Implanted Cardioverter-Defibrillator (ICD) Pocket Infection due to Candida auris

Candida auris has been increasingly recognized as an emerging fungal pathogen. It is associated with nosocomial infections and poses a serious global health threat given its multidrug-resistant susceptibility pattern. ICD pocket infections due to Candida are rare with a high mortality. We present a case of an 86-year-old woman with an ICD pocket infection caused by C. auris, complicated by right ventricular vegetation or thrombus.

An 86-year-old woman with medical history significant for heart failure with reduced ejection fraction status post ICD for primary prevention, deep vein thrombosis with an inferior vena cava filter given recent gastrointestinal bleed, and failure to thrive presented for worsening dehiscence of ICD pocket site with exposed device. The patient had empirically been given amoxicillin and doxycycline when it was first observed two weeks prior. Despite antibiotics, protrusion of the device worsened, and she presented to the emergency department for evaluation.

Patient was hypotensive, tachypneic and tachycardic on presentation. Labs were significant for leukocytosis of 14.66 K/mcL and lactic acid 3.3 mmol/L. Physical examination revealed a cachectic woman with protrusion of ICD device with associated erythema, tenderness, and purulent discharge. Chest x-ray showed cardiomegaly with right pleural effusion. Blood and wound cultures were obtained, and empiric Vancomycin was initiated. Given comorbidities, electrophysiologist opted for generator removal and lead capping without lead extraction. Wound cultures obtained from both bedside and intra-operatively initially revealed Candida albicans and Fluconazole was initiated. The following day, cultures revealed C. auris resulting in Fluconazole discontinuation, anidulafungin initiation, and contact isolation.

Transthoracic echocardiography was nondiagnostic. Transesophageal echocardiography revealed an irregular mass measuring 17 x 4.2 mm attached to the defibrillator wire in the right ventricle, suspected as thrombus or vegetation. Blood cultures were finalized negative. Infectious diseases recommended six weeks of anidulafungin followed by suppression depending on susceptibility pattern, however patient unfortunately decompensated during discharge. CT angiogram chest showed pulmonary embolism within the right main and upper lobar pulmonary arteries. Heparin treatment was initiated, however the patient continued to decompensate, and the family opted for comfort care.

Extensive review of the literature failed to reveal any cases of ICD pocket infection due to C. auris. C. auris association with nosocomial spread with hospital outbreaks are increasingly being documented. Our patient had several admissions at an outside facility as well as admission to a rehabilitation center placing her at risk of acquiring this organism. In patients with ICD or permanent pacemaker infections associated with fungal organism, the entire device should be removed. The patient in this case was deemed too high risk given comorbidities and the chronicity of the device. Susceptibilities were pending at the time of patient’s death; however initial treatment of choice is an echinocandin. C. auris could develop resistance to echinocandins with their use and if the patient is failing current therapy, addition of another antifungal medication should be considered. This case highlights the complexity of treatment of C. auris as well as the high mortality associated with Candida infections due to ICDs.
Beyond the Norm: A Case of Arrhythmogenic Right Ventricular Dysplasia Presenting with Multivessel Disease and Undergoing Staged Coronary Intervention

Introduction:

Arrhythmogenic Right Ventricular Dysplasia (ARVD) is an inherited myocardial disorder characterized by fatty infiltration of the right ventricular free wall. This condition typically presents between ages 10 to 50, with an average age of diagnosis around 30 years, affecting 1/2000 to 1/5000 adults. ARVD is often associated with ventricular tachycardia or sudden cardiac death, although heart palpitations is also a common initial complaint. In this report, we present a unique case of acute coronary syndrome in a 67-year-old male patient with a known history of ARVD.

Case Description:

A 67-year-old Caucasian man with a notable medical history of ARVD, post-ICD implantation in 2015, and atrial fibrillation post-radiofrequency ablation in 2017, presented to the emergency department with acute, severe substernal chest pain. The pain was sudden in onset with 8/10 severity and described as a burning or pressure-like sensation radiating to both arms and the back. Initial management at an outlying facility included aspirin and sublingual nitroglycerin. However, his initial EKG revealed a normal sinus rhythm with incomplete right bundle branch block, and initial labs demonstrated elevated troponins, prompting his transfer to our hospital for advanced cardiac care. The patient’s family history was significant for sudden cardiac death and ARVD. His cardiac history was further complicated by atrial fibrillation, which was managed with cardioversion and ablation and ongoing treatment with metoprolol succinate, dofetilide and warfarin. A comprehensive workup including CXR, CBC, CMP, lipid panel, and TSH revealed no acute abnormalities but an elevated HbA1c of 8.2 and elevated troponin of 0.247. An echocardiogram showed an ejection fraction of 60% and a mildly increased right ventricular size, indicating a preserved systolic function. Given the patient’s unique presentation and history of ARVD, an urgent left-heart catheterization was performed. This revealed a proximal 85% eccentric lesion in the left anterior descending artery with TIMI-3 flow and a critical 99% stenosis in the right coronary artery with TIMI-2 flow. The latter was successfully addressed with percutaneous transluminal coronary angioplasty and drug-eluting stent placement, achieving 0% residual stenosis and improved TIMI-3 flow. A staged intervention for the proximal LAD lesion was conducted subsequently, achieving optimal results with 0% residual stenosis and TIMI 3 flow. The patient was initially managed with Ticagrelor and low-dose aspirin. Due to the complexity of this case, the patient’s Aspirin and Ticagrelor were stopped, and he was discharged home on Clopidogrel. His anticoagulation regimen was switched from warfarin to Eliquis 5 mg twice daily.

Conclusion:

This case illustrates how the variability in patient’s medical history, in this case ARVC, can lead to changes in how patients care is managed due to additional considerations and risks. ARVC is a rare, genetic myocardial disorder that predisposes patients to ventricular tachycardia or sudden cardiac death. ICD is a mainstay to prevent sudden cardiac death and as these patients age, even coronary interventions, when indicated, need to be tailored accordingly due to their complex condition.
Medical Student Poster # 03  

Category: Research

Medical School: Central Michigan University
Presenter: Benjamin Herdman
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Exploring the potential benefits of self breast examinations for underserved communities: A systematic review

Introduction/Background: Breast cancer is one of the most common cancers in the United States. Previously, self-breast examination (BSE) was recommended to help detect breast cancer at an early stage to improve prognosis. Since a couple of decades ago, however, it is no longer being recommended in the US due to the findings that suggest that it fails to significantly decrease mortality while leading to an increase in biopsy cases, thus causing unnecessary harm. Despite the change in the US breast cancer screening recommendations, international researchers have continued to investigate the benefits of BSE in medically underserved regions. These studies raise the possibility that BSE could also be beneficial in specific populations in the US, including rural America, where people face higher mortality from chronic diseases, like cancer, compared to the general population.

Objectives: The study goal was to determine if BSE might have benefits for medically underserved populations in order to inform a potential reevaluation of current breast cancer screening recommendations. Thus, the primary research question asked if BSE is associated with benefits including increased breast cancer detection rates, increased breast cancer screening, and decreased mortality.

Methods: To assess the benefits of BSE in medically underserved populations, a systematic review was conducted using a set of terms to search across multiple databases for articles that are relevant to identifying studies on breast cancer survival and BSE in rural and/or underserved populations within the past ten years.

Results/Conclusion: The search yielded over 200 articles across three databases and they were further screened to include studies that show 1) effects of BSE in breast cancer diagnosis and/or mortality of breast cancer patients, 2) factors contributing to the efficacy of BSE, 3) factors that affect women’s willingness to perform BSE, and 4) effects of BSE on breast cancer awareness/behaviors to seek further screening. The final synthesis from 12 papers suggests that BSE is associated with early breast cancer detection (4 out of 12 papers), increased accessibility to breast cancer screening (2 out of 12 papers), and positively influence women to seek further breast cancer screening in rural populations (3 out of 12 papers). In addition, it also identifies a potential need for improved education on breast cancer and breast cancer screening, including BSE practices, to promote early breast cancer detection (3 out of 12 papers). In sum, the work here suggests that the reevaluation of the current recommendations to determine if exceptions should be made to specific populations would be helpful in addressing late detection and poor prognosis in medically underserved American populations.
RAC P29S Mutation in HT-144 Melanoma Cell Lines Enhance Proliferation, Migration, Invasion, and Attenuation of These Biological Processes Driving Melanoma Using PI3K

Introduction: Melanoma is a malignant cancer accounting for roughly half of all skin cancer-related deaths annually. It has the highest mutation rate of any cancer, and its incidence rates are increasing. While current immunotherapies include BRAF and MEK inhibitors, treatment resistance is a growing concern. A BRAF mutation in RAC1, a Rho GTPase in the RAS pathway, has shown enhanced proliferation and increased resistance to treatment and is present in 9.2% of sun-exposed melanomas. Furthermore, when RAC1’s downstream protein Phosphoinositide 3-kinase (PI3K) becomes overactive, melanoma cells exhibit increased oncogenic effects.

Objectives: We hypothesized that overexpression of mutant RAC1 would enhance HT144 cell proliferation, migration, invasion, and anchorage-independent growth, and that treatment with a PI3K inhibitor (LY294002) would attenuate these oncogenic phenotypes.

Methods: Using HT144 melanoma cells, we investigated specific RAC1 mutations, P29S and Q61L. We investigated the effect of the P29S and Q61L mutation on cell proliferation using MTT assay for proliferation, matrigel assay for invasion, wound healing for migration, soft-agar assay for anchorage-independent growth, and western blot for RAC1 expression compared to the wildtype Rac-1 (Wt). Separately, we attempted to target RAC1 by means of PI3K inhibition, which we propose to be a possible synthetic lethal pairing to RAC1.

Results: By day 4, Q61L cell proliferation increased by 25% compared to Wt cells. On the soft-agar assay, treatment with the PI3K inhibitor led to an 82% reduction in colony formation for P29S cells, compared to a 64% reduction in Wt cells. When treated with the PI3K inhibitor, P29S cells demonstrated 78% reduced invasion in the Matrigel assay when compared to untreated P29S cells. On western blot analysis, P29S and Q61L cells demonstrated increased expression of RAC1 compared to Wt, and treatment with the PI3K inhibitor significantly decreased the expression of RAC1 in P29S and Q61L cells.

Conclusions: Overexpression of RAC1 in HT144 melanoma cells increased proliferation, invasion, and anchorage-independent cell growth. Additionally, treatment with a PI3K inhibitor demonstrated decreased proliferation, invasion, anchorage-independent cell growth, and expression of mutant RAC1 in P29S cells. This data highlights the key role RAC1 plays in attenuating melanoma proliferation and identifies a potential treatment using a PI3K inhibitor.

Significance: While the RAC1 mutation P29S is not fully understood, its implication in treatment-resistant melanomas makes it a desirable target for alternative treatments. By demonstrating the oncogenic effects of this mutation and their attenuation through PI3K inhibition, future research is warranted to evaluate the viability of PI3K inhibition as a novel therapy for treatment-resistant melanomas.
Another STEP in the right direction: Should exercise be implemented as part of medical education to improve physician health?

Learning objectives:

1. Measure the beliefs & attitudes of medical students with exercise.
2. Identify motivating factors and barriers to exercise in medical school.
3. Determine if physician health can be improved by adding exercise into the curriculum.

Project objective/background: Due to external factors such as burnout in medical school, physicians may enter their practice feeling dissatisfied or unwell. An explanation for this can be due to the rigorous nature of medical school, which doesn’t allow medical students to prioritize exercise in their own lives. This may then translate into having a poor work-life balance when medical students become physicians and are unable to prioritize their health in the workplace.

Methods/approach: A prospective needs assessment study was employed using three validated surveys to determine if exercise should be implemented as part of medical education to improve physician health. The first validated survey determined the knowledge of medical students regarding the recommendation for exercise (AAKERS- Adult Knowledge of Exercise Recommendations Survey). The second validated survey discovered the amount of time and exercise level that medical students devoted to their schedule (IPAC- International Physical Activity Questionnaire). The third validated survey measured the beliefs and attitudes of medical students towards exercise (SEES- Self-Efficacy for Exercise Scale). An open-ended survey was also used to support the SEES survey by identifying the motivating factors and barriers towards exercise.

Results: Descriptive statistical tests were used to analyze the responses. Results from the AAKERS survey displayed a mean total score of 16.13/20 (n= 52). Results from the IPAQ survey found that medical students engaged in 21.82%, 9.09%, and 69.09% of high, moderate, and low levels of exercise, respectively (n = 55). Results from the SEES survey displayed a mean total score of 51/90 (n = 55). Quantitatively, the results from the open-ended survey found that the main motivating factor involved improvements to mental health, while the main barrier involved having difficulty balancing exercise with academic load.

Conclusion: Responses from the AAKERS survey were similar to the national average score of 16/20 (n= 2002), suggesting that medical students have similar knowledge as the average population when defining correct recommendations for exercise. Responses from the IPAQ survey suggested that medical students preferred engaging in lower intensity exercise during the academic year. Responses from the SEES survey and open-ended survey suggested that medical students were unable to balance exercise with their academic courses, even though they experienced an increase in their mental health when participating in exercise. Through additional studies using the same surveys and a larger sample size, a discussion can be warranted for incorporating exercise into medical school curriculums to improve physician health.
Emergent Quadriplegia with Loss of Sensation in Guillain-Barré Syndrome: Lessons from an Axonal Variant Case

Introduction:

Guillain-Barré syndrome (GBS) is one of the most common causes of acute, acquired weakness and is often provoked by a preceding infection. Acute inflammatory demyelinating polyneuropathy (AIDP) is the most common form of GBS affecting the myelin sheath. Acute motor axonal neuropathy (AMAN) and acute motor and sensory axonal neuropathy (AMSAN) are the axonal targeting and less commonly reported forms of GBS.

Case presentation:

A 56-year-old male presented to the hospital with complaints of bilateral upper and lower extremity weakness. One week prior, he had an acute non-bloody diarrheal illness followed by bilateral hand weakness and progressive bilateral leg weakness that intensified 48 hours prior to arrival to the emergency department. On presentation, the patient had pain and quadriparesis of all four extremities, most severe in the lower extremities and right upper extremity. Neurological examination revealed significant lower facial muscle weakness, bilateral hand grip strength at 0/5, an inability to raise bilateral upper extremities above the bed, bilateral hip flexion/extension sat 0/5, and bilateral plantar flexion at 2/5 and dorsiflexion at 0/5. Additionally, the patient had reduced sensation and absent reflexes in all four extremities. Laboratory findings were significant for an elevated erythrocyte sedimentation rate at 39 and low magnesium level at 1.5. Arterial blood gas analysis was unremarkable. Brain and spinal magnetic resonance imaging were negative for acute abnormality. Lumbar puncture with cerebrospinal fluid (CSF) analysis showed albumin-cytologic dissociation with a protein count of 68 and white blood cell count of zero. While in the emergency department, he developed shortness of breath and swallowing difficulty. Urgent pulmonary function testing revealed an elevated negative inspiratory force at > -20 cmH2O and suboptimal tidal volume. Given the rapid progression of quadriplegia towards quadriplegia along with sensory involvement and newly developing bulbar involvement, the patient's presentation was suggestive of GBS. The patient was started on intravenous immunoglobulins (IVIG), and he was immediately admitted to the intensive care unit (ICU). The patient was placed on non-invasive ventilation and remained in the ICU for respiratory monitoring where he received daily IVIG but did not require intubation. Diagnosis of AMSAN was confirmed after electromyography findings showed diminished compound muscle action potentials with suppression of sensory nerve action potentials in the upper extremities. CSF analysis came back positive for anti-ganglioside antibodies anti-GD1A and anti-GD1B. The patient had only slight improvement with IVIG and was started on plasma exchange. His overall condition remains stable with slow improvement of quadriplegia and respiratory function.

Discussion:

This case of Guillain-Barré Syndrome (GBS) is notable for the rarity of its axonal variant following a diarrheal illness and the unusual onset of upper extremity symptoms before lower extremity involvement. The simultaneous motor and sensory nerve affection emphasizes the crucial role of electrodagnostic studies and cerebrospinal fluid analysis in distinguishing GBS subtypes.
**Intricate Relationship between Cholelithiasis, Hemolytic Anemia, and Pernicious Anemia**

**Background and Clinical Significance:**

Pernicious anemia is considered a rare disease with its prevalence being 1.9% in patients over the age of 60. Additionally, pernicious anemia rarely presents with pancytopenia, hemolytic anemia, and cholelithiasis. The link among the aforementioned diseases have not been explored extensively in literature. The goal of our case report is to offer a framework upon which further research can be conducted.

**Patient Description:**

This report presents a comprehensive evaluation of a 72-year-old female with a medical history of CAD, COPD, hypertension, hyperlipidemia, and hypothyroidism. She presented with generalized weakness and progressive exertional dyspnea for 4 months. She endorsed chronic diarrhea of 1 year duration and 1-2 months of weight loss (18 lbs) with night sweats. Laboratory results revealed severe anemia (hgb 4.4), leukopenia (1,800), thrombocytopenia (80,000), elevated bilirubin (2.2) and elevated LDH (4250). Intrinsic factor antibody was positive. One day after discharge the patient returned with RUQ abdominal pain and nausea. Her labs showed elevated total bilirubin, elevated direct bilirubin, and urobilinogen on UA. US revealed cholelithiasis with gallbladder wall thickening and dilated CBD.

**Interventions and Patient Outcome:**

During her first admission, she received 3 PRBC transfusions and vitamin B12 injections. On second admission, bone marrow aspirate demonstrated hypercellular marrow with mildly megaloblastoid erythroid hyperplasia. Diagnosis was pancytopenia secondary to vitamin B12 deficiency caused by pernicious anemia. Subsequent presentation of cholelithiasis was attributed to pigmented gallstones due to ineffective erythropoiesis/intramedullary hemolysis. After 2 weeks of vitamin B12 injections, repeat CBC showed WBC count normalized to 5.1, hemoglobin improved to 8.7, and platelet count improved to 613.

**Discussion:**

Pigment gallstones are associated with hypersecretion of bilirubin conjugates triggered by hemolysis and ineffective erythropoiesis. In cases of vitamin B12 deficiency, the pathologic enterohepatic cycling of unconjugated bilirubin contributes to this phenomenon. This case highlights the intricate relationship between severe vitamin B12 deficiency, ineffective erythropoiesis, and the increased risk of pigmented gallstones. The patient's hemolytic anemia, attributed to vitamin B12 deficiency, underscores the complex interplay between hematological and gastrointestinal disorders in clinical presentations, emphasizing the potential link between these conditions.
A Case of Melanoma in the Breast Masquerading as Breast Cancer

In 2023, a 48-year-old female with no significant past medical history noticed a lump in her left breast. At the time, she was outside of the United States and saw a provider overseas. She was found to have a 2.5cm lobulated soft tissue nodule in the upper outer quadrant of the left breast. A bilateral mammogram was performed at the hospital. It demonstrated a high-density circumscribed mass seen in the upper outer quadrant. A biopsy was performed and revealed ER/PR/Her2 negative breast carcinoma (triple-negative breast carcinoma, TNBC). A CT scan done overseas showed no suspicious left axillary lymphadenopathy, internal mammary, or supraclavicular lymph nodes.

Subsequently, she returned to the States to receive follow-up care. A review of the available slides at the department of pathology in our institution revealed immunohistochemical staining that is unusual for TNBC. Therefore, a repeat biopsy was recommended at tumor board. While awaiting for the biopsy results, the patient started neoadjuvant therapy with carboplatin, paclitaxel and pembrolizumab (Keynote-522). The biopsy performed in our institution favored a diagnosis of melanoma rather than TNBC, as all epithelial immunohistochemical stains were negative and immunohistochemical staining was positive for melanoma markers (S100 and SOX10). The case was sent to Memorial Sloan-Kettering Cancer Center for a second opinion and for additional immunostaining. The final pathology report came back as BRAF wild-type melanoma. The patient’s treatment was then transitioned to standard of care ipilimumab and nivolumab. The patient was recently evaluated by dermatology, and a biopsy of the right posterior ear was taken for concerns of melanoma. Otherwise, no definitive primary melanoma site was discovered. The biopsy revealed an intradermal melanocytic nevus. The patient was referred to gastroenterology for colonoscopy and esophagogastroduodenoscopy to assess for possible mucosal primary melanoma origin. We present a unique case of melanoma in the breast that was originally misdiagnosed as triple-negative, poorly differentiated breast cancer. This case emphasizes the importance of follow-up care and comprehensive evaluation of a patient presenting with atypical findings, whether clinically or histopathologically.
Unraveling the Enigma: Pigment Gallstones Amidst the Complexity of Hemolytic Anemia in the Setting of Newly Diagnosed Pernicious Anemia

Introduction:
Pernicious anemia is an autoimmune disorder due to formation of anti-intrinsic factor antibodies and subsequently lack of vitamin B12 (cobalamin) absorption in the terminal ileum. Vitamin B12 is water soluble and a necessary cofactor for metabolic processes including the synthesis of DNA and RNA. Here we present a rare case of pernicious anemia presenting as vitamin B12 deficiency precipitating hemolytic anemia leading to pigment gallstones formation.

Case Presentation:
A 72-year-old female with a history of coronary artery disease and hypothyroidism presented to the hospital with worsening sharp right upper quadrant abdominal pain associated with chronic diarrhea of one year duration and current nausea without vomiting, fever, or chills. On presentation, the patient was afebrile with stable vitals. Laboratory tests were remarkable for WBC 1.8, RBC 2.26, hemoglobin 4.4, MCV 116, hematocrit 22.6, platelets 52, total bilirubin 2.2 (direct 0.9, indirect 1.3). Urinalysis was positive for blood, leukocyte esterase, and urobilinogen of 4. Abdominal ultrasound was done and revealed cholelithiasis with gallbladder wall thickening and dilated common bile duct indicating possible cholecystitis. Due to these findings, a magnetic resonance cholangiopancreatography was ordered, and it demonstrated a dilated biliary tree with gallstones and sludge but no evidence of choledocholithiasis. Further workup included a peripheral blood smear that showed macrocytic hyperchromic RBCs with anisocytosis and few teardrop cells, leukopenia with absolute neutropenia and hyperlobated neutrophils, and thrombocytopenia. Due to concerns over possible hematologic malignancy versus pernicious anemia, a bone marrow biopsy was done and revealed pancytopenia secondary to severe B12 deficiency. Patient was tested for anti-intrinsic factor antibodies, and it was elevated at 7.4, which is consistent with pernicious anemia. The patient was managed with blood transfusions and vitamin B12 injections and was instructed to follow-up with heme/onc on discharge.

Discussion:
Vitamine B12 is a cofactor for methionine synthase, which catalyzes the conversion of homocysteine to methionine. Therefore, vitamin B12 deficiency can cause dysfunction of methionine synthase leading to decreased DNA synthesis and ineffective hematopoiesis and pancytopenia. Vitamin B12 deficiency also causes a buildup in homocysteine, which if elevated, will cause hemolysis. Here we present a rare case of severe vitamin B12 deficiency complicated by hemolytic anemia and pigment gallstones formation.

Conclusion:
While vitamin B12 deficiency is a well-known cause of megaloblastic anemia, its association with hemolysis and gallstone formation is uncommon and noteworthy. This case highlights the complexity of hematological disorders and the importance of considering less common etiologies when faced with atypical clinical presentations. Early diagnosis and appropriate management of vitamin B12 deficiency are critical to prevent potentially life-threatening complications, such as hemolytic anemia and gallstone formation. Understanding the diverse manifestations of vitamin B12 deficiency and its potential hematological consequences can aid healthcare providers in delivering timely and effective care to patients with similar clinical profiles.
Pericardial Effusion Unveiling: An Uncommon Complication Arising from Psoriasis-Induced Pericarditis

Introduction

Autoimmune rheumatologic conditions, such as psoriasis, can manifest with cardiovascular complications including valvular disorders and pericarditis. Inflammatory cytokines in the pericardial fluid may cause local autoimmune and inflammatory events including progression to pericardial effusion. Here we present the case of an uncommon cause of pericardial effusion secondary to psoriasis-induced pericarditis.

Case Report

A 55-year-old female with a history of nonischemic cardiomyopathy, hypertension, asthma, and psoriasis presented to the emergency department with shortness of breath, orthopnea, and bilateral leg swelling for two weeks. The patient had a history of psoriasis, which was treated with a topical steroid at baseline. There were active psoriatic lesions noted on her arms. She had a history of positive ANA, and her CRP was elevated at 1.4. U1RNP and ENA were both positive. Upon arrival, the patient’s blood pressure was 200/100, troponins were 22, and BNP was 700. A chest x-ray demonstrated mild interstitial pulmonary edema and cardiomegaly. The patient was initially treated with furosemide, spironolactone, and carvedilol. Cardiology and rheumatology services were consulted, a transthoracic echocardiogram was done and showed left ventricular ejection fraction (LVEF) of 30-35% with moderate, mostly posterior, pericardial effusion without clear echocardiographic evidence of tamponade. It was determined that the patient had pericarditis secondary to her underlying psoriasis; therefore, she was treated with Colchicine 0.6mg BID for 8 weeks. A 2 month follow-up echocardiogram showed reduction in the size of the pericardial effusion from moderate to small and improved LVEF of 50-55% consistent with patient symptomatic improvement.

Discussion

Patients with autoimmune rheumatologic conditions are at an increased risk of cardiovascular disease. Specifically, individuals with psoriasis have been shown to have higher rates of occurrence of valvular anomalies and pericardial effusions. This case highlights the importance of considering dermatologic conditions in the evaluation of pericardial effusion. As a result of the increased concomitant incidence of heart pathology and rheumatologic diseases, dermatologic history should be obtained from each patient and testing should be performed in association with clinical suspicion. Conducting a comprehensive patient history, including dermatologic and rheumatologic aspects, is crucial in cases of pericardial effusion of unknown etiology. This patient’s presentation underscores the need for a holistic approach to diagnosis ensuring that potential rheumatologic and dermatologic connections are explored in evaluation of pericardial effusion.

Conclusion

This case emphasizes the significance of recognizing the potential association between autoimmune rheumatologic conditions and cardiovascular complications, such as pericardial effusions. Clinicians should be vigilant in obtaining a thorough patient history, including dermatologic and rheumatologic elements, when faced with pericardial effusions of unknown origin. A multidisciplinary approach and appropriate testing guided by clinical suspicion are crucial in achieving accurate diagnoses and optimizing patient care.
EHR Simulation to Enhance Addiction Medicine Training and Reduce Stigma

Substance use disorder (SUD) training and Electronic Health Record (EHR) training have been targeted for improvement within undergraduate and graduate medical education programs. Physicians spend approximately 35% of their time documenting patient information, and in some specialties, physicians spend more than half of their time within an EHR system. Additionally, stigmatizing views and attitudes of medical professionals toward individuals with SUDs are common and hinder access to quality health care.

Due to a lack of EHR training systems, we created a free and customizable platform utilizing Google Sites to simulate an EHR user experience specific to patients with SUDs in a primary care setting. The purpose of this pilot project is to decrease the stigma around opioid use disorder and improve medical education on appropriate, safe opioid prescribing practices across a diversity of patient cases and scenarios.

Our training program, Harnessing Electronic Records to Organize and Illustrate Cases (HEROIC), simulates the EHR user experience, including managing in-basket messages, chart reviews, and accessing prescription drug monitoring information. As an assignment, HEROIC has been designed to cover topics focused on improving proficiency in opioid prescription management, patient and interprofessional communication, EHR task management, and reducing stigma. Each patient case in the HEROIC assignment centers on diverse individuals with SUD or at-risk for developing a SUD, highlighting decision-making skills and communication for complex circumstances.

To assess the strengths and weaknesses of the assignment format and learning experience from student perspectives, free-response questions were included for qualitative evaluation. We implemented pre-port surveys with the previously validated Substance Abuse Attitude Survey (SAAS) questions to assess the impact of the HEROIC assignment on student attitudes and perspectives related to SUDs. Response averages were calculated for the 10 Likert scale survey questions to identify trends in student attitudes and for pre-post comparison of means.

Preliminary qualitative results for strengths of the assignment and learning platform included “The unique interface of the exercise was enjoyable,” and “It was realistic and detailed.” Preliminary results for weaknesses included “Some of the questions were redundant,” and “clicking back and forth from the chart.” Preliminary survey data on student attitudes and perspectives included 51 pre-assignment survey responses and 19 post-assignment survey responses. Of the 19 post-assignment responses, no statistically significant differences were found in comparison to the pre-assignment responses.

The development and implementation of the HEROIC assignment established a new learning platform for student learning and engagement in undergraduate medical education. The preliminary data from the pre-post assignment surveys did not reveal a change in student attitudes and perspectives following the completion of the HEROIC assignment. This may indicate that our educational materials and exposure to non-stigmatizing example cases did not decrease the stigma related to patients living with SUDs. However, there were significant limitations to the current analysis, including a small sample size and self-selection of students who opted into completing the optional assignment. Student experiences from qualitative responses were supportive of the assignment and areas for improvement were identified for future cohorts.
Revealing Uncommon Grounds: A Case Report on Thyrotoxicosis Induced by MSSA Bacteremia Complicated with Thyroid Abscess

Introduction
Thyrotoxicosis is a condition of inappropriately high levels of serum thyroid hormones that induce a hypermetabolic state with a significant risk for fatal complications. Although the thyroid gland is an uncommon site for hematogenously-spread infection due to its strong lymphatic drainage, high vascularity, fibrous encapsulation, and significant iodine concentration, bacterial colonization can lead to subacute thyroiditis and destruction of thyroid tissue causing massive release of thyroid hormones.

Case Report
A 45-year-old male with a history of polysubstance abuse who was brought to the ED after being found unresponsive. Upon arrival, the patient was disoriented, tachycardic, and hypertensive. Blood work was significant for K 7.9, Cr 2.22 (baseline 0.6), AST 269, ALT 207, lactate 1.8, CPK 6613. Urine drug screen was positive for amphetamines, methamphetamines, cannabinoids, benzodiazepines, and cocaine; urine myoglobin was also positive. The patient was medically managed for AKI and rhabdomyolysis. Patient initially improved; however, on day 11 of admission, he developed fever, CRP of 45, and ESR of 86 hence sepsis workup was done. Blood cultures grew MSSA for which he was treated with cefazolin and Flagyl. As the patient’s fever persisted, he began complaining of severe throat pain, which prompted a CT neck that showed interval enlargement with diffuse inhomogeneity consistent with acute vs subacute thyroiditis. Thyroid function tests were significant for TSH 0.03, T4 free 4.29, and T3 free 6.1. Final pathology report of fine needle biopsy showed benign follicular cells with abundant inflammation suggestive of abscess. The patient was placed on levothyroxine as his condition progressed to the hypothyroid stage of thyroiditis. His AKI and rhabdomyolysis resolved, and was discharged with outpatient endocrinology follow-up.

Discussion
Thyrotoxicosis commonly occurs in association with Graves’ disease, toxic multinodular goiter, or toxic adenoma. Clinical findings may include tachycardia, palpitations, fatigue, weight loss, and fine tremor. The condition more likely arises in individuals with pre-existing thyroid dysfunction or in the setting of immunocompromise. This case of thyrotoxicosis in the setting of MSSA bacteremia provides a unique presentation of the condition and provides insight into disease progression. Individuals with a history of intravenous drug use may face heightened vulnerability to thyrotoxicosis in the setting of bacteremia particularly from staphylococcus aureus and streptococcal species. This presentation of disease raises concerns for the development of acute suppurative thyroiditis and its associated complications including the risk for airway obstruction, vocal cord paralysis, and permanent thyroid damage. In instances of thyrotoxicosis with an unidentified origin, it is imperative to explore the association with bacteremia and carefully consider potential sequelae.

Conclusion
This case demonstrates a rare presentation of thyrotoxicosis in the setting of MSSA bacteremia and thyroid abscess in a patient with history of polysubstance abuse. The manifestation of thyrotoxicosis ranges from subclinical to severe with the capability of causing life-threatening thyroid storm. Early diagnosis and management of thyrotoxicosis in the setting of MSSA with appropriate antibiotics and thyroid hormone supplementation is critical due to the considerable risk of morbidity and mortality of the untreated course.
Medical Student Poster # 13  

**Category:** Clinical Vignette  

**Medical School:** Michigan State University College of Osteopathic Medicine  
**Presenter:** Celine Adriano  
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**Trauma Obscuring Leukemia - A Unique Case Report**  

**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.  

Although treatments will vary, systemic chemotherapy remains a mainstay of treatment. Stem cell transplants can also be employed. Patients diagnosed with AML have a poor prognosis, with a five-year survival rate of 28.3%.  

**Conclusion**  
At its core, this is a case of missed diagnosis and the need to emphasize holistic workup. Although the patient experienced a traumatic event that could have accounted for his symptoms, he was seen by several facilities without thorough care, causing a delay in the diagnosis and treatment.
Severe Paenibacillus Sepsis in a Premature Neonate

Paenibacillus sepsis is extremely rare in humans. Paenibacillus is an anaerobic Gram negative bacillus that is ubiquitous in nature. We report a case of Paenibacillus meningo-encephalitis in a preterm neonate that resulted in poor neurological outcome.

Our patient was born at 33 weeks gestation to a 30 year old mother who developed severe pre-eclampsia during pregnancy. The fetus was prenatally diagnosed with growth restriction and bilateral cleft lip and palate. The infant was delivered via cesarean section and required intubation after birth for respiratory distress. He was transferred to level IV neonatal intensive care unit for evaluation and management of cleft lip and palate. As a part of work-up for his cleft lip and palate and to exclude any associated anomaly, an echocardiogram, head ultrasound and ophthalmological exam were performed and were reported to be normal for his age. The infant was extubated after 11 days and placed on nasal cannula. He received a dental plate at 2 weeks of age and remained clinically stable. A week later, he developed fever and at that time a complete septic work up was initiated including spinal tap. CSF cell count was concerning for meningitis. He was started on broad spectrum antibiotics. The infant's respiratory status deteriorated and he required escalation of support and was placed on high flow nasal cannula. Blood culture grew gram negative bacilli and a DNA probe assay detected Paenibacillus sp. An MRI of the brain obtained at that time revealed extensive medullary vein thrombosis mainly involving the left posterior frontal and parietal deep and subcortical white matters with associated diffusion signal abnormality. Infant was treated with Vancomycin, Meropenem and Ceftazidime for 2 weeks and later switched to Ampillin. He was also anti-coagulated with Heparin. Repeat MRI showed bilateral asymmetric macrocystic encephalomalacic changes in the parieto-occipital lobe left greater than right which was likely representing propagation of the previously demonstrated asymmetric large areas of venous infarct due to underlying medullary vein thrombosis. Subsequent MRI’s showed development of ex-vacuo hydrocephalus. The infant was discharged home at 4 months of age and required G-tube for feeding. He was re-admitted at 6 months of age with seizures and found to have hypsarrhythmia and infantile spasms on EEG. He was diagnosed with global developmental delay and he continues to follow up with neurology and is receiving occupational and physical therapies.

Despite timely diagnosis and aggressive management, our patient suffered significant neurological damage. In conclusion, Paenibacillus is an important organism that could cause severe infection resulting in devastating consequences for immunological compromised preterm infants.
A Rare Case of Gastric Variceal Bleeding and Idiopathic Non-Cirrhotic Portal Hypertension in the Absence of Splenic Vein Thrombosis and Liver Cirrhosis

Portal hypertension (PHT) is commonly caused by cirrhosis or obstruction within the portal vein or splenic vein. Idiopathic non-cirrhotic portal hypertension (INCPH) arises in the absence of any clear etiology. Gastric varices in particular, are strongly associated with splenic vein thrombosis and an endoscopic discovery of gastric varices should prompt a workup for splenic vein thrombosis.

We present a case of a 45-year-old female with no prior history of liver disease who presented to the ED with two episodes of gross hematemesis associated with intermittent, sharp, left-sided chest pain. On presentation, she was found to be severely anemic with a hemoglobin of 2.5. Cross-sectional imaging of the abdomen revealed multiple splenic, gastric, and small para-esophageal varices. Doppler ultrasonography demonstrated flow through the splenic and portal vascular systems. Additional workup including liver function tests, hepatitis panels, and autoantibodies were unremarkable. After stabilization, Esophagogastroduodenoscopy (EGD) showed isolated gastric varices requiring treatment with embolization of the gastro-renal shunt performed by interventional radiology as well as balloon-occluded retrograde transvenous obliteration for secondary prophylaxis.

This case highlights the importance of being attentive to the presence of gastric varices during an EGD when encountering a patient with hematemesis, despite splenic vein thrombosis or liver cirrhosis not being evident. Before assigning the diagnosis of INCPH, it is essential to eliminate other typical causes of PHT.
Not-So-Typical Neurologic Effects: Unraveling Lithium Toxicity

Introduction: Lithium is one of the mainstay treatments for bipolar disorder. Given its narrow therapeutic window, acute and chronic toxicity can develop. Acute toxicity includes gastrointestinal and cardiac manifestations whereas chronic toxicity manifests as neurologic, thyroid, and renal dysfunction. The therapeutic range of lithium is 0.6 to 1.2 mmol/L. With chronic use, lithium may be toxic at 1.2 mmol/L or greater (1). Neurological toxicity includes tremors, hyperreflexia, nystagmus, ataxia, confusion, and even seizures, however, rare reports have shown peripheral neuropathy as a manifestation.

Case: A 40-year-old male with a history of bipolar disorder complicated by lithium induced chronic kidney disease stage 3 and hypothyroidism presented for acute on chronic severe bilateral lower extremity burning pain and paresthesias. The patient described a gradual two month progression of worsening painful paresthesias beginning in the great toes bilaterally and spreading up both feet to the level of the ankles. The pain worsens with weight bearing. He took no pain medication for his symptoms. The patient was evaluated previously in the outpatient setting a few months prior where no metabolic correlation was found and electromyography demonstrated severe bilateral lower extremity neuropathy and atrophy. Due to the on-going severity of his symptoms, he presented to the hospital for evaluation. On exam, sensation to pinprick was decreased distally and hyperesthesia and allodynia to light touch were evident in bilateral feet. There was no evidence of focal motor deficit, upper extremity or facial symptoms. No sensory level was appreciated, strength, tone and bulk were adequate, and reflexes were symmetric bilaterally.

CT and MRI of the brain were negative for acute intracranial process. MRIs of the spine showed degenerative changes out of proportion to his symptom severity. Laboratory studies for an inflammatory/autoimmune process were negative making vasculitis less likely. The patient was started on Gabapentin. The neuromuscular team was consulted who expressed concern for lithium induced peripheral neuropathy considering the patient’s hospital admission for lithium toxicity (levels up to 2.14 mEq/L) three months prior coincided with his symptom onset. Given the patient’s concurrent lithium induced thyroidopathy and nephropathy, recommendation to permanently discontinue lithium is maintained with concern that this peripheral neuropathy, although rare, is secondary to toxic lithium levels.

Discussion: The differential diagnosis of peripheral neuropathy is broad including metabolic, systemic, structural, and even toxic causes. Despite being extremely rare, Faravelli et al. has reported that chronic lithium use at therapeutic levels has been associated with peripheral neuropathy (1). Here, we present this atypical neurologic symptom that must be considered in an individual taking lithium with no other overt cause as prompt discontinuation of the treatment is imperative. Given that this patient is fairly young and healthy, we anticipate that he will have a much better recovery from his peripheral neuropathy than someone of advanced age with comorbidities, again pointing to the importance of early suspicion.

Aggressive Renal Cell Carcinoma in a Patient With a Rare Genetic Syndrome

Hereditary leiomyomatosis and renal cell carcinoma (HLRCC) is an exceedingly rare genetic disorder characterized by skin and uterine leiomyomas and an increased risk of renal cell cancer. HLRCC follows an autosomal dominant pattern and is due to inactivating germline and somatic mutations in the gene encoding the fumarate hydratase enzyme (FH) involved in the citric acid cycle. The rarity and wide phenotypic variation in the presentation of HLRCC complicates suspicion and testing for this syndrome.

We describe the case of a young woman who presented with pathological fracture secondary to bone metastasis from renal cell carcinoma. The patient presented to the emergency department with complaints of sudden and severe right arm pain. Right humerus radiography revealed permeative, “moth-eaten” lesions of the distal humeral diaphysis. Magnetic resonance imaging of the upper right arm supported suspicion for metastasis given a history of suspected renal cell carcinoma in childhood. The patient underwent successful particle embolization of the metastatic renal cell carcinoma in the distal humerus and follow-up angiogram demonstrated significant reduction of the neovascularity and tumor stain involving the humerus.

Chest CT demonstrated gross splenomegaly and enlarged retroperitoneal lymph nodes. Biopsy of the left retroperitoneal lymph nodes revealed high-grade metastatic renal cell carcinoma with features suggestive of fumarate hydratase-deficient hereditary leiomyomatosis renal cell carcinoma. PET/CT revealed additional metastases in the right hepatic lobe. The patient was urged to undergo genetic testing with a multi-gene panel, which confirmed a germline pathologic variant of the fumarate hydratase gene.

Additional history revealed the patient was diagnosed with polycystic kidney disease in childhood. At the time, bilateral nephrectomy was recommended due to biopsy suspicious for renal cell carcinoma but was not undertaken. Several years later, the patient developed biopsy-confirmed leiomyomas on the skin of her legs and was advised by her dermatologist, who suspected Reed’s Syndrome (Multiple Cutaneous and Uterine Leiomyomatosis), to undergo genetic evaluation for a variant of the FH gene; however, genetic testing was not undertaken at that time.

Due to the rarity of HLRCC, there are currently no established guidelines on the treatment of this type of renal cell cancer. Limited studies, however, have demonstrated immune checkpoint inhibitors and anti-angiogenic molecules have proven beneficial and the patient was recommended to start a combination of immunotherapy and targeted treatment.

This case highlights the importance of proceeding with screening of pathologic variants of the fumarate hydratase gene in patients with a history of leiomyomas and atypical renal pathology findings. Early identification can lead to appropriate surveillance for renal cell carcinoma and prevent an aggressive disease course.
Moonlighting: a pilot project to assess impact of extracurricular activities on career satisfaction and quality-of-life in graduate medical education

Background:
Moonlighting is the delivery of healthcare outside of training programs in exchange for compensation. The Accreditation Council on Graduate Medical Education allows individual training programs to permit or restrict moonlighting, which leads to variation in prevalence and practice. Guidelines and regulations for moonlighting vary greatly across graduate medical education programs. Yet, limited research has been done to determine resident and fellow physician attitudes towards moonlighting and its impact on quality-of-life.

Objective:
This pilot study investigates how participating in moonlighting and extracurricular activities may impact resident and fellow physicians’ self-perceptions of quality-of-life and career satisfaction.

Methods:
442 residents and fellow physicians were invited via email to participate in an anonymous REDCap survey during the spring of 2023. Only fully completed survey responses were included for final analysis which was performed using R Statistical Software. Responses were reviewed in aggregate and with stratification by particular participant characteristics.

Results:
A total of 93 responses were received (response rate 21%) and seventy-four completed responses (response rate 17%) were included for analysis. Trainees who moonlighted versus trainees who did not had no significant differences in age, gender, race, number of dependents, and marital status. 53 trainees indicated their program allowed moonlighting, of which 36 individuals moonlighted. Those who moonlighted were more likely to respond that moonlighting somewhat or very positively impacted their career satisfaction, quality-of-life, and ability to function effectively in their program (86%, 77%, 75%, respectively) compared to those who did not. No significant associations were noted between ratings of how participating in moonlighting specifically versus extracurricular activities in general impacted job satisfaction or quality-of-life.

Conclusions:
Participating in moonlighting may confer certain beneficial outcomes to resident and fellow physician participants. It seems, however, that the financial gain of moonlighting alone did not generate significant differences in self-perceptions of job satisfaction or quality-of-life compared to other forms of community involvement. Exploration into other potential contributing traits such as acquisition of translatable skills should be considered.
Rare Case of Hemorrhagic Pericardial Effusion Due to Coxsackie B Pericarditis

Introduction

Acute pericarditis is caused by inflammation of the pericardial sac. Amongst the vast number of potential causes, viruses tend to trigger pericarditis most frequently. Some of the more common viral causes are coxsackie A/B, echovirus, adenovirus, CMV, HSV, and HIV.1-3 Pericarditis often presents with signs and symptoms such as a friction rub, pain relieved with sitting up and leaning forward. ST segment elevations and PR segment depressions are seen on EKG. Laboratory tests showed increased inflammatory markers and WBC count. Pericardial effusion is a common complication and can be visualized on echocardiogram. In some cases, the pericardial effusion can be hemorrhagic in nature, which is extremely rare in the setting of viral pericarditis. The most common causes of hemorrhagic effusion are myocardial infarction, trauma, aortic dissection, or CABG surgery. Pericardial effusion can sometimes result in serious complications such as cardiac tamponade.4-6 NSAIDs, colchicine, and in some cases, prednisone can be used to treat pericarditis. In cases of significant pericardial effusion, pericardiocentesis may be required.6-8 We present an interesting case of pericarditis caused by the Coxsackie B virus causing significant hemorrhagic pericardial effusion requiring pericardiocentesis in a young patient.

Presentation

A 37-year-old female with no past medical history presented with substernal chest pain radiating to the left arm and shoulder that improved with leaning forward and dyspnea for two weeks. She also had a 2-week history of a cough, dysphagia, fever, and chills that started 2 days prior to the presentation. Vitals were remarkable for tachycardia in the 110s and tachypnea when breathing room air. Physical exam was unremarkable with no signs of edema, jugular venous distension, or muffled heart sounds. EKG showed widespread ST elevations and this is consistent with a diagnosis of pericarditis. Echocardiogram suggested acute pericarditis with a large pericardial effusion. 350mL of fluid was removed by pericardiocentesis. Cell count showed 201,000 RBCs/mcL and 9,350 nucleated cells/mcL. Cytology negative for malignancy. Cultures were negative for bacteria and fungi. Serum serology showed elevated inflammatory markers, CRP 140 mg/L and ESR 112 mm/hr. Increased Coxsackie B antibody titers, 1:160 for type 2 and 1:320 for type 3. Troponin levels were normal. She improved with pericardiocentesis and had minimal recurrence after drain removal. She was started on NSAIDs and colchicine.

Discussion

This is a unique case showing that while small exudative pericardial effusions may occur with viral pericarditis, viral infections can also cause a significant hemorrhagic pericardial effusion. Most Coxsackie virus infections are benign; however, there are a few documented case reports of hemorrhagic pericardial effusion from Coxsackie B causing tamponade and death.1,9 This case highlights the importance of considering viral infections as a potential cause of hemorrhagic tamponade, especially in young males during autumn and winter months, as those patients are at the highest risk.10
Circulatory Conundrum: Investigating the Intersection of an ST-Elevation Myocardial Infarction with Myocardial Bridge

A myocardial bridge (MB) is defined by the deviation of a coronary artery segment from its typical epicardial course, instead traversing through the myocardium. Individuals afflicted with myocardial bridges may manifest symptoms including exertional chest pain, dizziness, diaphoresis, and shortness of breath, which cannot be attributed to a secondary etiology. The prevalence of myocardial bridge is approximately 19%, although post-mortem studies have revealed a prevalence of 42% since most cases are asymptomatic. We report the case of a 58-year-old Caucasian male who presented with chest pain. A 58-year-old male with PMHx of ESRD due to polycystic kidney disease—on hemodialysis, anemia of chronic disease, HTN, and brain aneurysm was transferred to our hospital for evaluation of a coronary artery bypass grafting (CABG) vs. high-risk percutaneous coronary intervention (PCI). Over the last month, he experienced SOB and intermittent burning-pressure like chest pain. His vitals on presentation were stable. EKG showed ST elevation in leads III and AvF with reciprocal changes in the anterior leads, consistent with an acute inferior STEMI and the cath lab was immediately activated. Oral aspirin, sublingual nitroglycerin, and IV morphine and heparin were administered. Labs showed elevated Troponin I at 0.77ng/mL. CXR showed mild pulmonary congestion. Urgent catheterization via right groin showed severe 3-vessel CAD with left main involvement. The LCx and RCA were 100% occluded and PCI failed. The LAD showed moderate disease with bridging mid-segment. Balloon angioplasty of the left main was done with a 2.5mm balloon to improve flow to the LAD and collaterals. An intra-aortic balloon pump (IABP) was placed and he was transferred to our CVICU facility in stable condition. A myocardial bridge is characterized as a congenital cardiac anomaly wherein one of the coronary arteries traverses a segment of the myocardial tissue. In the typical cardiac anatomy, coronary arteries are situated directly atop the myocardium. This arrangement facilitates the unimpeded perfusion of blood to septal arteries, thereby nourishing the myocardium. In the prenatal developmental phase, a muscular band may emerge encircling one of the coronary arteries, resulting in the formation of a myocardial bridge over the affected artery. During each myocardial contraction, the bridge applies pressure, inducing constriction in the specified artery. Consequently, this constriction may precipitate diminished blood flow to the heart. Effectively addressing symptomatic myocardial bridge poses a considerable challenge in clinical management. Clinicians are advised to meticulously evaluate the patient’s symptoms, cardiac anatomy, extent of ischemia, and the presence of concurrent comorbidities. Presently, comprehensive cardiovascular society guidelines pertaining to the diagnosis or management of MB do not exist. Nevertheless, cardiac catheterization offers insight into the assessment and diagnosis of MB. Traditionally, angiography stands as the primary diagnostic modality for myocardial bridge. The identification of systolic narrowing or "milking" of the vessel during angiography serves as a key indicator of MB. In cases where patients experience intolerable or worsening chest pain, a surgical unroofing procedure represents a viable intervention. To enhance patient care for myocardial bridge, we aim to show the collaborative, multidisciplinary approach between medicine and cardiology in its assessment.
Celiac Disease Masking Crohn’s: A Case of Delayed Diagnosis

Introduction:

Crohn’s disease and celiac disease are chronic inflammatory conditions that share similar clinical features of abdominal pain and diarrhea but differ significantly in pathophysiology. Diagnostic protocol for celiac primarily involves positive tissue transglutaminase (tTG)-IgA antibody findings and further confirmation with tissue biopsy with esophagogastroduodenoscopy (EGD). Similarly, colonoscopy and lab findings of elevated inflammatory markers are needed in patients with Crohn’s disease. Celiac disease is managed with a gluten-free diet and iron supplementation in cases of anemia. However, crohn’s disease entails a complex plan involving corticosteroids and biologics to prevent disease progression. We discuss an important case of a 38-year-old woman who was diagnosed and treated with celiac disease and presented with bowel perforation likely due to undiagnosed crohn’s disease emphasizing the importance of understanding the overlap between the two diseases with very different work-up and treatments.

Case Presentation:

A 38-year-old woman with a history of psoriasis and diagnosed with celiac disease four years prior to presentation via clinical symptoms, positive celiac panel, EGD, and histopathology. On presentation, she was following a gluten-free diet, however, she had persistent intermittent diarrhea and abdominal cramps. After almost four years of diagnosis with celiac disease, she presented with chief complaints of fever and abdominal pain for two weeks that worsened two days prior to the presentation. Vital signs on presentation were significant for elevated temperature (Tmax: 38.8°C), and tachycardia (HR: 111/min). Physical exam was positive for soft, non-distended abdomen with severe generalized tenderness with guarding and a large mass in the right lower quadrant suggestive of acute abdomen. Pertinent labs were elevated WBCs (17.1 K/mcL) and low Hb (9 mg/dL). CT abdo-pelvis revealed pancolitis with proximal colon perforation and right lower quadrant collection of irregular fluid and gas suggestive of phlegmon. She underwent emergent subtotal colectomy, small bowel resection, and end ileostomy. Histopathologic exam showed severe transmural inflammation and alternating bands of stricture and dilation with acute patch suppurative serositis and perforation consistent with fulminant Crohn’s disease. She tolerated the procedure well and the final diagnosis was Crohn’s disease complicated by bowel perforation. She was started on adalimumab on outpatient follow-up with gastroenterology after discharge and repeat colonoscopy did not show endoscopic or histopathological evidence of active Crohn’s disease.

Discussion:

This case underscores the importance of recognizing the overlapping symptoms of these conditions and advocates for appropriate early testing and intervention. Timely diagnosis is crucial to prevent complications and improve patient outcomes, emphasizing the need for increased awareness within the medical community. A retrospective study by Yang et al, analyzed 455 patients with celiac disease, and concluded that IBD was significantly more common in patients with celiac disease as compared to the general population. Surveillance colonoscopy guidelines should be considered for patients with refractory celiac disease to assess for crohn’s sooner.

In conclusion, celiac disease, seemingly benign, can mask symptoms of a more insidious crohn’s disease. As clinicians, we must be vigilant about this possible concomitant overlap, especially in patients with refractory celiac disease.
Primary Polycythemia Vera: A Rare Case of Portal Vein Thrombosis

Introduction:

Primary polycythemia vera is a myeloproliferative disorder characterized by elevated levels of red blood cells, white blood cells, and platelets. This condition carries a significant risk of complications, with bleeding and thrombotic events being the most concerning. Thrombotic complications associated with polycythemia vera commonly involve deep venous thrombosis (DVT), pulmonary embolism (PE), Budd-Chiari syndrome, splenic vein thrombosis, stroke, and arterial thrombosis.

While these sites are typically associated with thrombotic events in patients with polycythemia vera, we present a unique case involving a young patient who presented with right upper quadrant pain. Further investigation revealed portal vein thrombosis, along with occlusion of the splenic and proximal superior mesenteric veins. This presentation of thrombosis in unusual locations adds to the complexity of the disease and highlights the importance of considering atypical manifestations in the diagnosis and management of polycythemia vera.

Case Report:

The patient is a 21-year-old Caucasian female with a history of polycythemia vera with a JAK-2 mutation, essential thrombocythemia, acquired von Willebrand factor deficiency, pseudotumor cerebri, and anxiety. She presented to the emergency department with sharp stabbing right upper quadrant pain that started the day before her presentation. She had previously been on treatment for her essential thrombocythemia with hydroxyurea and aspirin. However, the patient had not received treatment for her hematological disorders for the past 4-5 years prior to her presentation. Upon workup, her complete blood count was significant for an elevated red blood cell and platelet count. Her CT abdomen was significant for chronic portal vein, splenic vein and proximal superior mesenteric vein occlusions small volume ascites and splenomegaly consistent with portal hypertension. In the setting of portal hypertension she underwent an endoscopy to rule out esophageal and gastric varices. She was ultimately started on subcutaneous enoxaparin for management.

Discussion:

While portal vein thrombosis is frequently associated with cirrhosis, it can occur in any prothrombotic condition, including polycythemia vera. Diagnosing this condition typically involves the use of CT/MRI or Doppler ultrasound imaging. However, it is important to maintain a high level of suspicion for portal vein thrombosis in individuals presenting with a prothrombotic state. Complications of this condition encompass portal hypertension, intestinal ischemia, and septic portal vein thrombosis. Early diagnosis is crucial in order to minimize morbidity and mortality. Currently, available treatment options include anticoagulation, thrombolysis, and thrombectomy.

Conclusion:

Thrombotic events in patients with primary polycythemia vera can have significant implications for morbidity and mortality. The occurrence of thrombosis in the portal vein is considered atypical but not unheard of. Although some patients may present without pain, it is imperative to achieve early diagnosis and initiate prompt treatment to prevent complications. Fortunately, the availability of CT/MRI and ultrasonography imaging studies enables convenient diagnosis and appropriate management, leading to a notable reduction in mortality rates.
**Medical Student Poster # 23**

**Category:** Continuous Quality Improvement/Evidence-based Medicine

**Medical School:** University of Medicine and Health Sciences Basseterre

**Presenter:** Jessica Lutkenhoff

**Additional Authors:** Dhairya Salvi, Nour Aldaoud, Vamsi Krishna Lavu, Abdullah Yesilyaprak, Vesna Tegeltija

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**Standardization of Inpatient Hypertension Management**

**Problem Statement:** Hypertension is a frequent occurrence during hospitalization for any medical problem and physicians are routinely called to treat hypertensive events despite the asymptomatic nature of patients. Although guidelines about outpatient management of hypertension are available, management strategies for asymptomatic inpatient hypertension are lacking. Without a standardized approach, physicians treat asymptomatic hypertension utilizing inconsistent strategies including intravenous antihypertensives (IVAHs). The use of IVAHs has shown to have adverse effects that are associated with increased morbidity and mortality as well as longer length of stay.

**AIM:** The aim of this project is to standardize the approach to inpatient asymptomatic hypertension and minimize use of IVAHs by 25% in 6 months.

**Method:** Using the IHI Model, a quality improvement project was initiated. Root cause analysis was done and revealed a lack of standard approach to managing asymptomatic hypertension amongst hospital physicians and residents. PDSA cycle was used to test change. For PDSA 1, a standardized approach algorithm was created, and residents were educated about criteria for assessing symptoms, any contributing factors/secondary hypertension, and if they had a history of hypertension/home meds. For PDSA 2, we expanded education for the nursing staff and provided them with the treatment flowchart. We also included the pharmacy department in our initiative, to form a multidisciplinary approach. For PDSA 3, we included Nurse Practitioners in our education and provided them with our standardized algorithm as well.

**Results:** Prior to PDSA 1, 24 charts were analyzed and over 58% of patients (14/24) had received inappropriate IVAHs. Post-intervention, 31 charts were analyzed and showed a 32.5% reduction in IVAH use. Prior to PDSA 2, another 36 patient charts were analyzed and 25% of patients (9/12) had received inappropriate IVAHs. Post-intervention, 21 charts were analyzed and showed an 11% reduction in IVAH use. For PDSA 3, 19 charts were analyzed pre-intervention and 53% of patients had received inappropriate IVAHs. Following intervention, 12 charts were analyzed, and 42% of patients had received inappropriate IVAHs. Our post-intervention analysis is ongoing, and although data suggests a promising decrease in the use of inappropriate IVAHs, it has been inconsistent, and sustainability will become a challenge.

**Conclusion/Next Steps:** Our project led to the implementation of a standardized algorithm, thus decreasing inappropriate use of IVAHs and minimizing cost. Management of asymptomatic hypertension in the hospital begins with addressing contributing factors such as pain or anxiety, then reviewing held home medication. With the implementation of a standardized algorithm, we found some success in the reduction of use of IVAHs. Working with a multidisciplinary team led to a greater impact of our efforts. Our next steps include implementing the algorithm in the EMR system to have an alert system to clarify when IVAHs are indicated and when other options should be reviewed. We plan to continue our multidisciplinary efforts with the pharmacy and EMR staff along with new admission PA/NP service. Re-education throughout the year will also be implemented to improve sustainability.
Atypical Case of Marchiafava Bignami Disease without Risk Factors

Educational objectives

> Recognizing and understanding the etiology, pathogenesis and manifestations of Marchiafava Bignami Disease

> Identify and evaluate Marchiafava Bignami Disease in patients without typical risk factors

Introduction:

Poorly controlled diabetes can present with a wide range of clinical manifestations ranging from hypoglycemia to seizures and coma. In this vignette, we investigate hypoglycemia in a 56 year old male, who presented in a comatose state, resulting in the discovery of a rare neurological brain condition.

Case Presentation:

A 56-year-old man with an extensive medical history of uncontrolled Type 2 Diabetes Mellitus, Chronic kidney disease, Congestive heart failure, Coronary artery disease, Hypertension and Obesity with BMI of 43 kg/m2 presented due to unresponsiveness with a serum glucose of 37 mg/dl. In the ER, the patient received 1 amp of dextrose, was intubated and transferred to intensive care. On examination, the patient had a Glasgow Coma Score of six but intact corneal and gag reflex. Labs were remarkable for an hemoglobin A1c 7.0%, elevated C-peptide 14.3 ng/ml (1.1-4.4) and insulin 4.0 uIU/mL (< 25) with corresponding blood glucose of 141 mg/dl. Patient’s family confirmed the patient’s overall medication noncompliance but no history of alcohol intake or malnutrition. Of note, the patient was consistent with his sulfonylurea medication which is known to cause hypoglycemia in patients with renal failure if not used with caution. Extensive testing including Computed Tomography of the Head, Computed Tomography of the Chest and Abdomen, Cerebrospinal Fluid analysis for infections and malignancies yielded no significant results. Magnetic Resonance Imaging of the Brain revealed diffusion restriction/acute ischemic infarct involving splenium of corpus callosum, confirming the diagnosis of Marchiafava-Bignami Disease (MBD). Despite extensive treatment with IV dextrose infusion, octreotide and thiamine, the patient was found to be persistently hypoglycemic and unresponsive. The patient was eventually transferred to hospice care and expired.

Discussion:

MBD is a rare and fatal neurological disorder characterized by demyelination of the corpus callosum most commonly in the setting of poor nutrition or chronic alcoholism. T2 weighted . Magnetic Resonance Imaging of the Brain is the most sensitive diagnostic method used to confirm the diagnosis. This case highlights the importance of considering atypical risk factors such as hypoglycemia in the differential diagnosis of MBD in patients without alcohol use or malnutrition. It has been studied that fluctuations in serum osmolality in the setting of uncontrolled diabetes mellitus can increase oxidative stress and lipid peroxidation in the brain, thus leading to structural brain abnormalities as seen in this case. MBD has poor prognosis unless treated early in the course of disease.
Medical Student Poster # 25  
Category: Research

Medical School: University of Michigan
Presenter: Grace Herron
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Effectiveness of DOAC prescribing oversight by research or clinical teams at improving evidenced-based DOAC prescribing

While direct oral anticoagulants (DOACs) may be viewed as simpler to manage than warfarin, they present their own unique management challenges resulting in frequent off-label dosing. It is unknown to what extent off-label dosing occurs when a patient is started on a DOAC versus later in their treatment. We evaluated data from the Michigan Anticoagulation Quality Improvement Initiative (MAQI2) registry from 2018 – 2022 on the number of “alerts” that are generated in response to dosing that deviates from evidence-based guidelines. Among a sample of 1,261 – 1,563 annual patients in the MAQI2 registry, off-label dosing was relatively common. There were on average 147 dosing alerts per year. Dosing alerts occurred more frequently during a follow-up and accounted for 69% of alerts on average versus at baseline when the DOAC was first prescribed (31% of alerts on average). After initial review, an average of ~18% of alerts resulted in contact to the prescriber. When the prescriber was contacted, it led to an intervention ~75% of the time. The most common intervention was a change in DOAC dosing. This study demonstrates the benefit of DOAC prescribing oversight using a registry-based intervention to monitor for off-label dosing for the entirety of the time period a patient is prescribed DOAC as deviations in evidence-based prescribing occur frequently during the follow-up period.
Improving Treatment of Obstructive Sleep Apnea (OSA) in Geriatric Patients

Introduction:

Obstructive Sleep Apnea (OSA) is increasingly prevalent in older adults. Untreated OSA is associated with poor blood pressure control and cardiovascular risk. Despite demonstrated benefits of CPAP/BiPAP therapy, adherence remains poor across the age spectrum. Patients may not adhere to prescribed CPAP/BiPAP therapy for a variety of reasons, including improper mask fitting, dry nose/mouth, and nasal congestion. We sought to increase the proportion of (1) discussion of OSA therapy between older patients with comorbid OSA and hypertension and their primary care providers (PCPs) and (2) referral to sleep medicine specialists for follow-up.

Methods:

In May 2023, we performed a chart review to identify all geriatric patients with comorbid hypertension and untreated OSA in a large academic medical center geriatric clinic. We then split our cohort into a pre-post comparison of patients who had a visit with their PCP from 1/1/2023-4/29/2023 (baseline visits) compared to patients with a future PCP appointment scheduled from 6/1/23-11/30/23 (post-intervention visits). For intervention patients, we emailed PCPs with a brief reminder about untreated OSA in advance of visits and placed the following flag in the PCP schedules for the intervention visit: “Untreated OSA. Discuss Sleep Medicine Referral.” We then reviewed PCP notes and orders for two outcomes: (1) documented discussion of OSA therapy, and (2) referrals to sleep medicine specialists for follow-up. We compared the pre- and post-intervention patients for the outcome using chi-squared tests. Lastly, we followed the subsequent care resulting from the OSA treatment discussions in the intervention group.

Results:

Of 178 baseline visit patients, 88 (49%) had not discussed OSA therapy with their PCP. In the post-intervention visits, 53/83 (64%) of patients discussed OSA therapy with their PCP, a 13% relative improvement (p=0.044). In 21/53 (40%) of the intervention patients, a referral to a sleep medicine specialist was placed; of these 21, 3 patients (14.3%) have attended their sleep medicine appointment as of 1/20/23, and 2 have had CPAP/BiPAP titrations and reported using their CPAP/BiPAP nightly although have not subsequently seen their PCP to assess blood pressure control. 7/12 (58%) patients previously not using their home CPAP/BiPAP reported agreeing to OSA therapy following discussion with their PCP, although all declined a visit with a sleep medicine specialist.

Conclusion:

In this quality improvement study, the use of flags in PCP schedules was effective at improving the rate of discussion regarding OSA therapy for geriatric patients with comorbid hypertension. We plan to conduct future investigation into the relationship between sleep medicine specialist follow-up for OSA therapy, blood pressure control, and antihypertensive dosing regimens.
Portal Biliopathy is an Underrecognized Condition Resulting from Non-Cirrhotic Portal Hypertension

Portal Biliopathy (PB) is defined as the presence of abnormalities in the biliary tree in patients with non-cirrhotic/non-neoplastic extrahepatic portal vein obstruction. It occurs as a result of chronic occlusion of the portal vein and/or superior mesenteric vein which precipitates peribiliary collateral vessels to tether and compress the common bile duct (CBD) near the pancreatic head.

Our patient is a 67 y/o female with a PMHx significant for polycythemia vera (PV) complicated by chronic portal vein thrombosis (PVT), noncirrhotic portal hypertension with ascites esophageal varices, splenomegaly s/p splenectomy, among others. She was routinely seen by a hepatologist for management and noted to have rising liver chemistries. Notably, her ALP was 811; this led to her being referred for magnetic resonance cholangiopancreatography (MRCP) which noted a CBD stricture as well as stones in the proximal CBD and the cystic duct. She was then referred for an endoscopic retrograde cholangiopancreatography (ERCP) which was notable for choledocholithiasis and a tight lengthy CBD stricture. Stones were removed and a stent was placed. As the etiology of her stricture remained in question, follow-up ERCP with biopsies were negative for malignancy. The stricture was treated with dilation and stenting. A subsequent ERCP was performed: the CBD stricture was dilated again, however this was complicated by life-threatening hemobilia. A fully covered metal stent was placed which achieved hemostasis. CT angiography was obtained and showed no evidence of an arterial source for bleeding supporting portal biliopathy as the source of hemobilia. At her follow-up ERCP, her covered metal stent was removed successfully without further bleeding and her biliary stricture had resolved.

PVT, resulting from non-cirrhotic portal hypertension, is a risk factor for PB. The recurrent and frequent biliary strictures are a result of the bile duct and portal vein’s relative location immediately adjacent to each other. Gallstones can result from RBC breakdown in the setting of PV and hypersplenism. In addition, bile duct stones can form in patients with chronic biliary strictures. If there is portal vein occlusion, collateralization can cause peribiliary cavernous transformation of the portal vein. This transformation can stricture the biliary tree by extrinsic compression or result from ischemic damage secondary to altered biliary vasculature.

While PB seen with MRCP can be asymptomatic, about 5-38% are symptomatic presenting similarly to our patient with abdominal pain, elevated LFTs, and choledocholithiasis. The initial focus in the treatment of PB is to reduce portal hypertension which can be achieved by placement of a transjugular intrahepatic portosystemic shunt (TIPS) or surgical portosystemic shunt. The biliary changes may then resolve if due to extrinsic compression from portal collaterals. If biliary changes are due to ischemia, patients may require multiple endoscopic procedures to dilate strictures, placement of stents to aid in drainage, and removal of bile duct calculi. While portal biliopathy is an uncommon condition, it should be included in the differential diagnosis of patients with non-cirrhotic portal hypertension from portal vein thrombosis and biliary strictures. Recognition of PB will reduce morbidity and improve treatment approaches.
Medical Student Poster # 28

Category: Clinical Vignette

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Case Report of Lepromatous Leprosy in Michigan, USA.

Introduction: We report a case of lepromatous leprosy in Michigan, USA, in an individual with no known traditional risk factors. Despite increased reports of leprosy (Hansen’s Disease) in southern regions of the United States, to our knowledge, there have been no cases of leprosy in Michigan as recorded by the National Hansen’s Disease program in the past 7 years. Hansen’s disease is caused by Mycobacterium leprae and M. lepromatosis frequently contracted by patients after travel to endemic areas or present in foreign-born individuals after a long incubation period. Presentation of disease is remarkably heterogeneous ranging from skin to ocular to gastrointestinal involvement. The lepromatous form of leprosy (LL) primarily presents with multiple maculopapular skin lesions occurring in a symmetrical distribution. However, as the following case demonstrates, due to the considerable overlap of symptoms the clinical presentation can vary. Potential variances in disease presentation could arise from differences in medical history. As we demonstrate in this case, low clinical suspicion can delay timely diagnosis and appropriate management. We present our approach for developing an appropriate differential and recent advances in LL management.

Case History: A 73-year-old male, born and raised in the Upper Peninsula of Michigan, initially presented to the Dermatology Clinic in 2018 with a generalized rash. An initial biopsy revealed non-necrotizing granulomas. Due to overlapping non-specific pulmonary and neural symptoms, the patient was treated for presumed sarcoidosis with methotrexate, phototherapy, Remicaide infusions, and infliximab. The patient experienced another eruption of hypopigmented skin lesions a year after the first biopsy which prompted a return to Dermatology. The only travel endorsed by the patient was a remote history to Mexico and Jamaica restricted largely to the beaches. He did not report any known contact with armadillos or persons infected with leprosy. The case was discussed with Infectious Disease and a punch biopsy of the lesions was performed. Pathologic findings were positive for acid-fast bacilli and ulcerated granulomatous tissue reaction consistent with lepromatous leprosy.

Discussion: This report highlights the first case of lepromatous leprosy recorded in Michigan with an anomalous presentation for a patient with no known exposure or historical risk factors. While leprosy or Hansen’s Disease has been reported in the United States at a steadily increasing rate of approximately 200 cases per year since 2021, these cases have largely aggregated in the southern regions. We present this case to highlight the potential of transmission in Michigan and to prompt clinical suspicion for ambiguous presentations in the mid-western states. We also discuss advances in treatment options and management strategies. This information reminds healthcare providers that leprosy/HD may have re-emerged as an insidious albeit rare public health concern, and that a careful surveillance program remains necessary, even in non-endemic areas.
Small Cell Carcinoma Originating in the Gallbladder

A 65-year-old female presented to the emergency department with a 2-week history of right upper quadrant pain radiating to the back. Labs were significant for elevated AST of 104 and lipase of 540. ALT and bilirubin were within normal limits. CT abdomen and pelvis showed a 1.9 cm gallbladder mass, mildly dilated common bile duct, and an enlarged porta-hepatic lymph node measuring 1.5 cm. Right upper quadrant ultrasound demonstrated a heterogenous 3 cm mass in the gallbladder fundus suspicious for neoplasm. Tumor markers including CAE, CA19-9 and alpha fetoprotein were all negative. The patient was admitted for further workup of the gallbladder mass.

MRCP showed an irregular gallbladder mass and wall thickening measuring 3.1 cm located in the fundus with invasion into the hepatic parenchyma (segment 4b) as well as necrotic porta hepatic lymph nodes concerning for metastasis. CT chest was completed for staging which showed no evidence of metastatic disease. Endoscopic ultrasound was performed to further evaluate the mass at which time the enlarged porta hepatic lymph node was biopsied via fine needle aspiration. Cytology results from the lymph node biopsy were consistent with metastatic small cell carcinoma. A multidisciplinary tumor board discussion determined the patient was not a candidate for surgical resection. The patient was subsequently admitted to begin chemotherapy with carboplatin and etoposide.

A PET scan was done for further staging which showed no signs of metastatic disease. The patient continues to receive chemotherapy with carboplatin and etoposide along with radiation therapy.

Small cell carcinoma of the gallbladder is a rare malignancy, accounting for only 0.2% of all neuroendocrine tumors.1 Prognosis is typically poor, with a median survival of 8-13 months after diagnosis.1 Treatment may include a combination of chemotherapy, radiotherapy, and surgical resection.

References:

Restarting Warfarin Therapy with No Bridging Agent after an Acute Ischemic Stroke in a Patient with a Mechanical Mitral Valve

Introduction:
Patients with mechanical mitral valves require warfarin for anticoagulation due to higher thromboembolic risk. While initiating or restarting warfarin, bridging therapy with heparin is usually recommended to prevent paradoxical hypercoagulability. We present a case of an acute stroke patient with a history of heparin-induced thrombocytopenia (HIT) who was consequently restarted on warfarin with no bridging agent.

Case: A 27-year-old male with a history of hypertrophic cardiomyopathy, mechanical mitral valve on warfarin, hyperlipidemia, and HIT, presented with a witnessed event of acute weakness and dysarthria while playing drums at church. Exam showed right upper and lower extremity weakness, right-sided facial weakness, and dysarthria. Systolic blood pressure was 251. Initial NIHSS was 10, and a Code Stroke was called; labs showed a subtherapeutic INR of 1.38 and blood pressure improved with nicardipine. CT brain perfusion demonstrated hypoperfusion in the left posterior frontal parietal region and intravenous TNK was given. However, the patient’s symptoms worsened. CT head showed no acute changes. Neuroendovascular proceeded with intra-arterial infusion of tPA to the left MCA. Repeat head CT 24 hrs post TNK showed subtle hypodensity in the left parietal lobe most consistent with subacute infarct. Echocardiogram showed EF of 60-65% with a grade II LV diastolic impairment and a mildly dilated left atrium, no valvular thrombus. The patient reported a recent increase in salad intake and self-adjustment of his warfarin dosages before monthly INR checks.

The patient was placed on permissive hypertension protocol of up to 180 systolic for 48 hrs, and repeat CT head showed no hemorrhagic conversion. Cardiology recommended against bridging with argatroban as both argatroban and warfarin increase the prothrombin time. Warfarin therapy was started without bridging. Pharmacy recommended 10 mg of warfarin for two days (with no heparin bridge) until Cardiology cleared the patient for discharge. Pharmacy recommended 7.5 mg warfarin for the next two days until outpatient INR follow-up. The patient was discharged with no sequelae of his embolic event.

Discussion:
The protocol for restarting anticoagulation after ischemic stroke is individualized based on inciting factors and risk for hemorrhagic conversion. In this case, the size of the infarct, risk of thromboembolism with a mitral MHV, and inability to bridge with heparin due to history of HIT were considered. Multiple meta-analyses argue that the common practice of bridging warfarin with heparin is not based on true evidence of benefit to the patient in reducing thromboembolism risk, yet bridging therapy is associated with an observable risk of increased bleeding1. Though the evidence is even more limited for high-risk thromboembolism subgroups such as our patient, one study on acute ischemic stroke patients with MHV showed that the rate of symptomatic intracranial bleeding in heparin bridging patients was similar to that of non-bridging patients2.

Conclusion: Evidence supporting bridging therapy in acute ischemic stroke patients with MHV is limited and should be considered on an individualized basis.

Acknowledgement: Patient requested the authors express his acknowledgement to God and his medical team for his recovery.
Acute colonic pseudo-obstruction (Ogilvie’s syndrome) resolved with methylnaltrexone

Introduction: Abdominal distention has a wide range of differential diagnoses. Ogilvie’s syndrome is characterized by acute distention of the colon in the absence of mechanical obstruction. We report a refractory case of abdominal distention due to Ogilvie’s syndrome which improved with administration of methylnaltrexone.

Case: A 55-year-old male with a history of chronic back pain managed by daily NSAID use and previous DVTs on warfarin, presented with one day of hematochezia and hematemesis, resulting in multiple episodes of syncope. Initial trauma workup revealed an acute compression fracture of T12. An EGD revealed ulcerative esophagitis and ulcerative gastritis. The patient then underwent a kyphoplasty due to continued thoracic back pain requiring nearly around-the-clock opiates for 48 hours. Following the procedure, the patient complained of abdominal distension, absence of bowel movements, and flatus. His abdomen was tympanic, but bowel sounds were auscultated. Conservative treatment with a gradually intensifying bowel regimen was initiated. Subsequent abdominal x-rays showed marked dilation of the large colon to 11 cm. A Gastrografin contrast enema showed no mechanical obstruction, and lactic acid was within normal limits, confirming Ogilvie’s syndrome. On the sixth day following the kyphoplasty procedure, the patient was transferred to the SICU for Neostigmine treatment. However, the patient continued to experience distension without adequate bowel movements. A literature search revealed that methylnaltrexone had been utilized in a few case reports for pseudo-obstructions in patients with opiate exposure, but it is contraindicated in mechanical obstructions.¹ The patient received subcutaneous administration of 12 mg of methylnaltrexone, resulting in a significant 2 cm decrease in colon dilation observed in the subsequent daily abdomen X-ray, and positive flatus and bowel movements. A repeat dose of 12 mg was given, and the patient was discharged with the abdominal distension resolving.

Discussion: Ogilvie Syndrome is an unusual occurrence, and while there are a variety of theories, no clear pathophysiology has been agreed upon. Risk factors include advanced age with multiple chronic diseases and poor underlying functional status or immobility. Additionally, while all surgeries increase the risk of Ogilvie, major orthopedic and obstetric procedures are the highest risk for the development of Ogilvie.² There is common agreement that there is an impairment of the colonic motor system, with dysfunction or imbalance of the autonomic nervous system being the most likely cause. Therefore, causes that affect the autonomic system such as spinal trauma or corrective spinal surgery with posterior instrumentation, spinal anesthesia, and pharmacological agents have been identified as risks.³ Given the rapid response to methylnaltrexone, if this therapy was administered prior to neostigmine, we likely could have avoided a transfer to higher a level of care for the neostigmine administration.

Conclusion: In the setting of abdominal distension, Ogilvie’s syndrome should be considered as a differential diagnosis. In the absence of obstruction, the administration of methylnaltrexone is safe and should be considered in patients with opiate exposure, acute or chronic, and recalcitrant Ogilvie’s syndrome. Further studies are warranted to investigate the role of methylnaltrexone in the treatment of Ogilvie’s syndrome.
Amniotic Bladder Therapy for Treating Interstitial Cystitis/Bladder Pain Syndrome: a 6 Month Follow Up

Introduction: Interstitial Cystitis/Bladder Pain Syndrome (IC/BPS) is a debilitating condition marked by chronic pelvic pain and concurrent lower urinary tract symptoms. Our prior research established the efficacy of Amniotic Bladder Therapy (ABT) in offering symptomatic relief to refractory IC/BPS patients for a duration of up to 3 months. This study aims to delve into the durability of ABT, examining its effects over a more extended period, specifically up to 6 months.

Materials and Methods: Consecutive IC/BPS patients participated in the study, receiving intra-detrusor injections of 100mg micronized amniotic membrane. Comprehensive clinical evaluations were complemented by patient-reported outcome measures, encompassing the Interstitial Cystitis Symptom Index (ICSI), Interstitial Cystitis Problem Index (ICPI), Bladder Pain/Interstitial Cystitis Symptom Score (BPIC-SS), Overactive Bladder Assessment Tool, and SF-12 Health Survey.

Results: The study involved twenty-five consecutive recalcitrant IC/BPS patients, with an average age of 47.4 ± 14.4 years (29-67 years). After ABT, IC/BPS symptoms exhibited a gradual improvement up to 3 months in all patients. There was a substantial average improvement in ICSI, ICPI, and BPIC-SS scores of 72.8%, 71.9%, and 66.6%, respectively, at the 3-month mark. At the 4-month interval, seven patients experienced a resurgence of symptoms, prompting additional ABT. This secondary intervention resulted in significant improvements in IC/BPS symptoms after 2, 4, and 8 weeks. For the eighteen patients receiving a single injection, IC/BPS symptoms remained significantly reduced at 5 and 6 months compared to baseline. This persistence suggests a durable effect, as evidenced by sustained improvements in ICSI, ICPI, BPIC-SS, and OAB questionnaire scores.

Conclusions: Amniotic Bladder Therapy (ABT) emerges as a promising intervention, providing enduring relief from pain and lower urinary tract symptoms for up to 6 months post-treatment in certain refractory IC/BPS patients. These findings underscore the potential of ABT as a durable and effective therapeutic option for individuals with IC/BPS unresponsive to conventional treatments.
Peripheral Ulcerative Keratitis associated with Corneal Melt, secondary to Hidradenitis Suppurativa

Introduction: Hidradenitis Suppurativa (HS) is a chronic skin condition due to blocked and infected hair follicles, causing painful draining skin abscesses in the groin, breasts, and axilla. HS is a relatively common disease in the United States; however, ophthalmologic manifestations are much less common. Peripheral ulcerative keratitis (PUK) refers to thinning of the cornea of the eye due to inflammation. If untreated, PUK can eventually cause corneal melt, or perforation of the cornea, and eventual blindness. A few cases of PUK associated with HS have been reported, and of these, they coincide with severely advanced dermatologic disease.

Case Description: A 38-year-old female with a past medical history of controlled HS presented to the emergency department with intermittent episodes of bilateral painful loss of vision, eye swelling, eye redness, and associated photophobia over the past four months. The patient reported that she had a previous diagnosis of PUK but had difficulty following up with her outpatient ophthalmologist and nonadherence with her prescribed steroid regimen due to difficulty with making it to her appointments. Physical exam was significant for eye pain on extraocular movement, injection of conjunctiva, 90% loss of visual fields, and photophobia. Slit lamp photography showed severe bilateral corneal thinning with active perforation in the right eye. Lab workup was only significant for elevated ESR. The patient was diagnosed with corneal melt secondary to HS. Due to severity of disease and difficulty with outpatient follow up, the patient was admitted for three days of intravenous methylprednisolone, oral doxycycline for its anti-collagenase effects, and a topical antibiotic regimen including moxifloxacin drops and clindamycin drops. Prophylaxis included eye protection with a shield at all times. The patient had regular follow up with ophthalmology, where she was noted to have continual improvement in symptoms and corneal edema of both eyes and patient is planned to escalate to infliximab therapy for HS and PUK.

Discussion: This case report on corneal melt illustrates how HS is a systemic chronic condition and that its symptoms extend further than just the characteristic dermatologic lesions. Other HS ophthalmologic complications include peripheral ulcerative keratitis, uveitis, and scleritis. Steroids are standard medical management for flares, but often a transition to immunosuppressive therapy, such as infliximab or TNF-a, is required for remission. This case report also emphasizes the importance of delving deep into patient history and knowing all their chronic conditions to help diagnose the current disease process. Further discussing this patient’s history helped the physicians understand that even though her HS lesions had mostly resolved, this manifestation of corneal melt on her eyes was related to her HS. Other case reports cover patients with PUK associated with severe dermatological manifestations of HS, however, this patient had minimal dermatological disease burden. Although corneal melt is a rare complication of HS, it is important to recognize its symptoms, begin immediate steroid and antibiotic treatment, and educate patients on compliance to steroids or other immunosuppressive therapy to prevent irreversible blindness.
An Rare Case of Type 1 Gaucher Disease Associated with Dilated Cardiomyopathy

Introduction:
Gaucher disease is an autosomal recessive lysosomal disorder that leads to a deficiency of glucocerebrosidase. The resultant accumulation of glucocerebroside most commonly affects macrophage lineage cells in the liver, spleen, and bone marrow. Variants of Gaucher disease can also lead to CNS involvement. While type 1, or non-neuropathic Gaucher disease, can grow to involve other organs such as the lungs, even fewer cases of cardiac involvement have been reported. We report a rare case of infiltrative cardiomyopathy with associated heart failure in a patient with Type 1 Gaucher disease.

Case Presentation:
A 26-year-old Iraqi male presented to clinic for Gaucher disease testing after an extensive family history of the disease in four siblings. Clinically, he reported a history of frequent nosebleeds since childhood and easy bruising with minor trauma. He also reported increased occurrences of joint pain in his ankles, shoulder, and knees. On physical exam, the spleen was palpated 1-2 finger-breadths below the costal margin. Labs showed mild iron deficiency anemia and thrombocytopenia, with a platelet level of 64. Bone marrow biopsy confirmed Gaucher disease with the presence of Gaucher cells. An abdominal ultrasound revealed splenomegaly with the spleen measuring 20cm in sagittal dimension. He was started on Velaglucerase alpha enzyme replacement therapy from 2016 to 2017. Due to various social challenges, periodic treatment delays occurred between 2017 to 2021. Velaglucerase was re-initiated in 2022.

Seven years after diagnosis, the patient presented to the clinic stating that he had gone abroad for an elective surgery when abnormalities were noted on pre-operative cardiac evaluation. He had also been experiencing shortness of breath on exertion to moderate activity for about a year prior. Cardiac MRI revealed severe left ventricle enlargement with global hypokinesis and a left ventricular ejection fraction (EF) of 28%. The right ventricular function was also reduced with EF of 33%. There was extensive, patchy, predominantly mid-myocardial late gadolinium enhancement most consistent with an infiltrative cardiomyopathy. Of note, repeat serum protein electrophoresis and immunofixation revealed no abnormalities. He was diagnosed with AHA Stage C, Systolic Congestive Heart Failure with Dilated Cardiomyopathy. The patient’s non-ischemic cardiomyopathy was presumed to be a result of Gaucher disease. He was started on goal directed therapy for heart failure along with Velaglucerase.

Discussion:
Cardiac involvement in Gaucher disease is more commonly found as valvular abnormalities associated with the rare Type 3c variant. Dilated cardiomyopathy progressing to heart failure can be caused by various factors, including genetic mutations, infections, autoimmune disease, and metabolic causes; however, its association with infiltrative cardiomyopathy presumed to be from Gaucher disease has not been reported. A common cause of infiltrative cardiomyopathy is amyloidosis, but this is often seen with restrictive cardiomyopathy. While a confirmative cardiac biopsy was not performed to differentiate Gaucher’s from other causes of infiltrative cardiomyopathy, Gaucher disease cannot be excluded as a direct cause. This case describes the importance of cardiac assessment and monitoring in Type 1 Gaucher disease due to the complexities of multi-organ involvement.
Understanding Vaccine Hesitancy and Barriers to Vaccine Access in Latin American Communities of Metro Detroit

INTRODUCTION
The Latinx population in the United States is growing, yet insurance coverage of this group is not growing proportionally. Data estimates that as many as 25% of Latinx individuals are uninsured, compared to approximately 8.6% of non-Hispanic whites. Gaps in insurance coverage often lead to poorer health outcomes and may affect accessibility of essential health behaviors, including vaccination. Additional factors affecting vaccination in the Latinx community may include vaccine hesitancy, which has grown in the post-pandemic era. The objective of our study was to understand vaccine hesitancy and barriers to vaccine access in the local Latinx community through the lens of the influenza vaccine.

METHODS
Our study assessed the unique knowledge, barriers to access, and levels of trust regarding the influenza vaccination among local Latinx communities. To investigate these variables, we developed a 5-part survey that also gathered information regarding respondent demographics and previous vaccination history. The survey consisted of 22 multiple-choice questions, followed by a final optional, open-ended question regarding reasons for influenza vaccine refusal. The survey was administered at a local community center to voluntary participants in both English and Spanish.

RESULTS
A total of 25 individuals responded to the survey, and 23 (92%) completed it. The target demographic was adequately represented: 23 (92%) respondents identified as Hispanic/Latin, and 19 (76%) stated their preferred language was Spanish.

Overall, the findings show that local Latinx communities have sufficient knowledge, access, and trust regarding the influenza vaccination. Additionally, most respondents had extensive history of previous vaccination. For instance, 18 (72%) respondents stated that they had either gotten the flu vaccine “a few times” or “every year” during the past 5 years. 22 (88%) respondents stated that they believe vaccines are necessary, and 20 (80%) stated they would get a vaccine if their doctor recommended it. Individuals understood that if they are healthy, they still need the flu vaccine (16, 67%) and did not feel they needed insurance to get the vaccine (15, 65%). 14 (61%) agreed that the flu vaccine “prevents people from getting sick” and 15 (65%) also agreed that “government influenza vaccination programs are trustworthy.”

Notably, the most reported barriers to vaccination included “lack of time” (9, 39%) and “other” (8, 35%), while just 3 (13%) stated “lack of insurance” was a barrier and 0 cited cost as a limiting factor. Also, no significant reasons for vaccine refusal were reported.

In summary, these findings reflected a strong vaccination history, knowledge, and trust regarding the influenza vaccination in local Latinx communities, with few notable barriers to access reported.

CONCLUSION
Our study sought to describe vaccine hesitancy and barriers to vaccine access in local Latinx communities. The findings demonstrate that Latinx individuals generally understand and trust the flu vaccine, and have sufficient access to vaccination. However, Latinx patients do experience time-related and other unspecified barriers to vaccination, prompting need for further investigation. Additionally, many individuals not surveyed may still feel reluctance regarding vaccination, indicating need for specific efforts to reach these individuals and explore their reasons for vaccination hesitance.
Hemolytic Anemia and Pancytopenia in the Setting of Vitamin B12 Deficiency

Vitamin B12 deficiency can cause ineffective erythropoiesis and intramedullary hemolysis, manifesting as life-threatening hematological abnormalities, including a rare combination of pancytopenia and hemolytic anemia. In the case below, we describe a female with a more severe presentation than previously reported of hemolytic anemia and pancytopenia from vitamin B12 deficiency attributed to pernicious anemia.

A 36-year-old female with a past medical history of thyroid disease presented with confusion after a one-week history of emesis, decreased oral intake, weakness, and shortness of breath. The patient had no significant social history. She denied recent sick contacts or travel and new medication or herbal supplements.

Physical examination was only remarkable for a heart rate of 112 beats per minute, a respiratory rate of 33 breaths per minute with 100% oxygen saturation on room air, and altered mental status requiring endotracheal intubation.

The patient’s initial labs included hemoglobin of 1.6 g/dL with mean corpuscular volume of 86.9 fl, platelets of 15 K/uL, white blood cells of 7.5 K/uL, lactate of 22.8 mmol/L, creatinine of 1.79 mg/dL, alanine transaminase of 3263 IU/L, aspartate transaminase of 1687 IU/L, total bilirubin of 4.7 mg/dL, alkaline phosphatase of 71 IU/L, HCO3- of 6.7 mmol/L, anion gap of 32.3, ammonia of 112 umol/L, thyroid stimulating hormone of 6.7 ulU/mL, and free thyroxine of 0.74 ng/dL. Urinalysis, urinary drug screen, acute viral hepatitis screen, acute hepatitis panel, alcohol level, urinary pregnancy, and COVID-19 and influenza swabs were negative, and salicylate level was unremarkable.

Computed tomography scan of the head revealed no acute intracranial abnormalities.

Hepatology was consulted for concern of acute liver failure with extensive workup for clear etiologies of liver failure all being negative, including acetaminophen levels, viral and autoimmune hepatitis, Epstein-Barr virus, Cytomegalovirus, Herpes simplex virus, and Wilson’s disease. Ultrasound of liver with dopplers was also negative.

Hematology was consulted to evaluate pancytopenia and rule out iron deficiency anemia, microangiopathic hemolytic anemia, thrombotic thrombocytopenic purpura, autoimmune hemolytic anemia, hemolytic uremic syndrome, leptospirosis, and Wilson’s disease. Peripheral blood smear revealed hypersegmented neutrophils, nucleated and macrocytic red blood cells, dacryocytes, and no schistocytes. Coombs test and workup for rheumatologic disease were negative. Vitamin B12 was noted to be 121 pg/mL, and folic acid level was 11.7 ng/mL. Intrinsic factor and gastric parietal cell antibodies were positive, indicating pernicious anemia. The patient’s blood counts, liver function tests, and coagulopathy improved after resuscitation, initiation of vitamin B12, and transfusion of blood products. She was extubated with improved mentation. Bone marrow biopsy was deferred by hematology due to establishing vitamin B12 deficiency and response to treatment.

The patient received vitamin B12 injections and was discharged with instructions to follow up with hematology.

Our case highlights multiorgan failure and critical illness driven by vitamin B12 deficiency. While the constellation of hemolysis and multiorgan failure typically brings forward thrombotic microangiopathies (TMA) as the leading differential, it is vital to recognize that vitamin B12 deficiency in its most severe form can present similarly catastrophically. Despite its severity, prompt recognition and treatment led to timely resolution.
Battling Biases and Brain Fog: Unmasking Autoimmune Encephalitis Amidst Extensive Drug Use History

Autoimmune encephalitis (AE) is a neurological disorder where the body's immune system mistakenly attacks the nervous system. It leads to a wide range of neuropsychiatric symptoms, including epilepsy and limbic encephalitis. This case report focuses on the second most common subtype of AE, associated with glutamic acid decarboxylase 65 (GAD-65) antibodies. GAD-65 antibodies, usually found in various autoimmune conditions, pose a unique diagnostic challenge, particularly in patients with multiple comorbidities.

The patient is a 60-year-old black male with a complex medical history including hypertension, diabetes, polysubstance use (cocaine and heroin), and extensive methadone use. He presented with altered mental status, cognitive decline, and neurological symptoms, leading to multiple admissions across different hospitals (A, B, C). Initial misdiagnoses at hospitals A and B included vascular dementia and seizure disorders. Patient presented to hospital C for progressive encephalopathy for 4 months. On physical exam, patient was alert and oriented to location. Patient followed simple commands but was otherwise confused, selectively mute, and uncooperative. Patient had exaggerated startle response and myoclonic jerks with intact consciousness. Lumbar puncture at hospital B with routine CSF studies demonstrated elevated protein at 188. Comparative review of 2 MRI-Head/Brain obtained 3 months apart from hospital A and B demonstrated rapidly progressive brain atrophy. After ruling out infectious and malignancy concerns, patient’s initial differential was concerning for Creutzfeldt-Jakob Disease, rapidly progressive Alzheimer’s disease or vascular dementia, seizure disorder, and autoimmune or paraneoplastic encephalitis. Repeat LP was sent for CJD, autoimmune, and paraneoplastic studies.

Patient was empirically treated with a 5-day course of IVIG and IV methylprednisolone (IVMP). He experienced little improvement with IVIG but significant return to baseline with IVMP. CJD was ruled out with LP. The patient’s elevated anti-GAD antibody levels were notable, though the absence of corresponding elevation in the cerebrospinal fluid (CSF) added complexity to the diagnosis.

This case highlights the diagnostic and treatment complexities of AE, especially in the presence of multiple comorbidities and a history of substance use. Misdiagnosis is common in AE, as symptoms often overlap with other neurological conditions. However this misdiagnosis was particularly harmful given that AE was successfully treated with return to baseline, while vascular dementia would not offer the same prognosis. The patient’s documented polysubstance use history potentially introduced bias, limiting a comprehensive differential. The 20-year history on methadone raised concerns for overdose or withdrawal at hospitals A and B, leading to a delay in AE evaluation. At these hospitals, the patient’s family overheard discussions of providers attributing the deterioration to likely extensive drug use, which reveals the impact of biased perceptions.

Treatment with immunotherapies like IVIG and methylprednisolone showed varying efficacy, which is consistent with existing literature on AE management.

This case emphasizes the importance of considering AE in differential diagnoses of progressive neuropsychiatric disorders. The case illustrates the need for healthcare professionals to avoid biases, especially regarding substance use history, to ensure timely and accurate diagnosis and treatment, particularly in cases where overlooking alternative explanations can have profound consequences for patient outcomes.
Mucor Empyema in a Middle-Aged Female Patient with Hereditary Hemorrhagic Telangiectasia on Bevacizumab

Mucormycosis is a rare but clinically significant fungal infection that can infect numerous systems. Pulmonary mucormycosis in particular is rapidly progressive associated with hematologic malignancies. Other risk factors include immunocompromised status, diabetes mellitus, dexoferamine use, and iron overload. Mucormycosis is associated with high morbidity and mortality. Bevacizumab is a monoclonal antibody that inhibits vascular endothelial growth factor (VEGF) and is used in the treatment of hereditary hemorrhagic telangiectasia (HHT) due to its anti-angiogenic properties.

We present a case of a 55-year-old female with a history of HHT on bevacizumab, tranexamic acid, and aspirin, multiple arteriovenous (AV) malformations status post bilateral pulmonary vessel embolization, and anxiety who presented to the emergency department endorsing dyspnea and right-sided pleuritic chest pain. Vitally, the patient was afebrile but hypoxic to 83%. Initial laboratory workup was pertinent for a white blood cell count elevated to 12.5 K/mcL. Initial chest x ray (CXR) suggested a right sided pleural effusion and follow up computed tomography (CT) of the chest showed a loculated right pleural effusion with moderate focal opacity in the medial right middle lobe, suspicious for necrotizing pneumonia and infected bulla in the right upper lobe measuring 1.4 x 1.0 cm with an air-fluid level. Due to concern for empyema, thoracotomy and decortication was performed; cultures revealed Mucor species, and the patient was started on amphotericin and anidulafungin. Subsequently, the patient was noted to have a hard palate ulcer and a nasal biopsy revealed fungal species; CT sinus was pertinent for invasive fungal sinusitis without bony destruction. Further assessment revealed no neurological involvement, and a repeat CT sinus showed no progression, and accordingly the patient was discharged on 3 months of isavucanozole.

Pulmonary mucormycosis is a rare but clinically significant cause of Fungal pneumonia. While our patient does not have any of the known risk factors for mucor infection, she is on bevacizumab, which has been previously associated with invasive aspergillosis and the association between anti-angiogenic agents and invasive fungal infections has been postulated. The main pathogen causing mucormycosis are Rhizopus species, of which are found in about 70% of mucormycosis cases. While the most common presentation of mucormycosis is rhinocerebral, about 30% of mucormycosis cases are pulmonary in nature. It commonly presents as a rapidly progressive pneumonia; symptoms include fever, cough, and pleuritic chest pain. Biopsy reveals angioinvasion and tissue necrosis. Pulmonary mucormycosis most commonly involves the upper lobe. Radiographic findings include consolidation on chest x ray that is progressive. Cavitation is common in CT, being present in approximately 40% of pulmonary mucormycosis cases. Mortality is very high in pulmonary mucormycosis, estimated to be up to 87%. Management is both medical and surgical, with prompt tissue debridement as well as antifungal therapy with amphotericin B. More studies are needed to determine other risk factors for pulmonary mucormycosis, including its association with HHT, anti-VEGF therapy, autoimmune disease, and hospitalization. In the future, clinicians should be cognizant of risk factors of pulmonary mucormycosis, as early diagnosis and management are crucial in care for patients with this infection.
Entangled Pathways: Duodenal Obstruction Caused by Celiac Artery Dissection

Introduction:

Spontaneous celiac artery dissection represents a rare subtype of visceral arterial dissection. While it may be incidentally discovered in asymptomatic individuals, it typically manifests with acute, severe abdominal pain attributed to the dissection or presents with symptoms indicative of intestinal ischemia and malperfusion. Our case introduces a novel diagnosis involving extrinsic duodenal obstruction resulting from celiac artery dissection and hematoma formation.

Case Presentation:

A previously healthy 61-year-old man with no medical history presented with a subacute nausea, vomiting, and progressive constipation, leading to an inability to tolerate any food or drink. Upon presentation, his vital signs were within normal range, and abdominal tenderness was absent upon physical examination. Initial laboratory studies yielded unremarkable results. A CT scan of the abdomen and pelvis revealed a 6.8 x 3.6 cm loculated collection beneath the duodenum, causing external compression. Further evaluation through MRCP unveiled dilation and aneurysm of the celiac trunk, accompanied by a heterogeneous hemorrhagic mass enveloping and constricting the duodenum, resulting in obstruction. Esophagogastroduodenoscopy (EGD) identified a narrowed, erythematous portion of the duodenum just beyond the second segment, with all biopsies demonstrating benign pathology. Subsequent CTA exposed celiac artery dissection with preserved flow and a 4 cm pancreaticoduodenal artery pseudoaneurysm. Interventional radiology successfully performed embolization of the pancreaticoduodenal pseudoaneurysm using microcoils. The patient was then managed with a nasogastric tube and bowel rest. After several days of monitoring, the obstruction improved, allowing for discharge with a liquid diet.

Discussion:

The presentation of our patient with celiac artery dissection and the consequent pancreaticoduodenal pseudoaneurysm leading to duodenal obstruction is remarkably uncommon. Celiac artery dissection, a seldom-seen form of visceral arterial dissection, typically manifests with acute, intense abdominal pain attributable to the dissection itself or exhibits signs and symptoms of intestinal ischemia. This case underscores the necessity of broadening the scope of differential diagnoses for duodenal obstruction to encompass pathologies associated with the surrounding vasculature.
Rare Presentation of Myocardial Ischemia in a Patient with an Anomalous Right Coronary Artery

Introduction:
Coronary anomalies are coronary patterns that occur rarely in the general population. The most common type of these anomalies is the emergence of the right coronary artery (RCA) from the left coronary sinus, which in most patients is either asymptomatic or leads to sudden cardiac death (SCD).

The Case:
This case report presents the case of a 35 year old male patient who had an inferior ST elevation myocardial infarction (STEMI) for which percutaneous coronary intervention was attempted and failed due to the patient going into cardiac arrest. The patient was subsequently managed medically in the cardiac intensive care unit and followed up outpatient with a cardiologist. The patient experienced symptoms of exertional angina and had a CT angiography of the heart and coronary vessels that identified an anomalous RCA emerging from the left coronary sinus. He was diagnosed with an anomalous RCA with malignant take-off from the left coronary sinus, and subsequent management was surgical with a coronary artery bypass graft (CABG) x1 using the right internal mammary artery (RIMA to RCA). Intraoperatively, the patient was found to have sclerosis of the RCA proximally for about 5cm. The patient did well post-operatively and had no symptoms of exertional angina upon outpatient follow up with both his cardiologist and cardiothoracic surgeon.

Discussion:
Most cases of coronary anomalies have been found to be either completely asymptomatic or lead to SCD, especially in young patients, however very few cases in literature identified patients with this anomaly presenting with STEMI such as in this case. Another significant finding in this patient is sclerosis of the RCA, where although some articles propose that anomalous coronary arteries may be more susceptible to atherosclerosis, others conclude that there may be no association; concluding that factors such as intramural course, slit-like origin, and acute angle take-off might be more significantly associated. Moreover, patients with coronary anomalies rarely experience cardiac events solely due to their anomaly, such as with this patient. Finally, this patient’s significantly young age at 35 years old is significant given the mean age of patients undergoing CABG being 60.8 years in one cross-sectional study. Current management of symptomatic patients with these anomalies is under debate, with many patients undergoing CABG such as in this case, although one study shows no significant difference in mortality or 10-year survival with surgical intervention despite having higher surgical intervention rates. Percutaneous coronary intervention (PCI) of patients with coronary anomalies has been shown to be difficult or unsuccessful in most studies, although a few cases in literature were successful with tools such as multidetector CT (MDCT). Awareness of these anomalies and their possible presentation, risk factors, and radiologic findings is important especially given their potential for SCD. More research is needed to further elucidate the best treatment options and perhaps even increase the potential for non-invasive interventional techniques such as PCI.
Pain from Chest to Scrotum: An Initial Presentation of Aortic Dissection

Introduction: Acute aortic dissection (AAD) is a rare yet highly lethal phenomena with a mortality rate of 50% within 48 hours (1). Timely diagnosis and management of AAD is crucial, yet often is delayed by ambiguous symptomatology. Most commonly, patients present with extreme chest pain, dyspnea, and abdominal pain, but clinical presentation can also include focal neurological defects, altered mentation, and syncope (1).

Case Description: A 56 year old man with a history of hypertension, asthma, polysubstance use, insecure housing, and seizure disorder presented to the emergency department complaining of abdominal pain. During this admission, his workup included basic labs, lactic acid, and chest x-ray that were all unremarkable. He was given fluids, Droperidol and subsequently discharged. Three days later, the patient presented again to the emergency department complaining of chest pain, shortness of breath, abdominal pain, generalized fatigue, and a new productive cough. Furthermore, he mentioned a severe (10/10) shooting pain all the way from his chest to his scrotum, across his shoulders and diffusely throughout his abdomen. Of note, upon further investigation, the patient smoked heroin three days prior to his initial presentation to the hospital.

In the emergency department, the patient was afebrile and hemodynamically stable. Laboratory findings demonstrated elevated lactic acid and troponins. Upon examination, he was found to have a bounding S1 and S2 with a holosystolic murmur at the left upper sternal border, and a visible and inferolaterally displaced point of maximal impulse. These findings, in conjunction with his chest x-ray showing a prominent aortic notch, prompted an transthoracic echocardiogram which showed pericardial effusion and a dissection flap. An emergent CT-Angiogram showed an Type A aortic dissection spanning from the ascending aorta to just proximal of the renal arteries. Vascular Surgery was emergently consulted, and the patient was taken to the operating room for an emergent Type A aortic dissection repair.

Discussion: Aortic dissection is commonly misdiagnosed, with one-third of aortic dissection patients receiving an incorrect initial diagnosis (2). The case underscores the need for clinicians to consider AAD in patients presenting with acute chest pain, dyspnea, abdominal pain and scrotal pain, even in the absence of classic distress. Furthermore, the association between substance use, particularly heroin, and AAD warrants further investigation. Awareness of atypical presentations and a comprehensive diagnostic approach are crucial for timely identification and intervention in AAD cases.

Conclusion: This case report emphasizes the diagnostic challenges associated with atypical presentations of AAD. Clinicians should maintain a high level of suspicion, especially in patients with diverse symptomatology and risk factors. Further research is needed to explore the relationship between substance use, such as heroin, and the development of AAD. Timely recognition and intervention are pivotal for improving outcomes in this life-threatening condition.

References:


A unique case of progressive monocular vision loss secondary to Ramucirumab and Docetaxel therapy

Introduction: Ramucirumab and Docetaxel combination regimens are increasingly being utilized in advanced non-small cell lung cancer (NSCLC) following disease progression on immunotherapy and platinum-based therapies. While the side-effects of both chemotherapy and immunotherapy are vast, we present a unique case of monocular vision loss following the initiation of Ramucirumab and Docetaxel.

Case: Here we present a case of a 66-year-old male with metastatic NSCLC to bone and brain who experienced progressive right eye visual loss shortly after initiation of Ramucirumab and Docetaxel. Following induction of therapy, the patient noted increasingly blurred vision. 3 months later, after beginning cycle 5, the patient was admitted for an acute worsening of visual disturbances such as black spots that were increasing in size, flashes of light, and daily headaches. Initial examination showed 2+ right optic disc edema, right afferent pupillary defect and significant deficits in visual acuity. Fundoscopy demonstrated diffuse disc edema of the right eye with large vessel obscuration and small cups bilaterally. Lab results on presentation were remarkable for elevated inflammatory markers (ESR 89, CRP 2.7), and showed no evidence of infectious etiology. Despite low concern for possible giant cell arteritis, the patient was started on prophylactic IV steroids.

CTA imaging did not support stenosis or arteritis in the head or neck. MRI brain demonstrated stable metastatic disease with no evidence of vasogenic edema. MRI orbits showed no enhancement of the optic nerve; globes, optic nerve sheath complexes, extraocular muscles and orbits were all unremarkable. Furthermore, the patient's cerebrospinal fluid was negative for malignant cells and oligoclonal bands.

As the patient’s vision loss showed moderate symptomatic improvement on steroid treatment, and imaging showed stable disease burden of the brain, it was deemed unlikely that his presentation was secondary to tumor infiltration. Thus, this posed more credence to an alternative diagnosis. Given the timing of the vision change relative to the initiation of the systemic therapy regimen, and low clinical suspicion for giant cell arteritis, a diagnosis of toxic optic neuropathy could not be ruled out; thus, his therapy regimen was temporarily discontinued. In addition, due to the patient’s small discs, non-arteritic ischemic optic neuropathy (NAION) remained on the differential.

Since discharge, the patient has continued a gradual steroid taper. At 2 months post-presentation, he endorses symptomatic improvement; however, visual acuity tests showing persistent deficits and optic disc edema suggest incomplete resolution of pathology. While there are rare cases reporting possible Docetaxel induced optic neuropathy, our patient’s unusual presentation along with case reports linking anti-VEGF agents to NAION suggest a potentially mixed etiology of optic neuropathy secondary to combination Docetaxel and Ramucirumab therapy.

Discussion: While the association between chemotherapy, immunotherapy, and ocular disturbances remains an area of active research, this case illustrates a unique instance in which Ramucirumab and Docetaxel combination therapy resulted in visual deficits. Any visual disturbances following this treatment regimen's initiation should be followed up with prompt ophthalmologic evaluation and treatment to prevent debilitating long-term visual loss.
Assessment of Breastfeeding Education in Medical Training

In recent years, inadequate education of physicians on breastfeeding and lactation has been well-documented.1,2,3 Most physicians, especially in primary care, will encounter children or women of childbearing age throughout their career, and a baseline knowledge about this topic should be the expectation for medical school graduates. As such, in 2019, the Academy of Breastfeeding Medicine issued a revised set of educational objectives for medical trainees at various stages of education.4 In this statement, objectives for medical school were broken down into preclinical and clinical training objectives. In this project, our goal was to assess the degree to which medical training at our medical school, Wayne State University School of Medicine, is addressing these aims.

The assessment of breastfeeding education was accomplished by surveying medical students at various stages of training (M1-M4) and eliciting their level of confidence in and understanding of the topics outlined in the objectives by the Academy of Breastfeeding Medicine. Our survey also included some assessments of knowledge, background information of participants, and optional free response questions to generate qualitative feedback and suggestions for improvement. Participants were also given an option for providing feedback on the survey quality itself. Surveys were sent to students via email.

We used ANOVA and regression analysis to examine the quantitative data and, with the qualitative data, identified themes of student suggestions for improvement. Our analysis showed that breastfeeding objectives are not being met across all student populations, but there is improvement over time. Both reflected in the quantitative and qualitative portions of our survey, students are learning anatomy and physiology related to breastfeeding but instruction on mechanics, guidelines, and social factors is limited. Our research exemplifies the inconsistency between student’s perceived competency and the current educational guidelines and charts the course for potential curricular changes as identified by students. These changes could be trialed at medical schools anywhere, which, in turn, will give medical students a more well-rounded education that allows them to be better providers to a diverse patient base in the future.

References:


INTRODUCTION

Staphylococcus lugdunensis (SL) is a species of coagulase-negative staphylococci found as part of normal skin flora. In recent years, S. lugdunensis has been identified as an invasive disease with similar virulence to S. aureus. It is most associated with infective endocarditis and foreign body-related infections. We report a case of a 72-year-old female with multiple comorbidities presenting with native joint septic arthritis. Direct inoculation or hematogenous spread is known to infect joints. There have been many reports describing the strong association of SL and prosthetic joint diseases, most of these infections were reported between 6 weeks and 4 years after implantation.

CASE DESCRIPTION

We describe a 72-year-old female with a past medical history significant for end-stage renal disease on hemodialysis via permcat, heroin, and cocaine use disorder, essential hypertension, seizure disorder, and previously treated Hepatitis C infection. Initially, the patient presented with acute knee pain secondary to a mechanical fall. She was treated for opioid withdrawal and received NSAIDs for knee pain. An aspiration was performed which showed no growth but before any further workup could be performed, the patient left against medical advice. She later presented two weeks after for similar knee pain. On examination she was afebrile, her right knee was warm, erythematous, tender to touch, with a reduced range of motion. She was unable to bear weight on the affected leg. She was started empirically on IV Vancomycin and Cefazolin. Joint aspiration was repeated, and the culture grew S. lugdunensis with resistance only to penicillin. Sequential blood cultures did not show any growth. The patient received two arthrotomies with irrigation and debridement and was discharged after eight days of hospital stay with a one-month course of doxycycline. She yet again presented to the emergency department three days later with worsening right knee pain. The patient revealed that she did not receive her antibiotics after being discharged due to transportation and insurance issues. She received two repeat arthrotomies once again with irrigation and debridement and was restarted on a 21-day course of doxycycline. She was discharged to a subacute rehabilitation facility following symptom resolution.

DISCUSSION

There are few documented cases of SL septic arthritis of native knee joint, with even fewer cases of incomplete symptom resolution due to treatment noncompliance. Socio-economic determinants can significantly affect sequelae of S. lugdunensis septic arthritis.
Dental Abscess Complicated By Ludwig’s Angina

Case Presentation:

A 52-year-old immunocompetent male presented with 3 days of right neck pain, swelling, and dysphagia, and a right lower molar cavity. Workup was notable for leukocytosis, and CT neck was suspicious for a submandibular abscess with possible extension to the oropharyngeal wall, palatine, and lingual tonsils with resorption of the posterior mandibular molar. A panoramic radiograph showed a deeply carious tooth #32 with periapical radiolucencies involving the mesial and distal root. The patient was started on Unasyn for his submandibular infection and Ludwig’s angina. Repeat CT neck showed increased fluid collection at C6 which tracked inferiorly to the right parapharyngeal and retropharyngeal spaces. Right submandibular gland enlargement with mass effect on the airway was noted, and incision and drainage (I/D) of the right pterygomassesteric, parapharyngeal, and peritonsillar space abscesses was performed with extraction of teeth 28, 29, and 32. Abscess culture was positive for S. lugdunensis and S. constellatus. The patient was discharged on amoxicillin/clavulanic acid, and recovery was uncomplicated.

Discussion:

Ludwigs angina is an aggressive infection of the floor of the mouth with rapid spread within the submandibular, sublingual, and submental spaces, more common in immunocompromised patients or those with poor dentition. Over two-thirds of patients with Ludwig angina have a dental source of infection, usually involving the 2nd or 3rd mandibular molar teeth. Infection also can stem from tonsillar abscesses, trauma, or sialadenitis [1].

The classic presentation of Ludwigs Angina shows painful, bilateral submandibular swelling and edema, fever, dysphagia, and muffled voice. On physical examination, patients have tender, symmetric, and "woody" induration, with palpable crepitus in the submandibular area [2]. If left untreated, severe, life-threatening complications such as airway edema and sepsis can occur.

In our patient’s case, the submandibular abscess progressed to involve the parapharyngeal and retropharyngeal spaces within 5 days of symptom onset, highlighting the importance of rapid detection and treatment. Worth noting, our patient’s wound cultures were positive for S. lugdunensis and S. constellatus, two organisms not commonly isolated in cases of Ludwig’s angina but have been shown to have invasive potential [3]. This may be because many odontogenic infections do not progress to abscess formation, making wound culture and isolation of the causative organism difficult. Although S. constellatus is considered to be part of “normal dental flora” rarely does infection lead to abscess formation [3]. However, there have been documented cases of S. constellatus leading to invasive respiratory infections requiring surgical interventions [4,5,6].

Conclusion:

This case report highlights the significance of recognizing Ludwing’s angina early in its course. Timely intervention, including surgical drainage and antibiotic therapy, can significantly improve patient outcomes and reduce risk of complications associated with this aggressive infection.
Atrial Fibrillation Saved My Colon

Gamella haemolysans is a facultatively anaerobic, gram-variable cocci and commensal flora within the human upper respiratory tract, gastrointestinal tract, and genitourinary tract. Although the Gamella species rarely cause infections in humans, the most commonly reported infection caused by Gamella haemolysans is infective endocarditis. Other reported infections include meningitis, keratitis, spondylodiscitis and osteomyelitis. We discuss a 75-year-old male who presented with melena and Gamella haemolysans bacteremia leading to a biopsy-proven diagnosis of colon adenocarcinoma.

A 75-year-old male with type 2 diabetes, non-alcoholic cirrhosis with known grade 1 esophageal varices and ascites, chronic lymphocytic lymphoma (CLL) on ibrutinib, sick sinus syndrome with remote pacemaker implantation and recently diagnosed atrial fibrillation presented to the emergency department (ED) with melena after starting apixaban 1 month ago. He also reported fevers, chills, and malaise. He denied weight loss, back pain, change in vision, and headaches. Initial vitals were: temperature 38.2 Celsius, blood pressure 130/49, heart rate 100 and respiratory rate 22. Laboratory evaluation revealed BUN 29 mg/dL and creatinine 1.55 mg/dL along with decreased hemoglobin to 6.7 g/dL from baseline 13.5 g/dL. Iron studies showed ferritin 58 ng/mL, iron level 50 ug/dL, and total iron binding capacity 243 ug/dL. Blood cultures obtained on presentation grew Gamella haemolysans.

He was empirically treated for presumed cirrhotic upper gastrointestinal bleed thought to be provoked by apixaban with IV pantoprazole, octreotide, ceftriaxone and a transfusion of packed red blood cells. Esophagogastroduodenoscopy redemonstrated known grade I esophageal varices, normal stomach and duodenum without stigmata of bleeding. Colonoscopy revealed an ulcerated 2.5 cm mass with friable, erythematous mucosa in the proximal ascending colon with biopsy confirming adenocarcinoma. CEA was 1.8 ng/mL (<6.1 ng/mL), staging CT imaging did not disclose metastasis and orthopentogram did not show dental disease. Transesophageal echocardiography ruled out infective endocarditis and repeat blood cultures did not grow any organisms. He underwent uncomplicated robotic right hemicolectomy and was discharged 3 days later in stable condition with apixaban restarted for stroke prevention.

Gamella haemolysans is a rarely causes bacteremia. However, it has been implicated as an opportunistic pathogen in the elderly, such as this patient. This patient also had risk factors for immunocompromise, increasing his susceptibility to opportunistic pathogens.

Antimicrobial susceptibilities for Gamella haemolysans are not routinely performed, however isolates demonstrate good susceptibility to penicillins and ceftriaxone. Gamella haemolysans is most commonly reported in infective endocarditis and dental infections. Although this patient was found to have ulcerated colon adenocarcinoma as the likely source of his bacteremia, other investigations such as an orthopentogram and transesophageal echocardiogram were completed to rule out other sources and/or complications of bacteremia.

Streptococcus bovis infection is commonly associated with an increased likelihood for colon cancer. Clostridium septicum infection has also been associated with colon cancer. Enterococcus and other bacteria may also be associated with colon cancer, however are not well studied. The presence of an ulcerated colon adenocarcinoma with concurrent Gamella haemolysans bacteremia in this patient suggests that colon cancer may be associated with an increased risk of this infection, particularly in the immunocompromised.
Legionnaires’ disease: more than just your average CAP

Introduction:

Legionnaires’ disease, most commonly caused by Legionella pneumophila serotype 1, varies in its clinical manifestations. While typically presenting as community-acquired pneumonia (CAP), it can also present with various clinical and laboratory findings ranging from mild to severe that can guide diagnosis. A high index of suspicion and careful history-taking is paramount, including accounting for exposures to contaminated water or travel. Morbidity significantly increases if not treated with appropriate antibiotics. This case describes a patient who presented with nonspecific respiratory symptoms and extreme myalgias who was found to have Legionnaires’ disease featuring concomitant extrapulmonary findings.

Case Presentation:

A 34-year-old female with a history of smoking presented with 3 days of progressive dyspnea, fatigue, muscle soreness, and cough productive of rust-colored sputum. On presentation, she was afebrile, tachypneic on room air, and her exam was remarkable only for mild crackles in the left lower lung base. Laboratory results were significant for leukocytosis (14.6 K/μL) and electrolyte derangements including hyponatremia (128 mmol/L), hypokalemia (2.6 mmol/L), and hypomagnesemia (1.7 mg/dL). Additional laboratory findings included an elevated creatinine (1.66 mg/dL), transaminitis (ALT 58 IU/L, AST 75 IU/L), and an up-trending creatine phosphokinase (CPK) of 3,334 IU/L that later peaked at 10,860 IU/L. Imaging demonstrated hazy opacities concerning for multifocal pneumonia and the patient was thus started on CAP coverage. The patient also received IV fluids for her acute kidney injury and electrolytes were aggressively repleted.

On obtaining further history, the patient noted that she had a malfunctioning AC unit for several months, suggesting a potential source of exposure. This, coupled with the patient’s unique presentation of pneumonia complicated by rhabdomyolysis and AKI prompted high clinical suspicion for waterborne pathogens, specifically Legionella pneumophila. Subsequent urine antigen testing was found to be positive, thereby confirming the diagnosis of Legionnaires’ disease. Antibiotics were adjusted to a 10 day course of Azithromycin for enhanced coverage for Legionella spp. CPK down-trended, and acute kidney injury resolved with fluids. Overall, the patient had significant clinical improvement and was discharged home.

Discussion:

Having a clinical suspicion for Legionella infection demands meticulous history-taking. Lacking a pathognomonic presentation, Legionella manifests in various ways. Urine antigen testing is sensitive, but only detects serotype 1 out of 5. Other clues to diagnosis include changes in electrolytes, creatinine and transaminitis. Rhabdomyolysis, as found in our patient, has also been reported on rare occasions to accompany Legionella infections. This patient’s unique presentation of pneumonia, rhabdomyolysis, and acute kidney injury in the context of environmental exposure highlights the importance of timely detection and initiation of appropriate treatment. This is especially relevant in the context of CAP, which is more commonly encountered and can result in Legionella’s atypical pneumonia to be overlooked. Thus, a nuanced approach to history-taking and diagnostic testing is necessary for all clinicians, even in common presentations of pneumonia.
The Beast Unveiled: A Clinical Vignette of Anabolic Steroid-Induced Hepatotoxicity

We present a case of anabolic steroid-induced liver injury in a previously healthy male leading to cholestasis and hyperbilirubinemia.

A 34-year-old male with no medical history presented with 4 days of jaundice, pruritis, dark urine, and pale stools. Physical exam demonstrated scleral icterus and jaundice. He had been using an anabolic steroid, “Anabolic Beast Blend”, which is composed of Testosterone E 100 mg, Trenbolone E 100 mg, and Drostanolone E 100 mg. He was initially injecting 300 mg intramuscularly every other day for 3 weeks followed by twice daily injections for 2 weeks. Labs showed elevated aspartate aminotransferase (AST) of 40 IU/L, alanine transaminase (ALT) of 126 IU/L, alkaline phosphatase (ALP) of 149 IU/L, total bilirubin (Tbil) of 13.2 mg/dL, and direct bilirubin of 10.8 mg/dL. Hepatitis panels, Alpha-1-Antitrypsin phenotyping, ceruloplasmin, and autoimmune hepatitis panels were negative. He did not exhibit signs of encephalopathy and international normalized ratio (INR) remained unaffected. Ultrasound revealed echogenic liver nodules, without gallbladder or bile duct pathology. Computed tomography showed ill-defined hepatic lesions, which were defined on magnetic resonance imaging as incidental hepatic hemangiomas. A needle biopsy was performed on hospital day 3. While results were pending the patient was treated with two 40 mg injections of methylprednisolone. Though AST and ALT quickly stabilized, ALP and bilirubin continued to increase, so ursodiol 600 mg twice daily was started. Shortly after, his ALP and Tbil reached a maximum, then decreased. Pathology revealed severe cholestasis and inflammatory infiltrate consistent with drug-induced liver injury. The patient was discharged in stable condition with ALP of 72 IU/L, AST of 28 IU/L, ALT of 62 IU/L, and Tbil of 28.9 mg/dL. Outpatient appointments were made with hepatology, but the patient was lost to follow-up.

Anabolic steroids are used therapeutically in conditions such as hypogonadism but are often misused for performance enhancement. They are associated with acute liver injury, and chronic use has been linked to hepatic adenomas and hepatocellular carcinoma. Possible mechanisms of injury include oxidative damage, increased synthesis of bile acids, and hepatocyte hyperplasia. Most reported cases of associated drug-induced liver injury have described cholestasis on biopsy and have occurred in men between 20-40 years of age, as is consistent with our case. Reported injuries are most often reversible.

The use of anabolic steroids for aesthetic and athletic purposes has risen recently, prompting the need for further evaluation of potential risks. Risk is increased by supraphysiologic dosing and blends of multiple steroids and other active components, as observed in this patient. Additionally, there are several reports of over-the-counter performance supplements testing positive for anabolic steroids. This case highlights the importance of inquiring about patients’ use of supplements, both over the counter and illicit. Physician awareness of potential side effects is essential to promote discussion with patients about risks. Understanding potential complications is essential to effectively screen for unintended consequences.
A presentation of familial hypokalemic period paralysis syndrome

Hypokalemic periodic paralysis (HPP) is a rare channelopathy of skeletal muscle that primarily affects adolescents in the first or second decade of life. Patients present with sudden episodes of paralysis associated with severe hypokalemia. Recognizing this rare condition is important in both avoiding potentially lethal complications such as respiratory failure and helping patients prevent future episodes of paralysis.

A 20-year-old man with a history of HPP presented to the ED with significant focal weakness in his left arm and right leg. He had regular follow-ups with pediatric neurology since his diagnosis of HPP in 2022 and genetic testing was positive for mutation in CACNA1S. This variant is a common familial form due to a mutation in the dihydropyridine-sensitive calcium channel gene in skeletal muscle. He had previous hospital admissions with similar presentation, including admissions involving intubation for respiratory insufficiency. His last admission was one month prior, when he was found hypokalemic. After aggressive potassium replacement, his paralysis resolved. The patient was discharged on potassium chloride 40 mEq five times daily and spironolactone 25 mEq qAM and 50 mEq qPM. He reports adherence to the medication regimen throughout the interval leading to the current presentation.

Upon presentation, he denied any fever, chills, chest pain, shortness of breath, nausea, vomiting, or abdominal pain. Initial labs were notable for a potassium of 2.1. Given his history of intubation, he was admitted to the medical intensive care unit (MICU) for airway monitoring. In the MICU, he was given an additional 200 mEq of potassium to the 100 mEq received in the ED. With the aggressive repletion of potassium, he reported resolution of his symptoms. He was then stable for transfer to the general practice unit for continued monitoring of electrolytes and eventual discharge.

This case illustrates the sudden paralysis seen in patients with HPP and the necessary precautions one should take. It is important to monitor closely for possible diaphragm paralysis and airway collapse. Although HPP is rare, affecting only 1 in 100,000 individuals, recognition and prompt treatment are vital to stabilizing patients. Additionally, one should monitor electrolytes and maintain potassium in a safe range. Recognition of this disease, its management, and proper patient education is crucial to reduce the number of patient hospital admissions and improve quality of life.

Ref:


Salmonella enterocolitis causing Intussusception and Bacteremia in a patient with AIDS

Introduction:

Rates of Gram-negative bacterial enteric infections are at least 10 times higher among adults with HIV than in the general population, especially in patients not on antiretroviral therapy (ART). In addition to a broad exposure and sexual history, clinicians must consider whether additional complications may occur and have a low threshold to search for them.

Here, we present a 30-year-old female with AIDS who presented with mild enteritis that later worsened resulting in diagnosis of invasive Salmonella enterocolitis with intussusception and bacteremia. The complexities of this case serve as an example of the intersection between infectious diseases, HIV, and gastrointestinal pathology.

Case presentation:

A 30-year-old lady with a history of HIV not on antiretrovirals presented to the emergency department (ED) with nausea and non-bilious, non-bloody emesis. Intravenous fluids and anti-emetics led to symptomatic improvement and she was discharged with stable vital signs.

One week later, she returned to the ED with a new worsening of nausea, vomiting, abdominal pain, and one episode of hemoptysis. Physical exam was significant for right upper quadrant and epigastric tenderness without guarding, as well as oral thrush. She was started on empiric antibiotics (cefepime, vancomycin, and metronidazole) and blood cultures were obtained. Computer tomography of the abdomen and pelvis revealed small bowel intussusception in the left hemiabdomen with enterocolitis. Labs showed no leukocytosis, but CD4=92 cells/mm3. On day #2 anaerobic blood cultures had gram negative rods which grew as black colonies on hektoen enteric agar confirmed to be Salmonella enteritidis on molecular testing. Repeat blood cultures were negative and A transthoracic echocardiogram showed normal valves. Surgeons monitored her and the intussusception self-resolved on repeat imaging. Antibiotics were de-escalated to ceftriaxone and then high dose Bactrim DS 2 tablets twice a day for total 14 days along with an appointment to re-establish care in the HIV clinic.

Discussion:

Intussusception is a gastrointestinal pathology involving the translocation of one part of the bowel into an adjacent segment, commonly known as “telescoping.” In adults, it is extremely rare. Bacterial enteritis has been shown to significantly increase the relative risk of intussusception in children, however, no such data exists in adults.

In this case, salmonella enterocolitis led to bowel inflammation and an intussusception with bacteremia. Notably, HIV infection increases the risk of Salmonella bacteremia 20 to 100 times and mortality as much as 7 times compared to people who do not have HIV. Although repeating blood cultures is not standard for gram-negative bacteria, Salmonella has the ability to adhere to damaged endothelium, predisposing to endovascular complications and thus documentation of clearance was imperative in this immunocompromised patient presenting from the community. The optimal duration of therapy for HIV-related Salmonella infection has not been defined. Our patient received 14 days due to her low CD4 count and invasive disease provided the clearance of bacteremia was documented. The value of this secondary prophylaxis has not been established and must be weighed against the risks of long-term antibiotic exposure.
Spontaneous Pectoral Hematoma: A Case of Polypharmacy and Adverse Medication Effects

Tendinopathy is a rare side effect of fluoroquinolone use that is more common in older adults and those with preexisting renal disease and diabetes. Although infrequent, patients taking concurrent anticoagulation medications can develop spontaneous hematomas associated with significant disability and potentially life-threatening bleeding.

A 67-year-old man with past medical history of heart failure, hypertension, gout, type 2 diabetes mellitus, and paroxysmal atrial fibrillation was admitted to the hospital for acute left hip, knee, and back pain. Orthopedic surgery began workup for suspected polyarticular osteoarthritis. Medication reconciliation revealed ciprofloxacin for a recently diagnosed urinary tract infection, heavy NSAID use for one week, as well as long-term apixaban use for atrial fibrillation. On the fifth day of admission, the patient reported new onset right upper chest pain and swelling with no apparent trauma or inciting event. Chest ultrasound revealed a 6cm pectoral hematoma and the decision was made to discontinue apixaban. Hemoglobin was noted to have dropped to 8.2 from 14.1 at admission. Cardiothoracic surgery, interventional radiology, and vascular surgery were consulted at this time. On the sixth inpatient day, chest CT revealed an enlarging, 14cm intramuscular hematoma within the right pectoralis major muscle with foci of active hemorrhage. Interventional radiology successfully embolized the second and third branches of the thoracoacromial artery, following which, the hematoma did not increase in size and repeat imaging showed no active bleeding. Hematology was consulted for spontaneous hematoma of unknown etiology and acute blood loss anemia. They reported that the spontaneous hematoma could be secondary to fluoroquinolone-associated tendon inflammation, with severe bleeding secondary to apixaban and NSAID-induced platelet dysfunction. A conversation was then had with the patient to determine future management of paroxysmal atrial fibrillation. The patient had a CHA2DS2-VASc score of four with high risk of stroke and was found to be a strong candidate for a Watchman device. The patient was amenable and apixaban was not restarted. He was discharged in stable condition with cardiology and orthopedic surgery follow up.

This case illustrates the potential for severe bleeding complications with use of direct factor Xa inhibitors, and the value of a complete medication reconciliation. Although there were no specific medication interactions, the patient’s specific risk factors increased his risk for side effects, and his anticoagulant and NSAID use increased the severity of this event. Identifying the specific indication for each medication, as well as the patient’s individual risks and benefits, is critical to prevention and treatment of potentially life-threatening bleeding events.
Diagnostic Blind Spots: How Homelessness and Polysubstance Abuse can Contribute to Delayed Cholangiocarcinoma Diagnoses

Cholangiocarcinoma, a rare but aggressive malignancy, presents diagnostic challenges, particularly in patients where clinical concerns may be obscured by social determinants of health and historical health behaviors.

A 71-year-old man experiencing homelessness with a six-month history of abdominal pain, significant weight loss, and polysubstance use (heroin/cocaine), presented to the emergency department with acutely worsening abdominal pain and novel chest pain. The patient was admitted for acute heroin withdrawal. Upon further workup, the medication-assisted treatment team agreed that given the patient’s history of polysubstance use, and negative workup for acute abdomen, his pain was likely secondary to acute withdrawal. Furthermore, the elevated liver enzymes found on complete metabolic panel seemed consistent with chronic alcohol use. He was subsequently started on buprenorphine 8mg BID. After a day of buprenorphine therapy, the patient’s pain was unremitting. Treatment was advanced to methadone 2.5 mg BID by palliative care, but still failed to control his pain.

On physical exam, the patient was an older gentleman with apparent cachexia, temporal wasting, and unkempt facial hair. He was curled in bed, recoiling with palpation to his abdomen, chest, and extremities. Based on the physical exam and polysubstance use history, additional etiologies of his abdominal pain were explored. Subsequent labs identified underlying hepatitis B and C infections – both conditions that were unknown to the patient. Further workup showed elevated liver enzymes (aspartate aminotransferase (AST) of 154 IU/L, alanine aminotransferase (ALT) of 57 IU/L), alkaline phosphatase (ALP; 246 IU/L), and bilirubin (3.5 mg/dL). Given this cholestatic pattern with hepatocellular damage, an abdominal CT was ordered, revealing an 18 cm hepatic mass which was suspected to be hepatocellular given the history of hepatitis B and C, as well as an AFP (alpha-fetoprotein) of 52,000 ng/mL. The rest of the hospital course was directed at stabilizing the patient for an outpatient biopsy of the mass.

On outpatient follow-up, the liver biopsy showed neuroendocrine features that were most consistent with cholangiocarcinoma. The tumor was positive for cam 5.2, synaptophysin, and arginase. Given the size and nature of the tumor, the patient opted for hospice care and passed in subsequent weeks.

This case underscores the complex interplay between substance use disorders, homelessness, and malignancy. As similar cases are increasingly reported across the US, various metropolitan cities such as Boston and Philadelphia have identified cancer among the leading causes of death in homeless patient populations. While this remains an area of active research, there are studies showing increased disease burden and significantly lower survival rates compared to propensity score–matched reference groups in major counties across Southeast Detroit. Our case supports the importance of considering hepatobiliary malignancy as a differential when chronic abdominal pain and weight loss are reported in this patient population.
Coexisting Langerhans Cell Histiocytosis and Erdheim-Chester Disease: A Complex Case Report with Systemic Implications

Introduction: Mixed Histiocytosis (MH), the coexistence of Langerhans cell histiocytosis (LCH) and Erdheim-Chester disease (ECD), is a rare and intricate phenomenon in the spectrum of histiocytic disorders. LCH, characterized by granulomatous lesions primarily composed of langerin-positive (CD207+) histiocytes, can affect diverse organ systems. It occurs in both children and adults, often associated with the V600E BRAF mutation. In contrast, ECD features the excessive accumulation of foamy histiocytes, predominantly affecting the long bones, cardiovascular, retroperitoneal, endocrine, and orbital systems. It is more common in individuals aged 55-60 years and is derived from monocyte lineage.

MH represents an intersection of these two disorders, sharing a common CD34+ progenitor cell but exhibiting diverse clinical characteristics. This case report presents the diagnosis and management of systemic MH in a middle-aged female.

Case Presentation: A 44-year-old female with a history of LCH presenting with abdominal pain, particularly in the upper abdomen and periumbilical region. Notably, she experienced a visible mass protruding from her umbilicus upon coughing, with subsequent migration of pain to the right lower quadrant.

Initial diagnostic evaluation through computed tomography (CT) imaging revealed lung abnormalities, including volume loss, consolidation in the middle lobe of the right lung, and multiple small pulmonary nodules at the lung bases. Additionally, symmetric and bilateral peri- and pararenal soft tissue infiltration was observed, extending to involve the adrenal glands, renal pedicles, and extensive vascular infiltration, notably in the thoracic aorta, abdominal aorta, inferior mesenteric artery, and inferior vena cava. Hepatosplenomegaly was also noted, raising suspicion of ECD.

A CT-guided peri-nephric tissue biopsy confirmed the diagnosis, displaying ECD morphology and immunohistochemistry positive for CD163, Cyclin D1, Factor 13a, and the BRAF-V600E mutation.

Discussion: MH represents a diagnostic challenge. Our patient’s case was incidentally uncovered during a hernia workup. Its multisystemic impact spanning the skeletal, cardiac, and central nervous systems necessitates meticulous diagnostic evaluations and a multidisciplinary approach to management.

Treatment strategies for MH are complex, primarily focused on addressing ECD. Targeted therapies like Vemurafenib have shown promise in inhibiting the BRAF V600E mutation. In this case, personalized treatment effectively stabilized the patient's condition over two years.

This case underscores the complexity of MH, emphasizing the need for individualized treatment and multidisciplinary collaboration. It highlights the importance of thorough diagnostic assessments and imaging to determine the systemic nature of the disease. Further research and discourse within the realm of mixed histiocytosis are warranted to enhance our understanding and management of this intricate condition.
Riding the Rollercoaster of Platelets: A Decade-Long Journey with Cyclic Thrombocytopenia

Introduction: Cyclic thrombocytopenia (CTP) is a rare hematological disorder marked by drastically oscillating platelet counts. It is characterized by phases of thrombocytopenia that can cause symptoms such as easy bruising and prolonged bleeding, interspersed with periods of normal or increased platelet levels, which carry the risk of excessive clotting. CTP presents a diagnostic challenge due to its similarities with Immune Thrombocytopenia (ITP) in clinical presentation. Notably, the condition is distinguished by its exclusive impact on the platelet line, unlike other cyclic hematological disorders that affect multiple blood cell lines. This report discusses a case of CTP managed over nearly a decade, highlighting our approach to the challenges in the diagnosis and treatment of this rare hematological disorder.

Case Presentation: We report a 54-year-old woman who presented at age 45 with petechial rash, heavy menstruation and severe thrombocytopenia with a platelet count of < 5,000 x 10^9/L. She was initially diagnosed with immune thrombocytopenia (ITP). However, she did not respond to standard ITP therapy with corticosteroids, intravenous immunoglobulin, and thrombopoietin (TPO) mimetics. Her severe thrombocytopenia was followed by rebound marked thrombocytosis, ultimately leading to clinical diagnosis of idiopathic cyclic thrombocytopenia (CTP). She also did not respond to other treatment modalities including rituximab, splenectomy, and immunosuppressive medications. Her platelet count fluctuated from <5,000 to >1,000,000 x 10^9/L approximately every 4-6 weeks. Her only effective management has been platelet transfusion support for platelet count below 10,000 x 10^9/L. She has not experienced any bleeding or thrombotic complications thus far.

Discussion: Clinical evidence demonstrates that the etiology of CTP is complex, with potential associations to TPO expression, megakaryocyte dysfunction, unstable peripheral control mechanisms, hormonal imbalances, infections, or autoimmune factors. This disorder exclusively affects platelet counts, differentiating it from other cyclic hematological disorders. The rarity and elusive nature of CTP contributes to frequent misdiagnoses, complicating efforts to accurately determine its incidence and prevalence.

Our case, unresponsive to conventional ITP treatments, highlights the importance of vigilant platelet level monitoring in unexplained thrombocytopenia to distinguish CTP from ITP. Precise diagnosis allows CTP patients to avoid unwarranted therapies, such as immunosuppressants, hormonal mimetics, and/or splenectomy, and their associated side effects. The case presented here underscores the diagnostic and management complexities associated with CTP, emphasizing the need for research to unravel CTP’s etiology and develop more effective treatment strategies. Lastly, we advise providers to be on the lookout for recurrent, immunosuppressant-resistant, episodes of thrombocytosis, followed by rebound thrombocytosis, especially in the female patient population.
Medical Student Poster # 55  
Category: Clinical Vignette

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Cardioversion induced Takotsubo cardiomyopathy  

Introduction: Takotsubo cardiomyopathy (TC), also known as stress-induced cardiomyopathy or "broken heart syndrome," is a rare and reversible condition characterized by transient left ventricular dysfunction. It is typically triggered by acute emotional or physical stressors that induce catecholamine release, resulting in microvascular dysfunction and cardiotoxicity from intravascular calcium overload. Other risk factors include genetics and estrogen deficiency, with the most common patient population being postmenopausal women. Patients generally present with chest pain and dyspnea, mimicking coronary artery disease (CAD). Therefore, the diagnosis of TC involves 1) ruling out significant CAD, 2) presence of temporary hypokinesis, dyskinesis, or akinesis in LV segments with or without apical involvement, 3) possible recent changes detected on ECG (like ST-segment elevation and/or T-wave inversion) or significant elevation of serum cardiac troponins, and 4) non-existence of pheochromocytoma or myocarditis.

Case Description: A 77 year old obese woman with a history of hypertension, hyperlipidemia, chest pain with abnormal stress test, and atrial fibrillation (AF) presented with sporadic palpitations and fatigue. Vital signs revealed tachycardia and the physical exam was normal. Holter monitoring revealed 100% burden of AF (average HR of 112 bpm). Coronary angiography showed no obstructive disease. TEE showed normal global systolic function, mild to moderately dilated atria, and moderate mitral and tricuspid regurgitation. Cardioversion successfully converted AF to sinus bradycardia. Patient was discharged home on amiodarone 400 mg BID and dabigatran (Pradaxa) 150 mg BID. Four days later, the patient presented with substernal chest pressure, exertional dyspnea, mild weight gain, and lower extremity edema. She also noted an increase in family stress in the past few months. Exam findings included 2/6 holosystolic murmur in the mitral position, elevated JVP, diminished breath sounds, and bilateral lower extremity pitting edema. BNP was elevated at 1204 and troponin peaked at 0.227. ECG showed sinus rhythm at 62 bpm and T wave inversions. Echocardiogram demonstrated a significant decrease in ejection fraction (EF 20-25%) with hypokinesis of several regions, consistent with a possible LAD infarct or atypical TC. Chest CT ruled out acute pulmonary embolism. Patient was started on IV furosemide, was switched from carvedilol to metoprolol, and continued on amiodarone. Two days later, the patient's symptoms resolved. Cardiac MRI showed improved left ventricular ejection fraction at 35%.

Discussion: Electrical cardioversion is a procedure used to treat irregular heart rhythms like AF. This procedure is generally considered to be very safe. The incidence of cardioversion-associated TC in patients with AF is fairly low. Among 154, 919 patients admitted with AF who underwent electrical cardioversion in the National Readmission Database from 2018, 0.027% were readmitted with TC. Acute heart failure due to apical type TC is the most common presentation within 48 hours. However, it is important to consider cardioversion induced TC in patients even beyond the 48 hour window. The recovery time is generally 1-2 weeks.
Cholecystectomy and Its Role in the Intersection of Hereditary Elliptocytosis and Gilbert Syndrome: A Clinical Insight

Introduction:

Hereditary Elliptocytosis (HE) is a rare genetic disorder characterized by mutations in the alpha spectrin gene (SPTA1), resulting in structural abnormalities and compromised membrane stability of red blood cells (RBCs). Gilbert Syndrome (GS), on the other hand, is an autosomal recessive condition defined by reduced activity of uridine diphosphate-glucuronyl transferase-1A1 (UGT1A1), leading to elevated levels of unconjugated bilirubin (UCB). Although both conditions are typically asymptomatic, the interplay between their coexistence, especially in conjunction with other comorbidities like chronic kidney disease (CKD) is not well-documented. This report presents a unique case of concurrent HE and GS in a patient with CKD and examines the implications of cholecystectomy in the management of these conditions.

Case Presentation:

Our case involves a 62-year-old African American female with a medical history including stage 3B CKD, congestive heart failure, hypertension, and a previous cholecystectomy. She presented for the management of chronic normocytic anemia with a hemoglobin level of 8.5 g/dL. A blood smear examination identified the presence of elliptocytes, and subsequent genetic testing revealed a mutation in the SPTA1 gene, leading to a diagnosis of HE. Additionally, the testing incidentally uncovered two pathogenic mutations in the UGT1A1 gene, leading to a diagnosis of GS. Interestingly, the patient had no history of jaundice or hyperbilirubinemia following her cholecystectomy, which was performed 22 years prior to her presentation.

Discussion/Conclusion:

HE leads to the breakdown of RBCs, while GS impairs the liver’s ability to conjugate bilirubin. These coexisting conditions can elevate levels of unconjugated bilirubin, consequently increasing the risk of bilirubin gallstone formation. A study involving a pediatric patient with GS and a hemolytic disorder suggested a synergistic effect in elevating the risk of bilirubin gallstones and hyperbilirubinemia. Post-cholecystectomy, the patient in the study showed significantly reduced bilirubin levels and prevented the recurrence of gallstones. Our patient’s experience aligns with these findings; she did not encounter any hyperbilirubinemia episodes following her cholecystectomy. This outcome can be attributed to the bypass of bile storage in the gallbladder, thereby preventing the accumulation of excessive unconjugated bilirubin in the bloodstream. We propose that patients diagnosed with both GS and HE should be informed about the risk of jaundice and advised to avoid potential stressors that could precipitate a hyperbilirubinemia crisis and gallstone formation. In certain scenarios, a cholecystectomy may be a valuable intervention in managing these conditions effectively.
Medical Student Poster # 57  Category: Research

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Optimizing Patient Care: Bridging the Gap Between Micronutrient Deficiencies and Clinical Practice through Comprehensive Vitamin Panels

Identifying vitamin and mineral deficiencies offers opportunities to optimize patient treatment and health. However, vitamin panels are currently underutilized as a clinical tool for establishing diagnoses and providing patient care. This study highlights the need to incorporate vitamin panels when indicated as a crucial part of patient care in clinics and hospitals.

The biological importance of micronutrients has been well studied, and various pathologies related to micronutrient deficiency have been identified. Well-established associations include vitamin A and its role in vision, vitamin D and healthy bone formation, vitamin E as an antioxidant protecting cell membranes from oxidative damage, and Vitamin K supporting anticoagulation. The B vitamins are essential in various physiological processes, such as metabolism and ATP synthesis. Lastly, Vitamin C is essential for collagen synthesis and norepinephrine production.

This retrospective cohort study included 583 patients who presented to Grace Health in Battle Creek, Michigan between August 2019 - June 2023. Inclusion criteria included all patients ages 18 to 99 who presented to Grace Health with systemic dysfunction and a history of insufficient nutritional intake as defined by the “Healthy Eating Index.”

The primary objective of Phase I of this study aimed to identify the prevalence of vitamin deficiencies within Grace Health. Grace Health IT completed data retrieval, which collected data on patients with insufficient nutritional intake and related pathologies. Most recent lab values of Vitamin C, B1, B3, B5, B6, B9, B12, D, A, magnesium, zinc, ferritin, iron, and selenium were obtained. Secondary objectives included identifying associations of vitamin deficiencies with multiple patient variables, such as housing status, education level, BMI, activity level, tobacco use, drug use, and clinical markers. Clinical markers included periodontal disease, depression, easy bruising, malabsorption, iron deficiency, fatigue, memory loss, chronic pain, and paresthesia. A manual chart review was then conducted using REDCap to confirm the accuracy of IT data retrieval. Phase II is currently in the process of expanding the current patient database.

432 (74%) patients who met the inclusion criteria were shown to have one or more micronutrient deficiencies. Notable observational associations included mood disorders with vitamin B9 (88.2%, N=15), zinc (76.9%, N=40) and B6 (73.9%, N=99) deficiencies; tobacco use with vitamin C (80%, N=136) and B9 (76.5%, N=13); periodontal and oral disease with vitamin B9 (76%, N=13); papular rash with vitamin B6 (73.9%, N=99). This study also showed observational associations between vitamin deficiencies and social factors, such as Medicaid status and sedentary lifestyle.

Many common pathologies are impacted by vitamin and mineral deficiencies; therefore, obtaining appropriate vitamin levels when clinically warranted is critical for comprehensive care. This study warrants further investigation into the current use of micronutrient deficiency panels by healthcare providers upon patient presentation of symptoms. Future studies include identifying the relationship between chronic pain and vitamin C deficiency and establishing a vitamin panel protocol for hospitalized patients to decrease readmission rates; however numerous facets of researching these relationships remain open for exploration.