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New York Chapter
American College of Physicians

Annual Scientific Meeting

Poster Presentations

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Albany, NY

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Annual Scientific Meeting

Medical Student Clinical Vignette

Poster Presentations

Medical Student Clinical Vignette

Mahaim Fiber–Mediated Tachycardia in Childhood: Acute Management and Durable Long-Term Outcome Following Catheter Ablation

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Introduction:

Mahaim fibers are a rare subset of accessory pathways, accounting for < 3% of pre-excitation syndromes. Normally, electrical impulses travel from the atria to the ventricles through the AV node and His-Purkinje system. Accessory pathways bypass this route and can cause supraventricular tachycardia (SVT). Unlike rapidly conducting bidirectional pathways in Wolff-Parkinson-White syndrome, Mahaim fibers only conduct anterograde (atria-to-ventricle), slowly and decrementally, conducting slower as heart rate increases – normally only seen in the AV node. These pathways are often atriofascicular, connecting the right atrium to the right bundle branch. Patients typically present in young adulthood, with palpitations and left bundle branch block (LBBB) wide-complex tachycardia. Adenosine can terminate tachycardia by blocking accessory pathway conduction. Catheter ablation provides definitive therapy.

Case Presentation:

A 19-year-old woman reported a tachyarrhythmia at age 10, characterized by sudden-onset chest pressure. ECG revealed regular, wide-complex tachycardia at around 160 bpm with LBBB morphology and QRS duration 152 ms, suggestive of an accessory pathway. Vagal maneuvers and adenosine 6 mg IV were unsuccessful, while procainamide IV restored sinus rhythm. A second episode occurred with a heart rate around 170 bpm. Vagal maneuvers and adenosine 6mg IV were again unsuccessful – adenosine 12mg IV terminated the arrhythmia. Electrophysiology study confirmed Mahaim fiber-mediated SVT. Catheter ablation successfully eliminated the accessory pathway. The patient remained asymptomatic without recurrence nine years post-ablation.

Discussion:

Mahaim fibers were first described by Ivan Mahaim in 1938. “Fiber” reflects the histopathologic identification of discrete conducting tracts, as with His bundle and Purkinje fibers. Estimated population prevalence is approximately 0.5–1 per 10,000

individuals. Palpitations are the most common presenting symptom; younger patients with SVT may present with chest pain, as seen in the present case.

Because Mahaim fibers often insert into the right bundle branch, the electrical signal during tachycardia activates the right ventricle before the left, producing a LBBB pattern on ECG. The tachycardia is antidromic, traveling anterograde through the accessory pathway and retrograde via the AV node, completing the reentrant circuit. Accordingly, Mahaim-mediated SVT may be terminated by vagal maneuvers acting on the AV node (retrograde limb), procainamide acting on the accessory pathway (anterograde limb), or adenosine blocking both.

While adenosine 6mg was ineffective in both episodes, adenosine 12mg terminated the arrhythmia during the second – consistent with the known adenosine-sensitivity of Mahaim fiber conduction. During the first episode, procainamide IV infusion successfully restored sinus rhythm. Procainamide is preferred for antidromic tachycardia as it slows accessory pathway conduction. Either procainamide or higher-dose adenosine are standard options when initial adenosine fails.

Definitive management is achieved with electrophysiology-guided radiofrequency catheter ablation, typically targeting the pathway along the tricuspid annulus. The patient remained arrhythmia-free nine years later, consistent with excellent long-term outcomes reported following successful ablation.

Medical Student Clinical Vignette

The Missing Interval: Exertional Syncope Revealing Concealed Short QT Syndrome in a Collegiate Athlete

Anokhi Amara

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Short QT syndrome (SQTS) is a rare inherited cardiac disorder, with fewer than 200 reported cases worldwide, associated with atrial and ventricular arrhythmias, syncope, andsudden cardiac death. SQTS is a genetically heterogeneous channelopathy: SQT1-3 result fromgain-of-function potassium channel mutations that accelerate repolarization, and SQT4-5 involveloss-of-function calcium channel mutations that shorten the action potential plateau, bothincreasing susceptibility to malignant arrhythmias. Athlete-specific data remain scarce, a gapacknowledged by the 2024 Heart Rhythm Society consensus statement, highlighting the limitedevidence guiding diagnosis and return-to-play decisions in this population.

A 22-year-old male collegiate athlete with no significant medical history presented after aseven-minute loss of consciousness during a game with convulsive activity, oral foaming, andurinary incontinence. He awoke alert without postictal confusion. Family history was negative for sudden death. EMS ECG showed paroxysmal atrial fibrillation at 150 bpm that spontaneously converted to sinus tachycardia shortly after admission. Serial ECGs showed short QT interval (<360 ms), right bundle branch block, and possible prior inferior wall myocardial infarction. Troponins peaked at 166.8 ng/L. Echocardiogram demonstrated LVEF 50-55% with normal RV function. Cardiac MRI revealed mildly reduced biventricular function (LVEF 44.2%) without regional wall motion abnormalities. He remained hemodynamically stable with no recurrent arrhythmia or syncope; anticoagulation was not initiated (CHA_2DS_2-V ASc score of 0). Recommendations included activity restriction, wearable cardioverter-defibrillator (LifeVest),and referral to an electrophysiology center of excellence, where SQTS was confirmed, genetic testing was recommended, and quinidine was initiated. No recurrent syncope was reportedduring follow-up.This represents a rare presentation of SQTS unmasked by exertional syncope and paroxysmal atrial fibrillation in a competitive athlete. Exercise-induced sinus tachycardia cannormalize pathologically short QT intervals due to impaired QT rate adaptation in SQTS, potentially obscuring diagnosis on initial ECGs. Because SQTS may only become apparent at lower heart rates, diagnosis can easily be missed without repeat ECG evaluation. Serial ECGs at lower heart rates are therefore critical to reveal concealed QT shortening. A QTc ≤330 ms is diagnostic, while QTc <360 ms requires supporting clinical, familial, or genetic features.

Notably, paroxysmal atrial fibrillation in a young athlete without structural heart disease may represent an early manifestation of SQTS and should prompt evaluation for inherited arrhythmia syndromes. Early recognition is critical given the risk of sudden cardiac death. Quinidine remains the only pharmacologic therapy with demonstrated long-term efficacy, and ICD implantation may be required in high-risk patients or those with inadequate response to medical therapy. Genetic testing also plays an important role by confirming the diagnosis, guiding family cascade screening, and informing genotype-specific management. Clinicians evaluating exertional syncope in athletes should maintain suspicion for channelopathies and obtain serial ECGs at varying heart rates when initial findings are inconclusive.

Medical Student Clinical Vignette

LDL PARADOX: LOW LDL AS A PROGNOSTIC MARKER AND THERAPEUTIC CHALLENGE IN CHRONIC LYMPHOCYTIC LEUKEMIA

Amrin Chowdhury

Altered lipid metabolism is increasingly recognized in hematologic malignancies, including chronic lymphocytic leukemia (CLL). Emerging evidence suggests that low-density lipoprotein (LDL) and high-density lipoprotein (HDL) levels may serve as prognostic indicators in lymphomas such as diffuse large B-cell lymphoma and peripheral T-cell lymphoma, which complement established tools such as National Comprehensive Cancer Network International Prognostic Index. These alterations may reflect tumor metabolic activity and, in some cases, precede overt clinical progression.

A 67-year-old male with a history of coronary artery disease and hypertrophic cardiomyopathy, was found to have leukocytosis during evaluation for elevated prostate-specific antigen. Laboratory studies revealed a white blood cell count of 17.9 x 109/L, hemoglobin of 14.8 g/dL, and platelet count of 203 x 109/L. Peripheral blood flow cytometry demonstrated 47% circulating lymphocytes expressing CD19, dim CD20, CD5, dim CD23, and CD43, consistent with CLL. The patient was classified as Rai stage 0 and managed with observation.

The patient remained on high-intensity atorvastatin 80 mg daily throughout observation. Longitudinal lipid data demonstrated a progressive decline in LDL over time: 97 mg/dL (2015), 76 mg/dL (2017), and 60 mg/dL at diagnosis in 2022, followed by marked reductions to 39 mg/dL, 6 mg/dL, and 3 mg/dL in 2025. HDL levels remained persistently low throughout this period, ranging from 18-35 mg/dL.

Progressive reductions in LDL and HDL may reflect altered tumor metabolism and subclinical disease activity that is not captured by traditional staging systems. LDL can hence be used as a biomarker for diagnosis and prognosis of lymphoma. Additionally, cholesterol starvation can be used as novel technology to kill lymphoma cells. Prior studies have shown normalization of lipid levels following successful lymphoma treatment, supporting a relationship between lipid metabolism and disease burden. The coexistence of lymphoma-associated hypolipidemia and cardiovascular disease in this case raises important management questions regarding lipid targets, interpretation of lipid biomarkers, and the role of continued statin therapy when LDL levels become profoundly suppressed. Current evidence does not support discontinuation of statin therapy solely because of lymphoma-associated hypolipidemia; however, these patients may benefit from

individualized risk assessment and collaborative management between hematology and cardiology.

Medical Student Clinical Vignette

CHRONIC BILATERAL KNEE EFFUSIONS WITH LYMPHOCYTE-PREDOMINANT SYNOVIAL FLUID: AN ATYPICAL PRESENTATION OF LYME ARTHRITIS DIAGNOSED BY SYNOVIAL FLUID PCR AFTER A FIVE-YEAR DELAY

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Purpose: To report an unusually prolonged and atypical presentation of Lyme arthritis featuring bilateral knee involvement, lymphocyte-predominant synovial fluid, normal inflammatory markers, transatlantic Borrelia exposure, and a five-year diagnostic delay ultimately resolved by synovial fluid polymerase chain reaction (PCR).

Methods: A retrospective single-patient case review was conducted. Clinical data, laboratory results, and serial synovial fluid analyses were obtained from the electronic medical record. The diagnosis was established by two-tier serologic testing and synovial fluid PCR for Borrelia species DNA.

Results: A 33-year-old male presented with a five-year history of recurrent bilateral knee effusions. The initial episode involved significant left knee swelling and stiffness causing painful ambulation without erythema. Over five years, the patient underwent multiple aspirations with consistently negative cultures and received repeated corticosteroid injections without antibiotic therapy. Exposure history revealed four years of residence near Cologne, Germany—a well-established Lyme-endemic region—immediately preceding symptom onset, followed by relocation to Western New York. Despite frequent outdoor activities, he denied any recalled tick bite, erythema migrans, or systemic symptoms. Serologic workup revealed positive Lyme antibody screen with positive IgG Western blot, confirming seropositive Lyme disease by standard two-tier testing. Antinuclear antibody, rapid plasma reagin, and HIV screens were negative. Erythrocyte sedimentation rate was 2 mm/h and C-reactive protein was less than 3 mg/L. Left knee synovial fluid analysis revealed a cloudy aspirate with 14,975 nucleated cells/µL and an atypical lymphocyte-predominant differential (20% neutrophils, 52% lymphocytes, 28% monocytes), diverging from the neutrophil predominance characteristically reported in Lyme arthritis. Synovial fluid PCR returned positive for Borrelia species DNA, providing

definitive confirmation. The patient was treated with oral doxycycline 100 mg twice daily for 28 days with meaningful symptomatic improvement.

Conclusions: This case illustrates several atypical features that collectively delayed diagnosis: bilateral knee involvement, lymphocyte-predominant synovial fluid, normal serum inflammatory markers despite significant synovial inflammation, transatlantic exposure to geographically distinct Borrelia species, and absence of recalled tick bite or erythema migrans. The normal ESR and CRP with 14,975 cells/µL in synovial fluid further distinguish this presentation from septic arthritis and most inflammatory arthritides. The IDSA/AAN/ACR 2020 guidelines recommend synovial fluid PCR as a valuable adjunctive diagnostic tool in seropositive patients. Delayed recognition may result in prolonged morbidity and increased risk of antibiotic-refractory Lyme arthritis.

Medical Student Clinical Vignette

CYTOMEGALOVIRUS REACTIVATION PRESENTING AS PERSISTENT FEVER IN A CRITICALLY ILL PATIENT

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Cytomegalovirus (CMV) reactivation is an underrecognized cause of persistent fever and clinical deterioration in critically ill patients. Distinguishing clinically significant CMV reactivation from asymptomatic viremia remains challenging.

Case Presentation

A 57-year-old man with a history of bipolar disorder and polysubstance use (cocaine, alcohol, and cannabis) presented with acute encephalopathy, agitation, and acute hypoxic respiratory failure requiring intensive care unit admission and mechanical ventilation. He met sepsis criteria with leukocytosis (23 × 10^9/L), and chest radiography demonstrated bilateral pulmonary infiltrates concerning for multifocal pneumonia. Blood cultures grew methicillin-sensitive Staphylococcus aureus (MSSA). Despite appropriate antimicrobial therapy, his prolonged ICU course was complicated by recurrent infections, failed extubation requiring tracheostomy, PEG placement, and persistent high-grade fevers without an identifiable source.

An extensive infectious evaluation, including repeat blood cultures, cerebrospinal fluid studies, CT imaging, fungal testing, HIV testing, and repeat respiratory cultures, failed to identify an alternative cause of persistent fever. Although leukocytosis resolved, persistent lymphocytosis prompted evaluation for CMV reactivation. CMV serologies were positive, and quantitative plasma CMV PCR demonstrated a viral load of 102,000 IU/mL.

Given persistent fevers despite appropriate antimicrobial therapy, high-level CMV DNAemia, and multiple risk factors for reactivation, including prolonged critical illness, mechanical ventilation, corticosteroid exposure, recurrent bacterial infections, and prolonged ICU hospitalization, intravenous ganciclovir (5 mg/kg every 12 hours) was initiated.

Within 48 hours of treatment initiation, the patient's fevers resolved completely, and his lymphocytosis normalized. Serial quantitative CMV PCR demonstrated a decline in viral load from 102,000 IU/mL at diagnosis to 627 IU/mL after one week of therapy and to <200 IU/mL following completion of a 14-day treatment course.

Conclusion

This case highlights the importance of considering CMV reactivation in critically ill patients with persistent fever despite appropriate antimicrobial therapy. The combination of persistent fever despite appropriate antimicrobial therapy, extensive negative infectious evaluation, high-level CMV DNAemia (102,000 IU/mL), and rapid clinical and virologic response to ganciclovir strongly supported presumed clinically significant CMV reactivation. Early recognition and targeted viral testing may facilitate timely diagnosis and treatment in selected critically ill patients.

Medical Student Clinical Vignette

A HIDDEN SEQUEL: INTRADURAL EXTRAMEDULLARY SPINAL TUBERCULOMA AFTER TREATMENT

Erica Greiner

Erica Greiner MS, Anna Racanelli MS, Alekhya Kunaparaju DO, T. Charles Martin DO

Tuberculosis (TB) is one of the most common infectious pathogens affecting approximately 10.7 million new individuals worldwide each year. Central nervous system (CNS) involvement is a rare presentation and spinal tuberculoma is an even rarer subtype. Despite the uncommon nature of intradural, extramedullary tuberculoma, this disease presents a diagnostic and therapeutic challenge to clinicians. Symptoms can be nonspecific and largely driven based on compression of the mass on surrounding structures. While cerebrospinal fluid analysis and culture can aid in diagnosis, their utility is limited due to low bacterial load and long incubation times. Additionally, imaging can help raise clinical suspicion and localize the lesion; however, it is insufficient to make a definitive diagnosis. We present the case of intradural, extramedullary spinal tuberculoma following guideline-directed anti-tuberculous therapy for CNS TB.

A man in his 30's with a past medical history of Tuberculosis (TB) meningitis and pulmonary TB presented with 3 weeks of worsening lower extremity sensory deficits and a burning sensation on the right side of his abdomen next to his umbilicus. On exam, he had decreased sensation in his left lower extremity with preserved strength and proprioception. This patient was originally diagnosed with TB about 1 year prior and started anti-tuberculous treatment with isoniazid, rifampin, pyrazinamide, and ethambutol. This regimen required modification due to drug-induced liver injury, and the patient went on to complete 9 months of therapy on rifampin, pyrazinamide and levofloxacin.

He had been off antibiotics for 4 months when he presented to the emergency department. Magnetic resonance imaging demonstrated homogenously enhancing intradural, extramedullary foci within thoracic spinal canals at T3-T4, T5, and T8, likely indicating tuberculoma. The patient was restarted on anti-tuberculous therapy consisting of pyrazinamide, rifampin, ethambutol and moxifloxacin and was taken to the operating room on admission day 7 for a T8 laminectomy and resection of intradural extramedullary mass. Pathology results from the specimen demonstrated fibroconnective tissue with necrotizing granulomatous inflammation consistent with tuberculoma.

This case highlights the potential for persistent CNS TB despite guideline directed therapy and underscores the importance of maintaining high clinical suspicion in

immunocompetent patients. importance of aggressive treatment of CNS TB. It also emphasizes the need for aggressive treatment with close follow-up and careful consideration of treatment duration in these patients.

Medical Student Clinical Vignette

Disseminated Histoplasmosis Masquerading as Leukocytoclastic Vasculitis

Esha Gupta

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Introduction

Disseminated Histoplasma capsulatum is a potentially fatal opportunistic infection that can mimic autoimmune disease in immunocompromised patients. Cutaneous involvement is variable, and gastrointestinal manifestations are rare, often delaying diagnosis and appropriate treatment.

Case Description

A 71-year-old retired physician with history of overlap systemic lupus erythematosus and rheumatoid arthritis presented with five weeks of progressive painful desquamation of the right second and third digits. His rheumatologist recently transitioned him from belimumab to adalimumab. Prior to admission, he was treated with doxycycline and amoxicillin-clavulanate. He denied fevers or constitutional symptoms. MRI of his hand excluded osteomyelitis, vascular ultrasounds showed normal blood flow, blood cultures were negative, and inflammatory markers were elevated. Punch biopsy of the lesion demonstrated leukocytoclastic vasculitis. The presumed adalimumab-induced vasculitis was treated with methylprednisolone followed by high-dose prednisone and rituximab.

Shortly after discharge, he developed a perforated jejunal diverticulum managed with small bowel resection at an outside hospital. Days later, he was readmitted with profound fatigue, dyspnea, and worsening desquamating and new onset nodular skin lesions. On admission, he was febrile and tachycardic with no localizing infection. Bone marrow biopsy showed no evidence of hematologic malignancy. Infectious Diseases was consulted and recommended repeat skin biopsy with testing for microbiologic culture, fungi, mycobacteria, and Nocardia.

Preliminary results again showed vasculitis. However, one week later, Grocott methenamine silver staining of the collected specimen demonstrated yeast forms consistent with Histoplasma capsulatum. Retrospective fungal staining of the jejunal resection tissue and bone marrow biopsy was also positive, revealing the disseminated nature of his infection. Amphotericin B was initiated with signs of early clinical improvement. His course was complicated by recurrent bowel perforation due to an

anastomotic dehiscence. Ultimately, he elected to transition to comfort-oriented care and passed away.

Discussion

Immunocompromised patients presenting with atypical cutaneous lesions require a broad differential work-up. Disseminated Histoplasmosis should be regarded with high clinical suspicion as histopathology consistent with an immunological etiology cannot rule out an infectious source. Step up from TNF-α inhibition, high-dose corticosteroids, to B-cell depleting therapy was likely additive and increased susceptibility to a disseminated infection in this patient. Gastrointestinal Histoplasmosis is an uncommon manifestation, presenting with bowel perforation in this case, leaving the diagnosis more ambiguous. This case also highlights the importance of tissue stewardship. While infection remains suspicious, biopsy specimens should routinely undergo microbiological and fungal cultures and special stains, which were not sent on the initial tissue sample. Previously obtained pathology should be re-analyzed when the clinical course does not align with the presumed diagnosis.

Conclusion

Early recognition of an opportunistic infection, such as disseminated Histoplasmosis, requires broad tissue evaluation and pathology review. This approach is essential to avoid diagnostic anchoring, prevent inappropriate escalation of immunosuppression, and facilitate timely antifungal therapy.

Medical Student Clinical Vignette

ANTICOAGULATION MANAGEMENT IN NEW ONSET EVANS SYNDROME SECONDARY TO SYSTEMIC LUPUS ERYTHEMATOUS WITH UNDERLYING TRIPLE POSITIVE ANTI-PHOSPHOLIPID SYNDROME: A CASE REPORT

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Background: Evans syndrome is a rare autoimmune disorder characterized by immune-mediated cytopenias, most commonly immune thrombocytopenia (ITP) and autoimmune hemolytic anemia (AIHA). It is often triggered by underlying autoimmune disease such as systemic lupus erythematosus (SLE). Antiphospholipid syndrome (APS) is characterized by increased risk for arterial and/or venous thrombosis. The coexistence of both syndromes concurrently necessitates careful management considering bleeding and thrombotic risk.

Case Presentation: A 50-year-old Spanish-speaking female with extensive medical history of SLE on mycophenolate mofetil and hydroxychloroquine, thromboembolism secondary to triple-positive APS now on warfarin, and thrombotic thrombocytopenic purpura (TTP) status post exchange transfusion & rituximab in 2017 presented to the ED with 3 days of mucosal bleeding, petechiae, and ecchymosis. On admission, the physical exam was significant for oral ulcers and petechial rash involving the bilateral lower extremities. Laboratory findings at the time revealed profound thrombocytopenia (PLT 1, N≥150), as well as undetectable haptoglobin, elevated reticulocyte count, RBC panagglutination, and a positive direct antiglobulin test demonstrating hemolytic anemia. There was concern for TTP recurrence given the patient's prior history of TTP and profound thrombocytopenia. However, a normal ADAMTS13 activity assay (91%, N ≥ 61%) ruled it out. These results narrowed the diagnosis down to ITP with concurrent AIHA, which is consistent with the diagnosis of Evans syndrome. Given her comorbidities, intense treatment modalities were initiated with IVIG, dexamethasone, and rituximab with favorable response. Her management was complicated by a significant thrombotic history, triple-positive APS, and high beta-2-glycoprotein I IgG antibody level (IgG anti-β2GPI 150 SGU, N ≤ 20 SGU). Her concurrent high bleeding and thrombotic risk complicated her anticoagulation management. As it was determined that the patient's thrombotic risk is greater in clinical significance than her bleeding risk given her history, anticoagulation with warfarin was continued. There was discussion regarding a trial of heparin while the patient’s thrombocytopenia recovered given its superiority in reversibility in the event of a

hemorrhage. However, the patient was maintained on warfarin due to its ease of laboratory surveillance and titration.

Conclusion: Evans syndrome is a rare autoimmune condition that requires individualized management due to complications from comorbid conditions. Management in this patient is particularly challenging due to underlying APS and an extensive history of thromboembolisms, which necessitate careful balancing of immunosuppression, anticoagulation, and hemorrhagic risk. This highlights the complexities of medical management in patients with multiple concurrent autoimmune conditions.

Medical Student Clinical Vignette

A Case of Syncope as an Atypical Presentation of Löfgren Syndrome Mimicking Lymphoma in a Hispanic Male

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Introduction:

Löfgren syndrome (LS) is an acute variant of sarcoidosis characterized by the classic triad of erythema nodosum, arthritis, and bilateral hilar lymphadenopathy. Diagnosis can be challenging when laboratory markers are noncontributory or imaging suggests malignancy. Syncope is a rare presenting symptom of LS and may obscure recognition of the underlying inflammatory process.

Case Presentation:

A 33-year-old Hispanic male presented after a syncopal episode while taking a hot shower. He reported fever, malaise, night sweats, productive cough, and painful lower extremity ulcerations. On examination, he was febrile with left ankle swelling and erythema nodosum. Laboratory studies revealed leukocytosis and elevated inflammatory markers. There were normal calcium and angiotensin-converting enzyme (ACE) levels. An elevated 1,25-dihydroxy to 25-hydroxy vitamin D ratio suggested granulomatous inflammation. Chest imaging demonstrated massive bilateral hilar and mediastinal lymphadenopathy with scattered pulmonary nodules, raising strong concern for lymphoma. Bronchoscopy was inconclusive, showing only reactive lymphoid changes. Given persistent systemic inflammation and bulky lymphadenopathy, malignancy remained a leading differential. A subsequent mediastinoscopic lymph node biopsy revealed noncaseating granulomas without malignant cells, confirming sarcoidosis. The patient was treated with prednisone and naproxen, with rapid resolution of fever, joint pain, and rash.

Discussion:

This case illustrates syncope as an uncommon presenting feature of Löfgrensyndrome and underscores the diagnostic complexity of sarcoidosis when clinical findings overlap with lymphoma. Although elevated ACE levels are often associated with sarcoidosis, normal ACE levels should not lower clinical suspicion, as they may be normal in up to 40% of cases. In this patient, the elevated vitamin D ratio was the most reliable biochemical marker of granulomatous disease. Despite the presence of the classic clinical triad of erythema nodosum, arthritis, and bilateral hilar lymphadenopathy, the extensive

mediastinal involvement mimicked lymphoma and necessitated tissue biopsy for diagnosis. Sarcoidosis is less commonly reported in Hispanic individuals, making this presentation particularly notable. Clinicians should maintain a high index of suspicion for sarcoidosis when the classic triad is present, despite inconclusive laboratory results or when malignancy is suspected.

Keywords: bilateral hilar lymphadenopathy, pulmonary sarcoidosis, Löfgrensyndrome, lymphoma

Medical Student Clinical Vignette

PRIMARY LOW-GRADE LEIOMYOSARCOMA OF THE BREAST: A CASE REPORT

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BACKGROUND: Primary leiomyosarcoma of the breast is a rare malignant mesenchymal neoplasm, representing one of the least common subtypes of primary breast sarcoma. Owing to the limited number of reported cases, evidence-based recommendations regarding diagnosis, surgical margins, adjuvant therapy, and long-term surveillance remain poorly defined. We present a case of a screening-detected low-grade breast leiomyosarcoma that highlights the diagnostic limitations of core needle biopsy and the importance of complete surgical excision for definitive diagnosis.

CASE PRESENTATION: A 62-year-old postmenopausal woman presented after an abnormal screening mammogram revealed a 10 mm nodular opacity in the left breast. Targeted ultrasound demonstrated a 9 x 8 x 7 mm well-circumscribed hypoechoic mass classified as BI-RADS 4. Ultrasound-guided core needle biopsy demonstrated an atypical smooth muscle neoplasm with mild nuclear pleomorphism, low mitotic activity, and a rare atypical mitotic figure. Immunohistochemical staining was positive for desmin, smooth muscle actin, estrogen receptor, and progesterone receptor. Because low-grade leiomyosarcoma could not be excluded, the patient underwent wire-localized lumpectomy. Final pathology revealed a 1.2 cm low-grade leiomyosarcoma (FNCLCC grade 1) composed of atypical spindle cells arranged in intersecting fascicles with marked nuclear atypia, a mitotic rate of six mitoses/mm2, focal infiltration into the surrounding adipose tissue, and no tumor

necrosis. Immunohistochemical staining was positive for smooth muscle actin, desmin, caldesmon, and estrogen receptor and negative for ALK, S100, and pancytokeratin. Surgical margins were negative by at least 5 mm, and regional lymph nodes were not sampled.

DISCUSSION: This case illustrates several diagnostic challenges associated with primary breast leiomyosarcoma. Despite malignant histopathology, the lesion presented as a small, asymptomatic mass with benign-appearing imaging characteristics. Core needle biopsy suggested an atypical smooth muscle neoplasm but was insufficient for definitive diagnosis. These findings underscore the importance of maintaining suspicion for malignancy despite benign-appearing imaging and demonstrate the limitations of core needle biopsy in spindle cell lesions. The rarity of this malignancy continues to limit the development of evidence-based recommendations regarding surgical margins, postoperative surveillance, and the role of adjuvant therapy.

CONCLUSION: Primary breast leiomyosarcoma can present with benign-appearing imaging findings and indeterminate core needle biopsy results. This case demonstrates that complete surgical excision is essential for establishing a definitive diagnosis when core needle biopsy findings are inconclusive for spindle cell neoplasms.

Medical Student Clinical Vignette

Pseudo-Thrombotic Microangiopathy as the Initial Presentation of Severe Vitamin B12 Deficiency Secondary to Seronegative Pernicious Anemia

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Introduction:

Vitamin B12 deficiency is a well-established cause of macrocytic anemia and in severe cases, may cause ineffective erythropoiesis and intramedullary hemolysis mimicking thrombotic microangiopathies (TMA) such as thrombotic thrombocytopenic purpura (TTP) or hemolytic uremic syndrome (HUS). Etiologies include malnutrition, parasitic infections and gastrointestinal malabsorptive disorders. Pernicious anemia secondary to autoimmune atrophic gastritis is among the most common causes and is typically associated with autoantibodies. However, seronegative disease is increasingly recognized.

Here we present a rare case of pseudo-TMA secondary to severe vitamin B12 deficiency caused by seronegative pernicious anemia.

Case Presentation:

A 59-year-old man with vitiligo presented with several months of progressive dyspnea and fatigue. Examination revealed pallor and vitiligo without neurological deficits.

Laboratory evaluation showed pancytopenia with profound macrocytic anemia (hemoglobin 5.3 g/dL, mean corpuscular volume 114.2 fL), thrombocytopenia, markedly elevated lactate dehydrogenase (LDH), undetectable haptoglobin, indirect hyperbilirubinemia, negative Coombs test and schistocytes on peripheral smear, raising concerns for TMA. Rare immature myeloid forms also prompted consideration of myelodysplastic syndrome (MDS).

Further evaluation revealed an undetectable vitamin B12 level with markedly elevated methylmalonic acid and homocysteine, confirming severe cobalamin deficiency. Pseudo-TMA secondary to profound B12 deficiency was hence strongly suspected. Daily intramuscular cyanocobalamin was initiated on hospital day 1. The patient also received three units of packed red blood cell transfusions with an appropriate increase of hemoglobin to 8.2 g/dL. Given his history of vitiligo and absence of dietary risk factors, pernicious anemia was suspected despite negative anti-parietal cell and anti-intrinsic factor antibodies. A subsequent esophagogastroduodenoscopy (EGD) demonstrated

chronic atrophic autoimmune gastritis with focal intestinal metaplasia. Helicobacter pylori staining was negative.

ADAMTS13 activity and infectious studies were unremarkable. Serial laboratory testing showed stable blood counts without further transfusion requirements, progressive LDH decline, and appropriate reticulocytosis. The patient was discharged on hospital day 4 with outpatient follow-up arranged.

Discussion:

Severe vitamin B12 deficiency can mimic TMA and should be considered in patients presenting with neurological symptoms, microangiopathic hemolytic anemia, and thrombocytopenia as prompt recognition can prevent unnecessary treatment with plasma exchange and high-dose corticosteroids for presumed TTP/HUS. Serial hemolytic markers are useful for monitoring response to parenteral vitamin B12 therapy. LDH is typically the earliest and most rapidly responsive marker and may help guide discharge planning in conjunction with clinical recovery.

Careful history and physical examination remain essential for identifying the underlying etiology of B12 deficiency. Pernicious anemia secondary to autoimmune atrophic gastritis is among the most common etiologies of cobalamin deficiency and is typically associated with anti-parietal cell antibodies and anti-intrinsic factor antibodies, although seronegative disease is increasingly recognized. Therefore, when clinical suspicion remains high despite negative serologies, EGD with gastric biopsies should be pursued, when feasible, to confirm the diagnosis and facilitate appropriate long-term surveillance.

Medical Student Clinical Vignette

Blamed but Not Noncompliant: Refractory Hypothyroidism with Recurrent Decompensation Secondary to Severe Levothyroxine Malabsorption

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Introduction: Oral levothyroxine is the mainstay of hypothyroidism management. Although absorbed in the jejunum and ileum, its uptake depends on gastric acidity, motility, and emptying, which govern tablet dissolution. When patients remain refractory despite high doses, gastrointestinal malabsorption is an emerging and often overlooked cause. We describe a patient repeatedly labeled nonadherent who was ultimately found to have profound, objectively confirmed levothyroxine malabsorption driven by a convergence of gastrointestinal conditions.

Case Description: A 51-year-old woman with type 2 diabetes mellitus complicated by gastroparesis, chronic pancreatitis, and IBS-C with chronic constipation developed severe hypothyroidism following total thyroidectomy for multinodular goiter. Despite supratherapeutic oral levothyroxine (totaling 3,000 mcg/week) plus liothyronine 20 mcg daily, she remained biochemically and clinically hypothyroid, presenting with profound fatigue, weight gain, cold intolerance, worsening constipation, and bilateral lower extremity edema. Admission laboratory studies demonstrated severe hypothyroidism with TSH 110 mIU/L, free T4 <0.1 ng/dL, and total T3 12 ng/dL. She ultimately received intravenous levothyroxine after failure of escalating supervised oral therapy. Failure of oral therapy prompted an evaluation for impaired absorption. Celiac serologies were negative. A supervised levothyroxine absorption test using a 1,500-mcg oral loading dose demonstrated only 2.7% absorption (expected ≥60%), objectively confirming severe levothyroxine malabsorption. Esophagogastroduodenoscopy revealed gastritis with retained gastric contents consistent with gastric stasis. Following IV treatment, she was transitioned to liquid levothyroxine but continued to demonstrate an inadequate biochemical response. She is currently being considered for subcutaneous levothyroxine therapy.

Discussion: Refractory hypothyroidism is most often attributed to nonadherence; roughly 21% of treated patients have an elevated TSH, making noncompliance a reflexive and often premature explanation. Her malabsorption stemmed not from a classic mucosal etiology but from converging gastric and pancreatic conditions: diabetic gastroparesis delaying

emptying and tablet dissolution; gastritis with stasis altering gastric pH; and chronic pancreatitis leading to pancreatic insufficiency and altered secretions. Together these overlapping disturbances plausibly account for her strikingly low absorption despite negative celiac serology, illustrating that functional and motility-related gastrointestinal disease alone can produce clinically significant levothyroxine malabsorption. When oral therapy fails, liquid or soft-gel formulations that bypass dissolution may improve uptake, yet she remained refractory even to liquid therapy, underscoring her malabsorption severity. Because repeated IV therapy is impractical for chronic outpatient care, subcutaneous levothyroxine—which can achieve sustained euthyroidism, though cost and access remain barriers—represents a promising alternative. This case underscores the value of objective absorption testing to distinguish true malabsorption from noncompliance before escalating to parenteral therapy.

Medical Student Clinical Vignette

A Wolf in Neurologic Clothing: Occult Signet Ring Cell Carcinoma Mimicking Primary Neurologic Disease

Mukund Pasapuleti

Brittany Butcher, MBA; Shaikh Sufyan, MD; Chris Elsayad, MD

Occult colorectal carcinoma can rarely manifest as rapidly progressive neurologic decline, particularly when extensive neurologic and infectious evaluations fail to reveal the underlying diagnosis.

A 39-year-old woman with IBD diagnosed nine years earlier was brought to the ED for acute unresponsiveness after two weeks of intermittent headaches and vomiting. Despite multiple prior evaluations and extensive testing at outside facilities, no unifying diagnosis had been identified before presentation, but prednisone was prescribed for presumed migraine. On arrival, she was minimally responsive, moaning, incontinent, afebrile, hemodynamically stable, and unable to follow commands. Examination showed sluggish pupils without meningeal signs and spontaneous movement of all extremities. Initial studies showed leukocytosis, thrombocytosis, anemia, and an elevated lactate. Head CT and Head and Neck CTA showed no acute pathology. Lumbar puncture, urine toxicology, chest radiography, and electrocardiography were unrevealing. She was admitted to the medical ICU for presumed CNS infection or seizure-related encephalopathy and was treated empirically while neurology and infectious disease evaluations were pursued. By day 3, her mental status improved, creating brief reassurance that the working diagnosis of meningitis or encephalitis was appropriate. However, improvement was short-lived. After transfer to the medical floor, she developed visual complaints and neck pain, followed by progressive bilateral vision loss, prolonged pupillary dilation, dysphagia, and generalized weakness. As her symptoms evolved, the differential broadened to include neuromyelitis optica, autoimmune encephalitis, posterior reversible encephalopathy syndrome, reversible cerebral vasoconstriction syndrome, vasculitis, and functional neurologic disorder. Corticosteroids were initially deferred, then later administered with transient improvement in vision, swallowing, and ESR. Within 48 hours, her vision and dysphagia worsened again, IVIG was started, and her respiratory status declined. Imaging was expanded due to clinical deterioration, anemia, and hypoxemia. CT of the abdomen and pelvis revealed cecal wall thickening and ascites with extensive retroperitoneal and mesenteric lymphadenopathy. CEA was mildly elevated, and lymph node biopsy demonstrated mucinous signet-ring cell carcinoma, revealing a previously occult colorectal malignancy that had remained clinically hidden despite extensive investigation.

She was transferred to a tertiary center, where her course was complicated by cardiac arrest, anoxic brain injury, and transition to DNR/DNI.

This case illustrates how colorectal carcinoma may remain clinically concealed while presenting as a rapidly progressive neurologic syndrome, evading diagnosis despite extensive infectious, neurologic, and systemic evaluation. The final lymph node biopsy demonstrated mucinous signet-ring cell carcinoma, an aggressive colorectal cancer subtype often associated with younger age at presentation, advanced-stage disease, and poor prognosis. In patients with IBD, persistent diagnostic uncertainty, unexplained systemic abnormalities, and rapidly evolving neurologic symptoms should prompt reconsideration of an underlying occult malignancy and possible paraneoplastic disease when extensive investigations fail to identify a primary cancer.

Medical Student Clinical Vignette

ANORECTAL EPSTEIN-BARR VIRUS–POSITIVE DIFFUSE LARGE B-CELL LYMPHOMA MIMICKING PERIANAL INFECTION

Shaan Patker

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INTRODUCTION: Epstein-Barr virus (EBV)-positive diffuse large B-cell lymphoma (DLBCL) may arise in the setting of impaired immune surveillance. Anorectal involvement is rare and may resemble cellulitis, abscess, ulceration, or hemorrhoidal disease, delaying tissue diagnosis. We report EBV-positive anorectal DLBCL presenting as persistent perianal ulceration during prolonged immunosuppressive therapy.

CASE PRESENTATION: A 69-year-old woman with type 2 diabetes mellitus and presumed systemic lupus erythematosus and rheumatoid arthritis, for which she had received four immunosuppressive agents, presented with one month of rectal pain, constipation, and worsening perianal ulceration. She was admitted for suspected perianal cellulitis. Examination revealed abnormal ulcerated perianal tissue. Previous imaging and clinical assessments had attributed the lesions to infection; however, persistent symptoms prompted biopsy of an anorectal mass. Histopathologic examination demonstrated large atypical lymphoid cells expressing CD20, CD79a, PAX5, MUM1, BCL6, and BCL2. CD10 and cytokeratin were negative. The Ki-67 proliferation index was approximately 85%, and Epstein-Barr virus–encoded RNA in situ hybridization was strongly positive, supporting EBV-positive DLBCL. Cross-sectional imaging showed no widespread nodal disease. The patient was referred for positron emission tomography/computed tomography staging and oncologic treatment planning.

DISCUSSION: Anorectal lymphoma can closely mimic common infectious and inflammatory disorders. In patients receiving immunosuppressive therapy, persistent, atypical, or treatment-refractory anorectal lesions should prompt early diagnostic reconsideration. Clinical examination and imaging may not reliably distinguish infection from malignancy, making tissue biopsy essential.

CONCLUSION: Failure of presumed perianal infection to improve should prompt early biopsy, particularly in patients with impaired immune surveillance. Timely tissue diagnosis

can prevent repeated ineffective treatment and expedite care for rare extranodal lymphoma.

Medical Student Clinical Vignette

GEMELLA HAEMOLYSANS BACTEREMIA WITH AN AORTIC VALVE MASS: INFECTION OR THROMBUS?

Shaan Patker

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INTRODUCTION: Gemella haemolysans is a gram-positive facultative anaerobic coccus of the oropharyngeal and gastrointestinal flora that rarely causes bacteremia or infective endocarditis. In patients with advanced malignancy, a valvular mass may represent either infective endocarditis or nonbacterial thrombotic endocarditis. We describe G. haemolysans bacteremia accompanied by an aortic-valve mass in a patient with metastatic lung cancer.

CASE PRESENTATION: A 70-year-old man with stage IV metastatic non–small cell lung cancer, coronary artery disease after percutaneous coronary intervention, hypertension, hypothyroidism, and deep venous thrombosis presented with two weeks of progressive dyspnea. He was tachycardic and hypoxemic, with an oxygen saturation of 89% on room air. Chest radiography demonstrated near-complete opacification of the right hemithorax with mediastinal shift. Computed tomography angiography revealed a large right-lung mass, extensive consolidation, and a large pleural effusion containing a thick rind and gas pockets concerning for empyema. Blood cultures grew G. haemolysans. Transthoracic echocardiography demonstrated a mobile 1.1 × 0.4-cm amorphous mass on the ventricular surface of the aortic valve, raising concern for vegetation versus thrombus. Intravenous cefepime and vancomycin were initiated, with metronidazole added for anaerobic coverage. Given his advanced malignancy and do-not-resuscitate/do-not-intubate status, the patient and his family elected comfort-focused care and home hospice. Antibiotics were transitioned to oral amoxicillin-clavulanate. Transesophageal echocardiography and chest-tube drainage were not pursued.

DISCUSSION: G. haemolysans bacteremia with a mobile aortic-valve mass raised concern for infective endocarditis. However, the patient’s metastatic malignancy and history of venous thrombosis also increased the plausibility of nonbacterial thrombotic endocarditis. Transesophageal echocardiography and further microbiologic evaluation would ordinarily

help distinguish these entities. In this case, diagnostic certainty was limited because additional invasive testing was inconsistent with the patient's goals of care.

CONCLUSION: A valvular mass in a patient with bacteremia and advanced malignancy should not automatically be attributed to either infection or thrombosis. This case highlights the diagnostic uncertainty between infective and nonbacterial thrombotic endocarditis and the importance of balancing further investigation with procedural burden and patient-centered goals.

Medical Student Clinical Vignette

Acquired Hemophilia A Presenting as Lower Gastrointestinal Bleeding in Elderly Female

Sofia Perez

Anthony Corsi MD

Introduction: Hemophilia A is a bleeding disorder caused by autoantibodies directed against Factor VIII. Patients typically present with spontaneous bleeding and isolated prolongation of activated partial thromboplastin time (aPTT) and without family history of bleeding disorders. This case demonstrates acquired hemophilia A presenting as refractory lower gastrointestinal bleeding requiring massive transfusion and escalation of care.

Case Description: 70-year-old female with past medical history of multiple sclerosis (MS) complicated by optic neuritis, prior CVA, CKD stage III, HLD, and HTN presented after a fall and intermittent episodes of blood in stool over the past 3-4 months. Vitals on admission: Temp 99.4F, BP 114/60, HR 91, O2 Sat 100% on RA. Labs: WBC 9.70, Hgb 11.2, Plt 157, PT 8.1, aPTT 69.6, INR <0.90. Past surgical history included hemorrhoidectomy several years before complicated by rectal artery pseudoaneurysm formation. Patient was not on any home medications. Esophagogastroduodenoscopy showed mild esophagitis without bleeding, and sigmoidoscopy revealed abundant stool and blood with active rectal bleeding, without identification of source. CTA of the abdomen and pelvis revealed active arterial bleed in the rectum and left-sided colonic diverticulosis without diverticulitis. Due to active rectal bleeding, the patient received multiple units of PRBC, FFP, and platelets. 24 hours after her initial rectal bleeding, patient became hypotensive and massive transfusion protocol was activated. Further work-up revealed vWF Ag of 192% [63-172%], vWF activity of 273% [43-126%], factor VIII activity of 1.1% [>45%], factor IX activity of 104.2 [>52%], and factor XI activity of 50.4% [>45%]. Mixing studies revealed aPTT 100% of 59.8s, aPTT 50/50 of 34.6s, and aPTT 50/50 2hr of 55.4s. The patient was diagnosed with acquired hemophilia A and began on prednisone 1mg/kg QD and recombinant factor VIII. Repeat colonoscopy revealed arterial bleeding at level of anal verge; sutures were applied to the bleeding site, and Blakemore balloon was placed however these interventions failed to control the bleeding. IR could not embolize due to impaired coagulation. She received >20 blood products and transferred for higher level of care. After this admission she was started on increased prednisone (2mg/kg) and 4 cycles of rituximab. Bleeding slowly stopped, factor VIII level increased and she was eventually discharged.

Discussion: Acquired hemophilia A is due to autoantibodies against Factor VIII that either inhibit function or increase clearance of the factor. Most patients are older adults (median age ~73 yo) with uncontrolled bleeding, elevated aPTT not corrected by incubated mixing study and have reduced Factor VIII activity. An estimated 30-50% of patients with acquired hemophilia A have an underlying condition such as malignancy and autoimmune disorder. Our patient met the expected age range and has a past medical history of an autoimmune disorder (multiple sclerosis).

Medical Student Clinical Vignette

Numerous suspicious-looking Nipple Calcifications: A Radiological Imaging Case Study of Recurrent Breast Cancer

Uday Sappati

Dr. Michelle Teresa Wiciak

Dr. Shailaja Reddy Sappati

Introduction: In the United States of America, breast cancer is the second most common cancer in women. Cancer rates vary between races and ethnicities, highlighting a significant healthcare disparity, especially in Black/African American women. Typically, many cases can be detected by palpable breast lumps, but many early diseases are caught through routine mammogram screening. Sometimes, cases present without lumps or other symptoms, underscoring the importance of regular mammograms, especially for high-risk individuals.

Case Report: A 71-year-old African American woman presents for her yearly routine screening mammogram. She has a past medical history of right-sided breast cancer in the upper outer quadrant that was diagnosed in 2004 and treated with lumpectomy, axillary lymph node dissection, chemotherapy, and radiation with no evidence of recurrence. Previous mammograms and available ultrasounds showed benign findings. Her most recent mammogram and ultrasound showed new suspicious right-sided calcifications in the retroareolar region, without a discrete hypoechogenic mass. The image was deemed BI-RADS 4, suspicious for DCIS. A biopsy was performed of the calcification and determined to be positive for invasive mammary carcinoma and DCIS (intermediate grade) with ER+/PR-/HER2- status and a histology grade 1. Mastectomy is the recommended treatment plan for this patient.

Discussion: This case provides various educational points that providers and students should know and consider for all patients. For one, routine mammograms are critical in the detection of breast cancer, especially in patients who have a higher likelihood of getting breast cancer. Despite 20 years of remission, a recurrence of breast cancer is still likely, although the risk is lower than in the first 5 years post-treatment. Furthermore, it emphasizes the importance of recognizing that breast cancer does not always present with a palpable lesion, and can have atypical presentations, such as this case, which is more consistent with DCIS. Lastly, this patient is an African-American woman, and rates of late diagnosed breast cancers are higher in this population, contributing to the poorer outcomes, particularly mortality. Some late diagnoses may be due to limited access to

healthcare facilities, transportation, education, and health insurance, ultimately affecting screening and follow-up care. Thus, working with patients to keep up with routine screening, like this patient, can help catch breast cancer early and promote better health outcomes and quality of life.

Conclusion: Despite breast cancer being associated with palpable masses, it is possible to have breast cancer without overt signs and symptoms. Thus, it is important to ensure that patients, especially high-risk patients and those in populations with demonstrated healthcare inequities, receive their routine mammogram screenings.

Medical Student Clinical Vignette

A Multisystem Masquerade: Bartonella Prosthetic Valve Endocarditis Beyond the Heart

Shruthi Suresh

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Introduction: Bartonella species are an underrecognized cause of culture-negative infective endocarditis (IE), particularly in patients with prosthetic valves. Diagnosis is often delayed due to negative standard blood cultures and nonspecific systemic features. We describe a case of Bartonella henselae prosthetic valve endocarditis presenting with pancytopenia and ANCA-associated vasculitis with renal involvement, diagnosed using microbial cell-free DNA sequencing.

Case Description:

History: A 72-year-old man with severe aortic stenosis status post transcatheter aortic valve replacement (valve-in-valve), atrial fibrillation, and coronary artery disease presented with exertional dyspnea and 20-lb unintentional weight loss over one month.

Physical exam: Afebrile, no new murmur, no splenomegaly, and no peripheral stigmata of endocarditis.

Labs: Pancytopenia (hemoglobin 7.1 g/dL, platelets 51 K/µL) and worsening acute kidney injury (creatinine 2.8 to 4.2 mg/dL) with hyperkalemia requiring hemodialysis. Urinalysis showed active sediment with hematuria and cellular casts. Four blood cultures and a urine culture were negative. Bone marrow biopsy showed a hypercellular marrow without evidence of marrow infiltration.

Diagnostic workup: Transesophageal echocardiogram revealed a vegetation on the bioprosthetic aortic valve, redirecting evaluation toward endocarditis despite negative cultures. P-ANCA, ANA, and rheumatoid factor were positive with low C3/normal C4, while anti-dsDNA, ENA, and anti-GBM antibodies were negative, supporting endocarditis-associated ANCA vasculitis over primary connective tissue disease; renal biopsy was deferred given instability, thrombocytopenia, and the fact that management was unlikely to change. HIV screening immunoassay was reactive with undetectable HIV-1 RNA and no confirmatory differentiation testing performed, felt to be a discordant result of uncertain significance possibly related to polyclonal B-cell activation from chronic infection. Microbial cell-free DNA testing identified Bartonella henselae (128,558 copies/mL),

confirmed by markedly elevated B. henselae IgG (>1:65536) and cross-reactive B. quintana IgG (>1:2048), establishing the diagnosis of Bartonella prosthetic valve endocarditis.

Treatment: Cardiac surgery was considered but deemed not feasible given surgical complexity. He was treated with doxycycline and rifampin. Serial serology downtrended and repeat echocardiogram showed resolution of the vegetation; thrombocytopenia improved, but renal function did not recover and he remained hemodialysis-dependent.

Discussion: This case highlights Bartonella as an important, likely underdiagnosed cause of culture-negative prosthetic valve endocarditis, capable of presenting without classic peripheral stigmata and with unexplained pancytopenia and renal failure as the initial clues rather than cardiac symptoms. It underscores the association between Bartonella IE and ANCA-associated vasculitis mediated by circulating immune complexes rather than direct renal invasion. Microbial cell-free DNA sequencing was instrumental in establishing the diagnosis when cultures and serology alone were ambiguous. Clinicians should maintain a high index of suspicion for Bartonella in culture-negative IE with multisystem involvement, as early recognition may allow timely antimicrobial therapy and potentially prevent irreversible renal and hematologic damage.

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Poster Presentations

Medical Student Research

Targeting the YAP/TAZ-TEAD Axis as a Novel Therapeutic Treatment for Anaplastic Thyroid Cancer

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Background:

Among all thyroid cancers, approximately 5% undergo de-differentiation, leading to a loss of sodium-iodide symporter (NIS) expression, resistance to radioactive iodine (RAI), and poor clinical outcomes. Anaplastic thyroid cancer (ATC) is the most aggressive de-differentiated thyroid malignancy with a median life expectancy of 3-5 months. The transcriptional co-activators yes-associated protein (YAP) and transcriptional coactivator with PDZ-binding motif (TAZ) are downstream effectors of the Hippo signaling pathway that regulate gene expression through TEAD transcription factors. Because YAP/TAZ activity has been implicated in thyroid cancer progression and regulation of NIS, the YAP/TAZ-TEAD axis represents a promising therapeutic target. Multiple TEAD inhibitors are currently in Phase I and II clinical trials in other cancer contexts. We hypothesized that inhibition of YAP/TAZ-TEAD would reduce ATC cell viability and promote thyroid marker re-differentiation.

Methods:

Human ATC 8505C cells were treated with the TEAD inhibitor MGH-CP1 (0-20 µM) or transfected with pooled YAP/TAZ siRNA. TEAD transcriptional activity was measured using a dual-luciferase reporter assay. Cell viability was assessed by CCK8 metabolic assays and Hoescht nuclear counts after 96 hours of treatment. Western blots evaluated cancer cell proliferation (PCNA) and apoptosis (cleaved caspase-3 and PARP) while expression of YAP/TAZ target genes and thyroid differentiation markers, such as the SLC5A5 gene that encodes NIS, were quantified by RT-qPCR. Statistics were calculated using GraphPad Prism by running one-sample t tests, unpaired t tests, or one-way ANOVA analyses with statistical significance defined as $p \leq 0.05$.

Results:

Pharmacologic TEAD inhibition significantly reduced YAP/TAZ transcriptional activity. Both pharmacologic inhibition and YAP/TAZ knockdown significantly decreased 8505C cell

viability after 96 hours, demonstrating that YAP/TAZ-TEAD signaling is required for ATC cell viability. Reduced viability was associated with decreased expression of PCNA without detectable levels of cleaved caspase-3 or PARP. This indicates that the reduction in viability was due to impaired cellular proliferation rather than apoptosis. Furthermore, both pharmacologic and genetic inhibition significantly reduced expression of YAP/TAZ target genes but did not significantly alter expression of thyroid differentiation markers, including SLC5A5, suggesting that YAP/TAZ inhibits tumor growth independent of effects on thyroid differentiation.

Conclusion:

The YAP/TAZ-TEAD axis is a critical regulator of anaplastic thyroid cancer cell viability. Although YAP/TAZ inhibition did not restore thyroid differentiation or SLC5A5 expression, it impaired tumor cell growth by reducing proliferation. These findings support TEAD inhibition as a promising therapeutic avenue for patients with anaplastic thyroid cancer for whom re-differentiation therapies have proven unsuccessful. This study effectively broadens the potential clinical utility of Hippo pathway-directed therapies in a malignancy with few effective treatment options.

Medical Student Research

IMPACT OF REFERRING CLINIC TYPE AND CLINICAL FACTORS ON OUTPATIENT COLONOSCOPY PREPARATION QUALITY: A RETROSPECTIVE ANALYSIS

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Purpose

We aimed to determine whether referral from trainee-led or other subspecialty clinics independently predicts inadequate bowel preparation (IBP) in outpatient colonoscopy.

Methods

We performed a retrospective cohort study of 736 randomly selected patients undergoing outpatient colonoscopies at a single academic center from 2020 to 2025. Patients were stratified by referral source: gastroenterology (GI) attending clinic, GI fellow clinic, internal medicine (IM) attending clinic, IM resident clinic, and other clinics. Adequate preparation was defined as complete mucosal visualization with or without intraprocedural cleansing; IBP was defined as suboptimal visualization despite cleansing necessitating a shortened surveillance interval. Multivariable logistic regression identified independent predictors of IBP, adjusting for demographics, insurance, comorbidities (opioid use, constipation, tricyclic antidepressant use), prior colonoscopies, and preparation formulation.

Results

Among 736 patients, 23.1% of referrals from the GI fellow clinic had IBP, compared with 11.8% from the GI attending clinic. Compared with the GI attending clinic, the GI fellow clinic served more Spanish-speaking (21.2% vs. 5.1%) and Medicaid/uninsured patients (28.9% vs. 7.5%), and prescribed polyethylene glycol 3350 with electrolytes-based preparation more frequently (21.2% vs. 12.8%). After adjustment, referral from the GI fellow clinic was independently associated with higher odds of IBP (aoR 2.25; 95% CI 1.03–4.88; P

= 0.04). Male sex (aOR 2.34; P < 0.01) and constipation (aOR 3.03; P < 0.01) were also associated with IBP. Referral from IM clinics did not reach statistical significance.

Conclusions

Referral from the GI fellow clinic was independently associated with IBP, suggesting clinic-specific workflows or patient education may influence outcomes beyond demographics. Differences among IM clinics may have gone undetected due to limited sample size. The higher proportion of Spanish-speaking patients in the GI fellow clinic may represent a modifiable target, as guidelines recommend language-concordant instructions. Future interventions should standardize patient education in trainee-led clinics and develop tailored bowel preparation protocols for high-risk patients.

Medical Student Research

THE HEART OF THE MATTER: IMPROVING PROVIDER AWARENESS OF ASCVD RISK IN PATIENTS WITH SUBSTANCE USE DISORDER

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BACKGROUND

Cardiovascular disease (CVD) remains a significant cause of morbidity and mortality among individuals with substance use disorder (SUD). Use of tobacco, alcohol, cannabis, cocaine, and amphetamines has been associated with up to a 9-fold increase in premature atherosclerotic cardiovascular disease (ASCVD) and cardiovascular mortality. While cardiovascular mortality rates have declined in the general population, deaths in which substance use was identified as a contributing factor have increased by approximately 4% annually over the past two decades, with recent estimates demonstrating acceleration to 6.2% annually. Despite this growing burden, cardiovascular risk assessment and preventive management remain underrecognized in many behavioral health settings.

OBJECTIVE

The objective of this project was to improve ASCVD risk assessment, risk stratification, and implementation of evidence-based preventive cardiovascular care among patients with SUD through a targeted educational intervention designed to increase provider knowledge and awareness of cardiovascular risk.

METHODS

This quality improvement educational project was conducted within the Addiction Medicine Service at BronxCare Hospital and included multidisciplinary healthcare providers, including psychiatry residents, nurse practitioners, and physician assistants. Participants completed a 16-item pre-intervention survey assessing knowledge of ASCVD prevalence among individuals with SUD, mechanisms underlying increased cardiovascular risk, ASCVD risk assessment and stratification, and evidence-based preventive therapies. Survey content was reviewed by addiction psychiatry faculty for clinical relevance and

educational validity. Participants then completed a 20-slide educational session covering the epidemiology of ASCVD in SUD, cardiovascular risk mechanisms, ASCVD risk assessment using the American Heart Association PREVENT calculator, risk stratification, and preventive management strategies. A post-intervention survey was administered following the educational session.

RESULTS

Thirty participants completed both the pre- and post-intervention surveys and were included in the matched analysis. Overall knowledge scores improved significantly following the educational intervention (mean score 6.23 vs. 10.10, p = 1.21 × 10^{-9}). Using a multi-objective mapping approach, statistically significant improvements were observed across all educational domains. Participants also reported significant improvements in screening frequency and confidence in ASCVD management. Mean screening frequency increased from "sometimes" (2.1) to "often" (3.4) following the intervention (p = 0.000005), while provider confidence improved from "somewhat comfortable" (1.87) to "very comfortable" (2.5) (p = 0.000288).

CONCLUSIONS

This study demonstrates that a targeted educational intervention can significantly improve provider knowledge, ASCVD screening practices, and confidence in cardiovascular risk assessment and preventive management among patients with SUD. These findings support the incorporation of cardiovascular risk education into addiction medicine and behavioral health training programs and highlight the potential role of provider education in improving preventive cardiovascular care for this high-risk population.

LIMITATIONS

Limitations include the small sample size, single-center design, short-term assessment period, and reliance on self-reported behavioral outcomes.

Medical Student Research

Exercise Intolerance in Ehlers Danlos Syndrome and Hypermobility Spectrum Disorder is Associated with Greater Multisystem Symptom Burden Despite Similar Exercise Stress-Test Findings

Naman Kapoor

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Context:

Exercise intolerance is a disabling manifestation of Ehlers-Danlos Syndrome (EDS) and Hypermobility Spectrum Disorder (HSD). Both are associated with dysautonomia, chronic pain, deconditioning, and other multisystem manifestations that may reduce exercise capacity. The purpose of this study is to determine whether multisystem symptom burden predicts exercise intolerance in patients with EDS/HSD and whether exercise-intolerant patients demonstrate impaired EST performance.

Methods:

A retrospective review of patients diagnosed with EDS/HSD between January 2019 and November 2024 at the Long Island Heart Rhythm Center was conducted. Patients were grouped by the presence or absence of exercise intolerance based on chart documentation. A 10-point multisystem symptom burden score (presyncope, syncope, weakness/fatigue, headaches, edema, palpitations, shortness of breath, vision changes, chest tightness/pain, and nausea) was compared between groups. Logistic regression assessed whether symptom burden predicted exercise intolerance. Secondary analyses compared abnormal cardiac EST results, maximum heart rate, peak systolic and diastolic blood pressure, exercise duration, and peak metabolic equivalents (METs) among patients with stress-test data. Continuous variables were reported as mean ± SD or median [IQR] and categorical variables as counts (%). Mann-Whitney U and chi-square tests were used for group comparisons; $p < 0.05$ was considered statistically significant.

Results:

164 EDS or HSD patients were identified (age 37.1 ± 12.8 years; [9.1%] male, [90.9%] female). Of these, 74 patients reported exercise intolerance, and 90 did not. Patients with exercise intolerance had higher total symptom burden scores than those without (6.0 [4.0-7.0] vs. 4.0 [3.0-6.0], U=2226.5, $p<0.001$). Exercise-intolerant patients more frequently reported nausea ([50.0%] vs. [20.0%], $p < 0.001$; OR 4.00, 95% CI 2.01–7.97),

weakness/fatigue ([71.6%] vs. [41.2%], p = 0.011; OR 2.31, 95% CI 1.20–4.44), and headaches ([37.8%] vs. [20.0%], p = 0.011; OR 2.43, 95% CI 1.21–4.89). Shortness of breath (p=0.052) and vision changes (p=0.067) trended toward statistical significance. Presyncope, syncope, edema, palpitations, and chest tightness/pain were not significantly associated with exercise intolerance. Each 1-point increase in symptom burden increased the odds of exercise intolerance by 34% after adjustment for age, sex, postural orthostatic tachycardia syndrome diagnosis, and EDS/HSD subtype (adjusted OR 1.34, 95% CI 1.13–1.58, p<0.001). Among 44 patients with available EST results (24 exercise-intolerant and 20 exercise-tolerant), both groups had identical percentages of abnormal stress-test results (25%, p=1.000). Maximum heart rate, blood pressure response, total exercise time, and METs were not statistically significant between groups.

Conclusions:

Self-reported exercise intolerance independently predicts greater multisystem symptom burden in patients with EDS/HSD. Despite this, exercise-intolerant patients demonstrated similar stress-test performance to exercise-tolerant patients. This suggests that conventional exercise stress testing may not adequately capture the mechanisms underlying exercise intolerance or cardiovascular performance in this population. Prospective studies using larger cohorts and more comprehensive physiologic assessments are warranted.

Medical Student Research

FROM MEDICALLY READY TO DISCHARGE READY: AN INTERACTIVE DISABILITY AND TRANSITIONS OF CARE CURRICULUM FOR THIRD YEAR INTERNAL MEDICINE STUDENTS

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Purpose: An interactive, case-based curriculum on disability and transitions of care for third-year internal medicine (IM) students to address educational gaps in disability and transitions of care was developed and implemented from August to October 2026.

Methods: Third-year IM students participated in a 60-minute workshop centered on discharge planning for a patient recovering from a COPD (Chronic Obstructive Pulmonary Disease) exacerbation with mobility limitations. The curriculum involved three interactive stations: 1) mobility and assistive devices, 2) adult inpatient pain management, and 3) selection of appropriate post-acute care settings. The session concluded with a facilitated interdisciplinary debrief with physical medicine and rehabilitation faculty. Curriculum evaluation was conducted using pre- and post-session surveys with 5-point Likert scales to measure learners' frequency of exposure to patients with disabilities, comfort managing the care of patients with disabilities, confidence in discharge planning, and preparedness to utilize rehabilitation resources.

Results: Program implementation and data collection are currently underway. Analysis will compare pre- and post-session survey responses to quantitate changes in learner confidence, preparedness, and understanding of disability and transitions of care.

Conclusions: For patients living with disabilities or those who develop new functional limitations during hospitalization, successful transitions of care depend on more than resolution of the acute illness. Mobility, accessibility, assistive devices, caregiver support, home environment, and access to rehabilitation and post-acute services all influence a patient's ability to recover outside the hospital, maintain functional independence, and successfully return to their daily lives. Despite their importance, disability and transitions

of care remain underrepresented in medical education, leaving many students underprepared to recognize and address the functional barriers that shape discharge planning. Effective discharge planning also requires physicians to recognize and utilize multidisciplinary resources, including physical therapy, occupational therapy, case management, social work, respiratory therapy, and community based services, to develop individualized care plans that are feasible for each patient. This structured, interactive curriculum provides formal education on the functional, environmental, and social factors influencing hospital discharge, addressing gaps in traditional and variable clerkship experiences. It equips students with practical skills to support equitable care transitions, optimize resource utilization, improve continuity of care, and better address the needs of patients with disabilities and other complex discharge plans.

Medical Student Research

ASSOCIATION BETWEEN NOREPINEPHRINE-ENHANCING PSYCHIATRIC MEDICATIONS AND SYMPTOM BURDEN IN PATIENTS WITH POSTURAL ORTHOSTATIC TACHYCARDIA SYNDROME

Emily Lin

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Background:

Postural Orthostatic Tachycardia Syndrome (POTS) is a disease of orthostatic intolerance that is often comorbid with psychiatric conditions, including depression and attention-deficit hyperactivity disorder. Norepinephrine-enhancing psychiatric medications (NEPMs), defined as antidepressants and stimulants that increase synaptic norepinephrine, can be prescribed to treat these conditions. Evidence regarding the effects of NEPMs on POTS symptom burden is limited. NEPMs may worsen orthostatic tachycardia through augmented noradrenergic signaling, while also improving POTS symptoms through enhanced peripheral vasoconstriction. This study aims to determine whether POTS symptom burden differs between patients with POTS taking NEPMs compared to patients with POTS not taking NEPMs.

Methods:

A retrospective matched chart review was conducted using data collected between 2019-2026 from the Long Island Heart Rhythm Center. Twenty POTS patients receiving NEPM therapy were matched 1:1 with twenty POTS patients not receiving NEPMs by age (±10 years), sex, and concomitant POTS medical therapy (beta blockers, midodrine, and ivabradine). POTS symptom burden was quantified by a derived composite symptom score based on overlapping symptom domains from four POTS assessment instruments: Malmö POTS Score, Vanderbilt Orthostatic Symptom Scale, Orthostatic Grading Scale, and COMPASS-31. Symptoms represented in at least three of the four instruments were included in the composite score. The presence of each symptom at the patient's initial POTS clinical evaluation was assigned one point. Cumulative points generated an individual symptom burden score. Evaluated POTS symptoms included cardiovascular, gastrointestinal, neurologic, dermatologic, and constitutional manifestations. NEPMs were defined as psychiatric medications that increase synaptic norepinephrine concentrations

through norepinephrine reuptake inhibition or reverse transport. A two-sided paired samples t-test was calculated with statistical significance set at $p \leq 0.05$.

Results:

Forty POTS patients were identified, 20 taking NEPMs (mean age 29.00 ± 7.40 years; M/F [10%]/[90%]) and 20 not taking NEPMs (mean age 27.95 ± 7.47 years; M/F [10%]/[90%]). Among patients taking NEPMs, medications included amphetamine/dextroamphetamine (n = 9), methylphenidate (n = 5), bupropion (n = 3), nortriptyline (n = 2), amphetamine sulfate (n = 1), dexmethylphenidate (n = 1), and atomoxetine (n = 1); two patients received combination therapy. POTS patients taking NEPMs had a significantly higher composite POTS symptom burden score than patients not taking these medications (5.65 ± 2.87 vs. 4.00 ± 1.97; p = 0.037).

Conclusion:

Use of NEPMs was associated with a significantly higher POTS symptom burden score compared to patients not receiving these medications, suggesting that NEPM therapy may be associated with increased POTS symptom severity. Larger prospective studies are needed to conclusively determine this association and examine the potential confounding effects of comorbid psychiatric conditions and other neurotransmitters modulated by NEPMs, such as serotonin and dopamine, on POTS symptom burden.

Medical Student Research

Trends in Extraintestinal Manifestations Among Hospitalized Adults With Inflammatory Bowel Disease: A National Inpatient Sample Analysis (2018–2022)

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2. Sabrina Iqbal, BS

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Introduction

Extraintestinal manifestations (EIMs) contribute substantially to morbidity in inflammatory bowel disease (IBD), yet contemporary national trends across organ systems remain incompletely characterized. Defining these patterns may clarify the evolving multisystem burden of IBD and inform anticipatory multidisciplinary care.

Methods

We performed a cross-sectional analysis of adult, non-elective IBD hospitalizations in the National Inpatient Sample from 2018–2022. Annual prevalence estimates with 95% confidence intervals were generated for musculoskeletal, dermatologic, ocular, hepatobiliary, venous thromboembolic, renal/urologic, pulmonary, pancreatic, oral, bone/metabolic, neurologic, cardiovascular, and hematologic/immune EIMs. Linear temporal trends were evaluated using survey-weighted logistic regression.

Results

Among more than 350,000 weighted U.S. hospitalizations, the prevalence of any EIM increased from 13.7% in 2018 to 15.3% in 2022 ($P < 0.01$; OR 1.03, 95% CI 1.01–1.05). Hepatobiliary EIMs increased consistently (OR 1.10, 95% CI 1.04–1.16), including primary sclerosing cholangitis (OR 1.09, 95% CI 1.02–1.17) and autoimmune hepatitis (OR 1.13, 95% CI 1.03–1.25). Renal/urologic EIMs also increased, including nephrolithiasis (OR 1.06, 95% CI 1.02–1.11), as did pulmonary manifestations (overall OR 1.09, 95% CI 1.01–1.16; bronchiectasis/chronic bronchitis OR 1.14, 95% CI 1.01–1.28). Oral manifestations increased as well, including aphthous stomatitis (OR 1.10, 95% CI 1.03–1.17). Cardiovascular EIMs, including pericarditis, also increased (OR 1.12, 95% CI 1.02–1.23). Metabolic bone disease declined slightly (OR 0.96, 95% CI 0.92–0.99), while dermatologic, ocular, neurologic, pancreatic, and hematologic/immune EIMs remained largely stable.

Discussion

The overall burden of EIMs among hospitalized adults with IBD increased over the study period, with notable rises in hepatobiliary, renal/urologic, pulmonary, oral, and cardiovascular involvement. These findings suggest an increasing multisystem burden of IBD and highlight the need for proactive, multidisciplinary management. The observed patterns are concordant with emerging data demonstrating increased recognition of hepatobiliary and thromboembolic complications in IBD. In contrast, some non-Western cohorts have reported stable or declining EIM rates, suggesting that geographic variation, evolving disease recognition, differences in clinical practice, and changes in coding over time may influence observed trends.

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Poster Presentations

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Polymyositis Complicated by Antisynthetase Syndrome Presenting with Severe Bulbar and Respiratory Muscle Weakness: A Case Report

Muhammad Moeez Badar

Arjun Jayakumar, Ishan Mahesh Parmar, Aina Mahmood, Shiv Shankar, Indu Meena, Muhammad Moeez Badar, Azouba Gulraiz

Background:

Antisynthetase syndrome (ASS) is a rare autoimmune condition characterized by antibodies directed against aminoacyl-tRNA synthetases and is commonly associated with inflammatory myopathies. Clinical manifestations include myositis, interstitial lung disease, arthritis, Raynaud phenomenon, and mechanic's hands. Diagnosis can be challenging when patients present with atypical features or incomplete syndromic manifestations.

Case Presentation:

A 22-year-old woman with no significant past medical history presented with progressive proximal muscle weakness, fatigue, and diffuse myalgias for six weeks. Symptoms initially affected the lower extremities and later involved the upper limbs, resulting in difficulty climbing stairs, rising from a seated position, and combing her hair. She also reported dysphagia to solids, exertional dyspnea, and an unintentional 5-kg weight loss over two months.

Physical examination demonstrated symmetric proximal muscle weakness involving the shoulder and hip girdles with preserved sensation and reflexes. Laboratory evaluation revealed markedly elevated creatine kinase (8,940 IU/L), aldolase, lactate dehydrogenase, and transaminases. Antinuclear antibody testing was positive at high titer with a speckled pattern. Electromyography showed an irritable myopathic process, while MRI of the thighs demonstrated diffuse bilateral muscle edema consistent with inflammatory myositis. High-resolution CT of the chest showed subtle bibasal ground-glass opacities without established fibrosis.

The patient was initially diagnosed with polymyositis and treated with pulse intravenous methylprednisolone followed by oral prednisone. Further myositis antibody testing demonstrated positivity for anti-Jo-1 and anti-Ro52 antibodies, confirming antisynthetase syndrome. Methotrexate was subsequently initiated, leading to substantial improvement in muscle strength, swallowing function, and respiratory symptoms during follow-up.

Discussion:

Antisynthetase syndrome can present primarily as inflammatory myopathy before the development of classic pulmonary manifestations. Early recognition is important because respiratory involvement significantly affects prognosis. The presence of anti-Jo-1 antibodies in a patient with inflammatory myopathy should prompt evaluation for antisynthetase syndrome even in the absence of overt interstitial lung disease.

Conclusion:

This case emphasizes the importance of considering antisynthetase syndrome in patients presenting with inflammatory myopathy and bulbar symptoms. Early antibody testing and prompt immunosuppressive therapy facilitate timely diagnosis and improve clinical outcomes.

Resident/Fellow Clinical Vignette

Serum Beta-Hydroxybutyrate–Confirmed Euglycemic Diabetic Ketoacidosis in Septic Shock and Acute Kidney Injury Without SGLT2 Inhibitor Exposure

Ojas Balaji

Aryeh Deutsch, Yisrael Feman, Rajwani Aakash

Introduction:

Euglycemic diabetic ketoacidosis (euDKA) is an uncommon but potentially life-threatening variant of diabetic ketoacidosis characterized by significant ketonemia and high–anion gap metabolic acidosis despite normal or only mildly elevated serum glucose. Although most commonly associated with sodium-glucose cotransporter-2 (SGLT2) inhibitors, euDKA may also occur in critically ill patients without SGLT2 inhibitor exposure, making diagnosis challenging. We present a case of severe euDKA in the setting of septic shock, acute kidney injury, and emphysematous cystitis in which diagnosis depended on serum beta-hydroxybutyrate despite negative urine ketone testing.

Case Presentation:

An elderly woman with type 2 diabetes mellitus treated with saxagliptin and low-dose prandial insulin, severe protein-calorie malnutrition, recurrent urinary tract infections, dementia, prior cerebrovascular accident, hypertension, hyperlipidemia, and a history of pulmonary embolism presented from a long-term care facility in profound shock. She was hypotensive (66/33 mmHg), hypothermic (86.2°F), tachycardic, and unresponsive, requiring intubation and norepinephrine. Laboratory evaluation demonstrated leukocytosis, acute kidney injury (creatinine 1.6 mg/dL from a baseline of 0.7 mg/dL), blood urea nitrogen of 140 mg/dL, severe high–anion gap metabolic acidosis (anion gap 29–33; bicarbonate 6–9 mmol/L), arterial pH 6.98, and lactate of 1.4 mmol/L. Despite glucose ranging from 104–300 mg/dL, serum beta-hydroxybutyrate was markedly elevated at 8.0 mmol/L, while nitroprusside urine ketones were negative, establishing the diagnosis of euDKA. Computed tomography demonstrated emphysematous cystitis with bilateral hydroureteronephrosis and obstructive uropathy. Management included insulin infusion with dextrose-containing intravenous fluids until anion gap closure, aggressive resuscitation, vasopressor support, broad-spectrum antibiotics, Foley catheter placement, and bilateral percutaneous nephrostomy tubes for urinary decompression. The patient improved clinically with successful discontinuation of vasopressors and extubation within 24 hours.

Discussion:

This case highlights the diagnostic limitations of nitroprusside urine ketone testing and emphasizes the importance of serum beta-hydroxybutyrate measurement in patients with unexplained high–anion gap metabolic acidosis despite relatively normal glucose levels. The absence of SGLT2 inhibitor exposure, combined with septic shock, acute kidney injury, severe malnutrition, and ongoing saxagliptin therapy, made this presentation particularly unusual.

Conclusion:

Clinicians should maintain a high index of suspicion for euDKA in critically ill patients presenting with severe metabolic acidosis even in the absence of marked hyperglycemia or SGLT2 inhibitor use. Early recognition using serum beta-hydroxybutyrate, prompt insulin with dextrose therapy, electrolyte management, and definitive source control are essential for favorable outcomes.

Resident/Fellow Clinical Vignette

When Protection Becomes Injury: SGLT2 Inhibitor-Associated Acute Tubular Necrosis - A Two-Case Clinical Vignette Demonstrating Reversible Kidney Injury

Prateek Bali

Ali Ahmadi, MD; Rahul Kumar, BS; David B. Thomas, MD; Fausto C. Bolanos, MD; Michael Chang, MD; Joseph A. Vassalotti, MD

Introduction: SGLT2 inhibitors are widely used in chronic kidney disease (CKD) and provide established cardiorenal benefits. Although they reduce acute kidney injury (AKI) risk at the population level, individual cases have been reported, most attributed to volume depletion. SGLT2 inhibitor initiation commonly causes a transient eGFR decline through tubuloglomerular feedback that is hemodynamic and reversible. Rare histopathologic associations, including acute interstitial nephritis and osmotic nephrosis, have been described, but acute tubular necrosis (ATN) has not previously been reported with SGLT2 inhibitor use. We present two cases of ATN associated with SGLT2 inhibitor therapy, one biopsy-proven and one supported by urine microscopy, both reversible after drug discontinuation.

Case 1: A 61-year-old man with CKD stage 3b, type 2 diabetes, hypertension, and coronary artery disease with reduced cardiac output developed AKI following SGLT2 inhibitor exposure and ARB initiation, with creatinine rising from a baseline of 1.5 to 7.3 mg/dL. Creatinine partially improved to 3.0 mg/dL after ARB discontinuation but climbed again to 5.1 mg/dL after SGLT2 inhibitor dose escalation. MR angiography excluded renal artery stenosis. Renal biopsy sampling 26-30 glomeruli demonstrated severe acute tubular epithelial injury, moderate interstitial fibrosis, 28% global glomerulosclerosis, and severe arteriosclerosis. After SGLT2 inhibitor discontinuation, creatinine improved to 1.4 mg/dL over three months.

Case 2: A 62-year-old man with CKD stage 3b, diabetic kidney disease, hypertension, and nephrolithiasis, maintained on benazepril, developed a progressive creatinine rise from 1.2 to 1.8 mg/dL over three months following SGLT2 inhibitor initiation. Urine microscopy revealed granular casts consistent with ATN. The SGLT2 inhibitor was discontinued, and creatinine recovered to 1.3 mg/dL without further intervention.

Discussion: These cases suggest SGLT2 inhibitors may rarely induce true structural tubular injury, a previously unreported association. We hypothesize that concurrent RAS blockade and SGLT2 inhibition initiated injury by reducing intraglomerular perfusion pressure, with

additional insult from renal medullary hypoxia as increased distal solute delivery raised tubular workload in a region of inherently low oxygen tension. Notably, injury occurred without traditional second hits such as volume depletion or NSAID exposure, suggesting increased metabolic demand alone may precipitate ATN in susceptible individuals.

Conclusions: SGLT2 inhibitors may rarely cause ATN in high-risk CKD patients. Because discontinuation led to recovery in both cases, early recognition is critical. Granular casts on urine microscopy are a useful diagnostic clue, and renal function should be monitored closely after initiation and dose escalation in patients with CKD, particularly those on concurrent RAS blockade. Further studies are needed to identify predictive risk factors.

Resident/Fellow Clinical Vignette

UNMASKING SEROTONIN SYNDROME: DIVERGENT PRESENTATIONS IN A HUSBAND AND WIFE FOLLOWING FENTANYL-TRAZODONE CO-INGESTION

Jacob Batuhan

Michael Leone, MD

Natoushka Trenard, MD

Introduction:

Fentanyl, in addition to its opioid effects, has direct agonist activity at serotonin 5-HT1A and 5-HT2A receptors and promotes serotonin efflux. When combined with other serotonergic agents such as trazodone, a serotonin antagonist and reuptake inhibitor, fentanyl can precipitate serotonin syndrome (SS). Diagnosing SS in this setting is challenging because autonomic instability and agitation overlap substantially with opioid withdrawal (OW). This overlap is further complicated in opioid-tolerant patients, in whom the expected respiratory depression of fentanyl toxicity is absent, and co-ingestants may attenuate the neuromuscular findings that distinguish SS from OW.

Case Description:

A 45-year-old woman and her 53-year-old husband presented to the emergency department after co-ingesting intranasal fentanyl and trazodone. Both exhibited severe autonomic instability, including hypertension (systolic blood pressure >190 mmHg), tachycardia (heart rate >140 beats/min), diaphoresis, and fever. Quantitative serum toxicology (LC-MS/MS) confirmed fentanyl concentrations within ranges reported in fatal overdoses (10.8 ng/mL and 12.7 ng/mL) with elevated norfentanyl levels (3.7 ng/mL and 3.9 ng/mL), consistent with chronic, high-frequency use. Despite near-identical ingestions and autonomic findings, their neurologic exams diverged sharply. The husband met Hunter Serotonin Toxicity Criteria, exhibiting inducible ankle clonus, generalized hyperreflexia, and rigidity. Fentanyl-induced rigidity ("wooden chest syndrome") was considered but was less consistent with his clonus and hyperreflexia. The wife, who admitted daily non-medical use of up to 2 mg alprazolam and had a benzodiazepine-positive urine drug screen, presented with a masked exam featuring normal reflexes and no clonus. Sympathomimetic and anticholinergic toxicity were excluded in both patients by negative urine toxicology and profound diaphoresis. Both were managed in the medical intensive care unit with

intravenous fluids, antihypertensives, electrolyte repletion, methadone for withdrawal, and dexmedetomidine and lorazepam for agitation. Both stabilized and were discharged.

Discussion:

This case offers rare antemortem pharmacokinetic confirmation that profound opioid tolerance can permit survival despite fentanyl concentrations otherwise associated with death. This extreme tolerance prevented early respiratory collapse, thereby unmasking life-threatening serotonergic toxicity. It also illustrates a clinically important pitfall: benzodiazepines, a first-line SS treatment, may blunt the neuromuscular findings that define the syndrome, as likely occurred in the wife. Clinicians evaluating an agitated, opioid-tolerant patient should not default reflexively to a diagnosis of withdrawal; a directed exam for clonus and hyperreflexia is essential, and co-ingestants that may mask these findings should be specifically sought.

Resident/Fellow Clinical Vignette

ANCHORED BY SMOKE, DISTRACTED BY SHADOW: DIAGNOSTIC COGNITIVE ERRORS UNMASKING SUPERIOR MESENTERIC ARTERY SYNDROME

Jacob Batuhan

Kevin Chaverri, MD

Michael Leone, MD

Nikita Mundakel, MD

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Introduction

Superior mesenteric artery (SMA) syndrome is a rare duodenal obstruction caused by compression of the third duodenal segment between the aorta and the SMA. Its symptoms—epigastric pain, vomiting, and weight loss—overlap with functional conditions, leading to diagnostic delay often exacerbated by cognitive biases. Anchoring bias and incidentaloma-driven distractions can completely obscure underlying mechanical pathology. We present a case where a positive urine tetrahydrocannabinol (THC) screen anchored clinicians to cannabinoid hyperemesis syndrome (CHS), while an incidental pituitary microadenoma created a major diagnostic distraction, delaying the diagnosis of SMA syndrome across six emergency department (ED) visits and two hospitalizations over ten weeks.

Case Description

A 23-year-old female with microcytic anemia presented with epigastric pain, bilious emesis, and a 14.8 kg weight loss over three months (a 24% decline from her 62 kg baseline to 47.2 kg; BMI 15.81 kg/m2). She was discharged from four successive ED visits with diagnoses of infectious diarrhea or gastritis despite objective starvation ketosis (urine ketones 80 mg/dL, anion gap 19 mmol/L).

During her first admission, a positive urine THC screen led the team to anchor on CHS—despite 45 days of documented cannabis abstinence, far exceeding the typical symptom resolution window. A scheduled esophagogastroduodenoscopy (EGD) was aborted when the patient developed bilious emesis while strictly NPO, a finding mechanistically inconsistent with CHS that should have prompted evaluation for proximal obstruction. Focus was further diverted by an incidental 4.1 mm pituitary microadenoma abutting the

optic chiasm; the EGD was cancelled to prioritize neuroendocrine workup. She was discharged on oxycodone-acetaminophen, further impairing motility. Within ten days, she returned to the ED twice; haloperidol administered for presumed CHS precipitated acute oromandibular dystonia.

On her second admission, EGD revealed 100 mL of trapped bilious gastric fluid with mild gastritis. A radiology addendum to the initial abdominal CT—previously reported as unremarkable—identified an aortomesenteric angle of 7 degrees and distance of 0.6 cm with proximal duodenal prominence. Gastric emptying scintigraphy confirmed 95.8% retention at 120 minutes, and an upper gastrointestinal series demonstrated a transition point at the third duodenal portion. She was successfully managed with structured enteral nutritional rehabilitation.

Discussion

This case illustrates how sequential cognitive errors establish a pathophysiological feedback loop. Anchoring on CHS delayed recognition of mechanical obstruction, perpetuating vomiting-induced weight loss that further depleted the mesenteric fat pad and worsened aortomesenteric compression. The incidental pituitary lesion created a distraction fixation, diverting resources from gastrointestinal evaluation. Iatrogenic opioid therapy compounded duodenal dysmotility, while neuroleptic administration caused significant extrapyramidal morbidity. Crucially, the diagnostic vascular metrics were present on CT imaging weeks earlier, underscoring the necessity of measuring the aortomesenteric angle on sagittal reconstructions in cachectic patients with refractory vomiting.

Resident/Fellow Clinical Vignette

When Plant Sterols Make Things Worse: Sitosterolemia Unmasked in a Patient with Hypercholesterolemia

David Belkin

Tahmid Rahman, MD

Introduction

Sitosterolemia is a rare inherited disorder of plant sterol metabolism caused by pathogenic variants in ABCG5 or ABCG8 that impair regular physiologic excretion of sterols.1 Because plant sterols are widely promoted as cholesterol-lowering supplements, failure to recognize this condition may paradoxically worsen symptoms and dyslipidemia.

Case Presentation

A man in his forties presented for evaluation of persistently elevated cholesterol levels dating back to his twenties. He had not tolerated several statins due to fatigue, malaise, and muscle aches, and discontinued evolocumab after developing urticaria during an acute viral illness. Despite an active lifestyle and vegetable-rich diet, he remained markedly hyperlipidemic.

Seeking additional benefit, he began over-the-counter plant sterol supplements. Rather than improving his lipid profile, they worsened his fatigue, “head fog,” myalgias, and overall energy. These symptoms resolved promptly when he stopped the supplements. On evaluation, his LDL-C measured 383 mg/dL. Genetic testing, obtained due to the severity and chronicity of his hypercholesterolemia, revealed a pathogenic LDLR mutation consistent with heterozygous familial hypercholesterolemia and an abnormal ABCG8 allele associated with increased sterol absorption, raising concern for sitosterolemia. Coronary calcium scoring showed a score of 18.

He was advised to avoid all plant sterol–containing products and was started on ezetimibe 10 mg daily. At follow-up, he reported dramatic improvement, including resolution of cognitive fog and muscle aches, improved energy, and better exercise tolerance. Repeat lipid testing is pending.

Discussion

This case illustrates the importance of recognizing ABCG8-associated sitosterolemia in adults with severe hypercholesterolemia or unexpected clinical responses to supplements. Excessive sterol absorption and impaired biliary excretion allow plant sterols

to accumulate, producing fatigue, cognitive slowing, and myalgias. Increased sterol delivery to the liver also elevates LDL-C by disrupting cholesterol homeostasis, promoting sterol-rich LDL particle formation, and reducing LDL receptor activity, an effect exacerbated in individuals with concurrent LDLR mutations.2,3 Because plant sterols are widely marketed as “heart-healthy,” patients with unrecognized sitosterolemia may inadvertently worsen both symptoms and LDL levels. Ezetimibe, which inhibits NPC1L1-mediated sterol absorption, directly targets the underlying defect and led to rapid symptomatic improvement in this patient.4,5,6,7 Clinicians should consider sitosterolemia in patients with severe hypercholesterolemia or paradoxical worsening after plant sterol supplementation, as genetic testing can clarify the diagnosis and guide effective therapy with ezetimibe and avoidance of plant sterols.

Resident/Fellow Clinical Vignette

Title: Suspected Felty Syndrome Mimicking Lymphoproliferative Disorder: A Diagnostic Challenge

Samruddhi Bhoyar

Gatsak, Dmytro

Feoktistov, Aleksander

Background: Felty syndrome is a rare syndrome characterized by the triad of neutropenia, splenomegaly, and long-standing erosive rheumatoid arthritis. Patients can have FS even with minimal to absent symptoms of RA making it challenging to diagnose. FS can overlap with lymphoproliferative disorders such as LGL, NHL, and MUDS/MM which share similar features of cytopenia, splenomegaly, and constitutional symptoms. Accurate differentiation is critical for appropriate therapy.

Case Presentation: A 60-year-old female with a PMH of sickle cell trait, diabetes mellitus, and H. pylori infection s/p triple therapy, was referred to Hematology for splenomegaly, anemia, decreased appetite and weight loss. A peripheral blood flow cytometry was obtained but was negative for malignancy. However, paraprotein studies showed IgM lambda M spike of 0.3 with a mild elevation in lambda FLC of 3.31. PET scan showed splenomegaly, bilateral pulmonary nodules, and paratracheal LAD. Bronchoscopy with EBUS was non-diagnostic. Bone marrow biopsies done twice were negative for clonal abnormality. She was later hospitalized for LUQ pain and fever. CTAP showed splenomegaly and a new splenic infarct. Peripheral smear and CBCs revealed neutropenia. RF was negative but anti-CCP antibodies were highly positive (>250 U/mL). Patient reported fleeting polyarthralgias of bilateral wrists, hips, and ankles three years ago but currently asymptomatic. Rheumatology was consulted for FS, and she was discharged on a prednisone taper with outpatient follow up. Outpatient splenic biopsy was negative for hematologic malignancy. She was later hospitalized for diffuse body pain. Inpatient cervical lymph node biopsy was negative for suspected NHL. The case was presented at tumor board. Rituximab was recommended for Felty syndrome and low probability of B cell lymphoma. She was restarted on prednisone 60 mg daily with a weekly taper and received four weekly Rituximab doses with improvement in her abdominal pain and neutropenia.

Discussion: This case highlights the diagnostic complexity of FS, which can present with neutropenia and splenomegaly before overt arthritis. Atypical features — negative RF, highly positive anti-CCP, and remote joint symptoms can lead to delays in diagnosis and misclassification as hematologic malignancy. MGUS/MM was suspected due to IgM M

spike (0.3). However, multiple bone marrow biopsies were negative for clonal cells. NHL was ruled out with negative bone, spleen, and lymph node biopsies. LGL, linked to RA and often missed on bone marrow biopsy, was excluded by negative clonal T-cells on peripheral smear and flow cytometry making FS the leading diagnosis. Rituximab was appropriate therapy given its role in B-cell suppression in both refractory FS and lymphoproliferative disorders.

Conclusion: FS can mimic lymphoproliferative disorders, leading to diagnostic delays. High-titer anti-CCP antibodies in the appropriate clinical context support the diagnosis of FS. Careful interpretation of diagnostic studies is essential to avoid misdiagnosis and guide appropriate therapy.

Resident/Fellow Clinical Vignette

A Unique Case Of Acute Decompensated Liver Failure In A Patient With Hyperthyroidism and Right Heart Failure

Sean Callagy

Andrey Moiseyenko, D.O. PGY-IV, Robert Burns MS-IV, Toni Esposito MS-IV, Nikolaos Hatzis, M.D.

Acute-on-chronic liver failure (ACLF), also referred to as acute decompensated liver failure, involves the development of hepatic with or without extrahepatic organ failure in patients with existing chronic liver disease (1). No specific therapies exist for ACLF currently and up to 40% of patients will have no identifiable cause (1). The following case demonstrates a patient with acute decompensated liver failure due to methimazole use, infection, and right heart failure, complicated by multiorgan failure.

Our patient is a 47-year-old male with a past medical history of atrial fibrillation, hyperthyroidism, hypertension, non-alcoholic steatohepatitis (NASH), prior hepatitis C infection, morbid obesity, and chronic lower extremity edema who presented for progressively worsening dyspnea on exertion, abdominal distension, orthopnea, yellowing of his sclera, and brown colored urine. The patient's history was also notable for mild ascites and lower extremity edema at baseline, along with a family history of liver cirrhosis in his biological mother despite no personal history of liver disease. On initial evaluation, the workup was consistent with acute decompensated liver failure and right heart failure. Methimazole was discontinued due to suspected drug induced liver injury. The patient ultimately became unstable following an episode of cardiac arrest, acute respiratory failure requiring mechanical ventilation, hypotension requiring vasopressors, and acute renal failure requiring emergent hemodialysis. All workup remained unremarkable for potential causes of liver disease, though did reveal pneumonia and a urinary tract infection. The patient was ultimately transferred to a liver transplant center for further evaluation.

A diagnosis of ACLF requires that a patient has underlying chronic liver disease. Despite this patient having no prior diagnosis of cirrhosis and a non-cirrhotic liver on imaging, he did possess known NASH, a known prior hepatitis C infection that was successfully treated, a family history of liver disease, and endorsed chronic mild ascites and lower extremity edema. Methimazole induced liver injury is classically

cholestatic in pattern and has been documented to cause acute liver failure requiring transplantation, though the risk remains low at 0.4%. Injury usually occurs in the first few days to months after initiation of the medication, unlike this patient who had been taking the same dose of this medication for many years and then subsequently stopped shortly before this hospitalization (2,3,4,5). Hyperthyroidism, when left untreated, can also cause abnormal liver function testing, typically a cholestatic pattern that is reversible (6).

This case demonstrates ACLF likely due to drug induced injury from methimazole use in the setting of underlying infection and heart failure. As many cases of ACLF ultimately have no identifiable precipitating factor, further research will be needed to determine best practices for workup and treatment.

Resident/Fellow Clinical Vignette

BEYOND MONONUCLEOSIS: SPLENIC INFARCTION IN ACUTE EPSTEIN-BARR VIRUS INFECTION

Andres Chaponan-Lavalle

Jorge Alave, MD, Roberto Alexander Ramirez Hurtado, MD, Nelson Diaz, MD

Introduction: Epstein Barr Virus (EBV) is the most common cause of Infectious mononucleosis (IM), which commonly presents with fever, pharyngitis, and lymphadenopathy. Splenic involvement is a well recognized feature of IM; however, splenic infarction is a rare and likely underrecognized complication. We describe a previously healthy young woman who developed splenic infarction early in the course of acute EBV infection.

Case presentation: A 22-year-old woman with recent travel to Bolivia, Chile and the Peruvian Amazon presented to the emergency department with two-day history of left upper quadrant abdominal pain, painful cervical lymphadenopathy, and polyarthralgia. Five days earlier, she had developed malaise, subjective fever, and sore throat. Examination revealed an erythematous pharynx, diffuse tender lymphadenopathy, and left upper quadrant tenderness without palpable hepatosplenomegaly.

Laboratory studies demonstrated leukocytosis (16,480/µL) with lymphocytic predominance (67%) and 16% reactive lymphocytes, mild thrombocytopenia (143,000/µL), and elevated liver enzymes and lactate dehydrogenase. Contrast-enhanced abdominal computed tomography revealed splenomegaly with multiple peripheral wedge-shaped hypodense lesions consistent with splenic infarctions (Figure 1). EBV viral capsid antigen IgM and IgG were positive, confirming acute EBV infection. Testing for HIV, hepatitis B and C, cytomegalovirus, toxoplasmosis, dengue, brucellosis, and respiratory viruses was negative. Transthoracic echocardiography showed no intracardiac thrombus, valvular vegetation, or structural abnormality. No laboratory evidence of an underlying thrombophilic or hematologic disorder was identified.

The patient was managed conservatively with intravenous fluids and analgesia. Anticoagulation was deferred given the absence of ongoing thrombosis, an embolic source, or an underlying prothrombotic condition. She improved clinically without surgical intervention.

Discussion: Splenic infarction is an uncommon complication of acute EBV infection and may be overlooked because abdominal pain can be attributed to splenomegaly alone.

Proposed mechanisms include increased splenic metabolic demand from lymphoid hyperplasia, transient activation of coagulation pathways, and vascular compression related to splenic enlargement. In this case, infarction occurred only five days after symptom onset, showing that this complication can develop earlier than traditionally recognized. Contrast-enhanced computed tomography is essential when patients with suspected infectious mononucleosis develop focal left upper quadrant pain.

Conclusion: Splenic infarction should be considered in patients with acute EBV infection presenting with focal left upper quadrant pain. Early recognition may guide appropriate management.

Resident/Fellow Clinical Vignette

When the Immune System Strikes in the Waiting Room: A Case of Mixed Autoimmune Hemolytic Anemia.

Sara Chekalil

Nailah Sayeed, MBBS; Sadia Bari, MBBS; Nasir Gondal, MD

Introduction: Mixed autoimmune hemolytic anemia (AIHA) is a rare subtype comprising 5–10% of cases, characterized by both warm IgG and complement-mediated hemolysis. It is associated with severe anemia, high transfusion requirements, and frequent refractoriness to standard therapy [1].

Case Presentation: A 23-year-old previously healthy male presented with lightheadedness, dyspnea, abdominal pain, vomiting, and hematemesis. Symptoms began while accompanying his wife for labor and delivery in the waiting room, prompting evaluation. He was tachycardic but hemodynamically stable.

Laboratory evaluation showed severe anemia (hemoglobin 4.9 g/dL), elevated lactate dehydrogenase, indirect hyperbilirubinemia, and low C4. Direct antiglobulin testing was positive for IgG and C3, consistent with mixed AIHA. Cold agglutinins were negative. Mycoplasma IgM was elevated. Anti-β2 glycoprotein antibodies were positive; remaining autoimmune and hematologic workup, including PNH FLAER, serum protein electrophoresis, and flow cytometry, was unremarkable.

He required ICU admission and transfusion of 10 units of packed red blood cells. Initial treatment with high-dose corticosteroids, IVIG, plasmapheresis, and doxycycline failed to control hemolysis. Rituximab (375 mg/m² weekly for four doses) was initiated on hospital day 5. Bone marrow biopsy showed erythroid hyperplasia without malignancy.

At 33-day follow-up, hemoglobin normalized to 13.4 g/dL with resolution of hemolysis and successful steroid taper.

Discussion: This case highlights the severity and complexity of mixed AIHA [1]. Dual IgG and C3 positivity with low C4 supports complement-mediated hemolysis contributing to profound anemia [1,2]. Although Mycoplasma pneumoniae typically causes cold agglutinin syndrome, this presentation with negative cold agglutinins and refractory disease suggests an atypical immune trigger [1,2].

Positive anti-β2 glycoprotein antibodies raise concern for evolving autoimmune disease and warrant repeat testing at ≥12 weeks [4]. Corticosteroids remain first-line therapy;

however, consensus guidelines support early Rituximab in severe or atypical cases [3]. Plasmapheresis may provide temporary control in critical illness as a bridge to immunosuppression [5]. The patient's sustained response to Rituximab underscores its role in refractory disease.

Conclusion: Mixed AIHA may present with life-threatening anemia refractory to standard therapy. Early recognition and escalation to Rituximab, with adjunctive plasmapheresis when needed, are critical to achieving remission.

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Resident/Fellow Clinical Vignette

The Splinter That Was Not Remembered: Recurrent Abdominal Pain From an Occult Wooden Foreign Body Without Intraoperative Bowel Injury- A Case Report

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Background

Wooden foreign bodies in the gastrointestinal tract or abdominal cavity are diagnostically challenging due to their radiolucency, subtle appearance on imaging, and frequent lack of patient recall. Sharp objects such as toothpicks or splinters may traverse the gastrointestinal tract and lead to complications, including localized inflammation, abscess, phlegmon, fistula formation, or perforation. However, presentation as an inflammatory abdominal mass without a clear history of ingestion, trauma, or overt perforation remains uncommon.

Case Presentation

A 74-year-old woman with a history of appendectomy, hysterectomy, hypertension, hyperlipidemia, mild dementia, and prior removal of a wooden foreign body from the right lower quadrant two years earlier presented with four weeks of epigastric pain and two days of nausea. She had been hospitalized two weeks prior for similar symptoms, during which esophagogastroduodenoscopy and colonoscopy reportedly revealed no abnormalities.

On presentation, she was afebrile and hemodynamically stable, with moderate to severe tenderness in the epigastric, left upper, and left lower quadrants. Laboratory studies demonstrated no leukocytosis, normal lipase and lactate, with mild normocytic anemia and thrombocytosis. Computed tomography of the abdomen and pelvis revealed a stable 2.7-cm linear hyperdensity within the greater omentum along the greater curvature of the stomach, associated with surrounding fat stranding and phlegmon, concerning for a retained foreign body. A sealed gastric perforation could not be excluded.

The patient was treated empirically with piperacillin-tazobactam and underwent operative exploration. Intraoperatively, a large phlegmonous mass adherent to the anterior abdominal wall, stomach, and transverse colon was identified. A wooden-appearing foreign body was visualized and removed. Careful inspection of the stomach and colon revealed no evidence of perforation. Pathologic examination showed soft tissue with abscess formation and Gram-positive, GMS-positive branching filamentous organisms,

morphologically suggestive of Actinomyces or Nocardia. Despite repeated history-taking and psychiatric evaluation, the patient denied ingestion of wooden objects or prior trauma.

Discussion

This case underscores the diagnostic difficulty of wooden foreign bodies, particularly in patients without a clear ingestion history. Prior studies demonstrate that many patients do not recall ingesting toothpicks, and detection on imaging may be limited. CT remains the preferred modality, though identification depends on clinical suspicion, thin-slice review, and recognition of secondary inflammatory changes. The absence of an identifiable perforation suggests either a healed or contained transmural migration, prior unrecognized extraluminal migration, or an alternative traumatic mechanism. The presence of branching filamentous organisms further raises the possibility of Actinomyces or Nocardia infection, warranting clinicopathologic correlation.

Conclusion

Wooden foreign bodies should remain in the differential diagnosis of unexplained, recurrent abdominal pain with focal inflammatory findings, even in the absence of reported ingestion or trauma. Subtle CT findings, such as linear hyperdensity with surrounding inflammation, should prompt careful evaluation. Surgical exploration may be both diagnostic and therapeutic in persistent cases.

Resident/Fellow Clinical Vignette

When Horses Break the Heart and Mind: Disseminated Zoonotic Endocarditis Complicated by Mycotic Aneurysm Rupture

Miracle Chukwuma

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Introduction: Streptococcus equi subsp. zooepidemicus is a rare zoonotic pathogen associated with equine exposure and an uncommon cause of infective endocarditis (IE). Human infection can lead to severe invasive disease with metastatic and neurologic complications.

Case Presentation: A 48-year-old man presented with headache, bilateral shoulder and hip pain, fatigue, blurry vision, and low back pain. He had recently begun working at a racetrack and reported exposure to horses infected with strangles. Examination revealed a diastolic murmur at the right second intercostal space. Laboratory studies demonstrated leukocytosis, thrombocytopenia, and markedly elevated inflammatory markers. Initial CTA head/neck and chest radiograph were unremarkable. Blood cultures grew Streptococcus equi subsp. zooepidemicus. Aspiration of the left hip and shoulder grew the same organism, and MRI lumbar spine demonstrated findings concerning for paraspinal abscess. Transesophageal echocardiography revealed aortic valve infective endocarditis with echodensities involving the right coronary cusp. The patient additionally developed endogenous endophthalmitis requiring an anterior chamber tap and intravitreal vancomycin. After four weeks of targeted antimicrobial therapy, he improved clinically and was discharged. Two days later, he returned with altered mental status, unresponsiveness, a blown left pupil, and right-sided weakness. CT head demonstrated a left intracerebral hemorrhage, while CTA revealed a ruptured mycotic aneurysm involving the M2 branch of the middle cerebral artery with active extravasation and uncal herniation. Neurosurgical intervention was deemed futile. Despite supportive therapy, the patient progressed to brain death and died three days later.

Discussion: S. zooepidemicus is an uncommon but highly invasive zoonotic pathogen capable of causing fulminant infective endocarditis with widespread hematogenous dissemination. This case highlights the importance of obtaining a detailed occupational and exposure history when evaluating atypical infective endocarditis. It also demonstrates that catastrophic neurologic complications, including ruptured intracranial mycotic aneurysms, may occur despite initial clinical improvement and discharge. Mycotic aneurysms may develop and rupture despite appropriate antimicrobial therapy,

emphasizing the need for continued vigilance and close follow-up in patients with disseminated infective endocarditis.

Resident/Fellow Clinical Vignette

HIDDEN IN PLAIN SIGHT: AN UNUSUAL CASE OF NEUROCYSTICERCOSIS

Dilja Mary David

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Intraventricular Neurocysticercosis (NCC) may present without seizures and can mimic primary headache syndromes or cranial neuralgias while causing obstructive hydrocephalus. NCC, caused by the larval stage of Taenia solium, is a leading cause of acquired epilepsy in endemic regions and is increasingly encountered in non-endemic countries due to migration and travel. In the US, population-based hospitalization analyses for NCC show a markedly higher hospitalization rate in Hispanic populations compared with non-Hispanic Whites, reflecting imported infection and local transmission from tapeworm carriers.

A 68-year-old woman with hypertension, hyperlipidemia, prior right V1 herpes zoster, and a self-reported recent cerebrovascular event presented with years of severe right-sided headache and shooting facial pain (V1–V3). Headache is constant, sometimes associated with Nausea and vomiting. Facial pain is exacerbated by yawning and chewing. Physical examination was unremarkable.

The patient was evaluated in ED for headache and facial pain around five months ago. At that time, patient self-reported a stroke 8 months prior in her native El Salvador that affected her right limbs and gait. She also stated that headache had been persistent since the stroke. She further stated that facial pain was similar to that she had 3 years ago due to shingles. CT head showed small vessel ischemic changes, but no evidence of infarction was present. The patient was discharged from ED with pain medications and a referral to Neurology. Patient was seen in neurology clinic a month ago and was diagnosed with right trigeminal neuralgia in all 3 dermatomes. Patient was started on oxcarbazepine, however, reports no significant improvement.

Initial diagnoses favored zoster-related neuralgia, and neuropathic medications provided minimal relief. Brain MRI revealed a predominantly cystic, non-enhancing lesion filling the fourth ventricle with ventriculomegaly consistent with chronic obstructive hydrocephalus. Progressive ventricular enlargement prompted neurosurgical intervention. Suboccipital craniotomy with cyst excision and external ventricular drain placement was performed. Pathology demonstrated calcified cysticercus structures, confirming NCC, and serum cysticercus IgG was positive. Following surgical intervention, patient was treated with albendazole and corticosteroids, with improvement in headache.

Follow-up evaluations in primary care, neurosurgery, and infectious disease clinics demonstrated significant clinical improvement, with resolution of headaches and return to normal activities.

Fourth ventricular NCC may masquerade as chronic unilateral headache or trigeminal neuralgia, and progressive ventriculomegaly should prompt consideration of intraventricular NCC in at-risk patients. Early recognition is essential to prevent decompensated hydrocephalus and guide appropriate neurosurgical and antiparasitic management. This case also highlights the importance of considering NCC in patients from endemic regions who present with chronic headache syndromes plus imaging evidence of ventricular obstruction, even when competing explanations (post-zoster neuralgia, reported stroke) exist.

Resident/Fellow Clinical Vignette

Comparative Critical Outcomes of Acute Pancreatitis with Concomitant Heart Failure Versus Acute Pancreatitis Alone: A Propensity Score–Matched Retrospective Cohort Study

Stephen Djanie

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Background: Acute pancreatitis (AP) is a leading cause of hospitalization and may progress from mild inflammation to multiorgan failure. Heart failure (HF) may complicate AP management because impaired cardiac reserve and the need to balance adequate fluid resuscitation with avoidance of volume overload present significant therapeutic challenges. Evidence regarding the independent impact of HF on cardiovascular outcomes in AP remains limited. We hypothesized that patients hospitalized with AP and preexisting HF experience worse clinical outcomes compared to those without HF.

Methods:

We conducted a retrospective cohort study using the TriNetX Research Network. Adult patients diagnosed with AP between January 2005 and March 2026 were identified and stratified into two cohorts: AP with HF (AP+HF) and AP without HF. Propensity score matching (1:1) was performed using demographics, comorbidities, and medications, achieving balanced cohorts (n = 40,399 per group; all standardized mean differences <0.1). The primary outcome was 30-day all-cause mortality. Secondary outcomes included sepsis, septic shock, acute respiratory distress syndrome (ARDS), acute kidney injury (AKI), and venous thromboembolism (VTE). Outcomes were assessed using Cox proportional hazards models and reported as hazard ratios (HRs) with 95% confidence intervals (CIs).

Results:

After matching, patients with AP+HF had significantly higher 30-day mortality compared to those without HF (4.1% vs 3.6%; HR 1.11, 95% CI 1.03–1.19; p = 0.005). The HF cohort also demonstrated increased risks of sepsis (HR 1.72, 95% CI 1.53–1.93; p < 0.001), septic shock (HR 2.09, 95% CI 1.74–2.51; p < 0.001), ARDS (HR 2.37, 95% CI 1.75–3.21; p < 0.001), AKI (HR 1.69, 95% CI 1.52–1.88; p < 0.001), and VTE (HR 1.83, 95% CI 1.54–2.16; p < 0.001).

Conclusion: Preexisting heart failure is independently associated with worse outcomes in patients hospitalized with acute pancreatitis, including increased 30-day mortality and higher risks of sepsis, organ failure, and thromboembolic events. These findings highlight the heightened vulnerability of this population, likely driven by impaired cardiac reserve and complexities in fluid management. Early risk stratification and tailored management strategies are warranted to improve outcomes in this high-risk group.

Resident/Fellow Clinical Vignette

Gastrointestinal Mucormycosis After Autologous Peripheral Stem Cell Transplantation in Relapsed Hodgkin Lymphoma

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Introduction:

Invasive mucormycosis is an angioinvasive fungal infection typically associated with allogeneic transplantation, graft-versus-host disease, and prolonged neutropenia. Gastrointestinal involvement is rare, and cases following autologous peripheral stem cell transplant (auto-PSCT), a population that does not routinely receive antifungal prophylaxis, are exceptionally uncommon. We report a case in which a nonspecific gastrointestinal presentation delayed diagnosis of invasive mucormycosis in an auto-PSCT recipient without classical risk factors.

Case Presentation:

A 51-year-old man with type 2 diabetes and relapsed classical Hodgkin lymphoma, previously treated with (ABVD) doxorubicin, bleomycin, vinblastine, and dacarbazine and radiation, received salvage pembrolizumab plus gemcitabine, vinorelbine, and liposomal doxorubicin (GVD) chemotherapy followed by auto-PSCT with lomustine, cytarabine, cyclophosphamide, and etoposide (LACE). He developed neutropenia on day 3 and neutropenic enterocolitis with abdominal pain, distention, and constipation on day 4, managed supportively in the intensive care unit. Symptoms recurred on day 7, and by day 9 he had persistent fever despite negative blood cultures and empiric liposomal amphotericin B; filgrastim was escalated for delayed engraftment. Abdominal symptoms worsened by day 22, though computed tomography was limited by poor oral contrast uptake. On day 25, he developed metabolic acidosis and septic shock; repeat imaging revealed bowel ischemia with patent vasculature. Exploratory laparotomy showed gangrenous small bowel and abdominal wall musculature requiring resection. Histopathology demonstrated angioinvasive fungal disease with broad, ribbon-like, non-

septate hyphae, and tissue cultures grew Mucorales species, confirming gastrointestinal mucormycosis. Despite continued liposomal amphotericin B, he developed progressive multiorgan dysfunction and died on day 30.

Discussion:

Gastrointestinal mucormycosis carries especially high mortality because of its nonspecific presentation and rapid progression. Classical risk factors, including prolonged neutropenia, GVHD, corticosteroid use, iron overload, and voriconazole prophylaxis, are typical of allogeneic, not autologous, transplant recipients, yet a substantial proportion of cases occur without any of these features, as in this patient. His multiple treatment lines, underlying diabetes, and transplant-associated immunosuppression likely contributed despite falling outside the usual auto-PSCT risk paradigm. Diagnosis is difficult because radiographic findings are nonspecific and neutropenia often precludes early endoscopic evaluation, as occurred here. Emerging molecular diagnostics, particularly serum Mucorales qPCR, may allow earlier, non-invasive detection, having turned positive days before microbiologic, histopathologic, or radiographic confirmation in prior series, though further validation is needed.

Conclusion:

We should maintain a high index of suspicion for gastrointestinal mucormycosis in immunocompromised patients with persistent, antimicrobial-unresponsive abdominal symptoms, even in the absence of classical GVHD or allogeneic-transplant risk factors, and should pursue early tissue sampling whenever feasible, as the lack of routine antifungal prophylaxis in auto-PSCT recipients may otherwise delay diagnosis.

Resident/Fellow Clinical Vignette

WHEN FRAGILITY PERSISTS: MISSED OSTEOMALACIA IN AN ELDERLY PATIENT WITH X-LINKED HYPOPHOSPHATEMIC RICKETS

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INTRODUCTION:

X-linked hypophosphatemic rickets (XLH) is a rare, X-linked dominant phosphate-wasting disorder caused by pathogenic variants in the phosphate-regulating endopeptidase homolog, X-linked (PHEX) gene, with an estimated prevalence of 1 in 20,000 individuals. Excess fibroblast growth factor 23 (FGF23) activity results in renal phosphate wasting, impaired 1,25-dihydroxyvitamin D synthesis, and defective bone mineralization. While XLH is typically recognized in childhood, its manifestations in older adults are frequently misattributed to osteoporosis, leading to delayed recognition of osteomalacia and suboptimal management. This case highlights how persistent hypophosphatemia and recurrent fractures in late adulthood can be overlooked.

CASE DESCRIPTION:

A 78-year-old female with a history of osteoporosis, hypertension, and osteoarthritis was referred for evaluation of recurrent fragility fractures. She had been diagnosed with hypophosphatemic rickets in childhood and underwent bilateral tibial osteotomies for progressive bowing. She sustained multiple fragility fractures, including a femoral trochanteric fracture (2008), tibial and ankle fractures (2013), metatarsal fractures (2012), and a proximal ulnar fracture (2020), with chronic gait impairment requiring a cane.

On examination, she had short stature, scoliosis, and limb bowing. She lost her teeth in her 40s requiring dentures. Laboratory evaluation showed hypophosphatemia (1.3 mg/dL), elevated alkaline phosphatase (170 U/L), elevated parathyroid hormone (94 pg/mL), normal calcium, normal 25-hydroxyvitamin D, and elevated FGF23 (126 pg/mL). Bone densitometry demonstrated low T-scores (−2.9 spine, −1.9 hip). As bone densitometry cannot distinguish impaired mineralization from reduced bone mass, osteomalacia may present with low T-scores similar to osteoporosis.

Genetic testing confirmed a pathogenic PHEX variant (c.1949T>G; p.Leu650Arg), establishing XLH.

The patient had been treated intermittently with calcitriol alone without consistent phosphate supplementation and was lost to follow-up. She was restarted on calcitriol, cholecalciferol, and phosphate, with burosumab under consideration.

DISCUSSION:

This case highlights a key diagnostic pitfall: fragility fractures in older adults are often attributed to osteoporosis, while osteomalacia due to chronic hypophosphatemia remains underrecognized. Persistent hypophosphatemia, elevated alkaline phosphatase, and recurrent fractures should prompt evaluation for phosphate-wasting disorders.

Delayed recognition and inconsistent treatment contributed to cumulative skeletal morbidity. While conventional therapy may partially correct abnormalities, burosumab offers targeted treatment by inhibiting FGF23 and improving phosphate homeostasis. Therefore, early recognition, close follow-up, and multidisciplinary care remain critical for lifelong management of this rare disorder.

Resident/Fellow Clinical Vignette

HMG-CoA Reductase Antibody-Positive Immune-Mediated Necrotizing Myopathy Presenting With Severe Bulbar Dysfunction

Bolaji Elutilo

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Introduction

Anti-HMGCR immune-mediated necrotizing myopathy (IMNM) is a rare autoimmune myopathy typically associated with statin exposure. It is classically characterized by marked creatinine kinase(CK) elevation, histopathological muscle necrosis, and progressive proximal myopathy. Although dysphagia may occur, severe bulbar involvement as an initial presentation is uncommon and may delay diagnosis.

Case Presentation

A woman in her mid-fifties with hypertension, hyperlipidemia, nonalcoholic fatty liver disease, asthma, and prediabetes presented with myalgias, sore throat, and subjective dyspnea. She was initially treated for a presumed viral illness and discharged. Four days later, she returned with worsening myalgias, malaise, dysphagia, and difficulty swallowing solids and liquids. She reported recent statin use, which was discontinued two weeks prior due to reported numbness. Laboratory studies revealed CK 14,600 U/L, transaminitis, leukocytosis, and thrombocytosis. Computed tomography of the neck was unremarkable. She was managed for rhabdomyolysis with aggressive intravenous fluids and discharged after her symptoms and CK improved.

Five days later, she was readmitted with progressive dysphagia, reduced ability to control or swallow oral secretions, dysphonia, chest pressure, and recurrent myalgias. CK increased to 15,505 U/L, ESR was 66 mm/hr, AST was 780 U/L, ALT was 663 U/L, and troponin was 264 ng/L. Anti-HMGCR IgG antibody returned positive at 430 U/mL. Extensive structural imaging was unrevealing. Speech-language pathology evaluation demonstrated severe oropharyngeal dysfunction with high aspiration risk. MRI STIR imaging showed diffuse muscle edema involving the left upper extremity and chest wall. Muscle biopsy revealed necrotic myofibers undergoing myophagocytosis, confirming immune-mediated necrotizing myopathy. Due to severe dysphagia, a percutaneous endoscopic gastrostomy tube was placed for nutritional support. She was treated with pulse-dose corticosteroids, intravenous immunoglobulin, rituximab, and supportive care.

Discussion

Anti-HMGCR IMNM should be considered in patients with unexplained rhabdomyolysis and progressive dysphagia, particularly following statin exposure. This case is uniquely characterized by severe bulbar dysfunction, including dysphagia, dysphonia, and inability to manage oral secretions, which preceded overt proximal muscle weakness and initially mimicked a viral illness. Although dysphagia occurs in approximately one-quarter of patients with anti-HMGCR IMNM due to involvement of the oropharyngeal and proximal esophageal musculature, severe bulbar manifestations as the presenting feature are uncommon and may delay diagnosis. The underlying mechanism mirrors the pathophysiology of limb muscle disease, with anti-HMGCR autoantibodies triggering complement-mediated injury and macrophage-associated myophagocytosis, resulting in myofiber necrosis. Furthermore, recovery of swallowing function may be prolonged because anti-HMGCR antibodies impair myoblast fusion and muscle regeneration, limiting restoration of damaged muscle fibers. Diagnosis in this patient required integration of serologic testing, MRI evidence of diffuse muscle edema, and a confirmatory muscle biopsy demonstrating necrotizing myopathy. Early recognition and aggressive immunosuppressive therapy, including corticosteroids, intravenous immunoglobulin, and rituximab, are essential to reduce morbidity, prevent permanent functional impairment, and improve long-term outcomes.

Resident/Fellow Clinical Vignette

The Varied Presentation of Secondary Hemophagocytic Lymphohistiocytosis: A Case Series

Harry Fruchtman

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Background: Hemophagocytic lymphohistiocytosis (HLH) is a rare, life-threatening hyperinflammatory syndrome characterized by uncontrolled immune activation and cytokine release. Secondary HLH (sHLH) occurs most commonly in association with autoimmune disease, malignancy, or infection, and carries high mortality due to delayed recognition and its heterogeneous clinical presentation. Management depends on identifying and treating the underlying trigger, making early diagnosis essential.

Cases: We present three patients with HLH secondary to distinct etiologies, each requiring a different therapeutic approach. The first patient, a man in his early 30s, presented with fever, rash, polyarthralgia, hyperferritinemia, hypertriglyceridemia, splenomegaly, and hemophagocytosis on bone marrow biopsy, fulfilling diagnostic criteria for HLH. He also met Yamaguchi criteria for adult-onset Still's disease (AOSD). Treatment with high-dose corticosteroids and Anakinra an interleukin-1 receptor antagonist resulted in rapid clinical improvement and improvement of inflammatory markers. The second patient, a man in his early 30s with prior histiocytic necrotizing lymphadenitis, developed persistent fevers, cytopenias, hepatosplenomegaly, hyperfirritinemua and elevated soluble transferrin 2 receptor. Despite corticosteroids and Anakinra, his disease progressed until splenic biopsy revealed T-cell/histiocyte-rich large B-cell lymphoma. Following initiation of lymphoma-directed chemotherapy, his symptoms resolved and laboratory abnormalities improved. The third patient, a man in his late 30s with relapsed/refractory T-cell/histiocyte-rich large B-cell lymphoma, presented with fever, splenomegaly, pancytopenia, hyperfieritinemia, hypertriglyceridemia, complicated by disseminated intravascular coagulation and multiorgan failure. Initial treatment with corticosteroids and Anakinra failed, and lymphoma-directed therapy with etoposide was initiated; however, his condition continued to deteriorate, and he ultimately died after transition to hospice care.

Conclusion: These cases demonstrate the marked heterogeneity of adult sHLH and underscore the importance of identifying the underlying etiology to guide treatment. While immunomodulatory therapy successfully controlled macrophage activation syndrome associated with AOSD, lymphoma-associated HLH improved only after treatment of the underlying malignancy. In advanced malignancy complicated by infection and multiorgan

failure, prognosis remained poor despite aggressive intervention. Given the diverse triggers and clinical presentations of sHLH, a standardized treatment algorithm may not be appropriate. Instead, prompt recognition, multidisciplinary evaluation, and individualized therapy directed at the underlying disease are critical to improving outcomes.

Resident/Fellow Clinical Vignette

A Tongue In Peril: Ischemia Beyond Infection

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Introduction: Giant cell arteritis (GCA) is the most common large-vessel vasculitis in older adults, and delayed recognition can lead to irreversible ischemic complications. We describe a biopsy-confirmed case of GCA initially treated as oropharyngeal candidiasis that rapidly progressed to ischemic tongue necrosis.

Case Presentation: A 74-year-old woman with diabetes, hypertension, and osteoporosis presented with four weeks of worsening tooth pain, throat pain, and dysphagia, preceded by six weeks of fatigue, exertional dyspnea, and presyncope. She had recently completed two courses of amoxicillin-clavulanate for presumed odontogenic infection without improvement. She was afebrile; an oral exam showed creamy-white plaques over the dorsal tongue with halitosis. She denied headache, scalp tenderness, or visual changes. Severe hyperglycemia (glucose 420 mg/dL, HbA1c 10.5%) favored candidiasis. CT neck was negative for infection but revealed incidental pulmonary emboli, prompting anticoagulation.

Despite antifungal therapy and improved glycemic control, tongue pain worsened and plaques evolved into yellow-green discoloration involving most of the dorsal tongue. Bedside biopsy showed necrotic tissue without fungal elements, excluding candidiasis. Vascular imaging showed no vasculitis. Rheumatology was consulted, and empiric IV methylprednisolone was started given markedly elevated inflammatory markers (ESR peaking at 145 mm/hr, hsCRP 18.21 mg/dL) and leukocytosis; ANCA, antiphospholipid antibodies, and infectious serologies were negative. Despite unrevealing vascular imaging, bilateral temporal artery biopsy confirmed GCA.

Necrosis progressed to black, devitalized tissue but stopped advancing after corticosteroids were started; the necrotic tip and anterior left dorsal tongue sharply demarcated and auto-amputated, exposing viable tissue. She required multimodal

analgesia, achieving relief with liquid morphine, and was discharged on prednisone with plans for outpatient tocilizumab. At two-month follow-up she had preserved tongue function, though speech was mildly affected after losing the tongue tip.

Discussion: Tongue ischemia and necrosis are exceptionally rare in GCA, occurring in fewer than 1% of cases, owing to the tongue's rich collateral blood supply — which is why it should raise concern for systemic vasculitis once common causes are excluded. Hyperglycemia, multiple courses of antibiotics and white lingual plaques initially pointed toward candidiasis. A systematic review of 55 cases found lingual necrosis was the presenting manifestation in nearly a third of patients, often without cranial features, reinforcing that neither absent cranial symptoms nor negative imaging excludes GCA. Temporal artery biopsy remained diagnostic despite being performed within one week of steroid initiation.

Conclusion: This case illustrates why GCA should remain on the differential for older adults with rapidly progressive oral lesions mimicking oral candidiasis, even absent cranial symptoms. Early recognition, prompt immunosuppression, and multidisciplinary care are key to preventing irreversible ischemic damage.

Resident/Fellow Clinical Vignette

Metastatic Gastric Adenocarcinoma Mimicking Advanced Gynecologic Malignancy: Diagnostic Value of the CA-125/CEA Ratio

Svetlana Gasparyan

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Background:

Gastric adenocarcinoma can rarely present with clinical and radiographic features that mimic advanced gynecologic malignancy, particularly in postmenopausal women with ascites and adnexal masses. This overlap increases the risk of diagnostic anchoring and delayed identification of the primary tumor. Tumor marker patterns may provide critical diagnostic guidance in distinguishing gastrointestinal from gynecologic malignancy in such cases.

Case Presentation:

A 55-year-old postmenopausal woman presented with progressive abdominal distention, bilateral lower-extremity edema, unintentional weight loss, pelvic pain, dysphagia, nausea, and vomiting. Imaging revealed large-volume ascites and bilateral complex adnexal masses, raising concern for primary ovarian malignancy. Initial paracentesis demonstrated lymphocyte-predominant ascites.

Tumor marker evaluation demonstrated elevated cancer antigen 125 (CA-125) at 75 U/mL, carcinoembryonic antigen (CEA) at 48.3 ng/mL, and markedly elevated cancer antigen 19-9 (CA 19-9) at 3076 U/mL. The CA-125/CEA ratio was approximately 1.5 (<20), favoring a gastrointestinal rather than gynecologic primary. Esophagogastroduodenoscopy identified a gastric mass, and biopsy confirmed metastatic gastric adenocarcinoma. Ascitic fluid cytology was consistent with malignant ascites.

During hospitalization, the patient developed rapidly progressive cytopenias with severe thrombocytopenia refractory to transfusion, worsening anemia, and evolving coagulopathy. Positive heparin-induced thrombocytopenia (HIT) antibodies raised concern for HIT; however, the overall presentation was less consistent with isolated HIT and more suggestive of malignancy-associated disseminated intravascular coagulation or thrombotic microangiopathy. The clinical course was further complicated by intracranial hemorrhage, and the patient was ultimately transitioned to comfort-focused care.

Discussion:

This case illustrates how metastatic gastric adenocarcinoma can closely mimic advanced gynecologic malignancy in postmenopausal women presenting with ascites and adnexal masses. Elevated CA-125 may reinforce an initial gynecologic diagnosis, increasing the risk of diagnostic anchoring.

Tumor marker patterns can provide critical diagnostic guidance in distinguishing gastrointestinal from gynecologic malignancy. In particular, a CA-125/CEA ratio <20 has been associated with gastrointestinal rather than gynecologic malignancy. In this case, the markedly elevated CA 19-9 and low CA-125/CEA ratio were key indicators of a gastrointestinal source and may have prompted earlier endoscopic evaluation.

Additionally, malignancy-associated coagulopathy may further complicate clinical assessment. Although positive HIT antibodies raised concern for heparin-induced thrombocytopenia, the overall presentation was more consistent with malignancy-associated disseminated intravascular coagulation or thrombotic microangiopathy, given profound thrombocytopenia refractory to transfusion, worsening anemia, and evolving coagulopathy.

Conclusion:

Metastatic gastric adenocarcinoma should be considered in postmenopausal women presenting with ascites and adnexal masses, particularly when tumor marker patterns are atypical. Recognition of CA-125/CEA ratio trends and awareness of malignancy-associated coagulopathy are essential to avoid diagnostic delay and guide appropriate management.

Resident/Fellow Clinical Vignette

Hyporeninemic Hypoaldosteronism in a Vietnam Veteran: A Possible Endocrine-Renal Sequela of Agent Orange Exposure

Svetlana Gasparyan

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Background:

Hyporeninemic hypoaldosteronism (HH) is a well-recognized cause of hyperkalemia, most commonly seen in patients with diabetes mellitus and/or chronic kidney disease (CKD). Agent Orange, a herbicide used during the Vietnam War, contains dioxin (TCDD), which has been associated with malignancies, endocrine disorders, and kidney injury. However, its role in hyporeninemic hypoaldosteronism remains poorly understood.

Case Presentation:

A 77-year-old Vietnam veteran with a history of hypothyroidism, hyperlipidemia, chronic back pain, and tremor presented with recurrent episodes of orthostatic hypotension and near-syncope. He had known exposure to Agent Orange during military service and no history of diabetes mellitus, adrenal disease, or use of medications affecting the renin–angiotensin–aldosterone system.

On examination, he had significant orthostatic hypotension without compensatory tachycardia. Laboratory evaluation revealed suppressed plasma renin activity and inappropriately low aldosterone levels, consistent with hyporeninemic hypoaldosteronism. Further workup demonstrated stage 2 CKD with preserved glycemic control (HbA1c 4.9%). Adrenal insufficiency was excluded with an appropriate response to cosyntropin stimulation testing, and pheochromocytoma was ruled out with normal plasma metanephrines.

The patient was treated with fludrocortisone, with subsequent improvement in orthostatic symptoms and normalization of potassium levels.

Discussion:

While CKD is a recognized contributor to HH, the absence of diabetes and other common etiologies in this patient raises concern for an alternative underlying mechanism. Dioxin exposure has been associated with renal injury, including tubular dysfunction, as well as endocrine disruption through activation of the aryl hydrocarbon receptor. These effects may impair both renal handling of electrolytes and aldosterone synthesis.

Epidemiologic data in veterans have demonstrated increased risks of CKD with Agent Orange exposure, independent of traditional risk factors. In this context, the patient's clinical presentation suggests a possible link between dioxin-associated renal injury and disruption of the renin–angiotensin–aldosterone system.

Conclusion:

This case highlights a rare presentation of hyporeninemic hypoaldosteronism in a non-diabetic patient with known Agent Orange exposure. Environmental toxins should be considered in the evaluation of unexplained RAAS abnormalities, particularly in patients without traditional risk factors.

Resident/Fellow Clinical Vignette

Improving Pulmonary Consultation Compliance for Hospitalized Patients Receiving BiPAP Through a Multidisciplinary Quality Improvement Initiative

Ayesha Habib

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Purpose

To improve the rate of timely pulmonary consultations for hospitalized patients initiated on bilevel positive airway pressure (BiPAP) through a multidisciplinary quality improvement (QI) intervention after an electronic medical record (EMR) reflex pulmonary consult order alone failed to improve compliance.

Methods

This single-center QI project was conducted at St. John's Episcopal Hospital between 2024 and 2026. Adult inpatients initiated on BiPAP were included, while canceled BiPAP orders were excluded. Compliance was defined as placement of a pulmonary consultation during the same hospitalization as the initial BiPAP order. A baseline review demonstrated that the previously implemented EMR reflex pulmonary consult order alone failed to improve consultation rates because of persistent workflow barriers, including limited provider awareness, inconsistent recognition of consultation criteria, competing clinical priorities, alert fatigue, difficulty contacting the pulmonary service, and the absence of a standardized process to reinforce early pulmonary involvement.

Beginning in February 2026, a multidisciplinary intervention was implemented to address these barriers. The initiative included brief physician education sessions, laminated consultation algorithms, posters, quick-reference cards, standardized pulmonary consultation criteria, chief resident reminder emails, and daily reinforcement during interdisciplinary team rounds. Education targeted Internal Medicine and Family Medicine residents, hospitalists, emergency physicians, respiratory therapists, nursing leadership, and case managers. Respiratory therapists identified patients receiving BiPAP without pulmonary consultation and notified the primary team, while case managers reinforced early consultation for patients expected to require home BiPAP to facilitate discharge

planning. Pulmonary consultation compliance during the pre-intervention, awareness, and full implementation phases was compared using chi-square analysis.

Results

The prior EMR reflex consult order alone did not improve compliance, with pulmonary consultation rates remaining essentially unchanged (50% vs. 51%). During 2025, compliance averaged 44.8% across 927 inpatient BiPAP encounters, with monthly rates ranging from 34.6% to 54.5%, demonstrating persistent variability without sustained improvement. Following implementation of the multidisciplinary intervention, compliance increased from 45.9% during the pre-intervention period (56/122 patients) to 47.1% during the awareness phase (49/104 patients) and reached 56.2% during full implementation (36/64 patients), representing the highest compliance observed during the study period and a 22% relative improvement from baseline.

Conclusion

A multidisciplinary intervention combining provider education, standardized workflows, visual decision-support tools, and interdisciplinary collaboration improved pulmonary consultation compliance for hospitalized patients initiated on BiPAP, demonstrating the value of combining educational and system-based strategies to strengthen adherence to evidence-based care.

Resident/Fellow Clinical Vignette

Warm Intravenous Fluids as a Novel Adjunctive Therapy for Refractory Cannabinoid Hyperemesis Syndrome

Tatiana Hellwig

Hallie Buddendeck

Mahima Gill

Cannabinoid hyperemesis syndrome (CHS) is characterized by recurrent nausea and vomiting in the setting of chronic cannabis use. Relief with hot showers is a hallmark feature and is thought to result from activation of transient receptor potential vanilloid 1 (TRPV1) receptors. While topical capsaicin has emerged as a treatment option, the role of in-patient warmed intravenous (IV) fluids has not been described.

A 15-year-old female with recent THC use presented with one week of intractable nausea, vomiting, and abdominal pain. She reported temporary relief with prolonged hot baths but failed outpatient antiemetic therapy. Evaluation was notable for a positive urine cannabinoid screen, and she was admitted for presumed CHS. Despite treatment with multiple antiemetics, capsaicin, IV hydration, and supportive care, she continued to experience severe nausea and recurrent emesis requiring readmission shortly after discharge. During her second hospitalization, symptoms remained refractory to conventional therapy. The patient repeatedly reported improvement with heat exposure at home but was unable to obtain similar relief in the hospital setting. Given the established association between heat and symptom improvement in CHS, warmed IV fluids were initiated as an adjunctive measure. Within 24 hours, she experienced complete resolution of emesis and significant improvement in nausea, allowing advancement of oral intake. Although symptoms later fluctuated during her prolonged hospitalization, the temporal relationship between warmed fluid administration and symptomatic improvement was notable, as well as symptoms later worsening again soon after warmed IV fluids were discontinued, further supporting a temporal association between systemic heat exposure and symptom relief.

This case highlights warmed IV fluids as a potential adjunctive therapy for refractory CHS. The observed improvement following initiation of warmed fluids raises the possibility that systemic heat exposure may provide benefit through mechanisms like hot bathing, including TRPV1 receptor activation. While causality cannot be established from a single case, this observation suggests a low-cost, low-risk intervention that warrants further

investigation in patients with refractory CHS, particularly when access to heat-based symptom relief is limited.

Resident/Fellow Clinical Vignette

Cystic Neutrophilic Granulomatous Mastitis: A Diagnostic Challenge in a Young Woman with Persistent Mastitis

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Granulomatous breast disease is a rare benign inflammatory breast disorder first described in 1972. It most commonly affects young parous women during pregnancy or lactation; however, it can also occur in non-lactating women. The disease has been reported more frequently among women of Hispanic and Mediterranean descent. It is characterized by chronic necrotizing granulomatous lobulitis of unknown etiology and often presents a diagnostic challenge because it closely mimics infectious mastitis and even breast malignancy. Cystic neutrophilic granulomatous mastitis (CNGM) is a rare histopathologic subtype that is increasingly recognized and is frequently associated with Gram-positive bacilli, most commonly Corynebacterium species.

We report the case of a 36-year-old Hispanic female who presented with a painful left breast mass associated with persistent breast discharge. Breast ultrasonography demonstrated a focal, poorly defined area of altered architecture with ill-defined echogenicity. An ultrasound-guided core needle biopsy revealed lobulocentric granulomatous inflammation with a mixed inflammatory infiltrate, including numerous neutrophils, along with characteristic oval cystic spaces rimmed by neutrophils. Gram staining demonstrated Gram-positive bacilli within the cystic spaces. These histopathologic findings were unusual for conventional mastitis and raised suspicion for cystic neutrophilic granulomatous mastitis.

Following review of the histopathologic findings and a literature search, a diagnosis of CNGM was considered. The patient was initially treated with doxycycline for 14 days. However, at follow-up she reported worsening pain, and the breast mass had increased in size. Prednisone therapy was subsequently initiated because of concern for an ongoing inflammatory process. Despite one month of treatment with antibiotics and corticosteroids, she experienced only minimal clinical improvement. She was therefore referred to the Infectious Disease and Rheumatology services for further evaluation and management. Given her persistent symptoms and inadequate response to initial therapy, methotrexate was initiated.

At subsequent follow-up visits, the patient reported significant improvement, including a marked reduction in the size of the breast mass and complete resolution of the breast discharge. She continues to be closely monitored and has expressed substantial symptomatic relief with her current treatment regimen.

This case highlights the importance of recognizing CNGM as a distinct clinicopathologic entity in patients with persistent mastitis that fails to respond to conventional antibiotic therapy. Early recognition of its characteristic histopathologic features, particularly the presence of neutrophil-lined cystic spaces containing Gram-positive bacilli, can facilitate prompt diagnosis and appropriate multidisciplinary management. Increased awareness of CNGM among clinicians and pathologists may help reduce delays in diagnosis, avoid unnecessary interventions such as surgery, and improve patient outcomes by guiding timely initiation of immunomodulatory therapy when indicated.

Resident/Fellow Clinical Vignette

A Diagnostic Pitfall in the GLP-1 Era: Tirzepatide-Associated Gastrointestinal Symptoms Obscuring Occult Gastroesophageal Junction Cancer

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Introduction:

Tirzepatide commonly causes gastrointestinal side effects including nausea, diarrhea, and abdominal discomfort. Patients are counseled to expect these symptoms upon initiation of the medication. Clinicians anticipate these side effects and are not surprised when they are reported by patients. Given that gastrointestinal symptoms occur in a variety of conditions and as a side effect of many medications, this raises concern for GLP-1 RA-induced gastrointestinal symptoms masking and delaying recognition of potentially serious conditions such as malignancy (1).

Case Presentation:

A 44 year old female with history of GERD, Diabetes, started on Tirzepatide 3 months prior, presented to PCP with upper abdominal pain ongoing for 1 week. The pain was sharp, constant, 9/10, radiating to the back, and improved with passing gas/ bowel movement. Patient also reported nausea and loose stools for 1 month, and weight loss since starting Tirzepatide. Labs and CT abdomen were ordered but not completed.

15 days later, patient presented to the ED with worsening pain, green diarrhea, and inability to tolerate food due to severe nausea. CT Abdomen showed mild mucosal thickening of the distal esophagus and small bowel, moderate ascites, bilateral adnexal cystic lesions, cholelithiasis, and right hydronephrosis without an obstructing stone. Home Pantoprazole dose was increased and an urgent referral to outpatient GI was made.

12 days later, GI suspected GLP-1 induced gastroparesis. The dose of Tirzepatide was lowered. Additionally, Pantoprazole was switched to Esomeprazole.

Further investigations by PCP included an US pelvis which showed bilateral ovarian cysts (hemorrhagic cysts vs ovarian neoplasm). Patient was referred to outpatient Oncology.

One month after the first ED visit, patient presented to the ED with worsening abdominal pain, nausea, vomiting, and abdominal distension. Patient was admitted. CT Abdomen showed similar findings to previous, plus large ascites and heterogeneous omental nodularity. CT Chest showed small pericardial and left pleural effusions, and cardiophrenic lymph node enlargement. US gallbladder showed cholelithiasis without cholecystitis. MRCP was consistent with CT and US findings. EGD showed large, nodular, ulcerated, and friable lesion in the gastroesophageal junction causing decreased dispensability. Diagnostic and therapeutic paracenteses were done. Cytology indicated malignancy. Omental biopsy was consistent with gastroesophageal junction primary HER 2 3+ adenocarcinoma. Patient was started on palliative chemotherapy, however due to development of AKI and intolerance of hemodialysis, patient transitioned to comfort care, and subsequently passed away.

Discussion and Conclusion:

Gastrointestinal side effects are common following initiation of GLP-1 receptor agonists such as Tirzepatide. However, these symptoms should not be automatically attributed to medication effects. Persistent or progressive symptoms warrant evaluation to exclude potentially fatal conditions, in this case malignancy. Further studies are needed to better understand clinical presentations and outcomes in patients prescribed GLP-1–based therapies who may have more serious underlying conditions.

Resident/Fellow Clinical Vignette

To Screen or Not to Screen Asymptomatic H. pylori Infection in a High-Risk Urban Setting

Fatima Khan

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Background

Helicobacter pylori (HP) causes gastric cancer (GC) but is often asymptomatic, so infection can go undetected and untreated, particularly in high-risk populations such as immigrants from high-incidence regions. Early detection improves prognosis and helps prevent transmission, yet no U.S. guidelines recommend routine screening of asymptomatic patients for HP. We conducted a prospective cohort study in an urban primary care population to determine HP prevalence and associated risk factors and to evaluate an investigational point-of-care serologic test (POCT) that detects HP antibodies, compared with stool antigen testing (SAT), which detects active infection.

Methods

We recruited adults > 18 years from three primary care clinics in Brooklyn, NY. Participants completed a questionnaire on clinical history and symptoms, then underwent POCT testing. We asked all participants, regardless of POCT result, to collect and submit a stool sample for confirmatory SAT. We treated SAT-positive patients for HP and retested them to confirm eradication.

Results

We enrolled 296 patients between August 2024 and March 2026; all underwent POCT testing, and 58% were asymptomatic. Among asymptomatic patients, 35% (n=61) completed stool collection for confirmatory SAT, and this interim analysis is limited to those 61 patients with paired POCT and SAT results.

SAT confirmed active HP infection in 34% of this subgroup. Among SAT-positive patients, 62% were under age 50 and 33% were US-born. Using SAT as the reference standard, the POCT showed a sensitivity of 81%, specificity of 70%, a positive predictive value (PPV) of 59%, and a negative predictive value (NPV) of 87.5%.

Conclusion

In this interim analysis, active HP infection was present in one-third of asymptomatic patients who completed stool testing. Ongoing enrollment and confirmatory stool antigen testing will increase the number of paired samples, allowing more precise estimates of H. pylori prevalence and associated risk factors for asymptomatic infection in this urban primary care population.

Acknowledgements

This study was awarded a grant by the Maimonides Research Development Foundation. Figurelabs was used to create the illustration in Figure 1.

Table 1. Patient Demographics with SAT and POCT Results

Characteristic	Overall (N=61)	SAT+ (N=21)	POCT+ (N=29)
Age <50, n (%)	33 (54.1)	13 (61.9)	17 (58.6)
US immigrant, n (%)	40 (65.6)	14 (66.7)	23 (79.3)
Female, n (%)	34 (55.7)	12 (57.1)	18 (62.0)

Abbreviations: SAT = stool antigen test; POCT = investigational serologic point-of-care test.

Resident/Fellow Clinical Vignette

Partial Splenic Artery Embolization as a Bridge to Splenectomy in Refractory Immune Thrombocytopenia

Umair Khan

Background

Immune thrombocytopenia (ITP) is an autoimmune disorder characterized by platelet destruction and impaired platelet production. Though several medical therapies are available and well tolerated, some patients develop refractory disease complicated with critical bleeding. While splenectomy is often the final step and can provide disease control, in settings of severe thrombocytopenia and bleeding it can be unsafe. Partial splenic artery embolization may provide a safe transition point and can serve as a bridge to definitive splenectomy.

Case Presentation:

We present a case of a 78-year old man with extreme refractory immune thrombocytopenia whose case was complicated by spontaneous hemorrhages requiring partial splenic embolization as a bridge to total splenectomy. The patient was first diagnosed with ITP in May 2025 when he first presented to his PCP for gum bleeding with platelets of 17,000. He was initially treated with dexamethasone and IVIG with response, however within a week his counts had returned. His initial bone marrow biopsy revealed no acute pathology only showing increased megakaryocytic consistent with ITP. During the following six months, he remained refractory to repeated courses of pharmacologic management. A repeat bone marrow biopsy was done in January 2026 which was still consistent with ITP and his platelets at that time were 2,000. After the second bone marrow biopsy, the patient agreed to follow up with surgical oncology and was scheduled for planned splenectomy.

Before his operation, he presented in March 2026 noting weakness and difficulty ambulating. His platelets at the time were 3,000 and imaging revealed a posterior fossa subdural hematoma as well as a spinal canal hemorrhage causing severe compression at the L3-L5 region. Given his severe thrombocytopenia and active bleed the patient was precluded from immediate splenectomy. He was given several doses of emergency dexamethasone and IVIG which transiently peaked his platelets to 26,000 and Interventional Radiology was brought in for urgent IR embolization in which patient underwent embolization resulting in approximately 30-40% of splenic devascularization. Within 24 hours patient’s platelets went from 26,000 to 96,000 and patient was discharged

with a platelet count of 128,000, He underwent complete splenectomy April 2026 and his platelets have since remained normal.

Discussion

This case illustrates a challenging presentation of treatment-refractory ITP complicated by spontaneous intracranial and spinal canal hemorrhages. Despite numerous medical therapies the patient's platelet count remained critically low leading to consideration of surgery. In cases of acute complications, the role of splenic artery embolization may represent an effective rescue strategy and bridge to surgery in temporarily poor operative candidates. This case also demonstrates the importance of early collaboration among hematology, interventional radiology, surgery, and neurosurgery when critical bleeding limits conventional treatment options.

Resident/Fellow Clinical Vignette

When Seizures Aren't Seizures: Convulsive Syncope as The Initial Presentation of Megestrol Induced Secondary Adrenal Insufficiency

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Introduction

Megestrol acetate is commonly prescribed as an appetite stimulant but possesses glucocorticoid receptor activity that can suppress the hypothalamic-pituitary-adrenal (HPA) axis, resulting in secondary adrenal insufficiency. Although this adverse effect is recognised, presentation as convulsive syncope mimicking epilepsy is rare and may delay diagnosis.

Case Presentation

A 27-year-old woman with chronic migraine and no prior history of epilepsy presented with recurrent episodes of left-hand shaking followed by transient loss of consciousness. Episodes were preceded by dizziness and generalized weakness. Witnesses described rhythmic head and shoulder movements without tongue biting, urinary incontinence, or postictal confusion. Orthostatic vital signs demonstrated significant hypotension (blood pressure 115/67 mmHg supine to 90/68 mmHg standing) with compensatory tachycardia. Neurological examination was otherwise unremarkable.

Non-contrast head CT showed no acute intracranial abnormality, and continuous video electroencephalography captured recurrent events without epileptiform activity. Given persistent orthostatic hypotension despite a negative neurological evaluation, endocrine testing was pursued. Morning serum cortisol was undetectable (<0.4 µg/dL), and cosyntropin stimulation demonstrated an inadequate cortisol response (30-minute cortisol 10 µg/dL; 60-minute cortisol 11 µg/dL), confirming adrenal insufficiency. Further history revealed initiation of megestrol acetate 20 mg twice daily one month earlier for appetite stimulation, followed by amenorrhea. Megestrol was discontinued, physiologic hydrocortisone replacement was initiated, and the patient experienced resolution of orthostatic symptoms without further seizure-like episodes. The final diagnosis was megestrol-induced secondary adrenal insufficiency presenting as convulsive syncope.

Discussion

Megestrol-induced secondary adrenal insufficiency is an uncommon but reversible cause of orthostatic hypotension and convulsive syncope. Cerebral hypoperfusion during syncope may produce involuntary motor activity that closely resembles epileptic seizures, leading to extensive neurological evaluation and delayed recognition of the underlying endocrine disorder. This case highlights the importance of obtaining orthostatic vital signs, performing a thorough medication review, and considering adrenal insufficiency in patients with seizure-like episodes accompanied by normal neuroimaging and electroencephalography. Early diagnosis and glucocorticoid replacement resulted in rapid clinical improvement, avoided unnecessary antiseizure therapy, and facilitated appropriate endocrine follow-up.

Resident/Fellow Clinical Vignette

How Nocturnist–Resident Collaboration Prevented a Missed Diagnosis of Recurrent Hemothorax: A Case Highlighting Supervision Without Loss of Autonomy

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Purpose:

To illustrate how nocturnist oversight facilitated recognition of recurrent hemothorax during overnight clinical deterioration.

Case Presentation:

A 68-year-old man hospitalized with empyema and hemothorax underwent video-assisted thoracoscopic surgery (VATS), with a course complicated by respiratory failure requiring tracheostomy and kidney injury. Following stabilization, he was transferred to the medicine service.

The patient developed dyspnea and hypotension, with blood pressure declining from 154/95 mmHg to 86/59 mmHg overnight. Chest radiography demonstrated complete opacification of the left hemithorax with rightward tracheal deviation. Despite oxygen saturation of 100% on 4 L oxygen, he became progressively fatigued. Examination revealed left chest wall swelling, and hemoglobin decreased from 7.5 g/dL to 6.7 g/dL.

The resident reviewed the imaging and suspected recurrent pleural fluid accumulation and hemothorax. Thoracic surgery was consulted, and the covering surgical resident favored mucus plugging and considered bronchoscopy. Given persistent diagnostic uncertainty, the nocturnist reviewed the case. Reassessment of the clinical presentation, imaging, and hemodynamics shifted concern toward hemothorax. Bedside ultrasound evaluation supported a large pleural fluid collection, prompting ICU consultation. A pigtail catheter evacuated approximately 300 mL of serosanguinous fluid, confirming recurrent hemothorax with subsequent hemodynamic improvement.

Discussion:

The significance of this case lies less in the diagnosis than in how it was recognized. Faced with a complex postoperative patient and competing explanations, the resident team

appropriately generated a differential diagnosis, sought subspecialty input, and recognized the need for additional guidance. Because a nocturnist was immediately available on-site, attending-level input was obtained at a critical decision point. Collaborative review of the patient's hypotension, worsening anemia, chest wall findings, and radiographic abnormalities refined the differential and expedited escalation of care.

The nocturnist did not supplant resident decision-making. The residents identified the deterioration, interpreted the imaging, consulted thoracic surgery, and initiated the evaluation. The nocturnist provided additional diagnostic perspective and supervision during a high-risk scenario. This mirrors findings by Haber et al., who demonstrated that after nocturnist implementation residents contacted attendings more frequently during adverse events and before major management decisions without reducing perceived autonomy.

Although studies have shown mixed effects of nocturnist staffing on mortality, readmissions, length of stay, and medical error rates, such outcomes may not fully capture the benefits of overnight attending presence. Cases such as this suggest that nocturnists may exert their greatest impact through enhanced clinical decision-making at critical junctures, prevention of diagnostic delay, and supervision that strengthens rather than diminishes trainee autonomy.

Conclusion:

This case demonstrates how nocturnist-resident collaboration facilitated timely recognition of recurrent hemothorax during overnight decompensation. The value of nocturnist staffing may be best reflected in improved clinical decision-making, near-miss prevention, and effective supervision without sacrificing resident autonomy.

Resident/Fellow Clinical Vignette

PARADOXICAL EMBOLISM THROUGH A PATENT FORAMEN OVALE PRESENTING AS ALTERED MENTAL STATUS AND EMBOLIC STROKE IN THE SETTING OF EXTENSIVE DVT AND SADDLE PULMONARY EMBOLISM

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INTRODUCTION: Patent foramen ovale (PFO) is a congenital cardiac defect present in roughly 25% of adults. When right atrial pressure exceeds left atrial pressure, a PFO can permit right-to-left shunting, allowing venous emboli to cross into the systemic circulation, a paradoxical embolism. Most patients with a PFO remain asymptomatic and require no intervention, but recognized complications include cryptogenic stroke. We present a case in which a PFO served as the conduit for a paradoxical embolic stroke in a patient with concurrent extensive deep vein thrombosis (DVT) and saddle pulmonary embolism (PE).

CASE PRESENTATION: A 61-year-old woman with Alzheimer's dementia (baseline AOx1), hypertension, hyperlipidemia, type 2 diabetes, a chronic indwelling Foley catheter, and a recent hospitalization for falls and Aerococcus viridans bacteremia presented with acutely worsened altered mental status. D-dimer was markedly elevated at 7,650 ng/mL. CT angiography of the chest demonstrated a saddle PE with bilateral main, segmental, and sub segmental pulmonary artery involvement without right heart strain . Lower extremity venous duplex revealed acute DVT of the left common femoral, popliteal, and gastrocnemius veins. The patient was heparinized; Vascular Surgery and ICU were consulted, and the patient was managed on telemetry without need for acute surgical or critical care intervention. Given her altered mental status, further workup included brain MRI without contrast, which showed scattered foci of restricted diffusion consistent with acute-to-subacute embolic infarcts, and electroencephalogram, which showed mild-to-moderate multifocal cerebral dysfunction. Neurology and Neurosurgery were consulted; no acute neurosurgical intervention was indicated. Transthoracic echocardiogram showed preserved LVEF (55-60%) without right heart strain, but transesophageal echocardiogram identified a PFO , establishing the likely mechanism of paradoxical embolic stroke in the setting of venous thromboembolic burden. Cardiology was consulted and felt the patient

was a candidate for PFO closure; however, given the extent of her acute DVT/PE burden and underlying dementia, the decision was made to pursue medical management with anticoagulation rather than proceed with closure.

CONCLUSION: This case illustrates that in patients presenting with embolic stroke and concurrent venous thromboembolism, a PFO should be considered as a potential conduit for paradoxical embolization, particularly when no alternative arterial source is identified. Diagnosis requires a high index of suspicion and dedicated imaging (transesophageal echocardiogram), as transthoracic echocardiography alone may be insufficient. Management must be individualized, weighing PFO closure against medical therapy, based on the patient's overall thromboembolic burden, comorbidities, and goals of care.

Resident/Fellow Clinical Vignette

Undiagnosed Systemic Lupus Erythematosus in the Postpartum Period Presenting with Cardiomyopathy and Class IV Lupus Nephritis: A Case Report

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Background:

Peripartum cardiomyopathy (PPCM) is a rare cause of systolic heart failure that occurs during the last month of pregnancy and up to five months postpartum. The etiology of this condition is multifactorial and incompletely understood, with proposed contributions from genetic, hemodynamic, hormonal, and immune-mediated factors. Systemic lupus erythematosus (SLE) has been associated with PPCM and may present with overlapping clinical features, including myocarditis that can closely mimic PPCM.

We present a female patient who developed severe left ventricular systolic dysfunction three months postpartum in the setting of previously undiagnosed SLE with class IV lupus nephritis.

Case Presentation

A 25-year-old G1P1 female with a history of preeclampsia presented three months postpartum with progressive dyspnea on exertion, orthopnea, and worsening bilateral lower extremity edema, associated with headaches and elevated blood pressure readings at home. The patient had not been adherent to prescribed labetalol therapy and lacked primary care follow-up. On arrival, she was severely hypertensive (173/124 mmHg), with bilateral 2+ pitting edema without focal pulmonary findings.

Laboratory evaluation revealed anemia (hemoglobin 10 g/dL), hypoalbuminemia, and nephrotic-range proteinuria with a urine protein-to-creatinine ratio of 12.8 g/g. NT-proBNP was >8,000 pg/mL. Chest imaging showed increased pulmonary vascular markings, and the echocardiogram showed a left ventricular ejection fraction of 25–30% with severe global hypokinesis, grade I diastolic dysfunction, and a small pericardial effusion.

Further testing revealed positive antinuclear, anti–double-stranded DNA, and anticardiolipin antibodies, meeting EULAR/ACR criteria for SLE with renal involvement. Retrospective chart review revealed prior urinalysis several months earlier, demonstrating

significant proteinuria that had not been previously addressed, suggesting pre-existing renal pathology. Kidney biopsy was done, confirming class IV lupus nephritis.

Taken together, these findings established a diagnosis of previously unrecognized SLE with Class IV lupus nephritis presenting concurrently with severe left ventricular systolic dysfunction, most consistent with PPCM.

Discussion

This case highlights the diagnostic challenge of new-onset systolic dysfunction in the postpartum period. In this patient, the coexistence of severe left ventricular dysfunction and nephrotic-range proteinuria raises the possibility of SLE, which may initially raise concern for PPCM. Cardiac manifestations of SLE, including myocarditis and microvascular injury, can closely resemble PPCM, and distinguishing between these diseases remains challenging in the absence of advanced cardiac imaging or histopathology, as was the case in our patient.

In this case, the presence of biopsy-confirmed Class IV lupus nephritis, positive autoimmune serologies, and evidence of previously unrecognized proteinuria strongly suggests underlying active SLE preceding the onset of cardiac dysfunction. Given that SLE activity may persist or flare during pregnancy and the postpartum period, unrecognized autoimmune disease may have contributed to the patient's clinical presentation.

Recognition of potential autoimmune etiologies is crucial, as SLE-associated cardiac dysfunction may require immunosuppressive therapy in addition to guideline-directed heart failure management.

Resident/Fellow Clinical Vignette

Case Report: B-Cell Acute Lymphoblastic Leukemia Transformation in a 71-Year-Old Woman with Longstanding Polycythemia Vera

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Introduction: Polycythemia Vera (PV) is a myeloproliferative neoplasm (MPN) characterized by the overproduction of erythrocytes, typically driven by the JAK2 V617F mutation. While PV often progresses to myelofibrosis (MF) or transforms into acute myeloid leukemia (AML), transformation into acute lymphoblastic leukemia (ALL) is an exceedingly rare phenomenon, with only three cases reported in the last decade.

Case Presentation: A 71-year-old woman with a known history of JAK2-positive PV, previously managed with phlebotomy and hydroxyurea, presented to the emergency department with chest and epigastric pain. Clinical evaluation revealed stable vitals but significant laboratory findings, including a hemoglobin drop to 7.2 g/dL and hepatosplenomegaly on CT imaging. Although initial concerns favored transformation to myelofibrosis due to enlarging splenic lesions, a bone marrow biopsy (BMBx) confirmed a diagnosis of B-cell acute lymphoblastic leukemia (B-ALL). The patient was subsequently initiated on B-cell targeted therapy.

Conclusion: This case highlights a rare and enigmatic clinical progression of PV. The transformation into a lymphoid malignancy rather than the typical myeloid path underscores the complex clonal evolution of MPNs and the necessity for bone marrow evaluation in PV patients presenting with sudden clinical deterioration or cytopenias.

Resident/Fellow Clinical Vignette

A Rash Conclusion: When Thrombocytopenia Wasn't Drug-Induced

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Introduction

Drug-induced Thrombocytopenia (DITP) is primarily a clinical diagnosis supported by temporal association, exclusion of alternative causes, and platelet recovery after drug withdrawal. Failure of platelet recovery should prompt reassessment for alternative etiologies. Toxic Epidermal Necrolysis (TEN) causes profound immune dysregulation, and while secondary immune thrombocytopenia (ITP) is associated with autoimmune disease, lymphoproliferative disorders, and chronic infection, its co-occurrence with TEN is rarely reported. We present a case of severe ITP during TEN initially mistaken for DITP, highlighting diagnostic challenges of thrombocytopenia in critical illness.

Case Presentation

A 50-year-old male with hypertension and end-stage renal disease (ESRD) on hemodialysis presented with hypotension, fever, and diffuse erythematous pruritic rash. His labs showed leukocytosis (15.1) and eosinophilia (23%). He received empiric antibiotics and vasopressors for septic shock. As the rash progressed to involve mucosal surfaces, biopsy confirmed adverse drug reaction, with body surface area involvement consistent with TEN. Dialysis catheter-tip culture was negative post removal and he received intravenous immunoglobulin (IVIG).

By week four, platelets dropped to a nadir of 0–3 K/µL, without schistocytes on peripheral smear. We excluded common etiologies of thrombocytopenia, including thrombotic thrombocytopenic purpura and heparin-induced thrombocytopenia. Given exposure to multiple thrombocytopenia-associated medications, including vancomycin, cefepime, and piperacillin-tazobactam, DITP was suspected, with all medications discontinued. Despite multiple courses of IVIG, steroids, and platelet transfusions, platelets did not increase. Bone marrow biopsy showed increased morphologically normal megakaryocytes, indicating immune-mediated peripheral destruction. Persistence beyond the expected DITP timeline and concurrent immune dysregulation from TEN, the diagnosis was recategorized as secondary ITP. He was treated with romiplostim and rituximab, with gradual platelet recovery.

Discussion

In critically-ill patients, thrombocytopenia presents a diagnostic challenge, requiring differentiating DITP, secondary to multidrug therapy, from secondary ITP caused by underlying immune dysregulation during critical illness. In our case, medication timing initially supported DITP, but failure of recovery after drug discontinuation, despite accounting for delayed clearance in ESRD, prompted re-evaluation. Whereas TEN is typically drug-induced, the same medication could theoretically precipitate both the rash and thrombocytopenia. Bone marrow biopsy showed preserved trilineage hematopoiesis with increased morphologically normal megakaryocytes, supporting peripheral destruction rather than marrow failure or drug-related suppression.

Failure of first-line ITP therapies (IVIG, steroids) prompted second-line treatment, with platelets improving after rituximab and romiplostim. Hence, the overall clinical course was consistent with secondary ITP requiring second-line therapy4. This case underscores the importance of reassessing diagnostic assumptions when expected recovery does not occur in patients with overlapping risk factors and severe inflammatory states, as recognition of secondary ITP may enhance improved outcomes.

Conclusion

Persistent thrombocytopenia despite withdrawal of suspected medications should prompt reconsideration of DITP and evaluation for alternative immune-mediated etiologies. In patients with TEN, secondary ITP is a challenging diagnosis requiring prompt recognition and intervention.

Resident/Fellow Clinical Vignette

CASCADING DEFICIENCIES: A CASE OF INTRACTABLE VOMITING IN ADRENAL INSUFFICIENCY AND WERNICKE'S ENCEPHALOPATHY

Grace Macmillan

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A 41-year-old female with Cushing's disease secondary to pituitary adenoma status post transsphenoidal resection complicated by adrenal insufficiency presented with altered mental status, weakness, poor oral intake, and persistent nausea and vomiting for one week. She had missed multiple doses of chronic steroid therapy because of ongoing emesis and was recently hospitalized for suspected steroid withdrawal.

Examination was notable for tachycardia, confusion, nystagmus, and ophthalmoplegia. Computed tomography imaging showed no acute abnormalities. Differential diagnoses included metabolic encephalopathy, meningitis, and toxic-metabolic encephalopathy.

The patient was empirically started on broad-spectrum antimicrobial therapy while further evaluation was pursued. Stress-dose corticosteroids were initiated for suspected adrenal crisis. Lumbar puncture demonstrated elevated cerebrospinal fluid protein with otherwise unremarkable studies and negative meningitis-encephalitis PCR testing. Additional infectious workup was unrevealing. Electroencephalography demonstrated generalized slowing without epileptiform activity.

Magnetic resonance imaging of the brain revealed symmetric hyperintensities involving the bilateral medial thalami, mammillary bodies, and periaqueductal gray matter, findings suggestive of Wernicke's encephalopathy. Given the patient's prolonged vomiting and poor nutritional intake, severe thiamine deficiency was suspected. High-dose intravenous thiamine replacement was initiated with partial improvement in mentation and neurologic symptoms.

Thiamine deficiency can develop rapidly in patients with prolonged vomiting and malnutrition. Wernicke's encephalopathy classically presents with altered mental status, gait ataxia, and oculomotor dysfunction, although the full triad is often absent. While commonly associated with alcohol use disorder, this case demonstrates an uncommon presentation related to adrenal insufficiency and persistent emesis. Delayed recognition may result in irreversible neurologic injury and progression to Korsakoff syndrome.

This case highlights the importance of maintaining suspicion for thiamine deficiency in patients with adrenal insufficiency and prolonged vomiting presenting with encephalopathy. Early recognition and prompt thiamine replacement are essential to prevent permanent neurologic sequelae.

Resident/Fellow Clinical Vignette

STEMI with Multi-Territory Thrombosis and Transient Lupus Anticoagulant: The 12-Week Anticoagulation Dilemma

Brian Macutay

Dr. Brett Chen, MD

Acute myocardial infarction in young adults with thrombotic burden disproportionate to underlying atherosclerotic disease demands evaluation for hypercoagulable states, yet the choice of anticoagulant, vitamin K antagonist (VKA) versus direct oral anticoagulant (DOAC), is consequential given the inferiority of DOACs in confirmed antiphospholipid syndrome (APS) with arterial events. No existing guideline addresses antithrombotic management during the 12-week confirmatory window required to establish or exclude APS.

A 29-year-old male with hypertension, obesity, and family history of premature coronary artery disease presented with STEMI with diffuse ST elevations across inferior and precordial leads, consistent with a wrap-around LAD territory infarction. Urgent left heart catheterization revealed single-vessel disease with extensive thrombotic burden involving the proximal and apical LAD and ostial diagonal branch. He underwent aspiration thrombectomy with intracoronary nitroprusside; repeat catheterization with optical coherence tomography at 48 hours demonstrated thrombus resolution with residual 35% stenosis and underlying plaque. He subsequently developed acute lower extremity posterior tibial artery occlusion with preserved perfusion to the ankle level. Initial lupus anticoagulant testing, drawn prior to anticoagulation, was mildly positive, prompting heparin bridging to warfarin. Same-day repeat testing on heparin was negative, a result that cannot be considered definitive, as heparin is a recognized interferent with clot-based lupus anticoagulant assays. Comprehensive thrombophilia evaluation, including anticardiolipin antibodies, anti-beta-2-glycoprotein I antibodies, and genetic testing, was otherwise unremarkable. Given the negative repeat and negative ELISA-based antibodies, the patient was transitioned from warfarin to apixaban with clopidogrel, tailored to his residual coronary stenosis and ongoing peripheral arterial occlusion while awaiting confirmatory testing at 12 weeks. Echocardiography demonstrated an ejection fraction of 47% with subsequent improvement to normal systolic function.

This case highlights the therapeutic uncertainty created when a young patient presents with STEMI and multi-territory arterial thrombosis in the setting of transiently positive lupus anticoagulant. The antithrombotic strategy was iteratively adjusted as diagnostic

information evolved, from warfarin to apixaban with clopidogrel, itself a reflection of the guideline gap this report identifies. Meta-analytic data demonstrate a fivefold increase in arterial thrombotic events with DOACs compared to VKA in APS, yet committing to warfarin based on a single positive result that may prove transient imposes monitoring burden and bleeding risk without a confirmed indication. Prospective guidance for antithrombotic management during the APS diagnostic evaluation period is needed.

Resident/Fellow Clinical Vignette

HIDDEN IN PLAIN SIGHT: CT DIAGNOSIS OF DELAYED PACEMAKER LEAD PERFORATION DESPITE NORMAL DEVICE FUNCTION

Mohamed Mansour

Muhammed Iftikhar, MD, Mohamed Elhennawi, MD , Ahmed Ahmed, MD, Roshan Karki, MD

Introduction:

Cardiac perforation is a recognized complication of cardiac implantable electronic devices, with a reported incidence of 0.1–5.2%. Delayed perforation, occurring more than 30 days after implantation, is increasingly recognized and may be asymptomatic in up to 15% of cases, with device interrogation parameters often remaining deceptively normal. We report a case of incidentally discovered delayed right ventricular lead perforation identified on chest CT obtained for an unrelated indication.

Case Presentation:

An 87-year-old man with chronic obstructive pulmonary disease and complete heart block underwent dual-chamber pacemaker implantation with an active-fixation lead. Approximately three months later, he presented with two weeks of progressive dyspnea and cough without chest pain or signs of volume overload. He was hemodynamically stable. Laboratory studies demonstrated mild neutrophilic leukocytosis, while troponin I was mildly elevated (98 ng/L, decreasing to 88 ng/L). Electrocardiography showed sinus rhythm without ischemic changes, and chest radiography confirmed appropriate lead position. Non-contrast chest CT obtained to evaluate his respiratory symptoms unexpectedly demonstrated the right ventricular lead traversing the myocardial wall into the pericardial space. Electrophysiology was consulted urgently. Transthoracic echocardiography demonstrated a left ventricular ejection fraction of 42% without pericardial effusion, although the lead was not directly visualized. Device interrogation revealed an RV capture threshold of 2.25 mV, only minimally increased from a previous value of 2.125 mV, with low RV pacing dependence (1.4%). Given his hemodynamic stability, absence of pericardial effusion or attributable symptoms, and minimal pacing dependence, a multidisciplinary team elected conservative management with close clinical and echocardiographic follow-up rather than lead extraction.

Discussion:

This case highlights two important diagnostic challenges. First, perforation occurred three months after implantation, emphasizing that delayed lead perforation should remain in the

differential diagnosis well beyond the traditional 30- to 45-day period, particularly in patients with recognized risk factors such as advanced age and active-fixation leads. Second, the nearly unchanged capture threshold demonstrates that device interrogation alone cannot reliably exclude perforation. Cross-sectional imaging, particularly CT, remains the most sensitive and specific diagnostic modality. In carefully selected asymptomatic, hemodynamically stable patients without pericardial effusion or significant pacing dependence, conservative management with close multidisciplinary surveillance represents a reasonable alternative to immediate lead extraction.

Conclusion:

Delayed pacemaker lead perforation may be clinically silent despite reassuring device interrogation findings. Maintaining a high index of suspicion and a low threshold for cross-sectional imaging, coupled with multidisciplinary decision-making, is essential for timely diagnosis and individualized management.

Resident/Fellow Clinical Vignette

A TTP-Like Thrombotic Microangiopathy in Sickle Cell Disease: A Diagnostic and Therapeutic Dilemma, a Case Report

Mohammad Mansour

Lara Castaneda, Mohammad Mansour, MD, Alexander Feldberg, DO, Susan Collins, MD

Introduction:

Thrombotic microangiopathy (TMA) is a rare but life-threatening complication of sickle cell disease that can closely mimic thrombotic thrombocytopenic purpura (TTP). Diagnosis is particularly challenging because chronic hemolysis and overlapping sickle cell complications may obscure the clinical features of TTP. Both conditions can present with microangiopathic hemolytic anemia, thrombocytopenia, neurologic dysfunction, and end-organ injury, making early recognition and treatment critical.

Case Presentation:

A 70-year-old man with hemoglobin SS disease and chronic inflammatory demyelinating polyneuropathy treated with intravenous immunoglobulin presented with diffuse chest, back, and extremity pain typical of prior vaso-occlusive episodes. He was hypertensive but afebrile and initially oxygenating normally on room air. Laboratory testing showed hemoglobin 9.0 g/dL, platelets 89,000/µL, creatinine 1.23 mg/dL, lactate dehydrogenase 1,247 U/L, and total bilirubin 9.6 mg/dL. Chest radiography showed no infiltrate, and he was admitted for vaso-occlusive crisis.

Within 48 hours, he developed worsening chest pain, increasing oxygen requirements, leukocytosis, and a new left lower-lobe infiltrate concerning for acute chest syndrome. His hemoglobin fell to 5.6 g/dL, platelets to 30,000/µL, and creatinine rose to 2.50 mg/dL. Peripheral smear demonstrated schistocytes, lactate dehydrogenase increased to approximately 2,500 U/L, and haptoglobin was undetectable. His course was further complicated by gross hematuria and progressive encephalopathy.

Red-cell exchange was initiated, but multiple alloantibodies limited the procedure to four units of least-incompatible, sickle-negative packed red blood cells. The combination of microangiopathic hemolytic anemia, severe thrombocytopenia, acute kidney injury, and encephalopathy raised concern for TTP versus sickle cell disease-associated TMA. ADAMTS13 testing could not be completed because the specimen clotted. Given the high clinical suspicion and progressive multisystem involvement, therapeutic plasma exchange

was initiated empirically without awaiting confirmatory testing. He subsequently recovered neurologically and was discharged home approximately three weeks later.

Discussion:

This case illustrates the difficulty of distinguishing TTP from sickle cell disease-associated TMA. Chronic hemolysis limits the specificity of traditional TMA markers, while overlapping complications of sickle cell disease further obscure the diagnosis. Although severe ADAMTS13 deficiency remains the diagnostic hallmark of TTP, testing may be delayed or unobtainable. In this patient, progressive encephalopathy, worsening thrombocytopenia, microangiopathic hemolysis, and acute kidney injury supported empiric therapeutic plasma exchange while red-cell exchange addressed the concurrent sickle cell crisis and acute chest syndrome. His recovery highlights the importance of initiating treatment based on clinical suspicion when definitive testing cannot be obtained promptly.

Conclusion:

Clinicians should maintain a high index of suspicion for TMA in patients with sickle cell disease who develop rapidly progressive thrombocytopenia, hemolysis, neurologic dysfunction, or end-organ injury. When TTP remains a serious diagnostic consideration, treatment should not be delayed while awaiting definitive testing.

Resident/Fellow Clinical Vignette

BIOPSY-PROVEN PEMBROLIZUMAB-ASSOCIATED ACUTE INTERSTITIAL NEPHRITIS FOLLOWING RECENT PROTON PUMP INHIBITOR EXPOSURE

Mohamed Mansour

Haris Jawaid, MD; Adithya Nagendran, MD; Ahmed Zyara, MBBS; Faisal Pervaiz, MD; Michael LaBuzetta, MD; Anjali Satoskar, MD; Galina Mikhalina, MD

Introduction:

Immune checkpoint inhibitors (ICIs) have transformed cancer therapy but can cause immune-related adverse events involving virtually any organ system. Acute interstitial nephritis (AIN) is the most common cause of immune-mediated kidney injury in patients receiving ICIs, although it occurs in only 2–5% of patients receiving monotherapy. We report a case of biopsy-proven pembrolizumab-associated AIN in a patient with pre-existing chronic kidney disease (CKD) and recent proton pump inhibitor (PPI) exposure, highlighting the diagnostic challenges of this presentation.

Case Presentation:

An 85-year-old man with metastatic oropharyngeal squamous cell carcinoma, CKD, hypertension, and gastroesophageal reflux disease presented after routine laboratory testing revealed hyperkalemia and acute kidney injury. He had completed 12 cycles of pembrolizumab over approximately nine months, with the most recent cycle one week before presentation. Pantoprazole 40 mg twice daily had been initiated two weeks earlier for a gastroesophageal junction stricture. He denied vomiting, diarrhea, oliguria, fever, or rash. Examination demonstrated euvolemia without cutaneous findings. Laboratory evaluation showed potassium 5.5 mmol/L, creatinine 4.3 mg/dL (baseline 2.1 mg/dL), blood urea nitrogen 93 mg/dL (baseline 50 mg/dL), and fractional excretion of sodium of 2.64%. Urinalysis demonstrated mild proteinuria without pyuria or hematuria. Computed tomography of the abdomen showed no urinary obstruction. Pembrolizumab and pantoprazole were discontinued, and kidney biopsy demonstrated active interstitial nephritis with plasma cells, mononuclear cells, and scattered eosinophils. PD-L1 immunostaining showed focal tubular basement membrane expression, supporting an immune-mediated process. The patient was treated with prednisone 60 mg daily for 14 days, followed by a taper, with improvement in renal function (creatinine 2.4 mg/dL).

Discussion:

This case illustrates several important diagnostic challenges. Pre-existing CKD obscured recognition of superimposed immune-mediated nephritis, while the absence of the classic hypersensitivity triad of fever, rash, and eosinophilia further complicated diagnosis. The temporal relationship between prolonged pembrolizumab therapy and recent pantoprazole initiation supports the proposed "second-hit" mechanism in which checkpoint inhibition amplifies loss of immune tolerance to nephritogenic medications. Kidney biopsy was essential in distinguishing AIN from CKD progression and other causes of acute kidney injury. Although not routinely performed, PD-L1 immunostaining provided additional mechanistic support for immune-mediated renal injury.

Conclusion:

Pembrolizumab-associated AIN should remain a key diagnostic consideration in any patient receiving ICIs who develops otherwise unexplained acute kidney injury, even months after treatment initiation and in the absence of classic hypersensitivity features. Careful medication reconciliation, early kidney biopsy when indicated, and prompt corticosteroid therapy are critical to maximizing renal recovery.

Resident/Fellow Clinical Vignette

When the Heart Breaks the Brain: Acute NSTEMI Progressed to STEMI Complicated by Cardioembolic Stroke

Samy Matta

Cho Thet Zin, MD

Poe Ou Thant Sin, MD

Introduction

Acute myocardial infarction (AMI) complicated by cardioembolic stroke is a rare, high-stakes clinical scenario in which guideline-recommended therapies for one condition may directly conflict with the other. We present a case illustrating the diagnostic complexity of concurrent STEMI and cardioembolic stroke and the critical role of advanced neuroimaging in determining the temporal sequence of events.

Case Presentation

A 76-year-old woman with diabetes mellitus, hypertension, and a prior ischemic stroke with residual right-sided hemiparesis was brought to the emergency department after being found unresponsive and drooling, with a last-known-well time of 7.5 hours. Non-contrast CT head showed no acute intracranial abnormality. CTA revealed diffuse severe atherosclerosis without acute large-vessel occlusion. Initial ECG demonstrated normal sinus rhythm without ischemic changes; however, high-sensitivity troponin was markedly elevated at 1,440 ng/L.

Serial troponins rose steeply (1,440 → 6,192 → 17,860 ng/L). A repeat ECG revealed new ST-segment elevations in leads V3–V6, consistent with acute anterior STEMI. A multidisciplinary heart-brain team conference was urgently convened, and after shared decision-making with the patient's surrogate, therapeutic heparin and invasive evaluation were pursued.

Brain MRI with diffusion-weighted imaging, performed prior to catheterization, revealed acute multifocal bilateral ischemic infarctions in a pattern consistent with a cardioembolic mechanism — a pivotal finding that reframed the clinical narrative from stroke-heart syndrome to a primary cardiac event with secondary cerebral embolization. Coronary angiography demonstrated severe three-vessel disease with total LAD occlusion. Given the completed infarct and concurrent acute bilateral strokes, PCI was deferred in favor of

optimal medical management. Echocardiography showed a newly reduced LVEF of 30–35% with apical akinesis, compared with a normal baseline in 2022.

A comprehensive embolic workup including prolonged telemetry (>1 week), bubble study, and transesophageal echocardiography revealed no atrial fibrillation, PFO, or left atrial appendage thrombus, supporting the newly akinetic LV apex as the most likely embolic source.

Discussion

This case exemplifies the heart-brain axis, in which acute LAD occlusion led to severe LV dysfunction with apical akinesis — a well-established substrate for LV thrombus formation and embolization. The multifocal bilateral stroke pattern on MRI was the key diagnostic element, as multiterritory involvement implies a proximal embolic source rather than single-vessel atherothrombosis. LV thrombus complicates 6–15% of anterior STEMIs in the modern era, and embolization may occur before thrombus is detectable on imaging. This case underscores the importance of brain MRI in characterizing stroke mechanism, the value of a multidisciplinary heart-brain team approach, and the necessity of a comprehensive embolic workup when new cardiac structural abnormalities emerge during hospitalization.

Resident/Fellow Clinical Vignette

Balancing Heart and Neurosurgery: A Case of Acute STEMI Three Days After spine surgery

Samy Matta

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Ayman Fathalla, MD

Background: Dual antiplatelet therapy (DAPT) is the mainstay treatment following percutaneous coronary intervention for ST-elevation myocardial infarction (STEMI). We present a case of inferolateral STEMI which developed after three days of neurosurgery and the multidisciplinary challenges following drug-eluting stent placement.

Case presentation: A 40-year-old active smoker male with a history of hypertension, asthma, seizure disorder, and lumbar spinal fusion performed 3 days prior presented after a syncopal episode. The patient reportedly had significant postoperative back pain for 2 days which was worsening and had taken prescribed narcotics before losing consciousness. In the emergency department, the patient developed chest pain. Initial electrocardiogram demonstrated ST-segment elevations in the inferolateral leads, concerning an acute inferolateral STEMI- marked by a cumulative ST-elevation of 12 mm in leads II, III, aVF, V5, and V6 with reciprocal depression in V1 and V2. Blood work shows elevated troponin at 280.3.

Given chest pain, sum of ST elevation, and elevated troponin, Cardiology recommended emergent cardiac catheterization; however, the patient initially declined due to concern of bleeding and requested neurosurgery involvement prior to decision-making. The risks and benefits of intervention were extensively reviewed with the cardiology and neurosurgery teams. High sensitivity troponin levels increased from 280.3 to 48232.9 which raised concern for myocardial damage and thus the decision was made to proceed with revascularization. Patient underwent PCI with placement of a drug-eluting stent in the left circumflex artery, followed by aspirin and ticagrelor. His troponin peaked at more than 125,000, indicating significant myocardial damage. His hospital course was complicated by right lower quadrant pain, and computed tomography of the abdomen and pelvis revealed a right psoas hematoma. After evaluation by the general surgery team and repeat imaging demonstrating a stable hematoma, DAPT was continued without interruption.

Echocardiogram 1 year after the STEMI showed an ejection fraction of 30-35% with inferior wall hypokinesia.

Discussion: This case demonstrates the challenges of managing acute STEMI in the early postoperative period, where the necessity for urgent revascularization and dual antiplatelet therapy must be weighed against increased risk of postoperative bleeding complications. Current American heart association guidelines recommend delaying elective noncardiac surgery for 12 months after drug eluting stent placement and DAPT initiation, however, there is no clear roadmap for addressing reverse scenarios where the need for revascularization occurs within a few days of major surgery. This highlights the gap in current perioperative guidelines and reinforces that individualized multidisciplinary is essential to optimizing outcomes when standard protocols cannot provide a clear path.

Resident/Fellow Clinical Vignette

Recognizing BRASH Syndrome: A Cascade of Bradycardia, Renal Failure, and Shock in COVID-19 Myocarditis

Adrija Mehta

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Introduction:

BRASH syndrome (Bradycardia, Renal failure, AV-nodal blockade, Shock, Hyperkalemia) is an underrecognized iatrogenic cascade arising when AV-nodal blocking agents accumulate in renal impairment. It is frequently misattributed to primary cardiac or septic shock, delaying recognition of a reversible process.

Case Description:

A 55-year-old woman with asthma, hypertension, type 2 diabetes, and CKD stage 3 presented with two weeks of dyspnea, palpitations, and lower extremity edema, and was found to have atrial flutter with rapid ventricular response and SARS-CoV-2 infection. Initial labs showed acute kidney injury with creatinine 2.8 mg/dL, troponin 134 ng/mL, BNP ~30,982 pg/mL, and metabolic acidosis. Echocardiography revealed an ejection fraction of 22% and a left ventricular apical thrombus. Diltiazem and metoprolol were uptitrated for rate control, with amiodarone later added. Over the following days, her renal function continued to decline; by hospital day 5, creatinine had risen to 4.5 mg/dL with new potassium 6.5 mmol/L. That day she developed hypoglycemia (glucose 50 mg/dL) refractory to dextrose, hypotension (75/62 mmHg), and bradycardia (heart rate 30-40 bpm) unresponsive to atropine with EKG showing AV block — triggering recognition of BRASH. All chronotropic agents were stopped, vasopressors were started, and emergent hemodialysis was initiated, which led to improvement in electrolytes, acidosis, and kidney function. On hospital day 6, she suffered a pulseless electrical activity arrest and was subsequently found to have an acute ischemic stroke. She stabilized thereafter, and metoprolol was cautiously reintroduced at a low dose once renal function had partially recovered, and hemodialysis sessions were continued. She was discharged on rate control medications with close nephrology follow-up.

Discussion:

This case illustrates BRASH's pathognomonic triad — atropine-refractory bradycardia, fluid-refractory shock, and dextrose-refractory hypoglycemia — emerging despite compelling competing diagnoses of COVID-19 myocarditis and sepsis. Metoprolol's active

metabolites accumulate in renal impairment despite predominant hepatic clearance, prolonging its effective half-life several-fold; in this patient, baseline CKD combined with a creatinine that nearly doubled from admission to BRASH onset, and continued rising to a peak of 7.1 mg/dL even after hemodialysis began, meant that standard rate-control dosing, appropriate at admission, became a compounding drug burden as renal clearance narrowed daily. Recovery of glycemic control after drug cessation and dialysis, preceding any hemodynamic improvement, confirmed a pharmacologic rather than purely hemodynamic mechanism. Clinicians should maintain a low threshold to suspect BRASH in any renally impaired patient receiving AV-nodal blockers who develops this reversible triad, track creatinine trajectory as closely as heart rate when uptitrating these agents, favor hepatically-cleared alternatives or renally-adjusted dosing once acute kidney injury is evident, and integrate real-time renal function into every rate-control decision to prevent this potentially fatal but reversible iatrogenic complication.

Resident/Fellow Clinical Vignette

Immune Checkpoint Inhibitor–Associated Scleroderma-like syndrome: A Case Report of PD-1 Induced Fibrosing Autoimmunity.

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Background: Immune checkpoint inhibitors (ICIs), particularly PD-1 inhibitors such as pembrolizumab, have significantly improved outcomes in multiple malignancies but can trigger a wide range of immune-related adverse events (irAEs). Among these, ICI-induced scleroderma and scleroderma-like syndromes are rare, often seronegative, and may present with atypical patterns of skin thickening and edema, making diagnosis challenging.

Case Presentation: A 78-year-old woman with locally advanced squamous cell carcinoma of the right lower lobe received neoadjuvant gemcitabine, cisplatin, and pembrolizumab, followed by right lower lobectomy demonstrating minimal residual stage IA1 disease. She subsequently initiated adjuvant pembrolizumab. Early during therapy, she developed immune-mediated dermatitis that resolved with a short prednisone taper, and pembrolizumab was resumed. She was also treated for pembrolizumab-associated central adrenal insufficiency with hydrocortisone.

Several months into adjuvant therapy, she developed new bilateral lower-extremity pain and progressive edema, initially pitting and partially responsive to diuretics. Over time, she noted persistent, non-pitting skin thickening involving the lower extremities, anterior abdominal wall, chest, and posterior trunk. Symptoms worsened with prolonged inactivity and improved with ambulation. Despite improvement in edema, the skin changes progressed. She denied Raynaud's phenomenon, sicca symptoms, digital ulcers, or inflammatory myopathy. Creatine kinase, aldolase, and an extensive autoimmune panel including ANA, anti-Scl-70, anti-centromere, anti-RNP, anti-SSA/SSB, and dsDNA were negative. Complement levels were normal to mildly elevated. Imaging revealed edema, ascites, and a mild pericardial effusion without renal, hepatic, or cardiac pathology.

Given the diffuse, progressive skin thickening, seronegative profile, and temporal association with pembrolizumab, rheumatology evaluation strongly favored ICI-induced scleroderma. A Dermatologic biopsy was planned to confirm the diagnosis. High-dose

corticosteroids were initiated with partial improvement, and pembrolizumab was discontinued.

Outcome: Before biopsy could be obtained, the patient was hospitalized with acute abdominal pain and was found to have pneumoperitoneum due to intestinal perforation. Despite intervention, she died during the hospitalization.

Conclusion: This case illustrates a rare, severe presentation of pembrolizumab-induced scleroderma characterized by progressive, widespread skin thickening and negative serologies. The absence of classic systemic sclerosis markers and the overlap with early edema-dominant presentations can delay recognition. Early multidisciplinary evaluation and prompt immunosuppression are essential, as ICI-induced scleroderma may progress rapidly and carry significant morbidity and mortality.

Resident/Fellow Clinical Vignette

RAPID DECLINE FOLLOWING RASBURICASE INDUCED METHEMOGLOBINEMIA AND HEMOLYSIS

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Rasburicase, a recombinant urate oxidase used for tumor lysis syndrome (TLS), carries an FDA box warning for hemolysis and methemoglobinemia and is contraindicated in patients with Glucose-6-phosphate-dehydrogenase (G6PD) deficiency. In a systematic review of 43 patients with these side-effects, 90% of patients recovered and median hemoglobin nadir was 6.1g/dL. Traditional therapies such as methylene blue are contraindicated in G6PD deficiency. We present the case of a patient with unscreened G6PD deficiency and refusal of blood products secondary to religious beliefs, in which rasburicase treatment resulted in methemoglobinemia within 26 hours.

A 76-year-old Hispanic Jehova's witness with medical history of coronary artery disease and chronic kidney disease presented to the emergency department due to worsening renal function accompanied by back pain, fatigue, and five-pound weight loss over one month. Admission laboratory results were relevant for creatinine of 4.2mg/dl (baseline 2.5mg/dL), anemia (hemoglobin 8.8g/dL), and thrombocytopenia (platelets 89/µl). CT abdomen revealed a large mass in the left retroperitoneum encasing the ureter and renal pelvis, multiple enlarged para-aortic lymph nodes and innumerable hepatic lesions, raising concern for solid malignancy with metastasis. The patient declined blood products due to religious beliefs. Initial flow cytometry did not detect any abnormal lymphocyte populations. Lab results suggested TLS with uric acid level of 14.3mg/dL. Rasburicase was administered while awaiting G6PD deficiency testing. Eight hours after rasburicase administration, pulse oximetry showed a fixed oxygen saturation of 77%. The patient was asymptomatic and saturation did not improve with escalation of supplemental oxygen. Arterial blood gas revealed PaO2 of 203mmHg, lactate of 1.9mmol/L, and methemoglobin of 14.1%. Toxicology was consulted, care escalated to the critical care unit and anti-oxidative treatment with ascorbic acid initiated. Fourteen hours after rasburicase initiation methemoglobin remained elevated at 11.8%, hemoglobin dropped to 6.8g/dL and

potassium increased to 6.2mmol/L, consistent with hemolysis. The patient continued to decline transfusion. Methylene blue was deferred given concern for exacerbating hemolysis in the setting of suspected G6PD deficiency. Anemia progressively worsened and elevated lactate suggested end-organ damage. He developed profound acidosis, hyperkalemia, and declining mental status. Despite intubation and initiation of hemodialysis, he died 26 hours after rasburicase administration. Post-mortem laboratory results revealed G6PD deficiency.

This case illustrates a rare but fatal outcome of rasburicase-induced methemoglobinemia and hemolytic anemia in a patient with undiagnosed G6PD deficiency. It underlines the need for swift recognition and treatment of this condition. It is also notable given the patient developed TLS from an undiagnosed malignancy without initial suspicion for a hematologic malignancy, therefore highlighting the importance of universal screening for G6PD deficiency before rasburicase use, regardless of malignancy type. Finally, it supports considering patients' beliefs during medical decision making, as usually treatable conditions can turn fatal when therapeutic options are limited by patient preference.

Resident/Fellow Clinical Vignette

Vasospastic angina mimicking inferior wall STEMI

Claudiane Mouafo

Adusumalli, Snigda

Cerrud-Rodrguez, Roberto

Coronary artery vasospasm is a reversible cause of myocardial ischemia that can closely mimic acute coronary syndromes, including ST-elevation myocardial infarction (STEMI). Prompt recognition is essential to avoid unnecessary revascularization and to guide appropriate therapy. Vasospastic angina (VSA), also known as Prinzmetal or variant angina, is characterized by transient coronary artery constriction resulting in myocardial ischemia, often without fixed obstructive coronary disease. Nonatherosclerotic mechanisms, including vasospasm, account for approximately 5–25% of myocardial infarction presentations.

We report the case of a 57-year-old woman with multiple cardiovascular risk factors, including active tobacco use, type 2 diabetes mellitus, obesity, and chronic obstructive pulmonary disease, who presented with several days of intermittent chest pain radiating to the left jaw. Initial evaluation revealed mildly elevated troponin levels, and she was treated for non–ST-elevation myocardial infarction (NSTEMI) with antithrombotic therapy and statins. Shortly after transfer, she developed acute nausea, malaise, and significant bradycardia, followed by new ST-segment elevations on electrocardiogram consistent with inferior STEMI.

Emergent coronary angiography demonstrated severe diffuse vasospasm involving the left anterior descending and left circumflex arteries in a left-dominant coronary system, without evidence of obstructive coronary disease. Administration of intracoronary nitroglycerin resulted in rapid and marked vasodilation, confirming the diagnosis of coronary vasospasm. Anticoagulation was discontinued, and the patient was treated with calcium channel blockers and nitrates, with counseling on smoking cessation.

Vasospastic angina is associated with several risk factors, including female sex, smoking, endothelial dysfunction, and heightened autonomic reactivity. The condition is characterized by transient ST-segment changes that can mimic acute myocardial infarction, making diagnosis challenging. Coronary angiography, particularly with the use of intracoronary vasodilators or provocative testing, plays a central role in distinguishing vasospasm from atherosclerotic plaque rupture.

Management focuses on vasodilator therapy and risk factor modification. Calcium channel blockers are first-line agents, with nitrates used as adjunctive therapy. Beta-blockers are generally avoided, as they may exacerbate vasospasm. Recognition of this condition is critical, as appropriate treatment can prevent recurrent episodes and reduce the risk of adverse cardiovascular outcomes.

This case highlights the importance of considering coronary vasospasm in patients presenting with acute coronary syndrome features, particularly in the absence of obstructive coronary disease. Early identification and targeted therapy are essential to optimize outcomes and avoid unnecessary invasive interventions.

Resident/Fellow Clinical Vignette

A "masked " snake on the grass: a case of incidental masquerading bundle branch block

Claudiane Mouafo

#NAME?

Masquerading bundle branch block (MBBB) is a rare electrocardiographic finding characterized by a left bundle branch block pattern in the limb leads and a right bundle branch block pattern in the precordial leads. It is an often missed marker of severe high-grade conduction disease with an estimated prevalence of 0.004% and is associated with 41.4% four-year mortality rate and a 38.9% likelihood of requiring permanent pacemaker implantation.

A 74-year-old man with a history of coronary artery disease status post coronary artery bypass grafting (CABG) was referred to the cardiology clinic for evaluation of progressive exertional dyspnea that began six months earlier. Review of serial electrocardiograms obtained over the preceding year revealed a pattern initially interpreted as right bundle branch block with left anterior fascicular block. Transthoracic echocardiography demonstrated normal left ventricular systolic function. Ambulatory cardiac event monitoring identified sinus bradycardia with heart rates in the 50s. A nuclear stress test showed inferior ischemia, and the electrocardiographic component revealed a masquerading bundle branch block (MBBB) pattern. Subsequent coronary angiography demonstrated severe multi-vessel coronary artery disease with patent bypass grafts. Given the conduction abnormalities and symptomatic bradycardia, the patient was referred to electrophysiology for pacemaker implantation evaluation.

Masquerading bundle branch block (MBBB) is a rare and advanced electrocardiographic finding characterized by a right bundle branch block (RBBB) pattern in the precordial leads and a left bundle branch block (LBBB)-like pattern in the limb leads. Once described as LBBB mimicking RBBB, it is now considered a variant of RBBB with altered QRS vectors, often due to left anterior fascicular block (LAFB). Two types are described: the standard type, showing RBBB in precordial leads and LBBB-like limb-lead pattern due to left anterior forces; and the precordial type, with LBBB in V4–V6 and RBBB in V1–V3, often associated with ischemic heart disease, cardiomyopathy, or conduction system disorders. ECG features of standard MBBB include prolonged QRS duration, absent or small S waves in lead I, loss of terminal R' in frontal leads, and axis deviation around -60°. These patterns reflect high-grade conduction disease, often linked to adverse outcomes,

particularly in patients with structural heart disease. Misinterpretation is common, as in our patient, whose earlier ECG was labeled as simple RBBB with LAFB. Unrecognized MBBB may predispose patients to complete heart block or fatal arrhythmias. Awareness of this rare conduction abnormality is crucial for timely evaluation and management, including consideration for pacemaker implantation in high-risk individuals.

Resident/Fellow Clinical Vignette

Beyond Joint Pain: Hydralazine-Induced Lupus Presenting with Life-Threatening Cardiac Tamponade

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Introduction

Hydralazine-induced lupus erythematosus is a well-recognized autoimmune complication of prolonged hydralazine therapy. While musculoskeletal manifestations and positive antinuclear antibodies are common, clinically significant cardiac involvement is rare. Pericardial effusion progressing to cardiac tamponade is an uncommon but potentially life-threatening presentation. Because its clinical manifestations may mimic idiopathic systemic lupus erythematosus (SLE), infection, or malignancy, early recognition is essential for timely diagnosis and management.

Case Presentation

A 66-year-old Hispanic man with hypertension, stage 3 chronic kidney disease, hyperlipidemia, obesity, obstructive sleep apnea, pulmonary hypertension, and long-term hydralazine use was referred to rheumatology for progressive polyarthralgia involving the bilateral hands, wrists, shoulders, and knees. Laboratory evaluation demonstrated elevated inflammatory markers (ESR 42 mm/hr, CRP 13.6 mg/L), positive rheumatoid factor (24.8 IU/mL), high-titer ANA (1:1280, homogeneous pattern), elevated anti-double-stranded DNA antibodies, low complement C4 levels, and strongly positive antihistone antibodies. Anti-CCP, SSA, SSB, Scl-70, MPO, and PR3 antibodies were negative. Mild proteinuria was present without evidence of active lupus nephritis.

Hydralazine-induced lupus was suspected, and hydralazine was discontinued. Several weeks later, the patient presented with worsening chest pain and dyspnea. CT angiography, initially performed to exclude pulmonary embolism, demonstrated an enlarging pericardial effusion. Transthoracic echocardiography confirmed a large pericardial effusion with hemodynamic compromise consistent with cardiac tamponade. The patient underwent emergent pericardiocentesis with pericardial drain placement and was admitted to the intensive care unit.

Pericardial fluid cytology demonstrated chronic inflammatory cells without evidence of malignancy, and an infectious evaluation was negative. The overall clinical and serologic findings were consistent with hydralazine-induced lupus complicated by serositis leading to massive pericardial effusion and cardiac tamponade. Corticosteroid therapy was initiated, hydroxychloroquine was added, and hydralazine was permanently discontinued. Follow-up echocardiography demonstrated near-complete resolution of the effusion, with significant improvement in both inflammatory markers and arthralgias.

Discussion

Drug-induced lupus accounts for approximately 6%–12% of lupus cases, with hydralazine among the medications carrying the highest risk. Although antihistone antibodies are characteristic, clinically significant pericardial involvement is uncommon, and cardiac tamponade remains exceedingly rare. Hydralazine-induced lupus may also demonstrate overlapping serologic features, including positive anti-double-stranded DNA antibodies and hypocomplementemia, making differentiation from idiopathic SLE challenging. This case illustrates that severe serositis may develop despite discontinuation of hydralazine and should remain in the differential diagnosis of unexplained pericardial effusion in patients with compatible medication exposure and autoimmune serologies.

Conclusion

Hydralazine-induced lupus can closely mimic idiopathic systemic lupus erythematosus and, although uncommon, may present with life-threatening cardiac tamponade. Recognition of the characteristic serologic profile, medication exposure history, and exclusion of alternative etiologies are essential for timely diagnosis. Clinicians should maintain a high index of suspicion for hydralazine-induced lupus in patients receiving long-term hydralazine who present with unexplained serositis, as early diagnosis can be lifesaving.

Resident/Fellow Clinical Vignette

Unraveling a Rare Conundrum: Secondary Hemophagocytic Lymphohistiocytosis Induced by Severe Plasmodium falciparum Malaria in a Returning Traveler from West Africa

Henry Nabeta

Henry Nabeta, FNU Veena, Abel Akanyijuka, Neguemadji Ngardig Ngaba, Rayan Alaata, Asare Michael, Doda Manjola, Peachy Mathew, Rubin Philip, Khaja Misbahuddin

Introduction

Hemophagocytic lymphohistiocytosis (HLH) is characterized by a dysregulated immune activation and a cytokine storm, leading to macrophage activation, hemophagocytosis, and multiorgan dysfunction. Here, we present a rare case of secondary hemophagocytic lymphohistiocytosis (HLH) triggered by Plasmodium falciparum malaria in a 48-year-old male returning from Ivory Coast, West Africa. Despite parasitemia clearance, he had persistent fever, new onset hepatomegaly, and worsening acute kidney injury.

Case Presentation

A 48-year-old man with no significant medical history presented with a 6-day history of headache, rigors, diaphoresis, severe arthralgia, and vomiting, shortly after returning from a 2-month stay in Ivory Coast, where he reported numerous mosquito bites. On admission, he was febrile (99.9°F), tachycardic, and had QTc prolongation on ECG. Laboratory studies revealed thrombocytopenia (26 × 10^9/L), elevated creatine phosphokinase (CPK 5466 U/L), lactic acidosis (3.8 mmol/L), hypertriglyceridemia (300 mg/dL), hyperbilirubinemia (total bilirubin 4.1 mg/dL), transaminitis (AST 107 U/L, ALT 60 U/L), and a P. falciparum parasitemia of 9.7%. He was admitted to the intensive care unit for respiratory distress, tachycardia, and hypotension. Intravenous artesunate was initiated, and parasitemia cleared by hospital day 4.

Despite parasite clearance, the patient developed worsening renal function (peak creatinine 4.8 mg/dL), and persistent low-grade fever. Abdominal ultrasonography revealed hepatomegaly. Chest radiography showed mild pulmonary congestion. Head CT was unremarkable.

The persistent fever, hepatomegaly, cytopenias, hyperferritinemia, hypertriglyceridemia, and elevated sCD25 met five of the eight HLH-2004 diagnostic criteria. The H score was also highly suggestive of HLH. Bone marrow biopsy was deferred due to clinical stability and fulfillment of diagnostic criteria. Supportive care included intravenous fluids for acute

kidney injury, attributed to rhabdomyolysis and dehydration. Corticosteroids were withheld due to clinical improvement. Renal function improved from KDIGO stage 3 to 2, with creatinine decreasing by 0.9 mg/dL (19%) to 3.9 mg/dL. The patient was discharged on prophylactic anticoagulation.

Discussion

Malaria-associated HLH is a rare but potentially life-threatening complication. P. falciparum is the most common trigger, likely due to its capacity to induce a profound cytokine response. Persistence of HLH features after parasite clearance, suggests a post-infectious immune-mediated process. This case underscores the importance of considering HLH in malaria patients with atypical or persistent symptoms, even after parasite clearance.

Resident/Fellow Clinical Vignette

Acrodermatitis Continua of Hallopeau Progressing to Generalized Pustular Psoriasis: Recognizing a Rare Dermatologic Emergency

Henry Nabeta

FNU Veena, Henry Nabeta, Kyle Smith, Daviel C Rodriguez, Donald Rudikoff, Iwuaba Chinazor

Introduction:

Acrodermatitis continua of Hallopeau (ACH) is a rare, localized variant of pustular psoriasis characterized by chronic sterile pustules of the distal digits and nail apparatus. Delayed diagnosis may lead to permanent nail destruction, osteolysis, and progression to generalized pustular psoriasis (GPP), a potentially life-threatening inflammatory disorder. Because its early manifestations often resemble infection or drug-induced eruptions, ACH remains underrecognized. We report a case in which longstanding untreated periungual pustulosis culminated in an acute generalized pustular eruption, emphasizing the importance of early recognition and multidisciplinary management.

Case Presentation:

A 61-year-old man with no significant medical history presented with a four-day history of a rapidly progressive pustular eruption involving the trunk and extremities after failing an unspecified five-day outpatient treatment. He described a one-year history of a persistent pustular lesion affecting a fingertip with intermittent periungual scaling but denied preceding medication exposure, fever, mucosal lesions, recent infections, or other systemic symptoms.

Examination revealed diffuse erythematous blanching plaques studded with sterile pustules across the trunk and proximal extremities. Scaling pustular lesions involved the distal hands and periungual regions, accompanied by palmar hyperpigmented macules and marked bilateral auricular xerosis. Nikolsky sign was negative, with no epidermal

detachment or mucosal involvement.

Laboratory studies demonstrated leukocytosis (14.2 ×10^9/L) with neutrophilia, mild renal impairment (creatinine 1.4 mg/dL), severe vitamin D deficiency, elevated ferritin, and low serum iron. Respiratory viral testing and chest radiography were unremarkable. Urine toxicology was positive for cocaine. Dermatology recognized the chronic acral pustulosis preceding the diffuse eruption as ACH with progression toward GPP. Acute generalized exanthematous pustulosis, Stevens-Johnson syndrome, toxic epidermal necrolysis, and infectious pustular dermatoses were excluded based on clinical presentation and the absence of mucosal involvement, epidermal necrosis, or evidence of infection.

The patient was treated with acitretin 25 mg daily, cyclosporine 125 mg twice daily, and a 300-mg loading dose of secukinumab. Infectious Diseases advised against empiric antibiotics because there was no clinical or microbiologic evidence of infection. Close monitoring for renal dysfunction, hypertension, infection, and cardiovascular complications was performed, and latent tuberculosis screening was obtained before continuation of biologic therapy.

Discussion:

This case highlights an uncommon but clinically significant progression of chronic localized ACH to GPP. The year-long history of periungual pustulosis provided a crucial diagnostic clue that distinguished this presentation from drug-induced or infectious pustular eruptions and enabled prompt initiation of targeted immunomodulatory therapy. Early recognition of persistent acral pustulosis as a manifestation of ACH is essential, as delayed diagnosis may permit progression to generalized disease with substantial morbidity. This case demonstrates how multidisciplinary collaboration can expedite diagnosis, avoid unnecessary antimicrobial therapy, and facilitate timely biologic treatment for this potentially life-threatening psoriasis spectrum disorder.

Resident/Fellow Clinical Vignette

D-penicillamine-induced gigantomastia in Wilson's disease: Why partial dose reduction failed.

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Introduction: D-penicillamine-induced mammary hyperplasia is an exceptionally rare adverse effect, reported predominantly in rheumatologic disorders and only rarely in Wilson's disease. We report a case notable for its severity and for demonstrating why dose reduction alone is insufficient once tissue remodeling has begun.

Case Presentation: A 16-year-old girl with Wilson's disease, diagnosed at four years of age during evaluation for chronic hepatitis, remained clinically stable on D-penicillamine (250 mg three times daily) and zinc (50 mg three times daily) for 10 years, without neurological manifestations or Kayser-Fleischer rings.

At age 14, she developed progressive bilateral breast enlargement, greater on the left, well past normal pubertal timing for her Tanner stage. Pregnancy, thyroid dysfunction, and other endocrinopathies (e.g., McCune-Albright syndrome) were excluded clinically. Ultrasonography demonstrated multiple bilateral fibroadenomas, the largest measuring 7.4×3.3 cm. Serum prolactin, FSH, LH, and estradiol were normal. The dose was reduced from 750 mg/day to 50 mg once daily, but breast enlargement progressed over the next two years to massive bilateral gigantomastia extending to the upper thighs, causing severe postural discomfort, impaired mobility, and significant psychosocial distress. Examination revealed enormous pendulous breasts with multiple firm mobile masses bilaterally and mild hepatomegaly without decompensation.

MR mammography showed diffuse glandular hyperplasia with multiple enhancing fibroadenomas; fine-needle aspiration confirmed fibroadenoma. D-penicillamine was discontinued and the patient transitioned to trientine; zinc was continued. She underwent bilateral reduction mammoplasty with excision of four giant fibroadenomas (three left, one right), the largest measuring 11.5×9.5×3 cm. Histopathology showed juvenile

fibroadenomas with intracanalicular stromal hyperplasia. At 6-month follow-up, she had marked improvement in mobility, cosmesis, and psychological well-being, with no recurrence.

Discussion: The proposed mechanism is increased breast tissue sensitivity to circulating steroid hormones rather than endocrine dysfunction, consistent with this patient's normal hormonal profile. Partial dose reduction was insufficient to halt drug-induced tissue remodeling once activated, and normal endocrine investigations did not exclude drug-induced mammary hyperplasia — delayed recognition allowed progression to irreversible deformity requiring major reconstructive surgery.

Progressive breast enlargement in patients receiving D-penicillamine should prompt early consideration of this rare adverse effect, even with normal endocrine evaluation. Prompt, complete discontinuation — rather than tapering — with transition to an alternative chelator such as trientine may prevent irreversible deformity and avoid extensive reconstructive surgery.

Teaching point: gigantomastia can emerge even after a decade of drug tolerance, and the threshold for complete cessation, not tapering, should be low once it begins.

Resident/Fellow Clinical Vignette

When Ulcerative Colitis Becomes Crohn's Disease: Refractory Hemorrhage Unmasking Reclassification

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Introduction: IBD classification can evolve over time, with UC diagnoses revised to Crohn's disease (CD) upon disease progression or a refractory course. We present a 17-year-old male with UC diagnosed in 2021 who developed steroid-refractory, transfusion-dependent hemorrhage, ultimately reclassified as CD based on endoscopic and histologic findings.

Case Description: A 17-year-old male with beta thalassemia minor and a family history of UC was diagnosed with pancolonic UC in 2021 by colonoscopy and biopsy, with no ileal involvement or CD-type ulceration. He improved on mesalazine and steroids but self-discontinued therapy. He re-presented in 2024 with six weeks of hematochezia and abdominal pain. Workup showed hemoglobin 6.5 g/dL, fecal calprotectin 2,250, and leukocytosis 22,600. Colonoscopy showed acute colitis without chronicity; CT abdomen showed long-segment circumferential thickening of the distal ileum, cecum, ascending, and transverse colon with fat stranding. He received two PRBC transfusions and was started on prednisolone and mesalazine, with no response at one month. Repeat colonoscopy revealed large colonic ulcers with skip areas and deep punched-out terminal ileal ulcers; biopsy favored CD. He remained hemodynamically borderline despite transfusion, with persistent bright red blood per rectum and rising transfusion requirement despite maximal medical therapy, prompting escalation to biologic therapy. CT enteroclysis with angiography showed mural thickening without an arterial bleeding source. GeneXpert and latent TB workup were negative. Diagnosis was reclassified as CD. He then required eleven PRBC transfusions over eight days for ongoing hemorrhage. Infliximab did not control the bleeding, and he underwent limited terminal ileal/ileocecal resection with intraoperative enteroscopy, which controlled the hemorrhage; resection specimen histopathology confirmed CD, and his hemoglobin subsequently stabilized off further transfusion requirement.

Discussion: The 2021 findings were consistent with UC at the time; the 2024 skip lesions and punched-out terminal ileal ulcers represent a true phenotypic evolution to CD rather than an initial misdiagnosis. Non-adherence likely accelerated progression but does not fully explain it, as CD can declare itself years after a UC-pattern presentation regardless of adherence. The key teaching point: once a "stable" IBD relapse fails to respond to steroids, repeat endoscopy with deliberate terminal ileal intubation should be performed early to reassess the diagnosis itself, not just escalate therapy within the presumed disease. This case also underscores that a family history of UC does not predict phenotype on relapse, and that non-response to steroids plus mesalazine at one month is itself an indication for reassessment, not simply for adding a biologic. Reclassification should trigger immediate reassessment of therapeutic strategy alongside early, parallel surgical consultation rather than sequential escalation, given how quickly IBD hemorrhage can outpace medical therapy.

Resident/Fellow Clinical Vignette

THE UNRESOLVING BRUISE: A CASE REPORT ON DELAYED DIAGNOSIS OF RADIATION-INDUCED ANGIOSARCOMA

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Introduction

Radiation-induced angiosarcoma (RIAS) is a rare and aggressive cancer occurring in women with a history of radiotherapy that is often associated with delayed diagnosis. We present this case report to discuss the importance of early diagnosis and high index of suspicion.

Case

We present a 66-year-old postmenopausal woman with past medical history significant for stage IA left breast infiltrating ductal carcinoma, treated with lumpectomy, sentinel lymph node biopsy, and radiotherapy without chemotherapy. She then completed 5 years of hormonal therapy with exemestane.

During routine follow-up 9 years after radiation therapy, she was noted to have a new rash within the prior radiation field. According to the patient it had been present for approximately one year, initially attributed to trauma from her dog jumping on her but the “bruise” never resolved. On examination she did not have any distinct breast mass, discharge, nipple eversion or lymphadenopathy. She had a 4x5 cm non-tender purple discoloration of the skin of the left breast at the 9 o'clock position which was non-blanchable without fluctuance. Breast ultrasound followed by biopsy confirmed RIAS. Subsequent staging workup demonstrated no evidence of adenopathy, or metastatic disease. She underwent mastectomy and attempted sentinel lymph node biopsy which was aborted due to failure of radioactive tracer migration to the axilla.

Discussion

RIAS is associated with delayed diagnosis; in a retrospective series the mean interval between onset of cutaneous symptoms and diagnosis was 18 months. Understanding and improving factors that contribute to delay is important as overall survival (OS) is impacted by the stage and the possibility of surgery with intention of cure. A study demonstrated a 2-

year overall survival (OS) of 33.3% in unresectable vs 71.1% in resectable patients, and median OS of 18 months vs 37 months, respectively.

Delay in diagnosis is driven by its rarity, unusual presentation, lack of awareness, and lower level of clinical suspicion. RIAS occurs in about 1-in-18,000 women treated with breast radiotherapy but with increasing incidence felt to be secondary to increasing use of radiotherapy in breast cancer treatment. Its rarity poses a challenge in recognizing it as a possibility when suggestive symptoms develop, and is often ascribed to benign dermatological conditions, leading to delay like in our case. It is therefore important to educate patients, practitioners, and other healthcare providers on high index of suspicion of RIAS.

RIAS develops in older women about 7-to-10 years after radiotherapy, arising within the dermis and sub-cutaneous layer of the skin of the breast without necessarily involving the breast parenchyma. RIAS can appear like a bruise, rash, or skin thickening, and should be strongly considered in persons who have had radiotherapy within that region.

Resident/Fellow Clinical Vignette

YAMAGUCHI SYNDROME IN AN AFRICAN AMERICAN PATIENT PRESENTING WITH CHEST PAIN AND CONCURRENT AORTIC INTRAMURAL HEMATOMA

Rosanna Pineda

Purpose:

To highlight Yamaguchi syndrome as an underrecognized cause of chest pain in non-Asian patients and its potential coexistence with life-threatening aortic pathology requiring urgent intervention.

Methods:

We report a clinical case with diagnostic evaluation, multimodality imaging, management, and outcome.

Results:

An 84-year-old African American woman presented with acute chest pain. Electrocardiogram demonstrated a wide-complex tachycardia of presumed ventricular origin without ischemic ST-T changes. Chest radiograph revealed a widened mediastinum, raising concern for acute aortic pathology. The patient underwent emergent cardiac catheterization, which showed non-obstructive coronary arteries and a dilated aorta. Left ventriculography demonstrated marked apical hypertrophy with a characteristic “spade-like” configuration, consistent with apical hypertrophic cardiomyopathy (Yamaguchi syndrome). Transthoracic echocardiography confirmed apical hypertrophy and identified a crescentic echodense structure along the aortic wall concerning for intramural hematoma. Computed tomography angiography revealed an extensive ascending aortic intramural hematoma extending from the aortic root to the arch and into the abdominal aorta, with associated hemopericardium and moderate pericardial effusion. Given the high risk of progression to type A aortic dissection, the patient underwent emergent surgical repair with ascending aorta and hemiarch replacement. The patient tolerated the procedure and was stabilized postoperatively.

Conclusion:

Yamaguchi syndrome should be considered in patients with chest pain and non-obstructive coronary arteries, regardless of ethnicity. Clinicians should maintain a high

index of suspicion for concomitant aortic pathology, as multimodality imaging can identify this rare but critical combination and guide life-saving intervention.

Resident/Fellow Clinical Vignette

Cholestatic Liver Injury: Disentangling Drug-Induced Hepatotoxicity from Malignant Biliary Obstruction

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Srikar Uppaluri

Suresh Kumar Nayudu

Introduction:

Cholestatic liver injury (CLI) includes a broad spectrum of etiologies like drug-induced liver injury (DILI), choledocholithiasis(CDL), autoimmune hepatic disease(AHD) and pancreatico-hepatobiliary (PHB) malignancy. Among these, DILI remains a diagnosis of exclusion, requiring systematic evaluation to rule out competing etiologies especially CDL and malignancies. We present a diagnostically challenging case presenting with cholestatic liver injury initially diagnosed with DILI and subsequently diagnosed with pancreatic adenocarcinoma.

Case Report:

A 64-year-old woman with prior history of breast cancer, recurrent head and neck cancer presented with asymptomatic jaundice and markedly elevated liver enzymes in a cholestatic pattern with a R factor of 0.6. Extensive workup including viral hepatitis, AHD, and metabolic studies was unrevealing. Initial imaging with Ultrasound (US) and MRI/MRCP showed biliary ductal dilation but was negative for any pancreatic mass, obstructing stone, or filling defect. Liver biopsy was pathognomonic of DILI and offending agents were discontinued and treated with prednisone, ursodiol, and N-acetylcysteine as per GI with improvement in labs.

Three weeks later, she was readmitted with new onset vomiting and rising liver enzymes. Repeat imaging with US and CT revealed a 3.7 × 2.5 cm obstructing lesion at the pancreatic head—a finding not present on prior imaging. EUS-guided FNA confirmed pancreatic adenocarcinoma and underwent choledochoduodenostomy with stent placement with improvement.

Discussion

CLI is multifactorial as in this case with initial DILI diagnosis followed by subsequent pancreatic mass after 3 weeks supported by ACG guidelines which emphasizes that competing etiologies for cholestatic DILI are predominantly PHB in nature. However, as per literature negative imaging does not definitively exclude pancreatic tumors as the size can be below the initial detection threshold. Published data on missed pancreatic cancers demonstrate that approximately 42% of missed tumors were less than 2 cm in size and 44% were isoattenuation on imaging.

Conclusion:

This case highlights that clinicians should maintain a high suspicion of possible malignancy when clinical trajectories deviate from expected recovery or presence of new symptoms following initiation of appropriate therapy and should think of repeat cross-sectional imaging regardless of prior negative studies.

Resident/Fellow Clinical Vignette

A Mimic of Infarction: Stress Cardiomyopathy and Malignant Arrhythmia in Metastatic Paraganglioma

Fatima Qadri

Ryan Punsalan, MD, Aalia Khamis, MD

Abstract

This case reports a 77-year-old woman with metastatic paraganglioma presenting as a "pseudo-NSTEMI" with stress cardiomyopathy. Marked catecholamine excess (>17 x ULN) and PALB2/ATM mutations necessitated a multidisciplinary approach. It underscores the "alpha-before-beta" blockade rule to prevent hypertensive crisis and the role of precision oncology in rare neuroendocrine tumors.

Background

Paragangliomas are rare extra-adrenal neuroendocrine tumors with an annual incidence of 2–8 per million. While hypertension is common, malignant arrhythmias and stress cardiomyopathy are underrecognized complications. Excess catecholamine secretion causes cardiovascular toxicity through β-adrenergic myocardial stunning and α-mediated vasoconstriction.

Case Presentation

We report a 77-year-old woman with recurrent syncope initially attributed to vasovagal episodes after normal echocardiography and stress testing. She later presented with flank pain, troponin elevation 1.44→2.12→0.72 ng/mL), and biphasic T-wave inversions, suggesting non–ST elevation myocardial infarction. Coronary angiography showed nonobstructive disease, but imaging revealed a retroperitoneal mass, adrenal nodule, and hepatic lesions. Plasma metanephrines were markedly elevated (metanephrines 1,248 pg/mL (>17 x ULN]; total 4,194 pg/mL). FoundationOne® liquid biopsy identified PALB2, ATM, FGFR, ATRX, and DNMT alterations. DOTATATE PET confirmed extensive metastases, precluding surgery.

Following multidisciplinary review, she enrolled in a Phase II trial of olaparib and temozolomide (NCT04394858). She completed one cycle but was withdrawn due to recurrent hospitalizations. Months later, she presented with labile hypertension and atrial fibrillation. Echocardiography showed severe mid-to-apical hypokinesis (LVEF 30–35%), consistent with stress cardiomyopathy with partial recovery to 40–45% at four months. One

year later, she developed non-sustained ventricular tachycardia (NSVT) and a new bifascicular block. She is currently being evaluated for an ICD, with planned sequential α-adrenergic blockade followed by cautious β-blocker uptitration to minimize perioperative hemodynamic instability.

Discussion

Metastatic paraganglioma can mimic acute coronary syndrome, with an initially normal workup due to episodic catecholamine surges delaying diagnosis. Pathophysiology involves α-mediated coronary vasospasm and β-mediated myocardial toxicity, often causing atypical mid-to-apical hypokinesis rather than classic Takotsubo patterns. Management requires strict sequencing—initiate α-blockade before β-blockade to avoid hypertensive crisis from unopposed α-stimulation. Furthermore, identifying PALB2 and ATM mutations via liquid biopsy highlights the role of precision oncology in managing rare, non-resectable tumors.

Conclusion

This case emphasizes that functional paraganglioma must be considered a life-threatening cause of arrhythmias and stress cardiomyopathy. Effective management depends on the prompt identification of catecholamine-induced myocardial injury and rigorous compliance with sequential α-adrenergic blockade followed by β-adrenergic blockade to avert sudden cardiac death and hemodynamic failure. Furthermore, the identification of actionable mutations through genomic profiling highlights the critical role of precision oncology and multidisciplinary cardio-oncology vigilance in treating rare, metastatic neuroendocrine tumors where surgical options are precluded.

Resident/Fellow Clinical Vignette

Persistent Culture-Negative Flank Abscess Due to Ureaplasma parvum

Raghini Ranganathan

Charles Hyman

Ureaplasma species are normal components of the female genital microbiome. Due to their fastidious growth requirements and lack of a cell wall, they are not detected by Gram stain, do not grow on standard bacterial media, and are identified by specialized culture conditions or molecular techniques (16S rRNA sequencing or PCR). Although extragenital Ureaplasma infections are uncommon, increasing use of molecular diagnostics has led to the identification of these organisms as causes of deep soft tissue and musculoskeletal infections, particularly in patients with humoral immunodeficiency.

A 28-year-old woman with multiple sclerosis on ocrelizumab presented with a chronically draining left flank abscess. Five months earlier, she had been hospitalized with abdominal pain, and CT showed inflammatory changes of the descending colon and possible diverticulitis, which resolved following antimicrobial therapy. Three months before presentation, she underwent elective laparoscopic hysterectomy and cholecystectomy. Shortly after, she was readmitted with vomiting and diarrhea; a gastrointestinal pathogen panel identified non-typhoidal Salmonella, and her symptoms resolved with antibiotics. Two months before presentation, she developed a left flank abscess requiring CT-guided drainage. Initial cultures grew Escherichia coli, Salmonella group C, and Streptococcus anginosus and she was treated with amoxicillin-clavulanate. On repeat drainage, the aspirate remained culture-negative and a fistulogram was negative. She presented to our institution with increasing drainage from a wound in the left flank; she was evaluated with flexible sigmoidoscopy and MRI enterography, neither of which demonstrated an enteric fistula. Given the discordance between her clinical course and microbiologic findings, repeat CT-guided drainage was performed. Despite the presence of purulent material, Gram stain and standard bacterial cultures were negative. A sample submitted for broad-range 16S rRNA sequencing identified Ureaplasma parvum. Doxycycline was initiated, resulting in complete clinical resolution, with follow-up MRI confirming resolution of the abscess.

This case illustrates how several factors led to the development of Ureaplasma infection, including ocrelizumab-mediated B-cell depletion, pelvic surgery with possible iatrogenic inoculation and masking by polymicrobial infection. Standard cultures failed to establish the diagnosis, not because of prior antimicrobial exposure but because standard blood

culture media containing sodium polyanethol sulfonate inhibit its growth. Definitive diagnosis was achieved only through broad-range 16S rRNA sequencing. Following targeted doxycycline therapy, the patient experienced complete clinical and radiographic resolution of the infection. With the application of molecular diagnostics, invasive Ureaplasma infections are increasingly recognized as causes of culture-negative deep soft tissue and musculoskeletal infections in patients with humoral immunodeficiency. This case highlights the need for a high index of suspicion for Ureaplasma infection in B-cell-depleted patients with persistent culture-negative abscesses and supports early use of molecular diagnostics. Review of the literature indicates that in most reported cases, treatment with tetracyclines or fluoroquinolones uniformly results in clinical cure.

Resident/Fellow Clinical Vignette

Masquerading as a Bleed: Occult Metastatic Prostate Cancer Presenting as Thrombotic Microangiopathy and Dialysis-Requiring Acute Kidney Injury

Bassam Rao

Irfan, Rabbia; Mehdi, Syed; Mukhtar, Minahil

Introduction

Cancer associated thrombotic microangiopathy (TMA) is a rare paraneoplastic complication most often linked to mucin-producing carcinomas of the stomach, breast, lung, pancreas, and prostate, driven predominantly by microvascular ischemic injury rather than complement or ADAMTS13 mediated mechanisms. We present a case in which a trivial presenting complaint delayed recognition of an aggressive, occult malignancy-associated TMA.

Case

A 91-year-old man with sick sinus syndrome, prior TIA, and several months of weight loss presented with one day of vomiting and minor blood-streaked emesis, initially attributed to a Mallory-Weiss tear. CT incidentally showed lytic/sclerotic vertebral lesions, initially raising concern for multiple myeloma. Over the following days he developed progressive oliguric acute kidney injury (creatinine 2.2→7.8 mg/dL) with gross hematuria and worsening bicytopenia (hemoglobin 12.7→6.7 g/dL; platelets 108→26 ×10^9/L). Peripheral smear showed schistocytes and burr cells; haptoglobin was persistently <30 mg/dL and LDH peaked at 2378 U/L, though reticulocyte count and bilirubin rose only mildly, an atypically blunted hemolytic picture that initially muted suspicion for TMA. DIC, myeloma, TTP, autoimmune hemolysis, and HLH were excluded on fibrinogen, SPEP/free light chains, ADAMTS13, Coombs, and ferritin/triglyceride profile. FDG-PET showed diffuse marrow-based skeletal activity, and PSA returned elevated at 284 ng/mL without alkaline phosphatase elevation. A clinical diagnosis of metastatic prostate cancer-associated TMA was made, and androgen deprivation therapy was started empirically before biopsy. Eculizumab was considered but deferred given the blunted hemolysis and poor reported efficacy of complement inhibition in malignancy-associated TMA. Hemolytic markers and transaminases improved (LDH 2378→963 U/L; bilirubin 2.2→0.9 mg/dL) with platelet recovery, and renal function gradually recovered, with hemodialysis ultimately discontinued. Bone lesion biopsy immunohistochemistry showed tumor cells positive for

AE1/AE3, NKX3.1, androgen receptor, and PSA, and negative for TTF1, CDX2, and PAX8, confirming metastatic prostatic carcinoma.

Discussion

Our case highlights two points. First, anchoring on a trivial presenting complaint can delay recognition of a rapidly progressive, malignancy-associated TMA, particularly when blunted hemolytic parameters and mimicking differentials (myeloma, HLH, TTP) further obscure the diagnosis. Second, the patient's improvement with androgen deprivation alone, without plasma exchange or complement inhibition, is consistent with a multicenter retrospective analysis of 59 patients, in which urgent cancer-directed treatment was the only intervention associated with meaningfully improved 30-day survival, while plasma exchange alone offered no benefit over supportive care. A separate 15-year provincial cohort similarly found that patients with cancer-associated TMA are frequently exposed to unnecessary plasma exchange and delayed oncologic therapy, with newly diagnosed cancers carrying markedly worse prognosis than TMA arising in already treated disease. TMA should remain in the differential for AKI with thrombocytopenia and hemolysis evidence regardless of the presenting complaint, with systematic exclusion of mimics and prompt initiation of cancer-directed therapy once identified.

Resident/Fellow Clinical Vignette

ICD LEAD PERFORATION MASQUERADING AS LATERAL STEMI COMPLICATED BY NEW ONSET ATRIAL FIBRILLATION

Sima Rawal

Sean Stephenson, Indramani Pandey, Kent Stephenson, MD

Background: Cardiac Implantable Electronic Device (CIED), such as pacemakers and Implantable Cardioverter Defibrillator (ICD) lead perforations are rare with varying clinical features. Early manifestations may present as chest pain, dyspnea, and signs of hemodynamic collapse as seen with cardiac tamponade, however micro perforations can also present very subtle with only mild symptoms of acute pericarditis.

Case: This is a case of a 78-year-old male with PMH of coronary artery disease and resuscitated cardiac arrest who underwent a single chamber Biotronik ICD placement for secondary prevention followed by right ventricular ICD lead revision a week later with newly diagnosed Atrial Fibrillation (AF) who then presented within a week after ICD-lead revision with non-pleuritic chest pain, syncope, AF, elevated ICD lead threshold and low impedance despite no evidence of lead migration on chest x-ray. This case represents a dramatic clinical presentation of a patient with a subacute micro perforation initially masquerading as lateral ST-Elevation Myocardial Infarction (STEMI) complicated by newly diagnosed AF with resultant hemopericardium requiring surgical intervention.

Decision-Making: The ECG changes and newly elevated ICD lead threshold is what led to a high level of suspicion with workup of echocardiogram and CT Chest scan revealing the micro lead perforation with subsequent pericardial effusion and hemopericardium (likely exacerbated by intravenous heparin exposure).

Conclusion: CIED lead micro perforation can guise as myocardial infarction, new-onset AF, or acute pericarditis. Acute pericarditis evolves through its phases, particularly it can be easily missed in earlier stages, and as in our patient's case can look like a lateral STEMI in the setting of AF with rapid ventricular response. Early and timely diagnosis of lead micro perforation by CT Chest scan enabled rapid triage of the patient allowing for early life saving surgical intervention.

Teaching Points:

1. Lead perforation can mimic acute coronary syndrome or pericarditis—maintain high suspicion in the post-implant period even with a stable lead position on chest x-ray.

2. Early Device interrogation is critical in any patient with new symptoms post-ICD. But also, essential if the patient develops new arrhythmias early post-op, even without symptoms.

3. Anticoagulation in new-onset AF post-device implant should be individualized; extreme caution should be followed if the presenting patient has evidence of pericardial effusion and pericarditis.

4. Multimodal imaging (Transthoracic Echocardiogram, CT) and close monitoring are critical in unclear presentations despite normal lead position on Chest Xray.

5. Early transfer to tertiary care with Cardiothoracic surgical capability can be lifesaving when complications such as tamponade or perforation are suspected.

Resident/Fellow Clinical Vignette

Exceptional Long-Term Survival Following Post-Allogeneic Transplant Relapse in FLT3-ITD Acute Myeloid Leukemia

Amod Rayamajhi

Anush Patel, MD, FACP

Introduction: FMS-like Tyrosine Kinase 3 - internal tandem duplication (FLT3-ITD) mutations occur in approximately 25–30% of acute myeloid leukemia (AML) cases and are associated with a high risk of relapse and inferior survival, even after allogeneic hematopoietic cell transplantation (allo-HCT). Post-transplant relapse carries a dismal prognosis, usually measured in months. We present a case of exceptional long-term survival following post-transplant relapse managed with azacitidine, sorafenib, donor lymphocyte infusion (DLI), and concurrent graft-versus-host disease (cGVHD) therapy.

Case Presentation: A 55-year-old woman was diagnosed with FLT3-ITD-positive AML in June 2012 and underwent allo-HCT in November 2013. She ultimately developed chronic GVHD involving the skin, joints, and lungs, which was managed initially with ruxolitinib and rituximab. She relapsed in December 2016 and was treated with azacitidine plus sorafenib, a guideline-supported regimen for relapsed FLT3-ITD AML.

By June 2017, bone marrow biopsy demonstrated minimal residual disease with conversion to FLT3 negativity. She subsequently received DLI in December 2017 and continued azacitidine (dose-reduced to 50 mg/m² for 3 days every 6 weeks due to cytopenias) with daily sorafenib.

Subsequently, she underwent sequential therapies including belumosudil, abatacept, hydroxychloroquine, prednisone and IVIG every 6 weeks. Sorafenib was briefly discontinued in May 2025 after achieving FLT3 MRD negativity but was restarted in May 2026 for dual management of AML and GVHD. As of July 2026, nearly 10 years after relapse and 14 years after diagnosis, the patient remains in remission with an ECOG performance status of 1 and stable blood counts (WBC 11.4, Hgb 13.1, Plts 407).

Discussion: Several features of this case deserve emphasis. First, the combination of azacitidine and sorafenib may achieve durable, long-term disease control in relapsed FLT3-ITD AML following allo-HCT, with this patient's response far exceeding the durable remissions typically reported in the literature. Previous studies have demonstrated improved post-transplant outcomes with sorafenib, supporting its role in FLT3-ITD AML. Notably, sorafenib's immunomodulatory properties—including induction of IL-15

production in FLT3-ITD leukemia cells and promotion of CD8+ T-cell-mediated graft-versus-leukemia effects—may have acted synergistically in this case with the allogeneic graft and DLI to produce this exceptional response. Additionally, the patient's chronic GVHD, which itself required complex management, may have contributed to leukemia control through ongoing alloreactivity.

The optimal duration of FLT3-targeted therapy remains undefined. In this case, the brief discontinuation and subsequent reinitiation of sorafenib emphasize its potential dual role in AML and GVHD co-management.

Conclusion: This case suggests that durable remission after post-transplant FLT3-ITD AML relapse is possible with an individualized multimodal approach integrating targeted therapy, hypomethylating agents, donor lymphocyte infusion, and individualized chronic GVHD management.

Resident/Fellow Clinical Vignette

Beyond the Rash: Herpes Zoster Cervical Myelitis in an Immunocompetent Patient

Trisha Mae Riparip

William Bachman, MD

Purpose:

To report a rare case of herpes zoster-associated cervical transverse myelitis in an immunocompetent patient, highlighting the importance of early recognition and prompt treatment when neurological symptoms progress despite resolution of skin lesions.

Methods:

A single-patient clinical vignette with review of related literature.

Case summary:

A 62-year-old immunocompetent male presented with a painful vesicular rash involving the right neck, shoulder, upper chest and arm (C3-C5 dermatome). He was treated with oral valacyclovir and steroid taper. Approximately 1 week later, he developed progressive weakness of the right upper extremity, followed by bilateral lower extremity weakness more pronounced on the right side. Despite a second course of oral antivirals and steroids, his symptoms worsened even as his lesions healed and crusted. He was hospitalized for expedited workup. MRI of the cervical spine revealed T2 hyperintense lesions at the C3-C4 level. Cerebrospinal fluid analysis demonstrated mild lymphocytic pleocytosis (20 WBCs, 95% lymphocytes). Varicella Zoster Virus (VZV) IgM serology was elevated; however, VZV DNA PCR in CSF was negative, consistent with a post–infectious inflammatory process. During hospitalization, he also developed urinary retention. He was diagnosed with herpes zoster–associated transverse myelitis and started on high–dose intravenous acyclovir with partial improvement of symptoms. He was discharged on oral valacyclovir with outpatient rehabilitation. At 4-month follow–up, the patient demonstrated gradual neurological improvement.

Conclusion:

This case demonstrates a rare presentation of cervical VZV myelitis in an immunocompetent patient whose neurological symptoms progressed despite outpatient oral antivirals and corticosteroids, ultimately requiring hospitalization and intravenous acyclovir. Clinicians should suspect VZV myelitis when motor weakness, sensory changes,

or sphincter dysfunction develop in herpes zoster patients, even after rash resolution. Diagnosis relies on clinical context, MRI findings such as T2 hyperintense lesions, and CSF analysis showing lymphocytic pleocytosis; notably, VZV PCR in CSR may be negative beyond acute phase and/or after antiviral therapy. IV acyclovir is preferred for CNS involvement, as oral antivirals may be insufficient, and adjunctive corticosteroids may help reduce inflammation. Our patient's immunocompetent status and the cumulative antiviral and corticosteroid therapy he received likely contributed to his favorable recovery, reinforcing the importance of early treatment and timely escalation of care.

Resident/Fellow Clinical Vignette

BEYOND INFECTION CONTROL: A CASE OF MEROPENEM-INDUCED CHOLESTASIS

Shanna Rodriguez

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Background

Drug-induced liver injury (DILI) represents a diagnostic challenge requiring exclusion of competing etiologies, with cholestatic patterns—characterized by predominant alkaline phosphatase (ALP) elevation—accounting for 20–40% of cases and demonstrating particular predilection for older adults1. Although antimicrobial agents constitute the most frequently implicated drug class in DILI, with annual incidence estimated at 10–14 cases per 100,000 inhabitants, carbapenem-associated hepatotoxicity remains rarely reported despite widespread clinical use2. We present a case of meropenem-induced cholestatic liver injury in a patient with multiple comorbidities, highlighting the diagnostic approach and clinical implications.

Case presentation

A 50-year-old woman with end-stage renal disease on hemodialysis, diabetes mellitus, hypertension, and prior left lower extremity amputation presented with right foot infection and left stump complications. Imaging confirmed osteomyelitis, necessitating right heel debridement and subsequent left above-knee amputation. Baseline ALP was 217 U/L. Following meropenem initiation, ALP progressively increased to 449 U/L and eventually exceeded 1,200 U/L, demonstrating a cholestatic biochemical pattern with only mild transaminase elevation and preserved bilirubin.

Diagnostic evaluation

Comprehensive evaluation excluded alternative etiologies. Abdominal ultrasound and magnetic resonance cholangiopancreatography revealed no biliary obstruction. Viral hepatitis serologies were non-reactive, and autoimmune markers were unremarkable. Critically, ALP isoenzyme fractionation confirmed predominantly hepatic origin (liver 72%, macrohepatic 14%, bone 13%), thereby excluding bone-related elevation attributable to osteomyelitis or recent amputation3. The temporal relationship between meropenem exposure and ALP rise, combined with biochemical improvement following drug discontinuation, supported the diagnosis of DILI.

Discussion

Cholestatic DILI typically manifests within 2–24 weeks of drug exposure and may demonstrate prolonged recovery following withdrawal3. The pattern of isolated, marked ALP elevation with minimal transaminase abnormalities is consistent with cholestatic injury. The RUCAM (Roussel Uclaf Causality Assessment Method) score provides a standardized framework for evaluating causality, emphasizing temporal relationship, exclusion of alternative causes, and response to dechallenge4. Cholestatic DILI carries risk of progression to chronic injury or vanishing bile duct syndrome in rare cases, underscoring the importance of early recognition1.

Conclusion

Meropenem represents a rare but potential cause of cholestatic DILI. In patients with disproportionate ALP elevation during antimicrobial therapy and unrevealing hepatobiliary imaging, clinicians should maintain appropriate suspicion for DILI. Prompt identification and discontinuation of the offending agent remain essential to prevent progression to chronic liver injury and promote recovery.

Resident/Fellow Clinical Vignette

WALKING WITH FUNGEMIA: REFRACTORY CANDIDA AURIS CANDIDEMIA IN A CLINICALLY WELL APPEARING HEMODIALYSIS PATIENT DESPITE ECHINOCANDIN SUSCEPTIBILITY AND SOURCE CONTROL

Archa Roy

Agun Nukaya B.MED, Monica Ghitan MD, Vikash A Ramtahal MD

Candida auris is an emerging multidrug-resistant yeast associated with healthcare transmission, invasive devices, and persistent bloodstream infection. Persistent candidemia typically raises concern for inadequate source control or deep-seated infection and is often accompanied by significant clinical illness. We describe a unusual case of prolonged C. auris fungemia in a clinically stable hemodialysis patient without an identifiable infectious source.

A 71-year-old man with hypertension, end-stage renal disease on maintenance hemodialysis, and prior right subdural hematoma evacuation presented with encephalopathy. Brain imaging demonstrated a right temporal intraparenchymal hemorrhage with scattered bilateral acute and subacute infarcts concerning for embolic phenomena. During hospitalization, he developed new-onset atrial fibrillation. Transesophageal echocardiography showed no valvular vegetations, a patent foramen ovale with right-to-left shunting and multiple echogenic aortic arch plaques suspicious for thrombus or complex atheroma.Admission blood cultures grew Candida auris. All subsequent positive blood cultures during the hospitalization also grew C. auris, with serial susceptibility testing demonstrating preserved micafungin susceptibility throughout. Micafungin was initiated IV, and the tunneled dialysis catheter was promptly removed, with hemodialysis continued through a mature arteriovenous fistula. Despite catheter removal and appropriate antifungal therapy, the patient remained persistently fungemic, with approximately 15 positive blood cultures over 2.5 weeks. Micafungin was increased to 150 mg daily, and liposomal amphotericin B was added for refractory candidemia.Despite prolonged fungemia, the patient remained afebrile, hemodynamically stable, ambulatory, and without leukocytosis or other clinical manifestations of systemic infection. The first blood culture obtained after initiation of amphotericin B remained positive; with negative subsequent cultures and sustained clearance thereafter. Eye examination showed no evidence of fungal endophthalmitis. Vascular evaluation of the AV fistula showed no evidence of infection. Gallium scintigraphy, FDG-PET imaging, and cardiothoracic evaluation of the abnormal aortic arch findings failed to identify a definitive infectious

source or endovascular focus. The patient was discharged to complete 6 week course of IV micafungin.

This case highlights the striking discordance between persistent C. auris bloodstream infection and an unexpectedly benign clinical course. Although prolonged candidemia generally suggests uncontrolled infection or a deep-seated source, this patient remained clinically well despite nearly three weeks of persistent positive blood cultures and micafungin susceptibility. While occult endovascular infection could not be definitively excluded, persistent fungemia may reflect biofilm formation, antifungal tolerance, heteroresistance, or persistent colonization despite favorable in vitro susceptibility results. This case emphasizes that microbiologic persistence and clinical severity may not always correlate in C. auris infection.

Persistent Candida auris candidemia can occur in clinically stable patients despite prompt source control, preserved echinocandin susceptibility, and extensive evaluation without an identified source. In hemodialysis patients, persistent fungemia should prompt continued investigation for occult device-related or endovascular infection while recognizing that prolonged bloodstream positivity may occur even without overt sepsis or clinical deterioration.

Resident/Fellow Clinical Vignette

A RARE CASE OF PEMPHIGUS VULGARIS

Haleema Sadia

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Background: Pemphigus vegetans is a rare variant of pemphigus vulgaris, making up roughly 1–2% of cases [1]. It presents with vegetating plaques that develop in intertriginous areas, and when the clinical picture is atypical or complicated by infection, diagnosis and management can be challenging [2].

Case Presentation: A 63-year-old male patient presented to the emergency department with a 2 years history of worsening bilateral inguinal vegetating plaques with a cauliflower-like appearance, along with involvement of the oral mucosa. During this presentation, wound cultures revealed extended spectrum β-lactamase (ESBL)-producing organisms (Proteus and Klebsiella species), and he was treated with doxycycline 100 mg daily for 14 days, meropenem 1 g every 8 hours for 14 days, and Diflucan 200 mg daily for 7 days. Five months later, he presented to a dental clinic with ulceration of the oral mucosa extending to the lower lip. A pemphigus disorder was suspected, and histopathology revealed supranasal acantholysis. Furthermore, immunofluorescence showed intercellular Immunoglobulin G and Complement Component 3 deposition in the epidermis, and anti-desmoglein 3 antibody levels were markedly elevated at 88 units/mL, (reference 14–20 U/mL). The clinical course was further complicated by secondary bacterial infections with concurrent mycobacterial disease, including latent tuberculosis and Mycobacterium avium intracellular infection.

Management and Outcome: A multidisciplinary approach was employed, including targeted antimicrobial therapy and staged immunosuppression. The treatment plan is rifampin for 2 weeks followed by immunosuppressive therapy azathioprine vs mycophenolate mofetil with rifampin for 4 months. Rifampin treatment has been delayed due to insurance coverage issues, and the patient's outcome will be followed closely.

Conclusion: Pemphigus vegetans remains a diagnostically challenging and potentially life-threatening condition, particularly when complicated by secondary infections and coexisting mycobacterial disease [2]. Early recognition, comprehensive diagnostic

evaluation, and thorough infectious disease screening are essential before initiating immunosuppressive therapy. Modern treatment strategies, particularly rituximab-based regimens, offer improved outcomes with reduced corticosteroid burden and lower rates of severe adverse events [2]. This case underscores the importance of recognizing an exceedingly rare condition and a multidisciplinary approach in managing complex presentations of rare pemphigus variants.

Resident/Fellow Clinical Vignette

BEYOND THE USUAL SUSPECTS: DOCETAXEL-ASSOCIATED CAPILLARY LEAK SYNDROME PRESENTING WITH REFRACTORY HYPOXEMIA

Maryam Sardar

Amna Sardar, Muhammad Usman Siddique, Hassan Sardar, Kinzah Sardar

Introduction:

Docetaxel-associated capillary leak syndrome is a rarely reported, potentially life-threatening toxicity that may present as acute hypoxemic respiratory failure and mimic common cardiopulmonary conditions, leading to diagnostic difficulty.

Case Presentation:

A 70-year-old man with metastatic castration-sensitive prostate cancer on relugolix, darolutamide, and docetaxel, COPD, and prior left upper lobectomy for NSCLC presented with progressive dyspnea, subjective fever, and diffuse edema following chemotherapy. He was hemodynamically stable but profoundly hypoxemic with oxygen saturation in the 70s on room air, requiring high-flow nasal cannula oxygen. Arterial blood gas showed PaO_2 61 mmHg. Laboratory evaluation demonstrated leukocytosis (20.2 ×10^9/L), elevated BNP (1250 pg/mL), with normal troponin and negative viral panel; remaining laboratory studies were unremarkable. Leukocytosis was attributed to possible infection versus recent pegfilgrastim exposure. Empiric antibiotics were initiated for suspected pneumonia, and intravenous diuresis was started for possible volume overload. Despite therapy, hypoxemia worsened with escalating oxygen requirements. CTA chest was

negative for pulmonary embolism and demonstrated emphysematous changes with mild dependent atelectasis and small right pleural effusion, without consolidation. Transthoracic echocardiography showed preserved biventricular systolic and diastolic function. Given refractory hypoxemia and exclusion of infectious, thromboembolic, and cardiogenic etiologies, docetaxel-associated capillary leak syndrome was suspected. The patient received intravenous methylprednisolone 60 mg daily with continued diuresis, resulting in rapid clinical improvement over three days. He was successfully weaned off oxygen and transitioned to an oral prednisone taper. Docetaxel was discontinued.

Discussion:

Capillary leak syndrome is a systemic endothelial permeability disorder resulting in intravascular fluid extravasation and third spacing. Docetaxel-associated capillary leak

syndrome is a rarely reported, potentially life-threatening toxicity, with proposed mechanisms including cytokine-mediated endothelial injury and capillary hyperpermeability. Although most reported cases demonstrate reversibility with withdrawal of docetaxel and supportive care, initial attribution to more common etiologies may delay diagnosis and has been associated with worse clinical outcomes.

Conclusion:

Capillary leak syndrome should be considered in patients receiving taxane chemotherapy who present with unexplained hypoxemia and edema despite negative infectious, thromboembolic, and cardiogenic evaluation. Early corticosteroid therapy has been associated with rapid clinical improvement in reported cases.

Resident/Fellow Clinical Vignette

Beyond ANCA; Recurrent Life Threatening Diffuse Alveolar Hemorrhage Without Classical IgA Vasculitis Manifestations

Nailah Sayeed

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Diffuse alveolar hemorrhage (DAH) is a rare but life-threatening extrarenal manifestation of IgA nephropathy (IgAN) that is infrequently recognized and carries substantial morbidity and mortality. While IgA vasculitis classically presents with palpable purpura, arthralgia, abdominal pain, and nephritis, pulmonary capillaritis remains an underappreciated manifestation within the IgA-mediated disease spectrum. Early recognition is critical, as delayed treatment carries substantial morbidity and mortality.

We report the case of a 42-year-old Hispanic male with biopsy-proven IgA nephropathy progressed to end-stage renal disease (ESRD), alcoholic cirrhosis with portal hypertension, and hypertension, who presented with recurrent hemoptysis, acute-on-chronic anemia, hyperkalemia, and hypoxemia over a two-month period requiring three hospitalizations. Flexible bronchoscopy with bronchoalveolar lavage confirmed DAH with progressively hemorrhagic returns. An exhaustive infectious workup was negative, and comprehensive autoimmune serologies including ANCA, anti-GBM antibody, ANA, anti-dsDNA, and anti-Smith were all negative or clinically insignificant. Notably, the patient lacked the classic tetrad of IgA vasculitis (palpable purpura, arthralgia, abdominal pain, and nephritis), presenting instead with an atypical pulmonary-renal syndrome phenotype. The clinical constellation of bronchoscopically confirmed DAH in the setting of biopsy-proven IgAN, with comprehensively negative alternative etiologies, was consistent with IgA nephropathy-associated pulmonary capillaritis within the IgA-mediated disease spectrum. Treatment was initiated with pulse intravenous methylprednisolone followed by high-dose oral prednisone and rituximab, with favorable early clinical response.

This case adds to the limited literature on IgAN-associated DAH, highlights the importance of considering IgA-mediated pulmonary capillaritis in patients with IgAN presenting with unexplained hemoptysis and pulmonary infiltrates even in the absence of classic vasculitic features, and supports rituximab as a potential therapeutic alternative to cyclophosphamide in this rare but serious complication.

Resident/Fellow Clinical Vignette

Impending Paradoxical Embolism: Trans-Septal Thrombus Triggered by Massive Pulmonary Embolism

Nensi Shah

Samy Matta

Cho Thet Zen

Markos Endalew

Hejmadi Prabhu

Introduction: A trans-septal thrombus, or impending paradoxical embolism, is a rare, life-threatening condition where a venous clot lodges across a patent foramen ovale (PFO), bridging both atria. It requires immediate recognition to prevent systemic arterial embolization. Massive pulmonary embolism (PE) can acutely increase right-sided cardiac pressures, forcing open a dormant PFO and allowing thrombi to cross into the left atrium.

Case Description: A 71-year-old man presented to the emergency department following an episode of syncope. Due to immediate concerns for an acute ischemic stroke, an urgent neurological evaluation was initiated. CT imaging of the brain, alongside head and neck angiogram and perfusion studies, revealed bilateral areas of hypoperfusion (localized cerebellar hypoperfusion, greater on the right, and in the inferior left temporal lobe), heightening suspicion for a thromboembolic phenomenon. An electrocardiogram demonstrated atrial fibrillation with a right bundle branch block, further suggesting a cardiac origin. Crucially, the head and neck CT angiogram revealed an incidental pulmonary embolism captured at the lung bases. A subsequent CT angiogram of the chest confirmed extensive, bilateral pulmonary emboli with signs of severe right heart strain and a thrombus in the right atrium. A transthoracic echocardiogram definitively demonstrated a large right atrial thrombus attached to the interatrial septum, extending directly through the PFO into the left atrium. It also revealed severe dilation of the right atrium and right ventricle, with markedly reduced right ventricular systolic function. Venous Doppler ultrasonography of the lower extremities identified an acute, partially occlusive deep venous thrombosis in the popliteal vein. Given the imminent risk of systemic embolization, the patient was emergently transferred to a tertiary care center for advanced intervention.

Discussion: For stroke patients over 60 years old, or younger patients with atrial fibrillation, standard protocols assume an embolic source from the left atrial appendage; checking for a right-to-left shunt is not the standard of care. However, this case demonstrates that

clinicians must maintain a high index of suspicion for paradoxical embolism when right-sided heart pressures are acutely elevated. Massive PE with right heart strain can cause transient opening of a PFO, creating an operational shunt for venous clots. Early diagnostic imaging, specifically echocardiography, is vital in these settings to detect trans-septal thrombi and prevent catastrophic systemic embolic complications.

Resident/Fellow Clinical Vignette

INTRAHOSPITAL RESPIRATORY EMERGENCIES: THE PREDICTIVE CAPACITY OF OXYGEN REQUIREMENTS FOR ACUTE INPATIENT RESPIRATORY FAILURE

Brett Sherman

Sofia Toro Alvarez, MD; Andrea Ramirez Cazares, DO; Javier Ticona, MD; Jacob Bell, MD; Adam Rothman, MD

Purpose

The COVID-19 pandemic prompted expansion of non-invasive ventilation (high flow nasal cannula, CPAP, or BPAP) throughout the non-intensive care inpatient setting. In the years since, inpatient medical teams have displayed relative comfort in managing respiratory failure, often without critical care or pulmonary subspecialty involvement. This investigation aimed to evaluate whether patients' initial or progressive oxygen requirements were predictive of subsequent intrahospital mortality and to identify whether these patients may have benefitted from earlier intensive care involvement.

Methods

This retrospective review was conducted in a tertiary care hospital system in New York City. Patients were eligible for enrollment if assessed during a rapid response team activation for worsening respiratory distress between July 1, 2024 and June 30, 2025. Each patient's oxygen requirements were recorded for both the periods leading up to and following critical care team activation. An increase in oxygen requirement was defined as either a numeric increase in liters of oxygen per minute delivered or an increase in oxygen delivery method along the following hierarchy: nasal cannula, non-rebreather mask, high flow nasal cannula, BPAP, mechanical ventilation. Recurrent critical care activation(s) and final patient disposition were also examined, and correlation analysis was performed on the collected data.

Results

106 patients met inclusion criteria and were enrolled in this study. Of the study population, 57 were male (54%) and 49 (46%) were female, with an average age of 70 years. 40 patients (38%) expired during admission; of this subpopulation, 22 patients (55%) had an oxygen requirement of any kind upon initial hospital admission ($r=0.01$, $p=0.91$). There was also no significant correlation between oxygen requirement increase between or following rapid response activation and eventual patient mortality ($r=-0.01$, $p=0.95$).

Conclusions

Patient oxygen requirement at hospital admission or change in oxygen requirement following rapid response team activation for respiratory distress had no statistically significant correlation with subsequent patient mortality outside of the intensive care unit.

This analysis suggests that, for patients who experience acute respiratory decline during hospital admission, oxygen requirement alone may not be useful in identifying patients for whom early pulmonary or critical care involvement will impact final outcomes. Future review of secondary outcomes, including hospital length-of-stay and discharge disposition, is needed to confirm whether this holds true beyond the primary outcome of mortality.

Resident/Fellow Clinical Vignette

WHEN VISION REVEALS LEUKEMIA : LEUKOSTASIS-ASSOCIATED RETINOPATHY IN CHRONIC-PHASE CML

Hassan Sinan

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Background: Chronic myeloid leukemia (CML) is a clonal myeloproliferative neoplasm and typically presents in the chronic phase with nonspecific symptoms. Leukostasis is a rare and potentially life-threatening complication of hyperleukocytosis, most commonly associated with acute leukemias and a high blast count. Ocular manifestations are uncommon initial presentations of CML and leukostasis-related retinopathy in chronic-phase disease is particularly rare.

Case Presentation: A 55-year-old man presented with one month of progressive painless blurry vision in the left eye, unintentional weight loss, and night sweats. Ophthalmologic examination demonstrated bilateral optic disc edema, retinal hemorrhages, Roth spots, and vascular tortuosity, with visual acuity of 20/25 in the right eye and 20/30 in the left eye. Laboratory evaluation was notable for marked leukocytosis (431 ×10^9/L). Neuro-ophthalmic testing demonstrated generalized visual field depression and enlarged blind spots. Initial imaging raised concern for venous outflow abnormality, and lumbar puncture revealed elevated opening pressure. Peripheral blood flow cytometry demonstrated 1.5% circulating blasts, and bone marrow biopsy confirmed chronic-phase CML (CP-CML). MRI and MR venography excluded cerebral venous thrombosis and intracranial mass. Given the severe hyperleukocytosis, characteristic ophthalmologic findings and elevated intracranial pressure (ICP), leukostasis-associated retinopathy was suspected. The patient underwent emergent leukapheresis, was started on hydroxyurea for cytoreduction, and initiated on dasatinib. Neuro-oncology evaluation concluded that the elevated ICP was likely related to an impaired venous outflow secondary to hyperleukocytosis and leukostasis. Acetazolamide was added for ICP management. The patient experienced rapid clinical improvement, with resolution of visual symptoms, normalization of visual acuity to 20/20, and marked improvement in retinal findings. His leukocyte count decreased by >50% during hospitalization and normalized (6.3 ×10^9/L) within three weeks of discharge.

Discussion: This case highlights an uncommon presentation of chronic-phase CML: severe leukostasis resulting in retinopathy and elevated ICP. Leukostasis is uncommon in chronic-phase CML and is more frequently observed in acute leukemias with high circulating blast burden where impaired microvascular perfusion results from leukemic cell aggregation, endothelial activation and vascular obstruction. Ocular leukostasis may manifest as retinal hemorrhages, Roth spots, optic disc edema, and visual field deficits. Such findings are rarely the presenting manifestation of CML, particularly in patients with very low blast percentages and without the respiratory or overt neurological symptoms classically associated with leukostasis. Early recognition, prompt ophthalmologic evaluation, and timely initiation of cytoreductive therapy and tyrosine kinase inhibitor therapy are critical to achieving rapid clinical improvement and preventing irreversible organ damage.

Resident/Fellow Clinical Vignette

THE DOUBLE-EDGED SWORD: LINEZOLID TOXICITY MASKED AS SEPTIC SHOCK

Nikita Srivalsan

Ashlesha Chaudhary

Dikshya Khatiwada

Introduction:

Linezolid is an oxazolidinone antibiotic commonly used for resistant gram-positive infections, including methicillin-resistant Staphylococcus aureus (MRSA). Although effective, prolonged therapy has been associated with mitochondrial toxicity resulting in hematologic abnormalities, lactic acidosis, and peripheral neuropathy. Pure red cell aplasia (PRCA), characterized by profound suppression of erythroid production with severe reticulocytopenic anemia, is a rare but serious complication. We present a case of multisystem linezolid toxicity initially presenting as presumed septic shock.

Case Presentation:

A 47-year-old woman with insulin-dependent type 2 diabetes mellitus, peripheral neuropathy, rheumatoid arthritis on chronic prednisone, hypertension, hyperlipidemia, and peripheral arterial disease presented with fever, chills and right-sided abdominal hyperesthesia. Three weeks prior, she underwent incision and drainage for a left diabetic foot infection concerning for acute on chronic osteomyelitis and cultures positive for MRSA. Due to severe nausea and vomiting with vancomycin, linezolid was selected for a planned six-week course.

Three days prior, she was hospitalized for severe anemia with significant vaginal bleeding. She had inadequate response to four units of packed red blood cells and ultimately needed levonorgestrel intrauterine device placement.

On current presentation, she was tachycardic and developed hypotension requiring phenylephrine support. Labs revealed leukocytosis (12.1 K/µL), worsening anemia (hemoglobin 5.1 g/dL), thrombocytopenia (79 K/µL), acute kidney injury, and elevated lactate (2.7 mmol/L). Presumed recurrent sepsis was diagnosed; IV linezolid was continued, and cefepime was added. No clear infectious source was identified. Further evaluation revealed profound reticulocytopenia (absolute reticulocyte count 0 K/µL, index 0%) and along with her constellation of findings, linezolid toxicity was suspected.

Linezolid was discontinued and daptomycin was initiated. Within 72 hours, she demonstrated rapid clinical improvement with resolution of hemodynamic instability and symptoms.

Discussion:

Linezolid toxicity is an uncommon but important complication of prolonged therapy. Unlike most antibiotics, linezolid inhibits mitochondrial protein synthesis due to similarities between bacterial and human mitochondrial ribosomes. This impairment results in a characteristic spectrum of toxicity, including lactic acidosis, peripheral neuropathy, and bone marrow suppression.

Hematologic toxicity, predominantly thrombocytopenia, typically occurs after prolonged linezolid exposure of two or more weeks. However, severe erythroid suppression resulting in PRCA is exceedingly rare.

This case highlights the challenge of recognizing linezolid toxicity when alternative explanations are present. The patient's severe anemia was initially anchored to vaginal blood loss; however, the absence of a reticulocyte response revealed profound erythroid production failure. Additionally, the combination of elevated lactate, hemodynamic instability, and recent infection created a clinical picture suggestive of sepsis but ultimately attributable to medication toxicity.

Maintaining a high index of suspicion for linezolid-associated toxicity in patients receiving prolonged courses, particularly when new cytopenias, unexplained lactic acidosis, or neurologic symptoms develop. Early recognition and discontinuation of the offending agent may prevent severe complications and lead to rapid clinical recovery.

Resident/Fellow Clinical Vignette

Seronegative Paraneoplastic Encephalitis Heralding Occult Gallbladder Adenocarcinoma: A Diagnostic Odyssey

Tahsin Sultana

Yehia Abdelrahman MBBS

Introduction

Paraneoplastic neurological syndromes are exceedingly rare in gallbladder adenocarcinoma (GBC), with published reports limited to peripheral neuropathy, cranial nerve palsies, and opsoclonus. A frank paraneoplastic encephalitis has not, to our knowledge, been previously reported in GBC. Diagnosis is difficult because such syndromes typically precede recognition of the underlying malignancy and mimic more common entities, including stroke, CNS lymphoma, and infectious encephalitis. We report a case of rapidly progressive, antibody-negative paraneoplastic encephalitis that led to discovery of occult, biopsy-proven GBC.

Case Description

A 66-year-old man with Crohn's disease, type 2 diabetes, and hypertension presented with three days of progressive right-sided ataxia, numbness, and a band-like abdominal sensation. Initial MRI showed a left basal ganglia, thalamic, and corona radiata lesion attributed to infarction. Over the next four to six weeks, he developed bilateral visual loss, fluctuating ptosis, dysarthria, hemineglect, and fluctuating responsiveness. Repeat MRI demonstrated multifocal, evolving, non-enhancing T2/FLAIR hyperintensities with peripheral diffusion restriction involving the bilateral basal ganglia, thalami, brainstem, and cerebral peduncles — atypical for infarction or metastasis given the rapid progression of disease. CSF showed elevated interleukin-6, negative cytology, and a minute lambda-restricted B-cell population (MYD88 negative). An extensive infectious, autoimmune, metabolic, and toxic workup, including a paraneoplastic antibody panel, was unrevealing. Abdominal imaging incidentally identified a gallbladder mass with hepatic and lymphatic spread. Biopsy and radical cholecystectomy with partial hepatectomy confirmed moderately differentiated invasive adenocarcinoma with mixed clear-cell and biliary-type morphology, arising from high-grade biliary intraepithelial neoplasia, with lymphovascular and perineural invasion and metastasis to two pericystic lymph nodes. Given the temporal correlation with GBC discovery, seronegative paraneoplastic encephalitis was diagnosed as probable. He was treated with five sessions of plasma exchange, high-dose corticosteroids, and intravenous immunoglobulin, plus surgical resection; his

postoperative course was complicated by hemorrhage requiring reoperation. He demonstrated gradual, partial neurologic recovery with improved responsiveness and oral intake, though he required gastrostomy for persistent dysphagia.

Discussion

This case illustrates that GBC, though rarely linked to paraneoplastic neurological syndromes, should be considered in the differential of rapidly progressive, multifocal, non-enhancing encephalopathy once stroke, infection, CNS lymphoma, and demyelinating disease are excluded — particularly when antibody panels are negative, as most paraneoplastic encephalitides remain seronegative despite a genuine autoimmune mechanism. Previously reported GBC-associated neurologic phenomena include peripheral neuropathy, cranial nerve palsies, and opsoclonus with impaired consciousness; encephalitis with multifocal deep gray matter and brainstem involvement has not previously been described with this malignancy. Early empiric immunotherapy, tumor source control, and multidisciplinary collaboration were central to this patient's partial recovery. Clinicians should maintain a high index of suspicion for occult malignancy in patients with atypical, antibody-negative encephalitis, as timely tumor identification may carry both neurologic and oncologic importance.

Resident/Fellow Clinical Vignette

Semaglutide-Attributed Gastrointestinal Symptoms Masking High-Grade Serous Carcinoma: A Diagnostic Pitfall in the Era of GLP-1 Receptor Agonists

Asma Tameez ud din

Syeda Rukh

Nina Rizk

Introduction:

Semaglutide, a GLP-1 receptor agonist (GLP-1RA), is increasingly prescribed for obesity and related comorbidities. Gastrointestinal (GI) side effects — including nausea (25%–44%), vomiting (8%–24%), and diarrhea (19%–30%) — are the most commonly reported adverse events and typically occur during dose escalation. These symptoms overlap with the nonspecific presentation of advanced genitourinary cancer. We present a case in which semaglutide-attributed GI symptoms masked the diagnosis of high-grade serous carcinoma (HGSC) of tubo-ovarian or peritoneal origin, leading to life-threatening complications.

Case Description:

A 40-year-old woman with a history of heart failure with preserved ejection fraction (HFpEF), obesity hypoventilation syndrome, obstructive sleep apnea, and morbid obesity was admitted with hypochloremic metabolic alkalosis and acute kidney injury (AKI) after two weeks of persistent nausea, vomiting, diarrhea, and poor oral intake with hypotension following semaglutide dose escalation. She had been started on semaglutide and diuretics two months prior when she was diagnosed with heart failure after presenting with progressive dyspnea.

On admission, CT abdomen demonstrated large-volume ascites, bilateral pleural effusions, and omental thickening. Thoracentesis and paracentesis were performed, with ascitic fluid demonstrating a serum-ascites albumin gradient (SAAG) of 0.5, making portal hypertension unlikely. Fluid cytology and IR-guided biopsy of omental lesions identified high-grade serous carcinoma of tubo-ovarian/peritoneal origin. Pelvic exam was negative, and transvaginal ultrasound was nondiagnostic. CA-125 was markedly elevated; HE4 was not obtained due to AKI.

Given elevated CA 125, recurrent pleural effusions, ascites, and worsening AKI, gynecologic oncology initiated inpatient carboplatin/paclitaxel chemotherapy. She subsequently developed tumor lysis syndrome (TLS) requiring rasburicase and oliguric renal failure requiring continuous renal replacement therapy (CRRT) and transfer to the medical ICU. Her course was complicated by neutropenic fever, E. coli bacteremia, septic and hypovolemic shock requiring vasopressors, respiratory failure, and ultimately transition to comfort-focused care.

Discussion:

This case highlights a critical diagnostic pitfall in the era of widespread GLP-1RA prescribing: the attribution of cancer-related symptoms to known drug side effects. The GI adverse effect profile of semaglutide closely mirrors the nonspecific symptomatology of intraabdominal malignancy including genitourinary cancer, including nausea, vomiting, abdominal pain, bloating, and early satiety. The development of TLS following carboplatin/paclitaxel is exceedingly rare in ovarian cancer, with only one prior case reported. This case underscores the need for clinicians to maintain a broad differential when patients on GLP-1RAs present with persistent or worsening GI symptoms — particularly when accompanied by new ascites, effusions, or weight change, rather than reflexively attributing symptoms to the medication. As GLP-1RA prescribing continues to expand, awareness of this diagnostic blind spot is essential to avoid delays in cancer diagnosis.

Resident/Fellow Clinical Vignette

FAVORABLE RESPONSE TO EARLY EMPIRIC TREATMENT OF ERLICHIA-ASSOCIATED HYPERINFLAMMATORY RESPONSE IN THE MIDST OF DIAGNOSTIC UNCERTAINTY

Karis Tutuska

Naiji Gong

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Shabnam Zoeb, DO

Introduction: Ehrlichiosis, a tick-borne infection with an intracellular bacteria of the Erlichia genus, presents with fever, leukopenia, thrombocytopenia, and transaminitis. While most infections are self-limited, rare cases can result in multi-organ failure and secondary hemophagocytic lymphohistiocytosis (HLH), a severe hyperinflammatory response.

Case Description: A 91 year old man with CKD stage III presented with five days of headache and malaise (day 0). He was febrile to 39.2 C with a new oxygen requirement. Respiratory viral panel, urinalysis, and blood cultures were negative. Laboratory studies were significant for worsening AKI on CKD, anemia (Hgb low of 9 g/dL), thrombocytopenia (platelet low of 55 K/uL), and transaminitis. LDH was elevated to a peak of 1410 U/L with inappropriately low reticulocyte count and elevated inflammatory markers. TTP and other hemolytic processes were ruled out with normal haptoglobin and bilirubin. He was started on broad spectrum antibiotics with Vancomycin and Zosyn, however on day 1 additional history revealed a recent tick bite and he was switched to empiric Doxycycline, Atovaquone, and Azithromycin while pending tick borne illness panel (day 1). His clinical condition continued to worsen despite this regimen. On day 2 , ferritin resulted at 49,641 ng/mL with an associated D-dimer of 3,973 ng/mL, elevated triglyceride level of 219 mg/dL, and fibrinogen of 362 mg/dL. Due to cytopenia/hypertriglyceridemia below cut off values and lack of hypofibrinoginemia and splenomegaly, this presentation did not meet HLH-2024 diagnostic criteria. In the setting of clinical worsening on antibiotics, empiric treatment for HLH was initiated with high dose IV Dexamethasone (day 3). Hematology and Rheumatology were consulted, who considered HLH less likely but agreed with empiric high dose steroids in the setting of profound inflammation and diagnostic uncertainty. This resulted in dramatic clinical improvement, with ferritin and LDH significantly down-trending. On day 5, testing was positive for Erlichia. The patient completed a Doxycycline course and steroid taper with continued improvement.

Case Discussion: The rarity of secondary HLH due to Erlichia makes incidence hard to quantify, with one single-center study reporting a rate of 8%. These cases typically respond well to prompt initiation of Doxycycline. This vignette depicts a patient clinically worsening on empiric antibiotics for tick-borne illness with labs concerning for profound hyperinflammatory response. In the absence of a confirmed infection and despite not meeting full HLH-2024 diagnostic criteria, this patient was started on high dose Dexamethasone, which resulted in rapid clinical improvement. This prompts discussion of the utility of early evaluation for HLH and consideration of steroids in patients presenting with concern for tick borne illness without initial response to Doxycycline.

Resident/Fellow Clinical Vignette

More Than Meets the Sicca: When Sjögren's Presents as Anemia

Anushree Venkatesh Murthy

Anushree Venkatesh Murthy, Adithya Nagendran, Hazel Lever, Naga Rama Krishna Tangirala

Introduction: Autoimmune hemolytic anemia (AIHA) can precede the classic sicca symptoms of Sjögren's syndrome by weeks to months, particularly in older adults where an initial negative ANA may be falsely reassuring. We describe a case in which Coombs-positive anemia — without dry eyes, dry mouth, or dental caries — was the sole presenting feature of primary Sjögren's syndrome, later confirmed by a serositis flare.

Case Presentation: A 79-year-old female with hypertension, hyperlipidemia, and hypothyroidism was noted by family to have fatigue, poor appetite, and generalized deconditioning beginning in September, prior to presenting with weakness, dyspnea, and calf pain. Labs revealed severe normocytic normochromic anemia (Hgb 6.9, baseline 11.0) with monocytosis and lymphopenia, requiring transfusion, along with an occlusive superficial vein thrombosis of the right saphenous vein. Direct Coombs testing was positive for IgG and C3d, with haptoglobin rising from 81 to 261, and reticulocyte count improving from 0.3 to 0.6 over the admission. An extensive evaluation — including CT imaging, SPEP, free light chains, quantitative immunoglobulins, remote EBV IgG (positive, indicating past exposure) with negative active viral studies (CMV, parvovirus, HIV), and ANA (negative twice) — effectively excluded multiple myeloma and occult malignancy. Bone marrow biopsy showed a hypercellular marrow with mild dysmegakaryopoiesis and grade 1 reticulin fibrosis without excess blasts or a clonal population on flow cytometry; while myelodysplastic syndrome remained a differential pending mutational analysis, it did not account for the acute anemia or markedly elevated CRP and ESR. Despite the lack of a clear alternate etiology, empiric prednisone was trialed and the anemia improved.

The patient was readmitted eleven days later with acute pleuritic chest pain and was found to have a pericardial effusion. Pulmonary embolism was excluded by CT angiography and ventilation-perfusion scan, and troponins remained negative. Anti-SSA antibody returned positive with an elevated rheumatoid factor, and she was diagnosed with pericarditis secondary to autoimmune serositis. Corticosteroids produced a dramatic clinical response. Rheumatology confirmed primary Sjögren's syndrome with serositis and autoimmune hemolytic anemia requiring recurrent transfusions, notably without any sicca symptoms throughout her presentation. She was discharged on a prednisone taper,

hydroxychloroquine, and sicca prophylaxis, with close outpatient rheumatology and hematology follow-up.

Discussion/Conclusion: This case demonstrates that autoimmune hemolytic anemia — occasionally accompanied by monocytosis and lymphopenia — can be the initial and, for a time, isolated manifestation of primary Sjögren's syndrome, occurring without sicca symptoms and despite a negative ANA. A reassuring marrow biopsy should not preclude an autoimmune workup in Coombs-positive anemia responsive to steroids without another identifiable cause. Pursuing anti-SSA/SSB testing in this setting can accelerate diagnosis and guide durable, steroid-sparing therapy.

Resident/Fellow Clinical Vignette

The Hidden Storm: Relapsing Hemophagocytic Lymphohistiocytosis—A Case Report

Anushree Venkatesh Murthy

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Background

Hemophagocytic lymphohistiocytosis (HLH) is a severe hyperinflammatory syndrome caused by uncontrolled immune activation and cytokine release that can lead to multiorgan failure and death if not promptly recognized. Adult HLH often presents with nonspecific symptoms such as fever, cytopenias, transaminitis, and organomegaly, which frequently overlap with sepsis, liver disease, and hematologic malignancies, delaying diagnosis. Although historically described in children, HLH is increasingly recognized in adults with diverse triggers, including infections, malignancies, and autoimmune conditions. Early recognition requires a high index of suspicion and the use of structured diagnostic tools such as the H-score. Identifying and treating the underlying trigger is equally important, as etiology-directed therapy is often necessary to achieve sustained remission.

Case Presentation:

A 37-year-old Hispanic male presented with 2–3 weeks of fever, chills, night sweats, nausea, vomiting, and fatigue. Laboratory studies revealed pancytopenia, elevated liver enzymes, ferritin >2,000 ng/mL, LDH 968 U/L, haptoglobin <1 g/L, and a soluble IL-2 receptor level of 2,043 U/mL. Computed tomography demonstrated marked splenomegaly measuring 20 cm with associated mesenteric edema. Infectious workup was largely negative, except for low-grade Epstein–Barr virus (EBV) viremia. Bone marrow biopsy showed hemophagocytosis without evidence of lymphoma. Genetic testing for HLH-associated mutations using a Mayo Clinic 23-gene panel was negative.

Management and Outcome:

HLH was diagnosed based on bone marrow findings and an H-score indicating 97–99% probability. High-dose dexamethasone (10–20 mg/m²) led to rapid clinical improvement. He received Pneumocystis jirovecii prophylaxis. Soon after tapering steroids, he experienced relapse associated with rising EBV titers. He was subsequently treated with

etoposide and four weekly doses of rituximab. At one-month follow-up, he reported marked improvement, stable functional status, and manageable steroid-related side effects.

Discussion:

Adult HLH often presents with vague systemic symptoms, delaying recognition. In this case, early use of bone marrow biopsy and the H-score supported prompt diagnosis and initiation of appropriate immunosuppressive therapy.

Conclusion:

HLH should be considered in adults presenting with fever, cytopenias, hyperferritinemia, and organomegaly. Management of HLH in adults remains challenging due to heterogeneous etiologies, overlapping presentations with sepsis and malignancy, and a lack of standardized adult-specific protocols. In this case, timely use of bone marrow biopsy and the H-score supported early diagnosis, while the patient's relapse and rising EBV titers underscored the dynamic nature of adult HLH and the need for etiology-directed therapy, including rituximab for EBV-driven disease.

Resident/Fellow Clinical Vignette

RULING OUT TO RULE IN: UNMASKING HAMMAN-RICH SYNDROME IN DIFFUSE ALVEOLAR DAMAGE

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Introduction

Acute interstitial pneumonia (Hamman-Rich syndrome) is a rare, fulminant form of idiopathic diffuse alveolar damage (DAD) mimicking ARDS of infectious or autoimmune origin, with reported mortality as high as 60-100%.

Case Presentation

A 71-year-old man with rheumatoid arthritis on methotrexate, chronic prednisone, and treated latent tuberculosis, presented with dyspnea, brown sputum, and pleuritic chest pain. Chest radiography one month prior had been unremarkable; CT on admission demonstrated multifocal ground-glass opacities. Methotrexate was held and empiric corticosteroids initiated for suspected methotrexate-induced or eosinophilic pneumonitis. However, the patient continued to decline. Respiratory viral PCR identified a non-COVID coronavirus, and remdesivir was administered empirically given the rapid deterioration. Given significant immunosuppression, Pneumocystis jirovecii pneumonia was considered and empiric trimethoprim-sulfamethoxazole initiated, however, a normal beta-D-glucan argued against this diagnosis. Three acid-fast bacilli smears/cultures and a QuantiFERON assay were negative, excluding tuberculosis reactivation. Blood and sputum cultures were sterile; fungitell and Aspergillus galactomannan assays were negative, excluding invasive fungal disease, and the remaining respiratory viral panel was unrevealing. Antineutrophil cytoplasmic antibody, double-stranded DNA, and a myositis panel including anti-MDA5 antibody were negative, arguing against vasculitis or connective-tissue-disease-associated interstitial lung disease despite the patient's underlying rheumatoid arthritis. A pulse-dose methylprednisolone 1000 mg daily for three days was started in view idopathic diffuse aleolar damage (DAD). Despite this, the patient progressed to refractory hypoxemia requiring 90-100% FiO2 on high-flow support. Serial CT over two weeks demonstrated diffuse evolution of scattered ground-glass opacities with traction bronchiectasis and destruction of alveolar architecture, findings characteristic of DAD. With every secondary etiology excluded or empirically treated and no response to maximal immunosuppression, idiopathic acute interstitial pneumonia was established as the diagnosis of exclusion, as

patient denied bronchoscopic intervention. The patient, who had elected do-not-intubate/do-not-resuscitate status, subsequently died of fulminant respiratory failure.

Discussion

This case illustrates the systematic exclusion required to establish a diagnosis of Hamman-Rich syndrome, encompassing infectious (coronavirus, PJP, tuberculosis, bacterial, fungal), drug-induced, and autoimmune (eosinophilic pneumonitis, connective-tissue-disease-associated interstitial lung disease) etiologies, each independently pursued and treated. This mirrors prior cohorts describing a similarly older population (mean age ~69 years) with radiographic progression from patchy to diffuse bilateral disease over a comparable one-to-two-week interval, biopsy-confirmed DAD, and near-universal progression to advanced respiratory support despite pulse-dose steroids; histopathologic findings are indistinguishable from ARDS but lack an identifiable trigger. The radiographic evolution observed here, from ground-glass opacities to consolidation with traction bronchiectasis and architectural distortion, parallels the organizing-to-fibrotic phase of DAD described in the literature and portends a poor prognosis. This case underscores the importance of a thorough, protocol-driven evaluation in undifferentiated ARDS among immunosuppressed rheumatoid arthritis patients, given the stakes of distinguishing idiopathic acute interstitial pneumonia from treatable mimics, even when maximal therapy fails to alter a fulminant course.

Resident/Fellow Clinical Vignette

Multi-system Calciphylaxis with Constrictive Pericarditis Responsive to Sodium Thiosulfate

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Introduction

Calciphylaxis is a rare, life-threatening syndrome of microvascular calcification predominantly affecting patients with end-stage renal disease (ESRD). While cutaneous manifestations are well described, visceral involvement — including cardiac — remains underrecognized. Sodium thiosulfate (STS) is used off-label for calciphylaxis, with anti calcification, antioxidant, and vasodilatory properties. We present a case of multisystem calciphylaxis with suspected penial calciphylaxis and effusive-constrictive pericarditis that significantly improved following STS therapy.

Case Presentation

A 43-year-old man with ESRD on hemodialysis, insulin-dependent diabetes, and prior above-knee amputation presented with severe groin pain, jaundice, penile ulceration. Laboratory evaluation revealed mixed hyperbilirubinemia (total bilirubin 12.9 mg/dL, direct 8.4 mg/dL), cholestatic liver enzyme elevation, markedly elevated alkaline phosphatase (450–630 U/L), progressive hyperphosphatemia (5.0–9.0 mg/dL), and normocalcemia (7.3–9.2 mg/dL). Remarkable findings on Computed tomography of the abdomen and pelvis were extensively calcified peripheral aortic branch vessels, calcified vas deferens, and cardiomegaly with mild pulmonary vascular congestion. The echocardiography done revealed a moderate pericardial effusion with constrictive physiology, eggshell pericardial calcification, and severe diastolic dysfunction. Broad infectious work up done came negative, he was empirically treated for genital herpes but had no improvement with therapy. Liver biopsy done showed marked periportal ductular reaction with Biopsy done for penile ulcer demonstrated fibrous inflammation without malignancy. Pain management was challenging. A deep punch biopsy of penile ulcer was considered but later deferred due to the anatomic location of ulcer and chances of poor healing. A presumptive diagnosis of systemic calciphylaxis was made based on constellation of ESRD, diffuse vascular calcification on imaging, penile ulceration with fibrous inflammation on biopsy, pericardial calcification with effusive-constrictive physiology, intrahepatic cholestasis likely related to microvascular compromise, severe pain out of proportion to exam findings and markedly elevated alkaline phosphatase. Treatment included intravenous STS with

alternate-day hemodialysis. Although colchicine was recommended for pericarditis, the patient did not take it. On follow up repeat echocardiography two months later demonstrated significant improvement: decrease in pericardial effusion from moderate to trivial, diastolic dysfunction improved from severe to moderate and marked improvement in constrictive physiology. His groin pain also responded very well though the penile ulcers were the same.

Discussion

This case illustrates multisystem calciphylaxis involving cutaneous, cardiac, and hepatic manifestations. The improvement in effusive-constrictive pericarditis with eggshell calcification and pain symptoms following STS initiation — without concurrent anti-inflammatory therapy — is notable, as eggshell calcification suggests chronic rather than transient constriction. To our knowledge, this is the first reported case of effusive-constrictive pericarditis improving with STS therapy in calciphylaxis, suggesting STS may have beneficial cardiac effects beyond its established cutaneous applications.

Resident/Fellow Clinical Vignette

IMMUNE HEMOLYTIC ANEMIA FOLLOWING RIFAMPIN RE-EXPOSURE: WHEN THE DRUG IS THE DIAGNOSIS

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Introduction

Drug-induced immune hemolytic anemia (DIIHA) is rare but potentially life-threatening. Rifampin-associated reactions are particularly described after interrupted, intermittent, or restarted exposure, when pre-existing drug-dependent antibodies may trigger abrupt complement-mediated intravascular hemolysis. The resulting biochemical constellation — extreme hyperferritinemia, thrombocytopenia, and hypofibrinogenemia — may create diagnostic overlap with hemophagocytic lymphohistiocytosis (HLH), a high-stakes pitfall where misidentification risks inappropriate cytotoxic immunosuppression.

Case Description

A 67-year-old Filipino woman with latent tuberculosis infection (LTBI) was prescribed rifampin 400 mg daily. After three months of inconsistent use she discontinued the medication; after an approximately five-month drug-free interval, she accidentally re-ingested two tablets and presented within hours with nausea, vomiting, and abdominal pain. Hemoglobin declined from 15.4 to 11.8 g/dL within 24 hours, accompanied by indirect hyperbilirubinemia (total bilirubin 7.8 mg/dL), lactate dehydrogenase 1,312 U/L, and reticulocytosis — consistent with acute intravascular hemolysis. Thrombocytopenia (platelets 113 x 10^9/L), coagulopathy (INR 1.98, fibrinogen 69 mg/dL), and markedly elevated ferritin exceeding 33,511 ng/mL (laboratory upper limit 150 ng/mL) were identified.

The direct antiglobulin test (DAT) was positive for broad spectrum and C3, with negative IgG. Alkaline phosphatase was mildly elevated; aspartate aminotransferase and glucose-6-phosphate dehydrogenase activity were normal. Comprehensive infectious workup was negative.

HLH was considered given the extreme ferritin elevation, thrombocytopenia, and hypofibrinogenemia. The HScore was 143 — corresponding to an estimated HLH probability of 16–25%, prompting hematology evaluation. No fever or organomegaly was present, and bone marrow biopsy was deferred. Rifampin was discontinued and supportive

care provided. All laboratory abnormalities normalized within five days without HLH-directed therapy.

Discussion

This case illustrates the classic pattern of probable rifampin re-exposure DIIHA: abrupt onset within hours of reintroduction following a five-month drug-free interval, complement-mediated hemolysis with a C3-positive DAT, and complete biochemical recovery after drug withdrawal — together strongly favoring the diagnosis in the absence of formal drug-dependent antibody testing. The C3-positive, IgG-negative pattern reflects complement cascade activation by drug-dependent antibodies — mechanistically distinct from warm autoimmune hemolytic anemia in which IgG red cell coating predominates. Extreme ferritin elevation, while alarming, reflects acute inflammatory injury and should not be interpreted as pathognomonic for HLH. An HScore of 143 appropriately prompted HLH evaluation; however, absence of fever, organomegaly, and progressive bicytopenia — combined with the drug re-exposure timeline and rapid clinical improvement after drug withdrawal — favored DIIHA.

Conclusion

As four-month rifampin regimens expand for LTBI treatment, clinicians must recognize DIIHA as a serious re-exposure complication. A C3-positive, IgG-negative DAT in the setting of rifampin re-exposure and acute hemolysis is the serologic key to diagnosis. Drug discontinuation is definitive therapy — immunosuppression is not. Rifampin is permanently contraindicated, the allergy must be documented, and drug-dependent antibody testing pursued when available.

Resident/Fellow Clinical Vignette

Bullous Pemphigoid Presenting with Multiple Esophageal Strictures in Hemorrhagic Shock Patient

Bi Zhang

Nina Kulikovska, Rian Goding, Justin Poon, Larisa Garkusha, Antonio Costantino, Kyle Katona, Mary Thomas

Instruction

Bullous pemphigoid (BP) is a rare autoimmune skin disorder that primarily affects elderly between 60 and 80 years old. It is characterized by autoantibodies against hemidesmosomes at the basement membrane of the dermo-epidermal junction. Clinically, it presents with tense bullae and intense pruritus, although nonbullous presentations may occur. While BP primarily affects the skin, mucous membrane involvement is reported in 10-35% of cases, whereas esophageal involvement occurring in only approximately 4% of patients. Clinical manifestations range from asymptomatic to dysphagia, epigastric pain, hematemesis, and melena. We report an exceptionally rare case of multiple esophageal strictures in BP, incidentally discovered during emergent endoscopy for hemorrhagic shock, highlighting the diagnostic and technical challenges of endoscopic management.

Case presentation

An 80-year-old woman with a history of hypertension, hyperlipidemia, heart failure preserved ejection fraction (HFpEF), iron deficiency anemia, and severe bullous pemphigoid with diffuse cutaneous involvement presented with altered mental status and hypotension. She was admitted for septic shock secondary to stercoral colitis versus extensive skin/wound infection. The hospital course was complicated by acute kidney injury, hypernatremia, metabolic encephalopathy, and hypotension requiring vasopressor support. She subsequently developed hematemesis with hemorrhagic shock secondary to upper gastrointestinal bleeding (GIB), requiring endotracheal intubation, vasopressors, massive transfusion, and emergent EGD. EGD revealed multiple severe esophageal strictures involving the proximal, mid, and distal esophagus and GE junction, making scope advancement technically challenging. Careful balloon dilation was successfully performed. Two bullae were incidentally identified, confirming mucosal involvement by BP. Meanwhile, there were numerous gastric ulcers at varying stages of healing throughout the stomach with evidence of oozing. Endoclips and polypeptide hemostatic gels were applied to achieve adequate hemostasis. Following stabilization, the patient was successfully

extubated and transferred to the medicine floor, with dermatology and rheumatology consulted for long-term esophageal BP management.

Discussion

Esophageal involvement in BP is rare, and esophageal stricture is an exceptionally uncommon complication because, unlike mucous membrane pemphigoid (MMP), BP rarely results in significant scarring. Proposed mechanisms include extensive mucosal sloughing or erosions, severe localized infection extending into the submucosa with subsequent excessive inflammation and granulation tissue formation, preexisting gastroesophageal reflux disease, and the possibility of BP evolving into MMP. In our case, emergent EGD was technically challenging because of severe mucosal fragility, bullae formation, and multiple esophageal strictures during treatment of life-threatening GIB. Gentle endoscopic technique is essential to minimize iatrogenic mucosal injury. The diagnosis is typically established by histopathology and direct immunofluorescence. Systemic corticosteroids remain the first-line treatment, while steroid-sparing immunosuppressive agents such as azathioprine and rituximab may be used in refractory or severe disease. This case highlights early recognition of esophageal BP to prevent irreversible stricture formation and the critical role of meticulous endoscopic technique in safely managing these high-risk patients.

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CARDIOVASCULAR OUTCOMES WITH METOPROLOL VERSUS CARVEDILOL IN TRANSTHYRETIN CARDIAC AMYLOIDOSIS AND ATRIAL FIBRILLATION/FLUTTER

Nathaniel Herman

Sourish Rathi DO; Shivani Rana, DO; Alex Kaiteris, DO

PURPOSE: Transthyretin cardiac amyloidosis is frequently complicated by atrial fibrillation/flutter and need for rate control, yet comparative cardiovascular outcomes among patients treated with metoprolol versus carvedilol remain poorly defined.

METHODS: We performed a retrospective cohort study using the TriNetX Research Network. Adults with transthyretin cardiac amyloidosis and atrial fibrillation/flutter were stratified by initiation of metoprolol or carvedilol. Patients with prior exposure to the alternate beta-blocker were excluded, and beta-blocker initiation was defined as the index event. Follow-up extended up to 15 years. Propensity score matching was used to balance baseline demographics, cardiovascular and noncardiovascular comorbidities, concomitant cardiovascular medications, and prior cardiac interventions. Patients with prior occurrence of each studied outcome were excluded from the corresponding outcome analysis. Outcomes included cerebral infarction/transient ischemic attack, acute systolic or diastolic heart failure exacerbation, ventricular tachycardia, acute myocardial infarction, cardiogenic shock, and cardiac arrest. Risk ratios and Kaplan-Meier hazard ratios were calculated.

RESULTS: After matching, 943 patients were included in each cohort. Compared with carvedilol, metoprolol was associated with higher risk of acute systolic or diastolic heart failure exacerbation (21.6% vs 15.3%; risk ratio 1.41, 95% confidence interval 1.15–1.73; hazard ratio 1.32, 95% confidence interval 1.05–1.65; p=0.0008) and acute myocardial infarction (23.6% vs 18.0%; risk ratio 1.31, 95% confidence interval 1.09–1.58; hazard ratio 1.33, 95% confidence interval 1.07–1.66; p=0.0045). Ventricular tachycardia was more frequent with metoprolol by risk-based analysis (23.6% vs 16.6%; risk ratio 1.42, 95% confidence interval 1.17–1.73; p=0.0004), although time-to-event analysis did not reach statistical significance (hazard ratio 1.20, 95% confidence interval 0.98–1.49). No significant differences were observed in cerebral infarction/transient ischemic attack, cardiogenic shock, or cardiac arrest.

CONCLUSION: In this propensity-matched analysis, metoprolol was associated with higher long-term risk of acute heart failure exacerbation and acute myocardial infarction

compared with carvedilol among patients with transthyretin cardiac amyloidosis and atrial fibrillation/flutter. These findings suggest beta-blocker selection may be clinically relevant in this high-risk population and support prospective evaluation of comparative safety.

Resident/Fellow Research

CEREBROVASCULAR RISK IN CARDIAC AMYLOIDOSIS WITHOUT ATRIAL FIBRILLATION

Nathaniel Herman

Sourish Rathi DO; Shivani Rana, DO; Alex Kaiteris, DO

PURPOSE: Cardiac amyloidosis is associated with thromboembolic complications, particularly in the presence of atrial fibrillation, but whether it independently increases cerebrovascular risk in patients without atrial fibrillation remains uncertain.

METHODS: We performed a retrospective cohort study using the TriNetX Research Network. Adults aged 18 years or older with cardiac amyloidosis and no history of atrial fibrillation who had at least one inpatient encounter were compared with adults without cardiac amyloidosis or atrial fibrillation who had at least one inpatient encounter. Patients with prior transient ischemic attack or ischemic stroke were excluded. Propensity score matching was performed for age, hypertension, heart failure, diabetes mellitus, chronic kidney disease, and smoking history. The primary outcome was occurrence of transient ischemic attack or cerebral infarction identified using International Classification of Diseases diagnostic codes. Risk ratios, absolute risk differences, 95% confidence intervals, and p-values were calculated.

RESULTS: After propensity score matching, 16,683 patients with cardiac amyloidosis and 17,057 controls were included. Transient ischemic attack or cerebral infarction occurred in 960 patients with cardiac amyloidosis and 926 controls, corresponding to risks of 5.754% and 5.429%, respectively. The risk ratio was 1.06, with a 95% confidence interval of 0.971–1.157. The absolute risk difference was 0.326%, with a 95% confidence interval of −0.165% to 0.816%. There was no statistically significant difference in transient ischemic attack or cerebral infarction between cohorts (p=0.1932).

CONCLUSION: In this large propensity-matched analysis, cardiac amyloidosis was not independently associated with significantly increased risk of transient ischemic attack or ischemic stroke among patients without atrial fibrillation. Although event rates were numerically higher in the cardiac amyloidosis cohort, the difference was small and not statistically significant. These findings suggest that cardiac amyloidosis alone may not confer substantial cerebrovascular risk in the absence of atrial fibrillation and support further study to refine stroke risk stratification in this population.

Resident/Fellow Research

Association Between Clonal Hematopoiesis and Lung Cancer in Adults: A Systematic Review and Meta-Analysis

Umair Khan

Mark Batistick, MD, Amna Sher, MD

Introduction:

Clonal hematopoiesis of indeterminate potential (CHIP) is characterized by the expansion of hematopoietic clones harboring somatic mutations and has been associated with aging, inflammation, and tumor biology. Emerging data suggests a role in CHIP in lung carcinogenesis, however much of the data has come from limited study designs or single-centered institutes. We performed an updated meta-analysis to evaluate the association between CHIP and the incidence of lung cancer including the first with sub-groups analysis by variant allele frequency (VAF) and mutational subtypes.

Methods:

A systematic review of PubMed, Cochrane Central, Embase, and Web of Science was performed on all studies prior to April 6, 2026 for keywords related to CHIP and lung cancer. Observational studies with data that examined the association between the two were included. Studies that included pediatric populations, case reports, reviews, or conference abstracts without extractable data, or studies with data with the same cohort were excluded. When overlapping datasets existed, the most recent or most informative publication was preferred for the main analysis.

Results:

In total, 7992 articles were reviewed, and 5 were included in the meta-analysis. Analysis demonstrated a significant association for individuals with CHIP developing lung cancer (OR: 1.28 [95% CI, 1.17-1.39], $p < .05$) with a stronger association seen in individuals with high-VAF (OR: 1.48 [95% CI, 1.25-1.77], $p < 0.05$). In the mutational subtype analysis, an association was seen with ASXL1 and DNMT3A (OR: 1.72 [95% CI, 1.25-2.39], $p < 0.05$ and OR: 1.39 [95% CI, 1.22-1.59], $p < 0.05$) but not TET2 (OR: 1.16 [95% CI, 0.93-1.45], $p = 0.18$).

Discussion:

CHIP is associated with a statistically significant increase in lung cancer risk with a stronger association seen with high variant allele frequency. Notably, this is also the first meta-analysis to demonstrate a relationship that exists between CHIP mutational subtypes and lung cancer implicating ASXL1 and DNMT3A as potential drivers, although more data is likely needed. These findings overall support clonal hematopoiesis as a potentially meaningful biomarker of lung cancer susceptibility and reinforce a biologically plausible link between hematopoietic clonal expansion and lung carcinogenesis. Future work should be done to explore the mechanistic validation as well as to evaluate whether clonal hematopoiesis can improve lung cancer risk stratification beyond established clinical factors.

Resident/Fellow Research

Beyond Respiratory Disease: Cardiovascular Manifestations of Respiratory Syncytial Virus Compared with Influenza in Older Adults.

Sumitra Paudel

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Background: Respiratory syncytial virus (RSV) is an increasingly recognized cause of hospitalization among older adults and has been associated with adverse cardiovascular events. Following the availability of RSV vaccines for adults aged 60 years and older, understanding the cardiovascular manifestations associated with RSV infection has become increasingly important. We compared cardiovascular outcomes among adults aged 60 years or older hospitalized with RSV versus influenza using a nationally representative inpatient database.

Methods: We performed a retrospective cross-sectional analysis of the 2023 Healthcare Cost and Utilization Project National Inpatient Sample. Hospitalizations among adults aged 60 years or older with RSV or influenza listed in any diagnosis field were identified using ICD-10-CM codes and assigned to mutually exclusive groups. Hospitalizations were matched 1:1 using nearest-neighbor propensity score matching without replacement (caliper 0.01) based on age, sex, median household income quartile, and Elixhauser Comorbidity Index. The matched cohort included 20,342 hospitalizations, representing an estimated 101,710 weighted hospitalizations nationally. Survey weights were reapplied after matching, and survey-weighted multivariable logistic and linear regression accounting for the complex sampling design evaluated acute heart failure (HF), atrial fibrillation/flutter (AF), acute myocardial infarction (AMI), in-hospital mortality, and hospital length of stay.

Results: The matched cohort represented 50,855 RSV and 50,855 influenza hospitalizations. Compared with influenza hospitalizations, RSV hospitalizations had a higher prevalence of acute HF (41.6% vs. 37.6%) and AF (35.5% vs. 32.8%), whereas AMI occurred less frequently (7.9% vs. 9.9%). RSV hospitalizations were associated with higher odds of acute HF (adjusted odds ratio [aOR] 1.25, 95% confidence interval [CI] 1.17 to 1.33; $p<0.001$) and AF (aOR 1.14, 95% CI 1.07 to 1.22; $p<0.001$), while lower odds of AMI were

observed (aOR 0.77, 95% CI 0.70 to 0.86; p<0.001). No significant differences were observed in in-hospital mortality (aOR 0.97, 95% CI 0.84 to 1.11; p=0.635) or hospital length of stay (adjusted β = -0.08 days; p=0.557).

Conclusions: Among hospitalizations of older adults with RSV or influenza, RSV was associated with higher odds of acute HF and AF, whereas influenza was associated with higher odds of AMI. No significant differences were observed in in-hospital mortality or hospital length of stay. Although limited by the retrospective cross-sectional design and use of an administrative database, these findings suggest that RSV and influenza are associated with distinct cardiovascular manifestations and provide context for future studies evaluating the cardiovascular impact of RSV vaccination in older adults.

Resident/Fellow Research

PERCEPTIONS, BARRIERS, AND IMPLEMENTATION NEEDS FOR WEARABLE TECHNOLOGY AMONG HEALTHCARE WORKERS AT AN URBAN ACADEMIC MEDICAL CENTER

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Introduction:

Wearable health technologies, including smartwatches, continuous physiologic monitors, and ambulatory biosensors, are increasingly used to generate real-time health data with potential clinical value. However, their integration into routine healthcare delivery remains variable across clinical settings and institutions.

Methods:

We conducted a cross-sectional survey of employees at a large urban academic medical center between December 2025 and January 2026. The survey assessed professional role, healthcare experience, familiarity with wearables and personal use, current departmental use, perceived effectiveness, prior training, confidence interpreting device-generated data, support for institutional expansion, anticipated benefits, implementation concerns, and desired resources. Responses were summarized using frequencies and proportions. For multiple-response questions, percentages were calculated based on the number of respondents completing each item. Descriptive statistics summarized responses; chi-square tests compared attitudes across clinical roles (physician, nurse, allied health) and personal-use strata (regular, occasional, never). Significance was defined as $p<0.05$.

Results:

A total of 411 employees participated, including 83 physicians (20.2%), 69 nurses (16.8%), 47 allied health professionals (11.4%), 102 administrative staff (24.8%), 19 information technology staff (4.6%), and 91 respondents in other roles (22.1%). Most participants (60.3%) had more than 10 years of healthcare experience. Of 411 respondents, 365 (88.8%) were familiar and 33 (8.0%) were somewhat familiar with wearable health technologies. Among 409 respondents, 191 (46.7%) used a wearable regularly and 106 (25.9%) used one occasionally. Despite high personal adoption, only 59 of 410 respondents (14.4%) reported current departmental use of wearables for patient care; 246 (60.0%) reported no use and 105 (25.6%) were unsure. Wearables were considered very or somewhat effective in improving patient outcomes by 293 of 399 respondents (73.4%), while 253 of 404 (62.6%)

believed they improved clinical workflow and decision-making. Expanded institutional use was supported or strongly supported by 297 of 404 respondents (73.5%); however, only 40 of 404 (9.9%) reported receiving training or guidance on incorporating wearables into clinical workflows. Among respondents completing the multiple-response items, the most frequently perceived benefits were continuous monitoring (72.1%), early detection of clinical issues (71.0%), and data-driven care (52.1%). Major concerns included device reliability (58.6%), patient adherence (51.9%), data privacy and security (46.8%), and electronic medical record integration (43.0%). The most requested resources were training and education (80.2%), clear protocols and policies (55.3%), improved electronic health record integration (53.7%), and information technology support (53.2%).

Conclusion: The healthcare workforce demonstrated high familiarity, personal adoption, perceived clinical value, and support for wearable technologies, but limited departmental implementation and minimal workflow training. This enthusiasm-implementation gap suggests that institutions should prioritize structured education, governance, interoperability, technical support, and clearly defined clinical workflows before broad deployment.

Resident/Fellow Research

Impact of Intradialytic Exercise on Cardiovascular Markers in Adult Hemodialysis Patients: A Meta-Analysis

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Introduction

Cardiovascular disease is the leading cause of morbidity and mortality in patients undergoing hemodialysis. Intradialytic exercise has been proposed as a strategy to improve cardiovascular health; however, its effects on cardiac markers remain uncertain. This meta-analysis evaluated the effects of intradialytic exercise compared with no exercise on cardiac markers in adult hemodialysis patients.

Method

We conducted a systematic review and meta-analysis in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines. PubMed, Google Scholar, Scopus, Web of Science, and Embase were searched from inception to December 2025 to identify randomized controlled trials (RCTs) evaluating the effects of intradialytic exercise compared with no exercise/control among hemodialysis patients. Studies reporting cardiovascular outcomes, including pulse rate (bpm), systolic blood pressure (SBP), diastolic blood pressure (DBP), VO_2max (mL/kg/min), carotid-femoral pulse wave velocity (cfPWV; m/s), and ejection fraction, were included. Data were pooled using random-effects models to account for between-study variability. Continuous outcomes were analyzed as mean differences (MDs) using the generic inverse variance method. Statistical heterogeneity was assessed using the I² statistic.

Result

A total of 16 randomized controlled trials were included in the meta-analysis. Compared with standard of care, intradialytic exercise significantly reduced systolic blood pressure (MD: −9.05 mmHg; 95% CI: −14.70 to −3.34; p=0.002) and diastolic blood pressure (MD: −3.06 mmHg; 95% CI: −5.44 to −0.68; p=0.01). No significant difference was observed in pulse rate (MD: −0.13 bpm; 95% CI: −2.96 to 2.70; p=0.93). Similarly, intradialytic exercise did not significantly improve ejection fraction (MD: 0.86%; p=0.64), carotid-femoral pulse wave velocity (cfPWV) (MD: −0.71 m/s; p=0.14), or VO_2max (MD: 2.88 mL/kg/min; p=0.15) compared with standard care.

Conclusion

Intradialytic exercise appears to provide beneficial effects on blood pressure control in hemodialysis patients, significantly reducing both systolic and diastolic blood pressure compared with standard care. However, no significant improvements were observed in pulse rate, ejection fraction, cfPWV, or VO_2max. Further high-quality randomized controlled trials with standardized exercise protocols and longer follow-up periods are warranted to clarify the long-term cardiovascular benefits of intradialytic exercise.

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Resident/Fellow/Medical Student Quality, Patient Safety and Advocacy

HUMAN-CENTERED CARE IN THE AGE OF ARTIFICAL INTELLIGENCE: EXPANDING PALLIATIVE CARE ACCESS THROUGH POLICY REFORM

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Background

Artificial Intelligence (AI) is rapidly expanding to improve efficiency in healthcare, but its role in palliative care must be approached with caution. Palliative care serves vulnerable patients with serious illnesses and is dependent on nuanced human-centered communication, emotional intelligence, longitudinal trust-building, cultural sensitivity, and complex goals-of-care discussions that cannot be adequately replicated by algorithmic systems.

Early palliative care has been shown to improve quality of life while reducing healthcare utilization and costs, with a 25% cost reduction per patient and savings exceeding $4,600 when palliative consultation occurs more than four weeks before death (1, 2). Although an estimated 13.7 million Americans could benefit from palliative care services, access remains disproportionate in medically underserved communities. Inpatient palliative care services are available in about 96% of large hospitals but only in 34% of rural and 49% of for-profit hospitals (3). Adding to this disparity is the projected shortage of up to 18,000 palliative care physicians by 2030 (4).

AI is being used to address these healthcare gaps, like at Mayo Clinic, where an AI-driven “control tower” system is being tested to identify patients who may benefit from palliative consultation. However, clinicians note that such systems cannot adequately capture patient values or emotional complexity, reinforcing the need for clinician oversight (5). A scoping review found that while AI may improve symptom management and prognostic accuracy, implementation remains constrained by bias, privacy concerns, workflow challenges, and depersonalization risks (6). Studies evaluating AI chatbots found high accuracy in defining palliative terminology but poor credibility and readability, underscoring the need for clinician-guided communication in serious illness care (7).

Impact of Efforts

Recent federal initiatives, including the Palliative Care and Hospice Education and Training Act (PCHETA) and the Expanding Access to Palliative Care Act (SB2489), aim to reduce disparities through workforce expansion, clinician training, and improved access to services (8,9). However, a 2025 *JAMA Health Forum* study found that although 819 palliative care-related bills

were introduced between 2009 and 2023, comparatively few addressed workforce development and technological infrastructure needed to improve equitable access (10).

Simultaneously, rapid AI expansion has outpaced regulatory oversight, heightening concerns about inequitable implementation. Since early 2026, 43 states have introduced more than 240 AI-related healthcare bills following Executive Order 14365, which revoked a proposed federal moratorium on state AI regulation and emphasized minimally restrictive national framework (11,12). Without safeguards ensuring clinician oversight, transparency, and equitable deployment, AI-driven care models risk widening disparities by substituting technology for access to trained palliative specialists. Emerging policy efforts, including the New York Artificial Intelligence Ethics Commission Act, represent early steps toward ethical AI governance (13). Strengthening federal and state palliative initiatives remains essential to ensuring AI augments rather than compromises the human-centered foundation of palliative care.

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Resident/Fellow/Medical Student Quality, Patient Safety and Advocacy

Mandated Ratios, Missing Resources: Aligning Nursing Home Staffing Policy with Medicaid Reality in New York

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Background

Nursing homes disproportionately care for residents insured by Medicaid, including individuals with advanced chronic illness, dementia, and significant functional impairment. In 2022, New York implemented minimum staffing standards under the New York State Nursing Home Staffing Law, requiring facilities to meet defined hours per resident [1,2]. The resources allocated to nursing homes have not kept pace with the care demands of their residents. Workforce shortages have been compounded by inadequate Medicaid reimbursement that often fail to reflect resident acuity and complexity [2]. Understaffing is a driver of racial and socioeconomic disparities in New York nursing homes.

Challenges, Policy Making and Advocacy

Limited infrastructure to recruit and retain staff, coupled with post-pandemic burnout, has further strained the system. Inadequate Medicaid reimbursement hinders the opportunity for nursing homes to provide competitive wages to recruit and retain essential staff [3]. Understaffing is not merely a workforce issue—it is a structural driver of inequity. This jeopardizes the infrastructure required to provide adequate care for older adults. Facilities serving predominantly Medicaid-insured and minority residents are more likely to experience lower staffing levels and fewer financial reserves, reinforcing disparities in care quality[4].

Meaningful reform requires aligning staffing mandates with sustainable Medicaid investment. Policymakers should prioritize acuity-adjusted reimbursement models, direct funding for workforce recruitment and retention, and safeguards to protect safety-net facilities. Without structural financing reform, staffing mandates risk widening current disparities by penalizing under-resourced nursing homes rather than strengthening them. Addressing nursing home staffing is therefore not solely a regulatory obligation—it is a patient safety and health equity priority. Studies have shown that incentivizing appropriate levels of staffing can improve patient outcomes[5]. Lower staffing levels are linked to higher

rates of avoidable emergency department transfers and rehospitalizations, increasing overall healthcare expenditures[6]. These harms disproportionately affect Black and low-income residents, who are more likely to reside in under-resourced, Medicaid-dependent facilities. Higher staffing levels could reduce tens of thousands of annual hospital and ED visits, resulting in significant Medicare savings, approximately $187milion to $465 million[7].

Future Directions

Addressing nursing home staffing shortages in New York will require a coordinated strategy that expands workforce pipelines through scholarships, bonuses, and retention initiatives[8.] Investments made into the nursing home infrastructure have downstream implications such as improvement in quality of care and cost saving for health systems. Nursing homes are operating in deficits, ensuring adequate financial support would help facilities meet staffing mandates. Aligning workforce development with sustainable Medicaid investment is essential to strengthening staffing capacity, protecting resident safety, and providing equitable care.

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Resident/Fellow/Medical Student Quality, Patient Safety and Advocacy

MEDICATION RECONCILIATION MADE RIGHT: A QUALITY IMPROVEMENT INITIATIVE TO REDUCE MEDICATION ERRORS AT HOSPITAL DISCHARGE

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Accurate medication reconciliation plays a crucial role in patient outcomes and safety, yet studies show up to 60% of admitted patients have a medication reconciliation error, which can lead to delays in discharge and adverse health outcomes. Pharmacist involvement at various points of inpatient admissions has been shown to reduce medication errors. Therefore, our SMART Aim is to reduce the number of serious errors on discharge medication reconciliation by 20% over six months following the integration of pharmacists on hospital medicine resident teams at a large university-based hospital system.

Pharmacists were integrated into inpatient resident-led hospital medicine teams by assigning two pharmacists to two resident teams each (intervention group). Pharmacists alternated rounding with each team, consisting of an attending, senior resident, and two interns with up to 16 patients. Two teams (non- intervention group) did not have a pharmacist on rounds but one was available for questions. Pre/post surveys were distributed to providers to assess perceptions/attitudes surrounding medication reconciliation, communication, and efficiency in the discharge process. Pre/post intervention data on discharge medication errors was collected and categorized by error type and severity as adapted from the National Coordinating Council for Medication Error Reporting and Prevention by the pharmacists over two 10-week periods, July-September 2025 (pre-intervention) and January-March 2026 (post-intervention). As a balancing measure, nurses on two resident-led med-surg units were surveyed regarding perceptions/concerns with this intervention. Additionally, length of stay (LOS) was tracked and compared between intervention and non-intervention groups.

Pre-intervention, there were 174 total medication-related errors over a 10-week period, 55 of which were classified as serious. Post-intervention data showed a reduction in total errors to 99 (43% reduction) and serious errors to 40 (27% reduction). Pre/post survey responses revealed largely positive perceptions on the initiative. No clear difference in LOS was appreciated between intervention and non-intervention groups. Nursing survey results

identified a balancing measure, with 33% (24 total responses) reporting they agreed/strongly agreed that medication-related safety concerns increased on their unit without a unit-based pharmacist.

Overall, results have been encouraging, demonstrating a reduction in the number of both total and serious errors. Limitations include sharing pharmacists amongst teams, independent data classification by pharmacists, and reliance on pharmacist presence for data collection. While these could lead to slight variations in the data or possible missed data points, these were held constant pre/post intervention, therefore allowing for meaningful interpretation/comparison of data. Post-intervention, serious errors continue to account for a notable proportion of total errors. Therefore, future directions include further analysis of serious errors to determine additional interventions to target these. Lastly, we will continue collecting data on patient LOS to track improvement, monitor for effect on 30-day re-admission rates, and explore change implementation for nursing safety/workflow concerns.

Resident/Fellow/Medical Student Quality, Patient Safety and Advocacy

EVALUATION OF A BLOOD CULTURE DECISION ALGORITHM IN CRITICALLY ILL ADULTS

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Purpose:

Blood cultures are overused in ICUs despite a bacteremia prevalence of only 6–7%. Excessive ordering leads to false-positive results, unnecessary antibiotic use, increased resource utilization, and prolonged hospitalization. Clinical decision algorithms may optimize blood culture utilization while maintaining diagnostic accuracy. Our goal was to evaluate the diagnostic accuracy of a clinical decision algorithm for bacteremia in critically ill adults.

Methods:

This retrospective observational study evaluated a blood culture algorithm as a clinical decision support tool for detecting bacteremia in hospitalized adult ICU patients between August 1 and October 31, 2025. Culture episodes were classified as appropriate or inappropriate using an algorithm that incorporates clinical presentation, pretest probability of bacteremia, and risk factors to determine the need for blood cultures. Data were obtained through standardized chart review of clinical documentation, vital signs, and laboratory values by four internal medicine resident reviewers applying predefined criteria. The primary outcome was diagnostic accuracy (sensitivity, specificity, PPV, NPV); secondary outcomes included potential reduction in utilization and bacteremia yield. Estimates with 95% confidence intervals were calculated using the Clopper–Pearson exact method.

Results:

A total of 133 blood culture episodes were analyzed (102 initial, 31 follow-up). The algorithm classified 76 (57.1%) as appropriate and 57 (42.9%) as inappropriate. Isolated leukocytosis was the most common clinical finding in both groups (52.6% vs 36.8%),

followed by combined fever and leukocytosis (18.4% vs 33.3%) and isolated fever (10.5% vs 15.8%). All inappropriate cultures were negative for bacteremia. The algorithm demonstrated 100% sensitivity (95% CI: 69.2%–100%), 46.3% specificity (95% CI: 37.3%–55.6%), 13.2% PPV (95% CI: 6.5%–22.9%), and 100% NPV (95% CI: 93.7%–100%). No bacteremia cases were missed among algorithm-discouraged cultures. Overall bacteremia yield was 7.5%, increasing to 13.2% among algorithm-recommended cultures.

Conclusion:

These findings demonstrate that a blood culture stewardship algorithm can safely reduce unnecessary blood culture utilization without missed bacteremia in ICU patients. Prospective implementation may enhance diagnostic stewardship and reduce low-value testing in critical care settings. The algorithm has since been implemented in the ICU at Vassar, with post-implementation analysis currently underway.

Resident/Fellow/Medical Student Quality, Patient Safety and Advocacy

Beyond Access: Addressing Retention in Specialty Care for Systemic Lupus Erythematosus in New York City

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Summary:

Summary:

Systemic lupus erythematosus (SLE) is a chronic, multisystem autoimmune disease requiring sustained longitudinal management. However, retention in specialty care remains a critical gap in health system delivery. Inadequate continuity of care contributes to preventable disease flares, increased acute care utilization, and long-term morbidity, particularly in urban, socioeconomically diverse settings such as New York City.

Population based estimates from the Manhattan Lupus Surveillance Program provide a validated benchmark for disease burden, demonstrating an age standardized prevalence of approximately 62.2 per 100,000 persons and an incidence of 4.6 per 100,000 person years, with significant racial, ethnic, and sex based disparities (1). While this data establishes overall disease burden, it does not capture borough level variation in continuity of care or retention in rheumatology services across New York City.

Health services research demonstrates that patients with SLE frequently experience gaps in follow-up care and recommended laboratory monitoring even when insured, indicating that insurance coverage alone is insufficient to ensure sustained engagement in care (2, 3). These disruptions in continuity are associated with worse disease control and increased acute care utilization, underscoring the clinical consequences of inadequate longitudinal management. These gaps in retention reflect structural barriers that disproportionately affect medically underserved populations. In boroughs such as the Bronx, higher poverty rates and greater Medicaid reliance intersect with reduced access to subspecialty rheumatology care, limiting consistent follow-up and disease monitoring. Concurrently, the uneven geographic distribution of rheumatology providers across New York City further concentrates specialty care in Manhattan, reinforcing disparities in longitudinal disease management (4).

Taken together, these findings identify a critical gap in New York State health policy: existing frameworks emphasize insurance coverage and general access to care but lack targeted

mechanisms to ensure retention in specialty care and continuity of longitudinal management for patients with SLE.

Impact on Efforts:

Recent and proposed New York State policy initiatives reflect growing recognition of structural barriers to autoimmune disease care, though they do not yet directly address retention in care as a defined outcome. Telehealth policy, including expanded reimbursement frameworks under Public Health Law Article 29-G and Senate Bill S354, supports remote clinical care delivery and may improve continuity of care by reducing logistical barriers such as transportation constraints, scheduling limitations, and mobility challenges (5). In SLE, where regular monitoring and rheumatology follow-up are essential, telehealth serves as a key adjunct to traditional care that may improve longitudinal engagement, particularly in underserved communities.

Proposed legislation S6034 seeks to establish autoimmune disease treatment centers within public hospitals, expanding access to specialty care in safety-net settings that disproportionately serve Medicaid and low-income populations (6). This initiative may improve geographic access to rheumatology services in underserved boroughs such as the Bronx; however, its impact on retention in care will depend on workforce capacity, care coordination infrastructure, and integration with outpatient specialty follow-up systems.

Proposed legislation S5970, establishing a Center for Autoimmune and Blood Disorder Research at SUNY Downstate Medical Center, strengthens research infrastructure in a socioeconomically diverse and high-need borough (7). This initiative supports epidemiologic, clinical, and translational research that can better characterize patterns of disease burden, healthcare utilization, and barriers to continuity of care among populations disproportionately affected by autoimmune disease. While it does not directly modify care delivery, it provides an evidence base necessary to inform targeted interventions to improve retention in care and reduce disparities.

Collectively, these policy efforts represent meaningful progress toward addressing structural inequities in autoimmune disease care by expanding clinical access, strengthening research capacity, and integrating telehealth into care delivery. However, their impact on retention in care remains indirect, as none explicitly establish systems to measure or ensure sustained engagement in rheumatology care. Without such mechanisms, disparities in continuity of care are likely to persist despite improvements in infrastructure and access.

To meaningfully reduce inequities in SLE outcomes across New York City, future policy efforts must prioritize retention in care through integrated care coordination models,

expansion of rheumatology workforce capacity in underserved areas, and structured incorporation of telehealth into longitudinal disease management systems.

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Resident/Fellow/Medical Student Quality, Patient Safety and Advocacy

From Coverage to Care: Policy Efforts to Reduce Delays in Cancer Screening and Diagnosis

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Summary/Background: Cancer screening is an essential part of preventive medicine as it detects malignancies early and reduces mortality. Early screening for colon, lung, cervical, breast, and prostate cancer can slow disease progression and enable appropriate early treatment. However, prior authorization (PA) requirements create barriers to timely screening, guideline-recommended diagnostic imaging, and appropriate follow-up. PA was originally intended to ensure appropriate use of healthcare services. However, it has been shown to be an administrative burden for clinicians and patients alike, often resulting in financial and emotional stress. The 2025 American Medical Association PA physician survey showed that 88% of physicians said PA interfered with continuity of care. Additionally, physicians and their staff spend an average of 13 hours per week completing PAs. A study published in the American Society of Clinical Oncology showed that 50% of patients reported direct involvement by themselves or a family member in obtaining PA, with 50% spending 1-8 hours, 29% spending 9-24 hours, and 12% spending 41+ hours on a single incident. We reviewed current legislative initiatives in New York State addressing insurance coverage for cancer screening and diagnostic follow-up.

Impact of Efforts/Policy Making/Advocacy: Certain bills reflect ongoing policy efforts in the New York State legislature to reduce barriers to cancer diagnostic screening, follow-up, and treatment. SB2000 ensures mandatory insurance coverage for follow-up lung cancer screening or diagnostic services when appropriately recommended. Insurers cannot require patient cost sharing, which reduces financial barriers for patients. SB310 mandates broadened coverage for certain diagnostic procedures for breast cancer detection. SB586 established a mandatory 5-business-day deadline for insurers to respond to PA claims for cancer patients, which reduces delays in oncological care. If the insurer (private insurers or Medicaid) does not respond within 5 days, the physician is authorized to perform diagnostic imaging and/or treatment.

These legislative bills demonstrate how advocacy policies can support earlier cancer detection, improve access to guideline-recommended care, limit patients' out-of-pocket costs, and reduce administrative delays that may adversely affect patient outcomes.

Future directions: While these bills strengthen coverage requirements, prior authorization is likely to still limit their impact if administrative reviews delay screening. For instance, patients requiring covered breast imaging or lung cancer screening may face delays due to documentation requests or peer-to-peer reviews. Addressing PA barriers in cancer screening requires policy reforms that ensure coverage translates into timely access to diagnostic imaging.

Additional measures are needed to alleviate barriers and reduce the administrative and emotional burden. This could include standardized insurance criteria for guideline-recommended cancer screening and faster review timelines (such as the one provided by SB586) for not only oncology patients, but for primary care. These measures could play an important role in early cancer detection and in reducing associated morbidity and mortality.

Resident/Fellow/Medical Student Quality, Patient Safety and Advocacy

ENHANCING CLINICAL REASONING IN ROUTINE LABORATORY TEST ORDERING : A QUALITY IMPROVEMENT INITIATIVE AT MAIMONIDES MEDICAL CENTER - INITIAL PILOT STUDY

Archa Roy

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Introduction

Routine laboratory testing in hospitalized internal medicine patients is often influenced by established practice patterns rather than individualized clinical need. Excessive laboratory testing may contribute to iatrogenic anemia, patient discomfort, sleep disruption, false-positive results, and unnecessary downstream interventions. High-value care initiatives encourage thoughtful utilization of diagnostic testing while maintaining patient safety. We aimed to improve resident clinical reasoning in routine laboratory ordering through implementation of a clinical stability checklist.

Methods

This pilot quality improvement initiative was conducted on two internal medicine units at Maimonides Medical Center (Aron 5 and K7). Baseline laboratory utilization data were obtained through the Medical Information Systems (MIS) SharePoint database review of November 2025 data. A clinical stability checklist was introduced during daily interdisciplinary rounds to guide patient-specific decisions regarding routine complete blood count, basic metabolic panel, magnesium, and phosphorus testing. Residents received education on checklist use, with attending physician supervision to support clinical decision-making.

Weekly Plan-Do-Study-Act (PDSA) cycles were performed to evaluate laboratory utilization, workflow impact, and safety outcomes. Patients meeting predefined clinical stability criteria were included, while patients requiring frequent laboratory monitoring, hemodynamic support, or respiratory support were excluded. Safety monitoring was performed through retrospective chart review.

Results

Following implementation of the clinical stability checklist, routine laboratory utilization decreased by greater than 25% per patient-day. No adverse events were identified among

patients meeting inclusion criteria. During the monitoring period, one patient outside the study population developed hyponatremia requiring intensive care unit admission, prompting temporary suspension of the intervention and refinement of inclusion criteria through a repeat Plan-Do-Study-Act cycle.

The intervention promoted resident engagement in individualized laboratory decision-making and improved awareness of laboratory stewardship principles. Feedback identified the need for continued resident education, standardized eligibility criteria, and ongoing attending involvement to support safe adoption.

Conclusion

A clinical stability checklist integrated into daily rounds improved resident clinical reasoning and reduced unnecessary routine laboratory testing by more than 25% without identified safety concerns among eligible patients. This pilot highlights the role of structured decision-support tools in promoting high-value care and provides a framework for future expansion across internal medicine units.

Resident/Fellow/Medical Student Quality, Patient Safety and Advocacy

Bridging Policy and Practice: Physician Advocacy to Improve Medicare Preventive Services for Older Adults

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BACKGROUND

Older adults with multiple chronic conditions and social vulnerabilities often face significant barriers to accessing Medicare preventive services, including screenings, vaccinations, and Annual Wellness Visits (AWVs). Although these services are covered by Medicare, utilization remains inconsistent, particularly among patients with complex medical and social needs. Prior studies demonstrate that preventive care improves early disease detection, care coordination, and health outcomes, yet real-world uptake remains limited due to fragmented care, time constraints during visits, and social determinants such as transportation limitations, caregiver burden, and functional decline.[1–4]

At the policy level, growing attention has been placed on strengthening primary and preventive care to improve population health and reduce downstream healthcare costs. However, aligning policy design with real-world clinical practice remains a challenge. This advocacy internship explored how physician engagement in policy and legislative efforts can help bridge the gap between coverage and meaningful access to preventive care for older adults.

METHODS / ACTIVITIES

During this advocacy internship, I examined systemic and policy-level factors influencing Medicare preventive service utilization among older adults. Activities included:

- Reviewing Medicare data and peer-reviewed literature on preventive service utilization and Annual Wellness Visits.[1–6]
- Analyzing state-level legislative efforts in New York aimed at increasing investment in primary and preventive care, including Assembly Bill A8592 and pending Senate Bill S923.[7,8]
- Learning how organizations such as the Primary Care Development Corporation (PCDC) support clinics through funding, technical assistance, and policy advocacy to improve access and care delivery.[9]

- Synthesizing clinical experience with policy analysis to identify advocacy opportunities that reflect real-world patient and practice challenges.

RESULT

This experience highlighted that insurance coverage alone does not guarantee access to preventive services. Despite adequate reimbursement, AWVs and preventive care require time, coordination, and support that are often difficult to achieve in busy clinical settings caring for medically and socially complex older adults. Fragmented care, short visit lengths, and unmet social needs frequently shift focus toward urgent issues, limiting preventive care delivery.[2–4]

I learned that effective advocacy involves translating frontline clinical experience into policy discussions, ensuring legislation reflects patient realities. State efforts to increase primary care investment, including reporting and spending thresholds, may help support infrastructure, workforce capacity, and care coordination necessary to improve preventive care utilization.[7,8] Advocacy is inherently gradual and requires sustained engagement, collaboration, and persistence across multiple stakeholders.

CONCLUSION

Physician advocacy plays a critical role in aligning healthcare policy with real-world patient needs. By engaging in legislative analysis, collaborating with advocacy organizations, and contributing clinical perspectives to policy discussions, physicians can help improve access to Medicare preventive services for older adults. Integrating advocacy training into medical education can empower clinicians to support policies that promote equitable, patient-centered preventive care and healthier aging.

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