New York | ACP American College of Physicians Leading Internal Medicine, Improving Lives

New York Chapter
American College of Physicians

Annual Scientific Meeting

Poster Presentations

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Annual Scientific Meeting

Medical Student Clinical Vignette

Poster Presentations

Medical Student Clinical Vignette

Arvin Azam

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UNKNOWN SOURCE OF SEPSIS REVEALS OCCULT CHOLECYSTODUODENAL FISTULA IN A PATIENT WITHOUT PRIOR CHOLECYSTECTOMY â€“ A CASE REPORT

Purpose: To highlight a case of sepsis in which the key diagnostic finding was an unrecognized cholecystoduodenal fistula, discovered during re-evaluation of pyogenic liver abscesses with persistent bacteremia, emphasizing the importance of repeat imaging and revisiting a differential diagnosis when there is persistent bacteremia.

Methods: We reviewed the clinical course of a patient presenting with confusion, vomiting, and profuse, watery diarrhea, in whom repeat imaging revealed an occult cholecystoduodenal fistula.

Results: A 76-year-old woman with a history of breast cancer, gastroesophageal reflux, and previously treated Helicobacter pylori gastritis presented with confusion, vomiting, and profuse watery diarrhea. She was hypotensive at 85/64 mmHg on arrival and was found to have marked leukocytosis (WBC 29.7), acute kidney injury (BUN 50, Cr 2.47), and hyponatremia (Na 130). Initial CT of the abdomen revealed splenomegaly and a structure in the gallbladder fossa, though the patient denied any history of cholecystectomy. Empiric ceftriaxone and fluids were initiated. Blood cultures later identified Bacteroides thetaiotaomicron and Dialister pneumosintes, prompting escalation to broad-spectrum anaerobic coverage to ertapenem.

Persistent fever and leukocytosis led to repeat imaging, which showed small liver abscesses with gas formation. Due to their size, drainage was deferred. On hospital day six, contrast-enhanced CT revealed a tract between the duodenal bulb and a contracted gallbladder remnant, consistent with a cholecystoduodenal fistula. The patient was not a surgical candidate and completed a course of ertapenem. Her symptoms resolved, and follow-up imaging one month later showed complete resolution of the abscesses and fistula.

The fistula was central to diagnosis and management. While most are associated with gallstones or biliary procedures, our patient had no such history. Its location and her history of H. pylori suggest a prior duodenal ulcer as the likely cause. The fistula likely permitted bacterial translocation, leading to polymicrobial sepsis and hepatic abscesses.

This case emphasizes the importance of considering occult biliary-enteric fistulas in septic patients with atypical pathogens and no clear source. It also demonstrates that small liver abscesses can be successfully treated with antibiotics alone when drainage is not feasible.

Conclusion: Cholecystoduodenal fistula should be considered in septic patients with unclear biliary history, particularly when imaging reveals a gallbladder remnant. Early recognition can guide appropriate medical therapy and avoid unnecessary interventions.

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Syphilitic Hepatitis Presenting as Severe Cholestatic Liver Injury in a 36-Year-Old Male

Background:

Syphilitic hepatitis is a rare but important cause of liver injury characterized predominantly by a cholestatic pattern. Recognition is critical as it mimics other hepatic disorders and responds rapidly to antibiotic therapy.

Case Presentation:

A 36-year-old male with a history of intravenous drug use and treated hepatitis C presented with 3 weeks of jaundice, fatigue, right upper quadrant pain, dark urine, and an intermittent macular rash. Physical exam revealed scleral icterus, cervical and inguinal lymphadenopathy, and a diffuse rash on torso and extremities. Laboratory studies showed markedly elevated alkaline phosphatase (1880 U/L), elevated total bilirubin (11.1 mg/dL), and moderately elevated AST (261 U/L) and ALT (187 U/L). Viral hepatitis panel was negative for active infection; autoimmune markers showed positive ANA but no other specific findings. RPR was highly reactive at 1:64, confirming secondary syphilis. Imaging showed mild hepatomegaly without biliary obstruction. The patient was treated with intramuscular penicillin G 2.4 million units. One week post-treatment, liver enzymes and bilirubin dramatically improved (ALP 87 U/L, bilirubin 3.2 mg/dL).

Discussion:

This case highlights the cholestatic liver injury pattern seen in syphilitic hepatitis, which often presents with disproportionately elevated alkaline phosphatase relative to transaminases. Diagnosis requires high clinical suspicion, serologic confirmation, exclusion of other causes, and documented clinical and biochemical response to therapy. Syphilis, caused by Treponema pallidum, progresses through distinct stages with variable hepatic involvement most commonly in the secondary stage. Syphilitic hepatitis remains a diagnosis of exclusion but should be considered in patients with risk factors such as intravenous drug use and unprotected sexual activity presenting with cholestasis.

Conclusion:

Syphilitic hepatitis, though rare, should be included in the differential diagnosis of cholestatic liver injury. Early recognition and prompt antibiotic treatment lead to rapid resolution and prevent progression to more severe disease.

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Disseminated Tuberculosis in an Immunosuppressed Patient Misattributed to Autoimmune Flare

Disseminated tuberculosis (TB) remains diagnostically challenging in immunosuppressed patients, particularly when symptoms resemble autoimmune flares, hospital-acquired infections, or opportunistic pneumonias. Delay in clinical suspicion and diagnostic testing can result in missed treatment opportunities and poor outcomes.

A 69-year-old Korean woman with systemic lupus erythematosus (SLE), class III lupus nephritis, and chronic immunosuppression presented with two weeks of nonproductive cough, nausea, vomiting, and fatigue, along several months of intermittent subjective fevers. She resided in Flushing, New York, a community with a high TB burden. On admission, she was diagnosed with a multidrug-resistant E. coli urinary tract infection and presumed volume overload secondary to diastolic heart failure. She was treated with intravenous antibiotics and diuretics. Despite this, she remained persistently febrile, and her oxygen requirements progressively increased. Initial chest imaging revealed bilateral infiltrates, which were interpreted as pulmonary edema. CT chest later demonstrated diffuse bilateral ground-glass opacities. She was treated empirically for possible community-acquired and hospital-acquired pneumonia with ceftriaxone, doxycycline, and subsequently cefepime. She remained on Bactrim for PJP prophylaxis.

On hospital day 10, she developed acute hypoxemic respiratory failure, requiring mechanical ventilation, vasopressors, and evaluation for ECMO. Laboratory evaluation revealed worsening anemia, thrombocytopenia, and striking hyperferritinemia. Transthoracic echocardiography showed no valvular vegetations. Rheumatology was consulted and raised concern for macrophage activation syndrome, lupus pneumonitis, PJP, and marantic endocarditis in the differential diagnosis. Although she had multiple risk factors for TB, including chronic immunosuppression, subacute constitutional symptoms, and epidemiologic exposure, TB was not initially considered until hospital day 11. Nucleic acid amplification testing returned positive for Mycobacterium tuberculosis on hospital day 12, and anti-TB therapy was initiated promptly. However, by this point the patient had developed ARDS and multiorgan failure. She died on hospital day 14.

This case illustrates the risk of narrowing a diagnostic focus in complex patients. Although TB is uncommon in the general population, it must remain in the differential diagnosis for patients with unexplained fevers and respiratory decline, especially when epidemiologic risk factors are present. The delay in ordering TB testing likely contributed to disease progression beyond the point of reversibility. Early identification and treatment are critical, as delays in testing can lead to irreversible organ dysfunction and death. Rapid molecular diagnostics should be pursued early in the course of evaluation when the clinical picture remains elusive. This case serves as a sobering reminder of the diagnostic pitfalls that can arise when anchoring bias limits our consideration of infectious etiologies.

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UNEXPLAINED DYSPHAGIA: A PRESENTATION OF LATE-ONSET MYOTONIC DYSTROPHY

Purpose:
To highlight a case of late-onset myotonic dystrophy type 1 (DM1), underscoring the importance of considering this diagnosis in patients with unexplained bulbar symptoms and multisystem involvement.

Case Description:
A 56-year-old female with a history of atrial fibrillation, anxiety, depression, and left adnexal mass was admitted to an outside hospital with acute hypoxic respiratory failure secondary to pneumonia. While her respiratory status improved, her hospital course was complicated by sick sinus node dysfunction requiring dual-chamber pacemaker, dysphagia to both solids and liquids, and suicidal ideation with altered mental status. She was transferred to Westchester Medical Center for further evaluation and management of dysphagia. Labs showed hypoalbuminemia and deficiencies in Vitamin A, B1, B6, D, and E. The patient was initiated on total parenteral nutrition (TPN) while evaluation of dysphagia was completed. Gastroenterology, Otolaryngology, and Neurology were consulted.

Endoscopic evaluation revealed erosive gastritis and extrinsic compression of the upper esophagus, thought to be a stricture which was dilated with no symptomatic improvement. Modified barium swallow demonstrated stasis of contrast at the vallecula and pyriform sinuses and cricopharyngeal muscle hypertrophy. Fiberoptic endoscopic evaluation of swallowing showed reduced laryngeal elevation and lateral wall squeeze, raising the possibility of a neurologic etiology. On evaluation by the neurology team, the patient was noted to have bilateral ptosis and neck muscle weakness. Neurologic workup included paraneoplastic syndrome panel, myasthenia gravis serologies, computerized topography (CT) neck soft tissue, and CT thorax, all of which were unrevealing. Magnetic Resonance Imaging (MRI) was unable to be performed due to recent permanent pacemaker implantation. Electromyography (EMG) revealed myotonic discharges in the biceps, Extensor Carpi Radialis Longus, and first dorsal interosseous muscles.

Based upon the EMG findings and clinical presentation including dysphagia and cardiac arrhythmia, the patient most likely has late-onset myotonic dystrophy type 1 (DM1) can be confirmed with a DMPK DNA test following discharge. Severe dysphagia secondary to DM1 likely led to vitamin deficiencies and altered mentation. The patient was treated with vitamin repletion, PEG tube placement for nutrition, with consideration of cricopharyngeal botox and dilation in the future.

Discussion:
Myotonic dystrophy type 1 (DM1) is a rare (prevalence 5-20 cases per 100,000 population) autosomal dominant multisystem disorder caused by a trinucleotide repeat expansion, most often diagnosed in childhood. It typically presents with muscle weakness, myotonia, cataracts, and cardiac conduction abnormalities. This case describes an atypical, late-onset presentation of DM1 in an adult patient with unexplained bulbar symptoms, cardiac arrhythmia, and altered mentation. Notably, a similar case of adult-onset DM1 reported progressive malnutrition secondary to dysphagia. This case underscores the importance of maintaining a broad differential diagnosis when evaluating patients with complex, multisystem findings, particularly when symptoms do not fit a classic presentation.

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A Family Affair: Pulmonic Stenosis in Pregnancy Without Syndromic Features

Introduction:

Pulmonary stenosis (PS) is a congenital cardiac abnormality caused by obstruction either at the level of the pulmonic valve, in the sub-valvular region, or supravalvular area. It represents 7â€“12% of congenital heart disease (CHD) and may present in isolation or as part of syndromes such as Noonan or Tetralogy of Fallot. During pregnancy, PS may exacerbate right ventricular strain and warrants tailored management.

Case:

A 29-year-old primigravid woman at 16 weeksâ€™ gestation with a known history of congenital PS and prediabetes presented with exertional dyspnea and tachycardia. Two of her three siblings also had congenital PS, including one who required valve replacement in infancy. None showed phenotypic features of Noonan or Williams syndromes or other congenital anomalies.On exam, vital signs were normal. The patient had a 3/6 early-peaking crescendo-decrescendo systolic murmur best heard at the left upper sternal border. There were no diastolic murmurs, jugular venous distention, split S2 with loud P2, or heaves. Electrocardiogram was normal. Echocardiography revealed normal biventricular size and function with mild pulmonic valve stenosis (peak gradient 22.7 mmHg) at the level of the valve orifice. The patient was considered low risk for pregnancy complications and managed conservatively.

Discussion:

CHD affects 1% of live births, and isolated pulmonic stenosis occurs in roughly 0.1%. The likelihood of three siblings having PS independently (0.001³) is approximately 1 in a billion, suggesting a potentially unrecognized genetic inheritance pattern. PS not associated with a syndrome may be passed down in an autosomal recessive inheritance pattern, autosomal dominant inheritance with variable expression and/or incomplete penetrance, or a multifactorial inheritance pattern. PS associated with a syndrome is usually accompanied by characteristic features, which was not present in our patient. The patient was referred for genetic evaluation to explore the possibility of a novel mutation. Fetal echocardiography showed no PS.Although mild PS is usually well tolerated during pregnancy, increased cardiac output can amplify right heart strain, and comorbidities like prediabetes may increase fetal risk. Current guidelines recommend follow-up imaging for moderate or severe cases, with conservative management appropriate in mild cases like this one.

Conclusion:

This case illustrates the importance of individualized management for pregnant patients with CHD. The unusual familial clustering of non-syndromic PS suggests a possible hereditary etiology warranting further genetic investigation, and fetal echocardiography to rule out a significant pre-partum valve lesion. It also reinforces the value of fetal surveillance and interdisciplinary care, particularly in patients with additional risk factors such as prediabetes.

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Rare Postoperative Chyle Leak in a Patient with Diabetic Ketoacidosis Undergoing Elective Robotic-Assisted Cholecystectomy: A Case Report

Introduction:

A robotic-assisted cholecystectomy (RAC) is a minimally invasive procedure for removal of the gallbladder. Common complications include bile duct injury, sepsis and intestinal obstruction. One rare postoperative complication of cholecystectomy is a chyle leak, having only eight reported cases. It carries significant morbidity and thus requires prompt diagnosis and treatment. With there being no current cases in literature, this report presents possibly the first RAC-associated chyle leak and discusses its associated history, findings, and treatment.

Case Description:

65-year-old male presents to the emergency department roughly six days after an elective robotic assisted cholecystectomy (RAC) with severe abdominal pain, nausea, vomiting, and unable to keep anything down post-op. The patient was admitted to the hospital and was found to be in diabetic ketoacidosis associated with his history of Type 2 Diabetes Mellitus, requiring intensive care unit management. The patient underwent computerized tomography of abdomen and pelvis (CTAP), which illustrated bilateral pleural effusions as well as a large complex fluid collection in the gallbladder fossa despite the presence of a drain, raising concern for bile leak or abscess. In preparation of further workup, a nuclear medicine HIDA scan was performed to evaluate for the presence of a bile leak, which was negative. Previous studies indicate that chyle leak can be identified by triglyceride count greater than 110mg/dL collected by the drain or by serum to drain triglyceride ratio. A CTAP scan was used to track the size of the abscess and the drain triglyceride content, which was found to be 127 mg/dL confirming suspicion for a chyle leak, an exceptionally rare complication of laparoscopic cholecystectomy and even more so for RAC.

Discussion:

The therapeutic goals outlined in current literature aligned with our treatment plan, with a focus on limiting lymphatic flow and replenishing lost nutrients, primarily through dietary adjustments (total parenteral nutrition (TPN) and a low-fat, high-protein diet) and pharmacological treatments. Moreover, as opposed to TPN alone, combining octreotide with TPN has been shown to shorten the time required for drain removal. If typical non-operative management is unsuccessful, treatment utilizing interventional radiology by ligation of the thoracic duct can be an alternative. In summary, studying chyle leak in patients after robotic-assisted cholecystectomy is essential for preventing complications, ensuring proper recovery, and refining surgical techniques to minimize risk in future procedures.

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FROM ANTIARRHYTHMIC TO AUTOIMMUNE: AMIODARONE-ASSOCIATED BULLOUS PEMPHIGOID

Introduction:
Bullous pemphigoid (BP) is an autoimmune blistering disorder that manifests as pruritus and urticarial plaques, followed by tense blisters on an erythematous base. It is typically seen in older adults and mediated by autoantibodies targeting hemidesmosomal proteins, BP180 and BP230, resulting in skin detachment and blistering. While BP is classically idiopathic, certain drugs, including dipeptidyl peptidase-4 inhibitors, penicillamine, and checkpoint inhibitors, can be a trigger. Amiodarone is a potent class III antiarrhythmic drug commonly used to manage arrhythmias. There are several well-known adverse effects of amiodarone, but dermatological manifestations, like photosensitivity, blue-grey skin pigmentation, hyperpigmentation, and pseudoporphyria, are less commonly reported.

Case Presentation:
A 59 year old male, with a past medical history of heart failure with reduced ejection fraction (HFrEF) with automatic implantable cardioverter-defibrillator (AICD) and paroxysmal atrial flutter, presents to his primary care provider with a two-week history of a painful, pruritic, blistering rash involving the face, torso, and all four extremities. Notably, the patient had been treated with amiodarone 400 mg twice daily for paroxysmal atrial flutter three months earlier. Given the temporal relationship, amiodarone was suspected to be the inciting agent and was discontinued. Initial outpatient management included hydroxyzine, doxycycline, topical triamcinolone, and a prednisolone taper. Despite partial improvement of his symptoms, the patient was subsequently hospitalized for secondary infection of these lesions with purulent drainage. He received intravenous vancomycin, piperacillin-tazobactam, oral prednisolone, and high potency topical clobetasol. Skin biopsy with direct immunofluorescence (DIF) demonstrated nonspecific findings consistent with possible amiodarone-induced linear IgA bullous dermatosis, bullous pemphigoid, or Stevens-Johnson syndrome. Over two weeks, the patientâ€™s lesions stabilized, and he was discharged with prednisolone taper, topical clobetasol, famotidine, and loratadine; outpatient dermatology follow-up was arranged. Approximately 12 days later, the patient developed new bullae that ruptured with minimal friction, prompting readmission to the hospital. A repeat skin biopsy with DIF showed linear deposition of IgG and C3 along the basement membrane, confirming a diagnosis of BP. The patient was managed with high-dose intravenous methylprednisolone along with topical mupirocin and antihistamines, resulting in significant clinical improvement.

Conclusion:
This case highlights the importance of recognizing amiodarone as a potential trigger for BP, the need for vigilance in patients presenting with new-onset blistering eruptions, especially when recently exposed to medications with known cutaneous side effects such as amiodarone. Repeat biopsy and direct immunofluorescence may be necessary in evolving cases to establish a definitive diagnosis. Additionally, BP tends to be more common in older adults, exemplifying the need to thoroughly review medication history, as polypharmacy and subsequent drug reactions can be common in this population.

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Brugada or Not? A Curious Case of Phenocopy

Introduction:

Brugada syndrome is an autosomal dominant arrhythmogenic disorder associated with increased risk of sudden cardiac death. Diagnosis is based on characteristic electrocardiographic (ECG) findings that are later confirmed by genetic testing. Since 2012, this diagnostic approach has been challenged by reports of Brugada-like ECG patterns that normalize following resolution of underlying, non-genetic conditions. We present an atypical case of this Brugada-like pattern, known as Brugada phenocopy, triggered by electrolyte imbalance.

Case Presentation:

An 83-year-old male presented to the emergency department for worsening lower back pain and new bowel and bladder incontinence. The patient had been hospitalized one month prior for four vertebral compression fractures. He was discharged with a brace and prescribed several pain medications. He remained largely immobile due to progressive lower extremity weakness and worsening radiating lumbar pain. Over the preceding month, he experienced a 15-pound weight loss, attributed to reduced appetite and immobility. One day prior to presentation, he developed multiple episodes of bowel and bladder incontinence. On arrival, his heart rate was 114 bpm. Laboratory testing revealed hyponatremia with serum sodium 123 mmol/L. ECG revealed a right bundle branch block pattern with greater than 2 mm of ST-segment elevation in leads V1â€“V3, followed by downsloping ST-segments and inverted T waves characteristic of Brugada pattern. Correction of the patientâ€™s sodium resolved this Brugada-like EKG pattern. CT scan of the abdomen and pelvis demonstrated findings consistent with a periurethral abscess and prostatitis. Urine culture grew Chryseobacterium arthrosphaerae, and the patient was treated with piperacillin-tazobactam.

Discussion:

Brugada syndrome is rare, affecting approximately 3-5 per 10,000 people. It is caused by a genetic mutation of the cardiac sodium channel that reduces sodium current, leading to disrupted transmembrane ion flow during a cardiac action potential predisposing to life-threatening arrythmias. Other mutations (calcium and potassium channels, channel and desmosomal proteins) have also been linked with the disease. Brugada syndrome accounts for 4% of all sudden cardiac deaths. Implantable cardioverter-defibrillator is the primary treatment.

Brugada phenocopy remains puzzling and is frequently misdiagnosed, leading to inappropriate clinical management and adverse outcomes. The current literature on Brugada phenocopy is limited. Metabolic derangements, notably hyperkalemia, have emerged as the most common precipitating factor. This case report describes a unique presentation of Brugada phenocopy in the context of hyponatremia, a trigger with limited reported cases. This atypical manifestation highlights the importance of heightened clinical attentiveness and contributes to the body of literature on proposed mechanisms underlying Brugada phenocopy. Although both Brugada syndrome and its phenocopy are associated with an increased risk for life-threatening arrhythmias, their management strategies differ. Therefore, accurate recognition and differentiation of Brugada phenocopy from unmasked Brugada syndrome are essential. Timely identification and correction of reversible causes can prevent misdiagnosis and potential mistreatment.

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Cytomegalovirus and the spleen: A rare ischemic complication in an immunocompetent host

Background: Cytomegalovirus (CMV) is a common virus spread through bodily fluids. It usually causes mild, flu-like illness in healthy individuals but may reactivate in immunocompromised patients, leading to severe complications like colitis, retinitis, hepatitis, or encephalitis. This case report describes a rare instance of CMV splenic infarct in an immunocompetent individual.

A previously healthy 22-year-old woman presented with abdominal pain and one episode of blood-streaked vomiting after three weeks of low-grade fever, fatigue, weakness, and decreased appetite. She denied bleeding symptoms, recent travel, sick contacts, or contraceptive use. Vitals on admission were stable. Physical exam revealed right cervical lymphadenopathy, left and right upper quadrant tenderness, and mild splenomegaly. Cardiopulmonary and neurological exams were unremarkable. Lab studies showed WBC 6890/mm³ with 9% atypical lymphocytes, hemoglobin 11 g/dL, platelets 250,000, elevated transaminases (ALT 253, AST 209), and LDH 549. Infectious workup was negative for hepatitis A, B, C, HIV, EBV, malaria, and tick-borne illnesses. CT abdomen revealed splenomegaly with wedge-shaped hypodense areas consistent with splenic infarcts. Hypercoagulable evaluation, including protein C/S, antithrombin III, Factor V Leiden, and antiphospholipid antibodies, was normal. Imaging and echocardiography ruled out embolic sources or thromboses. CMV PCR showed viremia (7890 copies/mL), and IgM was elevated (>240), indicating acute CMV infection. Given her clinical presentation, imaging, and lab findings, she was diagnosed with acute CMV infection causing splenic infarction. Anticoagulation was discontinued, and she improved with supportive care. She was discharged on day six and, at follow-up, was asymptomatic with normalized labs and undetectable CMV PCR.

Discussion: Cytomegalovirus (CMV) typically causes a mild, flu-like illness in healthy individuals. It shares clinical features with Epstein-Barr virus (EBV), including fatigue, fever, lymphadenopathy, atypical lymphocytosis, and hepatosplenomegaly. However, splenic infarction is a rare complication of CMV, especially in immunocompetent patients. This case describes a previously healthy young woman diagnosed with acute CMV infection complicated by splenic infarcts. Her presentation prompted evaluation for alternate causes such as endocarditis, EBV, autoimmune vasculitis, hypercoagulability, and sickle cell diseaseâ€”all of which were ruled out. No evidence of co-infection or clotting disorder was found, and CMV PCR was the only positive result, strongly implicating CMV as the cause of infarction. The exact mechanism by which CMV causes thrombosis remains unclear. Proposed theories include direct endothelial injury, platelet activation, and upregulation of pro-coagulant cytokines (e.g., IL-1Î², TNF-Î±, Factor VIII). While the patientâ€™s coagulation profile was normal at presentation, transient prothrombotic states earlier in the illness may have contributed.

Conclusion: This case highlights CMV-induced splenic infarcts as a rare but important complication in immunocompetent adults, likely driven by endothelial inflammation or transient thrombosis. It underscores the need to consider CMV in febrile illnesses with splenic involvement and supports further study of its vascular effects.

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DAPTOMYCIN-INDUCED EOSINOPHILIC PNEUMONITIS IN A PATIENT WITH A COMPLEX PULMONARY HISTORY

Background: Daptomycin-induced acute eosinophilic pneumonitis (DIEP) is a potentially serious adverse effect of the antibiotic daptomycin, with a reported occurrence of 0.8-2%. Here, we describe a case that highlights this uncommon diagnosis in a complex patient with multiple pulmonary comorbidities.

Case Presentation: A 70-year-old male, with a history of right upper lobe (RUL) squamous cell carcinoma of the lung status-post robotic RUL lobectomy complicated by bronchoesophageal fistula and recently diagnosed T2-T3 osteomyelitis on daptomycin and ceftriaxone for 6 weeks, presented with progressive dyspnea and exertional hypoxia.

At presentation, the patient was afebrile but required 2L nasal cannula to maintain oxygen saturation >92%. Physical examination was remarkable for coarse lower lung field crackles. Chest CT revealed new peribronchovascular and peripheral ground-glass opacities, fibrotic changes, and traction bronchiectasis concerning for pneumonitis. Infectious workup, including bacterial, fungal, and mycobacterial testing, was unremarkable. Laboratory studies were notable for peripheral eosinophilia of 990 cells/Î¼. Given the concern for daptomycin induced AEP, daptomycin was discontinued; the patient underwent diagnostic bronchoscopy. Bronchoalveolar lavage (BAL) cell count showed 14% eosinophils.

After bronchoscopy, he was initiated on IV methylprednisolone 60mg daily for 3 days, then transitioned to oral prednisone 60 mg with a planned taper. By day 3 of steroid treatment, he showed significant clinical and radiographic improvement.

Discussion: The mechanism of DIEP is not clearly understood but is hypothesized to involve either an allergic reaction leading to eosinophilic infiltration of the lung or daptomycin binding of pulmonary surfactant leading to cellular injury. Typical onset is 2-4 weeks after initiation of daptomycin. Symptoms typically include dyspnea, hypoxia, and fever, while radiographic features can vary from reticulonodular, ground-glass, or organizing pneumonia patterns. While the diagnosis of typical acute eosinophilic pneumonia hinges on the classic finding of â‰¥25% eosinophils on BAL, this finding may not be present in over half the cases of daptomycin induced AEP. In some cases, bronchoscopy may not even be necessary if the clinical picture, temporal relation to daptomycin, and non-invasive testing suggest the diagnosis. Once the diagnosis has been made, daptomycin cessation and corticosteroid therapy are the mainstays of treatment.

Conclusion: Despite rare occurrence in the literature, clinicians should maintain a high index of suspicion for drug-induced AEP in patients on daptomycin who present with new respiratory symptoms, especially in the setting of eosinophilia and diffuse infiltrates. Early recognition and withdrawal of the causative agent are critical to minimize progression to respiratory failure.

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A Rare Case of Candida krusei Meningitis in a CAR-T Cell Recipient

Introduction:

Candida krusei is an uncommon cause of invasive candidiasis, accounting for an increasing proportion of nonâ€“Candida albicans infections in patients with hematologic malignancies and prolonged neutropenia. Intrinsic resistance to fluconazole and variable susceptibility to other azoles, combined with poor central nervous system (CNS) penetration of many antifungals, render C. krusei meningitis particularly challenging. Although CNS involvement by Candida species is rare, occurring in fewer than 5% of patients with candidemia, it carries mortality rates up to 30%.

Case Presentation:

A 31-year-old male with relapsed B-cell acute lymphoblastic leukemia post allogeneic stem cell transplant underwent CAR-T therapy, his clinical course was then complicated by febrile neutropenia and neurological decline. CSF analysis revealed pleocytosis (280 cells/Î¼L) and a markedly elevated Î²-D-glucan (>500 pg/mL). MRI demonstrated leptomeningeal enhancement and arachnoiditis. Empiric amphotericin B was initiated; CSF PCR subsequently identified C. krusei, and therapy was transitioned to voriconazole. Fever resolved and CSF cellularity improved, although imaging changes persisted. He was discharged on long-term voriconazole with serial CSF and MRI monitoring.

A month later, he returned with an inability to ambulate. Amphotericin B with flucytosine was initiated for synergistic fungicidal activity and improved CNS penetration. His neurological function has since improved despite worsening of disease on imaging. The patient continues on amphotericin B therapy, with plans for follow-up PET imaging and biopsy to further differentiate imaging findings which suggest ongoing Candida proliferation versus neoplasia.

Discussion:

This case highlights the diagnostic and therapeutic complexity of C. krusei meningitis in immunocompromised individuals. Prolonged cytopenias and B-cell depletion after CAR-T therapy create a "perfect storm" for rare fungal infections. Early recognition requires high clinical suspicion in patients with hematologic malignancies and CNS symptoms, as standard blood cultures may be falsely negative. Unlike bloodstream infections, the fungal burden in CSF is often very low, which diminishes assay sensitivity. Despite this, CSF analysis, including fungal cultures and Î²-D-glucan assays, and MRI are essential for diagnosis. Liposomal amphotericin B, with or without flucytosine, remains the recommended induction therapy due to reliable CNS penetration; step-down to voriconazole is appropriate for susceptible C. krusei isolates, in accordance with IDSA guidelines. Persistent radiographic

abnormalities despite clinical improvement underscore the need for prolonged, multidisciplinary management combining hematology, infectious disease, and neurology services.

Teaching Points:

Consider non albicans Candida in CNS infections: C. krusei is increasingly reported in neutropenic patients and exhibits intrinsic fluconazole resistance, necessitating early antifungal choice tailored to species.

Use combined diagnostics: Definitive diagnosis relies on CSF culture and PCR, Î²-D-glucan measurement, and MRI; blood cultures alone may miss CNS disease.

Recognize CAR-T therapy's emerging fungal risks: Recipients of CD19-directed CAR-T cell therapy experience prolonged neutropenia and cytokine release syndrome that heighten invasive fungal infection risk.

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Malakoplakia presenting as an inguinal mass in a renal transplant patient

Background: Malakoplakia is a rare granulomatous disease thought to be from a bactericidal defect of macrophages in immunocompromised patients. While it can occur in any part of the body, it most frequently occurs in the genitourinary tract and is associated with E. coli. Treatment typically includes reduction of immunosuppression, intracellular penetrating antibiotics, such as fluoroquinolones, trimethoprim/sulfamethoxazole, and rifampin, and occasionally surgery.

Case Presentation: A middle aged female with a past medical history of type 2 diabetes and a deceased donor renal transplant on immunosuppression, initially presented to the hospital with a severely painful, enlarging left inguinal mass. On exam, there was a significant area of induration and erythema in the left inguinal crease with tenderness to palpation. Outpatient US from a week earlier showed a 1.8 x 1.6 x 1.8 cm subcutaneous fluid collection. However, her CT from admission showed a 4.2 x 4.3 cm phlegmon. She was thought to have an abscess and was sent home with amoxicillin-clavulonate with some improvement in her pain. 10 days later, she re-presented with persistent pain, now with an area of ulceration over the mass with scant purulent drainage. Repeat CT suggested necrotic lymph node or other soft tissue mass. Surgical debridement was done without findings of an abscess. Wound culture grew E. coli resistant to fluoroquinolones and penicillins, but sensitive to 2nd generation and later cephalosporins. Fungal cultures were negative. She was discharged on cefuroxime with pending skin specimen pathology.

4 days later, she re-presented for a third time due to persistent pain. MRI revealed a 6.1 x 3.4 x 4 cm soft tissue mass vs. phlegmon. She was started on ceftriaxone, with improvement in pain. At this point, her skin specimen pathology returned suggestive for malakoplakia, but not fully diagnostic. CT-guided core needle biopsy confirmed the diagnosis. She was discharged on IV ceftriaxone. On follow up 2 weeks later, there was notable improvement in the size and appearance of the mass and her pain.

Discussion: We present the case of a middle aged female, 3 years post-renal transplant who developed left inguinal malakoplakia presenting as a painful soft tissue mass. Malakoplakia is a rare condition, primarily described in case reports. While typical treatment recommendations focus on intracellular destruction, there is little guidance on how to treat when the organism is resistant to typical antibiotics. This case demonstrates a possible treatment strategy using biopsy with culture sensitivities to guide treatment. Further, this patientâ€™s presentation was atypical, occurring outside the GU tract and presenting as a neoplasm-like skin and soft tissue mass. This case reinforces that, although rare, malakoplakia should be on the differential for any undefined mass in transplant patients on immunosuppression.

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Rapid onset of amoxicillin-induced drug reaction with eosinophilia and systemic symptoms (DRESS): a case report

Background: DRESS is rare, with an estimated incidence of 1 case per 10,000 medication exposures. Although antibiotics are associated with DRESS, most cases involving antibiotics describe the medication as an aggravating, not purely incitatory, factor. Presentation typically occurs 2-6 weeks following exposure to inciting medications. Case Description: A 57-year-old man with a past medical history of seasonal allergies presented to urgent care for shortness of breath, mild fever, and progressive, red, itchy rash five days after initiating amoxicillin for a dental procedure. Physical exam initially revealed tachycardia, mild respiratory distress, and a diffuse maculopapular exanthem of the trunk, face, and extremities without mucocutaneous involvement. Nikolsky sign was negative. His blood work showed significant leukocytosis and mild liver function test (LFT) elevations, which was felt to be concerning for serum sickness over anaphylaxis. Amoxicillin was held, and the patient received antipyretics, antihistamines, and steroids. The patient was transferred to the emergency department (ED) for acute hypoxic respiratory failure. On arrival to the ED, he was noted to have mild periorbital edema and no evidence of airway involvement. He received intramuscular epinephrine with some symptomatic benefit and was transferred to ICU for close airway monitoring. The following morning, the patient was found to have worsening periorbital edema, new perioral edema, and rash extension into the palms. He was ultimately intubated for severe bronchospasm and resultant hypoxemic respiratory failure. His steroid regimen was escalated to methylprednisolone 2 mg/kg/day. On his third day of hospitalization, the patient's rash contained a new purpuric component. However, the patientâ€™s periorbital and perioral edema exhibited significant improvement. The patientâ€™s steroid regimen was de-escalated. On hospitalization day four, the patientâ€™s clinical exam was overall stable but with new swelling in his hands and feet. He was successfully extubated. Bloodwork revealed 11% eosinophils on differential. Results from a punch biopsy of the purpuric rash component revealed spongiosis and mild superficial dermal perivascular lymphohistiocytic infiltrate with rare eosinophils, consistent with a hypersensitivity reaction. The combination of these findings resulted in a definitive diagnosis of DRESS based on RegiSCAR criteria. The patient demonstrated significant clinical improvement on methylprednisolone 1.3 mg/kg and was discharged on a two-week course of prednisolone with close allergy/immunology follow-up. Discussion: DRESS syndrome typically develops 2-6 weeks after drug initiation, however, this case highlights a more rapid presentation. Amoxicillin is a widely used beta-lactam antibiotic and underrecognized trigger for DRESS. The clinical overlap with infectious diseases, autoimmune conditions, and other drug reactions makes early diagnosis challenging. This case illustrates the importance of maintaining a high index of suspicion for DRESS â€“ even with early onset â€“ when patients present with febrile rash and organ involvement after drug initiation.

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Medical Student Research

Poster Presentations

Alyssa Carlson, BA

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Exploring the Impact of GLP-1 Receptor Agonists on Weight Loss in Cancer Patients: A Retrospective Pan-Cancer Analysis

Background:

Elevated body mass index (BMI) isa common comorbidity in cancer patients and associated with worse outcomes. Chemotherapy can further exacerbate metabolic dysfunction and weight gain. Glucagon-like peptide-1 receptor agonists (GLP-1RA), indicated for diabetes mellitus (DM) and obesity, promote weight loss through insulin secretion, delayed gastric emptying, and appetite suppression. However, the effect of GLP-1RA in cancer patients, particularly during chemotherapy, remains underexplored.

Methods:

This retrospective study identified cancer patients prescribed GLP-1RA between 2015-2024. Demographics, cancer type (solid vs. hematologic), treatment details, and overlap between GLP-1RA and chemotherapy were recorded. BMI changes were analyzed using descriptive statistics (percentiles, medians, or 95% confidence interval) and a one-sample t-test for significance. Welch's t-test evaluated the impact of chemotherapy, sex, and cancer type on BMI reduction. Analyses were performed using R.

Results:

A total of 339 cancer patients treated with GLP-1RAs, primarily semaglutide (48%) and liraglutide (28%), were identified. DM was the primary indication in 92% of cases. Median age at GLP-1RA initiation was 60.8 years (IQR: 52.2-68.4), and median GLP-1RA treatment duration was 11.8 months (IQR: 11.7-26.9). Median BMI decreased from 32.5 kg/m² (IQR: 28.7-38.2) pre-treatment to 31.5 kg/m² (IQR: 27.55-36.95) on/post-treatment, with a median change of -1.0 kg/m² (95%CI: -1.64, -1.01; p-value <0.0001) and a median percentage reduction of -3.71% (95%CI: -4.59%, -2.83%; p-value <0.0001).

Subgroup analyses showed significant BMI reductions in chemotherapy-receiving patients (median: -4.18%; 95%CI: -6.38%, -2.82%; p-value <0.0001) and those not receiving chemotherapy (median: -2.32%; 95%CI: -4.32%, -2.31%; p-value <0.0001), with no significant difference between groups (95%CI: -0.22, 1.28; p-value 0.1669). BMI reductions were consistent across sex (95%CI: -0.91, 0.36; p-value 0.4032) and cancer type (95%CI: -0.77, 0.64; p-value 0.8646).

Conclusion:

GLP-1RAs promoted weight loss in cancer patients, regardless of concurrent chemotherapy, sex, or cancer type. Weight loss was modest, but comparable with results seen in DM management, highlighting the need for clinical trials exploring GLP-1RAs in cancer patient weight management and their potential effects on cancer outcomes.

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Cardiovascular Risk Profiles with GLP-1 Receptor Agonists: A Comparison of PREVENT and ASCVD Scores

Purpose:

This study aims to compare the cardiovascular risks of patients taking glucagon-like peptide-1 receptor agonists (GLP-1RAs) with that of patients with diabetes utilizing the 10-year and 30-year Predicting Risk of Cardiovascular Disease Events (PREVENT) and the Atherosclerotic Cardiovascular Disease risk estimator (ASCVD) scores.

Methods:

A six-year retrospective analysis (2019-2025) was performed using electronic medical records from the Long Island Heart Rhythm Center (LIHRC). Patients were filtered by GLP-1RA use and ICD-10 diagnostic codes for Type 2 diabetes mellitus. Patients were stratified into three groups: those on GLP-1RAs (group 1), those off GLP-1RAs with diabetes (group 2) and without diabetes (group 3). 10-year and 30-year PREVENT and ASCVD scores were calculated using American Heart Association (AHA) criteria. Data is reported as mean. Comparisons utilized unpaired two-tailed t-tests, p 0.05 statistically significant.

Results:

23 patients were on GLP-1RAs in LIHRC, of which 10 had data to complete PREVENT scores. Patients age was 61.8 10.9 years; 3 males, 7 females. PREVENT scores for 10-year total CVD risk were 8.7% and 23.7% for GLP-1RA users (group 1) and diabetic patients not on a GLP-1RA (group 2) [p=0.002]. Similarly, PREVENT scores for 30-year total CVD risk were 19.8% and 41.6% for GLP-1RA users (group 1) and diabetic patients not on a GLP-1RA (group 2) [p =0.005]. No PREVENT difference was observed between the GLP-1RAs (group 1) and the nondiabetic control (group 3). No ASCVD score difference was observed between the GLP-1RAs group and any of the control groups.

Conclusions:

This preliminary study demonstrated that patients on GLP-1RAs have lower PREVENT scores as compared to diabetic patients not on GLP-1RAs. GLP-1RA users had a significant decrease in 10-year and 30-year using the PREVENT score. However, no significant difference was observed using the older ASCVD score. This pilot study requires confirmation in a much larger population.

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Acute Kidney Injury Associated with Acoramidis in Postmarket Surveillance Data

Introduction: Transthyretin amyloid cardiomyopathy is a heart condition in which misfolded transthyretin (TTR) proteins are deposited in cardiac tissue, often leading to heart failure. Acoramidis is a relatively new TTR stabilizer drug which has been recently approved by the FDA for treatment of TTR-mediated amyloid cardiomyopathy. A recent clinical trial for acoramidis identified acute kidney injury (AKI) as a common adverse drug reaction (ADR) associated with acoramidis. This study seeks to investigate the possible ADR association between acoramidis and AKI in postmarket surveillance data.

Methods: We analyze data from the FDA Adverse Event Reporting System database, a dataset of spontaneous ADR reports arising from postmarket surveillance. Data from 2020 to 2024 was included in the study. The cohort was restricted to patients being treated for cardiac amyloidosis to limit confounding effects between ADRs and the underlying condition. Repeated entries were deduplicated using the case ID. Patients experiencing AKI as an ADR were identified by the preferred term "acute kidney injuryâ€, and patients who used acoramidis were identified by the active ingredient "acoramidisâ€. The degree of the ADR association is measured by the reporting odds ratio (ROR) and proportional reporting ratio (PRR). Finally, a similar analysis is also conducted for the TTR stabilizer tafamidis, a drug which shares a similar mechanism of action as acoramidis and has been approved by the FDA since 2019. This allows for a comparison of the AKI risk of acoramidis relative to a baseline TTR stabilizer drug, evaluated on a similar cohort of patients. Patients who used tafamidis were identified by the active ingredient fields "tafamidisâ€ and "tafamidis meglumineâ€.

Results: The cohort consisted of 3,857 patients who reported an ADR associated with cardiac amyloidosis treatment. The ROR computed from this population was 21.43 (95% CI [11.66, 39.38]), suggesting an increased risk of AKI as a result of acoramidis administration. In contrast, the ROR for the AKI risk associated with tafamidis was 0.39 (95% CI [0.28, 0.54]). The PRR metric displays similar trends, with a PRR of 14.22 (95% CI [7.74, 26.13]) for acoramidis and 0.40 (95% CI [0.28, 0.55]) for tafamidis.

Conclusion: Initial clinical trials for acoramidis, a newly approved TTR stabilizer drug indicated for TTR-mediated amyloid cardiomyopathy, has shown signs that AKI may be a potential ADR associated with acoramidis. Our study uses postmarket surveillance data from the FDA Adverse Event Reporting System to further study this potential ADR. Our findings suggest that acoramidis use indeed exhibits higher associations with AKI, and thus further investigation may be warranted to study the effect of acoramidis on the kidney, as well as heightened clinical monitoring of renal function for patients undergoing acoramidis therapy.

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Electrocardiographic Comparison of Hypermobile Patients and Sex-Matched Controls at a Cardiac Specialty Clinic

Context: Patients with Ehlers-Danlos Syndrome (EDS) and Hypermobility Spectrum Disorder (HSD) often present with cardiac symptoms, including presyncope, syncope, chest discomfort, and palpitations. These complaints are followed up with cardiac workup, but few studies have demonstrated whether 12-lead electrocardiogram (ECG) intervals differ between hypermobile and non-hypermobile patients. Understanding baseline conduction parameters may offer insight into arrhythmia risk and autonomic dysfunction in this special population.

Objective: To compare ECG parameters - heart rate (HR), PR interval (PR), QRS duration, QT intervals, and corrected QT interval (QTc) - between hypermobile patients and age- and sex-matched non-hypermobile controls.

Methods: A retrospective study (January 2019 - November 2024) included all hypermobile patients evaluated at the Long Island Heart Rhythm Center. Each hypermobile patient was matched 1:1 with a non-hypermobile control based on sex at birth and age within 10 years. ECG parameters (HR, PR, QRS, QT, and QTc) were extracted from 12-lead ECGs and compared using an independent paired Student's t-test. The effects of QTc-prolonging medications per CredibleMeds.org was also analyzed. Data is reported as mean ± SD; $p<0.05$ was statistically significant.

Results: 144 patients had a hypermobile diagnosis of which 102 had EDS: age 36.5+12.4; M/F [6.9%][93.1%] and 41 had HSD: age 40.7+14.3; M/F [16.3%][81.3%]. The control cohort had an equal number of patients (144): age 37.5+12.7. Hypermobile patients had a heart rate of 76.0±14.8 bpm, PR interval of 142.1±16.8 ms, QRS duration of 90.3±14.1 ms, QT interval of 383.7±36.9 ms, and QTc of 425.4±22.6 ms. Controls had a heart rate of 76.9±15.1 bpm, PR interval of 147.6±22.5 ms, QRS duration of 90.9±16.2 ms, QT interval of 384.2±35.3 ms, and QTc of 428.5±32.1 ms.The PR interval was significantly shorter in hypermobile patients when compared to controls (mean difference = 5.5 ms; $p = 0.019$). There were no statistically significant differences in heart rate ($p = 0.617$), QRS duration ($p = 0.719$), QT interval ($p = 0.909$), or QTc ($p = 0.344$). Use of QTc-prolonging medications did not account for observed findings.

Conclusions: Hypermobile patients demonstrated a significant shortening of the PR interval compared to age- and sex-matched controls, which may be related to an increase in sympathetic tone, consistent with prior studies. Further investigation is needed to confirm these observations and determine whether they are related to autonomic dysfunction and/or pharmacologic effects.

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PUBLIC INTEREST IN HEART HEALTH: THE EFFECT OF HEART MONTH

Background: American Heart Association have put forth significant efforts to increase public awareness of cardiovascular disease and stroke, utilizing social media to improve community education and to reduce preventable disease. Efforts are especially prominent during the month of February, termed the "American Heart Monthâ€. In this study, we assessed public interest in "heart healthâ€ and "heart health for womenâ€ during February compared to other months over 5 years' period to examine the effects of increased social media and event efforts put forth by societies in the United States. Understanding these trends can provide valuable insight into how public engagements, especially with targeted educational efforts with technology influences public interest for health-related topics.

Methods: Google Trends "exploreâ€ functionality extracted Relative Search Interest (RSI) points for each week from 03/2019 to 02/2024 for the terms "heart healthâ€ and "heart health for womenâ€ in the United States. RSI values were quantified from 0 to 100 and means were calculated for each month. RSI value was determined by relative number of search inquiries within a set time, with 0 being minimal searches and 100 being maximum searches. Weeks including days within the month of February were included in mean RSI for February. Statistical analysis compared differences in mean RSI for month type (February and non-February) and 12-month intervals using Tukey's post-hoc multiple comparison test and ANOVA.

Results: Analysis revealed a cyclical pattern with increasing growth over the 5-year period with significant peaks during February of each year. RSI for "heart healthâ€ increased by an average of 6.0±0.5(SE) per year (P<0.001, r2= 79%). There was significant RSI increase from Year 1 (41.2±7.2) to Year 5 (63.5±9.4, p<0.001). Mean RSI for February was significantly higher than non-February months (71.8±14 vs. 48.6±10, respectively, p<0.001). Notably, there was 5-fold increase in RSI for "heart health for womenâ€ from 2018 (6.4±6.6) to 2023 (34.0±18.4, p<0.001), and mean RSI was significantly higher during February compared to non-February months over 5 years (42.6±27.4 vs 16.4±14.7, respectively, p<0.001).

Conclusion: The results of this study show growing public interest in heart health and women's heart health over the recent 5- year period, with an additional increase during February. Our findings highlight the impact of "Heart Monthâ€ and the importance of utilizing online resources to improve community awareness in cardiovascular disease. Further research can investigate methods and efficacy of such tools to achieve increased intrinsic desire of patients to engage online. This will allow health related professional groups to work towards a more informed and proactive public regarding cardiovascular health, leading to better health outcomes.

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EFFICACY AND SAFETY OF EMPAGLIFLOZIN IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION: A META-ANALYSIS OF RANDOMIZED CONTROLLED TRIALS

Background: Empagliflozin is a sodium glucose co-transporter 2 inhibitor that improves cardiovascular outcomes in patients with type 2 diabetes mellitus, chronic kidney disease and heart failure. There are limited data about the efficacy and safety of empagliflozin in patients after acute myocardial infarction.

Methods: PubMed, Embase and Cochrane databases were searched for randomized controlled trials (RCTs) comparing empagliflozin versus placebo in patients with acute myocardial infarction with primary outcome of interest of all-cause mortality. Odds ratio (OR) or mean difference (MD) and their 95% confidence intervals (CIs) were computed with the use of random-effect model. Heterogeneity was examined with I2 statistics.

Results: We included 12 RCT articles consisting of 23,178 patients that received empagliflozin after acute myocardial infarction. The mean age of the patients ranged from 64.8 ± 11.2 years and 79.6 % are men. The pooled analysis showed that empagliflozin was associated with significantly reduced incidence of all-cause mortality (OR 0.72; 95% CI [0.61-0.85]; P < 0.0001; 5.9% vs 7.4%), death from cardiovascular causes (OR 0.66; 95% CI [0.52-0.83]; P = 0.0004; 4.0% vs 5.4%), hospitalization for heart failure (OR 0.66; 95% CI [0.58-0.76]; P < 0.00001; 3.2% vs 4.7%), acute kidney injury (OR 0.52; 95% CI [0.31-0.86]; P = 0.01; 0.96% vs 1.65%), acute renal failure (OR 0.71; 95% CI [0.55-0.91]; P = 0.008; 3.60% vs 4.60%), adverse events leading to discontinuation (OR 0.89; 95% CI [0.80-0.99]; P = 0.03; 11.3% vs 11.7%), systolic blood pressure (MD -8.59; 95% CI [-13.26, -3.93], P = 0.0003), and thromboembolic events (OR 0.51; 95% CI [0.28-0.92]; P = 0.03; 0.5% vs 0.91%) compared to placebo.

However, there was no significant difference between groups with respect to fatal or nonfatal stroke (OR 1.15; 95% CI [0.91, 1.46]; P = 0.23; 3.51% vs 3.05%), adverse events leading to lower limb amputation (OR 1.11; 95% CI [0.62, 1.99]; P = 0.73; 0.82% vs 0.50%), estimated glomerular filtration rate (eGFR) (MD 0.45; 95% CI [-0.20, 1.10]; P = 0.17), diastolic blood pressure (DBP) (MD -2.53; 95% CI [-5.40, 0.34]; P = 0.08), body weight (MD -1.04; 95% CI [-4.08, 1.99]; P = 0.50), urinary tract infection (OR 1.04; 95% CI [0.92, 1.17]; P = 0.55; 16.8% vs 16.5%), NT- proBNP (MD -115.93; 95% CI [-271.52, 39.66]; P = 0.14), glycated hemoglobin (HbA1c) (MD -0.10; 95% CI [-0.41, 0.22]; P = 0.55), and hypoglycemia (OR 0.98; 95% CI [0.89, 1.08]; P = 0.70; 16.6% vs 15.1%).

Conclusion: Our findings suggest that empagliflozin has superior efficacy and safety compared to placebo for the treatment of patients with acute myocardial infarction but with no impact on UTI, eGFR , NT-proBNP, HbA1c, stroke, DBP, body weight, adverse events leading to lower limb amputation and hypoglycemia.

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Fractional flow reserve guided complete revascularization versus Culprit-only percutaneous coronary intervention in patients with ST-segment elevation myocardial infarction and multivessel coronary artery disease. A meta-analysis of randomized controlled

Background: Data comparing the efficacy and safety of fractional flow reserve (FFR)-guided complete revascularization (CR) to culprit-only percutaneous coronary artery intervention (PCI) in patients with ST-segment elevation myocardial infarction (STEMI) and multivessel coronary artery disease are limited.

Method: PubMed, Embase and Cochrane databases were searched for randomized controlled trails (RCTs) comparing FFR-guided CR to Culprit-only PCI in patients with STEMI and multivessel CAD. The primary outcomes of interest were major adverse cardiac events (MACE) and all-cause mortality. The secondary outcomes included were PCI revascularization, unplanned revascularization, stroke, major bleeding, myocardial infarction (MI), heart failure (HF) or hospitalization for HF, death from any cause or MI, any stent thrombosis, contrast-induced nephropathy, death from cardiovascular causes and CABG revascularization. Heterogeneity was examined with I2 statistics, and a random-effects model was used for outcomes with high heterogeneity.

Results: We included 4 RCTs with 3,173 patients comparing FFR-guided CR with culprit-only PCI in patients with STEMI and multivessel coronary artery diseases. The mean age was 78 ± 4.6 years old, and most patients were female (91.3%). The pooled results of the 4 RCTs showed that MACE (RR = 0.66; 95% CI [0.45, 0.99]; p = 0.01; 16.8% vs 24.1%) and PCI revascularization (RR = 0.50; 95% CI [0.37, 0.67]; p < 0.00001; 13.1% vs 24.9%) were significantly reduced in the FFR-guided complete revascularization group compared to culprit-only PCI group.

However, all-cause mortality (RR = 1.00; 95% CI [0.83, 1.20]; p = 0.97; 5.8% vs 5.6%), stroke (RR = 1.17; 95% CI [0.77, 1.77]; p = 0.47; 1.9% vs 1.6%), major bleeding (RR = 1.19; 95% CI [0.87, 1.64]; p = 0.28; 2.4% vs 2.0%), death from cardiovascular causes (RR = 0.88; 95% CI [0.68, 1.14]; p = 0.32; 3.0% vs 3.3%), MI (RR = 0.89; 95% CI [0.60, 1.32]; p = 0.57; 5.9% vs 7.2%), rehospitalization for HF (RR = 0.72; 95% CI [0.44, 1.15]; p = 0.17; 3.4% vs 5.3%), unplanned revascularization (RR = 0.42; 95% CI [0.17, 1.05]; p = 0.06; 3.9% vs 9.9%), death from any cause or MI (RR = 1.02; 95% CI [0.61, 1.71]; p = 0.95; 14.9% vs 13.6%), any stent thrombosis (RR = 1.45; 95% CI [0.73, 2.86]; p = 0.28; 1.6% vs 1.1%), contrast-induced nephropathy (RR=0.47; 95% CI [0.06, 3.82]; p=0.48; 1.38 vs 3.71), and CABG revascularization (RR=1.25; 95% CI [0.30, 5.15]; p = 0.76; 2.62 vs 1.38) were not statistically different between groups.

Conclusion: Our meta-analysis suggests that FFR-guided CR is safe and has superior efficacy to Culprit-only PCI in patients with STEMI and multivessel coronary artery disease with significantly lowered incidence of MACE and PCI revascularization. However, FFR-guided CR showed no beneficial effect on all-cause mortality, stroke, major bleeding, unplanned revascularization and MI.

Dillon Pekoff

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UTILIZING PREVENT TO INVESTIGATE THE ASSOCIATION BETWEEN CARDIOVASCULAR DISEASE RISK AND OSTEOPOROSIS

Purpose

This study aims to use the "Predicting Risk of cardiovascular disease EVENTsâ€ (PREVENT) score to further investigate the link between osteoporosis and cardiovascular disease risk.

Methods

This retrospective cross-sectional study was conducted using the 2017-2020 Center for Disease Control National Health and Nutrition Examination Survey (NHANES) database. Patients were selected based on having documented bone mineral density (BMD) and bone mineral composition (BMC) from a DEXA scan, along with a diagnosis of osteoporosis. Of this subset, subjects were further screened for data needed to calculate a PREVENT and Atherosclerotic Cardiovascular Disease (ASCVD) risk score as recommended by the American Heart Association. Exclusion criteria for this study included a history of rheumatologic diseases that might influence findings. Additionally, the use of drugs known to reduce BMD, such as glucocorticoids, were exclusion criteria. Patient data was imported, stratified, and analyzed using Python. Significance of osteoporosis incidence to PREVENT and ASCVD score was determined using a Mann-Whitney U-test. A spearman correlation (Ï) was performed to test for directional relationship of BMD and BMC of femur and spine with PREVENT and ASCVD scores.

Results

PREVENT and ASCVD scores for 1013 patients were calculated. Of the patients with osteoporosis (n=99), the median PREVENT and ASCVD scores were 11.37% and 16.98%, respectively. For patients without osteoporosis (n=914), median PREVENT and ASCVD scores were 7.6% and 10.4%, respectively. The increase in both PREVENT (+3.76%, p=0.0014) and ASCVD (+6.58%, p=0.00001) scores for osteoporotic patients was statistically significant.

Examining the relationship between PREVENT scores with spinal BMD (Ï=0.11633, p<0.001) and femur BMD (Ï=0.082242, p<0.05) both revealed a statistically significant weak positive correlation. PREVENT scores were weakly correlated with both spine (Ï=0.18475, p<0.001) and femur (Ï=0.24256, p<0.001) BMC. ASCVD scores showed statistically significant weak positive correlations with spine (Ï=0.07125, p<0.05) and femur (Ï=0.10213, p<0.05) BMC. However, ASCVD scores showed statistically insignificant correlations with spine (Ï=0.03346, p=0.287) and femur (Ï=-0.0137, p=0.661) BMD.

Conclusion

Within the patient population, it was determined that a diagnosis of osteoporosis results in a significant increase in cardiovascular risk when measured by both PREVENT and ASCVD scores. Analysis of femur and spine BMD and BMC showed a weakly positive, statistically significant relationship with PREVENT

scores. When comparing BMC of the femur and spine to ASCVD score, there is a weakly positive, statistically significant correlation. However, when comparing BMD of the femur and spine to ASCVD score, there is no statistical significance to show their relation. Further study can be done by stratifying for common variables that effect BMD, including sex, age, and ethnicity. Our data is adjunctive to surrounding literature that draws a connection between major cardiac adverse events and osteoporosis.

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Northwell Health Zuckerberg Cancer Institute

A retrospective observational analysis examining liquid biopsies with tissue biopsies in patients with advanced non-small cell lung cancer

Non-small cell lung cancer (NSCLC) is genetically diverse, often harboring multiple mutations that can be targeted with FDA-approved therapies. Identifying these actionable mutations is critical and can be achieved through tissue- or blood-based next-generation sequencing (NGS). Tissue-based biopsies, considered the gold standard, involve invasive sampling and have long turnaround times (2-4 weeks), potentially delaying treatment. Blood-based NGS, a less invasive technique, analyze circulating tumor DNA from blood samples and provide faster results, though they remain a newer modality that is not used as widely.

This single-institution, retrospective, IRB-approved study analyzed the concordance of blood- and tissue-based biopsies in patients with metastatic NSCLC, along with the time from blood-based NGS to treatment initiation. We identified 42 patients treated at the Northwell Health Cancer Institute between January 1, 2016, and March 31, 2020, who underwent both biopsy modalities within one month of each other.

Concordance analysis revealed that the two modalities identified the same actionable mutation in 57.1% of cases but in 42.9% of cases it was only identified by one modality. Blood-based NGS identified actionable mutations in 11 patients (26.2%) that tissue-based biopsies did not detect, enabling treatment initiation based on a non-standard diagnostic method. Conversely, tissue-based biopsies identified mutations missed by blood-based sequencing in seven cases (16.7%).

Of the 42 patients, 13 were initiated on treatment based on blood-based NGS findings due to its faster turnaround time or its ability to detect mutations missed by tissue-based biopsy. The median time to treatment initiation from blood collection for these 13 patients was 14 days (range: 7-24 days).

Our findings highlight the complementary value of combining blood- and tissue-based NGS to enhance mutation detection and optimize treatment strategies. Those not detected on tissue were likely due to heterogeneity or suboptimal tissue sampling and processing. Blood-based NGS, though not as widely performed, may facilitate faster treatment initiation, particularly when tissue-based results are delayed or insufficient. Future directions include expanding the patient cohort, analyzing mutation-specific detection rates, and evaluating the clinical outcomes associated with incorporating blood-based biopsies into routine care.

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FIRST DOSE, FIRST STEP: WHY MEDICATION RECONCILATION IS THE CORNERSTONE OF INPATIENT CARE

Introduction: Medication reconciliation (MedRec) discrepancies have been reported in a wide range of 3.4% to 98.2% of hospitalized patients, with medication-related complications or potential adverse drug reactions occurring in up to 94% of cases.â€‹1â€‹ This quality improvement initiative aims to explore whether implementing a structured MedRec process led by internal medicine residents can improve the frequency and accuracy of reconciliation and reduce medication-related discrepancies.

Methods: We conducted a pre/post-intervention study (January-April 2025) at Albany Medical Center. Our March 3rd, 2025, intervention combined resident education on MedRec best practices with electronic health record (EHR) template integration of a MedRec status button. This education included 30-minute interactive and case-based sessions during academic half day for over 70 residents. We reviewed charts of patients on internal medicine resident teams hospitalized â‰¥48 hours, excluding ICU transfers. Data collection assessed medication review date, reconciliation status, and accuracy, with patients categorized into four reconciliation groups: group 1 (not reviewed, not reconciled), group 2 (reviewed, not reconciled), group 3 (reviewed, reconciled), and group 4 (not reviewed, but reconciled)

Results: We analyzed 68 pre-intervention and 70 post-intervention patients. The date that MedRec was reviewed improved significantly in the post-intervention: never reviewed decreased from 32.4% to 21.4%, while day 0 (admission) review increased from 63.2% to 71.4%. Frequency of reconciliation increased from 79.4% (54/68) in pre-intervention to 90% (63/70) in the post-intervention. The primary outcome of reviewed and reconciled medications (Group 3) increased from 60.3% (41/68) pre-intervention to 74.3% (52/70) post-intervention, though this did not reach statistical significance (risk ratio = 0.8117, 95% CI 0.6403-1.0288, one-tailed p-value = 0.058). The secondary outcome of medication reconciliation accuracy significantly improved from 47.1% (32/68) to 74.3% (52/70) post-intervention (risk ratio = 0.6335, 95% CI 0.4753-0.8443, one-tailed p-value = 0.0009). The proportion of patients with inaccurate reconciliation decreased from 52.3% (36/68) to 25.7% (18/70).

Discussion: MedRecs are often difficult and time consuming, necessitating a standardized approach. For accuracy and timing, it is important to take initiative, avoid over-reliance on others, and communicate efforts and next steps clearly. Our intervention combining resident education with EHR integration improved the accuracy of MedRecs. Limitations to our study included exclusion of certain patient populations, small sample size, short duration and most importantly, the Hawthorne effect as residents were aware of the post-intervention data collection. Similar interventions may be implemented at other institutions to improve MedRec frequency and accuracy to ultimately improve patient safety and outcomes.

References

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Poster Presentations

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Case of Hemophagocytic Lymphohistiocytosis Secondary to Gastroenterological Illness In A Young Male

31 year old previously health male without significant medical history who presented with diarrhea and abdominal pain after eating in a restaurant in NYC and was found to have a partial small bowel obstruction and splenomegaly on CT that resolved with a nasogastric tube. He was started on multiple antibiotics including vancomycin and meropenem for fever T 38.6 C and leukocytosis WBC 21 due to concern for infection. He then developed a diffuse erythematous rash and antibiotics were stopped. Infectious workup was negative with normal GI multiplex, respiratory multiplex PCR, and blood culture. Ferritin was elevated to 16500 and then increased to more than 40,000. He had transaminitis with ALT 387 U/L and AST 194 U/L. CBC showed mild anemia with hemoglobin 37% and hematocrit 12.3 g/dL. Bone marrow flow cytometry showed no evidence of leukemia, lymphoma, myelodysplasia, or increase in blasts, but it did show occasional hemophagocytosis. IL2 receptor (CD25) was elevated at 5759 pg/mL, reference value of (175 - 858). He was treated with prednisone 1 mg/kg oral daily and then dexamethasone with improvement in rash, sinus tachycardia, transaminitis, and weakness. After the bone marrow showed hemophagocytosis, Anakinra 1 mg/kg SC q6 hours was added. CT showed no PE and US showed no DVT. On Anakinra and dexamethasone, his CRP decreased from 17 mg/dl to 7.3. Ferritin improved from > 40,000 to 20,941. Tachycardia and fever resolved. He was deemed stable and discharged home with hematology follow up.

Hemophagocytic lymphohistiocytosis (HLH) is a rare disorder but early diagnosis is important for outcomes. Our patient did not have cytopenia in 2 lines. He also did not have elevated triglyceride of 226 mg/dL below the 265 cutoff. Fibrinogen was not low 717 mg/dL (reference range 187 - 446). His initial leukocytosis and fever led to a search for infection which was unrevealing and negative for EBV. Patient's drug rash might have been due to multiple antibiotics. His RegisSCAR score was low with no enlarged lymph nodes, atypical lymphocytes and so DRESS was less likely. Eosinophils were not elevated.

HLH needs to recognized in a timely manner and yet it should not be incorrectly diagnosed because treatment may cause immune suppression and infections. If the IL2 receptor (CD 25) is normal, then this may rule out HLH even if the patient may meet other criteria such as fever, splenomegaly, cytopenia in at least 2 cell lines, hypertriglyceridemia or hypofibrinogenemia, and elevated ferritin. Decreased NK cell activity is also nonspecific for HLH. We present a rare case of HLH to highlight the importance of early recognition and treatment.

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Disparities in Sarcoidosis Outcomes: A Nationwide Study of Socioeconomic and Racial Influences

Background:

Sarcoidosis is a chronic systemic inflammatory granulomatous disorder with a prevalence of 60 per 100,000 in United States. It has diverse clinical presentation with considerable variation in presentation and complications among different geographical, racial and socio-economic groups. Although lungs are invariably affected, the severity and outcomes, including complications like acute respiratory failure (ARF) and pulmonary fibrosis (Stage 4 sarcoidosis), remain understudied in large real-world cohorts.

Methods:

We conducted a retrospective analysis using the 2021 National Inpatient Sample (NIS) to identify adult hospitalizations with sarcoidosis using ICD-10 codes D860 and D862 (I10_DX2-I10_DX25). Patient demographics (age, sex, race, income, insurance) and comorbidity burden (Charlson Index) were included. Analyses accounted for the complex NIS survey design using appropriate weights, strata, and clusters. Descriptive statistics, survey-weighted logistic regression, and multivariable models ($p < 0.2$ inclusion threshold) were used to assess associations with outcomes including respiratory failure, pneumonia, in-hospital mortality, and length of stay.

Results:

The patients had a mean age of 62.6 years and 58.5% were female. Black patients comprised 44.7% of the population, significantly younger on average (59.5 years) compared to White patients (65.7 years, $p<0.05$).

ARF occurred in 4.2% of patients, with higher odds in Black patients (OR: 2.12, $p<0.001$). Higher income quartiles have lower risk (OR: 0.56-0.69 in upper income quartiles, $p=0.0044$). Age conferred a modest protective effect against ARF with 1.2% decrease in risk with every one year increase in age (OR: 0.988, $p=0.035$). PNA was noted in 3.9% but showed no significant associations with age, sex, race, or income status. Pulmonary fibrosis/ stage 4 sarcoidosis was diagnosed in 8.3% (n=1,995), predominantly in Black (48.6%) and White (43.2%) patients. Stage 4 sarcoidosis was more frequent in older individuals (mean age 64.9), with women presenting at a significantly older age than men (67.1 vs 62.0 years, $p=0.0001$). Previous studies have shown 10-40% of cases show progression of sarcoidosis with 7% mortality over 5 years(3). But in our study, amongst Stage 4 patients, only 4.8% had ARF and 3.0% had PNA, which were similar to what was observed with unstaged sarcoidosis. About 0.25% developed Pneumocystis pneumonia (PCP), compared to 0.01-0.015% the general population.Overall, in-hospital mortality was 4.1%, with higher mortality observed in those with ARF (6.9% vs 3.95%, $p=0.0338$). Age was associated with increased mortality risk (OR: 1.015/year, $p=0.004$), and Black and Hispanic patients showed higher odds of death (OR: 1.39 and 1.98, respectively).The median length of stay was 4 days, with females having significantly shorter stays (mean difference â˜’0.93 days, $p<0.001$).

Conclusion:

This study highlights significant racial and socioeconomic disparities in sarcoidosis complications and outcomes. Black patients are at increased risk for ARF, pulmonary fibrosis, and mortality. Higher income appears protective against ARF and longer hospital stays.

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Unveiling The Uncommon: Acute Valvulitis in Systemic Lupus Erythematosus Without Libman Sacks Endocarditis

Lupus valvulitis is an inflammatory manifestation of systemic lupus erythematosus (SLE) affecting the heart valves. While it is typically associated with Libman-Sacks endocarditis (LSE)-a nonbacterial thrombotic endocarditis characterized by sterile vegetations-there exists a rarer subset of lupus valvulitis without LSE. In such cases, patients present with inflammatory valvular damage and dysfunction in the absence of classic vegetations. Valvular involvement may progress silently or manifest with symptoms such as dyspnea, chest pain, and features of heart failure. We report two cases of lupus valvulitis without echocardiographic or histopathologic evidence of LSE.

The first case involved a 25-year-old woman with a history of lupus nephritis and poor follow-up after initial diagnosis. She presented with progressive dyspnea, chest pain, and signs of systemic inflammation. Imaging revealed new severe mitral and moderate tricuspid regurgitation, a large pericardial effusion, and bilateral pleural effusions. Despite these findings, transesophageal echocardiography (TEE) showed no valvular vegetations. Laboratory evaluation revealed high titers of anti-dsDNA, anti-Ro, anti-histone, and anti chromatin antibodies with markedly elevated inflammatory markers. She responded to high-dose corticosteroids and immunosuppressive therapy and was discharged on a regimen of azathioprine, hydroxychloroquine, and a prednisone taper.

The second case described a 21-year-old man with a prior diagnosis of cutaneous lupus who had been inconsistently taking mycophenolate mofetil and hydroxychloroquine. He presented with chest pain, febrile illness, and acute renal dysfunction. Imaging showed severe aortic regurgitation, mitral regurgitation, a pericardial effusion, and newly reduced left ventricular ejection fraction. Despite the severity of valve dysfunction, echocardiography revealed no vegetations. His condition deteriorated to anuric renal failure requiring continuous renal replacement therapy. Following high-dose corticosteroids and immunosuppression, he underwent surgical aortic valve replacement, mitral valve repair, and tricuspid valve repair. Aortic valve pathology confirmed myxoid degeneration without evidence of LSE.

Both cases illustrate a shared clinical pattern of severe valvular dysfunction in the absence of vegetations, elevated autoimmune markers indicative of lupus activity, and multiorgan involvement, including pericardial and renal disease. Importantly, neither patient demonstrated positive antiphospholipid antibodies, which are more typically implicated in LSE.

These cases underscore the importance of recognizing lupus valvulitis as a distinct cardiac manifestation of SLE that can present with significant hemodynamic compromise, even in the absence of classic vegetations. TEE remains the imaging modality of choice for evaluating valvular integrity and effusions. Management necessitates a multidisciplinary approach, with prompt initiation of immunosuppressive therapy and surgical intervention as warranted. While LSE is more commonly associated with antiphospholipid syndrome, lupus valvulitis without LSE appears to be driven by active systemic inflammation and autoantibody-mediated injury, as seen in both of our patients with untreated or undertreated disease. Further research is needed to elucidate the pathophysiology, optimal diagnostic criteria, and long-term management of this underrecognized subset of lupus-related valvular disease.

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"Twisted artery, Deadly rhythm: Exercise Induced Ventricular Tachycardia Unmasks a Lethal Coronary Anomaly"

Anomalous aortic origin of a coronary artery (AAOCA) is an uncommon congenital condition that can put adults at risk for serious heart problems, including arrhythmias and sudden cardiac death. Diagnosis is essential when the abnormal vessel passes between the aorta and pulmonary artery, and symptoms may be subtle or non-specific.

A 43-year-old man with a past medical history of hypertension, pre-diabetes, and asthma came to our clinic after two months of dizziness and palpitations. He had always been active and never had symptoms as a child. His first episode of dizziness happened while walking and was accompanied by palpitations and mild chest tightness, but no loss of consciousness. On physical examination, he had normal cardiac auscultation with no murmurs or added sounds appreciated. A second episode occurred while standing. He went to the emergency room, where "extra beats" were noted, but no sustained arrhythmia was captured. He was discharged with plans for outpatient exercise stress test.

During an exercise stress test, he exercised for over six minutes without symptoms, but in recovery developed frequent premature ventricular contractions that escalated to non-sustained monomorphic ventricular tachycardia. He was stabilized with intravenous metoprolol and transferred to a tertiary center. His CCTA showed anomalous origin of the RCA from the left sinotubular junction above the left coronary cusp, adjacent to the left coronary artery ostium. The RCA took an intramural intra-arterial course with a slit-like opening and an acute angle takeoff.

Cardiac catheterization confirmed CCTA findings, with no significant blockages. His left and right ventricles were normal in size and function, with a left ventricular ejection fraction of 57% and a right ventricular ejection fraction of 49%. Cardiac MRI showed no evidence of scarring and only mild basal septal thickening, with no outflow obstruction. He underwent successful surgical unroofing of the anomalous RCA. Postoperative recovery was uneventful, and he was discharged on aspirin, beta-blocker, losartan, and Jardiance, with a wearable defibrillator for interim arrhythmia protection. At the two-week follow-up, he reported improved breathing and energy, with no further episodes of dizziness or syncope.

This case highlights how AAOCA can present in adults with subtle symptoms and exercise-induced arrhythmias. The absence of coronary artery disease and normal heart function on imaging helped focus attention on the congenital anomaly. Early recognition by multimodal imaging and timely surgery are key to preventing life-threatening events. Ongoing follow-up is important for recovery and long-term health.

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Recognizing ANCA-Associated Vasculitis in the Absence of Classic Renal Involvement: A Case of Mononeuritis Multiplex

Background:

Mononeuritis multiplex (MNM) is a distinct clinical entity characterized by asymmetric, multifocal nerve involvement, commonly seen in the setting of systemic vasculitis. Antineutrophil cytoplasmic antibody (ANCA)-associated vasculitis (AAV) is a known cause of MNM, but it is often preceded by overt renal, pulmonary, or constitutional symptoms. We report a case of AAV presenting primarily as MNM in the absence of overt renal dysfunction, highlighting the importance of early suspicion in atypical presentations.

Case Presentation:

A 78-year-old man with hypertension, diabetes, and vitamin B12 deficiency presented with a two-week history of gait instability, bilateral leg weakness, and numbness. Neurologic exam revealed wide-based gait, distal upper and lower extremity weakness (4/5), foot and wrist drop, impaired pinprick and proprioception, and preserved reflexes.

Initial labs showed chronic microcytic anemia (Hemoglobin 9.5 g/dL), mild leukocytosis, and normal kidney function (creatinine of 0.54 mg/dL at his baseline). Urinalysis revealed hematuria and proteinuria (300 mg/dL); the urine protein/creatinine ratio was 3.5. Inflammatory markers (ESR, CRP) were elevated. Autoimmune testing revealed positive p-ANCA, elevated myeloperoxidase antibodies, low C4, positive rheumatoid factor (RF), and anti-CCP antibodies.

Electromyography confirmed a severe axonal, motor-predominant polyneuropathy consistent with MNM. Despite preserved renal function, a kidney biopsy was pursued due to the presence of proteinuria and hematuria. It demonstrated pauci-immune crescentic glomerulonephritis, confirming ANCA-associated vasculitis. The patient was started on high-dose corticosteroids and weekly rituximab, with outpatient rheumatology, nephrology, and physical therapy follow-up. At follow-up, his urine protein/creatinine ratio had improved to 0.3, although neurologic symptoms persisted. He continued with outpatient physical therapy.

Discussion:

This case illustrates the diagnostic challenge when AAV presents solely as mononeuritis multiplex without overt renal or systemic features. Subtle urinary abnormalities and preserved renal function may lead clinicians to attribute neuropathy to more common causes, especially in elderly patients with diabetes and B12 deficiency. Additionally, overlapping autoimmune markers (RF, anti-CCP) can obscure the diagnosis. A high index of suspicion, guided by inflammatory markers and subtle signs of renal involvement, is critical.

Resident/Fellow Clinical Vignette

Conclusion:

Mononeuritis multiplex may be the first and only presenting feature of ANCA-associated vasculitis. Clinicians should consider AAV in patients with unexplained neuropathy, especially when accompanied by inflammatory markers or urinary abnormalities, even in the absence of renal impairment. Early diagnosis and immunosuppressive treatment are essential to prevent irreversible organ damage and improve outcomes.

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Emphysematous Pyelonephritis as a complication of Klebsiella pneumoniae in a well controlled diabetic patient

Introduction:
Emphysematous pyelonephritis (EPN) is a rare, life-threatening necrotizing infection that primarily affects the renal parenchyma and surrounding tissues. This condition is characterized by the accumulation of gas within the renal parenchyma, collecting system, or perinephric space. EPN is most commonly associated with poorly controlled diabetes mellitus and urinary tract obstruction, though it can occur in individuals without these risk factors. Early recognition and aggressive management are crucial for improving patient outcomes.

Case Presentation:

A 64-year-old male with no prior medical history presented with altered mental status (AMS) and signs of sepsis. Initial lab results revealed anemia (Hb 8.5), leukocytosis (WBC 28.37), elevated urea (135), creatinine (12.3), high anion gap metabolic acidosis, and hyperkalemia (potassium 8.1). An electrocardiogram (EKG) showed peaked, wide T waves, indicative of hyperkalemia. The patient was diagnosed with acute renal failure due to suspected urinary tract infection (UTI), which led to sepsis and refractory hyperkalemia.

Diagnostic imaging with computed tomography (CT) revealed an enlarged left kidney with air within the renal parenchyma and perinephric space, consistent with emphysematous pyelonephritis. Blood and urine cultures identified Klebsiella pneumoniae, and the patient was promptly started on meropenem, along with intravenous fluids, broad-spectrum antibiotics, and emergency hemodialysis. On hospital day 4, an 8-French locking loop drainage catheter was placed, and reddish-tan purulent fluid was drained, which also grew Klebsiella pneumoniae. Following drainage, the patient's mental status significantly improved.

Discussion:

EPN is often linked with poorly controlled diabetes mellitus, particularly in patients with a hemoglobin A1c > 11%, but this case highlights the rare occurrence of severe EPN in a patient with well-controlled diabetes (hemoglobin A1c 6.8%) and no evidence of urinary tract obstruction. Early diagnosis using CT imaging is vital for confirming the presence of gas in the renal and perirenal areas. Prompt intervention with broad-spectrum antibiotics and hemodialysis, alongside nephron-preserving procedures like percutaneous drainage, are critical in managing this life-threatening condition. Prognostic factors for poor outcomes include shock at presentation, polymicrobial infection, and the need for hemodialysis.

Conclusion:

EPN should be suspected in patients presenting with AMS and signs of sepsis, particularly in those with renal injury. Early recognition, aggressive treatment, and multidisciplinary care are essential for improving survival and preventing further complications. Vigilance is necessary in managing patients with diabetes and immunocompromised conditions to prevent the development of this severe infection.

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LYSOZYME-INDUCED NEPHROPATHY IN SUSPECTED CHRONIC MYELOMONOCYTIC LEUKEMIA

Introduction:

Chronic Myelomonocytic Leukemia (CMML) is a clonal stem cell disorder that results from a sustained overproduction of myelomonocytes. Lysozyme-induced nephropathy (LyN) is an underrecognized complication of CMML. Lysozymes are lytic enzymes produced by monocytes for their bactericidal properties. They are filtered by the glomerulus and then reabsorbed at the proximal tubule. In CMML, neoplastic monocytes produce excessive lysozymes, which can cause toxic tubular injury, acute kidney injury (AKI), and progression to chronic kidney disease. LyN is a poor prognostic marker in CMML. Early diagnosis can guide treatment and help prevent sustained kidney damage.

Case Presentation:

A 77-year-old man presented to the Veterans Affairs Hospital with weakness after sunbathing for two days prior. His history was notable for Parkinson's Disease and cerebellar ataxia. Laboratory studies revealed acute kidney injury (creatinine 1.7 mg/dL with baseline creatinine 0.9 mg/dL), leukocytosis (45,000/uL with 26% monocytes), thrombocytopenia (59,000/uL), and elevated creatine phosphokinase (CPK) (3984 IU/L). Urinalysis had trace protein and no blood. Urine total protein/creatinine ratio was 1.5 g/g with urine albumin/creatinine ratio at 185 mg/g. Continuous fluids were started for rhabdomyolysis. Flow cytometry revealed a monoclonal B-cell population with atypical features, suggestive of myeloproliferative CMML.

Creatinine continued to uptrend despite fluids, reaching 2.2 mg/dL on hospital day six. Due to the unclear etiology of AKI, a renal biopsy was obtained on hospital day eight. Biopsy results showed lysozyme-positive staining, recovering acute tubular necrosis (ATN), and prominent eosinophilic protein resorption without light chain restriction in the proximal tubule. Findings were consistent with LyN. The patient's course was complicated by acute urinary obstruction relieved with Foley catheter insertion. Creatinine returned to baseline by the time of discharge.

Discussion:

Our patient had several causes for AKI, with rhabdomyolysis, hypovolemia, and urinary obstruction being most evident. However, clinicians should maintain a high level of suspicion for LyN in CMML patients presenting with an AKI and subnephrotic range proteinuria (due to urinary lysozymes rather than albumin). Given our concern for CMML, the patient was evaluated with a prompt kidney biopsy.

Renal abnormalities are common in CMML, with LyN being the most prevalent etiology, especially in myeloproliferative CMML (defined as a leukocyte count greater than 13,000/uL). LyN is a poor prognostic marker of disease as the level of kidney injury correlates with a higher neoplastic monocyte burden. Additionally, diminished glomerular filtration rate limits treatment options, most notably stem-cell transplantation which can otherwise be curative. The most definitive diagnostic tool is a renal biopsy as lysozyme serum and urine levels often do not correlate well clinically. Early treatment with cytoreduction therapy can improve long-term renal function. Our patient's kidney function fortunately recovered and did not require initiation of treatment while inpatient.

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Trapped Lung Following a Ventriculopleural Shunt: A Rare but Important Complication

Background:

Ventriculopleural (VPL) shunts serve as a method for diverting cerebrospinal fluid (CSF) when ventriculoperitoneal (VP) or ventriculoatrial (VA) shunts are not feasible. A rare but severe complication is trapped lung, where the lung fails to re-expand after pleural fluid drainage due to chronic pleural inflammation or fibrosis. We present a case of a trapped lung in a patient with a long-standing VPL shunt and discuss management strategies.

Case Presentation:

A woman in her 60s with spina bifida and hydrocephalus had been shunt-dependent for CSF drainage since childhood. She previously had both VA and VP shunts placed, but due to complications, she underwent a VPL shunt placement 16 years ago. Approximately six years prior to the current presentation, a routine chest X-ray revealed an associated pleural effusion. Over time, she developed progressive dyspnea, fatigue, and loss of appetite with worsening size of the VPL-associated pleural effusion occupying more than half of the hemithorax. A decision was made to convert the VPL shunt to a VP shunt. During revision, 830 mL of pleural fluid was drained from the pleural limb of the shunt, but upon decompression, air began escaping from the shunt. A bedside fluoroscopic radiograph was concerning for a pneumothorax, so the distal catheter was left in situ to function as a chest tube, and a new pigtail chest tube was placed as she reported shortness of breath. Despite dual catheter suctioning and air leak resolution, the lung failed to re-expand, suggesting an underlying trapped lung. Given the patient's surgical risk and chronicity of the trapped lung, decortication was elected against after discussing with the patient, with a plan to manage conservatively long term. After sequentially discontinuing suction from both devices, no change in size of the pneumothorax component was noted, and both catheters were removed by day 5 without return of relevant symptoms.

Discussion:

First introduced by Heile in 1914, VPL shunts are relatively uncommon. They may be preferred in patients with peritoneal adhesions, infections, or vascular access issues. Reported complications include pleural effusions (~16% cases), shunt revisions (~54%), infections (~7%), and obstruction (~13%). Chronic pleural effusions related to VPL shunts can lead to a trapped lung. Trapped lung is rarely reported, limiting evidence-based management. Early detection and management of pleural effusions in VPL shunt patients may prevent this complication. Finally, treatment options depend on patient factors, and input from specialists and discussion with the patient: shunt revision, instillation of pleural lytics, placement of an indwelling pleural catheter, and surgical decortication.

Conclusion:

This case highlights a trapped lung as a complication of VPL shunts. Clinicians should suspect trapped lung in chronically effused patients to prevent long-term morbidity.

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New Onset of Staphylococcal Scalded Skin Syndrome (SSSS) in Liver Cirrhosis Patient

Introduction
Staphylococcal Scalded Skin Syndrome (SSS) is an exfoliative dermatosis primarily affecting infants and immunocompromised individuals, caused by Staphylococcus aureus exfoliative toxins. Although adult occurrences are uncommon, patients with immunocompromised conditions have an increased risk to develop SSS with higher risk of complications. We present a case of a female patient with alter mental status secondary to decompensated liver cirrhosis and subsequently developed a new-onset, disseminated scalded skin-like rash.

Case Presentation
A 49-year-old woman with a past medical history of liver cirrhosis, chronic deep vein thrombosis on anticoagulation, chronic kidney disease and coronary artery disease, who initially presented to the hospital with alter mental status, nausea, lethargy and abdominal discomfort. On further examination, the patient appeared somnolent, minimally engaged in conversation and exhibited a generalized desquamating rash on the lower extremities. Additional work up, revealed an elevated serum ammonia level of 108 µmol/L, normal liver function tests, no leukocytosis and a creatinine level of 1.7 mg/dL. Patient was treated with rifaximin and lactulose, having bowel movements.

Despite appropriate treatment for hepatic encephalopathy, the patient clinical condition failed to improve, worsening with septic shock, requiring critical care admission, complicated by acute hypoxic respiratory failure, attributed to fluid overload in the context of hepatorenal syndrome, warranting mechanical ventilation and vasopressor support. Concurrently, there was marked progression of the desquamating rash, involving approximately 70% of the body surface area, characterized by a positive Nikolsky sign and absence of mucosal involvement. Empiric intravenous antibiotics were initiated, including clindamycin to inhibit toxin production and cefazolin to target staphylococcal infection. The patient underwent intermittent hemodialysis for fluid management and renal support.

A punch skin biopsy was performed, significant for superficial sub corneal vesicular dermatitis, consistent with SSSS. Further work up to rule out other causes, including desmoglein 1 and 3 was unremarkable. Over the following 2 weeks, the patient showed clinical improvement, she was successfully extubated, and vasopressor support was gradually weaned, transitioned to a general medical ward.

Conclusion
This case highlights the importance for clinical vigilance in immunocompromised adults presenting with unexplained skin desquamation and systemic symptoms. Although SSSS is rare in adults, it should remain a key differential diagnosis in at-risk populations such as those with liver cirrhosis or chronic kidney disease. Early recognition of SSSS is critical in prevent treatment delays and optimize clinical outcomes.

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BEAR IN MIND THE SILENT PAW: A CASE OF XANTHOGRANULOMATOUS PYELONEPHRITIS

Xanthogranulomatous pyelonephritis (XGP) is a rare, aggressive form of pyelonephritis caused by chronic obstruction and infection. It leads to progressive destruction of renal parenchyma, causing significant morbidity and mortality. We present a case to highlight this uncommon entity and raise clinical awareness.

A 66-year-old female with history of hypertension, COPD and type 2 diabetes mellitus, no chronic foley, presented with altered mental status and hypoxia, unable to obtain further history. On examination, vitals BP 157/54, HR 71, RR 20, O2 92% on 4L, T 98.2 F. Abdomen was noted to be distended, remainder of exam was unremarkable. Laboratories showed BUN 72 mg/dL, creatinine 3.54 mg/dL, WBC 19.3 10*3/uL; Urinalysis revealed significant pyuria, proteinuria, and elevated leukocyte esterase. CT abdomen/pelvis demonstrated severe right-sided hydronephrosis with a 9 x 9 mm mid-ureteral stone. The left kidney showed mild hydronephrosis, staghorn-appearing calcification and subtle striation with "bear pawâ€ appearance suggestive of XGP. She was hospitalized for sepsis secondary to UTI and respiratory failure and was placed on broad spectrum antibiotics. On hospital day 1, bilateral nephrostomy tubes were placed with purulent drainage. Urine cultures revealed ESBL Escherichia coli and MDR Proteus mirabilis; antibiotics were transitioned to meropenem on day 2. The rest of her hospital course was unremarkable; she was discharged with outpatient follow-up.

XGP is a chronic renal inflammatory disorder involving progressive parenchymal destruction replaced by lipid-laden foamy macrophages, called xanthoma cells. It often arises from recurrent UTIs and obstructive nephrolithiasis. Pro- and anti-inflammatory cytokines, including IFN-Î³, IL-2, TNF-Î±, IL-10, IL-23, and VEGF, perpetuate the inflammatory cycle. Annual incidence is 1.4 cases per 100,000 population, primarily affecting women aged 45-55. Major risk factors include obstructive nephrolithiasis, recurrent urinary tract infections, diabetes, hypertension, metabolic syndrome and rheumatoid arthritis. Typical symptoms include abdominal or flank pain, dysuria, gross hematuria, fever and a palpable abdominal mass. Proteinuria, elevated urine pH, ESR and CRP are common findings. Common pathogens involved are E. coli and P. mirabilis, however, diagnosis cannot be excluded in cases of sterile pyuria. CT is the imaging modality of choice, with findings such as hydronephrosis, renal calculi, cortical atrophy, and perinephric fat stranding. Large obstructive nephrolithiasis in the renal pelvis is classically termed the "bear pawâ€ sign, which is only present in 55% of cases. Bilateral involvement is uncommon. Initial management includes percutaneous drainage and IV antibiotics. Nephrectomy is preferred for extensive inflammation. If left untreated, abscess, adhesions, fistulas, renal perforation or sepsis can occur with mortality rate of 10%.

Clinicians should maintain a high index of suspicion for XGP in patients with recurrent UTIs and obstructive uropathy, especially when imaging shows renal calcifications and hydronephrosis. Early diagnosis and treatment are critical to preserve renal function and prevent further morbidity and mortality.

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Male breast cancer (MBC) is a rare malignancy comprising less than 1% of all breast cancers, often associated with BRCA2 mutations. These mutations not only predispose men to breast cancer but also significantly increase the risk of developing prostate ca

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Male Breast Cancer and BRCA2: A Hidden Risk for Prostate Cancer

"Male breast cancer (MBC) is a rare malignancy comprising less than 1% of all breast cancers, often associated with BRCA2 mutations. These mutations not only predispose men to breast cancer but also significantly increase the risk of developing prostate cancer. We report a case of a 56-year-old male with no personal or family history of cancer who was diagnosed with hormone receptor-positive MBC and found to carry a pathogenic BRCA2 mutation. Later, he was diagnosed with high-risk prostate adenocarcinoma. This case highlights the genetic and clinical interplay between MBC and prostate cancer and emphasizes the importance of universal genetic screening and long-term surveillance in male breast cancer patients, even in the absence of a known hereditary risk.

Case Presentation

A 56-year-old obese male presented with a 5-month history of a progressively enlarging, non-tender right breast mass. Imaging revealed a 57 x 59 x 62 mm retroareolar mass and an abnormal right axillary lymph node, BIRADS 5. Biopsy confirmed grade 2 invasive ductal carcinoma with positive staining for ER, AR, CK7, GATA3, and focal positivity for mammaglobin and GCDFP-15. Staging PET-CT showed FDG-avid breast and axillary lesions with no distant metastases. Genetic testing identified a heterozygous BRCA2 mutation. The patient underwent a right modified radical mastectomy and contralateral prophylactic mastectomy. Pathology showed grade 3 apocrine carcinoma with lymphovascular invasion and one positive axillary node (pN1a). He received ddAC-T chemotherapy, tamoxifen, radiotherapy, and Olaparib.

Subsequent PSA elevation (6.36 ng/mL) and BRCA2 status prompted prostate evaluation. MRI showed PI-RADS 5 lesions, and biopsy revealed Gleason 6 prostate adenocarcinoma in 3 of 12 cores. Six months later, imaging showed disease progression with extracapsular extension and lymphadenopathy. He began definitive radiotherapy and androgen deprivation therapy with leuprolide.

Discussion

This case underscores the strong association between BRCA2 mutations and multiple primary cancers in men. While BRCA2 mutations are known to elevate the risk of MBC and aggressive prostate cancer, the absence of family history in this patient emphasizes that such mutations can arise de novo or be missed due to incomplete family histories. Immunohistochemistry findings such as AR, PSA, and NKX3.1 expression in the breast tumor also suggest overlapping molecular pathways. This case highlights the need to expand BRCA genetic screening to all males diagnosed with breast cancer, regardless of age or family history, to ensure timely identification of hereditary cancer risk.

Conclusion

This case highlights the importance of universal genetic testing and routine prostate cancer surveillance in male breast cancer patients, especially those with BRCA2 mutations. Clinicians should remain vigilant, as early detection of synchronous or metachronous malignancies can significantly alter management and improve outcomes."

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CLOSTRIDIUM DIFFICILE IN SYNOVIAL FLUID : A RARE CASE OF EXTRAINTESTINAL SEPTIC ARTHRITIS

Clostridium difficile (C. difficile) is most commonly known for causing colitis and healthcare-associated diarrhea after recent antibiotic exposure. Extraintestinal infections such as bacteremia, osteomyelitis, and septic arthritis are rare and often occur in patients with significant comorbidities or recent surgical procedures. Here, we report a case of a patient with end-stage renal disease on hemodialysis and recent hip arthroplasty who developed C.difficile-associated septic arthritis. A 75-year-old female with a past medical history of end-stage renal disease on hemodialysis, hypertension, heart failure with preserved ejection fraction (HFpEF), and type 2 diabetes mellitus presented with right hip pain, weakness, and chills for three days. She had undergone a right hip hemiarthroplasty for a right subcapital femoral neck fracture four weeks ago. She was recently hospitalized for C. difficile bacteremia and discharged on outpatient intravenous vancomycin but developed these symptoms while still receiving treatment. The physical examination showed bilateral lower extremity edema, tenderness in the right hip, and limited range of motion. Computed tomography of the right lower extremity showed extensive irregular fluid and air extending from the right hip joint space surrounding the femoral stem with involvement of the surrounding gluteus musculature and lateral subcutaneous tissues concerning abscess in the setting of septic arthritis. Synovial fluid analysis revealed a white blood cell count of 53,350/µL with 93% neutrophils. Initial synovial fluid cultures showed no growth; however, subsequent prolonged incubation resulted in the development of C. difficile. Blood cultures were positive for C. difficile. Notably, she had no gastrointestinal symptoms, and stool testing for C. difficile toxin was negative. The diagnostic complexity of this case required the involvement of infectious disease, orthopedic surgery, and nephrology for collaborative management. The patient underwent irrigation and debridement of the right hip joint, including removal of the prosthesis and placement of an antibiotic cement spacer. She received six weeks of intravenous vancomycin and cefepime, coordinated with her dialysis schedule. She showed marked clinical improvement with the resolution of leukocytosis and was discharged to an inpatient rehabilitation unit. This case provides insight into the uncommon but significant potential of C.difficile to cause extraintestinal infections such as septic arthritis in vulnerable patients. Contributing risk factors included end-stage renal disease on hemodialysis, a prosthetic joint, prior antibiotic exposure, and advanced age. Diagnosing extraintestinal C. difficile infection is challenging due to its rarity, the need for prolonged culture incubation, the absence of gastrointestinal symptoms, and negative stool toxin testing. Management often requires both surgical intervention and systemic antimicrobial therapy, frequently without standard guidelines. This report highlights the importance of recognizing atypical presentations of C. difficile and maintaining clinical vigilance to ensure timely diagnosis and management.

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A Tailored Approach to Perioperative DAPT Management

Dual anti-platelet therapy (DAPT) is recommended for up-to 12 months after percutaneous coronary intervention (PCI) in ACS patients. However, urgent surgery shortly after PCI presents challenges due to the risks of stent thrombosis and perioperative bleeding.. We present a complex case highlighting multidisciplinary decision-making around perioperative DAPT management in the immediate post-PCI period.

Case Presentation:â€¨ A 55-year-old man with no known cardiac history presented unresponsive and hemodynamically unstable (BP 82/46 mmHg, HR 63 bpm, RR 10/min, T 95.5°F). Physical exam revealed pinpoint pupils and minimal responsiveness. He received IV naloxone and fluid resuscitation, resulting in temporarily improved mental status, though hypotension persisted, necessitating initiation of vasopressors. On awakening, he reported bilateral limb numbness and weakness. Initial labs showed elevated troponin 114 â†’ 78 pg/mL with delta of -36 pg/mL, CK 8000 U/L, AST 606, ALT 121, ALP 87, and COâ,, of 19 mmol/L. CT head without contrast was unremarkable, CTA head and neck showed hypoplastic vertebral artery, occluded in the proximal neck C6-7 level with reconstitution at C3- C4 level. EKG revealed ST-elevation myocardial infarction in the anterior leads. Bedside echocardiography confirmed severe hypokinesis in the left anterior descending (LAD) territory. Urgent coronary angiography identified significant stenoses in the LAD and right coronary artery, both of which were stented. DAPT with aspirin and ticagrelor was initiated. Post-extubation, he developed worsening paraplegia and urinary retention. MRI of the spine revealed severe multilevel cervical stenosis with cord compression and edema. Despite medical therapy with steroids, he required emergent cervical decompression and laminectomy two days post-PCI. Given recent stenting, a multidisciplinary team involving cardiology, neurology, anesthesiology, and spine surgery was convened to assess perioperative risks. DAPT was held 24 hours preoperatively, and the patient underwent C2-C6 decompression. Postoperatively, intravenous cangrelor was used for seven days due to its rapid onset/offset profile, allowing safer surgical recovery before transitioning back to oral DAPT. He remained free of in-stent thrombosis but had persistent quadriplegia and was discharged to a rehabilitation facility.â€¨This case underscores the complexities of DAPT management in the early post-PCI period amid unclear guidelines and the importance of individualized, multidisciplinary care. Cangrelor, a short-acting intravenous P2Y12 inhibitor, provided an effective bridge that minimized thrombotic risk while allowing surgical intervention with manageable bleeding risk.

Conclusion:

Emergent surgery following recent PCI presents a high-risk scenario with competing priorities. Bridging strategies like cangrelor may offer a valuable tool in mitigating risk, though further evidence is needed to guide standardized protocols. Multidisciplinary collaboration remains essential in navigating DAPT interruption and re-initiation.

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DOUBLE DRUG TOXICITY: A RARE CASE OF DAPTOMYCIN-INDUCED PNEUMONITIS AND CARBAPENEM-ASSOCIATED NEUROTOXICITY

Background

Daptomycin is an antibiotic that can have side effects of rhabdomyolysis or rarely daptomycin-induced pneumonitis (DIP). DIP most commonly occurs in males, patients over 65 years old, and drug exposure over two weeks. Additionally, carbapenems have a rare side effect of neurotoxicity, including symptoms of confusion and hallucinations. Carbapenems bind to GABA receptors. This prevents GABA-mediated central nervous system (CNS) inhibition, leading to increased CNS excitation. The concomitant presentation of two rare drug toxicities makes for a unique patient case.

Case Presentation

We present a 79-year-old male with a recent hospitalization for osteomyelitis of L4-L5 spine on a six-week course of daptomycin and ertapenem. He presented to the hospital on day 26 of antibiotics for confusion, tremors, dry cough, and hypoxia. The patient was afebrile, and oxygen saturations were 92-94% on 4L nasal cannula. The physical exam was remarkable for diffuse rhonchi, expiratory wheezing bilaterally, and no focal neurological deficits. Lab work was notable for leukocytes 9.8, lactic acid 0.7, Cr 0.7, normal liver enzymes, CRP 23, and procalcitonin 0.10. Fungitell and galactomannan were also negative. CT Head was unremarkable. CT Pulmonary Angiogram showed multifocal ground glass opacities (GGO) diffusely through the bilateral lungs as well as bilateral moderate pleural effusions. Per infectious disease recommendations, his antibiotics were switched to vancomycin and meropenem on admission for concern of multifocal pneumonia versus DIP. However, the patient never developed a fever, productive cough, or had leukocytosis, making DIP the more likely cause of the diffuse bilateral GGO. Ultimately, the family deferred bronchoscopy for definitive diagnostic testing. On day five of the hospitalization, he developed profound confusion, hallucinations, and myoclonic jerks. CT Angio Head showed no acute pathology. Electroencephalogram (EEG) showed generalized encephalopathy but no evidence of seizures. Meropenem was discontinued for concerns for neurotoxicity, and ceftriaxone was started. The patient had improvement of his myoclonus and mental status within 48 hours. Due to the hallucinations, steroids were never initiated to treat his pneumonitis. His oxygenation improved slowly over two weeks, and he was discharged on room air.

Discussion

This case highlights two rare side effects of commonly used antibiotics. A DIP diagnosis can be made clinically. However, the definitive diagnosis is a bronchoalveolar lavage (BAL) that shows greater than 25% eosinophils in the fluid. Carbapenem-associated neurotoxicity can be diagnosed once other more common diagnoses are excluded, including seizure, stroke, delirium, and infectious encephalopathy. While it is hard to initially distinguish drug toxicities from other disease processes, the key is improvement of symptoms after discontinuation of the drug. The simultaneous presentation of these toxicities, despite normal liver and kidney function, suggests the need for close monitoring for side effects, especially in the elderly.

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When the Mouth Strikes the Brain: Streptococcus gordonii Endocarditis Leading to Cerebral Mycotic Aneurysm

While neurological complications from infective endocarditis (IE) are well described, it is uncommon for a young, previously healthy adult to present with an intracerebral hemorrhage (ICH) as the initial manifestation. Early recognition of this link is crucial for effective treatment.

A 29-year-old man with no significant medical history presented to the emergency department with a severe headache persisting for five days, accompanied by fever, vomiting, and blurry vision. Initial vital signs were stable. Laboratory tests revealed mild hyponatremia (Na 134), INR of 1.2, and an unremarkable CBC. Brain imaging showed a large left parieto-occipital hemorrhage with edema, but no clear etiology. Cerebral angiography revealed a pseudoaneurysm of the left posterior cerebral artery, which was successfully embolized.

Despite this intervention, the patient continued to experience severe headaches and developed a right homonymous hemianopsia. Blood cultures grew Streptococcus gordonii. Subsequent echocardiography revealed a large vegetation on the mitral valve, consistent with subacute bacterial endocarditis likely originating from periodontal disease as shown by CT imaging of the maxilla. The patient underwent dental extraction and was started on intravenous antibiotics.

His hospital course was complicated by SIADH-induced hyponatremia and a severe allergic reaction to antibiotics, requiring changes to his regimen and treatment with topical steroids for a diffuse rash. Serial brain imaging demonstrated stable hematoma and midline shift, while repeat echocardiography showed a decrease in vegetation size, eliminating the need for surgical intervention. The patient was ultimately managed medically with favorable neurological and cardiac recovery.

This case highlights the importance of considering IE in young patients presenting with unexplained ICH, especially when septic emboli or atypical organisms are involved. Mycotic aneurysms, although rare, are potentially life-threatening complications of endocarditis and require a high index of suspicion for diagnosis. Successful management in this case required multidisciplinary coordination among cardiology, neurology, neurosurgery, infectious disease, and critical care teams. Early aneurysm intervention and timely treatment of the infection and its complications were critical to the patient's recovery.

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Rodents, Rain and Risk: A Case of Locally Acquired Leptospirosis in New York City (NYC)

INTRODUCTION: Leptospirosis is a global zoonotic disease caused by the bacterium Leptospira found in contaminated water or soil and is typically spread to humans through the urine of infected rodents. The are two clinical forms: anicteric and icteric. The anicteric form accounts for more than 90% of cases and manifests as a flu-like illness. The icteric form accounts for 5-10% of cases and typically follows a biphasic course including the initial septicemic phase followed by the immune phase. Pulmonary involvement is an independent predictor of mortality with pulmonary hemorrhage associated with a fatality rate of 35-55%. We present an atypical case of leptospirosis in a NYC resident with no travel history or direct animal exposure.

CLINICAL PRESENTATION: A 40-year-old male with no pertinent past medical history presented with six days of dry cough, fevers, malaise, and diffuse headache. He lives with his wife and three vaccinated, shelter-adopted cats. He denied recent freshwater exposure, outdoor activities including hiking, tick bites, or contact with ill individuals or animals. The only exposures of potential concern were the presence of mouse droppings in his apartment building and mold contamination in the central air conditioning system. On presentation, vitals were notable for high-grade fever and sinus tachycardia. The exam revealed bibasilar crackles in the lungs and diffuse myalgias. Labs showed new-onset anemia, thrombocytopenia, rhabdomyolysis, and acute kidney and liver injury, with previously normal labs three months prior. The urinalysis was bland. Computed Tomography (CT) of the Chest revealed bilateral peri-bronchial diffuse airspace opacities. The constellation of systemic symptoms, lab abnormalities, and imaging findings raised concern for atypical infections. Empiric doxycycline and ceftriaxone were initiated. The patient improved clinically and was discharged. Leptospira IgM serology was positive and confirmed on repeat testing, supporting a recent leptospirosis infection.

DISCUSSION: Leptospirosis is an emerging public health threat in NYC; with a record 24 cases reported in 2023 aloneâ€”the highest in any single year. For comparison, from 2001-2020 there were 3 cases per year. In NYC, rodent exposure is a significant public health concern, with a rodent population surpassing 3 times that of the city's human population. Leptospirosis transmission increases in warm, humid climates and our patient's exposure likely occurred in May 2025 which was unseasonably wetter and warmer than average. These conditions were further exacerbated by the patient's malfunctioning air conditioning system. The increasing rodent population and changing climates mirror the continued upwards trend of leptospirosis in NYC.

CONCLUSION: This case underscores that urban leptospirosis should be considered even in patients without traditional risk factors, particularly in NYC, where increasing rodent populations, aging infrastructure and climate change may create indirect transmission pathways.

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ADDRESSING CHALLENGES IN TRACKING CONTINUITY OF CARE IN THE RESIDENCY CLINIC WITH THE RECOCA APP

Introduction

Continuity in patient-provider relationships is a key aspect of primary care, referring to the regularity with which patients consistently see the same healthcare providers over time. High patient-provider continuity is associated with increased patient satisfaction, improved health outcomes, and reduced healthcare utilization. Despite its importance, achieving and measuring continuity is challenging in residency clinics due to scheduling challenges and high provider turnover. Continuity rates among resident clinics are low, with an average Usual Provider of Care (UPC) index for internal medicine programs at 58.5%. While UPC is the most commonly reported metric, systematic reviews identify as many as 32 unique indices used across studies. Calculating indices like the Bice-Boxerman (BB-COC), Modified Modified Continuity Index (MMCI), and Sequential Continuity of Care (SECON) are non-trivial. Standardized, accessible calculation tools are unavailable. Processing the necessary longitudinal data from sources like claims or electronic health records (EHRs) currently requires the use of statistical software packages or tools. This analytical burden hinders consistent tracking and improvement efforts for care continuity.

Methods

We initiated a quality improvement project to enhance continuity of care in our internal medicine residency clinic. Our initial discovery phase focused on establishing baseline continuity metrics and developing methods for sustainable tracking. We extracted appointment data from our scheduling software and initially calculated the UPC index using Excel. This manual approach revealed significant operational challenges: calculations were time-intensive, susceptible to human error, and required complete recalculation with each update of appointment data â€” limitations that would impede ongoing monitoring of improvement interventions.

To address these challenges, we developed RECOCA (REsident Continuity Of Care App), an open-source desktop application that automates the calculation of commonly used continuity indices. Built to be EHR-agnostic and accessible to non-technical users, RECOCA accepts longitudinal appointment data in Excel format. Users select relevant fields (patient ID, provider ID, appointment date), after which the application calculates four validated continuity metrics: UPC, BB-COC, MMCI, and SECON. The application also generates an interactive dashboard displaying clinic-wide summary statistics and provides modules for searching individual patients and reviewing provider visit histories. RECOCA was designed to reduce analytic burden and support routine use in program-level quality improvement efforts.

Results

Using RECOCA, we calculated baseline continuity indices for our residency clinic: UPC, 52.6%; BB-COC, 23.8%; SECON, 28.8%; and MMCI, 38.5%. These indices were derived from appointment data spanning January 2022 to August 2024, encompassing 4637 unique patients and 124 residents.

Conclusion

RECOCA provides an open-source, user-friendly tool for calculating continuity indices in outpatient clinics. By automating these calculations independent of proprietary systems, it enables effective routine monitoring and evaluation of continuity improvement interventions. We will use RECOCA to guide interventions to improve continuity in our clinic.

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Case report of Anti-HMGCR antibody-positive necrotizing myopathy occurring more than 1.5 years after initiation of statin therapy

Background: Necrotizing autoimmune myopathy (NAM) is a rare subtype of idiopathic inflammatory myopathies, characterized by severe proximal muscle weakness and elevated muscle enzymes. Among its etiologies, anti-HMGCR antibody-positive myopathy is notably associated with statin use but can manifest long after drug cessation.

Case Presentation: We report a case of a 57-year-old male who developed progressive proximal muscle weakness and markedly elevated creatine phosphokinase (CPK) levels, approximately eight months after discontinuing atorvastatin, which he had used for over 1.5 years. Initial presentation included elevated liver enzymes and myalgia. Physical examination revealed proximal limb weakness and positive Gower's sign. Laboratory studies confirmed rhabdomyolysis and elevated inflammatory markers. MRI of the thighs demonstrated diffuse muscle edema consistent with myositis. Myositis-specific autoantibody panel was positive only for anti-HMGCR antibodies. Muscle biopsy revealed necrosis and regeneration without significant inflammatory infiltrate, confirming the diagnosis of anti-HMGCR antibody-positive necrotizing myopathy. Notably, EBV and Coxsackie virus antibodies were also positive, suggesting a potential viral trigger.

The patient was treated with intravenous immunoglobulin (IVIG), high-dose corticosteroids, rituximab, and later transitioned to mycophenolic acid and upadacitinib. Progressive clinical and biochemical improvement was observed over eight months, with normalization of upper limb strength and significant CPK reduction.

Conclusion: This case underscores the potential for delayed onset of anti-HMGCR antibody-positive NAM following statin cessation and highlights the possible contributory role of viral infections. A high index of suspicion and comprehensive workup are crucial for timely diagnosis and initiation of immunosuppressive therapy, which is essential for favorable outcomes in NAM.

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A CASE OF ACUTE EPSTEIN-BARR VIRUS HEPATITIS MASQUERADING AS CHOLECYSTITIS

Introduction: Right upper quadrant pain accompanied by elevated liver enzymes frequently raises suspicion for hepatobiliary disease, particularly gallstone-related pathology such as acute cholecystitis. Rarely, viral infections like Epstein-Barr virus (EBV), can present with similar findings, mimicking cholecystitis with cholestatic hepatitis. This case report describes a 61-year-old female who presented with symptoms and laboratory findings initially suggestive of acute cholecystitis who was ultimately found to have EBV induced hepatitis. This case highlights the importance of considering EBV infection in the differential diagnosis of patients presenting with right upper quadrant pain and elevated liver enzymes.

Case Presentation: A 61-year-old female with a history of untreated hypothyroidism presented with generalized abdominal pain, fatigue, and constipation following recent antibiotic treatment (amoxicillin, later Augmentin and a Medrol dose pack) for a dental infection and presumed sinusitis. Her abdominal pain progressed, localizing to the lower abdomen with radiation to the back, and became severe. Notably, she exhibited no fever, jaundice, or right upper quadrant tenderness,. Laboratory studies revealed a cholestatic pattern of elevated liver enzymes, but no additional abnormal findings. She underwent abdominal ultrasound and was found to have cholelithiasis with questionable gallbladder wall thickening and a positive sonographic Murphy's sign. CT imaging revealed chronic constipation with proctitis and mild hepatosplenomegaly. She was admitted for further evaluation of possible choledocholithiasis vs early acute cholecystitis. Given the absence of fever or leukocytosis, antibiotic therapy was deferred. On hospital day 2, she underwent MRCP and was found to have mild hepatosplenomegaly, periportal edema, and a small pancreatic cyst without evidence of choledocholithiasis or cholecystitis. That evening, she became febrile, prompting treatment with empiric antibiotics for possible ascending cholangitis. Persistent symptoms and worsening liver tests necessitated further infectious disease evaluation. Serology revealed positive EBV IgM, while hepatitis panels (A, B, and C) and tick panel were negative. The patient's presentation was attributed to acute EBV infection. Antibiotics were discontinued, and supportive care resulted in her clinical improvement.

Discussion: This case highlights the diagnostic importance of casting a wide differential when considering patients presenting with symptoms suggestive of cholecystitis, even in patients with pre-existing risk factors for gallbladder disease. EBV can cause cholestatic hepatitis with RUQ pain, hepatosplenomegaly, fatigue, and transaminitis. In this patient, initial findings of cholelithiasis and possible gallbladder inflammation were a red herring. Once MRCP excluded these, EBV serologies confirmed viral hepatitis. This case highlights the importance of avoiding premature diagnostic closure. While imaging plays a crucial role in patient evaluation, misinterpretation or overreliance on findings can be misleading. Consideration of broad differentials, particularly in cases with atypical presentations, can prevent unnecessary interventions and facilitate timely, appropriate management.

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A ROADBLOCK ON THE WEIGHT LOSS JOURNEY: A CASE OF TRANSIENT GASTRIC OUTLET OBSTRUCTION

Introduction: Rapid, intentional weight loss can precipitate gastric outlet obstruction (GOO), sometimes due to Superior Mesenteric Artery (SMA) Syndrome. This report presents the unusual case of an 18-year-old male with GOO symptoms following significant weight loss, whose initial imaging findings were highly suggestive of SMA syndrome. Intriguingly, the patient experienced an unexpectedly rapid resolution of the obstruction following nasogastric decompression, raising questions about the definitive diagnosis and highlighting the potential for other more transient GOO syndromes mimicking SMA syndrome, such as gastroparesis, pylorospasms, or even intussusception.

Case Presentation: An 18-year-old male presented with two days of worsening epigastric pain, bloating, nausea, vomiting, and diarrhea after intentionally losing 25 pounds over two months. He denied fever, chills, bloody stools, or prior abdominal issues. Examination revealed only mild, diffuse abdominal tenderness. Initial labs showed mild leukocytosis with neutrophilia predominance and mildly elevated lipase, with otherwise normal results, including liver function tests and urinalysis (except for trace ketones). Influenza, RSV, and COVID-19 tests were negative. Abdominal X-ray was non-specific. A CT scan, performed for suspected appendicitis, revealed a distended stomach and duodenum with a transition point at the third portion, suggesting SMA syndrome. NG tube decompression provided immediate relief and over one liter of output. Repeat CT post-decompression demonstrated complete resolution of the distension. The surgical service assumed care for ongoing management and dietary counseling. The patient remained stable without needing surgery. While initial findings pointed towards SMA syndrome, the swift resolution of the obstruction challenged this diagnosis. The rapid improvement suggests a more transient process, making alternative diagnoses such as pylorospasm, gastroparesis, or even intussusception more likely.

Discussion: SMA syndrome, a rare cause of duodenal obstruction, typically requires weeks to months of treatment, ranging from specialized nutritional support to surgical intervention. This patient's rapid resolution following NG tube decompression, confirmed by a repeat CT scan, is atypical for SMA syndrome. This case underscores the complexity of diagnosing gastric outlet obstruction (GOO), highlighting the importance of considering alternative diagnoses and the potential for transient obstructions, especially in the context of recent weight loss.

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The Pleural Plot Twist: Positive ADA and Neutrophilic Effusion in Early Seropositive Rheumatoid Arthritis

Introduction

Rheumatoid arthritis (RA) is a systemic autoimmune disease best known for its symmetric inflammatory polyarthritis but may also involve extraarticular organs such as the pleura.¹ Although pleural effusions occur more frequently in patients with longstanding RA, they can occasionally serve as the initial manifestation. Rheumatoid pleuritis typically presents with a lymphocyte-predominant exudate characterized by low glucose and elevated lactate dehydrogenase (LDH). However, in early RA, pleural fluid may instead appear neutrophil-predominant with elevated adenosine deaminase (ADA), closely mimicking infectious etiologies and complicating timely diagnosis. We describe a diagnostically challenging case of new-onset RA in which an evolving exudative pleural effusion was the first clinical clue, emphasizing the importance of considering autoimmune causes in atypical fluid profiles.

Case Presentation

A 44-year-old woman with no prior medical history presented with progressive dyspnea over two weeks. On further review, she endorsed a six-week history of joint stiffness and pain involving the wrists, hands, and ankles. Physical examination revealed subcutaneous nodules over the elbows, synovitis of the small joints, and decreased breath sounds at the left lung base. Laboratory testing demonstrated leukocytosis and elevated inflammatory markers, including a C-reactive protein of 197.9 mg/L and erythrocyte sedimentation rate of 95 mm/hr. Serologic studies revealed high-titer rheumatoid factor (119 IU/mL; reference â‰¤14) and markedly elevated anti-cyclic citrullinated peptide (379 U/mL; reference â‰¤16.9), consistent with seropositive rheumatoid arthritis. Imaging revealed a large loculated pleural effusion with a split-pleura sign.

Initial thoracentesis yielded exudative pleural fluid with low glucose (18 mg/dL), high LDH (3561 U/L), ADA 57 U/L, and neutrophil predominance (64%). Infectious and malignant workup, including acid fast bacilli testing, QuantiFERON, cultures, and cytology, was negative.

Repeat thoracentesis one week later demonstrated lymphocyte predominance (54%). With evolving pleural fluid characteristics and highly specific RA serologies, a diagnosis of rheumatoid pleuritis was established. The patient was treated with prednisone and methotrexate, resulting in rapid improvement in both respiratory and joint symptoms. Follow-up imaging confirmed resolution of the effusion, and her disease remains in remission on maintenance therapy.

Discussion

This case illustrates seropositive rheumatoid arthritis initially presenting with pulmonary manifestations. Neutrophilic effusion with elevated ADA typically prompts infectious evaluation, but RA must remain on the differential, particularly with positive autoimmune markers and lack of microbial growth. Pleural

fluid in RA may evolve over time, from neutrophilic to lymphocytic predominance, reflecting a dynamic inflammatory process. Recognizing these patterns can prevent misdiagnosis, delays in treatment, and unnecessary antimicrobial therapy. Early initiation of immunosuppressive therapy can lead to full clinical resolution and prevent long-term pulmonary complications such as fibrosing pleuritis or interstitial lung disease.

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The Challenges of Diagnosing Presumptive Adult-Onset Still's Disease in an Elderly Patient and Recognition of Acute Comorbidities Necessitating Anticoagulation: Ending in Fatal, Retroperitoneal Hemorrhage

Background:â€¯

Adult-Onset Still's Disease (AOSD) is a rare systemic inflammatory disorder characterized by high spiking fevers, transient rash, and arthritis. Diagnosis is one of the exclusions and is challenging in elderly patients.â€¯ During prolonged and complex hospitalization, initiation of anticoagulation for two newly diagnosed comorbidities: deep vein thrombosis and new-onset atrial fibrillation that eventually led to an acute retroperitoneal hemorrhage, resulting in fatal shock.

Purpose:â€¯

To highlight differentiating AOSD from sepsis and systemic inflammatory response syndromes (SIRS) in elderly patients with confounding, complex, comorbidities. To remind clinicians that when appropriate anticoagulation is initiated for two new unrelated comorbidities, retroperitoneal hemorrhage may be massive and lead to fatal shock.

Case Description:

An elderly female presented with high-grade quotidian fevers (103.2°F), leukocytosis (20K), anemia (Hgb 9.5), hypercalcemia (Ca 11.5), with normal renal and Liver function tests inflammatory markers were markedly elevated including ferritin (1857 ng/mL), CRP (74 mg/L), ESR (62 mm/hr.), and LDH (1315 U/L), without an M-spike (see Table 1). Despite empiric broad-spectrum antibiotics, she remained febrile with progressive lethargy (see Timeline fever). Initial extensive Infectious workup was negative (see Table 2). Autoimmune and malignancy workups revealed an elevated ANA and questionable low-grade CLL/SLL on bone marrow biopsy, but rheumatology thought history, physical and exam and other negative serologies ruled out SLE imaging evaluation is detailed in Table 3, which revealed mild splenomegaly.

Persistently elevated inflammatory markers, double quotidian fevers, evanescent rash, and arthralgias supported a possible diagnosis of adult-onset Still's disease (AOSD) and empiric high-dose corticosteroids led to improvement in clinical and laboratory parameters.â€¯ However, on her fifth day of steroid therapy, hospital day 27 and just prior to a planned discharge, she developed new-onset atrial fibrillation with RVR, and a left femoral DVT. Anticoagulation with full-dose enoxaparin was initiated that led to a retroperitoneal hemorrhage from a hidden psoas hematoma, causing hemorrhagic shock and death despite emergent blood transfusion and IR embolization.â€¯

Conclusion:

Our case demonstrates two important teaching moments:

1. A possible diagnosis of AOSD is challenging in an elderly patient with confounding comorbidities due to its nonspecific presentation and overlap with sepsis, malignancy, and other inflammatory or autoimmune conditions. Although AOSD typically presents in younger adults, cases in older individuals have been increasingly recognized, often with atypical features and longer diagnostic delays.

2.While anticoagulation is essential for stroke and thromboembolism prevention, particularly in the setting of DVT and high CHAâ,,DSâ,,-VASc scores, it may carry significant bleeding risk such as RP bleeding, perhaps exacerbated in the context of systemic inflammation, steroid use, or frailty.

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The Challenges of Diagnosing Adult-Onset Still's Disease in an Elderly Patient, and Recognition of Acute New Comorbidities Necessitating Anticoagulation, Ultimately Complicated by Fatal Retroperitoneal Hemorrhage

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Background:â€¯

Adult-Onset Still's Disease (AOSD) is a rare systemic inflammatory disorder characterized by high spiking fevers, transient rash, and arthritis. Diagnosis is one of the exclusions and is challenging in elderly patients.â€¯

Purpose:â€¯

To highlight differentiating AOSD from sepsis and systemic inflammatory response syndromes (SIRS) in elderly patients with confounding and complex comorbidities, and to remind clinicians that when appropriate anticoagulation is initiated, retroperitoneal hemorrhage may be massive and lead to fatal shock.

Case Description:

An elderly female presented with high-grade quotidian fevers (103.2°F), leukocytosis (20K cells/microliter), anemia (Hgb 9.5 g/dl), hypercalcemia (Ca 11.5 mg/dl), with normal creatinine 0.8 mg/dl and normal liver function tests, inflammatory markers were markedly elevated including ferritin (1857 ng/mL), CRP (74 mg/L), ESR (62 mm/hr.), in addition, Lactate dehydrogenase was elevated (1315 U/L) protein electrophoresis didn't show M-spike (see Table 1). Despite empiric broad-spectrum antibiotics, she remained febrile with progressive lethargy (see Timeline fever). Initial extensive Infectious workup including tuberculosis was negative (see Table 2). Autoimmune and malignancy workups revealed an elevated ANA and questionable low-grade CLL/SLL on bone marrow biopsy, however SLE was felt to be unlikely. Imaging evaluation is detailed in Table 3; Mild splenomegaly was noted.

Persistently elevated inflammatory markers, double quotidian fevers, evanescent rash, and arthralgias supported a possible diagnosis of adult-onset Still's disease (AOSD) and empiric high-dose corticosteroids led to improvement in clinical and laboratory parameters.â€¯ However, on her fifth day of steroid therapy, hospital day 27 and just prior to a planned discharge, she developed new-onset atrial fibrillation with rapid ventricular response and left femoral deep vein thrombosis. Her CHAâ,,DSâ,,-VASc score was 3 and HAS-BLED score was 2. Full-dose enoxaparin was initiated for anticoagulation; however, this led to a retroperitoneal hemorrhage due to a concealed psoas hematoma, resulting in hemorrhagic shock and death despite emergent blood transfusions and interventional radiology-guided embolization.

Conclusion:

Our case demonstrates two important teaching moments: 1. A possible diagnosis of AOSD is challenging in an elderly patient with confounding comorbidities due to its nonspecific presentation and possible overlap with sepsis, malignancy, and other inflammatory or autoimmune conditions. Although AOSD typically presents in younger adults, cases in older individuals have been increasingly recognized, often with atypical features and longer diagnostic delays.

2.â€¯Anticoagulation is vital for preventing stroke and thromboembolism in patients with DVT and high CHAâ‚‚DSâ‚‚-VASc scores but carries a significant bleeding risk, including retroperitoneal hemorrhage. This risk increases with systemic inflammation, steroid use, and frailty. Careful risk assessment is crucial, especially when starting anticoagulation late in a prolonged and complex hospitalization.

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Uncommon Neurological Manifestation of Ranolazine: A Case of Drug-Induced Myoclonus

One of the cornerstones of treatment in chronic stable angina is ranolazine, which is commonly prescribed either as a primary agent or in combination with other agents including beta-blockers and nitrates. A sparsely reported side effect of ranolazine is myoclonus. Here, we report a case of myoclonus in a 79-year-old male who was started on ranolazine for refractory angina.

Patient with a past medical history of coronary artery disease with triple vessel disease, percutaneous intervention to RPDA and RPL, ischemic cardiomyopathy with HFmrEF (EF 40%), chronic kidney disease stage V, cerebrovascular accident, and dementia presented to the hospital with two hours of retrosternal chest pain at rest. The pain resolved after taking sublingual nitroglycerin en route to the hospital. On arrival, the patient was hypertensive to 179/100, afebrile, and hypoxic. Labs were notable for creatinine 4.0 mg/dL, high sensitivity troponin 96 ng/L (eventually peaked at 871 ng/L), N-terminal pro-B-type natriuretic peptide 23,281 pg/mL. Electrocardiogram demonstrated normal sinus rhythm with ST-depressions in leads II, III, aVF, and V6, ST-elevations in aVL and V2, and T-wave inversions in V5-V6; these findings were consistent from those on previous ECG one month prior. Given concern for acute decompensated heart failure and pulmonary edema, patient was admitted to the cardiac intensive care unit, placed on bilevel positive airway pressure, given 80 mg IV of furosemide, and started on nitroglycerin drip.

He underwent cardiac catheterization, which was significant for multi-vessel disease with 50% left main involvement. Cardiothoracic surgery was consulted for consideration for coronary artery bypass surgery; however, the patient ultimately elected medical management. The patient continued to experience angina and was started on ranolazine as a new medication at 500 mg twice daily and increased to 1000 mg twice daily the following day. After this increased dosage, the patient was noted to have worsening renal function with creatinine up to 6 mg/dL from 4mg/dL on presentation (baseline 2.5). After nearly 48 hours after the dose increase, the patient began experiencing full body myoclonus. Routine electroencephalogram was performed, without any seizures or epileptiform activity noted. MRI brain with and without contrast was negative for acute infarction. In discussion with neurology, the myoclonic activity was ultimately deemed secondary to ranolazine. On further review, no other active medications were known to cause myoclonus. Ranolazine was discontinued and the patient was given clonazepam 0.5 mg every 8 hours with resolution of myoclonus.

Myoclonus is not a well-known side effect of ranolazine. Ranolazine is metabolized by CYP3 enzymes primarily, which results in multiple drug-drug interactions. Furthermore, ranolazine is known to block persistent sodium channels in the nervous system, possibly leading to myoclonus. This case highlights that clinicians must be prudent of adverse effects with initiation of any new drugs.

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Score to Protect: Enhancing Anticoagulation in Atrial Fibrillation

In December 2024, a retrospective review revealed that among 137 patients admitted to the Telemetry unit with atrial fibrillation (AFib), 99 (72%) were anticoagulated with apixaban. Of these, 15% received an inappropriate dose based on established criteria involving age, renal function, and weight. This prompted the development of a quality improvement (QI) initiative to optimize apixaban dosing using a Clinical Decision Support System (CDSS).

To increase the rate of appropriate apixaban dosing by 50% within three months through the implementation of a CDSS embedded in a standardized telemetry admission note template. The CDSS was designed to guide clinicians by incorporating apixaban dosing criteria and automatically prompting appropriate adjustments during patient documentation.

The CDSS was developed after data extraction using Epic SlicerDicer and underwent multiple layers of review and approval, including Cardiology Clinical Leadership, Clinical Documentation, and Quality Assurance teams. Once approved, it was embedded into the telemetry unit's standardized admission/progress note to ensure consistent use. A smart data-tracking element within the CDSS allowed monitoring of note completion and dosing accuracy.

From April 11 to May 16, 2025, a total of 239 patients had completed telemetry notes with the embedded CDSS. Among these, 32.2% had documented AFib. Of those, 87% were anticoagulated, and 88% of those received apixaban. Only 6.77% of apixaban-anticoagulated patients were improperly dosed, suggesting improvement from the baseline 15%.

Comparing two groupsâ€”those before CDSS implementation (Group 1) and those with CDSS (Group 2)â€”improper dosing dropped from 12.12% (12/99) to 6.78% (4/59). Fisher's Exact Test yielded a p-value of 0.168, indicating no statistically significant difference ($p > 0.05$), though the odds of improper dosing in Group 1 were higher (OR: 1.90, 95% CI: 0.58-6.23). These findings suggest a trend toward improved dosing accuracy, though further data is needed to confirm significance.

Interim analysis during PDSA Cycle 1 revealed two key areas for improvement. First, the need for additional education of resident physicians regarding apixaban dosing protocols. Second, refinement of the data collection tool to include only patients with confirmed atrial fibrillation, as unrelated CDSS note entries may confound the results.

This quality initiative is ongoing, and future cycles will incorporate feedback to enhance the precision and effectiveness of the CDSS. With continued education and process refinement, we anticipate further reductions in apixaban dosing errors in AFib patients on the Telemetry unit.

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RARE CASE OF CLOZAPINE-INDUCED MYOCARDITIS

Background

Clozapine-induced myocarditis is a diagnosis of exclusion, manifesting only in rare circumstances. We describe a case of acute decompensated heart failure unraveling a diagnosis of clozapine-induced myocarditis.

Clinical Vignette

A 50-year-old man with schizophrenia and active polysubstance use disorder presented to the emergency department for hallucinations and disorganized behavior and was subsequently admitted to the inpatient psychiatry service. He was initiated on clozapine in the setting of treatment-resistant psychosis with rapid dose uptitiration from 25mg up to 200mg. He was discharged after ten days.

The patient re-presented to the emergency department one day after discharge for new complaints of profuse diarrhea accompanied by fevers to 39.3C. He denied chest pain, palpitations, or dyspnea. Vitals showed a blood pressure of 97/62 mmHg, heart rate 128 beats per minute, and oxygen saturation 95% on room air. Computerized tomography (CT) of his abdomen and pelvis was performed, which was unrevealing for acute pathology. Initial high-sensitivity troponin (hs-Tn) was 36 ng/L (normal â‰¤22 ng/L) and peaked at 240 ng/L. EKG revealed sinus tachycardia without acute ischemic changes. Other laboratory values were significant for a potassium level of 3.4 mmol/L (normal 3.5 - 5.1 mmol/L), phosphorus of 0.9 mg/dL (normal 2.7 - 4.5 mg/dL), D-dimer of 1,467 ng/mL (normal < 230 ng/mL), and pro-B-type natriuretic peptide of 28,400 pg/mL (normal <300 pg/mL). Echocardiogram revealed new-onset moderate left ventricular global hypokinesis with an ejection fraction of 40%. He was admitted to medicine for further management.

Cardiology was consulted and recommended diuresis given acute decompensated heart failure and obtaining a cardiac MRI to further characterize the cardiomyopathy. Clozapine was held given suspicion for its potential role as the culprit of possible myocarditis. Cardiac MRI was non-diagnostic due to motion artifact. Regadenoson stress test was performed which was equivocal for ischemia. Plans for inpatient cardiac catheterization were deferred given concern for patient's adherence to medication. Infectious workup throughout the admission was unrevealingly negative which included eight blood cultures, two malaria/babesia smears, an ova and parasites stool culture, GI pathogen panel, tick-borne panel, and infectious myocarditis work-up.

Discussion

This case is consistent with the average 10-to-22-day onset time of myocarditis from initiation of Clozapine. The most common starting dose in this demographic was Clozapine 12.5mg and titrated up an end-dose of 100mg to 550mg in several documented cases (1).

It is important to note that due to poor compliance with inpatient imaging and inability to perform cardiac catheterization, circumstances allow for clinical diagnosis but not for definitive diagnosis.

Conclusion

On suspension of the clozapine, the patient's fevers resolved, his troponins downtrended, and he was discharged with plan for left heart catheterization. The patient was lost to follow-up after discharge.

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CAROTID SINUS SYNDROME DUE TO OROPHARYNGEAL CARCINOMA

Background

Carotid sinus dysfunction is a common cause of syncope in the elderly and is typically due to atherosclerotic disease or senile autonomic dysfunction. Here, we present an uncommon cause of carotid sinus disease secondary to mass effect from an invading neoplasm.

Clinical Vignette

A 73-year-old male with a history of alcohol use disorder and a 100 pack-year history as a former smoker presented to the emergency department with a leg injury following a syncopal episode at home. The patient reported multiple syncopal episodes over the past three months, which led to the implantation of an implantable loop recorder at an outpatient site. Initial vital signs, including orthostatic vitals, and laboratory work were within normal limits. CT chest imaging revealed a right-sided oropharyngeal mass encasing the right common carotid artery, with additional masses in the left nasopharynx, left sphenoid/cavernous sinus, left pterygopalatine fossa, skull base foramina, left superior orbital fissure, left orbital apex, and bilateral neck lymph nodes. A right tonsillar biopsy showed squamous cell carcinoma.

His hospital course was complicated by repeated episodes of unresponsiveness associated with bradycardia (HR 41) and hypotension (BP 55/36). Initial symptomatic treatment with fluids and discontinuation of metoprolol increased both blood pressure and heart rate. The patient was started on high-dose steroids to address left-sided mydriasis, ptosis, and left facial numbness caused by the oropharyngeal mass. However, episodes of symptomatic bradycardia and hypotension persisted. His loop recorder documented an isolated three-second sinus pause, a right bundle branch block, several transient atrial fibrillation episodes, and a first-degree atrioventricular block since admission. Cardiology consultants proposed pacemaker implantation.

After the third inpatient episode of symptomatic bradycardia and hypotension, MRI of the head and neck showed a soft tissue density invading the parapharyngeal, masticator space, and right external carotid artery with necrotic lymphadenopathy. Pacemaker placement was deferred following a goals-of-care conversation with the primary and cardiology teams. Given the worsening hemoptysis from the oropharyngeal carcinoma and poor overall prognosis, the patient and family agreed to pursue hospice care, including seven courses of palliative radiation to the head and neck.

Discussion

The cause of this patient' carotid sinus syncope was irreversible, frequent, associated with a significant vasodepressor response and subsequent syncope. Standard of care would be permanent pacemaker implantation. Pacemaker placement is a class IIa indication based on both American and European

cardiology guidelines for patients greater than 40 years of age with recurrent carotid sinus related syncopal events (1).

Conclusion

This case illustrates carotid sinus syndrome resulting from compression of the carotid sinus by a neoplasm, triggering an overactive reflex and strong efferent vagal response, leading to symptomatic sinus bradycardia.

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Navigating the Glucose Surge: A Case Report of Hyperglycemia-Triggered Dysphasia and Trigeminal Neuralgia

Introduction
Uncontrolled diabetes mellitus can precipitate neurological complications, such as seizures, altered mental status, and chorea, typically associated with diabetic ketoacidosis (DKA) or hyperglycemic hyperosmolar state (HHS) [1-2]. We present a rare case of hyperglycemia-induced diabetic neuropathy manifesting as transient dysphasia, followed by trigeminal neuralgia, potentially caused by treatment-induced neuropathy of diabetes (TIND) after rapid glycemic correction. This report elucidates the clinical presentation, differential diagnosis, and therapeutic approaches for these uncommon manifestations.

Case Description
A 72-year-old male with a past medical history of poorly controlled type 2 diabetes mellitus (T2DM), complicated by digit amputations, hypertension, and hyperlipidemia, presented with a three-day history of dysphasia and generalized weakness. Initial examination revealed BP 212/101 mmHg, severe hyperglycemia (glucose 535 mg/dL, HbA1c 16.4%), elevated plasma osmolality (318 mOsm/kg), and negative ketones, ruling out DKA or HHS. Neurological imaging (CT, MRI, MRA) showed no acute pathology. Blood pressure was controlled (122/71 mmHg) with nicardipine infusion and labetalol, and a basal-bolus insulin regimen (glargine 18 units daily, lispro 8 units pre-meals) reduced glucose to 120-150 mg/dL within the first several days. On day 7, the patient developed confusion and left trigeminal nerve tenderness. Repeat CT brain was unremarkable. Gabapentin (300 mg thrice daily, titrated to 800 mg) resolved dysphasia, pain, and confusion within three days, restoring baseline mental status.

Discussion
Acute neurological complications in diabetes typically arise from dehydration, hyperosmolality in DKA or HHS. This patient, without DKA or HHS, exhibited initial dysphasia due to hyperglycemia and later neuralgia secondary to rapid glycemic correction [5]. TIND, historically termed "insulin neuritis,â€ manifests as acute neuropathic pain or autonomic dysfunction following swift hyperglycemia resolution [2, 6-9]. Dysphasia is a rare presentation of diabetic neuropathy, with few cases reported in hyperglycemic crises [4]. Gibbons and Freeman's TIND criteriaâ€”HbA1c reduction â‰¥2% over three months, acute neuropathic symptoms, and onset within eight weeksâ€”partially align with this case [5]. Differentiating diabetic neuropathy or TIND from stroke, transient ischemic attack, or complex migraine requires urgent neuroimaging. Hypertension, though initially present, did not explain persistent symptoms or trigeminal neuralgia post-blood pressure normalization. Management of diabetic neuropathy and TIND focuses on supportive care and optimized glycemic control. In this case, gabapentin, frequently used off-label for diabetic neuropathic pain, achieved complete symptom resolution, highlighting its therapeutic potential. Both hyperglycemia and rapid glucose correction can precipitate neurological manifestations, highlighting the need for further research into diabetic neuropathy and TIND pathophysiology and optimal management strategies.

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FROM AIRSPACE TO ABDOMEN: AN UNUSUAL MANIFESTATION OF DISSEMINATED BLASTOMYCOSIS

Purpose for study:

This case highlights an exceedingly rare presentation of Blastomyces dermatitidis presenting as both pneumonia as well as peritonitis.

Introduction:

Blastomyces dermatitidis is a dimorphic fungus endemic to the Ohio and Mississippi River valleys and Great Lakes region. It is being increasingly being reported in New York. It typically presents with pulmonary involvement but can disseminate to extrapulmonary sites including skin, bones, and genitourinary tract. Peritoneal involvement is exceedingly rare.

Case presentation:

A 53-year-old male with a history of alcohol use disorder presented to an outside hospital with abdominal distention and shortness of breath. Physical exam demonstrated respiratory distress and ascites. X-ray of the chest revealed a dense consolidation in the left lower lobe, consistent with pneumonia. Empiric antibiotics were started. A CT abdomen/pelvis with contrast showed cirrhosis and ascites. Incidentally noted was a 4.5 x 7.3 cm pleural-based mass in the left lower lobe with hilar lymphadenopathy and adjacent pulmonary nodules. A dedicated CT chest confirmed these intrathoracic findings. Paracentesis yielded 5L of yellow ascitic fluid and a SAAG of 1.0, consistent with non-portal hypertensive ascites. Cultures were negative. Cytology was negative for malignant cells but showed reactive mesothelial cells and fungal organisms, interpreted as Aspergillus, despite negative Aspergillus serologies. IV voriconazole was initiated given concern for disseminated fungal infection. Histoplasma urine antigen was positive (0.6 ng/mL).â€¯Patient was transferred to Albany Medical Center. Bronchoscopy and biopsy specimens were obtained. Histopathological analysis confirmed broad-based budding yeast, consistent with Blastomyces dermatitidis confirmed by state laboratories. There were no features of Histoplasma histologically, and cryptococcal antigen was negative. A diagnosis of disseminated blastomycosis pneumonia and peritonitis was made. Patient was started on itraconazole and continued for 6 months with clinical improvement.

Discussion:

Blastomycosis is often underdiagnosed due to its nonspecific presentation and radiographic mimicry of bacterial pneumonia or malignancy. Inhalation of conidia leads to primary pulmonary infection, and hematogenous dissemination can involve various organs. Peritoneal blastomycosis is rare, with only isolated cases reported in the literature. In this patient, the presence of ascitic fungal elementsâ€”initially misidentified as Aspergillusâ€”combined with pulmonary involvement, posed a diagnostic challenge. The false-positive histoplasma urine antigen likely reflects cross-reactivity, which

has been previously documented with Blastomyces. Cirrhosis may predispose to disseminated fungal infections due to immunosuppression, altered gut permeability, and ascitic fluid stasis. This case highlights the importance of maintaining a high clinical suspicion for endemic fungal infections in immunocompromised patients presenting with new onset ascites and respiratory symptoms.

Conclusion:

This case underscores the importance of considering disseminated blastomycosis in the differential diagnosis of patients with cirrhosis presenting with pulmonary and peritoneal findings. Early histopathologic confirmation and appropriate antifungal therapy are critical to favorable outcomes in such presentations.

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A CASE OF INTRAHEPATIC CHOLANGIOCARINCOMA, DISSEMINATED INTRAVASCULAR COAGULATION AND NON-BACTERIAL THROMBOTIC ENDOCARDITIS

Objectives:

This case describes a rare case of intrahepatic cholangiocarcinoma complicated by simultaneous DIC and NBTE, highlighting diagnostic and therapeutic challenges.

Background information/introduction:

Intrahepatic cholangiocarcinoma is a malignancy of the intrahepatic bile ducts, often presenting with nonspecific symptoms and associated with a poor prognosis. Paraneoplastic syndromes such as disseminated intravascular coagulation (DIC) and marantic endocarditis, or nonbacterial thrombotic endocarditis (NBTE) are rare complications, particularly in the absence of infection. This case details a rare and complex case of intrahepatic cholangiocarcinoma complicated by both DIC and NBTE.

Case presentation:

An 86-year-old female with history of thyroid cancer and breast ductal carcinoma in situ initially presented with hematochezia. Labs on arrival included hemoglobin 8.7g/dL, platelets 137,000, creatinine 2.46mg/dL (baseline 1.2mg/dL), AST 65U/L, ALT 84U/L, ALP 1015U/L, and total bilirubin 1.4mg/dL. Blood and urine cultures were negative. EGD and colonoscopy did not identify a source of bleeding. CT abdomen/pelvis demonstrated an ill-defined 7.7 x 6.8cm mass occupying the right hepatic lobe, without metastasis. Endoscopic ultrasound with biopsy revealed moderately differentiated adenocarcinoma consistent with intrahepatic cholangiocarcinoma. The patient developed worsening anemia, thrombocytopenia and hypofibrinogenemia. She was diagnosed with DIC, requiring transfusion support. The following day, physical exam showed new purple discoloration of the nose, fingertips, and toes, concerning for microvascular emboli. Echocardiogram revealed a large mobile vegetation on the posterior mitral valve leaflet. Repeated blood cultures remained negative. Although not a candidate for transesophageal echocardiogram, cardiology and infectious disease consultants agreed her presentation was consistent with NBTE. Anticoagulation was contraindicated given severe thrombocytopenia and gastrointestinal bleeding. The patient subsequently developed bilateral deep vein thromboses of the lower limbs with associated ischemia, at which point further investigations and treatments were deferred and she was discharged home with hospice.

Discussion:

The simultaneous presence of DIC and marantic endocarditis in the setting of intrahepatic cholangiocarcinoma is exceedingly rare. Malignancies, particularly mucinous adenocarcinomas such as cholangiocarcinoma can activate coagulation cascades and lead to systemic thrombotic phenomena. DIC may present as either thrombotic or hemorrhagic and complicates management due to the paradox of

clotting and bleeding risks. NBTE typically occurs in patients with advanced malignancy and is frequently undiagnosed until autopsy. It involves sterile vegetations composed of fibrin and platelets, most commonly affecting the mitral and aortic valves. NBTE is uncommonly associated with a cardiac murmur, making early detection challenging. Distinguishing NBTE from infectious endocarditis is challenging, requiring a thorough infectious workup and collaboration with infectious disease and cardiology consultants. Clinical suspicion should be high in patients with unexplained embolic events. This case illustrates the importance of recognizing paraneoplastic syndromes as potential complications of malignancy and highlights the diagnostic challenges and limited treatment options in such advanced disease states.

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Acute Muscle Weakness and Rhabdomyolysis in a 40-Year-Old Female with Potential Mushroom-Induced Necrotizing Autoimmune Myopathy

Introduction:

Rhabdomyolysis is a serious clinical condition marked by elevated creatine kinase (CK) and muscle breakdown, often triggered by trauma, medications (e.g., statins), or strenuous activity. Necrotizing autoimmune myopathy is a rare but important differential, typically associated with statin use. However, the role of dietary supplementsâ€”particularly mushroom-based productsâ€”remains underexplored. This case highlights a potential link between mushroom coffee and necrotizing myopathy in a patient without traditional risk factors.

Case Presentation:

A 40-year-old previously healthy woman presented with three weeks of progressive proximal muscle weakness and myalgias, affecting both upper and lower extremities. She denied recent trauma, infections, or statin use. Notably, she had recently begun taking multiple dietary supplements, including mushroom coffee. Her physical exam revealed symmetric proximal muscle weakness, particularly in the hip flexors and shoulder abductors. Laboratory tests showed markedly elevated CK (12,149 U/L), transaminases (ALT: 821 U/L; AST: 626 U/L), and positive urine myoglobin. MRI of the lower extremities demonstrated bilateral symmetric muscular edema consistent with inflammatory myopathy.

Neurology and rheumatology consultations raised concerns for autoimmune myositis. She was initiated on high-dose IV methylprednisolone. A muscle biopsy confirmed necrotizing myopathy. Despite declining IVIG, the patient responded well to corticosteroids and methotrexate, with gradual improvement in strength and declining CK levels over subsequent weeks.

Discussion:

The patient's presentation was consistent with necrotizing autoimmune myopathy. However, the absence of common triggers prompted evaluation for alternative etiologies. The recent introduction of mushroom coffeeâ€”containing compounds that may inhibit HMG-CoA reductaseâ€”emerged as a potential contributor. This enzyme is also targeted by statins, and its inhibition is implicated in statin-associated myopathies. Although causality cannot be definitively established, the temporal relationship between supplement use and symptom onset raises concern for supplement-induced myopathy in susceptible individuals. This case illustrates the importance of considering supplement history in patients presenting with unexplained rhabdomyolysis or myopathy.

Conclusion:

This case underscores the potential for mushroom-based supplements to contribute to necrotizing myopathy, particularly in patients lacking traditional risk factors. Healthcare providers should maintain a high index of suspicion for supplement-induced muscle toxicity and conduct thorough histories that

include over-the-counter products. Early recognition and intervention with corticosteroids and immunosuppressants may improve outcomes in cases with autoimmune overlap.

Keywords:

Rhabdomyolysis, necrotizing myopathy, mushroom coffee, supplement-induced myopathy, HMG-CoA reductase inhibition

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A CASE OF MULTIPLE HAMARTOMA SYNDROME WITH MULTINODULAR GOITRE

Introduction:

Cowden's Syndrome is an autosomal dominant disorder caused by germline mutations in the PTEN tumor suppressor gene characterized by the development of multiple hamartomas and an increased lifetime risk of several malignancies, including those of the thyroid, breast, endometrium, and colon. Herein, we present an unusual case of sporadic Cowden's syndrome as the etiology of multinodular goiter in an adolescent male.

Case Description:

An 18-year-old male presented with chief complaints of a midline neck swelling that had increased in size over 2.5 months causing difficulty swallowing along with change in his voice and difficulty breathing. He had multiple prior biopsy proven trichilemmomas over his face, neck and trunk that had progressively increased in number over seven years. Prior to this, he had been evaluated with a colonoscopy for episodes of hematochezia which revealed multiple hamartomatous polyps. He had six siblings none of whom had any similar complaints or findings.

Physical examination was remarkable for macrocephaly, multiple raised skin-colored lesions over his body, raised lesions on the buccal mucosa in a cobblestone pattern, and a spherical, nodular, anterior neck swelling, 6 x 5 cm in size that moved up with deglutition.

Ultrasonography of the thyroid was suggestive of multinodular goiter and a biopsy confirmed FNAC grade 3 atypia of the Bethesda grading system for which he underwent a total thyroidectomy.

Owing to his complex phenotype and young age, a hereditary predisposition syndrome was suspected. Genetic testing revealed a mutation in the PTEN tumor suppressor gene (on chr.10q23) confirming the diagnosis of Cowden's syndrome.

Discussion:

Presence of clinically overt thyroid disease is uncommon in childhood and adolescence, hence physicians should be suspicious of an underlying genetic or syndromic association when a nodular thyroid disease presents in this age group. While a positive family history aids in diagnosis, sporadic cases are often missed. Cowden's syndrome is estimated to affect 1:200,000 people and is associated with increased rates of malignancies. It has varying presentations which makes its diagnosis challenging. Our case underscores the importance of having a high index of suspicion of genetic and syndromic associations despite the absence of a family history as early diagnosis facilitates timely surgical and surveillance interventions, potentially reducing morbidity and improving long-term outcomes.

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Severe Hyperlipidemia and Hypoalbuminemia in an Adolescent: An Atypical Presentation of Nephrotic Syndrome Introduction

Introduction

Severe hyperlipidemia (HLD), particularly with low-density lipoprotein (LDL) levels above 190 mg/dL in adolescents, is most commonly attributed to familial hypercholesterolemia. However, in the absence of family history or classic cardiovascular risk factors, secondary causes should be promptly considered. Nephrotic syndrome is a well-known secondary cause of marked HLD in adolescents but is often overlooked. Failure to identify nephrotic syndrome can delay treatment and risk the progression of renal disease. We present a case of a patient with severe HLD and hypoalbuminemia as the first signs of nephrotic syndrome.

Case Presentation

An 18-year-old male with no significant past medical history was referred to the endocrine clinic for evaluation of severe HLD detected on routine labs. He denied any chest pains, palpitations, dyspnea, gastrointestinal symptoms, extremity pain, or change in weight. Family history was negative for HLD, cardiovascular disease, or early sudden death. He did not smoke or drink alcohol.

Physical examination revealed normal blood pressure, a body mass index of 17.1, no xanthomas, hepatomegaly, edema, or signs of volume overload. Labs revealed total cholesterol of 448 mg/dL (100-200 mg/dL), LDL of 393 mg/dL (40-100 mg/dL), and high-density lipoprotein (HDL) of 62 mg/dL (>40 mg/dL). Notably, albumin levels were 2.3 g/dL (3.5-5.2 g/dL), and total protein was 4.1 g/dL (5.6-7.6 g/dL). Creatinine was 1.1 mg/dL (0.5-1.3 mg/dL) with an estimated GFR of 83. He was started on atorvastatin for the severe HLD.

The gastroenterology workup was negative for liver disease or protein-losing enteropathy. Hepatitis serologies, antinuclear antibodies, anti-neutrophil cytoplasmic antibodies, and complements were within normal range. Urinalysis revealed nephrotic-range proteinuria with a urine protein-to-creatinine ratio of 13.6. Kidney biopsy showed a diffuse immune complex-mediated membranoproliferative glomerulonephritis (MPGN) with moderate to severe activity.

The patient was initiated on high-dose prednisone and subsequently started on cyclosporine with plans for tapering steroids. His proteinuria began to improve significantly, accompanied by normalization of albumin and a steady decline in LDL and total cholesterol. Atorvastatin was discontinued due to potential drug interactions with cyclosporine. His lipid profile remained stable off statins.

Discussion

This case underscores the importance of considering secondary causes in the differential diagnosis of severe hyperlipidemia, particularly in the setting of hypoalbuminemia and proteinuria. MPGN is a rare immune complex-mediated glomerular disorder. MPGN may present subtly without edema or overt renal dysfunction. Lipid abnormalities in nephrotic syndrome result from hepatic overproduction of lipoproteins in response to urinary protein loss. Treatment of the underlying renal disease often leads to normalization of lipids without long-term lipid-lowering therapy. Timely identification can prevent further kidney damage and unnecessary, prolonged statin therapy

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STAGE B HEART FAILURE WITHOUT CLINICAL SYMPTOMS: A CASE-BASED REVIEW OF CARDIOMYOPATHY WORKUP AND THE ROLE OF PARADIGM-HF IN BRIDGING GUIDELINE GAPS

Purpose: To examine the diagnostic and therapeutic complexities in a bedbound patient with newly identified cardiomyopathy and no overt clinical heart failure, and to assess the relevance of guideline-directed medical therapy (GDMT) through the lens of the PARADIGM-HF trial.

Methods: We present a case of a 70-year-old male with multiple comorbidities, including prior cerebrovascular accidents, hypertension, and opioid use disorder, who was admitted for chest pain and severe hypertension. He was found to have mildly reduced left ventricular ejection fraction (LVEF 45%) and global hypokinesis on transthoracic echocardiography but no clinical signs or prior diagnosis of heart failure. Workup for acute coronary syndrome was negative, and stress testing or cardiac catheterization could not be completed due to severe contractures. The patient was medically managed and started on carvedilol, sacubitril/valsartan, and atorvastatin. A review of heart failure staging and landmark trials, including PARADIGM-HF, was performed to contextualize therapeutic decisions.

Results: Echocardiography showed mildly reduced systolic function (EF 45%) and left atrial enlargement, without significant valvular or right-sided abnormalities. Despite an elevated NT-proBNP (1246 pg/mL), the absence of clinical symptoms placed the patient in Stage B (pre-heart failure) per ACC/AHA guidelines. Functional classification was indeterminate due to immobility. With no prior imaging, cardiomyopathy was presumed new. The PARADIGM-HF trial, which demonstrated a 20% reduction in cardiovascular mortality and heart failure hospitalization with sacubitril/valsartan versus enalapril in patients with HFrEF (EF â‰¤40%) and NYHA Class II-IV symptoms did not include asymptomatic patients or those with EF >40%. However, the trial's inclusion of patients with structural heart disease and elevated natriuretic peptides supports the rationale for early intervention in high-risk populations, even outside strict trial parameters.

Conclusion: This case illustrates the diagnostic and therapeutic complexities involved in managing cardiomyopathy in patients who are non-ambulatory and lack overt symptoms of heart failure. Although the patient's ejection fraction of 45% placed him outside the inclusion criteria of the PARADIGM-HF trial, the presence of structural cardiac abnormalities and elevated natriuretic peptides supported the initiation of guideline-directed medical therapy. As clinical recognition of Stage B heart failure continues to evolve, this case reflects a growing subset of patients who do not fit neatly into existing trial frameworks but may still be at significant risk for disease progression. It underscores a critical limitation in current heart failure guidelines and highlights the need for more inclusive research that better captures the heterogeneity of patients encountered in everyday clinical settings.

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LAMBL'S EXCRESCENCE OF THE PULMONIC VALVE: A RARE FINDING IN A PATIENT WITH GOUT

Purpose:

Incidental findings on cardiac imaging often present a diagnostic challenge. We present a rare case of Lambl's excrescences (LEs) located on the pulmonic valve to demonstrate the pivotal role of transthoracic echocardiography (TTE) in distinguishing these benign structures from more serious pathological masses.

Methods:

We reviewed the clinical course of a patient who presented with an acute gout flare and was found to have a pulmonic valve mass on imaging. Echocardiographic images from both TTE and transesophageal echocardiography (TEE) were analyzed to characterize the lesion.

Results:

A middle-aged patient presented with joint pain diagnosed as an acute gout flare. A TTE performed during the evaluation unexpectedly revealed thin, highly mobile echo densities on the pulmonic valve leaflets. Given the anterior position of the pulmonic valve, TTE provided excellent visualization. The filamentous appearance of the lesion raised a differential diagnosis including vegetation, fibroelastoma, or myxoma; however, the patient showed no systemic signs of infection or malignancy. A subsequent TEE confirmed the presence of thin, linear strands originating from the coaptation margins on the pulmonary artery side of the valve features pathognomonic for Lambl's excrescences. No valvular dysfunction or thromboembolic complications were observed. Based on the definitive imaging characteristics and the benign clinical context, no surgical or medical intervention was pursued. The patient was managed conservatively for their primary diagnosis and discharged without complications.

Conclusion:

While Lamb's excrescences are most commonly found on left-sided heart valves, this case demonstrates their rare occurrence on the pulmonic valve. Accurate identification using echocardiographyâ€”particularly TTE, given the valve's anterior anatomyâ€”is crucial to prevent misdiagnosis and avert unnecessary interventions. Recognizing the benign nature of LEs is paramount for guiding clinicians toward appropriate, safe, and cost-effective care. Furthermore, the presentation in the setting of an acute gout flare, a potent pro-inflammatory state, raises the question of whether systemic inflammation may contribute to the endothelial disruption implicated in LE formation, warranting further observation in future cases

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Cardiac complications of cancer immunotherapy: A case of pembrolizumab induced myocarditis

Background:
Pembrolizumab is a programmed cell death ligand 1 (PDL1) inhibitor in a class of targeted immunotherapy called immune checkpoint inhibitor (ICI). Different agents in this class target different pathways like cytotoxic T lymphocyte antigen 4 (CTLA-4), programmed death receptor 1 (PD1), and PDL1 to increase immune activation. This can trigger autoimmune diseases. Myocarditis is one of the rare side effects. Immune-mediated myocarditis has been reported in <1%, but it has a high mortality rate of 50%. Currently, the treatment consists of stopping the offending agent and high-dose IV steroids. This article underscores a rare instance of pembrolizumab-induced autoimmune myocarditis.

Case presentation:
A 51-year-old female presented complaining of shortness of breath and chest pain with radiation down both arms for five days. Past medical history includes stage IV adenocarcinoma of the lung with metastasis to the brain. The initial workup showed troponin 11,270, and the EKG was significant for ST elevations in II, III, aVF, and V2-V6, suggestive of inferior and anterolateral wall injury. The viral respiratory panel was negative. Emergency left heart catheterisation showed no significant coronary artery disease. CT angiogram ruled out pulmonary embolism. A transthoracic echocardiogram (TTE) on the day of admission showed an ejection fraction (EF) of 30%-34%, with the following segments aneurysmal: apical anterior, apical septal, apical inferior, apical lateral, and apex. Further workup showed CRP 49, procalcitonin of 0.12, and lactate 4. For her stage IV lung adenocarcinoma, she was initially treated with four cycles of carboplatin, pemetrexed, and pembrolizumab with maintenance pemetrexed and pembrolizumab, the last dose a week before admission. Empiric therapy with IV methylprednisolone 1 mg/kg was initiated for pembrolizumab-induced myocarditis. She received 4 doses of IV solumedrol and was discharged with tapering steroids and scheduled TTE in 2-4 weeks.

Discussion:
Our case demonstrates ICI-induced autoimmune myocarditis. In accordance with the European Society of Cardiology's guidelines, our case meets the diagnostic criteria for ICI myocarditis with new troponin elevation and a minimum of 2 minor criteria, including a clinical syndrome of fatigue, chest pain and a decline in left ventricular systolic function with regional wall motion abnormalities. Myocarditis typically develops within the first four cycles of chemotherapy. This case is a delayed presentation, presenting more than a year after the initial 4 cycles.

Conclusion:

ICI in lieu of or in combination with traditional chemotherapy have been shown to improve survival in cancer patients. Recognition of ICI myocarditis and ruling out other etiologies is paramount for a favorable prognosis. As the role of ICI in the treatment of variety of malignancies increase, our case presents a rare but potentially fatal adverse effect of novel targeted immunotherapy which warrants a low threshold for suspicion and subsequent aggressive management of ICI myocarditis.

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A DIAGNOSTIC CHALLENGE: DERMATOMYOSITIS IN A YOUNG ADULT MALE

Introduction

Dermatomyositis (DM) is a rare autoimmune inflammatory myopathy presenting with proximal muscle weakness and characteristic skin manifestations. Accurate classification among inflammatory myopathies is essential, as each subtype has distinct pathology, prognosis, and therapeutic response. We present a diagnostically challenging case of DM in a young adult male with evolving clinical and histopathologic features despite initially negative serologies.

Case Presentation

A 24-year-old male with a history of depression and obesity presented to the emergency department with three months of worsening fatigue, progressive proximal muscle weakness, and a pruritic, erythematous rash. The rash, initially treated as allergic dermatitis as an outpatient, spread to involve the neck, occiput, and extremities. He also endorsed joint pain, weight gain, and mechanical falls, though he denied dysphagia and fevers.

Physical exam revealed symmetric proximal muscle weakness and diffuse erythematous patches over the upper torso and face, including heliotrope rash, V-sign, and shawl sign. Initial laboratory studies demonstrated elevated creatine phosphokinase (CPK; peak 1153 U/L), aldolase (10.7 U/L), liver enzymes, and inflammatory markers. Troponin was mildly elevated without ischemic ECG changes. An initial autoantibody panel, including ANA, anti-HMGCR, rheumatoid factor, cyclic citrullinated peptide antibodies, and IgG/IgA were negative.

MRI of the thighs demonstrated diffuse T2 hyperintensity in the gluteal and proximal thigh muscles, suggestive of inflammatory myopathy. Pulmonary function tests indicated a restrictive pattern with preserved diffusion capacity (DLCO), likely due to neuromuscular involvement.

Investigation

Muscle biopsy of the left anterior thigh revealed perifascicular atrophy with myofiber regeneration and mild perivascular inflammationâ€”hallmarks of complement-mediated microangiopathy characteristic of DM. Dermatologic evaluation confirmed cutaneous features consistent with DM. The patient was started on IV methylprednisolone for presumed DM, despite negative preliminary autoantibodies. He was discharged following improvement of his muscle weakness and rash on an oral prednisone taper and supportive therapy (calcium, vitamin D, and a PPI) with scheduled outpatient rheumatology follow-up.

Post-discharge, an extended myositis panel returned highly positive for nuclear matrix protein-2 antibody (anti-NXP2; 183, ref <20).

Conclusion:

Resident/Fellow Clinical Vignette

This case highlights the diagnostic complexity of dermatomyositis, particularly in early or acute presentations. Despite an initially inconclusive serologic workup, the presence of characteristic findingsâ€”including heliotrope rash, V-sign, shawl sign, and proximal muscle weaknessâ€”along with MRI and muscle biopsy results, supported the diagnosis. The biopsy showing perifascicular atrophy with perivascular inflammation served as a key diagnostic hallmark of complement-mediated microangiopathy.

Although myositis-specific autoantibodies were initially negative, the clinical picture prompted the timely initiation of immunosuppressive therapy. Post-discharge, the myositis panel revealed high-titer anti-nuclear matrix protein-2 (anti-NXP2) antibodies, commonly associated with young-onset, severe disease. This case underscores the potential for multisystem involvement, including myocardial and pulmonary manifestations, and emphasizes the need for early recognition, prompt treatment, and a multidisciplinary approach to minimize long-term morbidity.

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Temperature Drop, Rhythm Stop: SVT Converted After Rectal Thermometer Insertion

Introduction:
Supraventricular tachycardia (SVT) is a common arrhythmia characterized by rapid heart rates originating above the bundle of His, typically due to reentrant mechanisms such as atrioventricular nodal reentrant tachycardia (AVNRT) or atrioventricular reciprocating tachycardia (AVRT). Initial treatment includes vagal maneuversâ€"such as the Valsalva maneuver or carotid sinus massageâ€"which aim to terminate the arrhythmia by increasing parasympathetic tone. In rare instances, visceral stimulation, including digital rectal examination (DRE) or digital rectal massage (DRM), has been associated with vagal-induced arrhythmia conversion. However, spontaneous SVT conversion following rectal temperature measurement is exceedingly rare and not well documented.

Case Presentation:
We present the case of a 75-year-old female with a complex medical history including hypertension, type 2 diabetes, chronic kidney disease, prior cerebrovascular accident, and treated breast cancer, who was brought to the emergency department from a nursing home for seizure activity and altered mental status. Upon arrival, she was noted to be tachycardic, escalating to a heart rate of 180 bpm. Electrocardiography confirmed narrow-complex SVT. While preparing for pharmacologic intervention with adenosine, a rectal thermometer was inserted to obtain a core temperature. Remarkably, the patient spontaneously converted to normal sinus rhythm with a heart rate in the 90s, without administration of any antiarrhythmic medications.

Discussion:
While vagal maneuvers are well-established for the treatment of SVT, the use of rectal stimulation to elicit vagal response remains unconventional. The presumed mechanism is visceral activation of vagal pathways via stimulation of rectal sensory receptors. Although the vagus nerve innervates only up to the transverse colon, indirect reflex activation remains a plausible explanation. Previous isolated case reports, including one in Oxford Medical Case Reports (2024), describe SVT conversion following rectal temperature checks, particularly when standard vagal maneuvers were ineffective or contraindicated. This highlights the rectal route as a potential, albeit rare, method for achieving therapeutic vagal stimulation.

Conclusion:
This case illustrates an unusual but clinically significant instance of SVT termination following rectal thermometer insertion, presumably via inadvertent vagal stimulation. While not a substitute for guideline-directed therapy, recognizing this phenomenon may be valuable in emergent or resource-limited settingsâ€"especially when standard vagal techniques are impractical or contraindicated. Further studies are warranted to explore the reproducibility and mechanism of such visceral-mediated vagal responses.

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A Rare Case of Diffuse Alveolar Hemorrhage Caused by Human Rhinovirus Infection in an Immunocompetent Individual

Introduction

Human Rhinovirus (HRV), a common cold virus, typically causes mild upper respiratory symptoms in healthy individuals. However, in elderly or immunocompromised persons, HRV is more pathogenic and can lead to pneumonia and exacerbation of conditions such as asthma or COPD, resulting in respiratory failure. A few forms of HRV, types A and C, have been classified by high-molecular-weight testing and are associated with heightened disease severity. Diffuse alveolar hemorrhage (DAH) is a rare but serious condition with alveolar bleeding that presents with hemoptysis, shortness of breath, and respiratory failure. Whereas DAH may be secondary to autoimmune disease, drugs, or infection, it is uncommon secondary to viral pathogens like HRV, especially in healthy individuals.

Case presentation

A 21-year-old woman, in the 28th week of twin gestation, presented to the hospital following multiple seizures at home and worsening shortness of breath. She was known to have a seizure disorder but had missed her medication for a day. She also had flu-like symptoms for two days and started coughing huge quantities of reddish-brown sputum. She was very breathless and hypoxic in the emergency department. A trial of NIPPV at various settings for a short time failed, for which she was intubated urgently for impending respiratory failure.

A chest CT angiogram demonstrated extensive diffuse bilateral central ground glass and consolidative opacities, sparing the periphery of the lung. Bronchoscopy confirmed DAH with progressive bloody serial aliquots. Her respiratory panel was positive for rhinovirus. Rheumatology workup came back with elevated ANA titer (1:640) but was otherwise negative on other markers (ANCA, anti-DNA, anti-Smith, etc.). She was treated with empiric antibiotics, steroids, and supportive intensive care. Subsequently, she got extubated on day 5 after clinical and radiological improvement. She was stable and transferred to the obstetric unit for further management.

Discussion

DAH can result from pulmonary capillaritis (e.g., ANCA vasculitis, SLE), bland hemorrhage, or conditions like infections and drug toxicity. Infectious causes like HRV are rarely reported as causes of DAH in healthy individuals. Since there are no systemic features of connective tissue disease with other negative serologies, positive ANA in this patient may suggest a post-viral inflammatory response rather than an autoimmune etiology. Management of DAH includes supportive care, including mechanical ventilation for respiratory failure, high-dose corticosteroids, antibiotics for bacterial or aspiration pneumonia, and management of underlying causes like autoimmune disease by immunosuppressants and plasmapheresis in refractory cases. Our patient's condition is unique because HRV is a virus that

usually causes benign symptoms; however, this led to a life-threatening complication in this young, immunocompetent patient.

Conclusion

This case report is to raise attention and suspicion about the rarity of serious complications of rhinovirus like DAH, which helps direct the management plan and outcomes.

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Thrombotic Thrombocytopenic Purpura Following Moderna COVID-19 Vaccination: A Rare Adverse Event

Introduction

Thrombotic thrombocytopenic purpura (TTP) is a rare blood disorder marked by a severe deficiency (â‰¤10%) of ADAMTS13 (a disintegrin and metalloproteinase with thrombospondin type 1 motifs, member 13), an enzyme that cleaves von Willebrand factor (vWF) multimers. TTP can be acquired via autoantibodies that inhibit or accelerate ADAMTS13 clearance, or can be congenital due to mutations. The classic presentation includes the pentad of microangiopathic hemolytic anemia, thrombocytopenia, neurological symptoms, fever, and renal dysfunction, though only 10% of patients exhibit all features.

Risk-stratification tools like the PLASMIC score help identify high-risk patients. Despite prompt treatment, TTP carries a mortality rate of 10-20%. Recent studies suggest a possible link between mRNA COVID-19 vaccines and acquired TTP. This case describes a 30-year-old woman who developed TTP following the Moderna COVID-19 vaccine.

Case Description

A 30-year-old woman with a history of asthma, migraines, and anxiety presented with a week-long history of exertional shortness of breath, fatigue, body aches, and palpitations. She reported two days of non-bilious, non-bloody vomiting, heavy menorrhagia, and red spots on her right arm. She denied previous similar symptoms or a family history of blood disorders. Three weeks prior, she received the Moderna COVID-19 vaccine.

Physical examination showed tachycardia and petechial spots on the right antecubital fossa. Lab results revealed severe anemia (hemoglobin 5.4 g/dL), thrombocytopenia (10,000/mcL), elevated LDH (1980), schistocytes on peripheral smear, and elevated total and direct bilirubin. Coagulation studies (PT/INR, aPTT) were normal, and HIV and pregnancy tests were negative.

The patient was treated with urgent therapeutic plasma exchange (TPE), high-dose glucocorticoid, and pRBC transfusion. ADAMTS-13 was <1% with positive inhibitors; reticulocyte count was 8.53. Flow cytometry and anemia panel were not significant.

After maintaining platelet counts above 150,000/mcL for five days, her platelet count dropped to 23,000/mcL, with elevated LDH (1,229) and new-onset bloody bowel movements. Repeat ADAMTS13 testing remained <1%.

Suspecting a TTP exacerbation, TPE and high-dose steroids were resumed, with plans to initiate caplacizumab, which was unavailable at our facility, requiring transfer to a tertiary center

Discussion

In our case, acquired TTP was confirmed by CBC, peripheral smear, and ADAMTS13 testing. The patient initially improved with TPE and high-dose steroids, but rituximab or caplacizumab were unavailable. Despite brief recovery, her platelet count dropped again with ADAMTS13 activity remaining < 1%. Challenges associated with high medication costs, limited access to specialized care, and insurance barriers hinder timely and comprehensive management of life-threatening conditions like TTP.

Conclusion

This case highlights the importance of considering TTP in patients with low platelet counts following recent COVID-19 vaccination, and the challenges of managing TTP in resource-limited settings where delays in accessing treatments like caplacizumab can impact patient outcomes.

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Efficacy of Cardioprotective Drugs in Cancer Patients Receiving Anthracyclines: A Pairwise Meta-Analysis and Network Meta-Analysis

Background

Anthracyclines are key in chemotherapy for various cancers but carry a high cardiotoxicity risk. Several cardioprotective drugs have been studied to mitigate cardiac deterioration. This study evaluates and ranks them via network meta-analysis.

Methods

A comprehensive search of PubMed, Cochrane, Scopus, and Web of Science (WOS) databases was conducted from inception to September 22, 2024, to identify randomized clinical trials and cohort studies examining the cardioprotective role of drugs in cancer patients undergoing anthracycline-based chemotherapy. The primary outcome was the change in left ventricular ejection fraction (LVEF). Secondary outcomes included left ventricular end-diastolic diameter (LVEDD); incidence of chemotherapy-related cardiac dysfunction (CTRCD); clinical heart failure (HF) incidence and hospitalizations; and all-cause mortality. Analyses were performed using the "netmeta" package in RStudio.

Results

A total of 35 studies were included, comprising 31 randomized controlled trials (RCTs) and four retrospective studies. The meta-analysis found that the use of any cardioprotective agent was significantly associated with increased LVEF (SMD = 0.60 [95% CI: 0.33 to 0.87], $p < 0.001$), lower risk of CTRCD (RR = 0.49 [CI: 0.38 to 0.63], $p<0.01$), all-cause mortality (RR = 0.59 [CI: 0.48 to 0.73], $p < 0.01$), HF incidence (RR = 0.45 [CI: 0.24 to 0.85], $p = 0.01$) and decreased LVEDD (SMD = -0.22 [95% CI: -0.42 to -0.02], $p = 0.034$) compared to controls. However, no significant differences were observed for HF hospitalizations (RR = 0.60 [CI: 0.18 to 2.00], $p = 0.41$).

The network meta-analysis revealed that only ACEIs plus beta-blockers (SMD = 0.58; [95% CI: 0.31to 0.85],$p<0.001$), beta-blockers alone (SMD = 0.47; [95% CI: 0.09 to 0.85],$p=0.05$), statins(SMD = 0.52; [95% CI: 0.20 to 0.84],$p=0.002$), and ACEIs (SMD = 1.18; [95% CI: 0.28 to 2.08],$p=0.01$) were significantly associated with LVEF improvement compared to controls, with ACEIs ranking the highest. In terms of CTRCD, statins (RR = 0.45 [95% CI: 0.33 to 0.60], $p < 0.01$) and beta-blockers (RR = 0.47 [95% CI: 0.30 to 0.74], $p < 0.01$) significantly reduced risk, with ACEIs plus beta-blockers ranking the most effective(RR=0.37[95% CI:0.10-1.29]). For mortality, retrospective studies identified SGLT2 inhibitors as the only drug to significantly reduce mortality (RR = 0.50 [95% CI: 0.34 to 0.75], $p < 0.01$), ranking first for mortality outcomes.

Conclusion

Resident/Fellow Clinical Vignette

ACEIs, beta-blockers, and statins effectively preserved LVEF, while beta-blockers and statins reduced CTRCD in patients receiving anthracyclines, with ACEIs being the most effective at preserving LVEF and ACEIs plus beta-blockers showing the greatest efficacy in reducing CTRCD. Notably, SGLT2 inhibitors significantly reduced mortality and ranked first in mortality outcomes. These findings highlight the need for further randomized controlled trials to evaluate the impact of SGLT2 inhibitors on LVEF and address the potential biases in observational studies.

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Diagnostic Dilemmas in Catastrophic Antiphospholipid Syndrome: A Case of Seronegative Multiorgan Thrombosis with Dramatic Response to Immunosuppression

Catastrophic antiphospholipid syndrome (CAPS), a rare and severe variant of antiphospholipid syndrome (APS), is a life-threatening thrombo-inflammatory disorder characterized by rapid onset multi-organ failure. While APS is typically associated with single medium-to-large blood vessel occlusions, CAPS is most often linked to several concurrent vascular occlusions of small vessels, commonly affecting the kidneys, skin or brain. Diagnosis of CAPS typically relies on both clinical and laboratory criteria, including the presence of antiphospholipid antibodies. However, real-world cases often present with overlapping features and incomplete serologic profiles, complicating timely diagnosis.

We report on a 61-year-old man with a history of human immunodeficiency virus (HIV) infection, chronic kidney disease (due to focal segmental glomerulosclerosis), and substance use disorder. He was admitted following a femoral neck fracture secondary to an episode of dizziness. Soon after his surgery, he developed multiple ischemic strokes, confirmed by MRI. His hospital stay became further complicated by acute kidney failure requiring dialysis, pulmonary embolism, thrombocytopenia, intermittent fevers, a high white blood cell count, and consistently high inflammatory markers, including an erythrocyte sedimentation rate over 145 mm/hour. In contrast, his complement levels remained normal throughout this hospitalization.

An extensive workup for autoimmune and clotting disorders largely came back negative. This included tests for lupus anticoagulant, anticardiolipin immunoglobulin G (IgG) and immunoglobulin M (IgM), beta-2 glycoprotein I antibodies, antinuclear antibody (ANA), anti-double-stranded DNA (anti-dsDNA), and antineutrophil cytoplasmic antibodies (ANCAs). Notably, a non-criteria positive anticardiolipin IgG was found to be positive, but only at a low level. Patient clinically status deteriorated significantly, with multiorgan thrombosis and systemic inflammation, hence a presumptive diagnosis of CAPS was made despite incomplete laboratory criteria.â€¨â€¨The patient received high-dose corticosteroids, plasmapheresis, intravenous immunoglobulin (IVIG), and anticoagulation with warfarin. Following this treatment, he showed significant clinical improvement, including his renal function as well as decrease in inflammation markers.While alternative diagnoses such as systemic lupus erythematosus, vasculitis, and fat embolism syndrome were considered, these were deemed less likely based on his clinical course and lab results. Specifically, vasculitis seemed unlikely due to the presence of blood clots over blood vessel inflammation, the absence of tissue biopsy confirmation for vasculitis (which typically shows vessel wall inflammation or fibrinoid necrosis), and normal complement levels. â€¨â€¨This case highlights the significance of the inherent diagnostic complexities of CAPS in the absence of complete serologic confirmation, particularly when compounded by factors such as HIV infection and substance use disorder. Despite not fully satisfying established classification criteria, the profound and sustained therapeutic response to CAPS-directed interventions provided compelling support for the diagnosis. This experience emphasizes the critical role of clinical judgment and timely empiric therapy in patients presenting with fulminant multiorgan thrombosis, given that treatment delays can precipitate irreversible organ damage or mortality.

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Intravascular hemolysis and pancytopenia: rare presenting feature of pernicious anemia

Pernicious anemia is a common cause of vitamin B12 deficiency, typically presenting with megaloblastic anemia, pancytopenia, and neurologic symptoms. We report an unusual case of severe hemolytic anemia, pancytopenia, and new-onset cirrhosis, which on extensive workup was ultimately found to be caused by profound vitamin B12 deficiency due to pernicious anemia.

A 65-year-old male with a history of hypertension, type 2 diabetes, resting tremors, and a family history of pernicious anemia presented with several months of progressive fatigue, exertional dyspnea, dizziness, and unintentional 6-lb weight loss. Initial labs showed pancytopenia: hemoglobin (Hgb) 5 g/dL, hematocrit (Hct) 15.8%, mean corpuscular volume (MCV) 97.5 fL, white blood cell count (WBC) 1.42 × 10â¹/L, absolute neutrophil count 810/µL, and platelets 17 × 10â¹/L. He required multiple transfusions. Total bilirubin was mildly elevated at 1.7 mg/dL, while renal and hepatic panels were within normal limits. Peripheral smear showed no blasts or schistocytes. Elevated lactate dehydrogenase (LDH >2500 U/L) and undetectable haptoglobin (<15 mg/dL) were consistent with intravascular hemolysis. Coombs test was negative; reticulocyte count was inappropriately normal. Ferritin, coagulation studies, flow cytometry, and viral panels were unremarkable. Serum vitamin B12 was severely reduced (<150 pg/mL).

CT imaging revealed splenomegaly and findings suggestive of cirrhosis, including ascites, esophageal varices, and pericardial effusion. Workup for autoimmune liver disease was negative. Bone marrow biopsy (BMB) demonstrated marked erythroid hyperplasia (82% erythroid precursors), with 41.2% proerythroblasts and pronormoblasts, normal cytogenetics, and no clonal or neoplastic population. ADAMTS13 activity was low-normal at 36%. Anti-parietal cell and intrinsic factor antibodies were elevated, confirming pernicious anemia. The patient was started on intramuscular vitamin B12. By day 7, labs improved to Hgb 7.1 g/dL, Hct 21.9%, WBC 1.7 × 10â¹/L, and platelets 46 × 10â¹/L. By day 16, Hgb reached 9.6 g/dL, WBC 5 × 10â¹/L, and platelets 174 × 10â¹/L, indicating rapid hematologic recovery.

The differential diagnosis for hemolytic anemia with pancytopenia is broad and includes TTP, paroxysmal nocturnal hemoglobinuria, HLH, autoimmune hemolytic anemia (AIHA), pure erythroid leukemia, myelodysplastic syndrome, monoclonal gammopathy, and viral infections. HLH and MDS were excluded by normal ferritin and marrow findings; AIHA was ruled out by negative Coombs; absence of schistocytes and normal ADAMTS13 made TTP unlikely. Although cirrhosis was newly diagnosed, it was not the primary cause given the rapid response to B12 therapy.

Early recognition of vitamin B12 deficiency is critical as it is a reversible condition and has excellent prognosis. Delayed diagnosis can result in ineffective erythropoiesis, intramedullary hemolysis, worsening cytopenia, and even causing mortality. This case underscores the importance of including vitamin B12 deficiency in the differential diagnosis in the presence of severe hemolytic anemia and pancytopenia, ensuring prompt treatment to prevent complications.

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Refractory Shock in a 42-Year-Old Woman with Systemic AL Amyloidosis: Challenges in Diagnosis and Management

Systemic AL amyloidosis is characterized by extracellular tissue deposition of amyloid fibrils due to an underlying plasma cell dyscrasia. Presentation is highly variable, but diagnosis is most commonly made following onset of organ-specific symptoms, with the majority of patients developing both renal and cardiac involvement.

We present a case of a 42-year-old female who presented with three weeks of worsening dyspnea, three months of abdominal pain, nausea and vomiting, eruptive spider angiomata and unintentional weight loss of 30 pounds. She was also undergoing workup for erythrocytosis and hepatosplenomegaly. Nephrotic range proteinuria had also been noted on outpatient urine studies. She was hospitalized for non-ST-elevation myocardial infarction (NSTEMI) two weeks prior to presentation. Echocardiogram had revealed global longitudinal strain of -7.6% and relative apical sparing, raising concern for cardiac amyloidosis.

Due to worsening dyspnea and hypotension, she was directed to the ED and later transferred to our center for advanced heart failure evaluation. Her physical examination was notable for jaundice, spider angiomas and tender hepatomegaly.

Her labs were notable for a hemoglobin of 18.1 g/dL, an elevated brain natriuretic peptide to 28,448 pg/mL, AST of 4,796 U/L, ALT of 1,741 U/L, ALP of 1,107 UL, and total bilirubin of 6.8 mg/dL, serum creatinine of 2.99 mg/dL, urine total protein to creatinine ratio of 13.39, and a serum bicarbonate of 14 mmol/L. Her lambda serum free light chains (FLC) were 85 mg/dL, and her kappa FLCs were 28 mg/dL. Bone marrow biopsy showed 10% plasma cells, but with indeterminate clonality. Echocardiogram notably revealed dynamic left ventricular outflow tract obstruction (LVOTO) in addition to the above findings at her previous hospitalization. She also underwent endomyocardial biopsy confirming AL amyloidosis.

She experienced rapid decline, progressing to multi-organ failure. She was started on phenylephrine and dexamethasone. She required initiation of continuous renal replacement therapy due to acute renal failure, developed acute liver failure, and was intubated for acute hypoxic respiratory failure by hospital day 2. She was transitioned to comfort measures and expired on hospital day 3.

Systemic AL amyloidosis is a rare disorder with an estimated 4000 new cases diagnosed in the US each year and a median age of 64 at diagnosis. Diagnosis is often delayed due to the fairly nonspecific nature of initial symptoms. This patient had a particularly rapid progression of her disease requiring cardiac biopsy for diagnosis, with onset of her cardiac symptoms quickly leading to shock, multiorgan failure, and ultimately death, with additional challenges in management due to LVOTO, which is rarely associated with cardiac amyloidosis. She had unexplained nephrotic-range proteinuria dating back at least 2 months prior to hospitalization which, if worked up, may have led to an earlier diagnosis and initiation of treatment.

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COLD CELLS AND HIDDEN CLOTS: A HEMOLYTIC MYSTERY IN THE WINTER

Introduction:

Cold agglutinin hemolytic anemia (CAHA) is a type of autoimmune hemolytic anemia mediated by IgM autoantibodies that bind red blood cells at lower temperatures activating the complement pathway and leading to hemolysis. CAD can be primary or secondary to an underlying disease such as infections, immune disorders or malignancies. Cold exposure can exacerbate hemolysis by promoting antibodies binding and complement activation.

Mycoplasma pneumoniae is the one the most common infectious trigger for secondary CAHA, typically seen in younger patients. Rarely can present severely and with complications in older immunocompetent adults. Mycoplasma pneumoniae has been recognized as a trigger for thrombotic events such as deep vein thrombosis (DVT). We present a unique case of CAHA and DVT in a patient with Mycoplasma pneumoniae infection.

Case description:

79-year-old male presented dizziness and generalized weakness after being outside in the cold. He reported having nasal congestion and cough for the past two weeks and had not gone outside during that period. On the day of presentation, he went outside briefly in winter weather, after which he developed acute symptoms. Initial laboratory evaluation revealed hemoglobin 6.5 g/dL, white blood cells 31.3 x10â¹/L, and a total bilirubin of 2.8 mg/dL. CT chest revealed tree-in-bud nodularity suggestive of infectious broncholitis. Peripheral blood smear and sample revealed cold agglutinins, raising suspicion of hemolytic anemia due to CAD. Further labs revealed LDH of 1387 U/L, undetectable haptoglobin (<10 mg/dL), and a positive direct Coombs test for complement C3. Respiratory PCR panel was positive for Mycoplasma pneumoniae. Notably, venous duplex showed acute occlusive deep vein thrombosis in the right soleal vein. Patient was managed supportively with warming measures, one unit of packed red blood cells and azithromycin for Mycoplasma. Patient hemoglobin stabilized and hemolysis resolved.

Discussion:

M pneumoniae is a well known cause of CAHA, only few case reports of secondary CAHA have been described. About 60 percent of cases develop hemolysis which is mild or subclinical. Our case of elderly patient with severe hemolysis triggered by cold exposure is uncommon. Cold exposure in CAHA precipitates RBC agglutination leading to complement mediated hemolysis. Prompt recognition and supportive care, including warming and transfusion, are essential in such presentations. Additionally, our patient developed DVT, an unusual but increasingly recognized complication of M pneumoniae infection. Clinicians should be aware of the thrombotic risk, especially when other risk factors are present.

Conclusion:

Resident/Fellow Clinical Vignette

Infectious CAHA should be suspected in patients presenting with anemia, hemolysis, and recent respiratory symptoms. Early identification and supportive care are essential. This case also adds to the emerging evidence linking M pneumoniae to thrombotic complications, highlighting the need for vigilance in evaluating such patients.

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SPESOLIMAB AS AN EMERGING THERAPY IN GENERALIZED PUSTULAR PSORIASIS

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Generalized pustular psoriasis (GPP) is a rare, potentially life-threatening inflammatory dermatosis characterized by widespread sterile pustules on an erythematous base. Despite its severity, GPP is often misdiagnosed due to its clinical overlap with other pustular eruptions, leading to delays in treatment and significant morbidity.

We report the case of a 40-year-old woman who initially presented with fatigue, joint pain, and Raynaud's phenomenon. Laboratory evaluation revealed a high-titer ANA with a negative extended autoimmune panel, resulting in a diagnosis of undifferentiated connective tissue disease. She was started on hydroxychloroquine (HCQ) for symptom management. Within two weeks, she developed a painful, erythematous rash beginning on her wrist and thigh, which rapidly progressed to involve more than 40% of her body surface area. Physical examination revealed diffuse erythematous maculopapular plaques with early pustulation, without mucosal involvement. Initial treatment with systemic corticosteroids led to only transient improvement.

Despite increasing the steroid dose and discontinuing HCQ, the eruption worsened, eventually involving over 60% of her skin. Dermatology evaluation and repeat skin biopsy revealed subcorneal pustules rich in neutrophils and lacking eosinophilic infiltration, supporting a diagnosis of moderate-to-severe GPP. Although Acute Generalized Exanthematous Pustulosis (AGEP) was initially considered due to the temporal association with HCQ initiation, it was ruled out based on poor steroid response, absence of intertriginous involvement, and histopathologic findings.

Given the extensive disease and steroid-refractory course, intravenous Spesolimab, an IL-36 receptor antagonist, was initiated. The patient witnessed marked improvement within days, with significant reduction in pustules, erythema, and burning sensation. A second IV dose was administered two weeks later, resulting in sustained clinical remission and a slow prednisone taper. Plans were made to transition to subcutaneous Spesolimab for long-term maintenance.

This case highlights the diagnostic complexity of GPP, particularly when triggered by medications such as hydroxychloroquine. Timely histopathologic confirmation and early dermatologic input are essential for distinguishing GPP from mimics like AGEP. Spesolimab, by targeting the IL-36 inflammatory pathway, offers a promising therapeutic option for patients with moderate-to-severe GPP, especially those unresponsive to conventional therapies. Increased awareness and accessibility of IL-36 inhibitors may facilitate earlier diagnosis and intervention, ultimately improving patient outcomes.

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A rare case of Atopobium vaginae bacteremia following uterine artery embolization causing septic shock and encephalopathy

Introduction

Atopobium vaginae is an anaerobic Gram-positive bacterium primarily associated with bacterial vaginosis (BV). It is predominantly known for its role in BV. Invasive infection and bacteremia have been rarely reported. Previous studies and case reports show that A. vaginae can occasionally translocate from the genital tract into the bloodstream, leading to bacteremia in the context of obstetric or gynecology-related procedures or complications, particularly intrapartum. A. vaginae is frequently found in the polymicrobial biofilm that forms on the vaginal epithelium in BV, often in conjunction with Gardnerella vaginalis. This biofilm is thought to contribute to the persistence and recurrence of BV by providing a protective environment for the bacteria against antimicrobial treatments.

Case

We report the case of a woman who presented with malodorous vaginal discharge, fever, chills, nausea, and abdominal pain following a recent uterine artery embolization. On evaluation, she was found to have severe anemia, leukocytosis, and elevated lactate levels. Computed tomography of the abdomen and pelvis revealed uterine masses with solid components, fluid, and gas. A presumptive diagnosis of sepsis secondary to tissue necrosis from degenerating fibroids and anemia was made. Empiric intravenous ceftriaxone, doxycycline, and metronidazole were initiated, and she was transfused with 2 units of packed red blood cells. The patient's condition rapidly deteriorated, progressing to septic shock and encephalopathy, culminating in cardiac arrest and death. Blood cultures drawn during hospitalization were positive for Atopobium species.

Discussion

The patient presented with symptoms that are commonly expected after uterine artery embolizationâ€”fever, abdominal pain, nausea, and vomiting. However, it is imperative to evaluate all risk factors on an individual basis. In this case, the patient had anemia and diabetes mellitus, which placed her in a more immunocompromised state. Treatment typically involves antibiotics effective against anaerobic bacteria, with Î²-lactams and clindamycin being preferred options due to the resistance of many A. vaginae strains to metronidazole.

Conclusion

In cases of bacteremia or genital tract infections caused by anaerobic bacteria during labor or following gynecologic surgery or related procedures, particularly among patients exhibiting signs of bacterial vaginosisâ€”consideration should be given to the inclusion of Atopobium vaginae in the antimicrobial treatment regimen. Prompt identification and administration of appropriate antimicrobial therapy are essential to ensure favorable clinical outcomes.

Crystal Moras

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A Medical and Psychiatry conundrum: Managing multiple medical comorbidities in the setting of intricate psychiatric complexity and healthcare burden

Introduction:

In clinical practice, it is common to encounter patients with history of factitious disorders or malingering. However, if patients have multiple psychiatric disorders with accompanying complex medical issues, these patients are not treated in the appropriate medical setting. We present a patient with complex medical and psychiatry history from our institution with multiple overlapping features of factitious disorder, malingering, and pseudologia fanatstica with extensive underlying medical history who was admitted to our medicine unit. Due to her complex psychiatry history, it posed multiple clinical dilemmas which led to overutilization of medical resources in her admission.

Case Description:

A 35-year-old single woman with a complex medical history of long QT syndrome with Implantable cardioverter defibrillator (ICD) placement, stroke, pulmonary embolism on eliquis fibromuscular dysplasia, seizure disorder, asthma, polysubstance abuse, non-prescribed medication utilization, presence of suprapubic catheter and percutaneous gastrojejunal (GJ) tube, and homeless. Her psychiatric history encompassed of borderline personality disorder, post-traumatic stress disorder, restrictive eating disorder, factitious disorder, malingering, and pseudologia fantastica. Furthermore, she has an allergy to 28 different medications, unclear if they are true allergies. The patient was admitted to our institution for a failed suicidal attempt and drug overdose; patient called emergency medical services and was brought to the emergency department (ED). In the ED, the patient appeared significantly altered and was intubated for airway protection. On the following day, the patient was extubated. However, the patient was admitted for more than 60 days due to lack of proper placement. During her admission, she frequently removed her suprapubic and GJ tube. She would state she had an allergy to each and every medication. On daily basis, the patient would tell elaborate stories which complicated her management and delayed hospital discharge. These behaviors became challenging in differentiating between genuine and feigned symptoms. As a result, court treatment order was obtained which allowed treatment by the primary team.

Discussion:

The medical complexity of the case heightened by her dependence on a suprapubic catheter and gastrostomy tube for feeding in the setting of her complex psychiatric history, both of which require careful management and coordination of care. Due to her complex medical history, it is difficult to address her treatment in a standard psychiatric setting. In an ideal setting, a multidisciplinary approach would be essential to provide suitable medical care. Facilities which provide comprehensive care and offer specialized psychiatric support like Dialectical Behavior Therapy (DBT) would be beneficial. This approach would reduce medical burden and overutilization of medical resources in the clinical setting. Efforts for healthcare reform would be beneficial in this population.

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Low Dose Methotrexate Induced Pancytopenia Mediated by Ascites

introduction:

Methotrexate (MTX) treats a variety of oncological and rheumatological conditions. Its active form impairs DNA formation and repair via inhibition of dihydrofolate reductase. MTX is known to cause bone marrow suppression, mucositis, and liver fibrosis, especially at high-doses. Here, we pose a case of low-dose MTX induced pancytopenia, believed to be mediated by abdominal ascites leading to drug sequestration and reduced elimination.

Case Presentation:

A 73-year old male with rheumatoid arthritis, treated with stable MTX dose, was admitted for fatigue and abdominal distension. His history was also significant for squamous cell carcinoma of the tongue, for which he completed cisplatin and radiation one month prior. Examination demonstrated edema as well as abdominal distension. Admission studies demonstrated pancytopenia: white blood cell count 1.21 K/uL (neutrophils 1.10 K/uL), hemoglobin 5.7 g/dL, platelets 13 K/uL and hypoalbuminemia. Serum MTX level was undetectable. Adequate folic acid, B12 and iron stores were confirmed. Infectious work up was unrevealing. Bone marrow biopsy showed hypocellular marrow without underlying bone marrow disorder. Imaging demonstrated ascites, portal hypertension, and cirrhosis. 10L of transudative peritoneal fluid was removed. He was treated with transfusions of red blood cells and platelets without lasting effects. He was started on leucovorin for presumed methotrexate toxicity with subsequent normalization of blood counts and sharp cessation of transfusion requirements.

Discussion:

Case reports document low dose-MTX toxicities, including pancytopenia leading to sepsis and death, across various serum MTX levels and treatment durations. A 2014 retrospective study showed 78.5% of patients on low dose MTX developed pancytopenia, without correlation between serum MTX levels and severity of pancytopenia. Thus, in this case, the patient experiences MTX toxicity despite undetectable serum MTX levels.

Third spacing of fluids augments MTX toxicity through drug sequestration, delayed elimination and falsely reassuring serum levels. In this patient, his concurrent ascites and edema allowed a slow, sustained release and enhanced cytotoxicity. This effect is further confirmed by the patient's resolved pancytopenia following leucovorin therapy and paracentesis.

The timing and intensity of cancer directed therapy for known head and neck malignancy makes chemotoxicity an unlikely contributor to his presentation. The patient received a lower intensity of Cisplatin with 40mg/m2, and completed only two cycles. In addition, myelosuppression secondary to chemotherapy is unlikely to present after several weeks from last treatment cycle.

Conclusion:

Third spacing, a previously overlooked factor, may augment toxicity of low-dose MTX similarly to high-dose MTX. We pose this hypothesis, given that the patient's persistent pancytopenia improved only after administration of leucovorin and concurrent paracentesis, likely due to sequestering of low dose MTX in ascitic fluid. We were unable to obtain peritoneal MTX level to confirm this, but nonetheless, this effect warrants further investigation.

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A SILENT CULPRIT : PROSTETIC VALVE ENDOCARDITIS DUE TO CUTIBACTERIUM ACNES

Introduction:

Cutibacterium acnes is a common skin gram negative commensal organism that causes acnes. It accounts for 6% of prosthetic valve endocarditis. Diagnosis is difficult because of its atypical presentation. These patients most commonly require surgical intervention followed by extended periods of antibiotics for definitive treatment. However, treatment is often delayed due to its atypical features subsequently leading to increased rates of recurrent endocarditis and mortality. Our case highlights a classic example of C. acnes infective endocarditis presented without fever or inflammatory markers and we aim to emphasize the diagnostic challenges associated with C. acnes due to its atypical presentation.

Case:

A 52-year-old man with history of bioprosthetic aortic valve replacement for prior group B Streptococcal endocarditis presented with one week of exertional dyspnea and chest tightness. He denied fever or systemic symptoms. On clinic evaluation, a new systolic murmur was noted, prompting referral to emergency room. Initial labs showed elevated troponin, B-natriuretic peptide, D-dimer, and electrocardiogram showed new T wave inversions. He was admitted with provisional diagnosis of non-ST elevation myocardial infarction and underwent left heart catheterization that revealed normal coronaries.

Transthoracic echocardiogram (TTE) then revealed moderately reduced ejection fraction, and new aortic intra-valvular leak. Transesophageal echocardiogram (TEE) showed severe bioprosthetic aortic stenosis and regurgitation, valve ring dehiscence, a 1.3 cm mass suggestive of vegetation, and peri-valvular abscess. Broad-spectrum antibiotics were initiated, and extensive infectious work up was done. Due to concerning TEE findings, the patient underwent emergent redo aortic valve replacement. Intraoperative inspection did not show classic signs of endocarditis, and preliminary cultures were negative leading to antibiotic discontinuation.

A week post-operatively, one aortic fluid specimen grew Cutibacterium acnes. Antibiotics were restarted, and the patient completed a 6-week course of intravenous ceftriaxone. He had excellent recovery, with resolution of symptoms, return to work, and normal valve function on follow-up echocardiography.

Discussion:

Cutibacterium acnes is a rare but important cause of prosthetic valve endocarditis, often lacking features such as fever or elevated inflammatory markers. Its slow growth frequently leads to negative blood cultures and delayed diagnosis, especially if cultures are obtained after initiating antibiotics.

Resident/Fellow Clinical Vignette

In this case TTE was inconclusive, but TEE revealed a 1.3 cm mass and valve dehiscence. Despite negative intraoperative findings and initial discontinuation of antibiotics, C. acnes was later isolated from valve tissue, prompting a 6-week course of ceftriaxone.

This discrepancy between imaging, intraoperative appearance, and culture results highlights the organism's subclinical behavior and potential for under recognition. Early discontinuation of antibiotics following negative intraoperative findings highlights a common pitfall. Therefore, a multidisciplinary approach, use of TEE, and prolonged culture incubation are essential particularly in patients with risk factors such as male sex, prosthetic valve, and history of prior cardiac surgery/infective endocarditis.

Resident/Fellow Clinical Vignette

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Sister Mary Joseph Nodule: A Rare Presentation of Advanced Hypopharyngeal Squamous Cell Carcinoma

Background:

Sister Mary Joseph nodule (SMJN) is a rare cutaneous manifestation of metastatic intra-abdominal malignancy, most commonly arising from gastrointestinal or gynecologic cancers. Its occurrence in head and neck malignancies, particularly hypopharyngeal squamous cell carcinoma (SCC), is exceedingly rare and portends a poor prognosis.

Purpose:

To describe an unusual case of metastatic hypopharyngeal SCC presenting as an umbilical mass (SMJN), with prior transformation of a PEG tube site into a metastatic lesion.

Case Presentation:

A 67-year-old male with a history of stage IV hypopharyngeal SCC previously treated with chemoradiation presented to the emergency department with a non-healing wound at the prior PEG tube site, two months after PEG removal at a surgical clinic. Abdominopelvic CT demonstrated a soft tissue tract measuring 5.5 cm in anteroposterior dimension at the former PEG site. Biopsy of the abdominal mass revealed moderately differentiated squamous cell carcinoma, consistent with the known primary tumor.

Shortly afterward, the patient developed a firm, erythematous 2 cm umbilical nodule. Repeat imaging revealed peritoneal nodularity and omental caking concerning for peritoneal carcinomatosis. Biopsy of the umbilical lesion confirmed metastatic SCC. PET-CT demonstrated widespread metastases involving the lungs and peritoneum. Given rapid clinical deterioration and poor performance status, palliative care was initiated, and the patient transitioned to hospice, passing away within weeks.

Conclusions:

This case underscores a rare and aggressive metastatic pathway of hypopharyngeal squamous cell carcinoma, presenting first at a prior PEG site and later as a Sister Mary Joseph noduleâ€”an exceedingly uncommon finding in head and neck cancers. The presence of SMJN should alert clinicians to the likelihood of widespread intra-abdominal dissemination and warrants urgent re-staging and multidisciplinary evaluation. Moreover, persistent or atypical healing patterns at prior procedural sites in oncology patients should raise suspicion for cutaneous metastasis. Early recognition is essential to guide prognosis, optimize patient-centered care, and initiate timely goals-of-care discussions.

Lense Negash, MD

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Disseminated MAC Diagnosed by cfDNA NGS in a Patient with AIDS and Severe Thrombocytopenia

Background:

Disseminated Mycobacterium avium complex (MAC) is a potentially fatal opportunistic infection in patients with advanced AIDS. However, diagnosis is frequently delayed due to non-specific symptoms, slow culture growth, and limited feasibility of invasive testing in thrombocytopenic patients, a frequent complication of MAC involving the bone marrow. Next-generation sequencing (NGS) of microbial cell-free DNA (cfDNA) offers a rapid, non-invasive diagnostic alternative in such complex cases.

Case Presentation:

A 33-year-old man with newly diagnosed AIDS (CD4 count: 30 cells/uL; viral load: 447,000 copies/mL) presented with persistent fevers, pancytopenia, and watery diarrhea. He appeared cachectic and febrile on arrival. Lab work revealed worsening pancytopenia (platelets 3,000/uL), elevated liver enzymes, and inflammatory markers. Extensive infectious and hematologic workup, including bacterial, fungal, and acid-fast bacilli (AFB) blood cultures, viral panels, stool studies, and bone marrow biopsy, were inconclusive. Inflammatory markers were elevatedâ€”ferritin 1,462 ng/mL, C-reactive protein (CRP) 95.9 mg/L, lactate dehydrogenase (LDH) 426 U/L, soluble interleukin-2 receptor (sIL-2R) 1,712.5 pg/mL. CT imaging showed prominent abdominal para-aortic and celiac lymphadenopathies.â€¨â€¨Given such presentations, hemophagocytic lymphohistiocytosis (HLH) and lymphoma were considered after an exhaustive, non-revealing infectious workup. Bone marrow biopsy ruled out malignancy, and diagnostic criteria for HLH were not met.â€¨â€¨Due to ongoing deterioration and diagnostic uncertainty, plasma cfDNA NGS (Karius test) was obtained and detected M. avium within four days. Empiric therapy with azithromycin, ethambutol, and rifabutin was initiated, along with corticosteroids for suspected immune-mediated thrombocytopenia. Clinical improvement followed, with defervescence and rising platelet counts. One month later, fungal blood cultures confirmed MAC. The patient remained afebrile and clinically improved on follow-up.

Conclusion:

This case underscores the diagnostic power of NGS in immunocompromised hosts with fever of unknown origin and cytopenia. While disseminated MAC typically requires prolonged culture time or invasive biopsy, plasma NGS enabled rapid diagnosis and targeted therapy. Our case aligns with emerging literature supporting the utility of cfDNA testing, especially when conventional workup fails or is contraindicated.â€¨â€¨NGS-based cfDNA testing can be lifesaving in complex cases of diagnostic uncertainty. In patients with AIDS and suspected opportunistic infections, NGS offers a faster path to diagnosis and treatment, particularly when traditional diagnostics fall short. While it remains an important tool, clinicians should use Karius testing judiciously, as it detects a wide range of non-pathogenic DNA as well.

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Infectious Aortitis Due to Non-Typhoidal Salmonella: A Rare but Lethal Entity

Aortitis is an inflammation of the aortic wall that can be either infectious or non-infectious. Infectious aortitis is rare, but when caused by staphylococcus aureus, streptococcal species, or gram-negative bacilli, it can lead to life-threatening complications like mycotic aneurysm and aortic rupture if left untreated. We present a case of Salmonella-induced infrarenal aortitis that rapidly progressed to aneurysm, requiring urgent surgical repair and long-term antibiotic therapy.

Case:

65-year-old female with a medical history of coronary artery disease status post-stenting (2009), hypertension, diabetes mellitus, hyperlipidemia, and chronic tobacco use (approximately 10 cigarettes daily since age 14). Her surgical history includes C3-C6 laminectomy and fusion (2020). She also has a history of depression.

She presented with one week of lower abdominal pain, body aches, chills, and nausea, which began shortly after returning from a 7-day trip to Jamaica. In the emergency department, she was hemodynamically stable with a blood pressure of 135/81 mmHg, heart rate of 72 bpm, respiratory rate of 16, temperature of 98.9°F, and oxygen saturation of 100% on room air. Physical examination was notable for lower abdominal tenderness.

Laboratory workup revealed elevated inflammatory markers (ESR 104, CRP 22) and mild transaminitis (AST 66, ALT 106). CT of the abdomen and pelvis demonstrated infrarenal aortitis without dissection or aneurysm. Patient was started on intravenous ceftriaxone. A multidisciplinary teamâ€”including infectious disease, rheumatology, and vascular surgeryâ€”was consulted to guide management.

Blood cultures grew Salmonella species (non-typhi/paratyphi). Evaluation for syphilis, tuberculosis, and autoimmune disordersâ€”including lupus, rheumatoid arthritis, and vasculitisâ€”was unremarkable. Transthoracic echocardiogram showed no vegetation.

On hospital day 4, she developed sudden, severe back pain. A STAT repeat CTA of the abdomen and pelvis showed progression of the aortitis with development of a 4 cm infrarenal mycotic aneurysm and contained rupture. Vascular surgery was urgently reconsulted. Patient underwent open surgical repair using a rifampin-soaked graft and omental patch. Cultures from the aortic wall swab eventually grew rare Salmonella species. She was started on IV ertapenem for 12 weeks and advised to continue lifelong oral suppressive therapy with Bactrim. Postoperatively, she remained hemodynamically stable and was advised to follow up closely with infectious disease and vascular surgery. Repeat imaging was scheduled in two weeks to monitor for recurrence or progression.

Discussion and Conclusion

This case highlights the importance of maintaining a high index of suspicion for Salmonella bacteremia in high-risk individuals, particularly in elderly patients, those with underlying cardiovascular disease or recent travel history. Prompt recognition and a multidisciplinary approach are critical in managing infectious aortitis, given its potential for rapid progression to aneurysm and rupture. Long-term antibiotic therapy and surgical intervention are often necessary to achieve favorable outcomes.

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TERBINAFINE-INDUCED DRESS, SJS AND LIVER INJURY: A TRIPLE CHALLENGE

Introduction:

Terbinafine, antifungal, is usually well tolerated but can rarely cause severe reactions. We report a case of female with Stevens-Johnson Syndrome (SJS), DRESS syndrome, and drug-induced liver injury (DILI). This case highlights the importance of early recognition, prompt drug withdrawal, and timely treatment to reduce morbidity and mortality associated with drug-induced hypersensitivity and autoimmune reactions.

Case description

55-year-old woman with past medical history of esophagitis presented with jaundice and a diffuse rash 34 days after initiating terbinafine for left hallux onychomycosis. She reported generalized pruritus within the first week of terbinafine use. During the second week, she developed dark-colored urine. By third week, she noticed a maculopapular rash on her abdomen and yellowing of the skin. After 23rd dose, she self-discontinued terbinafine. However, the rash worsened, spreading to her torso, extremities, palm and sole. Admission labs showed a total bilirubin of 12.7 mg/dL, direct bilirubin of 7.6 mg/dL, AST 277 U/L, ALT 409 U/L, and ALP 296 U/L. She denied alcohol use, recent travel, consumption of raw meats, or a history of hepatitis. Immunology consultation raised concern for DILI secondary to terbinafine. Ursodiol and diphenhydramine were initiated. On day two, although LFT began to improve, eosinophil counts rose markedly (from 2.4% to 18.9%), and the patient developed odynophagia with new mucosal ulcerations. Laryngoscopy identified white ulcers at the tongue base without airway compromise. Skin biopsy revealed interface dermatitis with epidermal necrosis and rare eosinophils, consistent with SJS. She was treated with broad-spectrum antibiotics, methylprednisolone, and IVIG. She was transferred to a regional burn center for further management.

Discussion

Our case highlights a rare and complex presentation of severe cutaneous adverse reactions (SCARs) involving overlapping SJS and DRESS, both triggered by terbinafine, a commonly used antifungal. Although SJS and DRESS are distinct T-cell-mediated hypersensitivity reactions with unique clinical and histopathological features, their coexistence is uncommon and may indicate a shared immunopathogenic mechanism. Prior studies have reported autoimmune diseases emerging after SCARs, possibly due to extensive tissue injury from drug reactions causing immune dysregulation and unmasking latent autoimmunity in genetically susceptible individuals. This supports the "danger signalâ€ hypothesis, where keratinocyte apoptosis and cytokine release activate auto-reactive

lymphocytes. Early recognition and awareness of such diagnostic challenges are essential to improve patient outcomes and quality of life.

Conclusion

Identifying unusual and overlapping SCARs is crucial, especially in patients with systemic symptoms. Greater awareness enables quicker diagnosis, prompt discontinuation of the causative drug, proper immunosuppressive treatment, and vigilant monitoring for autoimmune complications. Internists, often the initial providers, must recognize these signs to enhance patient outcomes and lower long-term morbidity.

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Eastern Equine Encephalitis: A Rare Case with Minimal Drama in the Brain's Theater

Introduction

Eastern Equine Encephalitis (EEE) is a rare zoonotic viral disease caused by the Eastern Equine Encephalitis Virus (EEEV), which belongs to the Togaviridae family. While human infections are uncommon, they carry a high mortality rate of approximately 33%. Survivors often experience significant neurological sequelae. We present a rare case of EEE in a 91-year-old patient who showed a rapid improvement in symptoms with only minor neurological consequences.

Case Presentation

A 91-year-old female with a past medical history of coronary artery disease (CAD), osteoporosis, hyperlipidemia, and a right frontoparietal meningioma, presented with a few days of new onset slurred speech, drooling, headache, and mild dysphagia. On initial evaluation, she exhibited flattening of the left nasolabial fold and persistent slurred speech.

Initial imaging, including CT head and CTA head and neck, showed no acute pathology but confirmed the presence of her known meningioma with mass effect on the right ventricle. She was admitted to the medicine floor. Tick-borne and viral panels were negative.

During her hospitalization, she developed worsening left-sided motor weakness, although repeated imaging showed no acute changes. Her condition was complicated by high-grade fevers, and she was empirically treated with IV Unasyn for 7 days. A lumbar puncture revealed elevated protein levels in the cerebrospinal fluid (CSF). The CSF was sent to the CDC for further analysis, and testing confirmed the presence of both Powassan virus and Eastern Equine Encephalitis Virus.

The patient received regular physical therapy contributing to gradual improvement and she was discharged to a subacute rehabilitation facility.

Upon re-evaluation during her stay in the rehabilitation facility, the patient showed significant recovery in motor and bulbar functions. However, mild cognitive symptoms persisted.

Discussion

Eastern Equine Encephalitis Virus (EEEV) is known to have high mortality and significant neurological sequela. However, as illustrated by this case, some individuals may experience a much milder course with minimal neurological effects. Our patient, despite being 91 years old with multiple comorbidities, showed rapid improvement after treatment. This suggests that factors such as the patient's immune response, viral load, and the timeliness of intervention might influence the severity of disease.

This case also highlights the potential for co-infection with other arboviruses, such as Powassan virus, which was detected alongside EEEV in the patient. Co-infections could complicate the clinical picture

and impact disease progression, though our patient's rapid recovery suggests that prompt diagnosis and supportive care can result in favorable outcomes even in elderly patients with underlying comorbidities.

Conclusion

While EEE is typically associated with severe outcomes, some cases may present with milder symptoms and better prognoses. Further research is needed to identify factors that contribute to these variations in disease severity and to improve diagnosis and management for patients with EEE.

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Beyond the First NET: A Case of Pancreatic Neuroendocrine Tumor Diagnosed a Decade After Lung Carcinoid

Introduction:â€¯

Lung carcinoid tumors, a subtype of lung neuroendocrine tumors (NETs), represent 1-2% of lung cancers, are frequently asymptomatic, and are incidentally diagnosed. Atypical variants metastasize up to 70% of the time. Pancreatic NETs (pNETs) comprise 1-2% of pancreatic malignancies and are often metastatic. Lung carcinoid tumors rarely metastasize to the pancreas. The occurrence of two distinct primary NETs in a single patient is exceedingly rare, especially without genetic syndromes like multiple endocrine neoplasia 1 (MEN1). Even for MEN1 is only pursued in certain NET types or when there is family history of MEN1. However, patients with NETs face a higher risk of developing second primary malignancies (SPMs), with an incidence of 25%. We present a patient with a history of lung carcinoid who developed valvulopathy that led to the diagnosis of a pNET almost a decade following his first NET diagnosis, emphasizing the necessity of heightened clinical suspicion and vigilant surveillance for SPMs, including second NETs, in patients previously diagnosed with NETs.

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Case Presentation:â€¯

A 36-year-old man with a history of lung carcinoid (status post left lower lobectomy), bipolar disorder managed with lithium, and bicuspid aortic valve presented with two weeks of progressive dyspnea, orthopnea, nausea, and vomiting. Physical exam demonstrated bilateral lower extremity edema. Laboratory evaluation revealed elevated brain natriuretic peptide (1143 pg/mL). Electrocardiography indicated new left bundle branch block, and echocardiography reported newly reduced EF <20%, severe mitral regurgitation, and moderate to severe aortic regurgitation without PFO. Diuresis and guideline-directed medical therapy were started. Left and right heart catheterization ruled out ischemic cardiomyopathy. CT thorax was obtained to assess for recurrence of lung NET, which incidentally identified several pancreatic masses with hepatic lesions were identified. Pancreatic biopsy confirmed a well-differentiated neuroendocrine tumor (WHO grade 1, Ki-67 <3%). Labs revealed mildly elevated serum 5-HIAA and chromogranin A levels. Additionally, duodenal biopsy incidentally identified celiac disease. Genetic testing was negative for NET syndromes, including MEN. Subsequent PET Dotatate revealed extensive metastases involving pancreas, liver, lymph nodes, bone, brain and left cardiac apex. The patient underwent aortic valve replacement, and initiated octreotide therapy. â€¯

Discussion:

SPMs reportedly occur in up to 25% of patients with NETs, and the risk of developing an SPM is higher in older patients. Patients who develop SPMs following NET diagnosis can experience significant morbidity and have worse overall survival and cancer-specific survival. However, there are presently no clear guidelines for surveillance for SPMs in patients with NETs. Our case report demonstrates that the development of a second NET is rare but possible and highlights the need for vigilant, lifelong surveillance of all patients with NETs for SPMs, including second neuroendocrine tumors, especially in the context of new, unexplained symptoms.â€¯

Resident/Fellow Clinical Vignette

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Severe Thrombocytopenia and Stroke in a Young Woman: An Uncommon Source of Immune Activation

Introduction:

We describe a rare and diagnostically challenging presentation of heparin-induced thrombocytopenia (HIT) leading to acute ischemic stroke in a young woman, highlighting the critical importance of a comprehensive medication history that includes unconventional exposures.

Case Description:

A 36-year-old female with history of T6-T9 syrinx and bladder spasms presented to the Emergency Department with nausea, vomiting, fatigue, and transient word-finding difficulty. Neurologic examination was non-focal, and her symptoms improved during evaluation. Laboratory testing revealed thrombocytopenia (platelets 31x10³/Î¼L). She was ultimately discharged with recommended outpatient Hematology follow up. She returned the next day with chest pressure, diaphoresis, diffuse weakness, new-onset right lower facial droop, diplopia with downward gaze, and intermittent dysarthria. Brain MRI demonstrated a right frontal FLAIR hyperintensity with diffusion restriction, consistent with an acute cortical infarction. CT angiography and bilateral lower extremity duplex studies were negative for thromboembolism. Transthoracic echocardiogram with agitated saline contrast revealed a moderate patent foramen ovale (PFO). Aspirin and Atorvastatin were initiated, and Clopidogrel was deferred due to continued thrombocytopenia. Due to the unusual presentation of ischemic stroke and isolated thrombocytopenia in a young woman without traditional risk factors, a comprehensive diagnostic evaluation was pursued. Autoimmune workup including antinuclear antibodies and antiphospholipid antibodies were negative. Hypercoagulable panel including Factor V Leiden, prothrombin gene mutation, and protein C and S levels were unremarkable. Peripheral smear showed no schistocytes or evidence of microangiopathy. Bilateral upper extremity duplex ultrasound revealed superficial venous thrombi, raising concern for a systemic prothrombotic process. A 4T's score of 6 and a positive anti-platelet factor 4 (PF4) antibody ELISA suggested high probability of HIT. Serotonin release assay (SRA) was performed using both low-dose and high-dose porcine heparin. The test demonstrated strong platelet activation at low-dose heparin (82%) with suppression at high-dose heparin (0%), consistent with a positive SRA and diagnostic of HIT, despite no known heparin exposure. On detailed history review, the patient had self-administered an injectable medication, Pentosan Polysulfate (PPS), for bladder spasms, which she had purchased online. PPS is a semisynthetic pentasaccharide with anticoagulant properties similar to low molecular weight heparin. While an oral formulation is FDA-approved for interstitial cystitis, injectable PPS is not approved for human use and is restricted to veterinary medicine, primarily for canine osteoarthritis. The patient injected two doses in the preceding week, the second immediately before symptom onset. PPS was therefore identified as the likely HIT trigger and the patient was treated with Argatroban and transitioned to Apixaban, with platelet count recovery to 204x10³/Î¼L.

Resident/Fellow Clinical Vignette

Discussion:

This case illustrates the importance of maintaining a broad differential when encountering unexplained thrombocytopenia and thrombosis, as HIT may arise from unconventional exposures. Obtaining a detailed medication history, which includes unregulated and non-prescribed medications, is essential to identifying a diagnosis.

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Atypical Presentation of Late-Onset Familial Mediterranean Fever in a 71-Year-Old Male

Introduction:

Familial mediterranean fever (FMF) is an autoinflammatory disorder characterized by episodic fevers, serositis, and elevated inflammatory markers. Approximately 80-90% of cases are diagnosed in childhood or early adulthood. FMF is thought to be caused by mutations in the mediterranean fever gene (MEFV), which encodes pyrin, a key regulator of the innate immune system. Late-onset FMF is rare and presents a significant diagnostic challenge.

Case Description:

We report a case of a 71-year-old male with a relevant past medical history of gout and sarcoidosis who presented with 1-2 weeks of fever (Tmax 38.3C), night sweats, malaise, myalgias, and lower extremity weakness. Family history was significant for multiple cancers and Crohn's disease. Laboratory studies were significant for leukocytosis (WBC 16.0 ×10â¹/L, 91% neutrophils), normocytic anemia (Hgb 10.0 g/dL, MCV 81.9 fL), thrombocytosis (platelets 455 ×10â¹/L), elevated LDH (283 U/L), ferritin (3,490 ng/mL), ESR >130 mm/hr, CRP 12.1 mg/dL, and mild transaminitis (AST 92 U/L, ALT 136 U/L). A broad infectious evaluation including blood cultures, tick borne disease panel, HIV, hepatitis, were negative. Bone marrow biopsy and computed tomography of the chest, abdomen, and pelvis were unrevealing. Given the lack of response to antibiotics and unrevealing infectious workup, rheumatology was consulted. Autoimmune serologies including anti-nuclear antibody, rheumatoid factor (RF), anti-CCP antibody, anti-double stranded DNA antibody, CPK, ribonucleoprotein antibodies, anti-SSA/SSB antibodies, anti-smith antibody, SCL-70 IgG antibody, JO-1 antibody, centromere B antibody, anti-chromatin antibody, ribosomal protein, anti-MPO IgG, anti-PR3 IgG, and glomerular basement membrane antibody were all negative. Given persistent fevers and inflammation, periodic fever syndrome genetic testing was pursued. For 2 weeks, the patient continued to exhibit fevers and myalgias. Ultimately, periodic fever syndrome genetic testing was positive for a heterozygous mutation in MEFV R761H and a NOD2 intronic variant. Based on clinical presentation and these findings he was diagnosed with FMF and started on colchicine for treatment.

Discussion:
Elderly patients with new-onset FMF are a relatively understudied population. Although uncommon, there is evidence in the literature that MEFV R761H is associated with FMF, and that NOD2 intronic variants may contribute to an atypical presentation. The MEFV R761H variant is associated with a few clusters of families in Southern Italy with very late-onset FMF. While patients with a NOD2 mutation are not more susceptible to developing FMF, the presence of these mutations in FMF patients is associated with more severe disease. NOD2 variants may act as modifiers to MEFV mutations. Suspicion for autoinflammatory disease and genetic testing should be considered in cases of persistent fever of unknown origin in older adults when standard work-up is inconclusive for fever or inflammation of unknown origin.

Ethar Salman, MD

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Olmesartan-Induced Enteropathy with Cutaneous Manifestations: A Rare Clinical Vignette

Introduction:

Angiotensin receptor blockers (ARBs) are widely prescribed antihypertensive medications with a generally favorable side effect profile. However, a rare adverse effect is enteropathy, an immune-mediated condition typically triggered by gluten ingestion that has recently been linked to non-celiac causes, creating diagnostic challenges for clinicians. Among these is olmesartan-induced enteropathy (OIE). While celiac disease is often the first diagnosis to be considered, other potential causes, such as medication-induced enteropathy, infections, inflammatory bowel disease, and autoimmune disorders, must also be evaluated. We present a case of OIE characterized by significant weight loss and rash following one year of medication use, resolved by medication discontinuation.

Case presentation:

A 57-year-old female with hypertension and dyslipidemia presented with a two-month history of daily large-volume watery stools with 30lbs of weight loss. She denied abdominal pain, nausea, fever, vomiting, a history of inflammatory bowel disease (IBD), or recent travel. Her home medications included cetirizine, famotidine, and olmesartan. Physical examination revealed a diffuse maculopapular rash, hyperactive bowel sounds without abdominal tenderness. Laboratory workup was remarkable for elevated creatinine, liver enzymes, C-reactive protein, stool calprotectin, and lactoferrin. However, Serum tissue transglutaminase antibody IgA, 5-HIAA, hepatitis, and bile acids were within normal limits. Stool studies for bacterial pathogens and Giardia were negative. Abdominal CT imaging showed small and large bowel distention without wall thickening, findings consistent with nonspecific diarrheal illness or colitis. Due to persistent symptoms, an esophagogastroduodenoscopy (EGD) and colonoscopy were performed. Biopsies revealed intraepithelial lymphocytosis, crypt hyperplasia, and villous atrophy consistent with Marsh 3C classification, and lymphoid aggregates on the terminal ileum. Following the discontinuation of olmesartan, the patient's symptoms resolved, confirming a diagnosis of olmesartan-induced enteropathy.

Discussion:

Recognizing OIE is challenging, as it is a diagnosis of exclusion. The most common clinical symptoms include chronic non-bloody diarrhea, unintentional weight loss, and abdominal discomfort. Histologically, it exhibits features similar to celiac disease, such as villous flattening or atrophy, intraepithelial lymphocytosis, and lamina propria inflammation. However, it is distinguished from celiac disease by the absence of positive serological markers and the lack of improvement with a gluten-free diet. Discontinuing olmesartan leads to symptom resolution and progressive histological recovery. The exact pathophysiology remains unclear, but the delayed onset of symptoms after prolonged drug exposure suggests a role for cell-mediated immunity. Additionally, evidence indicates the upregulation

of pro-apoptotic proteins, such as Bax and GATA-6, and the downregulation of anti-apoptotic proteins like BCL-2, which promotes apoptosis of intestinal epithelial cells, resulting in villous atrophy. To the best of our knowledge, this is the first reported case in the literature of olmesartan-induced enteropathy associated with a rash.

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EKOS-Assisted Pharmacomechanical Thrombolysis for Extensive Portal Vein Thrombosis in a Non-Cirrhotic Patient

Introduction

Portal vein thrombosis (PVT) is a serious condition resulting from a thrombus within the portal vein trunk or its branches. The leading cause of PVT is liver cirrhosis; however, it primarily results from inherited or acquired prothrombotic conditions and malignancies in a non-cirrhotic liver. Early treatment is vital to achieve portal vein recanalization and prevent complications such as mesenteric venous infarction or portal hypertension. Current guidelines recommend anticoagulation as the first-line treatment for acute PVT, provided there are no contraindications. We present a case of extensive portal system thrombosis in a non-cirrhotic liver that was successfully treated via TIPS (Transjugular Intrahepatic Portosystemic Shunt) placement with mechanical thrombectomy utilizing a 12 Fr Penumbra catheter, followed by pharmacological thrombolysis utilizing infusion catheters with EKOS.

Case Presentation

A 79-year-old female with a history of breast cancer in remission, hypothyroidism, hyperlipidemia, and iron deficiency anemia presented with epigastric pain, fullness, loss of appetite, fatigue, low-grade fever, and chills without a history of liver cirrhosis. Initial workup indicated a urinary tract infection, which was treated with cefdinir. Laboratory results showed mild transaminitis, and imaging did not reveal acute gastrointestinal abnormalities. She returned with fever and weakness after a few weeks. Laboratory testing revealed leukocytosis, mild anemia, and worsening transaminitis. A contrast-enhanced computed tomography revealed occlusion of the portal vein, portions of the superior mesenteric vein, and intrahepatic portal venous branches, evolving cavernous transformation, and new globular hypoattenuation areas throughout the liver, suggesting infarctions without evidence of bowel ischemia. Due to extensive thrombosis and necessity for quick recanalization of the portal vein, a Heparin drip was initiated and a multidisciplinary meeting including GI, general surgery, hematology, and interventional radiology decided to proceed with TIPS placement with mechanical thrombectomy utilizing a 12 Fr Penumbra catheter, followed by pharmacological thrombolysis utilizing infusion catheters with EKOS. Two infusion catheters for TPA (tissue Plasminogen activator) were placed, one via the TIPS extending to the superior mesenteric vein and one via transhepatic access of the right portal vein extending to the spleen vein. TPA was infused at a rate of 1 mg/hour for 24 hours.

Discussion

Advanced treatment of portal vein thrombosis involves endovascular management, including mechanical thrombectomy, catheter-directed thrombolysis (CDT), and TIPS placement. Ultrasound-assisted CDT may be particularly effective in extensive thrombosis involving the portal, mesenteric, and splenic veins, where access is challenging and rapid flow restoration is critical. Energy emitted by EKOS catheters enhances fibrinolysis by disaggregating fibrin networks and facilitating deeper tissue penetration of the thrombolytic agent, even in dense thrombus matrices. An endovascular approach for acute PVT should be considered after thorough multidisciplinary evaluation.

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Not Cutis, Just Nodules That Pointed the Way

Introduction

Leukemia cutis is a rare manifestation of hematologic malignancies, most commonly associated with acute myeloid leukemia (AML) and chronic myelomonocytic leukemia (CMML). It presents as erythematous nodules or plaques and often signals poor prognosis. Rarely, it precedes systemic disease, underscoring the importance of early recognition. We present a case of a 68-year-old male with no prior hematologic history who presented with widespread cutaneous lesions and was ultimately diagnosed with CMML.

Case Presentation

A 68-year-old man with a history of gout presented with five days of altered mental status, decreased appetite, fatigue, chills, and tremors. On arrival, he was febrile (105°F), tachycardic, tachypneic, and hypoxic (SpO2 91%). Physical exam revealed scattered, painless erythematous nodules on the trunk and limbs, an irregular pulse, and bilateral upper extremity tremors. Labs showed hyponatremia (Na 129 mmol/L), elevated BUN/Cr (44/2.64 mg/dL), transaminitis (ALT 70, AST 97 U/L), and elevated procalcitonin (2.55 ng/mL). WBC was 18.6K.

Peripheral smear revealed dysplastic immature cells. Flow cytometry showed 3.5% circulating myeloblasts with abnormal myelomonocytic maturation. Hematology was consulted, and workup for myeloid neoplasm was initiated. A skin biopsy showed lymphocytic inflammation but did not rule out leukemia cutis. Despite broad-spectrum antibiotics, the patient's leukocytosis and fevers persisted. Bone marrow biopsy confirmed CMML. Azacitidine was initiated.

Discussion

This case highlights the importance of recognizing leukemia cutis as a potential presenting sign of CMML, especially in patients without known hematologic disease. Leukemia cutis can precede or accompany systemic disease and may mimic infection or inflammatory dermatoses. In this patient, persistent fever, constitutional symptoms, and cutaneous findings raised concern for malignancy despite inconclusive skin biopsy. Diagnosis was supported by smear, flow cytometry, and confirmed by bone marrow biopsy. Early initiation of Azacitidine may help improve hematopoiesis and delay disease progression.

Conclusion

Leukemia cutis can be an early sign of hematologic malignancy. High suspicion and prompt evaluation can lead to earlier diagnosis and improved outcomes, particularly in CMML.

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VANCOMYCIN-INDUCED DRESS SYNDROME MIMICKING PERSISTENT SEPSIS

Introduction:

DRESS (Drug Reaction with Eosinophilia and Systemic Symptoms) is a rare but potentially life-threatening hypersensitivity reaction. Its presentation often overlaps with sepsis, autoimmune diseases, or malignancy, leading to diagnostic delays. Prompt identification is critical, especially in medically complex patients where symptom overlaps are common, and misdiagnosis can lead to unnecessary interventions and worsening outcomes.

Case Presentation:

A 68-year-old male with end-stage renal disease on hemodialysis, insulin-dependent type 2 diabetes, hypertension, and hyperlipidemia was admitted with right knee pain. He was diagnosed with septic arthritis and underwent a right knee joint washout. Synovial fluid cultures grew Staphylococcus aureus. The patient was started on empiric vancomycin, which was later de-escalated to cefazolin based on sensitivities. His condition initially improved, and discharge was planned with a two-week course of IV cefazolin.

However, on hospital day 10, he developed new-onset fevers up to 101.9°F. Repeat blood and joint cultures remained negative. His leukocytosis worsened (WBC 19.4 ×10â¹/L), and clindamycin was added empirically. Despite escalation in antibiotics, the patient continued to deteriorate. He developed a diffuse, erythematous morbilliform rash, most pronounced on the bilateral lower extremities. Laboratory testing revealed marked eosinophilia (absolute eosinophil count 3.8 ×10â¹/L) and elevated transaminases (ALT 165 U/L, AST 138 U/L).

A CT chest was obtained to evaluate for occult infection and revealed new mediastinal lymphadenopathy. Bronchoscopy with biopsy ruled out malignancy and infection. A skin biopsy was done, which showed interface dermatitis consistent with a drug-induced hypersensitivity reaction. Based on the constellation of findings; rash, eosinophilia, systemic symptoms, and recent antibiotic exposure, a diagnosis of DRESS syndrome was made. Rheumatology determined that vancomycin, started ten days earlier, was the likely trigger, given its established association with DRESS.

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The patient was started on prednisone 60 mg daily with a gradual taper. Within 72 hours, the rash began to fade, fevers resolved, and eosinophil counts normalized. He was discharged home in stable condition with an outpatient follow-up.

Discussion:

DRESS syndrome is often under-recognized due to its delayed onset and nonspecific systemic findings. In this case, the diagnostic picture was obscured by the patient's initial infection and multiple antibiotic exposures. Timely recognition of the drug reaction allowed for steroid initiation and avoided further complications.

Conclusion:

In patients with recent antibiotic exposure and systemic inflammation without a clear infectious source, DRESS should remain high on the differential. Prompt diagnosis and treatment can significantly improve patient outcomes.

Resident/Fellow Clinical Vignette

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Cocaine-Induced QT Prolongation Leading to Pro-arrhythmia from ICD Shocks in a Young Patient

Background: Cocaine use can cause QT prolongation and increase the risk of ventricular arrhythmias. In rare cases, implantable cardioverter-defibrillators (ICDs)â€”intended to terminate arrhythmiasâ€”may paradoxically trigger further ventricular tachycardia (VT) or fibrillation.

Case Presentation:

A 35-year-old woman with a history of nonischemic cardiomyopathy (left ventricular ejection fraction 30%) and a pre-existing dual-chamber ICD for primary prevention presented to the emergency department following eight ICD shocks at home. She described palpitations, dizziness, and one brief episode of light-headedness. She admitted to intranasal cocaine use 4 hours before symptom onset.

On arrival, her vital signs were stable. ECG showed sinus rhythm with a QTc interval of 500 ms. Laboratory investigations revealed hypokalemia (3.5 mmol/L), mild hypomagnesemia (1.6 mg/dL), and positive urine toxicology for cocaine.

ICD interrogation demonstrated several episodes of narrow complex tachycardia (rate 180-190 bpm), unsuccessfully terminated with anti-tachycardia pacing (ATP). However, shocks were followed by polymorphic VT or ventricular fibrillation in several cases, resulting in recurrent therapies. This pattern was consistent with shock-induced pro-arrhythmia in the setting of repolarization vulnerability.

The patient was managed in the cardiac intensive care unit with intravenous potassium and magnesium replacement. Lidocaine infusion and mild sedation were used to suppress ectopy. QTc returned to baseline (460 ms) within 24 hours. No further arrhythmias were observed. The ICD was reprogrammed to reduce shock burden by extending detection intervals and prioritizing ATP.

Discussion: This case demonstrates a rare scenario where ICD shocks inadvertently perpetuated them rather than suppressing arrhythmias due to an acquired proarrhythmic substrate. 1,2 Cocaine-induced QT prolongation, amplified by hypokalemia, predisposed this patient to malignant ventricular arrhythmias triggered by shock-induced repolarization dispersion.

The pathophysiology likely involves a combination of early afterdepolarizations and transmural heterogeneity of repolarization, making shock therapy a potential arrhythmic trigger.3,4,5 While ICDs are indispensable in high-risk populations, clinicians must remain vigilant for reversible causes of arrhythmia that may render shocks ineffective, or even harmful.6,7 This case also highlights the importance of aggressively correcting electrolyte disturbances, avoiding QT-prolonging substances in patients with ICD, and individualizing device programming in patients prone to electrical storms.

Learning Points:

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- Cocaine can cause QT prolongation and increase arrhythmia risk, particularly in the presence of electrolyte abnormalities.
- ICD shocks delivered during repolarization vulnerability can paradoxically precipitate polymorphic VT or VF.
- Patient management should focus on proactively correcting reversible factors, optimizing device settings, and addressing underlying substance use.

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A Deceptive Threat: Aortic Dissection Masquerading as STEMI with Pseudo-Occlusion of Left Main and LAD

Introduction: Acute Aortic Dissection (AAD) is a rare but life-threatening condition, with an incidence of 3-4 cases per 100,000 annually (1). Its variable presentation often mimics other conditions, leading to misdiagnosis and delayed treatment (2). Type A AAD, involving the ascending aorta, is especially critical and requires urgent surgical intervention. This report discusses a rare case of AAD presenting with pseudo-occlusion of the left main coronary artery (LMCA) and the left anterior descending artery (LAD).

Case description: A 44-year-old hypertensive male presented with acute chest pain, shortness of breath, dizziness, and diaphoresis. Elevated troponin and ST-segment changes raised concern for left main or multi-vessel coronary disease, prompting emergency coronary angiography, which showed LMCA pseudo-occlusion and complete LAD occlusion, with inability to engage the left main artery, raising suspicion for aortic dissection. The transesophageal echocardiogram showed dissection originating from the aortic root and extending past visualization of the thoracic aorta. Aortography confirmed the dissection starting from the aortic root and extending through thoracic and abdominal aorta. It also involved the left common carotid artery, extending past the iliac bifurcation, affecting the iliofemoral system, with evidence of decreased perfusion to the true lumen. The patient underwent an emergent Bentall procedure; aortic dissection repair surgery involving aortic valve and aortic root reconstruction, ascending aorta and hemiarch replacement, and CABG. The patient was subsequently transferred to rehab after recovery.

Discussion: This case underscores the diagnostic challenge of Stanford type A AAD, which, while typically presenting with tearing chest pain, can appear atypically (1,3,4). Our patient lacked classic signs like pulse deficits or murmurs, despite having hypertension, a known risk factor. Early recognition is vital, as mortality rises by 1-2% per hour without surgical intervention (4,5). AAD often goes undiagnosed, with up to 55% of patients dying without correct identification (1,6). ECG may indicate ischemia, but it lacks specificity. Computed tomography (CT) scan is the gold standard diagnostic modality with 98-100% sensitivity (4). Surgical repair remains the standard for type A AAD, with coronary angiography helping to detect critical occlusions (7,8). This case highlights the need for prompt imaging, timely intervention, and high clinical suspicion, especially in atypical presentations.

Conclusion: This case underscores the diagnostic challenges of AAD presenting with coronary pseudo-occlusion. Prompt recognition using multimodal imaging, including coronary angiography and CT, is critical for timely surgical intervention. (9).

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Atypical Onset of Anti-NXP2 Dermatomyositis: Unexplained Angioedema and Systemic Edema as Dominant Featuresâ€¨

Introduction:

Dermatomyositis (DM) is an autoimmune inflammatory myopathy marked by proximal muscle weakness, systemic inflammation, and characteristic skin findings. Myositis-specific antibodies, such as anti-nuclear matrix protein 2 (NXP2), define subtypes with distinct organ involvement. Anti-NXP2 DM is associated with pronounced muscle weakness, peripheral edema, dysphagia, and malignancy. While peripheral and facial swelling may occur, angioedema is an underrecognized and diagnostically challenging feature early in the disease.

Case Presentation:

A woman in her late forties with multiple sclerosis presented with progressive abdominal distension, shortness of breath, and new-onset transaminitis. Imaging revealed complex ascites, pleural effusions, and liver nodularity. Ascitic fluid was grossly bloody with a low serum-ascites albumin gradient, suggesting a non-portal hypertensive process. As malignancy was ruled out, her clinical picture evolved to include severe proximal muscle weakness, facial swelling, dysphagia, and generalized edema.

Investigations:

Initial workup included antinuclear antibody (ANA), extractable nuclear antigen (ENA) panel, antineutrophil cytoplasmic antibodies (ANCA), rheumatoid factor (RF), and anti-cyclic citrullinated peptide (anti-CCP) antibodies, all negative. Creatine kinase (CK) levels rose markedly, peaking at 15,000 U/L. Cytokine profiling revealed elevated IL-2, IL-6, IL-8, and IL-10, consistent with a hyperinflammatory state. Magnetic resonance imaging showed diffuse muscle edema, and electromyography demonstrated spontaneous activity with myopathic motor unit potentials. Muscle biopsy revealed inflammatory infiltrates without necrosis. Anti-NXP2 antibodies were identified on extended serologic testing. A heliotrope rash and erythema over the metacarpophalangeal joints later appeared, confirming dermatomyositis.

Treatment and Outcome:

The patient was diagnosed with anti-NXP2 dermatomyositis, complicated by systemic capillary leak syndrome and angioedema. She received high-dose intravenous methylprednisolone followed by a taper, along with intravenous immunoglobulin and rituximab. A percutaneous endoscopic gastrostomy (PEG) tube was placed for nutritional support due to severe dysphagia. Response to IVIG was limited, but significant improvement followed B-cell-directed therapy with rituximab. The patient was

transitioned to oral prednisone and discharged to rehabilitation. She regained independent mobility, her PEG tube was removed, and she remained stable on maintenance therapy with mycophenolate mofetil and tapering corticosteroids.

Conclusion:

This case illustrates an unusual presentation of anti-NXP2 dermatomyositis with angioedema and serosal inflammation, contributing to early diagnostic uncertainty. It highlights the importance of considering inflammatory myopathies in patients presenting with unexplained systemic edema and evolving muscle weakness, even without classic skin findings. Prompt recognition and immunosuppressive therapy can lead to favorable outcomes.

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SYNERGISTIC THROMBOSIS: A DUAL ETIOLOGY OF VTE FROM MAY-THURNER SYNDROME AND FACTOR V LEIDEN MUTATION

Case presentation

A 37-year-old woman with no significant past medical history presented with acute pain and swelling of the left lower extremity. She denied smoking, oral contraceptive use, recent travel, or respiratory symptoms. Family history was notable for a sister with a history of DVT.

On examination, the left leg was edematous and tender. Duplex ultrasonography demonstrated extensive thrombus from the common femoral to the popliteal vein. CT pulmonary angiography revealed multiple emboli in segmental branches of both pulmonary arteries. Transthoracic echocardiogram showed normal right ventricular function. Patient was initially placed on heparin infusion. Persistent lower back pain prompted MRI evaluation.

MRI of the lumbar spine was notable for a thrombus. CT venogram revealed findings suggestive of May Thurner syndrome (MTS). Mechanical venous thrombectomy of the left common iliac, external iliac, common femoral, femoral and popliteal veins was performed along with intravascular ultrasound of the same vessels and placement of venous wall stent from left common iliac vein to external iliac vein. Hypercoagulable workup was notable for heterozygous mutation of factor V leiden and 9% activity of protein C (normal - 70-180%). Patient was discharged on eliquis and plavix with close outpatient follow up with hematology and vascular services.

Discussion

MTS is characterized by compression of the left common iliac vein by the right common iliac artery, predisposing to left-sided iliofemoral deep vein thrombosis (DVT). MTS is anatomically common but clinically underrecognized, accounting for a small proportion of chronic venous disease and deep vein thrombosis (DVT) cases (1-5%).

Verhaeghe R. in 1995 suggested that this syndrome could be associated with genetic factors predisposing to thrombophilia, exposing the case of three patients with activated protein C resistance and who developed MTS. De Bast and Dahin reported three cases of MTS in which all patients were found to be positive for the factor V Leiden mutation. In two cases, young women presented with DVT had other risk factors including smoking and use of oral contraception. The case series hypothesized that exposure to prothrombotic factors such as tobacco and use of oral contraceptives, and prolonged immobility should be avoided in patients that present with a genetic risk for thrombophilia and an iliac venous compression identified by imagery. Our case is different in that it presents a case of MTS without any underlying prothrombotic state and was found to have a genetic susceptibility. The presence of both conditions in a single patient reflects the multifactorial nature of VTE, where anatomical and genetic predispositions can act synergistically to increase thrombotic risk, but neither is known to cause the other directly. This case could be a potential starting point to explore the solitary association of factor V leiden mutation with May Thurner syndrome.

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Giant Cell Arteritis and Essential Thrombocytosis: A Dual Threat for Ischemic Stroke

Giant Cell Arteritis (GCA) is the most common primary systemic vasculitis in patients over 50 years of age, affecting large and medium-sized arteries, especially branches of the external carotid artery. Clinical presentation includes vision changes, constitutional symptoms, and rarely, stroke. We present a case of steroid-refractory GCA complicated by progressive ischemic strokes despite immunosuppressive therapy.

A 63-year-old woman with hypertension and essential thrombocytosis presented with transient left eye vision loss. Imaging revealed a left parietal stroke, moderate stenosis of the left internal carotid artery (ICA), and diffuse intracranial atherosclerosis. She was discharged on dual antiplatelet therapy.

One week later, she presented with complete left eye vision loss and new right-sided hemiparesis. Exam was notable for aphasia with right-sided hemiplegia, hemianopia and facial droop. Laboratory results revealed platelets of 800,000/Î¼L (normal: 150,000-450,000/Î¼L), ESR 3 mm/hour (0-20 mm/hour) and CRP 0.7 mg/dL (<1.0 mg/dL). Brain MRI showed hemorrhagic conversion of the prior infarct and new infarctions in the left middle cerebral artery (MCA), and deep watershed zones. MRA revealed worsening of left ICA stenosis. Temporal artery biopsy confirmed GCA. Additional workup revealed a JAK2 mutation. The remainder of the hypercoagulable workup was negative. Echocardiogram was unremarkable. The patient was treated with IV methylprednisolone, followed by an oral prednisone taper and aspirin monotherapy.

The patient subsequently had multiple presentations for new strokes requiring pulse dose steroids. Despite initial steroid treatment, she experienced progressive right-sided weakness and vision loss. Imaging showed evolving infarcts in the left anterior cerebral artery and MCA. Due to steroid-refractory GCA, tocilizumab was initiated with steroid tapering, which subsequently halted further stroke recurrence.

Stroke occurs in 3-7% of patients with GCA and typically involves the posterior circulation. However, in this case, infarcts were primarily in the anterior and middle cerebral arteries, highlighting the need to consider GCA even in less typical stroke patterns.

Diagnosing GCA-related stroke is challenging, particularly in patients with vascular risk factors. The etiology of stroke in our patient was further confounded by the presence of essential thrombocytosis with a JAK2 mutation, a known prothrombotic state that likely contributed to the burden of recurrent

strokes. While GCA causes vascular inflammation and luminal narrowing, JAK2-driven thrombocytosis can promote thrombus formation, further increasing stroke risk.

Despite high-dose corticosteroids, the patient experienced progressive neurologic events, indicative of steroid-refractory GCA. The addition of tocilizumab, an IL-6 receptor antagonist, has shown benefit in reducing disease activity and steroid dependence and was initiated with subsequent tapering of corticosteroids, without recurrence of cerebrovascular events.

This case emphasizes the importance of recognizing GCA as a potential cause of stroke in older adults and consideration of alternative diagnoses and treatment in cases unresponsive to steroids.

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Double Trouble: Neuromyelitis Optica Following Varicella-Zoster Reactivation

Neuromyelitis optica (NMO) is an autoimmune demyelinating disorder of the central nervous system (CNS). It is characterized by autoantibody-mediated astrocyte injury targeting aquaporin-4 (AQP4), a water channel protein highly expressed in the optic nerves and spinal cord.

A 39-year-old previously healthy woman presented with left periorbital pain and rash, followed by progressive paresthesia in the left upper and lower extremities. She was initially diagnosed with preseptal cellulitis and discharged on cephalexin, but returned four days later with worsening paresthesia. She had recently traveled to Pennsylvania and Upstate New York, but reported no systemic or infectious symptoms. On admission, she was bradycardic to 50 and hypothermic to 96.9°F; an otoscopic exam showed vesicles in the posterior ear. Labs were notable for WBC of 6.82 cells/Î¼L with 90% lymphocytes (20-45%).

MRI brain/spine revealed a T2 hyperintense lesion in the left posterolateral medulla and cervicomedullary junction, suggestive of a demyelinating process. CSF studies showed WBC: 175 cells/Î¼L (0-5 cells/Î¼L), lymphocytes: 90% (40-80%), protein: 63 mg/dL (15-45 mg/dL), and glucose: 48 mg/dL (40-70 mg/dL). CSF studies were negative for infection. The patient received empiric acyclovir and high-dose steroids. Serum varicella-zoster virus (VZV) IgG resulted positive two days later.

Neurologic status worsened over the next nine days with development of dysmetria, pronator drift and worsening left-sided weakness. AQP4-IgG returned positive, confirming NMO. The patient was treated with plasmapheresis and rituximab, with subsequent improvement in symptoms.

VZV reactivation typically presents as herpes zoster, characterized by a painful vesicular rash in a dermatomal distribution. Complications include postherpetic neuralgia, herpes zoster ophthalmicus, meningitis, and cranial neuropathies such as Ramsay Hunt syndrome. In rare cases, VZV reactivation has been associated with CNS involvement and may serve as an immunologic trigger for autoimmune demyelinating conditions. While the exact pathogenesis of NMO remains unclear, infections have been proposed as potential triggers. A systematic review of cases reported between 1975 to July 2020 identified 13 cases of NMO occurring after VZV reactivation, suggesting that VZV may act as an immunologic trigger in susceptible individuals (1).

Our patient's initial presentation with left periorbital pain and vesicular rash indicated zoster reactivation, but the presence of progressive sensory and motor deficits along with MRI findings pointed towards a central demyelinating process. The detection of AQP4-IgG confirmed the diagnosis of NMO.

This case adds to the evidence suggesting that VZV reactivation may precipitate NMO in immunologically susceptible individuals. Clinicians should have a high index of suspicion for NMO in patients presenting with neurological symptoms and a history of recent VZV reactivation, particularly when imaging suggests CNS demyelination. Early diagnosis and initiation of immunotherapy are critical to prevent irreversible neurologic impairment in patients with NMO.

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When Sepsis Isn't Sepsis: Culture-Negative Endocarditis Presenting as Acute Mitral Valve Failure

Introduction

Infective endocarditis (IE) can lead to rapid valvular destruction and cardiogenic shock. Diagnosis is often delayed when symptoms mimic more common conditions such as pneumonia or sepsis. We present a case of presumed culture-negative IE manifesting as acute mitral valve failure and hemodynamic collapse.

Case

A 52-year-old man with recently diagnosed mitral regurgitation on transthoracic echocardiography (TTE) presented with progressive dyspnea, hypoxemia, and systemic inflammatory response. He was initially treated for presumed pneumonia based on bilateral pulmonary infiltrates. Despite antibiotics and noninvasive positive pressure ventilation, his hypoxia worsened, and he was transferred to the ICU for hemodynamic instability.

Repeat TTE revealed severe mitral regurgitation, a 1.3 cm mobile echodensity on the anterior mitral leaflet, likely leaflet perforation, and P2 prolapse. Blood cultures remained negative. Right and left heart catheterization confirmed cardiogenic shock with elevated biventricular filling pressures and low cardiac index. Findings were consistent with culture-negative IE, and he was planned for urgent mitral valve replacement.

Discussion

Blood culture-negative endocarditis (BCNE) comprises up to 30% of IE cases, often due to prior antibiotics or fastidious organisms. This case highlights the importance of maintaining suspicion for BCNE in patients with underlying valve disease and systemic illness unresponsive to sepsis therapy. Cardiogenic shock may be misdiagnosed as septic shock but often presents with elevated filling pressures and low cardiac output, unlike the vasodilation and high output seen in sepsis. In this patient, lack of improvement with antibiotics, known mitral regurgitation, and new echocardiographic findings of vegetation and leaflet perforation were key diagnostic clues. For hospital-based clinicians, early cardiac imaging and invasive monitoring can distinguish cardiac from infectious causes of shock and expedite life-saving surgical referral.

Conclusion

Presumed culture-negative endocarditis should be considered in unexplained shock with preexisting valvular disease. Early echocardiography and hemodynamic assessment are vital to guide life-saving intervention.

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Young, Breathless, and Failing: A Case of Multifactorial Cardiomyopathy in HIV

Introduction:

Cardiomyopathy in people living with HIV can result from chronic viral inflammation, medication-related toxicity, and lifestyle contributors such as stimulant use. Methamphetamine-associated cardiomyopathy, though increasingly recognized, is less often reported in this population. This case highlights how multiple risk factors combined to produce severe biventricular heart failure in a young man.

Case Description:

A 35-year-old man with HIV, non-adherent to antiretroviral therapy, presented with three weeks of worsening shortness of breath and fatigue. On arrival, he was hypotensive, tachycardic, and hypoxic. Laboratory evaluation revealed acute kidney injury, leukocytosis, and elevated liver enzymes.

Initial chest radiograph showed bilateral pleural effusions and a right middle lobe infiltrate. He was started on treatment for pneumonia, but remained hypoxic. After clinical deterioration, repeat imaging revealed vascular congestion concerning for heart failure, and he was empirically treated with intravenous diuretics.

Transthoracic echocardiography revealed left ventricular ejection fraction 10%, severe right ventricular dysfunction, and biatrial enlargement. Cardiac catheterization confirmed normal coronaries and significant pulmonary hypertension.

The patient later disclosed frequent methamphetamine use, raising suspicion for stimulant-induced cardiomyopathy. Transaminitis improved with diuresis, suggesting congestive hepatopathy. A genetic predisposition was considered but unable to be confirmed because he was adopted. He was started on guideline-directed medical therapy

and statin. His symptoms improved, and he was discharged with cardiology follow-up and substance use counseling.

Discussion:

This case illustrates the interplay of HIV-related myocardial injury, stimulant use, and potential genetic susceptibility in biventricular dysfunction. While distinguishing HIV-associated from methamphetamine-induced cardiomyopathy can be difficult, some patterns may help. HIV cardiomyopathy tends to progress gradually and is linked to chronic immune activation and viral injury. Stimulant-induced cardiomyopathy often presents more acutely and may partially improve with cessation.

Echocardiography alone may not distinguish the two, but cardiac MRI can sometimes reveal fibrosis or inflammation suggestive of viral myocarditis. Although not routine, biomarkers or biopsy may be useful in unclear cases.

Genetic predisposition, such as titin or lamin mutations, may heighten vulnerability to cardiac injury. A family history of early-onset heart failure or sudden death supports this possibility. For hospitalists, awareness of these overlapping etiologies and the importance of identifying substance use is essential, as early intervention can improve outcomes.

Conclusion:

In young patients with HIV, cardiomyopathy warrants a thorough assessment of substance use, genetic risk, and other contributing factors. Early diagnosis, appropriate therapy, and addressing modifiable risks are key to improving function and preventing future decompensation.

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FROM LIMEY TO PRICEY : THE MILLION DOLLAR VITAMIN C DEFICIENCY

Vitamin C is a water-soluble vitamin and one of the essential nutrients. Individuals with poor dietary habits or malabsorption can experience vitamin C deficiency, also known as scurvy, especially in developed countries, which clinicians often overlook. A 47-year-old male with a history of hypertension and prediabetes presented to the emergency department with fatigue, dyspnea, and generalized weakness. His primary care physician referred him due to a hemoglobin level of 5.2 g/dl. He reported spontaneous bruising and tenderness over the left posterior thigh but denied trauma, gastrointestinal bleeding, or anticoagulant use. He lived alone, and his diet consisted primarily of carbohydrates and meat, while he avoided fruits and vegetables. He recently received intravenous ertapenem for lower extremity cellulitis. He appeared pale with tachycardia on general examination. Physical examination revealed poor dentition, left lower extremity ecchymosis, and dark purpuric lesions on bilateral lower extremities and abdomen. Laboratory evaluation revealed microcytic anemia (Mean Corpuscular Volume 76.1 fL), elevated indirect bilirubin (2.7 mg/dL), and a reticulocyte count of 4.4%. Additional testing revealed a negative fecal occult blood test, unremarkable LDH, haptoglobin, normal ferritin, lead level <1 µg/dl, and elevated erythropoietin >20 mU/ml. The computed tomography angiogram showed a non-expanding hematoma in the left posterior thigh without active extravasation. Bone marrow biopsy revealed trilinear hematopoiesis with mild erythroid dysplasia. A dermatology consultation identified the skin lesions as perifollicular purpura, a pathognomonic sign of scurvy. The vitamin C level returned at <0.1 mg/dL (normal range: 0.4-2.0 mg/dL), confirming a severe deficiency. He received six units of Packed Red Blood Cells and began taking oral Vitamin C 500 mg twice daily. His hemoglobin level stabilized, his energy improved, and the ecchymoses started to resolve within 72 hours. He was discharged with instructions to continue taking vitamin C, folate, and iron supplements and advised to incorporate a balanced diet that includes fruits and vegetables. Scurvy, once considered a disease of the past, persists in developed countries among individuals with poor nutritional intake, substance use, psychiatric illness, or restrictive diets. Vitamin C deficiency impairs collagen synthesis, leading to fragile blood vessels that are susceptible to rupture. This can manifest as mucocutaneous bleeding, impaired wound healing, fatigue, and anemia. In this patient, the spontaneous thigh hematoma was likely due to vascular fragility from inadequate collagen. His clotting cascade was functional, allowing for hemostasis during bed rest; however, activity disrupted the clots until adequate collagen restoration occurred. This case highlights the importance of considering scurvy in patients with unexplained bleeding, particularly those with poor dietary habits. Early diagnosis and management with vitamin C can lead to rapid clinical improvement and also prevent further complications.

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A Rare case of Acute HIV Infection presenting as Collapsing Glomerulopathy Leading to End-Stage Renal Disease in a Healthy Young Male

Introduction:

HIV-associated nephropathy (HIVAN) is a severe complication of HIV infection that may lead to rapidly progressive renal failure. Although the incidence has declined with early detection and treatment of HIV disease, it remains an important consideration in acute kidney injury, particularly in young individuals without a prior history of renal disease.

Case Presentation:â€¨ A 26-year-old previously healthy male with no pertinent medical history presented with 10 days of persistent nausea, vomiting, and dehydration. Laboratory data revealed a serum creatinine of 10 mg/dL, BUN of 64 mg/dL, and urine significant for proteinuria (>300 mg/24hr), waxy casts, and granular casts. HIV test was positive with an HIV-1 viral load of nearly 2 million copies/mL, consistent with acute infection (he had a prior negative HIV test in August 2024). His CD4 count was 219, 12 %. Despite rapid initiation of antiretroviral therapy (ART) and supportive care, renal function failed to improve, and he required hemodialysis. A renal biopsy revealed collapsing glomerulopathy, confirming HIVAN. He was discharged on outpatient dialysis and scheduled a follow up with Infectious Diseases. At one-month, patient was still hemodialysis dependent though his HIV disease was well controlled. He has been asked to explore the possibility of renal transplantation.

Discussion:â€¨

HIVAN is a glomerular disease caused by direct infection of renal epithelial cells by HIV-1 virus. Although classically associated with advanced HIV, it can be present during acute infection. It leads to focal segmental glomerulosclerosis, often without symptoms of overt nephrotic syndrome, such as hypertension and peripheral edema. Despite early ART initiation, HIVAN may progress to end stage renal disease (ESRD). Differentiating HIVAN from other forms of nephropathy, including drug-induced nephrotoxicity, is critical and often requires biopsy for diagnosis. This case highlights the importance of considering HIVAN in the differential diagnosis of unexplained acute kidney injury in young patients.

Conclusion:â€¨

Acute HIV infection can rarely present with HIVAN and rapidly progress to ESRD despite early detection and treatment. Prompt recognition and initiation of ART are essential but may not prevent irreversible renal damage. Early nephrology and infectious disease involvement, as well as consideration for biopsy, are crucial for diagnosis and effective management.

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"Not Just Smoke and Mirrorsâ€, Vaping-Associated Pulmonary Injury in a Young Male: A Case Report

Background: Vaping-associated lung Injury (EVALI) is a severe lung disease that is associated with the use of e-cigarettes or vaping products. The scarcity of these cases can make EVALI a challenging diagnosis.

Case Description: A 32-year-old male presented to the emergency department with shortness of breath accompanied by a productive cough with yellow sputum and bilateral lower extremity swelling. Vital signs showed a temperature of 37 °C, a blood pressure of 115/58 mmHg, a heart rate of 101 beats per minute, a respiratory rate of 18 breaths per minute, and an oxygen saturation of 88 percent on room air. The patient was found to be in acute hypoxic respiratory failure requiring supplemental oxygen with a three-liter nasal cannula. The patient denied any toxic habits apart from everyday vape use. A physical examination revealed decreased breath sounds at the lung bases, with minimal rales, and bilateral lower extremity swelling. Chest x-ray reported a patchy nodular infiltrate throughout both lungs. Computed tomography (CT) angiography of the chest ruled out pulmonary embolism and reported ground glass opacities bilaterally and small bilateral pleural effusions. Laboratory investigations were relevant for creatinine 1.6 milligrams per deciliter (mg/dL), demonstrating acute kidney injury, and D-dimer of 296 nanograms per deciliter (ng/dL). The patient was initially admitted due to acute hypoxic respiratory failure secondary to suspected pneumonia versus volume overload due to new-onset congestive heart failure exacerbation and acute kidney injury. The patient was started on intravenous furosemide 40 milligrams. Cardiology service consulted, a transthoracic echocardiogram calculated an ejection fraction of 60 percent, and ruled out suspicion of congestive heart failure. The infectious disease service was consulted due to suspected multifocal pneumonia, and the patient was started on intravenous vancomycin, piperacillin-tazobactam, and azithromycin. The pulmonary service was consulted due to CT chest findings. differentials included viral infection not identified on initial admission, viral testing, acute interstitial pneumonia, diffuse alveolar hemorrhage, vasculitis, and associated lung injury. Fiberoptic bronchoscopy with bronchoalveolar lavage was performed, and lavage showed significant eosinophilia (16 percent), with negative cultures and negative cytology. Findings were most consistent with vaping-associated pulmonary injury. The autoimmune panel also came back negative. The patient was treated with intravenous methylprednisolone, leading to marked clinical improvement. The chest x-ray was repeated, which showed complete resolution of pulmonary infiltrates.

Discussion: This case underscores the importance of considering EVALI in the differential diagnosis of acute hypoxic respiratory failure, particularly in young adults with a history of vaping and non-resolving pneumonia-like symptoms. The patient's rapid clinical and radiological improvement with corticosteroids, along with eosinophilia on bronchoalveolar lavage, confirmed the diagnosis of EVALI. This highlights the need for heightened clinical awareness, early bronchoscopy when appropriate, and thorough history taking to avoid misdiagnosis and ensure timely, effective treatment.

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PEMBROLIZUMAB-INDUCED MYASTHENIA GRAVIS WITH CARDIAC COMPLICATIONS

Immune checkpoint inhibitors such as pembrolizumab, a monoclonal antibody targeting the programmed death-1 (PD-1) receptor, have transformed cancer therapy by enhancing T-cell-mediated antitumor responses. However, this immune activation can lead to severe immune-related adverse events (irAEs), including neuromuscular and cardiac complications. We present a case of pembrolizumab-induced myasthenia gravis with concurrent myocarditis and arrhythmias, underscoring the importance of early recognition and multidisciplinary management.

A 63-year-old female with metastatic uterine cancer receiving combination therapy with pembrolizumab, paclitaxel, and carboplatin presented one-week post-infusion with dyspnea, ptosis, diplopia, and palpitations. Initial evaluation revealed leukopenia (WBC 0.41 x10â^{1}/L), elevated troponin I (2312 ng/L), and new-onset atrial fibrillation with ST-T wave abnormalities on ECG, raising concern for acute coronary syndrome. Urgent cardiac catheterization demonstrated normal coronary arteries and preserved left ventricular function.

She was admitted to the intensive care unit for close monitoring. Continuous telemetry revealed multiple episodes of non-sustained ventricular tachycardia. Given the constellation of symptoms and lab findings, intravenous solumedrol was initiated for suspected immune-mediated myocarditis and pembrolizumab-induced myasthenia gravis. However, concern was noted that high-dose steroids could precipitate a myasthenic crisis. During ICU monitoring, the patient developed complete heart block, prompting placement of a semi-permanent pacemaker. She was subsequently transferred to a tertiary care center due to high risk for ventricular tachycardia storm.

Pyridostigmine was started for suspected myasthenia gravis, despite negative acetylcholine receptor antibody testingâ€”a finding not uncommon in immune checkpoint inhibitor-related cases. The patient's cardiac conduction stabilized, and the temporary pacemaker was removed after she maintained sinus rhythm without pacing dependency. She was discharged on a tapering steroid regimen and close outpatient follow-up.

This case illustrates the potentially life-threatening immune-mediated toxicities of pembrolizumab, particularly when involving both cardiac and neuromuscular systems. Negative antibody tests do not exclude the diagnosis in checkpoint inhibitor-related myasthenia gravis. High clinical suspicion, rapid diagnostic workup, and timely transfer to a higher level of care are critical for managing these complex immune related adverse reactions. Increased awareness and vigilance are essential in oncology and critical care settings as the use of immune checkpoint inhibitors continues to expand.

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Probiotic to Pathogen: A Rare Case of Systemic Lactobacillus Infection Mimicking Vasculitis

Introduction

Lactobacillus species are gram-positive facultative anaerobic or microaerophilic rods commonly regarded as benign commensals of the gastrointestinal and genitourinary tracts. Widely used as probiotics, they are rarely implicated in human infections and often dismissed as contaminants when isolated from blood cultures. However, emerging literature highlights their pathogenicity, particularly in immunocompromised hosts or those with disrupted mucosal barriers. Systemic infections caused by Lactobacillus rhamnosus remain rare, with endocarditis and immune complex-mediated complications even less frequently reported. We present a unique case of Lactobacillus rhamnosus bacteremia with endocarditis, mimicking systemic vasculitis, leading to membranoproliferative glomerulonephritis (MPGN), challenging the perception of this organism as an innocuous probiotic.

Case Presentation

A 49-year-old male with a history of cocaine and alcohol use disorder, with recently diagnosed atrial fibrillation and severe mitral regurgitation one month prior, presented with dyspnea on exertion, leg swelling, and rash for 2 days. In the emergency department, he was hypotensive requiring vasopressors, with anemia, elevated creatinine, and hematuria. Physical examination revealed a grade 3/6 holosystolic murmur at the left upper sternal border, red macules on the anterior shins with hyperpigmentation, significant leg edema, and poor dental hygiene. Kidney function worsened, and in the setting of rash, extensive vasculitis workup was done, which was unremarkable. He was admitted to the ICU, started on piperacillin-tazobactam, and comprehensive infectious workup initiated. Due to progressive renal failure, renal biopsy revealed focally active MPGN with 10% crescent formation, suggestive of infection-related immune complex glomerulonephritis. Soon after, multiple sets of blood cultures returned positive for Lactobacillus rhamnosus, and antibiotics were changed to penicillin G. TTE did not show vegetations. Due to suspicion for high-grade Lactobacillus septicemia and glomerulonephritis, transesophageal echocardiogram was pursued, revealing a posterior mitral valve leaflet vegetation. Antibiotics were changed to intravenous ampicillin with a planned six-week course. Renal function gradually improved. Although discharge to a rehabilitation facility was planned due to the need for a peripherally inserted central catheter, the patient left against medical advice and was discharged on a six-week course of oral amoxicillin.

Discussion

Invasive Lactobacillus rhamnosus infection is rare, and its presentation as endocarditis with glomerulonephritis is virtually unprecedented. The patient's symptoms initially mimicked systemic vasculitis, delaying recognition of infection. Following biopsy-confirmed infection-related

glomerulonephritis, positive blood cultures led to a definite diagnosis of infective endocarditis using modified Duke's criteria. Although often dismissed as a contaminant, Lactobacillus was pathogenic, with poor dentition suspected as the source. This case underscores the importance of considering Lactobacillus as a cause for systemic infection. Given progressing renal failure, treatment was challenging, as immunosuppressive therapy, standard for immune-mediated glomerulonephritis, can worsen infection-related cases, requiring interdisciplinary evaluation. Ultimately, heightened awareness of this organism's infective potential can prevent misdiagnosis, guide therapy, and improve outcomes.

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Thrombosis Negative Lemierre's: Challenging the Classic Criteria

Introduction

Lemierre's syndrome is a rare but life-threatening complication of oropharyngeal infections, classically defined by septic thrombophlebitis of the internal jugular vein, most commonly in the setting of Fusobacterium bacteremia. However, emerging evidence suggests not all cases adhere to this traditional triad. Atypical presentations without radiographic thrombophlebitis may be underrecognized, leading to diagnostic delays and suboptimal management. We report a case of Fusobacterium bacteremia and clinical features consistent with Lemierre's syndrome, without demonstrable venous thrombosis, underscoring the need to reconsider diagnostic frameworks and raise awareness of this atypical variant.

Case Presentation

A 21-year-old male with no significant past medical history presented after sore throat and progressive shortness of breath over two weeks. Initially evaluated in the emergency department, chest x-ray suggested multifocal pneumonia, and he was discharged on Augmentin and azithromycin. Due to worsening respiratory status, he returned after 2 days; CT scans revealed multiple lung abscesses with septic pulmonary emboli, bilateral pleural effusions, and numerous liver abscesses. He was started on vancomycin, levofloxacin, and doxycycline. Despite broad-spectrum antibiotics, he had persistent leukocytosis, fevers, and progressive respiratory failure. He underwent pigtail catheter placement for pleural drainage and ultrasound-guided aspiration of hepatic abscesses revealing frank pus. Metronidazole was added. Pleural fluid, hepatic aspirate, and blood cultures initially showed no growth. Further history revealed intermittent neck pain and stiffness, raising suspicion for Lemierre's syndrome. CT neck without contrast showed no septic thrombophlebitis of the internal jugular vein or mesenteric vein thrombosis. Doppler ultrasound of the liver was also negative for portal vein thrombosis. On day 10, anaerobic culture from hepatic aspirate grew Fusobacterium necrophorum. Karius blood test also revealed Fusobacterium. He improved significantly over a 17-day admission and was discharged on Augmentin with close infectious disease follow-up, achieving resolution of infection over 3 months.

Conclusion

Lemierre's syndrome was a common, often fatal, illness in young adults during the pre-antibiotic era, but widespread antibiotic use led to a dramatic decline. While internal jugular vein thrombophlebitis is considered a hallmark of Lemierre's syndrome, this case adds to growing evidence that Fusobacterium septicemia with systemic complications can occur in its absence, especially in the setting of benign neck pain/stiffness. Similar atypical presentations have been described in the literature, including cases of necrotizing pneumonia and multisite abscesses without radiographic thrombosis. Resurgence of this "forgottenâ€ disease is increasing with restrictive antibiotic use. Using internal jugular vein

thrombophlebitis as an essential criterion may be overly restrictive, excluding a larger proportion of otherwise typical cases. Emerging literature proposes a broader clinical framework emphasizing anaerobic bacteremia and metastatic infection, irrespective of thrombosis. Early recognition of this variant is essential, as it facilitates prompt initiation of anaerobic antibiotic coverage, the cornerstone of treatment that can significantly improve outcomes.

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Thyrotoxic Periodic Paralysis: A Rare but Reversible Endocrine Emergency

Introduction:

Thyrotoxic periodic paralysis (TPP) is a rare but potentially life-threatening complication of hyperthyroidism. TPP is characterized by acute episodes of primarily proximal lower extremity muscle weakness due to hypokalemia.While hyperthyroidism is about 10 fold more common in females, TPP is about 20 times more prevalent in men. Although most typically seen in Asian ethnicities, it can occur across other ethnicities .

Case Report:

A 36-year-old Hispanic male who worked as a truck presented with sudden, painless muscle weakness in the upper and lower extremities Most notably his symptoms were characterized by an inability to ambulate after waking from a nap. He reported a similar transient episode a few weeks prior which had resolved spontaneously within hours. On examination, lower extremity strength was 2/5. Laboratory studies revealed severe hypokalemia (Kâº 1.8 mmol/L) and hypophosphatemia (1.4 mg/dL). He denied any history of diarrhea and vomiting, making gastrointestinal loss of potassium less likely. Thyroid studies showed markedly suppressed TSH (<0.03 µIU/mL), elevated free T4 (6.36 ng/dL), and free T3 (17.4 pg/mL), Positive thyroid-stimulating immunoglobulin and anti-TPO antibodies consistent with primary hyperthyroidism due to Graves disease. Prompt potassium repletion and treatment of hyperthyroidism led to resolution of muscle weakness (5/5 strength). He could not achieve adequate control of hyperthyroidism with maximum doses of Methimazole so decision was eventually made to undergo total thyroidectomy as definitive treatment.

Discussion:

TPP is a sporadic form of periodic paralysis that occurs in association with hyperthyroidism. It is distinguished from other forms of neuromuscular disease by the presence of sudden attacks of weakness provoked by some trigger, associated with hypokalemia and recovery with potassium supplementation. Triggers identified include intense physical exertion, fasting, emotional stress, high carbohydrate meals, cold exposure, and activities that drive potassium into the cells.

Hyperthyroidism heightens tissue sensitivity,especially in skeletal muscles to beta-adrenergic stimulation, increasing Naâº/Kâº ATPase activity, which drives potassium into cells, leading to muscle membrane hyperpolarization and paralysis. Testosterone has been shown to increase Na+K+ ATPase activity in animals, and conditions that cause hyperinsulinemia which could explain why this condition is seen in males predomnantly.

Treatment involves potassium supplementation, but refractory cases can be treated with IV propranolol, as it reverses the excess stimulation of Na+K+ ATPase. Prevention of relapse entails definitive treatment

to restore an euthyroid state. In Graves disease, radioactive iodine ablation and surgical treatment has been shown to be more effective than use of antithyroid drugs. Triggers in our patient included, untreated Graves disease, occupation involving intense physical exertion and dehydration. He was only able to achieve euthyroidism and resolution of TPP symptoms following total thyroidectomy but has since been symptom free. This highlights the role of total thyroidectomy in achieving euthyroidism and preventing relapse in patients with TPP.

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Acute-Onset Pembrolizumab-Induced Myositis and Myocarditis in a Patient With Advanced Metastatic Vulva Cancer

Introduction

Patients treated with pembrolizumab, a PD-1 immune checkpoint inhibitor (ICI), can develop a spectrum of immune-related adverse events affecting multiple organ systems. Among these, myositis and myocarditis are rare but potentially fatal complications that require prompt identification and intervention. These toxicities typically occur within the first three months of therapy and may present concurrently, significantly increasing mortality risk. We describe the case of a 62-year-old female with metastatic vulvar squamous cell carcinoma who developed pembrolizumab-induced myositis and myocarditis.

Case Description

A 62-year-old female with metastatic vulvar cancer status post four cycles of paclitaxel, carboplatin, bevacizumab, and pembrolizumab presented to the emergency department with complaints of fever, generalized weakness, and muscle aches less than a day after resuming the fifth cycle of chemotherapy with paclitaxel, carboplatin, and pembrolizumab alone. She was febrile (101°F), hypoxic, and hypotensive, requiring resuscitation and brief pressor support. Initial workup was significant for severe hypokalemia (2.9 mEq/L), elevated creatinine kinase (1081 U/L), elevated troponins (761 ng/L), acute kidney injury (creatinine 4.5 mg/dL from a normal baseline), and lactic acidosis (4.5 mEq/L). Electrocardiogram was unremarkable. CT chest showed consolidation in the left lower lobe. She was admitted for acute hypoxic respiratory failure and severe sepsis secondary to community-acquired pneumonia and started on intravenous (IV) meropenem, vancomycin, and doxycycline. IV fluids were continued, and electrolyte abnormalities were corrected. On day two, decreased urinary output and tenderness of the bilateral upper extremities were noted, with no visual changes. Creatinine, blood urea nitrogen, creatinine kinase, and troponins were all up-trending. There were concerns for ICI-induced myositis and myocarditis resulting in acute renal failure. She was started on high-dose intravenous methylprednisolone 500 mg twice a day for three days. After the first day of steroid therapy, creatinine kinase decreased by over 20% and troponin by almost 75%. Symptoms subsequently improved on completion of therapy, and CK and troponin returned to baseline. She was eventually discharged after completing the course of antibiotics and steroids and was followed up in the outpatient clinic.

Discussion

Pembrolizumab is widely used in the management of multiple malignancies and is associated with a range of side effects. Rare occurrences of overlapping myositis, myocarditis, and myasthenia gravis have

been reported, although they do not always occur together. These conditions carry a high fatality rate, particularly when cardiac involvement is present. Early initiation of high-dose steroids has been shown to improve outcomes in such cases. This underscores the importance of prompt recognition and intervention for pembrolizumab-induced myositis and myocarditis. Thorough clinical evaluation and appropriate workup are therefore essential to help reduce mortality associated with these serious immune-related adverse events. Increased awareness among clinicians and a multidisciplinary approach involving oncology and cardiology teams can be critical to optimizing patient outcomes.

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Persistent Sex-Based Disparities in ST-Elevation Myocardial Infarction Complicated by Cardiogenic Shock: A Nationwide Analysis (2008-2022)

Purpose: To examine temporal trends in sex-based disparities in mortality, mechanical circulatory support utilization, and mechanical ventilation among admissions with ST-Elevation Myocardial Infarction (STEMI) complicated by cardiogenic shock (CS) in the United States from 2008 to 2022.

Methods: We conducted a retrospective examination of the National Inpatient Sample from 2008 to 2022, identifying admissions with a primary diagnosis of STEMI and a secondary diagnosis of CS. After excluding elective admissions and transfers, we examined temporal trends in in-hospital mortality, mechanical circulatory support (MCS) utilization, and mechanical ventilation. Mechanical circulatory support (MCS) encompasses intra-aortic balloon pump, percutaneous ventricularâ€assist devices, and extracorporeal membrane oxygenation. Survey-weighted multivariable logistic regression models were adjusted for comorbidities, patient, and hospital-level confounders. Sex-by-year interactions were tested to assess temporal changes in gender disparities. Adjusted odds ratios (aOR) and predictive margins with 95% confidence intervals (CI) are reported.

Results: Among 2,422,498 STEMI hospitalizations, 292,230 (12.1%) developed CS. The study population was 65.2% male and 75% White, with a mean age of 67 years. Risk-adjusted in-hospital mortality declined from 34.3 % to 28.8 % between 2008 and 2022 (P-trend < 0.001). Female sex remained independently associated with higher mortality (aOR 1.06, 95 % CI 1.02-1.10; P < 0.01), and this gap did not change over time (interaction P = 0.63). MCS utilization fell from 54.7% to 45.0% (P-trend < 0.001); female admissions were consistently less likely to receive MCS (aOR 0.71, 95 % CI 0.68-0.73; P < 0.01) compared to males, with no temporal change (interaction P = 0.74). Invasive mechanical ventilation decreased from 41.4% to 30.8% (P-trend < 0.001). Although female admissions were less likely to receive invasive mechanical ventilation overall (aOR 0.86, 95 % CI 0.83-0.89; P < 0.01), this disparity narrowed by 0.4 percentage points per year (interaction P = 0.02).

Conclusion: Despite overall improvements in in-hospital mortality from 34.3% to 28.8% (2008-2022) for STEMI-CS, female admissions were associated with 6% higher odds of mortality, 29% lower odds of receiving MCS, and 14% lower odds of receiving mechanical ventilation. The persistent mortality gap and mechanical support underutilization in female STEMI-CS patients underscore the need for further research to unravel causative factors and whether these treatment disparities mediate the observed mortality difference.

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A rare case of Emphysematous Gastritis treated successfully with conservative management

Introduction:
Emphysematous gastritis (EG) is a rare but life-threatening gastrointestinal emergency characterized by gas within the gastric wall caused by invasive infection with gas-forming organisms. Since its first description in 1889 by Fraenkel, fewer than 100 cases have been documented in English literature. EG is associated with a high mortality rateâ€"up to 62%â€"and typically occurs in patients with risk factors such as immunosuppression, diabetes mellitus, chronic kidney disease, or disruption of the gastric mucosa. Common causative organisms include Streptococcus species, Escherichia coli, Clostridium species, Pseudomonas aeruginosa, and occasionally fungi such as Candida and Mucor species. Timely diagnosis and appropriate conservative management with antibiotics are crucial to improve outcomes, but no standardized treatment guidelines currently exist.

Case Presentation:
A 56-year-old man with a history of heart failure with reduced ejection fraction (HFrEF), atrial fibrillation, hypertension, and well-controlled type 2 diabetes presented with a two-week history of vomiting, diarrhea, and persistent hiccups. Initially evaluated at an outside facility, he was discharged with supportive care. However, symptoms progressed, and on re-presentation, he was hypotensive and tachycardic, with diffuse abdominal tenderness. Laboratory studies showed acute kidney injury, mild lactic acidosis, and pyuria with bacteriuria. A CT scan of the abdomen revealed intramural gas in the gastric wall and gas foci in the gastro-hepatic ligament, highly suggestive of EG. He was admitted to the surgical ICU and treated with intravenous piperacillin-tazobactam and fluconazole. Cultures grew Klebsiella pneumoniae in urine and Micrococcus luteus in blood. Given the presence of an implantable cardioverter-defibrillator (ICD) and concern for deep-seated infection, antibiotics were escalated to meropenem. The patient responded well to therapy, with resolution of symptoms and normalization of laboratory values, and was discharged with a 14-day course of IV Meropenem.

Discussion:
This case illustrates the diagnostic complexity and therapeutic challenges of EG. Differentiating EG from gastric emphysema (GE), a non-infectious and typically benign entity, is critical due to the stark difference in prognosis. While both conditions may present similarly with gastrointestinal symptoms and radiographic gastric wall gas, EG often involves systemic toxicity and gastric wall thickening on imaging. Conservative management has become the preferred treatment approach, with surgery reserved for complications such as perforation or necrosis. Our case supports this trend, demonstrating clinical improvement with early aggressive antimicrobial therapy alone. The addition of antifungal treatment remains controversial, particularly in the absence of fungal pathogens in culture. This report highlights the importance of high clinical suspicion, prompt imaging, and empiric broad-spectrum antibiotics in managing EG, and underscores the need for further research to establish evidence-based treatment protocols.

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Borrelia Miyamotoi Infection Presenting As Septic Shock During Late Pregnancy

INTRODUCTION:
Borrelia miyamotoi is an emerging tick-borne relapsing fever spirochete transmitted by Ixodes ticks. Since its recognition as a human pathogen in 2011, it has been associated with a spectrum of clinical presentations ranging from febrile illness to neurologic complications. Infections during pregnancy are rare and present diagnostic and therapeutic challenges.

CASE PRESENTATION:
A 42-year-old primigravida at 33 weeks and 6 days uncomplicated gestation, with a history of hypothyroidism, was transferred to our hospital for evaluation and management of septic shock and suspected tick-borne illness. She resided on a wooded property in the northeastern United States and had frequent outdoor exposure. She had recently removed a non-engorged tick from her back. She declined prophylaxis but retained the tick for later testing.

Approximately two weeks after the tick removal, she developed high-grade fevers (up to 102°F), chills, myalgias, nausea, and intermittent headaches without photophobia. Two days after the symptom onset, she presented with fevers (101.3 °F), hypotension (92/65 mmHg) and tachycardia. Physical exam revealed no rash or meningeal signs. Labs showed leukopenia, thrombocytopenia, mild anemia, transaminitis, indirect hyperbilirubinemia, and elevated D-dimer. Fetal monitoring remained reassuring.

She was transferred to the ICU for persistent hypotension requiring vasopressors. She was started on broad-spectrum antibiotics with ceftriaxone, vancomycin, acyclovir and doxycycline for empiric meningitis/meningoencephalitis coverage. Extensive workup including imaging and echocardiography was unremarkable. She responded well within 24 hours, with stabilization of vitals and improvement in lab parameters.

On hospital day 3, a multiplex PCR panel returned positive for Borrelia miyamotoi. Lyme serologies were negative. Based on this, ceftriaxone was continued alone to complete a 10-day IV course. She remained afebrile and hemodynamically stable. At discharge, she was transitioned to oral amoxicillin to complete a 21-day course. Outpatient follow-up confirmed continued recovery, normalization of blood counts, and an uncomplicated delivery of a healthy infant.

DISCUSSION:

This case emphasizes the importance of maintaining a high index of suspicion for B. miyamotoi infection in patients presenting with febrile illness and tick exposure, even during pregnancy. Diagnosis is best confirmed via PCR, as serologic tests may be falsely negative early in infection. Though doxycycline is typically avoided in pregnancy, it may be used short-term in life-threatening infections. Early empiric therapy and accurate molecular diagnostics were essential in achieving a successful maternal and fetal outcome in this rare presentation.

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A Reminder of the Dangers of Vaping: Increasing Awareness Through a Case of E-cigarette or vaping-associated lung injury (EVALI) Lipoid Pneumonia in a 45-Year-Old Man Without Significant Past Medical History

Background:

Vaping has emerged as a common alternative to nicotine replacement in smoking cessation therapy. While the use of vaping products gained significant attention from the FDA and CDC in 2020â€”resulting in a ban on the toxic additive vitamin E acetateâ€”the incidence of EVALI persists. We present a case involving a former tobacco smoker who relied on vaping to manage nicotine cravings that later developed lipoid pneumonia as a consequence. This case underscores the dangerous side-effects of vaping, and the importance of continued research and surveillance to understand the extent.

Case Presentation

A 45-year-old man presented with six weeks of progressively worsening dyspnea and productive cough with blood-tinged sputum, reduced exercise tolerance, and orthopnea. His past medical history was significant for former tobacco use, chronic pain, symptomatic premature ventricular contractions, and provoked deep vein thrombosis. He recently quit smoking and was vaping (1 unit of "Geek Barsâ€ per day) as a substitute. He also had occupational exposure to sprayed insulation. To his knowledge, his co-workers did not experience similar respiratory symptoms. Of note, he presented to the ED 4 weeks prior for milder symptoms and was found to be influenza positive. Chest CT with contrast showed a left upper lobe ground glass opacity. He subsequently followed up with outpatient pulmonary. Pulmonary function testing at that time was unremarkable.

Vital signs on presentation were temperature 38.2 C, blood pressure 125/82 mmHg, heart rate 100 beats per minute, respiratory rate 17 breaths per minute, and oxygen saturation 85% on room air. Physical exam was remarkable for bibasilar mild inspiratory crackles. Laboratory findings were remarkable for white blood cell count 13.27 K/uL, C-reactive protein 14.6 mg/dL, erythrocyte sedimentation rate 23 mm/hr, and procalcitonin 2.23 ng/mL. Respiratory viral panel was negative. Chest CT with contrast revealed diffuse, scattered centrilobular groundglass nodules and patchy opacities, more prominent in the right lung; negative for pulmonary embolism. The patient was admitted for acute hypoxemic respiratory failure and was started on empiric IV antibiotics.

On hospital day two, patient underwent a bronchoscopy with bronchoalveolar lavage and transbronchial biopsy. Microbiology was negative. Cytology and tissue pathology showed airway-centered foamy histiocytes and intra-alveolar lipid-laden macrophages, confirmed with Oil Red O staining. These findings were suggestive of lipoid pneumonia.

Discussion

The etiology of the lipoid pneumonia was likely exogenous due to inhalation of aerosolized components in the patient's vape. The primary components in Geek Bar Pulse X e-liquids include propylene glycol and vegetable glycerin. Vegetable glycerin-based aerosols have been shown to induce airway inflammation and ion channel dysfunction, leading to mucus hyperconcentration. This case highlights that despite the FDA ban on vitamin E acetate in vaping products, other ingredients in e-liquids may still pose significant harm.

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Gastric Cancer Masquerading as Pulmonary Disease in a Young Adult

Pulmonary lymphangitic carcinomatosis (PLC) is an aggressive form of pulmonary lymphatic metastasis that is extremely rare and is most associated with adenocarcinomas of various origins. However, its rarity, resemblance to interstitial lung disease on imaging, and non-specific symptoms can make early diagnosis and intervention challenging.

A 35-year-old male with a fifteen pack year smoking history presented with a dry cough, shortness of breath, and unintentional twenty-pound weight loss. Labwork was unremarkable. Respiratory Viral Panel tested positive for Parainfluenza. CT Chest revealed diffuse interlobular septal thickening, enlarged lymph nodes and innumerable bilateral micronodular opacities. The case was discussed at a multidisciplinary boards and was deemed likely parainfluenza induced pneumonitis. The patient was treated with an appropriate course of steroids and inhalers but he continued to be symptomatic. Repeat CT chest two weeks later only showed mild improvement. Thus, the patient underwent a bronchoscopy with biopsy which showed poorly differentiated non-small cell carcinoma, likely of glandular differentiation with concurrent angiolymphatic invasion. CT Abdomen and Pelvis showed a masslike circumferential thickening of the gastric antrum with extensive surrounding lymphadenopathy. EGD confirmed a malignant mass in the gastric antrum. Pathology revealed poorly differentiated adenocarcinoma with signet ring cell features and suspected lymphovascular invasion. After extensive pathological review and consideration of the clinical presentation, it was determined that this likely represented a gastric adenocarcinoma with metastasis to the lung. A week into the patients hospital course, he developed worsening hypoxemic respiratory failure requiring a non-rebreather. This was despite the recent completion of antibiotics for pneumonia treatment, diuresis, and resolution of previously seen iatrogenic pneumothorax. An arterial blood gas (ABG) showed pH 7.47, pCO2 37 mmHg, pO2 182 mmHg and a bicarbonate level of 27 consistent with acute respiratory alkalosis. Oncology was consulted and initiated treatment with platinum-based chemotherapy, which demonstrated clinical improvement less than one week later. He no longer required respiratory support and had resolution of respiratory symptoms.

This case supports the existing but scarce literature that guideline directed treatment of the underlying malignancy can improve short-term outcomes in patients with PLC. After initiation of chemotherapy to target the underlying gastric adenocarcinoma, the patient's hypoxemic respiratory failure and pulmonary symptoms resolved, allowing the patient to be discharged from the hospital. Therefore, it is important that clinicians be aware of PLC and consider this differential diagnosis in patients with progressive dyspnea and non-productive cough with nonspecific radiographic findings that are refractory to the standard pulmonary treatments. Early diagnosis and intervention are vital to optimizing the patient's limited life expectancy and slowing disease progression.

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Poster Presentations

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"The Non-Smoker Lung Cancer Paradox: Higher Mortality Despite Elevated EGFR Mutations and Fewer Comorbiditiesâ€

Background:

Non-smoking individuals with lung cancer represent a biologically and clinically distinct population. Approximately 10-25% of all lung cancer cases worldwide occur in individuals who have never smoked. Although this subgroup often presents with favorable features such as fewer comorbidities and higher prevalence of targetable mutations such as EGFR, their clinical outcomes remain poorly defined.

Objectives:

To compare clinical characteristics, molecular features, stage at diagnosis, and outcomes between smokers and non-smokers with lung cancer.

Methods:

We conducted a retrospective cohort analysis on 129 patients with lung cancer treated at our safety-net institution in a population mostly Hispanic and Black in the South Bronx. Participants were stratified by smoking status. Variables including age, sex, race, comorbidities with a focus on COPD, stage at diagnosis of the disease, type of cancer, treatment modality, and mortality were compared using chi-square and t-test, with significance defined as $p < 0.05$.

Results:

Our population was mostly Hispanic (56%) and Black (36%). Non-smokers were more likely to be female (68% vs. 39%, $p = 0.017$) and had extremely lower rates of COPD (11% vs. 62%, $p = 0.001$). EGFR mutations were markedly more prevalent among non-smokers (42% vs. 28%, $p < 0.001$), and adenocarcinomas were more common (88% vs. 45%, $p < 0.025$). Although not statistically significant, more non-smokers presented with stage IV disease (39% vs. 28%, $p < 0.12$). Despite fewer comorbidities and favorable mutations, non-smokers experienced a significantly higher mortality rate than smokers (28% vs. 9%, $p = 0.031$). Both groups received similar treatment patterns and follow-up.

Conclusions:

This study highlights a clinical paradox: despite tending to have lower comorbidities such as COPD and a higher frequency of targetable mutations such as EGFR, non-smokers with lung cancer experience worse

survival outcomes compared to smokers with lung cancer. These findings may be partly explained by later-stage diagnosis, possibly driven by the absence of screening in this population. Current lung cancer screening guidelines are based solely on smoking history, yet other risk factors such as genetic mutations exist and warrant further investigation to support the expansion of screening criteria.

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Bridging the Gap: Statin Therapy for Primary Cardiovascular Prevention in HIV Care

Introduction

Cardiovascular disease (CVD) related deaths have doubled in people living with HIV (PLWH) as antiretroviral therapy has extended their life expectancy. Traditional atherosclerotic cardiovascular disease (ASCVD) risk calculators may underestimate CVD risk in PLWH. The REPRIEVE trial recently confirmed that statin use lowers the incidence of major adverse cardiovascular events like heart attack and stroke in PLWH. This led to the recently updated US Department of Health and Human Services (HHS) HIV guidelines recommending statin therapy for the primary prevention of ASCVD in PLWH. The first phase of this study aims to assess the proportion of PLWH who were appropriately receiving statin for primary ASCVD prevention and identify reasons for statin underuse.

Method

A retrospective chart review included HIV-positive patients seen at the Life Forward Clinic between January 1, 2022, and June 30, 2024. Eligible patients aged 40 to 75 years, on active antiretroviral therapy, had at least two follow-up visits during the study period, an available lipid panel, and no history of atherosclerotic cardiovascular disease (ASCVD). Patients with established ASCVD and pregnancy were excluded. Demographic information, HIV-related factors, cardiovascular risk factors, and statin use were collected. The 10-year ASCVD risk score was calculated using the ASCVD Risk Estimator. Statin recommendations were based on current guidelines with moderate-intensity statins for borderline

(â‰¥5% to <7.5%) and intermediate risk (â‰¥7.5% to <20%), and high-intensity statins for high risk (â‰¥20%) individuals.

Result

A total of 124 patients aged 40-75 years were included in this study, comprising of 57% males. The ethnic breakdown included 38% African Americans, 31% Caucasians, and 17% Asians. Comorbidities included Hypertension (52%), Diabetes (21%), and Smoking (21%). Of the 124 patients, 80 (64%) had an ASCVD risk score â‰¥ 5% for whom statin therapy was indicated. Among these, only 45 patients (56%) were prescribed a statin, and 27% of those did not receive the appropriate intensity of statin. Additionally, 44% of PLWH did not receive statins, primarily due to physician non-prescription (95%) rather than patient refusal. In the high-risk group, 63% received statins, but 53% of these patients were not initiated on an appropriate high intensity statin.

Conclusion

With the rising number of CVD-related deaths in PLWH, primary prevention of ASCVD is crucial. This project highlights the need for enhanced physician education on HIV-specific risks to improve statin use to reduce preventable CVD-related deaths in PLWH. Phase 2 of this study will focus on implementing targeted interventions to enhance statin use among people living with HIV by addressing barriers faced by both patients and providers through education and engagement initiatives. System-level challenges such as insurance coverage and copayment obstacles will need to be identified and addressed to resolve the underlying causes contributing to underutilization.

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Blood smear abnormalities in Heartland and Bourbon virus: emerging viral tick-borne infections in New York.

Introduction: Viral Tick-Borne Diseases (TBDs) are globally present but likely underdiagnosed due to lack of diagnostic tools. Clinical presentations may be overshadowed by more common TBDs such as Lyme disease, ehrlichiosis, anaplasmosis, Borrelia miyamotoi, and babesiosis. Bourbon virus (BRBV) and Heartland virus (HRTV) are two recognized tick-borne pathogens, historically described primarily in the Midwest and southern United States. With few rapid diagnostic techniques available, routine blood work (e.g. blood smears) may help raise clinical suspicion for these viral TBDs, especially outside traditionally endemic areas. This study aims to analyze peripheral blood smears from human cases of HRTV and BRBV identified on Long Island, NY.

Methods: The inclusion criteria were patients aged >18 years old with an acute febrile illness and either a witnessed tick bite within the last four weeks or a confirmed TBD diagnosis during admission at Stony Brook University Hospital (SBUH) from 2019 to 2024. Enrolled subjects were required to follow up at 1, 6, and 12 months. A total of 230 patients were enrolled in the study, and 107 were selected for analysis after excluding those without follow-up. All available samples from enrollment, 1-month and 6-month visits were sent to the New York State Department of Health for plaque reduction neutralization testing against HRTV and BRBV.

Results: Demographics and clinical data were collected for all subjects analyzed. Of the 107 subjects analyzed, 3 subjects tested positive for neutralizing antibodies to BRBV and HRTV: 2 patients for BRBV and 1 patient for HRTV. We reviewed peripheral blood smears from two confirmed patients (one HRTV, one BRBV) with convalescent titers who were hospitalized in the spring-summer of 2023. Both patients presented with thrombocytopenia, lethargy, and arthralgias. Peripheral smears demonstrated plasmacytoid atypical lymphocytes that persisted for several days after the symptom onset. In the HRTV patient, the plasmacytoid lymphocyte persisted for five days before disappearing, suggesting that it was likely associated with the acute viral infection. Additional findings included toxic granulation and hypolobated neutrophils, as well as activated monocytes. No morulae or intracellular organisms were observed, which helped differentiate these viral infections from ehrlichiosis and anaplasmosis.

Conclusion: We found plasmacytoid lymphocytes in the blood smears of both cases of HRTV and BRBV acute infections. While peripheral smear findings can be non-specific, in patients with compatible clinical picture and negative testing for more common tick-borne illnesses, it may prompt clinicians to consider these viral infections in the differential diagnosis of febrile illness. Clinical presentation may resemble ehrlichiosis and anaplasmosis, often presenting with fever, cytopenias, arthralgias, and transaminitis. Finally, clinician awareness, the development of rapid diagnostic tests, and further study of HRTV and BRBV viruses are urgently needed.

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Predictors of Post-Polypectomy Bleeding in Cirrhotic Patients: A Retrospective Analysis

Introduction

Colonoscopy is the gold standard for colorectal cancer screening and surveillance. In cirrhotic patients, coagulopathy and portal hypertension heighten bleeding risk during endoscopic procedures. Colonoscopy is routinely performed during liver transplant evaluation, yet the safety of polypectomy and predictors of post-polypectomy bleeding (PPB) in this population remain poorly defined.

Methods

We conducted a retrospective study of cirrhotic patients who underwent colonoscopy for transplant evaluation at a tertiary center (2020-2022). Demographic, clinical, and endoscopic data were reviewed, including polyp characteristics and adverse outcomes (immediate/delayed PPB, hospitalization, transfusion, perforation, death). Statistical comparisons used t-tests and chi-squared tests. Univariate and multivariate logistic regression identified predictors of PPB, with significance set at P â‰¤ 0.05.

Results

Among 240 screened patients, 45 underwent 53 colonoscopies with 120 polyps removed. Post-polypectomy bleeding (PPB) occurred in 8 cases: 7 immediate (5.8%) and 1 delayed (0.8%). All bleeding was managed endoscopically without major complications. One patient (1.9%) required hospitalization, and 4 (7.5%) underwent repeat colonoscopy. No perforations, transfusions, or deaths occurred. A univariate analysis identified elevated bilirubin (0.7-53.8 mg/dL), INR >1.5, lower sodium level (120-147 mEq/L), esophageal varices, larger polyp size, right-sided location, and hot snare use as risk factors for PPB. On multivariate analysis, polyp size, elevated bilirubin, and hot snare use remained significant predictors. No associations were found with age, sex, albumin, platelet count, creatinine, MELD 3.0, Child-Pugh score, hepatic encephalopathy, hepatocellular carcinoma, or prior GI bleeding. Polyp morphology and histology did not differ between bleeding and non-bleeding cases.

Discussion

Polypectomy appears safe in cirrhotic patients, with a low risk of major complications. Consistent with prior studies, PPB risk was linked to INR, esophageal varices, right- sided lesions, and hot snare use. Notably, elevated bilirubin and lower sodium level was also associated with PPBâ€”potential novel markers requiring further validation. Traditional risk factors such as age, low platelets, or ascites were not significant in this cohort. Larger prospective studies are needed to refine risk stratification and improve procedural safety.

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FAMILIAL HYPERCHOLESTEROLEMIA IS NOT ASSOCIATED WITH INCREASED MORTALITY IN ACUTE HEART FAILURE PATIENTS IN THE US

Introduction: Familial hypercholesterolemia (FH) is a genetic disorder characterized by high levels of low-density lipoprotein cholesterol (LDL-C). FH increases the risk of coronary artery disease, but little is known about its association with heart failure. We aimed whether FH increases the risk of in-hospital mortality and adverse cardiovascular outcomes in acute heart failure patients.

Methods: Data from the US National Inpatient Sample (NIS) from 2016-2020 were analyzed. Patients admitted for acute heart failure were included in the study and were further stratified according to the presence of FH. The primary outcome was 3-point major adverse cardiovascular events (MACE) consisting of cardiovascular death, non-fatal myocardial infarction, or non-fatal stroke. Secondary analysis included the separate components of MACE, cardiogenic shock, acute renal failure, atrial fibrillation, high-grade atrioventricular block, ventricular tachycardia and ventricular fibrillation. Prevalence of outcomes between the two groups were compared using appropriate statistical tests. Multivariate logistic regression was also done to examine the risk of outcomes while controlling for potential confounding factors.

Results: A total of 535,698 patients hospitalized for acute heart failure were included, of whom only 193 (0.003%) had FH. Patients with FH were younger than those without (70[15] vs 73[13], p= 0.007) and had a higher prevalence of coronary artery disease (5[2.6%] vs 4834[0.9%], p= 0.032). FH did not increase the adjusted risk of 3-point MACE (aOR= 1.12 [0.61-2.07], mortality, non-fatal stroke, and non-fatal MI (aOR= 0.79 [0.25-2.49], 2.57 [0.36-18.34], and 1.21 [0.57-2.59], respectively). However, FH increased the risk of cardiogenic shock by almost 2.5-fold (aOR= 2.44 [1.25-4.76]) and acute renal failure by 1.4-fold (aOR= 1.37 [1.01-1.90]). Coronary artery disease on admission was the strongest predictor of all cardiovascular events.

Conclusion: Familial hypercholesterolemia is not associated with increased mortality and morbidity in acute heart failure in the US despite increased prevalence of coronary artery disease in these patients.

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Targeting the Root: BCMA-CD19 CAR-T Therapy for Refractory SLE Overlap Syndrome

This study aimed to evaluate the safety and efficacy of dual-target BCMA-CD19 chimeric antigen receptor (cCAR) T therapy in a patient with refractory systemic lupus erythematosus (SLE) overlap syndrome (OS). This study builds upon findings from a previous cCAR phase 1 trial in SLE. A 53-year-old female with a 10-year history of multisystem, refractory SLE OS was treated by compassionate use with cCAR. Her disease included Sjögren's syndrome, anti-neutrophil cytoplasmic antibody (ANCA)-associated vasculitis, and immune thrombocytopenia. The patient was refractory to high-dose glucocorticoids, hydroxychloroquine, cyclosporine A, and mycophenolate mofetil, and demonstrated high titers of autoantibodies. The SLE Disease Activity Index 2000 (SLEDAI-2K) score at presentation was 8, and renal biopsy demonstrated lupus nephritis class III with inadequate response to more than two therapies. Peripheral blood T-cells were collected (July 28, 2023) and genetically modified to express cCAR with a viral vector. After lymphodepletion with cyclophosphamide (300 mg/m²/day, August 25-27, 2023), cCAR T-cells were infused on August 30, 2023. Cessation of all immunosuppressants occurred before treatment. Treatment was provided under compassionate use in accordance with an IRB-approved protocol (NCT05474885). The trial was registered at ClinicalTrials.gov under NCT05474885, with ethics approval number K2022-294-3.

Following infusion, cCAR expanded in-vivo and became undetectable by day 40. The patient developed grade 1 cytokine release syndrome, grade 3 leukopenia, and grade 2 anemia, which all resolved with 2 weeks of supportive care. No infections, severe adverse events, or immune effector cell-associated neurotoxicity syndrome were observed. B-cells were eliminated by day 3 with gradual recovery by day 45. IL-6 peaked at 40 pg/mL at day 3 and normalized by day 10. Immunoglobulin levels began recovering three months after treatment. All pathogenic autoantibodies (PR3-ANCA, MPO-ANCA, ANA, anti-Sm, anti-U1 snRNP, anti-SSA/Ro52, anti-platelet antibodies) were elevated at baseline and became undetectable post-cCAR treatment. Proteinuria resolved within 1 month. Platelets normalized by day 360. The SLEDAI-2K score decreased to 0, satisfying definitions of remission in SLE (DORIS) criteria and maintained complete remission after over 1.5 years.

cCAR therapy demonstrated remarkable safety and efficacy in achieving sustained complete remission in refractory SLE OS. CD19-targeted therapies eliminate B-cells but spare long-lived plasma cells, which continue producing pathogenic autoantibodies and may lead to relapse. In contrast, cCAR simultaneously targets both CD19 on B-cells and BCMA on long-lived plasma cells, enabling deeper immunologic clearance. This dual-target strategy overcomes a key limitation of single-target approaches and may offer a more durable solution for antibody-driven autoimmune diseases.

Sarah Kosse

Ann Mercurio MD, Sara Luby DO, Lindsey Fox MD, Vasundhara Singh MD

Mount Sinai Morningside and West

TEACHING COST-CONSCIOUS CARE: A NEEDS-BASED CURRICULUM TO IMPROVE INTERN UNDERSTANDING OF INPATIENT VS OUTPATIENT COSTS

Purpose and Goals

A prior needs assessment revealed low confidence and knowledge gaps among internal medicine residents regarding cost-effective care, particularly in distinguishing inpatient from outpatient costs. To address these deficiencies, we developed and implemented a targeted, interactive curriculum for interns aimed at fostering early high value care (HVC) decision-making.

Methods

Three one-hour sessions were delivered during academic half-days over a three-month period. Topics included: (1) introduction to HVC and associated ethical dilemmas, (2) inpatient vs outpatient costs and discharge planning, and (3) barriers to HVC and the cost of diagnostic imaging. Sessions incorporated interactive "choose-your-own-adventureâ€ clinical cases, breakout discussions, and didactic teaching. Content was adapted from the ACP HVC curriculum and informed by needs assessment findings. Interns completed post-session surveys evaluating confidence, knowledge, and session effectiveness.

Results

Participation: 21 interns submitted reflections; 21 completed post-session 2 surveys, and 16 completed post-session 3 surveys.

Confidence: 94% of interns reported feeling "somewhatâ€ or "veryâ€ confident in applying cost-conscious care principles after the sessions; none reported feeling "not at all confident.â€

Engagement & Effectiveness: 69% rated the sessions as "veryâ€ or "extremelyâ€ effective in helping them overcome barriers to implementing HVC in clinical practice.

Knowledge Gains: The proportion of interns who correctly identified that inpatient CBCs are approximately three times more expensive than outpatient increased from 56% pre-curriculum to 63% post-curriculum, with a corresponding decrease in incorrect responses.

Qualitative Reflections: Interns reflected on ethical dilemmas involving prolonged hospitalizations versus arranging outpatient follow-up. Recurring themes included transportation limitations, insurance barriers, and balancing patient-centered care with hospital resource utilization.

Cost of Discharge Options: Knowledge of SNF coverage improved substantially. In the pre-survey, 33% correctly identified that SNFs are often covered by Medicare but may involve out-of-pocket costs. Post-curriculum, 63% accurately described Medicare's full coverage for the first 20 days and partial coverage through day 100â€”a 29% increase in accurate, clinically relevant knowledge.

Conclusion

This interactive, case-based curriculum effectively addressed knowledge and confidence gaps identified in a prior needs assessment. Interns demonstrated improved cost-awareness, ethical reasoning, and readiness to integrate patient-centered values into high value care decisions. This curriculum offers a scalable model for early HVC education within internal medicine residency training.

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Temporal Trends in Mortality Among U.S. Adults With Dermatomyositis and Polymyositis, 1999-2023: An Analysis of CDC WONDER Data

Introduction:â€¨Dermatomyositis and polymyositis are rare, immune-mediated inflammatory myopathies associated with significant morbidity and increased mortality, often due to complications such as interstitial lung disease, cardiac involvement, or malignancy. Despite advances in diagnosis and treatment, contemporary population-level data on long-term mortality trends remain limited. A better understanding of these trends is essential for identifying disparities, evaluating the impact of therapeutic advances, and guiding public health policy.

Methods:

Using the CDC WONDER database, we analyzed death certificates of US adults aged >25 years where Dermatopolymyositis (ICD-10 Codes: M33) was listed as underlying or contributing causes. Age-adjusted mortality rates (AAMRs) per 100,000 population and annual percent change (APC) were calculated and stratified by gender, race, age and geographical factors.

Results:

A total of 6444 dermatopolymyositis associated deaths occurred between 1999 and 2023. A significant decline in the AAMR was observed, decreasing from 0.17 per 100,000 in 1999 to 0.05 in 2023, with an average annual percent change (AAPC) of -4.3% (95% CI: -4.92 to -3.68; $p < 0.000001$). Women had consistently higher AAMRs than men and showed steeper declines i.e. 0.24 vs. 0.14 in 1999 to 0.07 vs. 0.04 in 2022. Non-hispanic White adults experienced the most consistent and significant reductions (AAPC: -4.47, $p<0.000001$). All four U.S. Census regions exhibited statistically significant downward trends, with the Midwest showing the steepest decline (AAPC: -4.21, $p < 0.000001$). These findings represent broad national improvements in adult mortality over the 25-year period, though with subgroup disparities.

Conclusion:

Mortality from dermatomyositis and polymyositis in the U.S. has declined substantially over the past two decades, reflecting progress in disease recognition, management, and multidisciplinary care. Women consistently exhibited higher age-adjusted mortality rates, and notable differences were observed across racial, ethnic, and geographic subgroups. These patterns point to underlying disparities that persist despite overall improvements. Continued research is needed to uncover the drivers of these inequities and to ensure that future advances in care benefit all patient populations equally.

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PREDICTORS OF CANCER-SPECIFIC SURVIVAL IN METASTATIC COLORECTAL CANCER PATIENTS UNDER 45 YEARS: A SEER ANALYSIS

Purpose:

Incidence of metastatic colorectal cancer (mCRC) in young adults is rising, yet prognostic factors in those under 45 are poorly defined. We sought to identify demographic, socioeconomic, and tumor-related predictors of cancer-specific survival (CSS) in this cohort using a large, national registry.

Methods:

We identified 6,609 patients aged <45 years with mCRC diagnosed from 2000-2022 in the SEER 17-registry database (SEER*Stat v9.040). Cancer-specific death (CSD) and survival time (ST, in months) were our event and time endpoints. Candidate predictors were marital status (MS), SEER abstract type (AT), median county household income bracket (MCIB), urban/rural classification (URC), primary tumor site (PTS), six metastasis indicators, and time from diagnosis to therapy initiation (TDI). Missing predictor values were handled via Multiple Imputation by Chained Equations (MICE) with 11 imputations. Cox proportional hazards models were fit within each imputed dataset and pooled by Rubin's rules.

Results:

There were 4,095 CSD events; median survival was 18 months. Compared to married patients, single status was associated with worse survival (HR 1.25; 95% CI 1.17-1.34; p<0.001), as was widowed status (HR 1.69; 95% CI 1.07-2.67; p=0.024). A linear trend across MCIB brackets showed increasing hazard with lower income (HR 1.50 per bracket; 95% CI 1.20-1.89; p<0.001). Metastatic burden strongly predicted CSD: bone metastases (BoM) conferred HR 1.96 (95% CI 1.74-2.20; p<0.001), and brain metastases (BrM) HR 1.69 (95% CI 1.30 2.21; p<0.001). Tumor site effects included higher hazard for ascending colon primaries (HR 1.41; 95% CI 1.24-1.60; p<0.001), hepatic flexure (HR 1.70; 95% CI 1.39-2.09; p<0.001), and transverse colon (HR 1.30; 95% CI 1.13-1.50; p<0.001) compared to tumors originating from sigmoid colon.

Discussion:

Use of SEER provides robust sample size and generalizability but lacks individual treatment details, which may introduce residual confounding. Employing MICE ensured that cases with incomplete data contributed information, improving statistical validity over complete-case analysis. The association of single or widowed status with poorer survival may reflect differences in social support, healthcare access, and adherence. The pronounced effect of metastatic extent underscores the urgency for earlier detection and intervention in young patients. Clinically, these predictors can inform risk-stratified surveillance and support services tailored to high-risk subgroups.

Conclusions:

In young adults with metastatic colorectal cancer, non-married status, lower socioeconomic status, and metastatic burden independently predict higher CSD, while primary tumor site modulates risk. These findings highlight vulnerable subpopulations who may benefit from intensified surveillance, multidisciplinary support, and personalized treatment strategies.

Ngaba Neguemadji Ngardig

Disha Jangra, Collin Tran, Anna O'Neil, Sheila-Alice Tayie, Salome Mary Samuel, Victoria Lovallo, Michael Asare, Henry Nabeta, Shagun Thakur, Riddick Osei Agyemang, Rayan Alataa, Fouad Kaddour Hocine, Abel Akanyijuka, Dennis Ansah, Akua Amoa, Shazia Khan, Khaja Misbahuddin.

Bronxcare Health System

Comparative Analysis of Zilebesiran and Conventional Antihypertensive Treatments: Exploring a New RNA Interference Strategy

Background: Hypertension is a major global health issue linked to increased cardiovascular risk. Traditional antihypertensives, such as thiazides, beta-blockers, and ACE inhibitors, have been essential in managing hypertension but often face challenges like suboptimal control and adherence issues. Zilebesiran, an innovative RNA interference therapy, targets hepatic angiotensinogen production and offers a novel approach with potentially improved efficacy and compliance due to its bi-monthly dosing. This systematic review and meta-analysis evaluate Zilebesiran's efficacy and safety compared to traditional classes, focusing on blood pressure reduction, safety, pharmacokinetics, pharmacodynamics, and cost-effectiveness, aiming to optimize hypertension management strategies.

Methods:We searched PubMed, Embase, Cochrane Library, and ClinicalTrials.gov for studies up to 2025, focusing on RCTs and observational studies. Keywords included "Zilebesiran," "RNA interference," and various antihypertensive classes. Inclusion criteria encompassed studies comparing Zilebesiran with traditional agents, reporting on blood pressure reduction, safety, and cost-effectiveness. Non-comparative studies and non-English publications were excluded. Data extraction was conducted by two independent reviewers, with meta-analysis using a random-effects model to assess pooled effect sizes and heterogeneity. Study quality was evaluated using the Cochrane Risk of Bias tool and the Newcastle-Ottawa Scale.

Results: Zilebesiran achieved superior blood pressure control in a higher percentage of patients compared to most traditional antihypertensives. It presented fewer adverse effects, primarily mild to moderate, with lower side effect frequency and severity than diuretics and beta-blockers. Long-term use of Zilebesiran showed no significant risks, unlike the renal and metabolic issues associated with prolonged diuretic and ACE inhibitor use. Zilebesiran's bi-monthly dosing, due to favorable absorption and distribution, improved patient compliance over daily dosing required by traditional agents. Minimal contraindications and fewer drug interactions were observed compared to ACE inhibitors and beta-blockers. Despite these advantages, Zilebesiran's higher cost and limited availability pose challenges.

Conclusions: Zilebesiran is a promising alternative for hypertension management, offering superior efficacy and a favorable safety profile. Addressing cost and accessibility is crucial for optimizing its clinical utility.

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Uncovering Risk Factors for Early-Onset Colorectal Cancer: A Real-World Multicenter Study

Introduction:

Colorectal cancer (CRC) incidence in adults under 50 has risen sharply, suggesting a shift in epidemiology. Identifying risk factors may improve early detection and guide screening efforts. Our aim is to identify clinical and metabolic predictors of early-onset colorectal cancer (EOCRC) in a real-world U.S. population.

Methods:

A retrospective case-control study was performed using TriNetX, a federated EHR database from 80+ U.S. healthcare institutions. Adults aged 30-49 with first-time CRC (ICD-10 C18-C20) between 2010-2023 were identified.

Exclusions: prior malignancy, hereditary CRC syndromes (e.g., Lynch, FAP), inflammatory polyposis, or colectomy before diagnosis.

Each case was matched 1:2 to cancer-free controls based on age (±2 years), sex, and year of encounter.

Predictors (within 2 years before diagnosis): obesity (E66), type 2 diabetes (E11), IBD (K50, K51), metabolic syndrome (E88.81), tobacco use (F17, Z72.0), physical inactivity (Z72.3), and family history of GI malignancy (Z80.0).

Multivariable conditional logistic regression was used; AUC evaluated model performance. Multiple imputation addressed missing BMI and HbA1c data.

Results:

3,217 EOCRC cases were matched with 6,434 controls (N=9,651). Mean age: 43.2; 52% male. EOCRC patients had higher prevalence of:

Obesity: 48.3% vs 29.1%

Diabetes: 32.5% vs 18.7%

Metabolic syndrome: 22.9% vs 12.8%

IBD: 9.7% vs 3.3%

Family history: 12.6% vs 5.1%

Inactivity: 18.4% vs 10.2% (all $p< 0.01$)

Independent predictors:

IBD: OR 2.43 (95% CI: 1.99-2.96)

Obesity: OR 2.08 (1.88-2.31)

Family history: OR 2.41 (2.02-2.87)

Metabolic syndrome: OR 1.77 (1.48-2.13)

Diabetes: OR 1.59 (1.36-1.85)

Smoking: OR 1.42 (1.22-1.67)

Model AUC was 0.81. Subgroup analysis revealed stronger associations in African American and Hispanic patients, and in those without prior CRC screening. Sensitivity analysis using Firth's correction confirmed robustness.

Conclusion:

EOCRC is associated with metabolic, inflammatory, and behavioral risk factors. These findings support EOCRC as a distinct entity and highlight the need for risk-based screening strategies for individuals under 50.

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Machine Learning Models for Risk Stratification of Major Adverse Cardiac Events in STEMI Patients Undergoing Primary Percutaneous Coronary Intervention

Purpose: To evaluate machine learning algorithms for risk stratification of in-hospital major adverse cardiac events (MACE) following primary percutaneous coronary intervention (PCI) in ST-elevation myocardial infarction (STEMI) admissions using contemporary national data.

Methods: Using the Nationwide Inpatient Sample (2016-2022), we identified 822,980 weighted STEMI admissions undergoing primary PCI. MACE was defined as mortality, ischemic stroke, or coronary artery bypass grafting after primary PCI. Six machine learning algorithms (HistGradientBoost, CatBoost, LightGBM, XGBoost, Random Forest, and logistic regression) were developed using patient demographics, clinical comorbidities, and institutional factors, with temporal validation performed on 2022 admissions. Model performance was assessed through discrimination (area under the receiver operating characteristic curve [AUROC] with 95% confidence intervals from 1,000 bootstrap iterations), calibration (Brier score), net reclassification improvement, and equity metrics across demographic subgroups.

Results: Among 822,980 admissions (28.5% female, 75.8% white), MACE occurred in 7.2%. HistGradientBoost (AUROC 0.864, 95% CI: 0.854-0.873) and CatBoost (AUROC 0.864, 95% CI: 0.854-0.874) achieved the highest discrimination, though HistGradientBoost demonstrated superior calibration (Brier 0.051 vs 0.152). LightGBM (AUROC 0.863, 95% CI: 0.854-0.873), XGBoost (AUROC 0.861, 95% CI: 0.851-0.870), and Random Forest (AUROC 0.858, 95% CI: 0.848-0.867) all maintained strong predictive accuracy (all Brier scores â‰¤0.052). HistGradientBoost significantly outperformed logistic regression ($p<0.001$), yielding a 2.4% net reclassification improvement. While sex-based performance was equitable (AUROC difference: 0.001), racial disparities emerged, with AUROCs ranging from 0.843 in Hispanic patients to 0.907 in patients classified as Other race.

Conclusion: Our findings suggest that HistGradientBoost may be a superior to traditional statistical approaches for identifying high-risk STEMI patients post-PCI. While HistGradientBoost and CatBoost showed equivalent discriminatory power, the marked calibration differences between them demonstrate that AUROC alone is insufficient when evaluating model performance. The model's variable accuracy across racial groups highlights the need for caution when considering the utility of machine learning in clinical practice. Further research is needed to evaluate the clinical utility of machine learning models in diverse populations.

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Title Automation of Inpatient Medicine with Large Language Model Driven Agents

Introduction

Inpatient medicine requires rapid synthesis of complex patient data, interpretation of evolving guidelines, and decision-making under pressure. This cognitive load, intensified by fragmented digital systems and high patient volumes, contributes to diagnostic error and clinician burnout. Alleviating this burden without compromising clinical accuracy stands to benefit both clinicians and patients.

Generative Large Language Models (LLMs), such as GPT-4o, offer the potential to function not merely as static chatbots, but as autonomous agents, i.e. systems that reason, utilize embedded clinical knowledge supplemented by verified external knowledgebases, and interact with real-world effector tools all in pursuit of a defined goal. In this work, we create a framework that allows GPT-4o to function as a medical agent capable of autonomous navigation through real-world inpatient cases.

Methods

We curated 25 representative clinical cases across cardiology, critical care, emergency medicine, internal medicine, and genetics. Each was converted into a structured file containing patient history, labs, imaging, and clinical guidelines. The LLM agent was equipped with tools simulating clinical capabilities such as lab retrieval, ordering new imaging and investigations, ECG review, and access to evidence-based guidelines.

The agent operated via iterative creation of an internal case narrative and interacted with tools through structured text interfaces. We incorporated a Retrieval-Augmented Generation (RAG) mechanism which allowed the agent to retrieve relevant, up-to-date clinical guidance once a working diagnosis was proposed. RAG helps mitigate LLM limitations like stale training data and improves alignment with current standards of care.

Two expert clinicians per specialty reviewed the model's outputs using four metrics: final-answer correctness, tool use, guideline adherence, and hallucination resistance.

Results

GPT-4o performed well across all domains. Average tool use scored 8.6/10, guideline adherence 8.2/10, and final-answer correctness ranged from 7.6 (emergency medicine) to 10.0 (critical care). RAG integration improved guideline adherence by an average of 20%, including a 52% increase in internal medicine cases (from 5.4 to 7.2). No hallucinations or unsafe recommendations were noted.

The agent demonstrated behavior that closely mimicked clinical reasoning: using ECGs early, ordering targeted labs, and querying guidelines only when appropriate. This structured, iterative workflow

contributed to accurate, guideline-concordant decisions while reducing the need for exhaustive prompt engineering.

Conclusion

LLM driven agents can autonomously execute key elements of inpatient clinical workflows with expert-level performance thereby offering a generalizable framework for clinical automation. Such systems may relieve cognitive burden, improve decision reliability, and serve as a foundation for scalable, adaptive decision support in modern healthcare.

New York | ACP American College of Physicians
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Annual Scientific Meeting

Quality, Advocacy and Patient Safety

Poster Presentations

Jessica Accardi MD

S. Hassan Rahmatullah, MD; Richard Gil, MD; Francisco Alvarez, MD; Andrea Porrovecchio, MD

Westchester Medical Center

FROM NOTES TO INSIGHTS: LEVERAGING AI TO OPTIMIZE CLINICAL DOCUMENTATION IN A HOSPITAL MEDICINE SERVICE

Background

As electronic health record (EHR) usage has expanded, the volume and complexity of data entered into these systems have grown considerably (1). Between 2009 and 2018, the median length of clinical notes increased by 60.1%, along with 10.9% rise in redundancy, a phenomenon referred to as "note bloatâ€ (2). This trend highlights the increasing documentation burden and inefficiencies within workflows. Despite the vast quantity of information stored within EHRs, much of it remains underutilized, particularly in time-pressured environments where rapid, focused chart review is critical to informed clinical decision-making (3). To address these challenges, we implemented a generative artificial intelligence (AI) tool within our EHR system. This AI solution analyzes clinical notes in real time and surfaces relevant, context-specific information to support clinician decision-making. By enhancing the accuracy and completeness of documentation, generative AI integration offers a promising strategy to strengthen both the safety and quality of patient care and clinical documentation.

Methods

In October 2024, the Department of Medicine at a large academic tertiary care center integrated a generative AI software into the existing EHR system to enhance clinical documentation. The initial rollout focused on internal medicine hospitalists, critical care physicians, fellows, physician assistants, and later expanded to internal medicine residents. Implementation involved group training sessions and live demonstrations conducted in collaboration with the software company and hospital leadership. After an initial period of independent use, individual follow-up sessions addressed questions and provided additional instructions. We then evaluated user adoption and assessed documentation-related outcomes, including complications or comorbidities (CC), major complications or comorbidities (MCC), severity of illness (SOI), Case Mix Index (CMI), and the Elixhauser Risk Adjustment Index.

Outcomes

Following the implementation of generative AI software within the EHR, adoption rates reached 100% among hospitalists on non-teaching services and 50% among those on teaching services. Comparative analysis between users and non-users of the AI tool demonstrated measurable improvements in documentation quality and clinical coding outcomes. Specifically, the capture rate of CC and MCC increased by 12.1%, with a 19.8% improvement per encounter. Additionally, SOI rose by 3.9%, and the CMI increased by 2.4%. The Elixhauser risk adjustment index per encounter also showed a 23.6% increase, reflecting enhanced documentation of patient complexity.

Impact on patient care

The integration of generative AI into the EHR system was associated with high user adoption rates over time through the use of large group and individual training sessions. Which in turn has led to significant improvements in documentation quality and clinical coding metrics. Thus leading to an improved ability to capture patient complexity, and support more informed decision-making highlighting its value as a strategy to improve the quality and safety of patient care.

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Development of an Electronic Based-Trigger to Identify Causes of Insulin-Induced Hypoglycemia in Hospitalized Patients

Background:

Hypoglycemiaâ€”defined as blood glucose levels less than 70 mg/dLâ€”is a common medication-related harm event resulting from insulin administration. It is highly prevalent in the inpatient setting and associated with adverse outcomes including prolonged hospital stays and increased mortality at severe levels. As part of a quality improvement project, we developed an electronic-based trigger to identify cases of hypoglycemia at NYU Langone Health, a large academic health center in New York City. The purpose was to identify trigger-positive cases and analyze reasons for insulin-mediated hypoglycemic events among hospitalized patients.

Methods:

A multi-step validation approach was used to develop an electronic-based trigger that identified cases of hypoglycemia among hospitalized patients between January 1 to December 31, 2023. Cases met trigger criteria if patients received insulin at least 24 hours prior to a recorded hypoglycemic event. After trigger validation, a subset of trigger-positive charts was qualitatively analyzed to generate a codebook with primary and secondary themes relating to reasons for hypoglycemia. The remainder charts were analyzed using the finalized codebook. A sub-analysis of severe hypoglycemic eventsâ€”defined as blood glucose less than 40 mg/dLâ€”was further performed to determine thematic variations from the main study group.

Results:

The electronic-based trigger identified 111 hypoglycemic events and 31 severe hypoglycemic events. We found that, in both hypoglycemia and severe hypoglycemia groups, the most common primary reason for insulin-induced hypoglycemia was related to errors in insulin orders. Common secondary reasons within this primary theme included providers starting or adjusting insulin at too high of a dose and failing to discontinue insulin despite falling glucose levels. Other primary themes that emerged included changes in dietary status, treatment for elevated potassium levels, and patients not being appropriate candidates for insulin or being more appropriate for oral medications.

Conclusion:

Triggers are effective tools to analyze large amounts of data from electronic medical records (EMR). We successfully developed an electronic-based trigger to identify and evaluate reasons for insulin-mediated hypoglycemic events among hospitalized patients at a large academic center. Given that multiple causative etiologies emerged in our analysis, we present multifaceted solutions to prevent hypoglycemia in hospitalized patients on insulin at our institution. Solutions include creating best practice alerts within our EMR to flag falling glucose levels and forming educational groups for providers. Short-term areas for improvement further include modifying our institution's protocol for treating elevated levels of potassium and improving communication among teams about changes in dietary status.

Chloe Krugel

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Implementation of an Ambulatory Practice Safety Briefing at an Academic Primary Care Network in Northeastern New York

Background

Although the majority of medical care occurs in the outpatient setting, previous efforts to improve patient safety have focused primarily on the inpatient setting. Common errors in the outpatient setting include communication errors, diagnostic errors, and medication errors (including vaccinations). Furthermore, children with special healthcare needs and medical complexity are more likely to experience an adverse event. Efforts to standardize error surveillance and develop a culture of safety are promising strategies to help address and minimize adverse events in the ambulatory setting.

Our academic institution in Northeastern New York has 4 primary care sites in the county, which serve a diverse patient population. Our parent and Children's Hospitals run a daily safety huddle, but outpatient medicine is not represented well in these briefings. After discussions with key stakeholders, including division chiefs, nursing leadership, executive staff and the Medical Director's office, the decision was made to establish a dedicated safety briefing to focus on outpatient safety issues.

Methods

Staff from 4 primary care practices (2 general pediatric sites, 1 Internal Medicine, 1 Med-Peds) developed an Ambulatory Practice Safety Briefing to be held virtually once weekly to review any major safety events, as well as perceived threats to patient and staff safety. One moderator, who is either a physician, nurse lead, or administrative lead, presents data weekly. This briefing started a soft go-live in December 2024, and a full go-live in January 2025. Administrative and nursing staff from each site, as well as representatives from security, IT, epidemiology, pharmacy, lab, and radiology present weekly. To evaluate the effect of these safety briefings on safety intelligence reporting, the number of Safety Intelligence (SI) reports have been tracked pre- and post-intervention.

Results

There was a noticeable increase in SI reports around the time of both go-live events, with an increase from about 30 reports a month pre-implementation to over 45 reports a month post-implementation. The largest increase was noted in the Internal Medicine practice. In addition, during the 1st quarter of 2025, 2 root cause analyses (RCAs) were planned based on outpatient safety events, compared with 0 in the year prior.

Conclusion

The development of the Ambulatory Practice Safety Briefing has increased error reporting in the outpatient setting. Initial feedback and data is promising, and continued efforts to encourage safety reporting in the outpatient setting is warranted.

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Demystifying the Scary Bugs and Confusing Drugs: Introduction of a Standardized Approach to Antibiotic Selection to Third-year Medical Students

Third-year medical students (MS3s) rotating through their Internal Medicine (IM) clerkship frequently encounter patients who require antibiotic therapy. Given the increasing threats of antimicrobial resistance, the need for antimicrobial stewardship cannot be understated. The clinical reasoning and medical knowledge required for initial antibiotic selection can be daunting. Appropriate antibiotic use is oftentimes a challenging concept to learn for students without a structured framework to help clinically reason through the decision. In order to better facilitate MS3s' understanding of antibiotic selection, we implemented a novel interactive, case-based session into the IM Clerkship academic half day. This 90-minute teaching session has been implemented in two cohorts of MS3s thus far (n = 56). A pre-intervention needs assessment was conducted and found that only 53% of students received formal teaching on antibiotic selection during their clerkship. Furthermore, only 15% were able to describe an approach they use for antibiotic selection, with a majority (58%) having no approach at all. This session helps address the variability and gaps in antibiotic teaching.

During this teaching session, students are introduced to a stepwise approach to clinical reasoning through selecting antibiotics based on host factors, pathogen factors, and other clinical risk factors. Students then work through two clinical cases (community acquired pneumonia & urinary tract infection) applying this framework. They work in small groups to answer questions at each step of the patient's clinical course and address any changes (e.g., clinical decompensation after empiric treatment initiation, finalization of culture sensitivities, etc.), while referencing the stepwise approach to demonstrate its utility and adaptability. Then students debrief with the 4th year student leader, IM residents, and clerkship faculty facilitators.

The first cohort's post-intervention survey focused on obtaining feedback on session duration, timing, and content difficulty. Overall feedback supported the current approach. The second cohort survey assessed how helpful students felt the session was. 89% found the session â€˜helpful' or â€˜somewhat helpful' for their learning and 83% anticipated utilizing the approach taught for future, applicable clinical situations. Students were also asked to self-assess their knowledge on key concepts pre- and post-intervention. They were provided with a rating scale for guidance, ranging from 0 to 100, with 100 being full proficiency. Ratings across all concepts increased 45% from an average of 50 (n=26) pre-intervention to 72 (n=18) post-intervention. These preliminary results demonstrate a positive impact on students' approach to antibiotic selection. Future directions could include antibiotic stewardship learning points across other AHD sessions and improving engagement through gamification.

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RUNNING A CODE: CARDIAC ARREST FOR THE RISING SENIOR

Purpose: In-hospital cardiac arrests are often identified, led, and coordinated by internal medicine residents, making effective code leadership a core competency. Despite completing a full year of medical training, individual resident confidence in ACLS and cardiac resuscitation remains low. This project utilizes an interactive lecture series to improve the confidence and skills needed for a rising PGY-2 to responsibly run a medical code (ACLS), recognize cardiac rhythms and execute appropriate interventions.

Methods: Pre-intervention confidence in organizing, leading, recognizing, and managing cardiac arrest was assessed using a 5-point Likert scale, and knowledge of ACLS and institutional protocols was evaluated through multiple-choice questions. A 60-minute interactive, small-group lecture was conducted, where residents worked in teams to work through cases that progressed into cardiac arrest. This approach offered a controlled, low-acuity environment for residents to develop and refine clinical reasoning and decision-making skills. A post-test was administered after the session to evaluate an improvement in both knowledge and confidence. 6-month retention data will be collected. In addition, this lecture will be used as a resource for the ICU consult elective, which will include high-fidelity simulation training and real time RRT/Code experience to improve cardiac resuscitation efficacy and experience.

Results: 30 rising PGY-2 residents participated in this lecture, 14 identified as female, 13 identified as male, and 3 did not report gender. Results showed a statistically significant increase in both overall confidence ($p= 1.24\times10^{9}$) and knowledge ($p= 8.99\times10^{9}$) following the session. Post-lecture confidence was notably higher in male residents ($p = 3.84\times10^{3}$), despite no significant differences in knowledge scores between genders at either the pre-test ($p = 0.851$) or post-test ($p = 0.764$). Six-month retention scores are still pending.

Conclusion: A structured, interactive educational session significantly improved both knowledge and self-reported confidence in cardiac arrest management among rising senior residents. The persistent gender-based confidence gap, despite equal foundational knowledge as well as post-intervention knowledge acquisition, underscores the need to integrate confidence-building strategies into clinical training. Long-term retention data will provide additional insight into the durability of this intervention and inform future curricular design. Furthermore, embedding this module within a longitudinal ICU elective, supported by simulation and real-time experiences, may help bridge the confidence gap and further prepare internal medicine residents to lead life-saving interventions with competence and clarity.

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PRIMARY CARE AND BEYOND: EVALUATING CULTURAL HUMILITY TRAINING FOR MUSLIM PATIENT CARE

PRIMARY CARE AND BEYOND: EVALUATING CULTURAL HUMILITY TRAINING FOR MUSLIM PATIENT CARE

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Purpose: To evaluate the impact of a cultural humility workshop on resident knowledge and confidence in caring for Muslim patients, and to compare outcomes between primary care specialties (PC) and non-primary care specialties (NPC).

Methods: This was a pre-post survey IRB-exempt intervention conducted across 8 residency programs (internal medicine, family medicine, anesthesia, general surgery, pediatrics, and emergency medicine) at three hospitals in New York State. Residents were invited to participate in a one-hour workshop on cultural humility in the care of Muslim patients. The session included didactics on cultural humility versus cultural competence, key considerations for Muslim patients, and an introduction to Madeline Leininger's Transcultural Care Model. Participants then engaged in facilitated case-based discussions applying the model to scenarios involving modesty, gender-concordant care, and Ramadan fasting in both inpatient and outpatient settings.

Pre- and post-workshop surveys assessed self-reported knowledge, confidence, and perceived clinical relevance using Likert-scale items. Statistical tests, including McNemar's chi-square test, were used to assess within-group change. Subgroup analyses compared responses between primary care (internal medicine, family medicine, pediatrics) and non-primary care residents.

Results: A total of 107 residents (61 PC, 46 NPC) completed the pre-survey; 22 identified as Muslim. Among non-Muslim residents, only half (50%) reported having a structured approach to addressing patients' cultural or religious preferences prior to the workshop.

Of the 86 residents who completed both surveys, improvements were observed across all knowledge areas related to culturally responsive care. Statistically significant gains were noted in accommodating same-gender personnel during examinations ($p = 0.0164$) and allowing family to remain in the room for support ($p = 0.0023$).

Post-workshop, over 80% of residents reported increased confidence in approaching patients with cultural humility, asking about religious and cultural needs, and modifying care plans accordingly. Primary care residents generally reported greater gains than their non-primary care peers. The majority (82.28%) indicated they were very likely to apply workshop content in clinical practice.

Conclusion: This study demonstrates that a cultural humility workshop significantly impacts resident knowledge and confidence in caring for Muslim patients. Although comparisons between PC and NPC residents were not statistically significant, the observed effect sizes suggest potential for more specialty-specific findings with continuation of this workshop. Importantly, the majority of residents found the workshop highly relevant and reported increased preparedness to deliver culturally responsive care. Most participants expressed strong intent to implement the knowledge and skills gained in their clinical practice, reinforcing the workshop's practical value.

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BOLSTERING CONFIDENCE AND PRESCRIPTION OF CONTINUOUS GLUCOSE MONITORS IN THE PRIMARY CARE CLINIC: A QUALITY IMPROVEMENT INITIATIVE

Purpose: To enhance provider confidence, knowledge, and prescribing rates of continuous glucose monitors (CGMs) within a large primary care system through targeted educational interventions.

Methods: A 7-minute educational video and a concise, one-page CGM prescribing guide were developed and distributed starting in October-November 2024 to six primary care clinics in Internal Medicine (IM) and Family Medicine (FM) at Stony Brook University Hospital (SBUH). The materials provided step-by-step prescribing instructions, shortcuts, and clarified insurance criteria. Provider confidence regarding CGM prescribing was assessed using matched pre- and post-intervention surveys on a Likert scale, analyzed with independent samples t-tests. Perceived barriers were assessed using free text responses in pre and post-surveys. Additionally, monthly CGM prescribing rates were tracked from January 2024 through May 2025 using data provided by the hospital IT department, capturing only new first-time CGM prescriptions and excluding refills, limited to the sites where the educational material was released.

Results: Fifty-two providers completed pre-intervention surveys (18 IM attendings, 14 FM attendings, 4 APPs, and 16 IM residents). Post-intervention surveys (n=27) demonstrated a significant increase in confidence scores, from the pre-surveys, from an average of 2.90 to 4.19 ($p < 0.001$, n=27, paired T test, using a 1 of 5 scale for providers). Open-text responses in post surveys highlighted persistent barriers to CGM prescription: insurance (18), visit time constraints (11), and patient education needs (12). Overall, new CGM prescriptions across SBUH primary care sites (affected by the intervention) increased from an average of 11.9 per month (Jan-Sep 2024) to 47.4 per month post-intervention (Dec 2024-May 2025, $p < 0.001$, two-sample independent T test). New CGM prescriptions specifically in high-risk patients with diabetes (A1C >9%) increased from average of 4.11 per month before the intervention (Jan-Sep 2024) to 15.0 after the intervention (Dec 2024-May 2025), with a two-sample independent t-test showing a statistically significant difference ($p < 0.001$).

Conclusions: A brief, structured educational intervention led to a statistically significant improvement in provider confidence and knowledge regarding CGM prescribing. The intervention included a targeted video module and an accompanying information sheet that addressed common barriers, such as uncertainty about insurance coverage and documentation requirements. Following implementation, CGM prescribing rates more than tripled across participating primary care clinics, with a comparable rise among high-risk patients with A1C >9%. These results demonstrate that concise, practical educational tools can effectively reduce provider-level barriers and promote the adoption of CGM technology. By clarifying insurance requirements, offering time-saving resources, and improving workflows, the intervention not only boosted provider confidence but also drove a substantial increase in prescribing, presenting a scalable model to improve diabetes care.

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IMPROVING DISCHARGE COMMUNICATION THROUGH A RESIDENT-LED EDUCATION MODEL

Background: Ineffective discharge education is a well-documented contributor to patient confusion, medication nonadherence, missed follow-up appointments, and preventable hospital readmissions. In many inpatient settings, discharge instructions vary in quality and clarity due to inconsistent communication practices, lack of a standardized process, and workflow challenges that limit time for patient education.

Objective: We implemented a quality improvement project to improve patient understanding and satisfaction with discharge instructions by implementing a structured, resident-led discharge education protocol embedded into the daily workflow of an internal medicine teaching service.

Methods: A mandatory educational session was conducted for all internal medicine residents and hospitalists. The training focused on the use of plain language and strategies for delivering clear and patient-centered communication. Residents reviewed a sample discharge summary and participated in small-group role-plays with hospitalists to practice effectively communicating discharge plans. A structured discharge education protocol was implemented, assigning senior residents on the primary team to provide consistent and comprehensive discharge teaching to patients prior to discharge. We surveyed patients' satisfaction with their discharge experience before the start of the intervention and also after it was in place using a 5-item Likert style survey. In addition, we also surveyed the residents before and after the training on their confidence and comfort in providing discharge education.

Results: Patients' post-intervention results showed improvements on all survey items. Specific improvements were in feeling their diagnosis and course of treatment was explained to them ($t(55)=2.91$, $p<.01$) and explaining their medications ($t(55)=2.20$, $p=.03$). Additionally, residents reported greater confidence and comfort in providing discharge education following training. Embedding the discharge education process into daily clinical workflow enhanced the consistency and quality of patient-provider communication.

Conclusion: Implementing a standardized, resident-led discharge education model significantly improved both patient satisfaction and provider confidence in delivering discharge instructions. By integrating the process into daily workflow, this intervention addressed common barriers to effective discharge communication and supported safer transitions of care.

Limitations: The project was limited by a small sample size and short duration. Patient satisfaction was measured through subjective surveys, which may be influenced by external factors. Additionally, clinical responsibilities sometimes limited senior residents' ability to consistently provide discharge education. Long-term outcomes such as medication adherence, follow-up appointment completion, and hospital readmission rates were not evaluated.

Next Steps: To enhance sustainability, future efforts will include training junior residents and monitoring long-term outcomes. Post-discharge follow-up via surveys or phone calls will also be used to gather ongoing patient feedback and assess the lasting impact of the intervention.

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Association Between Asthma Outcomes and County Health Factors in New York State

Purpose for study: County Health Rankings (CHR) program evaluates county-level health across the nation, providing a summary z-score for each county and determine county health rankings within the same state annually. This study aims to investigate the association between county health factors as measured by CHR, and asthma outcomes in NYS, including current adult (aged â‰¥18 years) asthma prevalence, emergency department (ED) visits, hospitalizations and mortality.

Methods: We obtained county-level data on adult asthma prevalence, ED visits, hospitalization and mortality rates for 2019-2021 from the New York State Department of Health (NYSDOH). Overall County health factors z-score was calculated by an equation using weighted measures in 4 different categories: Health Behaviors, Clinical Care, Social and Economic Factors, and Physical Environment. We examined NYS county health factors z-score from 2023 CHR report, which covers data for 2017-2021. The higher z-score indicates the worse county ranking. The associations between asthma outcomes and Overall County health factors z-score and each category z-score were analyzed by negative binomial regression.

Results: From 2019 to 2021, the adult asthma prevalence was 9.8%, an age-adjusted ED visit rate was 49.0 per 10,000, a hospitalization rate was 7.1 per 10,000, and a mortality rate was 12.3 per 1,000,000. Among NYS's 62 counties, Bronx had the highest z-score (the least healthy county) whereas Nassau had the lowest z-score (the healthiest county) for Overall Health Factors. In our analysis, counties with higher z-score of Overall Health Factors were significantly associated with increased rates of ED visits (Rate Ratio [RR]: 1.47, 95% Confidence Interval [CI]: 1.01-1.99, p = 0.011). In counties with high z-score

of Health Behaviors, there was an associated with increased asthma prevalence (RR: 1.23, 95% CI: 1.00-1.52, p = 0.049). Counties with high z-score of Social and Economic Factors were strongly linked to higher ED visits (RR: 2.35, 95% CI: 1.81-3.05, p < 0.001), hospitalization (RR: 1.68, 95% CI: 1.06-2.68, p = 0.027), and mortality (RR: 1.94, 95% CI: 1.20-3.15, p = 0.007). Similarly, counties with high z-score of Physical Environment were associated with increased ED visits (RR: 1.65, 95% CI: 1.15-2.41, p = 0.007) and significantly higher hospitalization rates (RR: 3.02, 95% CI: 1.83-5.03, p < 0.001).

Conclusion: County-level disparities in health factors behavioral, socioeconomic, and environmental domains are significantly associated with worse asthma outcomes in NYS. These findings suggest tailored public health interventions with strategic resource allocation for asthma should be implemented in disadvantaged communities to improve asthma management and reduce health disparities.

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MEDICAL STUDENT-RUN, COST-FREE TOBACCO TREATMENT PROGRAM MAINTAINS QUIT SUCCESS FOR HOSPITALIZED SMOKERS

The Rochester Model is a hospital-based quality improvement program that provides bedside and post-discharge tobacco cessation counseling. This study examined whether briefly trained medical students could assume those counseling duties from overstretched nurses while maintaining the program's benchmark six-month quit rates for hospitalized smokers.

Medical students trained for bedside and post-discharge counseling through two one-hour Zoom sessions run by a faculty member. These students participated in all elements of the program, including screening and enrolling patients from hospital units and assigning patients to counselors. Students performed bedside counseling, then called patients (at 3 and 6 weeks) along with New York State Quitline counselors after discharge. Counselors obtained the patients' smoking status during outcome calls at 4 weeks, 3 months, and 6 months.

Currently, 37 medical students participate. From 1/29/22 to 5/18/24, 250 patients enrolled, and counselors obtained outcomes up to 6 months. The 7-day point-prevalence quit rates for the as-treated (AT) patients were 66/121 (55 %), 37/77 (48 %), and 33/70 (47 %) at 4 weeks, 3 months, and 6 months, respectively; for the intent-to-treat (ITT) patients, these were 66/250 (26 %), 37/250 (15 %), and 33/250 (13 %). Before 2022, nurses had achieved AT quit rates of 50 %, 42 %, and 38 % (n = 178, 151, 143) and ITT quit rates of 23 %, 16 %, and 14 % (n = 385) at the same time points, respectively. Students showed no significant difference from nurses in quit rates at all time points (4 weeks, 3 months, 6 months) for AT ($p = 0.513, 0.441, 0.246$) and ITT ($p = 0.463, 0.846, 0.747$) groups.

With just two one-hour virtual trainings, medical students became effective bedside and post-discharge counselors, achieving six-month AT quit rates (47 %) that match prior nurse-led outcomes and nearly double the 25 % benchmark self-report AT quit rate for successful hospital programs. By off-loading counseling duties from overstretched staff, this student-powered model offers a scalable, cost-neutral solution for hospitals, all while giving future physicians early, hands-on experience in evidence-based tobacco treatment that will strengthen their clinical toolkit for years to come.

New York | ACP American College of Physicians Leading Internal Medicine, Improving Lives

New York Chapter
American College of Physicians

Annual Scientific Meeting

NYACP Advocacy Interns

Quality, Advocacy and Patient Safety

Poster Presentations

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The Incorporation of a Transgender Care Curriculum into Internal Medicine Training

Learning Objectives:

By the end of the session, learners will be able to:

1.Summarize key healthcare disparities affecting transgender individuals. (SBP2, PC5)

2.Illustrate strategies to promote inclusive care of transgender individuals during the clinical encounter. (SBP2, ICS1, PC5)

3.Rank as important the need to incorporate culturally competent, empathic, and inclusive care into clinical encounters with transgender patients. (SBP2, ICS1)

Methods: A needs assessment surveying our large urban IM program showed 82% of residents cited insufficient training (n=52) and 62% (n=39) cited fear of appearing intrusive as the main barriers to delivering optimal care to LGBTQ+ patients. We implemented a one-hour classroom-based session to approximately 40 internal medicine PGY2 and PGY3 residents across two sessions. Each session included a brief didactic overview (20 minutes) on terminology, health disparities, and evidence-based recommendations, followed by a 30-minute moderated panel discussion with transgender and gender-diverse individuals, and a 15-minute open Q&A. Pre- and post-intervention surveys assessed changes in resident attitudes and comfort using Likert scale questions, and responses were analyzed in aggregate using unpaired t-tests. Qualitative data were gathered via open-text responses and thematically analyzed.

Results: Survey results demonstrated a statistically significant improvement across all four attitudinal domains measured. The greatest change was observed in residents' comfort approaching the clinical encounter ($p < 0.001$) and their understanding of the transgender experience ($p < 0.001$). Thematic analysis of qualitative responses revealed three primary areas of value: (1) insight into lived transgender experiences, (2) appreciation for addressing an underrepresented topic in medical training, and (3)

acquisition of practical communication strategies including pronoun usage and gender-neutral language. Reported challenges included scheduling logistics and balancing audience size with intimacy.

Conclusions: Our findings support the transgender patient panel as an effective educational strategy for improving resident-reported comfort and awareness in delivering inclusive care. Incorporating lived experience into medical education fosters empathy, addresses common barriers such as fear of intrusiveness, and enhances practical clinical communication skills. These findings underscore the importance of ongoing curriculum development and advocacy initiatives aimed at closing persistent gaps in transgender health education and highlight centering patient voices and experiences as a meaningful strategy to address these gaps.

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ADVOCATING FOR BETTER ENFORCMENT OF THE N.Y. STATE (NYS) LAW PROHIBITING FLAVORED ELECTRONIC NICOTINE DELIVERY SYSTEMS (ENDS)

Summary: The World Health Organization defines “young people” as covering the age range of 10-24 years. Over 80% of adult smokers begin smoking by 18 years of age, with 99% of first use by 26 years of age. Tobacco companies have a long history of targeting young people, with documentation dating back decades. One example from 1983, shows R.J. Reynolds Tobacco Company stating in a confidential report that “younger adults are the only source of replacement smokers… today’s younger adult smoking behavior will largely determine the trend of industry volume over the next several decades.” The tobacco industry realized that if they don’t create new smokers around adolescence, they won’t ever start smoking. So, the industry spent a significant amount of time and resources in developing strategies to attract young people. A crucial tactic is the usage of flavored tobacco (e.g., candy flavors like chocolate, fruit flavors like mango, mint/menthol flavors, etc.) to lure young people.

In 2020, NYS banned ENDS (i.e., electronic/e-cigarettes, vapes, etc.) from selling flavored products. This is critical since 10.1% of American high school students reported current use of tobacco products, with 7.8% of high school students utilizing ENDS, 88.2% of which used flavored ENDS in 2024. ENDS can result in negative health effects like addiction, dual usage (i.e., concurrent cigarette use), cognitive disorders (e.g., ADHD), mental health effects (e.g., depression, anxiety, etc.), pulmonary/cardiovascular disease, and various other diseases. Unfortunately, the enacted ENDS flavor ban law has loopholes which limit its proper implementation. Loopholes like allowing the FDA’s pre-market tobacco product authorization to override NYS flavor bans, expanding flavor ambiguity via specific terms (e.g., ice, clear) or utilizing synthetic/analog compounds not covered by the law, allowing retailers to deny inspectors access to their premises without penalty, allowing retailers to claim any product in their possession is for “out-of-state sales” without proof, allowing wholesalers to continue to provide ENDS to NY retailers, and many other workarounds. Therefore, NYS bill A2182/S5196, which closes many of these loopholes, is essential towards allowing for proper enforcement of the current law and limiting young people’s use of ENDS.

Impact of Efforts:

By collaborating with a tobacco cessation coalition, 15 organizations were involved in an advocacy event held on 5/21/25, 48 organizations in total backed the bill, 8 of which wrote memorandums of support, and a detailed package promoting for the bill’s passage was used to advocate alongside in-person visits to a total of 27 NYS legislators. Many of which stated they would vote for the bill. However, this remains a long shot, since no significant non-budget anti-tobacco bills have passed through the assembly health committee since 2019. However, the true impact of these efforts will only be revealed with time.

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Telehealth at a Crossroads: The Critical Role of Advocacy in Shaping Virtual Care Policy

Prior to the COVID-19 pandemic, telehealth services were largely restricted to rural settings or special circumstances, with Medicare imposing significant geographic and coverage limitations. These restrictions excluded many homebound, urban, and suburban patients from receiving telemedicine as a covered benefit and confined services to a narrow network of providers. [1,2]

The pandemic dramatically transformed this landscape. Faced with unprecedented challenges, healthcare systems rapidly expanded telehealth to meet growing demands. Emergency measures granted payment parity, allowing telehealth visits to be reimbursed at rates equivalent to in-person encounters, and removed many geographic and originating site restrictions [3] to ensure continuity of care. Providers and patients alike came to appreciate the convenience, flexibility, and effectiveness of telehealth, cementing its role in modern healthcare delivery.

However, the future of telehealth remains uncertain. The expiration of many pandemic-era flexibilities has resulted in a fragmented patchwork of policies across federal, state, and private payers. This has fueled a robust advocacy movement aimed at ensuring telehealth's integration into standard healthcare practice.

State Level:

Multiple legislative efforts have been introduced to sustain and expand telehealth coverage. In New York, Assembly Bill AB6334 seeks to broaden telehealth services and grant discretion to the commissioner regarding covered services [4] . Assembly Bill AB1691 proposes extending existing reimbursement flexibilities from 2026 to 2028 [5] . These two bills respectively are in committee assembly review. Bills have that have passed the Senate include SB354 focuses on equitable reimbursement for telehealth services [6] , while SB4720 advocates for parity in telehealth reimbursement for individuals with developmental disabilities or traumatic brain injury [7].

Federal Level:

At the federal level, temporary telehealth flexibilities—initially set to expire in December 2024—were extended through September 30, 2025, by the Full-Year Continuing Appropriations and Extensions Act, 2025 [8]. This act also incorporated telehealth into the Medicare Diabetes Prevention Program. Several federal bills seek to make telehealth flexibilities permanent, including S.126 and S.763 which addressed Medicare telehealth and high-deductible health plan rules, and House bills such as HR 1407 which proposes elimination of geographic limitations, HR 1614 which authorizes telehealth services for physical, occupational therapy and speech pathology. HR1899 also proposes permanent audio-only coverage and HR1720

proposes expanding telehealth use in the setting of hospice care to ensure continual services [9] .

Advocacy efforts from healthcare professionals, community groups, and lawmakers remain critical as stakeholders work to protect and advance telehealth policy. With key decisions approaching, particularly the September 2025 deadline, sustained advocacy will play a pivotal role in shaping the future of virtual care and ensuring equitable access to healthcare services nationwide.

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Artificial Intelligence Expansion in Prior Authorization: A Policy Proposal to Address Rising Administrative Burden and Protect Equitable Access to Care

Background

In recent years, Artificial Intelligence (AI) and Machine Learning (ML) have been increasingly adopted by health insurers to automate claims processing, particularly regarding prior authorization (PA). While these technologies offer potential to streamline administrative workflows and reduce costs, their application in utilization review has introduced serious risks. AI-driven systems often operate with minimal human oversight and limited clinical nuance, leading to a rise in erroneous denials leading to delays in care and significant administrative burden on healthcare providers.

PA was originally designed as a cost-containment tool to ensure that medical services and prescriptions meet medical necessity prior to coverage. However, automation through AI has amplified existing issues within this system. In Medicare Advantage plans, algorithms restricted postacute care based on rigid models, ignoring clinician input (1). Lawsuits against insurers like UnitedHealthcare and Humana revealed that tools such as nH Predict triggered premature discharges and widespread denials, often justified by undisclosed proprietary logic. Over 90% of these denials were overturned on appeal, highlighting their inaccuracy (2).

These concerns are highlighted by broader survey data. A 2024 AMA survey found that physicians handle 39 PA requests weekly, consuming 13 hours of staff time. Nearly 40% employ dedicated PA staff, costing $2,161–$3,430 per physician annually. PA was linked to burnout and increased utilization by 89% of physicians, often driving ineffective care and avoidable hospitalizations (3). The black-box nature of many AI tools, reliance on biased or non-representative data, and inability to incorporate individualized clinical context further compromise both the integrity and equity of care decisions.

As AI/ML continue to be deployed in high-stakes coverage decisions, it is critical to address their current limitations, reinforce clinical oversight, and implement regulatory safeguards to ensure that technological efficiency does not come at the expense of patient safety or physician autonomy. California’s Senate Bill 1120 (SB 1120) offers a strong model. Effective January 2025, it prohibits insurers from relying solely on AI to deny or modify care, mandates licensed provider involvement, and requires transparency in algorithm use. It also enables regulatory oversight by the Department of Managed Health Care and the Department of Insurance, with penalties for non-compliance (4). These standards should be adopted nationally. These state level regulations remain at risk- the budget reconciliation law- One Big Beautiful Bill Act" (OBBBA), effective as of July 4, 2025 had previously included a 10-year moratorium on state AI regulation (5). The implications of AI expansion without regulation are concerning given AI's shortcomings in the prior authorization model in its nascent stages.

Impact of efforts

If properly regulated, AI has the potential to become a powerful tool to reduce the $335 billion spent annually on healthcare administration—particularly in streamlining prior authorization and claims

processing. With the right safeguards, AI can ease the administrative burden on physicians, allowing them to spend more time on patient care rather than paperwork.

By ensuring that care denials are reviewed by appropriately trained specialists, maintaining prior authorization validity for chronic conditions, and basing decisions on peer-reviewed clinical evidence, AI-driven systems could support—not supplant—clinical expertise. This would help restore physician autonomy, reduce delays in care, and improve overall system efficiency. Transparency measures such as requiring insurers to publicly report approval and denial rates and to clearly disclose how AI contributes to each denial would enhance accountability and trust.

Equity in patient care would also be advanced by mandating that AI systems be trained on diverse, representative datasets and regularly assessed for bias. Extending regulatory oversight—modeled after existing frameworks by the FDA, Health Canada, and the UK MHRA—to insurance-based algorithms would ensure that these tools are held to clinical standards of safety and fairness (6).

With meaningful oversight, AI can improve care delivery, reduce federal healthcare spending, and empower physicians to deliver timely, evidence-based care—ensuring that technology enhances, rather than undermines, the quality and equity of our healthcare system.

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Advancing Obesity Policy and Clinical Integration: A Legislative and Advocacy Agenda for the New York Chapter of the American College of Physicians (NYACP)

Summary

Obesity is a growing driver of chronic disease and health disparities across New York, yet it remains under-prioritized in both clinical care and public policy. Despite strong evidence supporting prevention and treatment, primary care physicians often lack adequate training, face time constraints, and encounter inconsistent reimbursement for obesity-related interventions. The New York Chapter of the American College of Physicians (NYACP) has identified key legislative and policy gaps that, if addressed, could significantly improve obesity outcomes statewide.

Education and Training Gaps

A 2018 survey of U.S. medical schools found that one-third lacked formal obesity education and only 10 hours on average were dedicated to the topic over four years. Less than 40% of schools covered core competencies in obesity care. Among internal medicine residency programs, while two-thirds recognized obesity as an educational objective, most lacked formal rotations and adequate training on topics such as weight stigma, psychosocial factors, and pharmacologic treatments.

To address these gaps, NYACP supports integrating obesity education into medical school and residency curricula, expanding faculty development, and prioritizing obesity as a critical component of chronic disease management. A relevant legislative effort is Senate Bill S4169 (2025), introduced by Senator Kevin Parker, which proposes creating a state Office of Nutrition and Fitness to lead public health education and promote nutrition-focused interventions.

Sugar-Sweetened Beverage (SSB) Policy

Excess sugar consumption—especially from sugary beverages—is a major contributor to obesity. SSB taxes are a proven public health tool that can reduce consumption, lower healthcare costs, and prevent disease, especially in vulnerable populations.

NYC has implemented several measures, such as the Sweet Truth Act (2023), requiring large chain restaurants to display sugar warnings on menu items with over 50 grams of added sugar. Although the city has not enacted a sugar tax, proposed state legislation includes:

• Assembly Bill A3490 (2025) – Sponsored by Assembly Member Karines Reyes, this bill would impose an SSB excise tax and create a Community Health Equity Fund.

• Senate Bill S2330 (2025) – Introduced by Senator Gustavo Rivera, mirrors A3490.

Food and Beverage Marketing Regulation

Aggressive marketing of unhealthy foods—especially to children—drives poor dietary habits and increases obesity risk. These practices disproportionately target low-income and minority communities.

To address this, Assembly Bill A4424 and Senate Bill S213B propose stricter oversight of misleading food advertisements, especially those targeting children. The bills empower the state attorney general to take legal action against violators. Although similar bills stalled in previous sessions, the current versions have gained momentum in the 2023–2024 legislature.

Insurance Coverage and Access to Treatment

Comprehensive coverage for obesity treatment remains limited, particularly under Medicaid. NYACP supports expanding access to medical nutrition therapy, behavioral counseling, bariatric surgery, and FDA-approved anti-obesity medications. Key legislative initiatives include:

• Assembly Bill A4211 (2025) and Senate Bill S8959 (2024) – Mandate Medicaid coverage for comprehensive obesity care.

• Assembly Bill A2715 (2025) and Senate Bill S5798 (2025) – Ensure Medicaid covers FDA-approved weight management medications for adults with obesity and related conditions.

Future Directions

NYACP's advocacy agenda includes supporting:

• Implementation of an SSB tax to reduce consumption and fund community health programs

• Integration of obesity education across medical training

• Regulations to curb unhealthy food advertising, particularly toward children

• Expansion of insurance coverage for evidence-based obesity treatments

By aligning clinical practice with policy reform, NYACP can lead a coordinated strategy to combat obesity, improve provider readiness, and promote equitable health outcomes across New York's diverse communities.

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Effort to Amend the Family Health Care Decisions Act to Apply to Persons with Intellectual and Developmental Disabilities

ABSTRACT

The Family Health Care Decisions Act (FHCDA) is a New York State law that governs how medical decisions are made for incapacitated patients who lack advance directives. It allows a surrogate, such as a spouse, child, parent, sibling, or close friend, to make medical and end-of-life decisions, including those involving life-sustaining treatment. However, individuals with intellectual and developmental disabilities (I/DD) are excluded from the FHCDA.

Instead, health care decisions for those with I/DD must follow a separate process outlined in Surrogate's Court Procedure Act (SCPA) Section 1750-b. This law requires a more complex set of steps, including physician certification of incapacity, possible court-appointed guardianship, and oversight by the Office for People with Developmental Disabilities (OPWDD) or a surrogate decision-making committee. These safeguards were designed in response to historical abuses, including the legacy of institutions like Willowbrook in Staten Island, New York.

While protections remain critical, the dual system results in confusion, delays, and inequitable care. Patients with I/DD may experience prolonged suffering at the end of life while families navigate legal and procedural hurdles. Medical teams often consult hospital legal counsel to interpret unclear or conflicting laws which further delays care.

In response, the New York State Task Force on Life and the Law and the New York State Bar Association (NYSBA) Health Law Section have recommended reform. A Special Advisory Committee concluded that the FHCDA should be extended to individuals with intellectual disabilities, while retaining select protections from SCPA 1750-b. This would streamline care, clarify processes for clinicians, and provide consistent and compassionate treatment for patients with I/DD.

We (the authors) are part of a coalition advocating for legislation to amend the FHCDA to include persons with I/DD. This effort seeks to reduce disparities, prevent unnecessary suffering, and ensure parity in decision-making rights. As physicians in geriatrics and palliative medicine, we believe this change would improve care quality while maintaining ethical oversight. We invite clinicians, families, attorneys, and politicians to join this effort. The next step is to introduce a bill in the New York State Senate and Assembly. By building a broad base of support, we can move this reform forward and close a harmful gap in health care law.

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First, Do No (Financial) Harm: The Impact of Medical Debt

Background: Medical debt has emerged as a significant crisis in the United States, particularly in New York State (NYS), where financial burdens increasingly interfere with patients' ability to seek necessary healthcare services. Approximately 41% of working-age Americans have medical debt, with the total estimated to exceed $195 billion nationwide. In New York specifically, more than 740,000 residents have medical debt in collections, averaging $1,945 per person.

Methods: We analyzed current legislative initiatives addressing medical debt in NYS, reviewed evidence on the impact of medical debt on healthcare utilization and outcomes and evaluated implications for internal medicine practice.

Results: Research from NYU Grossman School of Medicine found that New York patients with medical debt were 2.3 times more likely to delay treatment for chronic conditions and 3.1 times more likely to skip follow-up appointments. A Commonwealth Fund survey found 43% of New York respondents with medical debt reported postponing or avoiding necessary healthcare due to cost concerns.

NYS legislators have introduced several initiatives, including requiring facilities to publicly disclose all standard charges (S.7479); the Hospital Medical Debt Relief Program (S.1857), establishing a pilot program to cancel hospital debt for eligible residents; and prohibiting state-operated hospitals from suing patients (S.359).

Discussion: Medical debt disproportionately affects vulnerable populations, creating barriers to healthcare access and exacerbating existing health disparities. For internal medicine physicians, the implications are profound, as 78% report changing clinical recommendations due to patients' financial concerns, potentially compromising optimal care. Medical debt affects medication adherence, with patients more likely to skip prescribed medications or cut pills in half to save money, compromising treatment efficacy. Though the federal No Surprises Act and Hospital Price Transparency Rule provide some protections, significant gaps remain, particularly in NYS legislation.

While the proposed bills attempt to safeguard patients from aggressive collections tactics employed by many healthcare systems, they fail to outline monitoring and enforcement of suggested transparency requirements or address the incommensurate health care costs in NYS that are causing patients to go into debt in the first place. All the while, such policy approaches have generated controversy regarding implementation costs and potential impacts on healthcare providers' finances.

Conclusion: Addressing medical debt requires a comprehensive approach incorporating proposed NYS Senate and Assembly bills alongside additional policy measures and provider practices. Beyond legislation, solutions should include enhanced financial assistance screening, innovative and forgiving payment models, and provider training on discussing costs with patients. Effectively addressing medical debt is essential to ensure that financial barriers do not prevent New Yorkers from accessing needed healthcare and to improve health outcomes across the state.

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A SUMMARY ON REPRODUCTIVE HEALTH POLICY: MEDICOLEGAL CONTEXT AND IMPLICATIONS IN 2025

Reproductive health policy in the United States has long been an area of much legal contention and controversy. In the nineteenth century, common law recognized “quickening” or feeling fetal movement as the beginning of fetal existence, thus only providing abortion after quickening was considered illegal and punishable by law14,15. Lobbying by the nascent American Medical Association (AMA), which released a “Report on Criminal Abortion” that framed abortion as an immoral act of “murder”, lead to the theory that life began not at “quickening” but at conception.14

The advent of safer abortion practices such as vacuum aspiration and medication abortion lead to changes in physician and patient views. In 1973, Roe v. Wade established the constitutional right to abortion in defined circumstances.19 In 1992, Planned Parenthood v. Casey reaffirmed the constitutional right to abortion by emphasizing the importance of adhering to precedents, but replaced the trimester framework with an emphasis on fetal viability.17 However in 2022, the constitutional provision of the right to abortion was dramatically overturned by Dobbs v. Jackson Women’s Health Organization, in a historic reversal of prior case law.7 Since Dobbs, abortion access has been severely curtailed or banned in at least nineteen states, and has led to the criminalizing of a range of reproductive healthcare services, severely harming the patient-physician relationship.5,12,23

Of note, New York State legalized abortion in 1970 and reaffirmed it in 2019.16 Since then, the state has also created provisions to protect physicians who provide abortions from medical malpractice insurance companies and from extradition or arrest in other states related to abortions legally performed in this state also referred to as a ‘shield law’.20,21 Bills that have been introduced include a provision to allow prescription labels for medications to include the name of the prescriber’s practice rather than their own name, allowing pharmacists and RNs to dispense abortion medication, establishing an abortion access fund and allowing pharmacists to inject contraceptives.1-3,20-22

The history of abortion in the United States is tumultuous. The medical profession’s role as a body politic has varied at different times. Current expert medical opinion is best summarized by the ACP’s policy brief in 2023, which asserts unequivocal opposition to government restrictions that would erode equitable access to reproductive health care services including abortion, and laws that impose criminal or civil penalties for receiving, providing or facilitating clinically appropriate reproductive services. It supports one’s right to travel to obtain medical care, for medication to be appropriately mailed, and for the provision of finances by payers to patients who cannot obtain services in their locality.23 Policies such as expanding tele-health, allowing pharmacy dispensation of medications, and improving Medicaid reimbursement, will be those for physicians to focus on as we continue to advocate for our patients and navigate this new era of changing reproductive policy.

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