patient received 2 L lactated Ringer bolus in the ED. He was given a dose of cefepime. A left femoral central line was placed. He received hyperkalemia treatment in the emergency department. He was given another liter of Lactated Ringer bolus, started on maintenance fluids with normal saline at 100 cc an hour. Ventilator settings were Assist control 26/450/50/5. He was given intravenous antibiotics ertapenem and vancomycin as an empiric treatment. Later ertapenem was replaced by ceftriaxone 2gm daily and vancomycin was continued as per infectious disease expert recommendations. CT abdomen and pelvis showed no sign of peritonitis and chest xray showed no sign of pneumonia. He was transferred to medical intensive care unit and was started on CRRT for worsening metabolic derangements including persistent hyperkalemia. After several hours of CRRT treatment, the patient’s potassium remains in the 7 to 8 meq/l. Lactic acid climbed up to 15.49. Despite being max dose norepinephrine, vasopressin and phenylephrine, patient’s blood pressure continued to decline. He eventually had cardiac arrest due to septic shock and was pronounced deceased on the fourth day of admission. Later blood culture grew weeksella virosa which was susceptible to cefepime, ceftazidime, ceftriaxone, ciprofloxacin, piperacillin tazobactam, meropenem, and imipenem and gentamicin. Urine culture was positive for Citrobacter freundii and Enterococcus faecalis which were susceptible to ampicillin, vancomycin, cefepime, ceftazidime and few other antibiotics.

Discussion

Weeksella virosa, though rare, can induce fatal septic shock, particularly in immunocompromised patients with multiple comorbidities. Our case underscores the swift progression and fatality, emphasizing the crucial role of prompt recognition, IV fluids, empiric antibiotics, and blood cultures.
Atypical Reproducible Chest Pain secondary to NSTEMI

INTRODUCTION:

Non-ST segment elevated Myocardial Infarction (NSTEMI) most commonly causes anterior mid-sternal chest pain, often with radiation to the left chest wall, neck, and left upper extremity. Atypical chest pain is defined as pain secondary to acute coronary syndrome (ACS), which presents in locations other than that of typical chest pain. Reproducible chest pain is commonly seen in pathologies of musculoskeletal disorders and is not widely seen in ACS.

CASE DESCRIPTION:

A 59-year-old female with end-stage renal disease on hemodialysis via permcath present on her left upper chest presented to the emergency department with a chief complaint of chest pain. The chest pain was localized at the insertion site of the permcath and started approximately a week ago; it was constant, radiated to the left axilla, and worsened with movement of the left arm and upon lying down. It was associated with weakness and fatigue but no associated shortness of breath, chest tightness, cough, fever, or palpitations. On examination, the pain was reproducible on mild palpation and was maximally present at the insertion site of the permcath on the upper chest wall. On admission, the vitals were within normal limits except for a blood pressure of 193/93 mmHg, and the EKG showed T wave inversions in lateral leads, which was not present on the previous EKG. Her history was significant for hypertension and coronary artery disease with an RCA stent placed nine months ago. The patient was compliant with aspirin, statin, ticagrelor, and a beta blocker. The echocardiograph showed an overall LVEF of 60-65%, a global strain of 13%, and a diastolic filling pattern consistent with impaired relaxation and elevated filling pressure. High sensitivity troponin I on admission was 14, which increased to 21 on day 1 of admission. As the patient continued to complain of chest pain, a repeat EKG on day 2 showed no changes, and High sensitivity Troponin I levels were found to be 21,971. The patient underwent prompt cardiac catheterization that showed significant two-vessel coronary artery disease in the mid-right coronary artery (RCA) and the obtuse marginal artery (OM1). She underwent successful percutaneous coronary intervention with a drug-eluting stent (DES) placement. The patient reported resolution of the reproducible chest pain following the procedure and was discharged with dual antiplatelet therapy (DAPT).

DISCUSSION:

The sensitivity of troponin is more than 99%. Therefore, it is a reliable marker for ACS. In rare instances, it may be unremarkable during the initial phase of ACS. This case demonstrates the need for physicians to keep an increased level of suspicion of ACS even for reproducible chest pain with no significant EKG changes. High-sensitivity troponin I should be trended until it shows a decrease or is explained by another cause. It is crucial to diagnose ACS at the earliest as prompt treatment improves outcomes significantly.
An Interesting Yet Uncommon Presentation of Sensorineural Hearing Loss Due to Hypothyroidism

Introduction: Thyroid hormone is responsible for the development and function of the cochlea through fast-acting potassium channels. Thyroid-stimulating hormone (TSH) receptors also regulate the myelination of the cochleovestibular nerve and the integrity of the cochlear cytoskeleton. It has been suggested that there is a direct signal pathway from the thyroid to the inner ear spiral ganglion cells and inner and outer hair cells. Hypothyroidism disrupts the morphology of the outer hair cell and reduces the cochlear microtubules via decreased fibroblast growth factor (FGF) and coflin gene expression. An upset in the delicate balance of these signaling pathways may provide a link between thyroid dysfunction and sensorineural hearing loss.

Case Description: A 64-year-old female, with a past medical history of hypertension and hypothyroidism secondary to radioactive iodine ablation, presented after a fall. Home medications included levothyroxine 175 mcg daily; however, she was non-adherent with her medication. A thyroid profile obtained in 2022, one year preceding this hospital admission, showed TSH 54.80 micro IU/mL and free T4 0.3 ng/dl. A few months later, the patient developed sudden bilateral sensorineural hearing loss that was more pronounced in the left ear. Audiogram revealed moderate to profound mixed hearing loss on the right and moderate mixed hearing loss on the left, with asymmetry on her word recognition score - worse on the right. Marginal improvement in hearing following cerumen disimpaction was observed. During this admission, her thyroid profile demonstrated a TSH level of 44.05 micro IU/mL, free T4 < 0.25 ng/dl, and free T3 2.3 ng/dl. The patient was resumed on her home dose of levothyroxine. A week later, she reported subjective hearing improvement bilaterally with a repeat TSH of 19.88 micro-IU/mL.

Discussion: This case demonstrates that although thyroid disease has a high incidence globally with well-known symptoms, it may still present with uncommon manifestations. Studies have linked thyroid dysfunction with both sensorineural and conductive hearing loss. Treating hypothyroidism not only prevents hearing loss but also has the potential for complete or partial hearing restoration. Therefore, it is imperative to include TSH levels in the evaluation of hearing loss with uncertain etiology. Additionally, screening for hearing loss in newly diagnosed hypothyroidism can be taken into consideration.
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**Obscure Gastrointestinal Bleeding in a middle age man**

Introduction: The most common type of gastrointestinal bleed (GIB) is an upper GIB. Incidence of UGIB is approximately twice that of lower and presents with hematemesis and melena while lower GIB commonly presents with hematochezia. Previously, approximately 5-10% of patients were labeled as obscure bleed after no source of bleeding being found on standard endoscopic and radiology evaluation. However, in 75% of these patients, the source of bleeding is the small intestine that is not seen on standard evaluation. Advancements in technology have changed the definition of obscure bleed to comprise only patients with no identifiable bleed source after complete small bowel evaluation in addition to standard endoscopic and radiographic evaluations.

Case Description: A 59-year-old male presented to the emergency department (ED) after an episode of massive hematochezia and syncope. Patient has no history of prior GIB and did not report dizziness, dyspnea, or fatigue. Patient’s vital signs were initially stable with laboratory studies showing hemoglobin (Hb) of 9.1 g/dl. Endoscopy demonstrated a small arteriovenous malformation with no active bleeding and colonoscopy showed dark stool without active bleed. Patient continued to have hematochezia with a repeat Hb of 4.6 g/dl and received a total of 5 units of packed red blood cells (PRBCs). CT angiography (CTA) abdomen/pelvis, radionuclide GI bleed scan, and a gastrografin enema were all negative for any pathology or bleed. Hematochezia resolved and the patient was discharged with a Hb of 7.6 g/dl. Patient returned to the ED 2 days afterwards complaining of lethargy, dizziness, weakness, and melena. Patient was hypotensive on presentation (79/54 mmHg) with HR of 84bpm and digital rectal exam revealing black stool. Hb was 3.7 g/dl and the patient was admitted directly to the MICU. CTA abdomen/pelvis showed no active bleed and invasive angiograms of the celiac, superior mesenteric, and inferior mesenteric artery and their main branches revealed no active bleed as well. During the second admission, patient received 6 units of PRBCs with melena resolving, vitals stabilizing and the patient was eventually discharged. Patient was referred to a tertiary care center if symptoms returned. 5 days later, patient presented to an outside hospital with same symptoms of melena and syncope. Patient was resuscitated with 1 unit of PRBC and received capsule endoscopy that showed no active bleeding. Patient was discharged home with GIB etiology remaining unknown.

Discussion: This case demonstrates elusiveness in diagnosing etiology of GIB when it is not localized to the stomach or colon. Physicians need to consider transferring the patient to a tertiary care center for further evaluation if no upper gastrointestinal or colon etiology is found and the patient remains symptomatic. Lastly, this case exhibits the rapid deterioration that patients with GIB can present with and the vigilance physicians must exhibit in their management of these patients.
Wernicke-Korsakoff Syndrome w/reversibility w/High dose IV thiamine for >5 days
Introduction: Wernicke-Korsakoff syndrome has a mortality rate of 10-15% which can have noticeable difference within 2-3 weeks of treatment. The key is to give IV thiamine quickly, via IV, and a high dose. Some changes can occur over a long-term period if the patient continues to abstain from alcohol and supplementing maintenance dose levels of oral thiamine.

Case Description We presented a case of a 47-year-old female with known history of epilepsy nonadherent to her medications, debility, CAD, and a long smoking Hx who sustained a recent fall from her wheelchair a month prior and has been encephalopathic with weakness since that time. Her weakness in BL LE’s was markedly worse than her UE’s. Non-contrast MRI of head and full spine who found stenosis but did not think it explained her symptoms and her stroke work up and EEG did not reveal any acute findings. She presented to a second hospital with seizures, encephalopathy, hypertensive urgency and progressive ascending weakness with point tenderness over thoracic spine and a rash over her posterior torso.

Her LP was only remarkable for pleocytosis, and all serum inflammatory markers were negative. On repeat MRI w/w/o contrast of the brain and spine, she was found to have profound T2 hyper-intensity and enhancement of the mamillary bodies and T2 hyperintensity symmetrically involving dorsomedial thalami, consistent with acute Wernicke's encephalopathy. She had Old fracture at T5 and severe stenosis of L5 on MRI w/w/o contrast. We were able to get the same neurosurgeon to review her case and he compared her imaging and physical exam and thought that she was back in the same state he saw her. We spoke with poison control and continued high dose IV thiamine x 5 days and her mentation and strength have markedly improved daily. She went from being obtunded to answering questions with intermittent stages of confabulation and hallucinations. Eventually, the patient regained the upper extremity strength although much of the damage in the lower extremities.

Discussion: This case demonstrates the utility of empiric treatment with high dose IV thiamine earlier, and with longer duration than the standard 3 days in patients who present with acute encephalopathy with severe alcohol use disorder. Since thiamine water soluble w/<20% bioavailability, the adverse effects are low. Further, the slight increase in cost is worth attempting higher dose treatment, given the profound impact it can have on patients’ morbidity and mortality. This should become more widely used as the window of opportunity is small.

Sources:

Carotid Sinus Syndrome in a patient with advanced laryngeal Cancer

Introduction: An important regulatory mechanism for maintaining arterial blood pressure in the body is the carotid sinus reflex. Parry observed in 1799 that placing pressure on one of the carotid sinuses can slow down the pulse rate. Carotid Sinus Hypersensitivity (CSH) is the term for an amplification of the response to this maneuver. Rarely, head and neck tumors can cause Carotid Sinus Syndrome. We describe a case with advanced laryngeal carcinoma presented with syncope and intermittent episodes of bradycardia and hypotension secondary to carotid sinus syndrome.

Case Description: A 68-year-old male with past medical history of Stage 4 laryngeal squamous carcinoma, hypertension and dyslipidemia, presented to the hospital after syncopal attack. Four months prior to admission, patient was diagnosed with a right laryngeal mass which was consistent with invasive squamous cell carcinoma. On admission, his vitals were temperature 36.9 °C, heart rate 95/min, blood pressure 101/70 mmHg, respiratory rate 20/min, saturating at 100% on room air. On physical examination, the patient had a palpable 5 cm mass in the right submandibular region that was firm and fixed. A routine blood test and measurement of electrolytes, glucose, liver, kidney, thyroid function, and high-sensitivity troponin T were within normal. EKG showed normal sinus rhythm. CT head showed Encephalomalacia in the left thalamus as sequela of remote hemorrhage and no evidence of acute intracranial abnormality. Cerebral CT angiogram demonstrates normal contrast enhancement of the major intracranial arteries without occlusion, stenosis, or filling defects and symmetric distribution of the distal arterial branches without areas of abnormal vascularity. CTA neck showed a large heterogeneously enhancing mass in the right lateral neck extending from the clavicle to the supraclavicular region measuring 6.6 x 5.5 x 8.4 cm. The mass encases the right common and internal carotid arteries. During hospital course, the patient developed recurrent attacks of hypotension and bradycardia with full recovery in a few minutes. No specific triggers were apparent. No elevation of myocardial necrosis markers or electrolyte imbalance. Transthoracic echocardiography showed no abnormal findings. We noticed that during these attacks when we turned the patient’s neck to the left (opposite site of lesion), heart rate returned to normal. A diagnosis of carotid sinus syndrome from tumor compression was assumed. The management plan for our patient was to target the tumor itself and start radiotherapy; However, secondary to CTA findings of common and internal carotid artery encasement and compression by the tumor, Neuro-endovascular surgery was involved and carotid stenting was done. Patient was transferred to Neurocritical Care Unit for recovery. Next morning on POD1, the patient had a sudden pulseless electrical activity with unsuccessful CPR.

Discussion: Unexplained repeated episodes of bradycardia and hypotension in patients with head and neck cancer should notify the physician about the potential for carotid sinus compression. Effective collaboration among internists, cardiologists and oncologists is essential for accurate diagnosis, effective treatment, and a favorable result.
Recurrent Idiopathic Hypoglycemia in a middle age male

Introduction: Recurrent Idiopathic Hypoglycemia is an incomplete diagnosis with likely etiologies including: Adrenal Insufficiency, Hypothyroidism, Insulinoma, Sulfonylurea/Insulin use, Liver Disease, etc. Recurrent hypoglycemia can affect any age group. However, it is more prevalent in older populations. This case report explores the challenges encountered in managing a multifaceted clinical presentation.

Case Description: A 63-year-old African-American male, with a past medical history of hypertension, seizures, and hyperlipidemia, presents to the hospital following a fall at home attributed to hypoglycemia, he had previously undergone inconclusive evaluations for neuroendocrine tumors. The patient’s altered behavior and hypoglycemia led to hospitalization. Transferred to the MICU on day 9 due to acute respiratory failure, he received ventilatory support for 9 days alongside cefepime and vancomycin for aspiration pneumonia. Cosyntropin test on day 29 revealed adrenal insufficiency which was managed with stress-dose steroids. Despite steroid replacement he continued to have hypoglycemic episodes. An octreotide scan ruled out somatostatin-positive neoplasms. His sulfonylurea labs were negative, and IGF-1 lab was within normal limits.

The patient’s fasting serum insulin level was within normal limits at 4.3 while his blood glucose was low. Therefore, a clinical diagnosis of radiologically undetected insulinoma was made. Oral Diazoxide was started and his hypoglycemia improved. The patient was transferred to a sub-acute rehabilitation center.

Discussion: This case underscores the intricate nature of managing patients with diverse comorbidities. Collaborative efforts among specialties were pivotal in addressing challenges. Outpatient follow up is crucial for sustained management and monitoring potential underlying causes contributing to the patient’s complex clinical presentation.
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**Lower extremity Hemiparesis - It is not a pinched nerve but Neurosarcoidosis**

**INTRODUCTION**

Sarcoidosis is a multi-organ granulomatous disease of unknown etiology. It mostly affects middle-aged patients, and prevalence is higher in women compared to men. Neurosarcoidosis is relatively uncommon with a reported prevalence of 3-10%. Any part of the nervous system can be affected including brain parenchyma, cranial nerves, and meninges mimicking other neurological disorders causing a challenge in diagnosis.

**CASE DESCRIPTION**

A 34-year-old female with no significant past medical history presented to the Emergency Department complaining of pain and weakness in her right lower limb. The weakness worsened to the point where the patient could not ambulate. The patient had multiple hospital visits in the past 3 months with the same complaint during which differential diagnosis included complex regional pain syndrome, pinched nerve, and was started on Ketorolac and Methocarbamol with minimal symptom relief. During day 2 of her hospital stay patient also developed urinary retention requiring straight catheterization every 6 hours. She had stable vital signs. Physical examination demonstrated weakness (0/5) and absence of reflexes in RLE. CT head, CT of the lumbar spine and right Lower extremity were unremarkable. Laboratory studies showed elevated ESR 90 and CRP 37.9. Serum ACE was within normal limits. Autoimmune and vasculitis workup with ANA, RF, CCP, dsDNA, ENA, and PM-scl100 were within normal limits. CT chest abdomen/pelvis showed mediastinal and abdominal lymphadenopathy. Cerebrospinal fluid analysis was compatible with aseptic meningitis. Further investigation with MRI spine revealed diffuse poorly defined T2 hyperintensity in the lower thoracic and lumbar spinal cord, including the conus medullaris. Head MRI showed T2 hyperintense enhancing foci in the left corona radiata/caudate nucleus junction. These findings in a patient with pulmonary sarcoidosis are consistent with Neuro sarcoidosis. Iliac lymph node biopsy revealed the presence of multiple granulomas with focal necrosis, GMS and AFB stains are negative for fungal or mycobacterial microorganisms. The therapeutic approach included a short course of high-dose steroid treatment, and the patient was started on Azathioprine. The patient’s clinical status showed significant improvement, leading to discharge with a scheduled outpatient and appropriate rehabilitation.

**DISCUSSION**

Neurosarcoidosis presents a diagnostic challenge due to its rarity and diverse clinical manifestations, mimicking other neurological disorders especially in patients without a known diagnosis of systemic sarcoidosis. This case demonstrates the variable presentation of neurosarcoidosis and the challenges in diagnosing this disorder. ACE levels are not necessarily always elevated in sarcoidosis. Prompt diagnosis and treatment with high-dose steroids are advised to prevent permanent neurological deficits.
Heart failure outcomes in GDMT alone versus GDMT combined with revascularization in patients with HFrEF

Background:
Worldwide, 5.7 million individuals are affected by heart failure, with an expected increase to 8 million by 2030. Currently, guidelines dictate that all patients with heart failure be started on goal-directed medical management (GDMT). However, given that the most common cause of heart failure remains ischemic, this study aims to assess the benefits of adding revascularization as a treatment option for patients with HFrEF who are already on GDMT.

Methods:
A comprehensive literature search using databases such as PubMed and Elsevier was done, with a focus on randomized controlled trials (RCTs), systematic reviews, and meta-analyses published between 2013-2023. Inclusion criteria required articles to compare medical therapy with revascularization in patients with ejection fraction below 40% and reported on mortality outcomes. Two meta-analyses and two RCTs were selected after screening out duplicates and articles that did not meet inclusion criteria.

Results:
The first meta-analysis by Davoudi et al. suggests a significant reduction in all-cause mortality (RR 0.76, 95% CI: 0.62–0.93, I² = 0) with revascularization in patients with ischemic cardiomyopathy. The RCT of Panza et al. indicates that surgical revascularization benefits patients with advanced ischemic cardiomyopathy, particularly those with specific prognostic factors (hazard ratio [HR]: 0.71; 95% CI: 0.56 to 0.89; p = 0.004). Wolff et al.’s meta-analysis, covering 21 studies and 16,191 patients, highlights a significant mortality reduction with both CABG and PCI compared to medical treatment (HR, 0.73; 95% CI, 0.62–0.85; P<0.001; heterogeneity P = 0.96; I² = 0%), with CABG being more superior to PCI (HR, 0.82; 95% CI, 0.75–0.90; P<0.001; heterogeneity P = 0.01; I² = 47%). However, a contrasting RCT by Perera et al. involving 700 patients shows that PCI did not result in a lower incidence of death or hospitalization for heart failure as compared to optimal medical therapy (hazard ratio, 0.99; 95% confidence interval [CI], 0.78 to 1.27; P=0.96).

Conclusion:
Our findings suggest there is merit in revascularization for heart failure secondary to ischemic heart disease, with CABG potentially being superior to PCI, especially in severe cases. However, given the advances and additions to GDMT, coupled with the improved surgical revascularization techniques, there is a need for up-to-date, large-scale, multi-center RCTs to suggest one treatment modality over the other.
When chronic pancreatitis reaches the chest, a case of pancreaticopleural fistula and recurrent pleural effusion.

Introduction:

Pancreaticopleural fistula (PPF) is an infrequent complication arising in approximately 0.4% of pancreatitis cases, predominantly observed in patients with alcoholic chronic pancreatitis. This rare condition is characterized by an abnormal connection between the pancreas and the adjacent pleural cavity. It occurs due to a disruption of the pancreatic duct (PD), resulting in leakage of pancreatic fluid. Its symptoms primarily affect the thoracic region, posing challenges in diagnosis. Here, we describe a case of PPF in a patient with non-specific chest pain and chronic alcohol use.

Case Description:

The patient is a 64-year-old male with a history of asthma and alcohol use (pint of liquor daily) who presented with a chief complaint of chest pain. He described his pain as a sharp, stabbing sensation mainly over the left chest wall and his entire abdomen, associated with difficulty breathing, decreased oral intake, and constipation. The patient denied any fevers, chills, cough, vomiting, or recent sick contacts. Physical examination revealed tachycardia, tachypnea, and diminished air entry with decreased breath sounds and dullness to percussion over the left lower lung base. The initial lab workup was unremarkable. EKG showed sinus tachycardia with no ST segment changes, troponin was unremarkable. Chest X-ray revealed left basilar atelectasis and a moderate left pleural effusion. CT thorax showed a large left-sided pleural effusion with multisegmental atelectasis. CT abdomen showed subcentimeteric hypoattenuating lesions in the pancreas. Thoracentesis revealed an exudative effusion with LDH 521, protein 4.2, albumin 2.1, glucose 63, cholesterol 79, and amylase > 10000. Fluid culture and cytology were negative. Post-thoracentesis, the patient was discharged but had recurrent hospitalizations for similar presentations. Further workup included MRI abdomen and MRCP which revealed a cystic lesion in the pancreatic head. The patient was initially managed conservatively with repeat thoracentesis, bowel rest, and octreotide. The pleural fluid continued to reaccumulate despite medical therapy, so the patient underwent chemical and mechanical pleurodesis and the placement of a Pleur-x catheter. ERCP revealed pancreatic ductal disruption, a stent was placed successfully which resolved the patient’s recurrent symptoms.

Discussion:

Pancreaticopleural fistula (PPF) is an uncommon condition caused by the leakage of the pancreatic duct leading to the development of pleural effusion, typically seen in patients with chronic pancreatitis. The fistulous tract leads into the pleural space and causes large-volume effusions that accumulate rapidly. Symptoms usually include nonspecific chest complaints. Elevated amylase levels (>1000 U/L) in pleural fluid should raise suspicion for PPF. Diagnostic imaging includes CT, MRCP, and/or ERCP to identify the fistulous tract. In cases where the pancreatic duct is normal or mildly dilated, initial medical management includes chest drainage with thoracentesis, bowel rest to decrease pancreatic activity, and octreotide administration to decrease volume. If conservative management is unsuccessful, endoscopic or surgical interventions may be considered. This case highlights the importance of keeping a high index of clinical suspicion to allow for the identification of a rare diagnosis of pancreaticopleural fistula in patients who have a history of chronic pancreatitis or history of alcohol use disorder and present with nonspecific respiratory/cardiac symptoms.
Fixed versus Automatic CPAP in Adult Obstructive Sleep Apnea Management

Introduction:
Obstructive sleep apnea (OSA) involves repetitive pharyngeal airway collapse during sleep, leading to hypoxemia and hypercapnia. It is managed with either automatic positive airway pressure (APAP) or continuous positive airway pressure (CPAP). The objective of this study is to compare the efficacy of APAP and CPAP in the treatment of adult patients with OSA.

Methods:
A systematic review of literature from 2013-2023 in PubMed and Web of Science databases was conducted, focusing on APAP and CPAP in OSA management. Selection criteria included clinical trials, randomized controlled trials, and systematic reviews in patients greater than 18 years old. Duplicates were excluded via Rayyan.ai. The analysis covered aspects such as pressure titration, symptom improvement, patient adherence, and adverse effects.

Results:
Seventeen studies met the inclusion criteria. In three studies, APAP was found to require significantly lower pressure settings for optimal titration. Both APAP and CPAP showed equivalent symptom improvement in all studies included. A slight hemodynamic advantage was seen with CPAP concerning diastolic in two studies and systolic pressures in one study. Higher BMI and male gender patients showed increased airway pressures with APAP. Patients with lighter sleep or higher arousal thresholds may tolerate and adhere to CPAP better. No notable differences in adverse effects between the two methods were identified.

Conclusion:
While some differences in pressure titration and hemodynamics were observed, APAP and CPAP showed similar efficacy in symptom improvement and safety profiles. Therapeutic choices should consider individual patient characteristics and preferences. APAP would be a better choice for obese patients with higher BMI, while patients with high arousal index and percentage of stage N1 may benefit from CPAP.

Keywords: automatic positive airway pressure, continuous positive airway pressure, obstructive sleep apnea.
Antibiotic failure in Arcanobacterium tonsillitis – Case Report

Introduction:

Arcanobacterium Haemolyticum (A. Haemolyticum), a coryneform Gram-positive bacillus, is a rare cause of head and neck infections, pharyngitis, and sinusitis in teenagers and young adults. It is rarely isolated in most clinical laboratories due to difficulties in its recognition.

Case

A 29-year-old male presented with recurring sore throat and odynophagia occurring 3-4 times/year since the patient was 14 years of age. Patient has a 10-pack-year history of smoking and occasional alcohol and marijuana use. Patient presented with a peri-tonsillar abscess around 6 weeks ago at an outside hospital. The abscess was drained, and the patient was discharged on 2 weeks of Augmentin which he completed. He presented to Sinai-Grace Hospital (SGH) with recurrence of symptoms. Patient underwent repeat drainage, and the aspirate was sent for culture. Patient received one dose of 3 grams Unasyn along with dexamethasone 20mg in the ED, and discharged on 2 weeks of clindamycin with a referral to ENT.

Patient presented again to SGH with recurrence of symptoms. The patient had shortness of breath without stridor and throat pain. Patient also developed dysphonia and dysphagia. Vitals were stable. Patient endorsed completion of clindamycin course. On physical exam, exudates and enlargement of the right tonsils, as well as generalized erythema in the tonsillar region was noted. CT neck with contrast was performed which noted a right 1.5 x 1.0 cm tonsillar abscess without retropharyngeal abscess. Patient was started on Unasyn. Another I&D on right tonsil was performed with drainage of 2cc of purulence. Final culture report from previous I&D 3 weeks ago showed numerous Arcanobacterium species. Anaerobic culture also showed moderate Fusobacterium necrophorum species.

Based on the culture sensitivity reports patient was started on Ceftriaxone and Metronidazole. Unasyn was discontinued. On Day 3 of IV antibiotics, patient reported significant improvement in symptoms and was able to talk with more ease. Physical exam was consistent with decreased swelling of the tonsils. Patient was discharged on Flagyl 500mg TID and Azithromycin 500mg once daily for 2 weeks with instructions to follow up with outpatient ENT clinic to schedule a tonsillectomy.

Discussion

A. Haemolyticum, an aerobic, slow growing, catalase-negative, gram-positive bacillus, has been reported as an infrequent cause of peritonsillar abscess, pharyngitis, and tonsillitis in children and young adults. Risk factors for the development of this infection remain to be identified. The organism, moreover, has been isolated from chronic skin ulcers, soft tissue infections, deep tissue abscesses, meningitis, pneumonia, endocarditis, and bacteremia.

Case reports of Arcanobacterium hemolyticum have reported failure of treatment when penicillin or clindamycin were used for treatment. No failures are reported on cephalosporins and newer macrolides. Due to difficulties in its laboratory identification, clinicians should consider the possibility of Arcanobacterium especially with antibiotic failure.
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**Beyond the Thyroid: Retrosternal Goiter’s Intriguing Journey to Acute Stroke and Venous Thrombosis**

**Introduction:** The incidence of retrosternal goiter varies between 2% and 26% of all thyroidectomized patients. Internal jugular vein thrombosis is a very rare yet serious radiological finding that can happen spontaneously or secondary to mediastinal masses. Few cases have been reported in literature to be secondary to retrosternal goiter. Internal jugular venous thrombosis can lead to potentially life-threatening complications, including but not limited to: pulmonary embolism, ischemic stroke, superior sagittal sinus thrombosis, septic emboli, and superior vena cava syndrome, intracranial hypertension.

**Case Description:**

A 56-year-old African American male patient with a past medical history of essential hypertension and Type II Diabetes Mellitus was brought to the ED by his family with acute neurological symptoms potentially demonstrating acute stroke; presenting as right-sided weakness and dysarthria, that started 5 hours before admission. The patient actively smokes 1 pack of cigarettes per day, coinciding with occasional alcohol (red wine) usage, and regular use of crack cocaine, including the day before admission. Upon physical examination, elevated blood pressure was noted (157/105), as the patient experienced lethargy, yet was able to follow commands, while expressing comprehensive aphasia and dysarthria with anisocoria and vertical palsy OD > OS, left facial droop and right-sided weakness of 3/5. NIHSS Score on presentation was 11. A circumference of 43cm was noted on examination of the neck, with no signs of thyromegaly. Pemberton’s sign could not be assessed due to right arm weakness. Further neurological workup included a CTA head and neck which showed evidence of a massive retrosternal goiter compressing the bilateral brachiocephalic veins resulting in collateral venous return through the mediastinum, as well as bilateral internal jugular veins thrombus. MRI was consistent with the left para-median thalamus and left para-median superior mesencephalon marginating the periaqueductal gray matter and left red nucleus compatible with ischemic changes greater than 6 hours. CT chest confirmed a massive goiter that has a mass effect on the trachea, which is displaced to the right, bilateral brachiocephalic vein compression with collateral venous return. Plan of treatment included a multi-disciplinary decision among ophthalmology, neurology, general surgery, ENT, CT surgery, and interventional radiology. Although the stroke was outside the window to start tissue plasminogen activator, coinciding with an unresolved cause of this acute stroke, the patient was started on Aspirin, Plavix, and statins. A decision was made not to initiate a heparin drip as the internal jugular vein thrombosis was chronic. A surgical intervention decision to remove the thyroid gland was needed: laryngoscopy, cervical neck exploration, sternotomy, and cervicomediastinal goiter resection.

**Conclusion:**

Presentation of acute ischemic brainstem stroke accompanied by an incidental finding of retrosternal goiter with mass effect causing bilateral deep vein thrombosis and mediastinal collaterals was demonstrated. This rare case illustrates the importance of neck imaging by either CTA or carotid duplex ultrasound of mediastinal imaging in patients with acute stroke. The finding of internal jugular vein thrombosis carotid imaging may potentially be the initial sign of a space-occupying lesion in the thorax. This finding guides surgical approach in managing retrosternal goiter.
Shifting paradigms of platinum-resistant ovarian cancer - mirvetuximab and bevacizumab, an elixir or curse? A case report of treatment-induced pulmonary fibrosis in a 68-year-old female with epithelial ovarian carcinoma.

Introduction
Mirvetuximab (MIRV) is the first biomarker-driven antibody-drug conjugate to target the folate receptor alpha (FRα) antigen—a protein expressed in 90% of ovarian malignancies. Clinical trials have demonstrated this drug to have significant efficacy in the treatment of platinum-resistant ovarian cancer (PROC). Despite the promising efficacy and tolerable safety profile, the drug when used with bevacizumab is associated with a rare and devastating treatment-related adverse effect of pulmonary fibrosis.

Case Description
A 68-year-old female presented to the emergency department for a one-day duration of acute onset dyspnea with cough. She denied fever, hemoptysis, chest pain, or diaphoresis. Medical history was significant for high-grade serous ovarian carcinoma for which the patient had undergone neoadjuvant chemotherapy, cytoreductive surgery, adjuvant chemotherapy with 10 cycles of Bevacizumab, and maintenance therapy with Letrozole. She was later enrolled in phase II clinical trial 2022-076: Carboplatin/Mirv + Mirv maintenance with Bevacizumab which she was receiving at the time of presentation. Vitals were pertinent for elevated blood pressure, tachycardia, and hypoxemia with saturations restored to >92% after being placed on supplemental oxygen. Initial labs revealed WBC 7.8; D-dimer 7.54, and troponin 96 > 69. EKG was negative for any ischemic changes. ABG revealed pH 7.53, pCO2 36, and pO2 49. Respiratory pathogen panel was negative for flu and COVID-19. Chest x-ray revealed predominantly basilar reticular opacities with traction bronchiectasis. CT-PE ruled out pulmonary embolism but showed worsening fibrosis and pneumomediastinum.

The patient’s dyspnea was treated with bronchodilators, a prolonged course of steroids, and a course of ceftriaxone. Repeat CXR re-demonstrated previously noted diffuse bilaterally prominent reticular nodular opacities and interstitial pneumonia/pulmonary fibrosis. Pulmonology was consulted and noted worsening fibrosis was likely secondary to Mirvetuximab and recommended its discontinuation and a prolonged course of steroids. Bronchiolar lavage by fiberoptic bronchoscopy to rule out infectious etiology was subsequently conducted which was unremarkable. Cardiac workup which included an echocardiogram was also unremarkable and was less likely the cause of the patient’s dyspnea. Barium swallow was negative for esophageal perforation.

During hospital admission, the patient’s oxygen requirements increased to 8-10 L/min but was gradually weaned to 5-6 liter/minute. The patient was counselled for pulmonary rehabilitation and discharged to subacute rehabilitation with the recommendation to permanently discontinue Mirvetuximab therapy.

Discussion
Based on studies, patients with PROC have poor outcomes and suboptimal responses to therapy, median overall survival is <1 year. In clinical studies, MIRV showed significant antitumor activity in patients with FRα-high (>75%) PROC independent of bevacizumab exposure status. There is an overexpression of FRα in EOC compared to normal tissues, hence the increased tumor-selective activity. The Food and Drug Administration approved MIRV in November 2022 for FRα-positive PROC following the results of the SORAYA trial. MIRV has demonstrated a tolerable safety profile with the main adverse effects including low-grade gastrointestinal and ocular symptoms. For patients with FRα levels between 25-75%, MIRV is administered in conjunction with bevacizumab for optimal efficacy. However, coadministration of both systemic agents as this case highlights increases the risk for irreversible pulmonary fibrosis and treatment failure.
An Analysis of Ketamine Use in Mechanically Ventilated Patients: A Systematic Review

Introduction: Hemodynamic instability is one of the major side effects of many regularly used sedatives, which makes them problematic in the critical care situation. Ketamine is a new option that is gaining popularity due to its sedative effects on patients on mechanical ventilation. Nonetheless, a thorough analysis of its safety and adverse impact profile is necessary.

Materials and Methods: We searched the literature using PubMed with the terms "Ketamine" and "mechanical ventilation." The first search produced 34 results, and among the eligible trials were adult patients on mechanical ventilation who were continuously receiving an intravenous ketamine infusion for sedation. RCTs and observational studies including a total of 892 patients made up the final selection. The effect of ketamine use on the doses of sedative medications, particularly propofol, midazolam, and fentanyl, was closely examined.

Results: The results of our study indicate a clear upward trend in the use of ketamine among patients requiring mechanical ventilation, which highlights the increasing interest in the sedative properties of this drug. Notwithstanding, notable statistical and clinical fluctuations were noted in the utilization of ketamine, with concerns pertaining to the ideal setting, patient group, and dosage. Although there was inconsistent data from a retrospective investigation, ketamine did not reliably lower the doses of fentanyl or midazolam. Notably, in six trials, ketamine was linked to a considerable decrease in the dosage of propofol. Additionally, five studies found a significant correlation between longer ICU stays and ketamine sedation, although three studies found no significant differences in the duration of mechanical breathing. The incidence of delirium showed inconsistent results: two studies found a decline in delirium, two found an increase, and two found no discernible change.

Conclusion: In order to gain a more comprehensive understanding of the safety and effectiveness of ketamine use in patients on mechanical ventilation, our study highlights the urgent need for more randomized controlled trials. Keywords: Ketamine, prolonged sedation, adverse event.
Uncommon Positional Dyspnea Manifestation Associated with Incidentally Diagnosed Adenomatous Goiter

Introduction:

Adenomatous goiter is a common benign tumor of the thyroid gland. Most cases are asymptomatic but various clinical manifestations can occur, especially when nodules cause compression of adjacent structures including recurrent laryngeal nerve, trachea, and esophagus leading to hoarseness, dyspnea, and dysphagia.

Case Description:

A 55-year-old female with a past medical history of Hypertension, Seizure d/o, Migraine, and Obstructive sleep apnea, presented to the emergency department with a left-sided migraine headache with nausea and left-sided radicular pain in her neck. Approximately 2 weeks prior patient was seen in an outpatient clinic for complaints of upper extremity left-sided paresthesia, in the emergency department patient stated that it felt worse than her baseline. Her physical exam was within normal limits. She denied any recent trauma, syncope, dizziness, speech difficulty, difficulty in balance. She also suffers from positional dyspnea, mostly laying on her left side and leaning forward whereas she is comfortable on her back, right side and standing upright. CT scan revealed a multinodular goiter with a dominant nodule on the left, with mild left tracheal attenuation. Her thyroid stimulating Hormone (TSH), and thyroxine (T4) levels were normal; whereas triiodothyronine (total T3) was 80. calcitonin, Anti TPO Ab, and TSH receptor Ab were negative. A US-guided needle biopsy was performed which showed adenomatous goiter. The patient underwent a left thyroidectomy. The patient was started on gabapentin 100 mg 3 times daily, venlafaxine to 25 mg daily for left-sided brachial plexopathy due to neurogenic thoracic outlet syndrome. Malignancy was ruled out through biopsy. The patient was discharged on the above medication and was asked to follow-up outpatient with endocrinology, ENT. The patient was later followed up with and stated resolution of her symptoms.

Conclusion:

The case study showcased a correlation between adenomatous goiter and dyspnea, highlighting the significance of examining thyroid problems in clinical settings when faced with unexplained respiratory discomfort. Although adenomatous goiter seldom transforms into malignancy, it often induces compression symptoms due to its considerable size.
Salmonella causing prostatic abscess; Rare case report

Introduction:

Prostatic abscesses (PA) are a rare urological condition associated with chronic or acute bacterial prostatitis. Gram-negative bacilli (primarily E. coli) are the most prevalent, accounting for 40-50% of cases. Mostly, it is a diagnostic challenge as the patients present with non-specific symptoms, such as pelvic/perineal pain, dysuria, fever, chills, and urethral discharge. However, in this rare case, the cultured specimens revealed the presence of Salmonella Agona as the etiological agent. Salmonella is a rarely causative organism of prostatic abscess and is associated with an immunosuppressive state; in this case, the patient had uncontrolled Diabetes Mellitus type II.

We are reporting this case because the identification of a rare organism is vital: It changes the threshold for investigating complications common with this specific organism, such as enteric infections and bacteremia.

Case Description:

A 41-year-old male with a past medical history of uncontrolled DM-2 s/p Bilateral below-the-knee amputation secondary to gangrenous diabetic foot infection, CKD, anxiety disorder, major depressive disorder, anemia, and chronic debility presented to the emergency department with complaints of pelvic and lower abdominal pain aggravated by recent constipation, pneumaturia, chills, and fever. The patient was hemodynamically stable during the initial examination. The patient had suprapubic pain on palpation and no costovertebral angle tenderness on physical exam. Laboratory data did not reveal leukocytosis as expected. CT scan of the abdominal and pelvic revealed a large prostate abscess measuring 3.9 x 5.7 x 5.2 cm with bladder wall thickening and intraluminal air consistent with cystitis. Empirical antibiotic therapy was promptly initiated (Ceftriaxone on ED, then empirically changed to Vancomycin + Meropenem after CT report) to address this condition, and the patient underwent surgical intervention (transurethral unroofing) for abscess drainage and irrigation. Later, cultured specimens sent during the surgical procedure grew pure culture of Salmonella Agona. He also received adequate control of his glucose level. Over the following week, the patient’s condition improved; EGD for possible GI bleed, as the patient mentioned a melena episode, showed mild esophagitis-hyperemia, which may be associated with the nontyphoidal Salmonella as it is known to cause enteric infection, the patient was started on Pantoprazole 40mg daily with the improvement of his symptoms. After 7 days as an inpatient, he was discharged home with a prescribed regimen of oral antibiotics, Levofloxacin 750 mg PO daily for more than 4 weeks.

Conclusion:

The clinical symptoms of prostatic abscess often mimic those of several other diseases of the lower urinary tract, and the diagnosis may be difficult. Be aware of risk factors, as diabetes plays a crucial role in achieving an accurate diagnosis that can be obtained with the help of imaging. Although rare, prompt diagnosis is vital to improve survival and decrease life-threatening complications like sepsis. Also, identifying the causative organism is essential to monitor for expected complications related to the specific organism; in this case, the patient had associated GI symptoms, and the abscess was from an organism known to cause enteric infections.
Solitary plasmacytoma of bone presenting as atypical chest pain in an elderly male: A case report

Introduction: Plasma cell dyscrasias comprises a broad spectrum of disorders characterized by neoplastic proliferation of clonal plasma cells. Solitary plasmacytoma of bone (SPB) contains a single clone of plasma cells localized to a single bone lesion without any other plasma cell lesions in the body. The incidence of SPB is less than 450 cases annually, representing 0.3 cases per 100,000 person-years in the United States. SPBs mostly present with skeletal pain or pathological fracture of the bone involved. However, rarely, they may present as a palpable mass due to extension into the surrounding soft tissue. We offer you a unique case of SPB in an elderly male presenting with atypical chest pain.

Case Description: A 69-year-old African American male with a past medical history of hypertension, asthma/COPD, peripheral artery disease, carcinoma of larynx (pharynx) status post-resection presented to the emergency department with a painful lump in his left lower posterolateral chest exacerbated by touch and movement. The patient reported having a left paraspinal lump for a year which had been rapidly growing over a month and was associated with fever and night sweats. The examination was remarkable for a 5x8cm painful lump at the 11th left posterolateral rib level. Laboratory workup showed a normal troponin and BNP with an unremarkable 2D ECHO and stress test ruling out cardiac etiology. Urine analysis showed trace protein. Further, a CT thorax with contrast demonstrated a 9.4 cm expansile osteolytic mass involving the left 11th rib. The mass showed pronounced enhancement with invasion into the paraspinal and latissimus dorsi muscles, strongly indicative of primary bone malignancy. Multiple osseous erosions and pathologic fracture areas were present throughout the 11th rib. These findings were similar to the findings seen in the CT abdomen and pelvis done a few weeks back during a prior hospitalization. Given these findings, further work-up was done to rule out malignancy. Serum protein electrophoresis demonstrated an M spike with IgG lambda band on immunofixation. The bone survey done was found to be negative. A bone biopsy showed diffuse infiltration by atypical plasma cells having large nuclei, prominent nucleoli, and scant cytoplasm. Plasma cells were positive for CD138 and were found to produce lambda light change immunoglobulin on kappa and lambda in-situ hybridization. The plasma cells stained negative for AE1/AE3, CAM5.2, and LCA. The patient was treated symptomatically for pain with morphine and gabapentin as needed during the hospitalization. The patient was referred to establish care with a hematology-oncology for further management.

Conclusion: This case presentation of SPB shows an unusual presentation with symptoms of atypical chest pain in an elderly patient with multiple cardiovascular and pulmonary comorbidities which begs prompt diagnosis and treatment. The primary treatment for SPB is localized radiation therapy. Surgery may be considered in patients with structural bone instability, retropulsed bone or cord compression progressing rapidly. The median survival rate is approximately 10 years with an overall survival rate of 75 and 45 percent at 5 and 10 years respectively.
A case of self-inflicted thoracic empyema caused by E.Coli

E.coli is a part of intestinal flora that rarely causes infection in the pleural fluid. When isolated, it is most commonly due to seeding from bacteremia or aspiration of gastric contents. This case study reveals an unusual cause of aspiration.

We present a case of a 76-year-old male with a past medical history of pulmonary sarcoidosis, rheumatoid arthritis, prostate cancer and papillary renal cell cancer who presented to the hospital for shortness of breath of 1-month duration. On admission, patient was afebrile, normotensive, tachycardic, slightly tachypneic and saturating at 95% on room air. He had a cachectic appearance with clear lung sounds. Labs were unremarkable other than leukocytosis. CT scan of the chest showed a small to moderate-sized loculated left pleural effusion which was also present on an outpatient CT chest performed one week before presentation. He underwent thoracentesis with removal of 250cc of cloudy fluid. Pleural fluid analysis was exudative and suggestive of empyema. Due to persistent effusion, a left-sided pigtail catheter was placed followed by intra-pleural instillation of lytic agents. Pleural fluid culture grew E.coli. Histocytology was negative for malignancy or atypical cells. Blood culture and urine culture did not show any growth. Upon further questioning, he described one of his daily routines which involved siphoning water orally to irrigate his crops and also recalled having accidentally aspirated on several occasions. Speech therapy evaluation and oropharyngeal motility study were performed to rule out a swallowing disorder which came back negative. Patient remained hemodynamically stable and on room air throughout the hospital course and was discharged on a course of intravenous ceftriaxone with eventual removal of pigtail catheter in one week. He was also instructed to avoid oral siphoning of water.

This patient’s relatively benign presentation along with the presence of a loculated effusion on imaging prior to admission suggests that he may have had the infection developing in the pleural space for a significant amount of time. Isolation of E.coli from empyema with negative blood culture makes aspiration a more probable cause. On inpatient testing, there was no evidence of swallowing disorder that would inadvertently put him at risk for aspiration. All these points towards the fact that he had chronic microaspiration which was self-inflicted, although unintentional, from his high-risk behavior of water siphoning. Additionally, he was at increased risk given his underlying immunocompromised state and rheumatological disease that has a predisposition to affect the pleural space.

Certain behaviors may unknowingly put individuals at high risk of having a life-threatening infection like empyema and hence efforts should be made to identify the etiology in such cases.
Pyridostigmine’s Role in Increased Risk of GI Bleeding Events

Introduction:

Pyridostigmine, a cornerstone in Myasthenia Gravis therapy, and due to its renal excretion, may pose risks in patients with renal impairment. This case highlights a potential association between pyridostigmine (an AChE inhibitor) and increased risk of gastrointestinal bleeding, an area not extensively explored in current literature.

Case Description:

The patient, an 85-year-old male, has a medical history of coronary artery disease, end-stage renal disease on hemodialysis, and long-term management of Myasthenia Gravis with pyridostigmine and mycophenolate. He presented to the hospital with a two-week history of intermittent black, bloody stools. Upon admission, he was hemodynamically stable, but laboratory tests revealed severe anemia with a hemoglobin level of 6.9 g/dL and a slightly elevated INR of 1.2, without any other significant lab abnormalities. Notably, he had four similar admissions over the past three years, and previous endoscopies had revealed multiple gastric ulcers, arteriovenous malformations, and duodenal and gastric antral vascular ectasias. His treatment regimen on these occasions had included Argon Plasma Coagulation, discontinuation of any antiplatelet medications, and the initiation of high-dose proton pump inhibitors (PPI). During this admission, a repeat endoscopy identified active oozing from multiple arteriovenous malformations in the gastric antrum. Biopsy results showed chronic reactive gastropathy and mild nonspecific inflammation, with no evidence of H. pylori infection. An abdominal ultrasound revealed a slightly coarse hepatic echotexture, but subsequent imaging did not indicate any significant liver abnormalities or portal hypertension.

The patient’s management included the administration of high-dose PPIs, fluid resuscitation, and multiple blood transfusions. Additionally, a repeat Argon Plasma Coagulation procedure was performed, and pyridostigmine therapy was discontinued. Following the stabilization of his hemoglobin levels, he was discharged on a high-dose PPI regimen. For ongoing care, regular gastroenterology follow-ups were scheduled, including periodic Erbe coagulation sessions, to ensure the complete success of his ablation therapy. Concurrently, outpatient neurology follow-ups were arranged to explore alternative treatments for Myasthenia Gravis, with particular consideration of Efgartigimod alfa infusions.

Discussion:

Studies indicate that donepezil, another AChEI used for Alzheimer’s dementia, is associated with an increased risk of upper gastrointestinal hemorrhage. However, direct evidence linking pyridostigmine with gastrointestinal bleeding is not well-established. Literature notes that pyridostigmine may prolong PT and inhibit platelet aggregation. Importantly, 80-90% of pyridostigmine is excreted renally, and impaired renal function can affect the drug’s clearance, potentially elevating the risk of adverse effects. Although uremic bleeding could be a contributing factor, this case provides supporting evidence for a potential connection between reversible AChEIs and gastrointestinal bleeding. It emphasizes the importance for clinicians to be aware of the risks associated with pyridostigmine, especially when prescribed to patients with renal impairments. Early recognition of these risks could help in reducing hospital readmissions and improving patient outcomes.
Tirzepatide Associated Partial Small Bowel Obstruction: A case report

Glucagon-like peptide-1 receptor agonists (GLP1 RA) are quickly becoming an adjunctive treatment modality for people with type-2 diabetes and is approved for chronic weight management. Tirzepatide has a dual GIP/GLP-1 RA action and has a synergistic effect when administered together. GLP-1 RA are associated with improved cardiovascular and all-cause mortality outcomes in patients with type-2 diabetes. Tirzepatide is associated with gastrointestinal (GI) side effects like nausea, diarrhea, abdominal pain, decreased appetite, and constipation. This case report discusses a case of small bowel obstruction (SBO) that was noted after patient was changed from dulaglutide to tirzepatide.

Mr. X is a 61 y/o male with PMHx of type-2 DM, HTN, and HLD who presented to ER for severe epigastric pressure-like 7/10 abdominal pain. No association with food intake. He had 1 episode of semi-loose stools. Denied any abdomen cramping, sick contacts, nausea/vomiting, fever/chills, or constipation. His previous endoscopy was only relevant for benign colonic polyps, which were removed. He had been on GLP1 RA for more than a year, and previously on dulaglutide 3mg, which was switched to tirzepatide 2.5mg 5 weeks ago. He was able to tolerate the medication without significant side effects and was recently up-titrated to 5 mg. Labs showed WBC 7.2, Hb 13.6, Cr 1.45, BUN 12, lipase 25, and normal LFTs. Abdominal X-ray and CT abdomen were noted to be significant for a partial vs evolving SBO without any identifiable small bowel intrinsic pathology. Mr. X was managed conservatively with fluids, NPO diet, and pain medications. Serial abdominal images were obtained, and he was eventually advanced to soft diet. Tirzepatide was discontinued on discharge.

As a response to enteral nutrition, distal small bowel and colon release GLP-1 hormone. Exogenous and endogenous GLP-1 are associated with reduced gastric motility. GLP1 RA and dual GIP/GLP-1 RA are advised to be used with caution in patients with established gastroparesis, as they can worsen the symptoms. GLP-1 effect on small intestinal motility is not extensively studied, however, small bowel motility inhibition was noted in animal models. Some of these actions were dose-dependent. There are some case reports of SBO due to GLP-1 RA. An observational cohort study noted an increased incidence of SBO with GLP1 RA when compared to SGLT2i. They noted most incidents of SBO happened early within the initiation of therapy (within 3 months). At the time of writing this abstract, there are no published SBO case reports associated with tirzepatide. Further large-scale clinical trials need to be conducted to explore the association of SBO in patients with GIP/GLP-1 and GLP-1 RA. These studies will help guide if these drugs should be contraindicated or used with caution in people with a previous history of SBO or colonic obstruction.

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Seronegative Autoimmune Encephalitis as a sequela of Hyperosmolar Hyperglycemic Syndrome: A Rare Case

Educational Objective:

> Clinical presentation, diagnosis, and treatment of autoimmune encephalitis

> Role of plasmapheresis in encephalitis of unknown etiology

Introduction: Neurological disorders treated with plasma exchange, such as myasthenia gravis, Guillain-Barré syndrome, and chronic inflammatory demyelinating polyneuropathy, are typically associated with aberrant humoral immune responses. This case report explores a unique instance of encephalitis of unknown etiology following hyperosmolar hyperglycemic syndrome.

Case Description: A 61-year-old female with a history of non-insulin-dependent diabetes mellitus, hypertension, congestive heart failure, and chronic kidney disease Stage III, presented with altered mental status and a serum glucose level of 849 mg/dl. The family reported that the patient’s history was unremarkable except for non-compliance with her medications.

On admission, her Glasgow Coma Scale score was 8, necessitating intubation for airway protection and subsequent transfer to the intensive care unit. Despite initial treatment for Hyperosmolar Hyperglycemic Syndrome (HHS), including insulin infusion and electrolyte replenishment, her altered mental state persisted, necessitating an extensive workup and empirical treatment for potential meningoencephalitis. The evaluations, including laboratory tests, EEG, brain CT and MRI, and CSF analysis/culture, revealed no significant findings except for a high CSF protein count of 298. An autoimmune workup, which included screening for anti-N-Methyl-D-Aspartate antibodies, Acetylcholinesterase levels (ACE), ANA, anti-double stranded DNA, complement levels, anti-myeloperoxidase antibodies, proteinase 3 antibodies, and ANCA antibodies, was inconclusive. Nevertheless, considering a possible autoimmune etiology, a trial of plasmapheresis was initiated. This intervention led to a marked improvement in her condition, with successful extubation following two plasmapheresis sessions. The patient continued to improve and was discharged home after a total of five plasmapheresis sessions.

Conclusion:

Autoimmune encephalitis, a rare but complex condition, often presents with diverse symptoms ranging from confusion to seizures and even coma. Despite comprehensive investigations for bacterial, viral, and autoimmune causes, the etiology in 20-30% of encephalitis cases remains elusive. This often leads to the consideration of seronegative autoimmune encephalitis, a diagnosis of exclusion. There are reported instances where seronegative autoimmune encephalitis coexists with other autoimmune conditions. Our case raises a question about the role of severe hyperglycemia as a potential underlying cause. Further research is warranted to explore this potential link and to evaluate the effectiveness of plasma exchange in treating seronegative autoimmune encephalitis, especially in the context of hyperglycemic episodes.
In response to the high incidence of cardiopulmonary arrests, a leading cause of inpatient mortality, hospitals have implemented rapid response systems. This retrospective cohort study examines the role of residents in patient safety and the outcomes of rapid responses in a community-based teaching hospital.

Method:

Data from May 2022 to October 2023 were analyzed to assess the impact of rapid responses on inpatient mortality, code blues, and unexpected transfers to the ICU. The study aimed to standardize the diagnosis and treatment of patients with SIRS 2/4 as sepsis, aiming to reduce rapid responses due to SIRS >2/4 by 25% within six months. The analysis focused on whether patients with SIRS 2/4 were diagnosed and treated as sepsis before rapid response activation.

Results:

In our community-based teaching hospital, the Rapid Response Team (RRT) was swiftly activated 308 times over eighteen months, showcasing the proactive stance of our resident-led initiatives in patient care. Out of the 308 patients attended to, gender distribution revealed 178 females (57.8%) and 130 males (42.2%), reflecting the team's commitment to addressing diverse patient needs. The primary reasons for RRT activation encompassed cardiac (44.2%), neurological (25%), respiratory (20.8%), and other miscellaneous issues, illustrating the comprehensive scope of our rapid response approach. Remarkably, 9.4% of RRT activations occurred under direct resident supervision, underscoring the proactive role of our residents in patient safety. Moreover, the majority of activations (55.5%) transpired during daytime hours, highlighting the round-the-clock vigilance maintained by our resident-led team. Of the patients attended to, 33.4% were seamlessly transferred to the Intensive Care Unit (ICU) while 17.5% were directed to progressive care units, showcasing the decisive actions taken by our team to ensure optimal patient outcomes. Despite encountering systemic challenges, the mortality rate remained insignificant at 1%, a testament to the efficacy of our resident-led rapid response interventions.

Discussion: Root cause analysis identified a lack of standardized protocols for diagnosing and treating sepsis among hospital physicians and residents. To enhance effectiveness, protocols should be standardized for diagnosing and treating sepsis in patients with SIRS 2/4. Educational initiatives and quality improvement measures are essential to improve healthcare provider knowledge and skills. Foster interdisciplinary collaboration and data monitoring are crucial for optimizing rapid response team effectiveness.
A Resident-Led Initiative to Improve Heart Failure Readmission Rate by Targeting Factors of Social Vulnerability

Introduction: Heart Failure (HF) affects more than 6.5 million Americans with an estimated half-a-million new cases diagnosed annually. Readmission continues to be one of the highest burdens on the US healthcare system, with an average cost of ~$14,000 for each hospitalization, and $31 billion annually. HF poses the highest risk for rehospitalization among other medical conditions, with 2011–2014 data indicating a 30-day readmission rate of 22%. Factors that increase a patient’s HF readmission risk are often linked to social vulnerability factors (health care illiteracy, lacking transportation, etc.).

Methods: The Institute of Healthcare Improvement model was used and the Plan, Do, Study, Act (PDSA) cycle was used to format this project and test change. IRB exemption was obtained. In partnership with the HF nurse practitioners, a survey was distributed to all cardiology attendings at our hospital to identify the most pressing health care disparities in HF patients specific to our patient population. This data, along with American Heart Association and American College of Cardiology 2022 HF guidelines, was used to design the PDSA 1 education-based intervention. In January 2023, a peer-led interactive workshop was used to train residents on identifying and addressing health care disparities amongst heart failure patients. A dot phrase was created and shared with residents to document HF specific clinical status, factors that could contribute to increased risk of readmission, and patient-specific counseling that was conducted to mitigate HF exacerbation outpatient (changes in medications, appointments and follow ups, lifestyle changes, etc.). The primary outcome to measure change will be hospital-wide HF readmission rates. Post-intervention analysis will be done using simple descriptive statistics.

Results: The 2019 HF readmission rate (18.8%) at our hospital exceeded the target by 15.10%. Survey responses from cardiology attendings (n=5) showed that 60% felt patient education and understanding of the disease was the most crucial factor affecting HF readmission risk. Other causes reported were medication non-adherence and low income. When asked about which intervention would have the most impact on addressing health care disparities in HF patients, 60% of attendings highlighted patient education as a top priority. Data analysis revealed pre-intervention readmissions rate 32%, 20.8% and 22.6% for the months of November, December and January, respectively. Post-intervention readmissions rates were 6.1%, 21.7% and 28.6% for the months of February, March and April 2023, respectively. The average readmission rate for the last 6 months of 2022 was 22% prior to PDSA1. 3 months post-intervention readmission rate average was 18.8%. Further data analysis is ongoing to identify factors of social vulnerability contributing to readmission that are specific to our patient population.

Conclusion: Our project aimed to identify and address factors of social vulnerability contributing to high readmission risk in HF patients at our hospital. Future PDSA cycles of our initiative will focus on educating other health provider groups involved in patient discharge planning, e.g., floor nurses, and designing a more integrated EMR change such as mandatory HF documentation checklist linked to discharge diagnosis of HF exacerbation. Future interventions will focus on targeting disparities specific to our patient populations based on data analysis.
Exploring the Clinical Outcomes of End-Organ Damage in Hypertensive Emergency: A National Perspective

Background/AIM:
The number of emergency department visits for hypertensive emergency has been increasing and was reported to be 496,894 visits in 2013. Current recommendations are to admit these patients to the ICU and start on intravenous blood pressure-lowering medications. This study aims to assess the prevalence of each end organ damage and their effects on the outcomes during hospitalization.

Methods:
This study analyzed data from the 2016-2020 National Inpatient Sample (NIS) database, focusing on adult hospital admissions. We utilized International Classification of Diseases, 10th Revision, Clinical Modification (ICD-10-CM) codes to identify cases of hypertensive emergency (I16.1) and other relevant comorbidities. The primary objective was to assess the prevalence of end-organ damage and its impact on mortality. We employed multivariable logistic regression to adjust for demographics, comorbidities and other potential confounders.

Results:
Our comprehensive analysis included 1,032,860 hospitalizations due to hypertensive emergencies, with a notable increase in prevalence from 1,149 to 10,255 cases per 1 million patients between 2016 and 2020. The mean age of the cohort was 62.10 (± 15.97 years), with females constituting 50.7% and whites 44.97%. The most common chronic comorbidities identified were chronic kidney disease (CKD) at 44.87%, diabetes mellitus (DM) at 44.22%, coronary artery disease (CAD) at 42.49%, and congestive heart failure (CHF) at 42.46%. Among end-organ damages, acute kidney injury was most prevalent (33.37%), followed by acute heart failure/pulmonary edema (26.38%), Acute Coronary Syndromes (ACS) (15.33%), ischemic stroke (11.55%), hemorrhagic stroke (9.79%), hypertensive encephalopathy (5.30%), and aortic dissection (1.11%). In patients with hypertensive emergencies, the in-hospital mortality rate was 3.9%, and the median length of stay was 5.96 days (range: 5.91 - 6.01). Subgroup analysis revealed the highest mortality rates in patients with hemorrhagic stroke (20.0%), followed by those with aortic dissection (7.1%). Consistently, hemorrhagic stroke (OR 11.45, 95% CI 10.83-12.10, p<0.001) and aortic dissection (OR 3.11, 95% CI 2.54-3.80, p<0.001) emerged as the most significant predictors of increased in-hospital mortality after adjusting for demographics, comorbidities, and various types of end-organ damage.

Conclusion:
This study highlights a significant ongoing rise in hospital admissions due to hypertensive emergencies from 2016 to 2020, emphasizing the increasing burden of this condition. Our findings demonstrate prevalent comorbidities, end-organ damages and notable mortality rates among patients admitted with hypertensive emergencies. The study emphasizes the need for personalized patient care, underscoring the vital role of early detection and outpatient management of hypertension to prevent the onset of hypertensive emergencies and improve patient outcomes. With the rising incidence of hypertensive emergencies, it is imperative for healthcare systems to evolve, adopting new protocols and refining risk stratification criteria to effectively meet these emerging challenges.
Why didn’t this ‘pneumonia’ go away?

Introduction: It is estimated that 15% of inpatient Pulmonary Service consults are due to non-resolving pneumonia. The list of differential diagnoses for this condition is long, and in 20% of cases, the final diagnosis is non-infectious. We present a case of cryptogenic organizing pneumonia diagnosed in the context of non-resolving pneumonia.

Case Presentation: The patient is a 75-year-old gentleman with a history of cardiac failure with preserved ejection fraction, chronic kidney disease stage II, and polymyalgia rheumatic on long-term prednisone (2 mg daily) who presented to ED with persistent dry cough and shortness of breath. Five weeks prior, he had been diagnosed with community-acquired pneumonia after outpatient evaluation for cough and dyspnea. An initial CT angiogram of the chest (CTA) revealed a multifocal nodular consolidation in the right middle lobe and right lower lobe. The patient completed a 7-day course of amoxicillin-clavulanate and azithromycin with partial improvement of symptoms. Repeated CTA revealed worsening of the opacities in the right lung, and new opacities in the left lower lobe. A review of medications, along with a work-up for rheumatologic disease and infectious causes were negative. Bronchoscopy with transbronchial biopsy was negative for malignancy and infectious causes, however showed fibroblastic plugs with acute and chronic inflammation in some portions of the alveolar lung tissue. The diagnosis of cryptogenic organizing pneumonia was made. The patient was started on oral prednisone (60 mg daily) with significant clinical and radiological improvement at the 3-month follow-up.

Discussion: Non-resolving pneumonia is a common condition on the regular Internal Medicine floor. We must be aware of all the possible differential diagnoses and collect an adequate history and physical from the patient. Cryptogenic organizing pneumonia is a rare pulmonary condition that can be well controlled in 2/3 of cases when it is correctly diagnosed and managed.
Not just a headache: Spontaneous CSF leak presenting with migraine-like symptoms

INTRODUCTION

A cerebrospinal fluid (CSF) leak is a condition in which CSF escapes the subarachnoid space and is commonly associated with specific causes like trauma or surgical complications. Rarely, cases can occur spontaneously. Leakage of CSF causes symptoms of intracranial hypotension, classically including a postural headache. In this case, we describe a patient with a spontaneous CSF leak presenting with intractable headache. Initial testing included a brain CT which was remarkable only for low cerebellar tonsils, a non-specific finding.

CASE PRESENTATION

A 30-year-old woman with a history of primary hypertension and anxiety presented to the emergency department due to persistent throbbing headaches over the past few weeks. The patient reported experiencing multiple episodes daily, left-sided, accompanied by nausea, vomiting, photophobia, and visual disturbances. Despite trying various over-the-counter medications, she only obtained partial and temporary relief. Upon arrival at the emergency department, her blood pressure was elevated at 183/110 mmHg. Physical examination showed decreased strength on the left side, leading to her admission for intractable headache.

Initial tests included a normal chest x-ray and brain CT showing borderline low cerebellar tonsils, revealed no significant abnormalities. A subsequent CT angiogram of the head and neck was also unrevealing, and laboratory results were within the normal range. The following day, she developed significant burning pain throughout her left side, accompanied by a severe headache and further worsened left-sided weakness which prevented her to ambulate furthermore. Magnetic resonance imaging of the head and cervical spine revealed spontaneous intracranial hypotension associated with a CSF leak.

Neurosurgery was consulted and recommended a CT myelogram of the spine to pinpoint the leak’s location, which was identified around the T3 and T4 levels of the thoracic spine. Interventional radiology was then consulted and performed an epidural blood patch placement with the slow administration of 4 mL autologous blood. The patient had significant improvement following the procedure. She regained the ability to walk with minimal headache, and her left arm weakness significantly improved with barely perceptible weakness. The patient was discharged with follow-up appointments scheduled with her primary care physician and neurologist.

DISCUSSION

CSF leakage causes intracranial hypotension which can lead to headache and acute neurologic symptoms. The altered cerebral perfusion pressures and reduced cushioning of brain parenchyma due to low CSF volumes in intracranial hypotension can cause many complications including weakness and movement disorders, well represented by our patient. CSF leak is thought to be mainly caused by a meningeal disarrangement. Trauma is the most common cause of leak, followed by dural tear. Our patient had no history of those. Additionally, she did not report previous sinus surgery and did not present stigmata or symptoms of cranial CSF leak through the cribriform plate, which corroborates to the hypothesis of a spontaneous leak. It is important to value patient’s history and maintain a wide range of diagnoses in cases of worsening neurologic symptoms or refractory to therapy. Growing recognition of spontaneous intracranial hypotension among clinicians is critical for the early management of this condition.
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Autoimmune Paroxysmal Cold Hemoglobinuria with Syphilis: A Case Presentation

Introduction: We report a case of Donath-Landsteiner paroxysmal cold hemoglobinuria coexisting with syphilis.

Case Presentation: A 70-year-old female, known for chronic kidney disease stage IIIb, hypertension, hyperlipidemia, poorly controlled type 2 diabetes, and a history of an unprovoked saddle embolus on chronic anticoagulation, presented to the Emergency Department with shortness of breath and severe acute anemia (hemoglobin: 4.9g/dL). Anemia work-up revealed normal folic acid and vitamin B12 levels, a peripheral smear without red blood cell morphology changes, and neutrophils with hypersegmentation. Further investigations indicated late acute infection with positive Epstein-Barr virus (EBV) results, a positive direct antiglobulin test (DAT) for complement only (negative for IgG), cold agglutinin hemolytic anemia with low titers, and positive Donath-Landsteiner and syphilis treponemal antibodies.

Discussion: Donath-Landsteiner antibodies, associated with paroxysmal cold hemoglobinuria, trigger complement activation, and subsequent hemolysis. This case emphasizes the importance of considering paroxysmal cold hemoglobinuria in the differential diagnosis of acute anemia. The coexistence of syphilis adds a unique dimension to the clinical presentation, underscoring the need for a comprehensive approach to diagnostic evaluation in such cases.
Group C Streptococcus Species: A rare cause of Infective endocarditis

Infective endocarditis is defined by infection of the endocardial surface of the heart, heart valves, or indwelling cardiac device. It is commonly associated with Staphylococcus aureus, Viridans streptococci, and Streptococcus gallolyticus. This report describes a case of infective endocarditis attributed to Group C Streptococcus (GCS) or Streptococcus dysgalactiae, a rare causative agent.

A 61-year-old male with a history of psoriatic arthritis on Enbrel, DVT/PE, antiphospholipid antibody syndrome, and testicular cancer presented with fever, altered mental status, and lightheadedness for the past 3 days. He was febrile, hypertensive, and tachycardic. The physical exam was relevant for nail pitting, plaque psoriasis, Janeway lesions on the right hand and feet, and left foot swelling. Labs revealed elevated procalcitonin, thrombocytopenia, hyponatremia, and metabolic acidosis. Chest X-ray revealed retrocardiac infiltrate. Vancomycin, Tobramycin, and cefepime were started empirically. Blood cultures revealed Streptococcus dysgalactiae, hence antibiotics were deescalated to ceftriaxone. CT Chest/abdomen/pelvis revealed small pleural effusion, splenic infarct, and splenic abscesses. The patient was started on metoprolol for new-onset Atrial fibrillation with RVR. On day four, he developed right ankle tenderness. Venous Ultrasound did not reveal deep venous thrombosis. X-ray and MRI of the right foot were significant for cellulitic changes. Transesophageal echocardiography revealed endocarditis with two vegetations, the larger vegetation measuring 1.5 x 0.75 cm attached to the A1 scallop of the mitral valve and the smaller attached to the A2 scallop. It also noted trace mitral regurgitation and small patent foramen ovale. MRI brain showed multifocal strokes and acute hemorrhage in the right occipital lobe. Diagnostic angiogram showed no evidence of mycotic aneurysms however, a small right frontal arteriovenous malformation was noted, Spetzler-Martin Grade-2. Infectious Disease recommended valve replacement due to increased risk of major embolic events in group C endocarditis. Cardiothoracic surgery recommended valve replacement to the patient who elected against surgical intervention. He was discharged with a PICC line and ceftriaxone course of 4 weeks. Subsequent CT brain revealed improvement in ischemic insults with no new significant findings. Repeat TEE a month later revealed severe mitral regurgitation from previously healed endocarditis.

GCS-causing infective endocarditis can be devastating with one study reporting a 46% risk of embolization and 8% in mortality risk (1). Multiple reports describe the severe impacts of GCS, including embolization of vegetations on cardiac valves with one case report describing multiple brain embolisms despite antibiotic therapy (2). This case emphasizes the significance of a detailed history, thorough physical examination, and the inclusion of GCS as a tested organism in patients presenting with classical signs of bacteremia and infective endocarditis. Recognition of GCS as a potential causative organism is crucial for effective management given the substantial morbidity and mortality associated with the disease.

References:


Introduction
Euglycemic diabetic ketoacidosis (EDKA) is a clinical syndrome occurring both in type 1 (T1DM) and type 2 (T2DM) diabetes mellitus characterized by euglycemia (blood glucose less than 250 mg/dL) in the presence of severe metabolic acidosis (arterial pH less than 7.3, serum bicarbonate less than 18 meq/L) and ketonemia. Many risk factors are associated with EDKA, including starvation, alcohol use disorder, pregnancy, pancreatitis, glycogen storage disorders, surgery, infection, and the use of sodium-glucose cotransporter 2 (SGLT 2) inhibitors (1). We present a case of EDKA in the setting of cocaine abuse and insulin non-adherence.

Case Presentation
A 32-year-old female with medical history significant for non-compliant T1DM presented to the emergency department with a 2-day history of nausea, vomiting, and generalized abdominal pain. Upon arrival, she was tachycardic, hypertensive, lethargic and had extremely dry mucosal membranes on examination. Laboratory was significant for 4+ ketonuria, normal blood glucose of 86, a high anion gap metabolic acidosis with pH 7.28, anion gap 21, bicarbonate of 11, sodium 135, and potassium 3.9. Serum beta hydroxybutyrate was raised to 3.20. The urine drug screen was positive for cocaine and serum alcohol levels were normal. The patient was fluid resuscitated and then started on dextrose 5% plus half normal saline. 10 units of subcutaneous insulin were administered after fluid resuscitation. Shortly thereafter, the anion gap normalized without using IV insulin. Overall clinical status improved after the anion gap closed, and she was discharged with close follow-up with primary care and endocrinology.

Discussion
The incidence of EDKA has grown with the popularity of SGLT-2 inhibitors. Interestingly, cocaine has been reported to be an independent trigger for DKA in many diabetic patients. This is attributed to cocaine’s stimulatory effect on cortisol, epinephrine, and norepinephrine release from the adrenal gland [2,3]. This increase in counter regulatory hormones is the underlying cause for having DKA with hyperglycemia in such patients. Conversely, as cocaine has been shown to cause anorexic effect via suppression of feeding centers in the central nervous system [4]. This starvation effect leads to drop in the blood sugar to normal range. If the anorexic effects of cocaine are more pronounced, euglycemic DKA ensues. In conclusion, euglycemic DKA should be included in differential diagnosis in patients presenting with elevated anion gap metabolic acidosis and near-normal blood glucose, especially in the setting of cocaine abuse.

References
**Sporadic Creutzfeldt-Jakob Disease with Visual Disturbance: A Rare Presenting Symptom**

**Background:**

Creutzfeldt-Jakob disease (CJD) is a rapidly advancing neurodegenerative condition marked by dementia, myoclonus, and other neurological manifestations. While visual symptoms are present in 20% of CJD cases, very few patients present with an isolated visual disturbance without noticeable cognitive decline initially; we present one such case in our report.

**Case report:**

A 60-year-old woman presented to the emergency department (ED) for visual disturbance, worsening confusion, abnormal gait, and urinary retention. She had a history of chronic left visual impairment. Symptoms of decreased vision in the right eye had started about two months before: she had seen an ophthalmologist about four weeks after symptoms began, and a slit lamp and tonometry tests were normal; given normal C-reactive protein, serum copper, and B12 vitamin levels, she was advised to monitor symptoms at home. On three weeks follow-up, she had decreased visual acuity of 20/40 in her right eye, following which she developed progressive difficulty with word finding, memory, and wobbly gait, as well as urinary retention. In the ED, her physical exam was significant for lack of coordination, confusion, diffuse bradykinesia, abnormal extraocular eye movements, and hyperreflexia of left lower extremity, with significant motion tremors on finger-to-nose testing and diffuse spasticity. Basic blood work and computed tomography imaging of brain were largely unremarkable. MRI of the brain with and without contrast however showed mildly scattered white matter hyperintensities. Shortly after admission, the patient became non-verbal and had multiple episodes of repeated tonic-clonic seizures requiring intubation. An electroencephalogram (EEG) was performed, which showed several instances of sharp and slow waves suspicious of electrographic seizure. In the ICU post-intubation, a CSF analysis was performed, remarkable for 31 RBCs per u/L, with 10 WBCs per u/L (100% mononuclear), glucose 65 mg/dl, and protein 40 mg/dl, with negative culture, VDRL, Lyme PCR, oligoclonal bands, paraneoplastic antibodies and malignant cells. 14-3-3 protein testing on the CSF was initially inconclusive, but tau-protein was high (>20000 pg/ml). The initial Real-time quaking-induced conversion (RT-QuIC) assay was negative. Unfortunately, her encephalopathy progressed; repeat MRI of the brain with and without contrast showed cortical ribboning. Given the lack of diagnostic answers, repeat CSF collection was performed, and the RT-QuIC test resulted positive, confirming CJD. With family input she was transitioned to comfort care measures, extubated and passed away.

**Discussion:**

CJD is a disorder of poor prognosis and may be acquired through medical procedures, exposure to highly contaminated tissue or inherited genetically, but 80–90% cases are categorized as sporadic CJD, where the underlying cause remains unknown as in our case. The most common clinical manifestations of CJD are progressive cognitive decrease, dementia, myoclonus, and ataxia. When the disease involves predominant visual symptoms, it is termed as the “Heidenhain variant”. It must be considered a differential diagnosis in all patients who present with isolated visual complaints and either normal conventional brain neuroimaging or findings that do not completely correlate with the signs and symptoms. Early diagnosis can be useful in case of treatable dementia and may help in preventing iatrogenic transmission of CJD.