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Clinical Vignette Symposium 2026

Book of Abstracts

University of Oklahoma –Tulsa

School of Community Medicine

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Dear Colleagues,

It is my pleasure to welcome you to the 15th annual Clinical Vignette Symposium (CVS) hosted by the University of Oklahoma School of Community Medicine. At CVS, trainees present interesting and unusual medical case studies in a conference setting. We look forward to learning the stories behind these unique case reports.

In addition to poster and podium presentations at CVS, authors have an opportunity to upload their work to the Open Science Framework (OSF). Posters and podium presentations uploaded to OSF will be more widely disseminated to a global community. Authors will also be able to include these presentations as citations on their Curriculum Vitae. Awards will be given to the top scoring posters. These awards may be used toward travel or publication fee expenses to further disseminate their scholarly work.

I would like to thank the Tulsa County Medical Society for their generous donation to this year's event. I would also like to thank the presenters as well as those who organized and are hosting CVS this year. We hope you enjoy CVS 2026.

Sincerely,

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ACKNOWLEDGEMENTS

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ORDSA would like to thank all faculty and staff who contributed their time and energy to organizing the 2026 Clinical Vignette Symposium. ORDSA would also like to thank the library for their assistance. Finally, ORDSA would like to thank the Tulsa County Medical Society for sponsoring this event, and the OUSCM faculty who provide financial contributions to it.

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Oral Presentations

Abstract #37: Medical Child Abuse Operating under Guise of Neurodevelopmental Disorder and Pica

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Introduction:

Medical child abuse (MCA), recognized as factitious disorder imposed on another by the DSM-5, describes situations in which a child is subjected to unnecessary and potentially harmful medical care due to a caregiver's intentional exaggeration, fabrication, or deliberate induction of illness.[1] The true frequency of MCA is difficult to establish, as existing estimates primarily focus on only severe cases. While child protection agency data suggest an annual incidence of 0.4–1.2 per 100,000 children, population studies looking at consecutive hospitalizations indicate that milder, unreported forms of MCA have rates as high as 530 per 100,000 children.[2-4]

Case Description

An 11-year-old female with a reported history of Autism Spectrum Disorder, pica, developmental delays, ADHD, OCD, and anxiety was admitted to a pediatric hospital for suspected intentional ingestion of batteries. She had at least 17 prior foreign body ingestions, predominantly involving high-risk substances such as magnets and batteries, requiring repeated endoscopic and surgical interventions. Admission frequency had increased, with six episodes in the prior year and six additional episodes over six months. Imaging on admission consistent with small disc batteries. Child psychiatry consultation was requested, as the mother attributed ingestion to pica. During previous admissions, psychiatric evaluation declined by mother, and behaviors were considered consistent with historical diagnoses. Evaluation revealed discrepancies in mother's history, inconsistent attempts to limit patient's access to batteries, and insistence on unsupported psychiatric diagnoses. The patient demonstrated age-appropriate social reciprocity, intact perspective-taking, and no overt cognitive or developmental delays. Nursing staff observed maternal coaching prior to interviews. The patient consistently withdrew or stated "it is pica" when questioned regarding ingestion. The selective ingestion of only highly dangerous items was inconsistent with pica or OCD. Concerns of mother's primary and secondary gain surfaced during follow-up visits and community collateral. The behaviors posed significant medical risk and were not accounted for by reported diagnoses or developmental delays. Placement away from the primary caregiver with close outpatient psychiatric follow-up was recommended, and DHS approved removal from the home. Subsequent chart review indicated no ingestions over seven months, psychological testing consistent solely with ADHD, routine school attendance, and a monitored reunification plan with the mother.

Discussion

This case highlights how caregiver-attributed neurodevelopmental diagnoses and rare psychiatric disorders can obscure pediatric medical neglect and delay appropriate intervention. The removal of the primary caregiver leading to complete resolution of ingestions and later psychological testing only recognizing ADHD supports the validity of this interpretation.

Abstract #79: Multifactorial Metabolic Acidosis and its Fatal Consequences

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George Hunihan, MD - University of Oklahoma School of Community Medicine, Department of Emergency Medicine

Introduction

Metabolic acidosis is a pathological process that increases hydrogen ions and lowers bicarbonate. This can be a result from increased acid production, bicarbonate loss, or decreased renal acid excretion and is further subclassified by anion gap (AG) status. This paper discusses a case of increased AG acidosis precipitated by diabetic ketoacidosis, starvation, alcohol abuse, and increased metabolic demand and hypoperfusion of organs leading to anaerobic metabolism secondary to cocaine abuse.

Case Description

A 45-year-old female with a history of Type 1 diabetes with previous hypoglycemic events, hypotension, and panic attacks presented with sudden-onset emesis, tachycardia, and tachypnea. One hour prior to presentation she was asymptomatic; she attributed these sudden symptoms to a panic attack. Initial vitals showed a glucose of 117 mg/dL and normotension. Workup was initiated and she was treated with Ativan, Zofran, and 1L normal saline (NS).
The first lab abnormality to result was a WBC of 39.62. She was promptly reassessed and found to be hypotensive, diaphoretic, and pale with visible neck swelling. While O_2 saturation was stable, she was tachypneic and visibly fatigued. A stat chemistry showed a pH of 6.93, pCO_2 15.1, and HCO_3 3.21, representing metabolic acidemia with incomplete respiratory compensation. Lactic acid later resulted at 18.7 mmol/L.
Due to her fast deterioration, intubation was prioritized over further testing and imaging. She was resuscitated prior to delayed sequence intubation with two liters of normal saline and norepinephrine. Post-intubation she became hypotensive again, requiring vasopressin. Subsequent CT imaging showed oropharyngeal swelling and lung consolidation suggesting tonsillitis and pneumonia, respectively. Broad-spectrum antibiotics and a bicarb drip was started and she was admitted to the ICU. Toxicology later showed acute intoxication of cocaine and alcohol. The patient was extubated on day two of her ICU stay, received six days of antibiotics, and was discharged on day seven with complete symptom resolution.

Discussion

Metabolic acidosis mortality ranges from 17% to 88%, with survival heavily dependent on the cause and promptness of correction. This patient's acidemia was multifactorial: cocaine's vasoconstrictive and anorexic properties along with increased metabolic demand, compounded by type 1 diabetes, alcohol intoxication, and infection. These factors led to anaerobic metabolism and the accumulation of lactic acid and ketones.
Winter's Formula assesses respiratory compensation capability; with a pCO_2 of 15.1, she was at her physiological limit. Fatigue would have soon ended this compensation, causing fatal acidemia. This case underscores the need for prompt identification and correction of complex acid-base disorders.

Poster Presentations

Emergency Medicine

Abstract #5: Cerebral Venous Sinus Thrombosis Presenting as Hemorrhagic Stroke in the Setting of Polycythemia Vera

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Introduction

Cerebral venous sinus thrombosis (CVST) is a rare cause of stroke that presents with altered mental status, headaches and intracranial hemorrhage. In some cases, hemorrhage results from increased intracranial pressures. Anticoagulation is a mainstay of treatment; failure to promptly identify CVST and intervene may result in poor neurological outcomes. After stabilizing these patients, it is crucial to identify the etiology of the venous thrombosis to prevent recurrence.

Case Description

A 48-year-old male presented to the emergency department with multiple syncopal episodes, headache, nausea, vomiting, confusion and transient right upper extremity weakness. Past medical history was significant for hypertension and alcohol use disorder. Initial imaging revealed a left parietal intraparenchymal hemorrhage without evidence of aneurysm or large vessel occlusion. Blood work was notable for elevated hemoglobin and hematocrit. Additionally, he was found to be in a hypertensive emergency with a systolic blood pressure of 204 mmHg. He was started on clevidipine and transferred to the ICU for further management.

After admission to the ICU, he developed right homonymous hemianopia. Further imaging revealed a left parietal and occipital venous infarct and thrombosis of the superior sagittal, left transverse, and sigmoid sinuses, confirming CVST. Although the left parietal intraparenchymal hemorrhage was still present, heparin infusion was initiated with close monitoring since the etiology of hemorrhage was the thrombosis. Patient's hypertensive emergency had resolved, and blood pressure remained stable.

Serial imaging indicated a stable hemorrhage and neurologic checks were unchanged; however, right homonymous hemianopia persisted. Lab studies demonstrated persistent erythrocytosis with normal erythropoietin and platelet counts. This raised suspicion for polycythemia vera and JAK2 mutation testing was ordered. Appropriate anticoagulation and frequent neurological exams and imaging continued. There was gradual resolution of neurological symptoms with stable hemorrhage and thrombosis.

Discussion

This case highlights the importance of recognizing cerebral venous sinus thrombosis as a distinct cause of hemorrhagic stroke. In this case, early initiation of anticoagulation along with frequent monitoring was critical to prevent worsening of thrombosis, cerebral edema and hemorrhage expansion. Consequences of inadequate treatment includes worsening neurological function and potentially fatal complications like herniation.

Additionally, this case shows the importance of appropriate workup of coagulopathy in CVST patients. For this patient, erythrocytosis was present with normal erythropoietin levels indicating polycythemia vera. At the time, the patient was unaware of this diagnosis. If left untreated, he is at increased risk for recurrent thrombosis. Therefore, prompt identification and management of CVST is crucial to prevent neurological complications.

Abstract #21: Saddle Pulmonary Embolism Presenting as Inferior ST-Elevation

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Introduction

Massive pulmonary embolism (PE) is a well-recognized cause of obstructive shock and cardiac arrest, accounting for an estimated 5–10% of sudden cardiac deaths. Although PE classically presents with sinus tachycardia or nonspecific EKG findings, acute right ventricular (RV) strain can produce ST-segment elevations that mimic acute myocardial infarction, particularly following return of spontaneous circulation (ROSC). These pseudo-STEMI patterns are thought to arise from RV ischemia due to acute pressure overload, hypoxia, and decreased coronary perfusion. Early differentiation between PE and true coronary occlusion is critical, as management pathways diverge significantly and delays in definitive therapy may worsen outcomes. Point-of-care ultrasound (POCUS) offers rapid bedside assessment of RV structure and function and may be particularly valuable when traditional diagnostic modalities are unavailable or misleading during resuscitation.

Case Description

A 61-year-old female presented to an urban tertiary emergency department with altered mental status and respiratory distress. She deteriorated rapidly and experienced two episodes of pulseless electrical activity arrest. During pulse checks, POCUS demonstrated marked RV dilation with interventricular septal bowing, consistent with acute RV pressure overload and obstructive shock. Following ROSC, a repeat EKG showed inferior ST-segment elevations, prompting activation of the cardiac catheterization laboratory for presumed STEMI. Coronary angiography revealed no obstructive coronary disease but instead identified a large saddle pulmonary embolus. The patient underwent catheter-directed thrombectomy with subsequent hemodynamic improvement. She was stabilized in the cardiovascular intensive care unit and ultimately survived to discharge to inpatient rehabilitation.

Discussion

This case highlights a critical diagnostic dilemma in cardiac arrest care: post-ROSC EKG findings suggestive of STEMI may obscure alternative life-threatening etiologies such as massive PE. While EKG abnormalities in PE are well described, ST-elevation remains an uncommon and potentially misleading manifestation. Early recognition of RV strain on POCUS was the most informative finding in this case and aligned with the patient's ultimate diagnosis. Although prior literature supports the use of POCUS in identifying reversible causes of cardiac arrest, reports specifically describing its role in differentiating STEMI from PE in post-arrest patients remain limited. This case adds to the growing body of evidence supporting POCUS as a critical adjunct in resuscitation, emphasizing its role in broadening the diagnostic framework and informing time-sensitive decision-making when conventional markers conflict.

Abstract #41: Slow and Subtle: A Case of Digoxin Toxicity in the Emergency Department

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George Hunihan, MD - University of Oklahoma School of Community Medicine, Department of Emergency Medicine
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Introduction

Digoxin was once commonly used to treat tachydysrhythmias (e.g., atrial fibrillation) in congestive heart failure (CHF). Its side effect profile and narrow therapeutic window have reduced its use. Consequently, Emergency Department (ED) presentations of digoxin toxicity have become rare. Common precipitants of digoxin toxicity include renal impairment, electrolyte disturbances, and drug interactions.
Digoxin toxicity (acute or chronic) causes many non-specific symptoms throughout the body, but the most feared, at least in the emergency setting, relate to cardiac conduction problems. The most common cardiac manifestation is frequent premature ventricular contractions (PVCs) on EKG, but it can present as almost any dysrhythmia. It can also cause gastrointestinal, neurologic, or ocular symptoms. Diagnosis requires high clinical suspicion, as even normal levels between 0.5 and 2 can be falsely normal, especially with chronic ingestion.

Case Description

An 88-year-old female with a history of CKD with a baseline creatinine of 1.1 and atrial fibrillation on digoxin presented to the ED for left-sided chest pain. She sustained a fall a week prior. She presented for continued left-sided chest pain. Vitals on arrival were a BP of 140/91, HR of 58, RR of 20, and oxygen saturation of 98% on room air. Pertinent examination findings included normal bilateral breath sounds, left chest wall tenderness without bruising or crepitus, and a bradycardic, irregularly irregular rhythm. Pertinent initial labs included a potassium of 4.5 and a creatinine of 1.61. In the ED she developed multiple episodes of bradycardia into the 30s-40s with borderline hypotension. Serum digoxin concentration was 1.3 (reference range 0.8-2.0ng/mL). CT imaging showed multiple subacute left rib fractures without pneumothorax. She was admitted for pain control and cardiology evaluation for bradycardia and possible digoxin toxicity. Cardiology recommended discontinuing digoxin, but it was not discontinued until admission day 2 after additional episodes of bradycardia. She had bradycardic episodes until day 5 of admission, ultimately being discharged to a skilled nursing facility on admission day 8.

Discussion

As digoxin utilization has become more uncommon, the number of toxicity cases that present to the ED has decreased. Diagnosis is challenging because of the variety of nonspecific symptoms that can be present upon ED presentation. This is complicated even more by the limited utility of the digoxin concentration, often making this a clinical diagnosis supported by dysrhythmia on EKG. This case highlights the importance of maintaining a high index of suspicion for digoxin toxicity in any patient who presents to the ED on digoxin.

Abstract #49: ECMO Cannulation in Emergency Department due to Recurrent Dysrhythmia

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Introduction
Veno-arterial Extracorporeal Membrane Oxygenation (VA ECMO) is a critical and life-saving technique employed for patients experiencing severe heart and lung failure. The procedure temporarily assumes the functions of the heart and lungs by redirecting blood through a machine that oxygenates it and removes carbon dioxide

Case Description
A 58-year-old patient, with past medical history of hypertension, hyperlipidemia, diabetes mellitus, and coronary artery disease, arrived at the emergency department via EMS. Patient presented with complaints of slurred speech, left-sided weakness, and left sided facial droop, with the last known well time being 45 minutes prior to arrival. The patient met the criteria for a stroke code, which was promptly activated. CT scans of the head and neck showed no obvious acute pathology.
Upon arrival in the treatment room, the patient reported experiencing chest pain followed immediately by loss of pulse and apnea, prompting immediate initiation of CPR. Emergency physicians were notified and present at the bedside immediately. Multiple rounds of CPR, defibrillation, and medications were administered per ACLS guidelines. Due to refractory ventricular dysrhythmias, a decision was made to contact the CVICU for potential ECMO canulation. The CVICU team successfully performed VA ECMO cannulation at bedside. Remarkably, patient was awake and oriented the following day and extubated. It was noted that the patient required an Impella device, as his ejection fraction was less than 20%. Severe stenosis of native epicardial arteries was identified as a likely cause of the repeated ventricular dysrhythmias. Unfortunately, the patient went into refractory ventricular tachycardia. The patient articulated that he would not want any further aggressive care including defibrillation. Patient was noted to have willingly withdrawn from ECMO and died peacefully with comfort measures.

Discussion
When activated within guidelines, ECMO can help serve as an adjunct for arresting patients that otherwise would not be able to hemodynamically support themselves. Although this case ends with withdrawal from care, the modality gave the patient his best chance for recovery. VA ECMO offers an opportunity to relieve stress on the body while the underlying pathology is assessed, and further treatment options are considered. In this situation, potential life-prolonging measures included the prospect of LVAD placement as a bridge to cardiac transplantation. Ultimately ECMO allowed the patient the opportunity to verbalize his wishes.

Abstract #55: Thrombolytic Therapy in Patients with Known DOAC Usage

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Introduction

Estimations indicate that one in six patients with an acute ischemic stroke who would otherwise be eligible for intravenous thrombolytic therapy (IVT) are receiving direct oral anticoagulant (DOAC) therapy. Current guidelines designate DOAC use as an absolute contraindication to IVT due to the perceived increased risk of intracranial hemorrhage (ICH), thereby excluding a substantial proportion of patients from the only established acute treatment for ischemic stroke. However, emerging evidence from observational studies and target trial analyses suggests that the risk of ICH in this population may be lower than previously assumed, potentially paving the way for expanded therapeutic options in this high-risk subgroup.

Case Description

A 56 year old male was transferred to the ER from an outlying facility for concerns of an acute ischemic stroke. He is on daily Aspirin and Pradaxa and confirmed taking his dose this morning. He developed acute onset left hemiparesis, left facial droop, and headache that began at roughly 1:45pm. CT scans at the outlying facility were negative for intracranial hemorrhage but showed a possible distal right M2 occlusion. After discussion with neurology, Tenecteplase was administered prior to transfer. On arrival, the patient had resolved facial droop and mostly improved left sided strength. Repeat CT scans did not show any ICH and now a patent right M2. Patient was admitted for further monitoring and with MRI negative for cerebral vascular accident.

Discussion

Current guidelines regard recent use of direct oral anticoagulants (DOACs) as an absolute contraindication to intravenous thrombolytic therapy (IVT) in patients with acute ischemic stroke. Exciting evidence, however, suggests that the risk of symptomatic intracranial hemorrhage (sICH) following IVT may be comparable between patients with and without recent DOAC exposure. In a large global multicenter retrospective cohort, no signal of increased harm in terms of sICH was observed among 832 DOAC-treated patients compared with 32,375 non-DOAC controls, with sICH defined as worsening of at least four points on the National Institutes of Health Stroke Scale with radiological evidence of intracranial hemorrhage. There was also no difference between factor Xa inhibitor or factor IIa inhibitor-treated patients regarding the risk of sICH. Although these observational findings provide encouraging insight for traditionally excluded patients, randomized controlled trials with expanded outcome variables are needed before definitive changes to clinical practice can be recommended. Nevertheless, this shift adds optimism regarding the potential expansion of reperfusion therapy to a broader group of patients with acute ischemic stroke.

Abstract #59: Post Bronchoscopy Aspiration Pneumonia in Patient Presenting with Altered Mental Status

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Introduction

Cavitary lesions are commonly encountered in thoracic imaging and encompass a broad differential, including infections, malignancies, and autoimmune conditions. A mainstay of the diagnosis of these cavitary lesions is bronchoscopy, which is a relatively safe procedure. However, it is not without complication. Infection (4.5%), bleeding (0.58-4%), and pneumothorax (0.53-4%) are three of the most common complications seen on bronchoscopy.

Case Description

A 55 year old female presents to the emergency department from PACU after outpatient bronchoscopy for altered mental status. The procedure was being performed to evaluate the etiology of the patient's RUL cavitary lesion. Two transbronchial brushings and 10 biopsies were obtained during the procedure. Some slow oozing of blood was identified and treated with 20 ml of topical epinephrine, with cessation in bleeding. No active bleeding was identified bilaterally after therapeutic aspiration of blood clots at the end of the procedure. She was transferred to PACU in stable condition. Shortly after arrival in PACU, the patient began thrashing in bed, altered, and not following commands. Her mental status remained unchanged, and a rapid response was called; the patient was taken down to ED for further evaluation. CT PE of patient's chest showing patchy consolidation and ground-glass opacities throughout both lungs, greatest in the right upper lobe, where there is some cavitary consolidation measuring up 3.1 x 3.7 cm. These findings are consistent with some atypical or fungal pneumonia. She was intubated for airway protection. She was treated with vancomycin, cefepime, and micafungin and admitted to the ICU. Patient became febrile to 104 and had seizure-like activity. LP performed and negative for meningitis. After extubation, the patient admits to drinking 3-4 beers per day prior to hospitalization. Patient's RUL cavitary consolidation felt to likely be secondary to aspiration pneumonia in the setting of alcohol withdrawal.

Discussion

Although bronchoscopy is regarded as a relatively safe diagnostic procedure, emergency physicians must recognize that complications remain clinically significant and can present acutely to the emergency department. While this patient presented almost immediately post-procedure, not all patients do. Infectious complications, delayed pneumothorax, and hemorrhage can occur days to weeks after bronchoscopy. It is imperative for the emergency physician to be aware of any recent procedures a patient has had and to consider possible post-procedure complications as the cause of the patient's presentation to the emergency department.

Abstract #74: Have Caution When Diagnosing Cannabinoid Hyperemesis: A Pelvic Inflammatory Disease Case Report

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Introduction

Pelvic Inflammatory Disease (PID) is a common and frequently missed condition among young women in the emergency department. PID may be caused by a variety of pathogens, including normal vaginal flora, and the complications can be both deadly and disabling. Women may develop infertility (10%–20%), chronic pelvic pain (40%), and 10% will experience an ectopic pregnancy after PID. The most common cause of death is rupture of a tubo-ovarian abscess, which carries a 5%–10% mortality risk and is associated with peritonitis and sepsis. Perihepatitis may present with pleuritic right upper quadrant pain and referred pain to the right shoulder. Because presentations vary widely and symptoms may be subtle, maintaining a low threshold for diagnosis is critical.

Case Description

A 27-year-old, ill-appearing female presented with chronic lower abdominal pain, persistent nausea, vomiting, and weight loss.
Vitals: HR 94, BP 116/74, RR 20, Temp 99.5°F, O_2 99%.
She reported diffuse abdominal tenderness, worse in the pelvis. She denied pregnancy, STI exposure, changes in bleeding or discharge, and any drug or THC use. She refused many medications, stating, “They don’t work.”
Two days prior, she was seen in the ED with a lactate of 4, a urine drug screen positive for THC, and a CT scan significant only for a small ovarian cyst. Documentation from that visit referenced her as “a frequent flyer.” She was discharged with antiemetics and educated on cannabis hyperemesis.
During the current visit, multiple antiemetics produced only minor improvement. She declined a repeat CT and remained ill-appearing, at times lying on the floor. PID was discussed, and she agreed to a pelvic exam. The exam revealed diffuse pelvic tenderness and mucopurulent discharge. Endocervical swabs later returned positive for chlamydia. She was admitted and recovered uneventfully.

Discussion

PID must be included in the differential when reproductive-age women present with abdominal pain. It is a clinical diagnosis, and no single finding is sensitive or specific. Many women have mild, non-specific, or atypical symptoms, and some are asymptomatic. This case also illustrates how cognitive biases, including anchoring on prior THC use or labeling as a ‘frequent flyer,’ can delay recognition of a serious condition. Clinicians should maintain a low threshold to diagnose and empirically treat STI-related PID, even if endocervical swabs are negative or a pelvic exam is not feasible. When in doubt, TREAT.

Abstract #80: Metastatic Endometrial Cancer Presenting as Progressive Weakness

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Eric Lee, MD - University of Oklahoma School of Community Medicine, Department of Emergency Medicine
Nathan Wilks, DO - University of Oklahoma School of Community Medicine, Department of Emergency Medicine

Introduction

Malignant spinal cord compression is an oncologic emergency most frequently associated with breast, lung, and prostate cancers. Cervical spinal cord compression due to metastatic endometrial adenocarcinoma occurs in less than 1% of endometrial cancer and can present with rapidly progressive neurologic deficits. Prompt recognition is essential to guide management decisions and goals-of-care discussions.

Case Description

A 70-year-old woman with past medical history of CKD3a, HFpEF, DMII and recently diagnosed stage IV endometrial adenocarcinoma with known pulmonary and right iliac bone metastases, presented to the emergency department from inpatient rehabilitation with one month of progressive bilateral upper and lower extremity weakness. She had not yet initiated chemotherapy or radiation therapy due to deconditioned status. On examination, the patient was alert and oriented with intact cranial nerves II–XII. Neurologic examination revealed significant bilateral upper extremity weakness (1/5 strength) involving the biceps and brachioradialis, supinators, pronator teres, and wrist extensors. She also demonstrated 1/5 strength in bilateral dorsiflexion and plantarflexion with signs of progression to more proximal muscles. Sensation to light touch, sharp, and temperature was decreased in distal extremities but intact. MRI of the cervical spine with and without contrast revealed a probable metastatic mass in the posterior cervical soft tissues at the C6 level causing severe spinal canal stenosis with mass effect on the spinal cord from C5–C7 and associated compressive myelopathy. Metastatic involvement of the C6 vertebral body was also noted. MRI of the brain, thoracic, and lumbar spine showed no evidence of metastatic disease. Pulmonary metastatic lesions were stable compared to prior imaging. Neurosurgery was consulted and did not recommend surgical intervention given the patient's comorbidities and overall poor prognosis. Following multidisciplinary discussions with oncology, radiation oncology, and palliative care, the patient elected comfort-focused care and declined further oncologic treatment. She was transitioned to inpatient hospice for symptom management and eventually discharged after 16 days to Clarehouse Hospice House.

Discussion

This case illustrates an uncommon presentation of metastatic endometrial adenocarcinoma resulting in cervical spinal cord compression with rapidly progressive quadriparesis. The neurologic examination localized to the C5–C7 levels, correlated with imaging findings. In patients with advanced malignancy, early identification of spinal cord compression is critical not only to assess candidacy for intervention but also to facilitate timely goals-of-care discussions and the role of palliative care.

Family & Community Medicine

Abstract #13: Delayed Diagnosis of Cervical Artery Dissection After Manual Strangulation

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Introduction

Cervical artery dissection (CAD) accounts for approximately 2% (27 of 1368) of all ischemic strokes and 25% (79 of 322) in adults younger than 50 years. Isolated head or neck pain without cerebral infarction occurs in 8-12% (20 of 245, 21 of 169) of cases and contributes to diagnostic delay. Among CAD patients with probable misdiagnosis, 89% (194 of 218) had an emergency department visit for headache. A negative non-contrast head CT does not exclude clinically significant cervical vascular injury. Strangulation is a high-risk cervical trauma in which cerebrovascular risk may persist despite unrevealing initial imaging.

Case Description

A 35-year-old woman presented to the ED with a severe headache. Her history was notable for intimate partner violence, including manual strangulation with loss of consciousness 11 months earlier. Non-contrast head CT showed no acute findings, and she was discharged. Two days after discharge, she underwent cervical manipulation. Six days after her initial ED visit, she returned with a worsening headache, left posterior neck pain, visual disturbance, and left facial sensory symptoms. CT angiography revealed near-occlusive dissection of the distal left intradural vertebral artery, bilateral cervical internal carotid saccular aneurysms, and a small distal anterior cerebral artery aneurysm. Brain MRI showed no infarction. Findings were consistent with vertebral artery dissection and multivessel cervical arteriopathy. Antithrombotic therapy was initiated, and serial imaging demonstrated stable vascular findings without infarction or hemorrhage.

Discussion

CT-negative headache after strangulation warrants consideration of cervical vascular injury. Strangulation represents a high-risk cervical trauma and merits escalation beyond CT-only triage when symptoms persist or evolve. While cervical manipulation has been reported in up to 17% (20 of 116) of CAD cases, biomechanical causality remains unproven; temporal proximity alone does not establish causality. Multivessel involvement occurs in approximately 15.2% (149 of 983) of CAD cases and is more frequently associated with aneurysmal disease, supporting interpretation as part of a broader cervical arteriopathy rather than a single inciting event. A history of strangulation should be treated as a cerebrovascular risk factor, incorporated into early risk stratification, and prompt timely vascular imaging when head or neck pain persists despite negative initial CT.

Abstract #24: Imaging-Surgical Discordance in Pectoralis Major Rupture

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Introduction

Pectoralis major rupture is an uncommon but functionally significant injury that most often occurs during high-load eccentric contraction and frequently requires operative management. Operative planning depends on accurate identification of the anatomic tear location and the quality of remaining tissue. Injuries may be labeled as “distal” on imaging even when abnormalities appear near the musculotendinous junction rather than at the humeral insertion, which can obscure whether the tendon has truly avulsed from the bone. Literature demonstrates variability between imaging, clinical assessment, and intraoperative findings, with inconsistent application of classification systems and limited correlation with surgically relevant anatomy. Current labels collapse intratendinous, junctional, and humeral avulsion injuries under a single “distal” category. As a result, location-based labels may not reliably predict whether a surgically usable tendon-bone interface is preserved.

Case Description

During a 500-lb bench press one-repetition maximum attempt, the patient developed acute right chest injury with immediate pain and deformity. Preoperative MRI demonstrated a complete tear of the sternal head of the pectoralis major, interpreted as a grade 3 myotendinous injury with a distal tendon stump measuring approximately 30-mm and no surrounding edema. These findings suggested a proximally located tear with preserved distal tendon. Surgical exploration instead revealed a complete intratendinous rupture, leaving only a small tendon remnant attached to muscle, while tendon normally anchored to the humerus remained intact. The lack of sufficient tendon stump precluded standard tendon-to-bone fixation using anchors, leaving suture-based reconstruction as the only viable repair option.

Discussion

Preoperative imaging labeled this injury as a distal rupture and implied preservation of a repairable tendon, yet operative findings demonstrated an intratendinous failure that eliminated a usable interface. The presence or absence of a surgically usable tendon-bone interface determines operative strategy, but imaging-defined distal location does not reliably indicate whether a surgically relevant tendon remains available for fixation. This distinction is consequential, because healing biology and fixation options differ between intratendinous, junctional, and humeral insertion injuries, and these patterns are not interchangeable at the time of repair. Grouping these injuries under a single “distal” label masks differences in available techniques and limits interpretation of reported outcomes. This limitation supports further study of classification systems that anchor imaging interpretation to surgically confirmed tissue integrity, enabling operative decision-making to be meaningfully aligned with reported outcomes.

Abstract #34: Limits of Limb Salvage in Severe Forearm Trauma

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Introduction

Both bone forearm fractures are a relatively common injury that can occur following high-energy trauma. However, progression to vascular compromise and extensive soft tissue damage leading to compartment syndrome is much rarer. Early recognition and timely intervention are crucial in prevention of devastating outcomes such as amputation. We present a case that depicts a severe open forearm fracture that resulted in amputation during attempted limb salvage due to considerable neurovascular destruction and poor functional improvement.

Case Description

A 20-year-old male presented to the emergency department from an outside facility following a dirt bike accident that occurred at approximately 5:15PM. He arrived at 7:00PM and was found to have a Grade IIIB open segmental fracture of the right radius and ulna, absent distal pulses, and strong suspicion for acute compartment syndrome. Initial evaluation by the orthopaedic team revealed extensive soft tissue damage and limb ischemia, prompting emergent operative intervention for decompression and attempted limb salvage. The patient was taken to the OR at 9:24PM, with surgical intervention beginning at approximately 10:00PM. During compartment release of the forearm, extensive necrotic tissue was encountered, particularly at the site of the distal radius fracture. The radial artery and radial nerve were found to be severed, with contusion of the median nerve. Additionally, multiple muscles were noted to be necrotic or ruptured, including the brachioradialis, flexor carpi ulnaris, and palmaris longus. Vascular surgery was subsequently consulted, as the forearm appeared nonviable and without perfusion despite decompression. Given the extent of injury and irreversible neurovascular damage, it was agreed that acute forearm amputation was the most appropriate course, as revascularization and reconstruction were unlikely to yield a functional limb. A right forearm amputation proximal to the level of the radial and ulnar fractures was performed, followed by extensive irrigation and debridement and placement of a wound vacuum-assisted closure device. The patient was subsequently admitted for continued management.

Discussion

This case highlights the unique nature of the injury given the combination of a Grade IIIB both bone forearm fracture with extensive neurovascular compromise and soft tissue destruction ultimately leading to forearm amputation. Despite timely operative intervention aimed at limb stabilization and salvage, intraoperative findings and multidisciplinary evaluation rendered this approach futile. Early, decisive amputation was undertaken to reduce morbidity, prevent systemic complications, and allow for optimal rehabilitation. This case underscores the importance of continuous assessment of limb viability at presentation and intraoperatively to guide appropriate and ethical clinical decision-making.

Abstract #47: Restrained Motor Vehicle Accident Leading to an Open Pilon Fracture

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Introduction

Open pilon fractures (OPF) are typically seen in younger patients following high velocity trauma and are characterized by severe soft tissue compromise, potential neurovascular injury, and intricate articular involvement posing an imminent threat to the limb. OPF are also known as tibial plafond fractures referring to their involvement of the distal tibial joint surface. Axial compression drives the talus into the tibial plafond creating comminuted intra-articular fracture patterns. Mechanisms include motor vehicle accidents (MVA) or falls from height. These injuries carry high complication rates including deep infection (27%) and nonunion (22%)[2]. Treatment emphasizes immediate assessment, soft tissue damage control with spanning external fixation, and soft tissue coverage before later initiating definitive fixation.

Case Description

A 56-year-old male presents to the ED following a head-on MVA where he sustained an open right ankle fracture. Initial evaluation showed an obvious deformity with a poke hole on the ankle's medial aspect for which he received closed reduction and immobilization of the fracture in the trauma bay. The following day, surgical soft tissue debridement preceded fracture compression and external fixator placement to facilitate soft tissue recovery and minimize complications prior to definitive fixation. Four days later, the external fixator was removed prior to definitive fixation via open reduction internal fixation (ORIF) was performed. Post-operatively, the patient was given antibiotics and thrombotic prophylaxis. Follow-up visits ensured no signs of infection, a well-healed operative site, and future physical and occupational therapy.

Discussion

An OPF of this nature necessitates a staged management strategy to minimize complication and maximize outcomes. Initial damage control coupled with antibiotic administration within one hour is critical to prevent infection. For soft tissue optimization: surgical debridement, irrigation, and external fixation should precede delayed definitive fixation. An open fracture demonstrates significantly higher infection rates with acute fixation (38%) than with delayed staged fixation (20%)[2]. OPFs are reliably classified using the Ruedi-Allgower system[3] based on displacement and comminution; this patient's injury was a Type I fracture. Definitive fixation is commonly done via ORIF as it provides superior articular reduction and anatomical restoration of the joint surface. Additionally, this procedure is associated with better functional outcomes and faster recovery compared to other methods[1]. OPF carry clinically significant roles due to their association with high complication rates, reflecting the severity of both osseous and soft tissue injury. This case highlights treatment challenges and multidisciplinary management indications to reduce morbidity of a traumatic injury.

Abstract #50: Dermatitis Herpetiformis with Atypical Presentation and Co-Occurring Sjögren's Syndrome: A Diagnostic Challenge

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Introduction

Dermatitis Herpetiformis (DH) is a rare cutaneous manifestation of Celiac Disease (CD) characterized by intensely pruritic vesicles and papules on the elbows, knees, and buttocks. Due to the severe pruritic nature of the disease, patients may also present with excoriations, erosions, and crusted papules, often obscuring primary lesions. Although an association between CD and Sjögren's Syndrome has been described, there is limited information regarding coexistence between Sjögren's Syndrome and DH. Furthermore, up to half of patients with Sjögren's Syndrome develop cutaneous manifestations such as pruritic rashes, which may lead to misattribution of dermatologic findings to Sjögren's Syndrome rather than DH. DH diagnosis is further complicated by nonclassical lesion distribution and the frequent absence of intact primary lesions.

Case Description

A 48-year-old female with a 3-year history of Sjögren's Syndrome on hydroxychloroquine and azathioprine presented to the allergy clinic for a 20-year history of recurrent papulovesicular rash present on the arms, legs, and scalp, which worsened with sun exposure. At office visit, presentation of the rash was primarily found on extensor surfaces with excoriations. The blistering lesions had spread from hands to the rest of her upper extremities and upper trunk. The patient described the vesicles as painful and itchy. She previously trialed steroid creams such as triamcinolone and clobetasol, with minimal relief of the pruritic lesions. Due to extensive excoriations at the time of presentation, selecting a site for biopsy was challenging. Ultimately, dermatology was consulted, and one single blistered lesion was identified and sampled using a 3 mm punch biopsy. The pathology report confirmed the diagnosis of DH.

During the follow-up visit, the patient reported intermittent gastrointestinal symptoms, including diarrhea and abdominal discomfort, occurring inconsistently with gluten intake. A G6PD and CD panel was ordered (IgA, tissue transglutaminase IgA, Endomysial IgA), which are pending collection. In the setting of concurrent hydroxychloroquine and azathioprine therapy for Sjögren's Syndrome and the associated risk of peripheral neuropathy, methemoglobinemia, and hemolytic anemia, the patient opted against dapsone therapy. Management was pursued with a strict gluten-free diet.

Discussion

This case highlights the diagnostic challenges that can occur due to limited literature establishing the co-occurrence of Sjögren's Syndrome and DH. It also underscores the difficulty in diagnosing dermatologic conditions when primary lesions are obscured by excoriation, complicating both clinical and histopathologic evaluation. Lastly, given the atypical distribution and migratory pattern of the patient's lesions, this case reinforces the importance of maintaining a high index of suspicion for DH.

Abstract #54: Tibial Plateau Fracture with Metaphyseal-Diaphyseal Dissociation Managed with Staged Fixation

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Introduction

Tibial plateau fractures are periarticular fractures of the proximal tibia accounting for 1-2% of all fractures. They present management difficulties due to complex fracture patterns and associated soft-tissue injuries. Plateau fractures most commonly involve the lateral plateau, but can involve the medial or both, bicondylar. Bicondylar fractures account for 10-30% of all plateau fractures and commonly occur after combined coronal plane forces and axial load. In younger individuals, these injuries typically result from high-energy trauma, while in elderly patients they result from low-energy mechanisms with poor bone quality.

Case Description

42-year-old male presented to the emergency department after colliding into a trailer while riding an electric mini-bike. Evaluation demonstrated a two centimeter longitudinal wound over the mid-anterior left shin with obvious deformity. CT imaging demonstrated a bicondylar tibial plateau fracture and metaphyseal-diaphyseal dissociation, consistent with a Schatzker 6 fracture pattern. He underwent closed reduction in the trauma bay and later external fixator placement to allow recovery of soft tissues. Five days later, he underwent open reduction internal fixation (ORIF), indicated for bicondylar plateau fractures. The postero-medial aspect of the plateau was addressed first. Pes anserine tendons were protected and soft tissue attachments were freed from the postero-medial tibia. A postero-medial plate was placed for posterior column stabilization. Lag screw placement compressed the sagittal split between the anterior epiphyseal segment and posterior diaphyseal fragment. Next, the antero-lateral plateau underwent fixation for articular surface stability. The IT band insertion was protected and submuscular conduit was cleared. Sub-meniscal arthrotomy provided direct visualization of the joint surface. A lateral proximal tibia plate was positioned along the lateral joint surface and compressed to the diaphysis. Below the joint surface, lag screws were placed to achieve compression across the joint surface.

Discussion

Tibial plateau fractures are complex injuries requiring careful operative planning. Fractures with minimal displacement or depression can undergo closed reduction. Complex fractures with features seen in this case, such as bicondylar and medial plateau involvement, require ORIF. If there is significant soft tissue swelling or injury then staged ORIF is necessitated. Clinical outcomes after ORIF primarily depend on restoration of joint stability, requiring precise surgical placement of hardware.

Internal Medicine

Abstract #6: Hypothalamic-Pituitary Neurosarcoidosis Flare Manifesting as Psychosis: Success with Hydrocortisone and IVIG

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Introduction

Sarcoidosis is a multisystemic, immune-mediated granulomatous disease that most commonly affects the lungs but can involve nearly any organ system, including the central nervous system (CNS). It exhibits multiple phenotypes which are highly dependent on involved structures. When CNS involvement is seen, it is termed neurosarcoidosis (NS), a rare but potentially severe manifestation. Hypothalamic-pituitary involvement occurs in 0.5% of sarcoidosis cases. Clinical presentations are highly variable and evidence-based treatment guidelines are limited.

Case Description

We report a case of a 46-year-old male with a known history of HPNS with secondary adrenal insufficiency, hypopituitarism, and diabetes insipidus who presented with acute onset headache, tactile and visual hallucinations, and altered mentation. Past medical history includes transient ischemic attack, depression, and type II diabetes mellitus secondary to long-term steroid use. His symptoms developed three weeks after his most recent intravenous immunoglobulin (IVIG) infusion. Brain imaging revealed no acute pathology, and inflammatory markers were within normal limits. He was treated with his baseline hydrocortisone regimen and received two additional doses of IVIG. Symptoms resolved within 48 hours, and he was discharged in stable condition.

Discussion

This case highlights a flare of HPNS presenting with psychotic features in a previously stable patient. It underscores the psychiatric presentation of neurosarcoidosis and the role of immunosuppressive therapies including corticosteroids and IVIG in management. While corticosteroids are the current first-line treatment for NS due to its inflammatory nature, there is growing evidence to support the use of IVIG as well as other immunomodulating agents. Given the absence of randomized controlled trials for NS treatment, further investigation into standardized therapeutic approaches is warranted.

Abstract #19: When CIDP isn't idiopathic: A case of MGUS- associated demyelinating neuropathy

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Introduction

Chronic inflammatory demyelinating polyneuropathy (CIDP) is largely considered idiopathic; however, secondary causes must be excluded. Monoclonal gammopathy of undetermined significance (MGUS) is an important and frequently overlooked cause of demyelinating neuropathy and may initially present as CIDP. We present a case in which an initial diagnosis of CIDP prompted further evaluation that revealed an underlying MGUS, emphasizing the need for thorough diagnostic assessment.

Case Description

An 82-year-old man with a history of hypertension, hyperlipidemia, osteoarthritis, and prior L2–S1 spinal fusion presented with several years of progressive numbness, tingling, and pain in a stocking distribution involving both feet, gradually ascending to the distal lower extremities. He later developed mild hypersensitivity in the fingertips bilaterally and progressive gait instability with sensory ataxia. He denied diabetes, alcohol use, or family history of neuropathy.

Neurologic examination revealed symmetric stocking distribution sensory loss below the knees, weakness of intrinsic foot muscles with subtle ankle dorsiflexion weakness, and impaired tandem gait. Romberg testing was positive. Upper extremity strength, sensation, and reflexes were normal. Electromyography and nerve conduction studies demonstrated a widespread demyelinating motor and sensory neuropathy with conduction block and temporal dispersion, consistent with an acquired demyelinating process and supporting a diagnosis of CIDP. Given concern for a potentially treatable underlying etiology, further evaluation was pursued.

Laboratory studies showed normal vitamin B12, folate, copper, and thyroid-stimulating hormone levels, with negative Lyme and syphilis serologies. Myelin-associated glycoprotein (MAG) IgM antibody was markedly elevated at 19,995 BTU. Serum protein electrophoresis did not reveal a clear monoclonal spike; however, immunofixation identified a trace IgM kappa paraprotein, consistent with MGUS.

The patient was initiated on intravenous immunoglobulin (500 mg/kg/day for four days, repeated monthly for three cycles) in combination with mycophenolate mofetil.

Discussion

This case highlights MGUS as an important secondary cause of CIDP-like demyelinating neuropathy, particularly in older adults. Identification of a monoclonal gammopathy, especially in the presence of elevated anti-MAG antibodies, has diagnostic and therapeutic implications, as treatment response and disease course may differ from idiopathic CIDP. Comprehensive evaluation for paraproteinemia should be considered in patients presenting with acquired demyelinating neuropathy.

Abstract #22: Taking the Edge Off: Nebivolol for Sympathetic-Driven Hypertension with Comorbid Anxiety

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Introduction

Hypertension and anxiety disorders can often coexist, likely through a shared heightened sympathetic nervous system activity. Traditionally, Beta-blockers have been used for cardiovascular indications and situational anxiety, yet their effectiveness can vary. Nebivolol is a β1-selective blocker with additional nitric oxide-related vasodilatory properties; it may offer dual benefits for attenuating sympathetic drive and blood pressure control. This case highlights nebivolol's potential benefits in a patient with hypertension and general anxiety disorder. This presentation highlights Nebivolol's role in symptomatic and anxiolytic relief following transition from nonselective beta-blockage to nebivolol.

Case Description

A man in his 40s with a history of generalized anxiety disorder and hypertension presented to the clinic for severe episodic anxiety and fluctuating blood pressure readings. He reported near-normal readings upon awakening which increased throughout the day in association with anxiety symptoms, reaching 140s/90s maximum. He reported panic features of palpitations, chest tightness, and dyspnea. Workup for secondary causes was largely unremarkable with CBC, CMP, TSH, and office EKG all within normal limits. Metanephrines were ordered to rule out pheochromocytoma and remain pending, but clinical suspicion remains low.

Initial management included intermittent low-dose alprazolam for situational triggers, citalopram, hydroxyzine 25 MG PRN, and propranolol 20 MG. Despite dose escalation of propranolol to 40MG nightly with additional daytime dosage and adequate duration of therapy for citalopram, the patient reported limited symptomatic benefit and blood pressure remained elevated.

Following a new diagnosis of OSA—with minimal HTN improvement on CPAP—and a severe episode of chest pain attributed to panic, propranolol was discontinued, and nebivolol 5MG QD was initiated. Patient reported improvement in chest tightness and palpitations. Patient described a reduction in their cyclical escalation of cardiovascular sensations and anxiety awareness. Home blood pressures improved to 130s/80s, and nebivolol was increased to 5MG BID, with further improvement in blood pressure readings and continued anxiety reduction. Patient documented minimal anxiety, reduced racing heart sensations, and improved daily functioning.

Discussion

This case highlights the benefits of the adjunct use of cardio-selective beta-blockers as symptom-targeted therapy; it is not a recommendation for the replacement of other first-line therapies. We hypothesize that its benefit came from breaking the reinforced panic and subsequent blood pressure elevation through a reduction in adrenergic activation and in heart rate. In comparison to nonselective beta-blockers, such as propranolol, nebivolol's β1selecivity may improve tolerability by selectively modulating sympathetic tone. Ultimately, recognizing the pathophysiology of this patient's underlying conditions demonstrates the importance of individualized patient management.

Abstract #27: Marching to Its Own Beat - a Mysterious Case of Heart Block

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Introduction

Coxsackie virus is infamous for causing hand, foot, and mouth disease, but can also lead to more severe manifestations, including myocarditis, cardiomyopathy, and heart block. However, it is exceedingly rare for coxsackie virus to cause cardiovascular consequences in the absence of myocarditis. We present a young patient with a complete heart block secondary to coxsackie virus infection despite a normal cardiac MRI.

Case Description

A 31-year-old woman presented with a week of multiple syncopal episodes and shortness of breath. Past medical history included bulimia nervosa in remission and a recent mild upper respiratory illness. Initial labs including CBC, CMP, troponin, CK, and inflammatory markers were unremarkable. ECG revealed sinus rhythm with high-grade AV block. Extensive workup, including Lyme serologies and autoimmune testing for sarcoidosis, were negative. Infectious workup was also negative except for a positive coxsackie virus PCR. Cardiac MRI to evaluate for myocarditis was normal. Her complete heart block was therefore attributed to coxsackie virus in the absence of clinical or imaging evidence of myocarditis. She received supportive care and ultimately underwent placement of a leadless pacemaker. She was discharged in stable condition after ten days.

Discussion

This case presents a rare but potentially devastating expression of coxsackie virus. While heart block has been reported in coxsackie infections, it is almost always associated with myocardial inflammation, which was absent or potentially resolved in this case. Because coxsackie virus is so common, affecting millions of people across the globe yearly, knowledge about and suspicion for such grave manifestations is vital for timely diagnosis and treatment of these patients.

Abstract #35: From Valve to Bone: Musculoskeletal Sequelae of Persistent Endocarditis

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Introduction

Non-union of a fracture complicated by osteomyelitis is typically due to direct contamination or compromise of local tissue. Hematogenous seeding of bone from endocarditis is a much rarer phenomenon, but represents an important diagnostic consideration. Particularly, patients with multivalve infective endocarditis (IVE) are at increased risk of septic embolization and bacteremia which creates a favorable environment for secondary musculoskeletal infection. We present a case of distal fibular non-union in the setting of triple valve endocarditis, highlighting the interplay between uncontrolled endovascular infection and orthopedic pathology.

Case Description

A 40-year-old man presented to ED for right ankle pain and swelling, dyspnea, and systemic symptoms. Previous medical history is significant for intravenous (IV) drug use, opioid use disorder on methadone, untreated hepatitis C, prior tricuspid valve replacement, and hospitalization three months prior for a snake bite complicated by endocarditis. On admission, the patient was septic with leukocytosis and elevated procalcitonin of 4.4 ng/mL. Blood cultures were positive for gram positive organisms. CT of the right tibia and fibula showed a non-union fracture of the distal fibular diaphysis with surrounding lucency and rim-enhancing fluid collection consistent with osteomyelitis and possible abscess. CT angiogram revealed multifocal nodular infiltrates concerning septic emboli. Inflammatory markers were elevated with ESR of 57 mm/hr and CRP of 20.25 mg/L. The patient was started on IV vancomycin and broad-spectrum antibiotics. The patient was admitted and podiatry consulted. The patient was later taken to surgery for irrigation and debridement of the right ankle. Operative debridement was performed with sharp excision of necrotic tissue and periosteum and open bone biopsy of the distal fibula. Intraoperative findings included necrotic bone and pathologic fracture. Given persistent bacteremia and pulmonic septic emboli, transesophageal echocardiogram revealed large vegetative growths on the mitral, tricuspid, and aortic valves with possible aortic root abscess. Cardiothoracic surgery deemed the patient a poor surgical candidate. Despite persistent antibiotic therapy, the patient's condition declined and he was transitioned to comfort-focused care.

Discussion

This case demonstrates hematogenous seeding of distal fibular fracture non-union in persistent bacteremia from triple-valve endocarditis. Chronic infection likely impaired fracture healing, promoted abscess formation, and resulted in pathologic fracture and ulceration. Recognition of an endovascular source is critical in patients presenting with unexplained osteomyelitis or fracture non-union, particularly in those with risk factors for infective endocarditis. Early multidisciplinary collaboration is essential to address orthopedic complications and underlying endocardial infection. Delayed recognition can lead to irreversible systemic and structural complications.

Abstract #39: Hypercalcemic Pancreatitis in the Setting of Hodgkin Lymphoma

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Introduction

Hodgkin lymphoma (HL) is defined as a monoclonal lymphoid neoplasm divided into two distinguishable categories: classical Hodgkin lymphoma (cHL) and nodular lymphocyte-predominant Hodgkin lymphoma (NLP-HL). Classical HL most commonly presents with asymptomatic lymphadenopathy, mass on chest x-ray, or B-symptoms (fever, night sweats and unintended weight loss). B-symptoms are associated with a poorer prognosis and are present in up to 50% of patients with advanced disease. Symptoms generally progress slowly, with patients often experiencing symptoms for weeks to months before they are evaluated. Definitive diagnosis is through biopsy of a lymph node or affected organ. Pancreatitis due to hypercalcemia is rare, occurring in less than 10% of cases. Hypercalcemia related to Hodgkin lymphoma is also uncommon and occurs in less than 5% of cases. We present a rare case of acute pancreatitis secondary to hypercalcemia in the setting of HL.

Case Description

An 18-year-old female presented for flu-like symptoms and a 3.4 cm pancreatic mass, pulmonary nodules and hepatic lesions visualized on outlying facility CT imaging. History and labs revealed an unintentional weight-loss of 20 pounds in 2 weeks, non-tender and immobile left axillary lymphadenopathy, calcium of 11.8, lipase greater than 1000, hyperuricemia, serum creatinine of 14.3, 1,25-dihydroxyvitamin D of 84.3, and an elevated beta-hydroxybutyrate secondary to starvation ketosis. Repeat CT and MRI imaging only revealed significant pancreatic inflammation. She was admitted for the management of pancreatitis, acute kidney injury, and hypercalcemia. A left axillary lymph node biopsy was performed, which revealed nodular sclerosis and large, atypical cells with prominent bilobed nucleated cells. This result was confirmed through immunohistochemistry staining. These findings were consistent with Ann Arbor Stage IV Hodgkin Lymphoma. While in the hospital, she started chemotherapy with nivolumab, doxorubicin, vinblastine, and dacarbazine. At discharge, outpatient treatment was arranged.

Discussion

Among patients with HL, pancreatitis is an uncommon side effect. Mechanisms that can cause pancreatitis include pancreatic metastasis, bone metastasis, PTHrP production, or calcitriol overproduction as seen in our case. In HL, cells produce an excess of 1,25 hydroxyvitamin D by increasing the production of 1 alpha-hydroxylase through upregulation of its enzyme CYP27b1. The combination of HL and acute pancreatitis may cause significant harm, and prompt diagnosis can allow for earlier intervention and treatment. Aggressive intravenous hydration, glucocorticoids, calcitonin and bisphosphonates are essential to decrease serum calcium and treat acute pancreatitis. After their condition has improved, definitive treatment for Hodgkin lymphoma must be started to prevent recurrence.

Abstract #40: Colonic Ischemia as a Manifestation of Stem Cell Transplantation-Associated Thrombotic Microangiopathy

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Introduction

Hematopoietic stem cell transplantation-associated thrombotic microangiopathy (HSCT-TMA) is a severe complication of hematopoietic stem cell transplant (HSCT) associated with a mortality rate of 50 to 60%. The reported incidence of HSCT-TMA varies widely from 2 to 39% among patients who have previously completed stem cell transplantation. Due to its rarity, it is often missed as a diagnosis or misdiagnosed. This is a case that exemplifies how this process can present and provides helpful diagnostic tools.

Case Description

A 58-year-old male presented for left lower quadrant pain and hematochezia. Past medical history includes multiple myeloma with two previous autologous stem cell transplants, one seven years ago and another eight months prior to presentation. At presentation, hemoglobin was 7.1; platelets were 51,000. On CT abdomen and pelvis, he was noted to have diffuse mural thickening of descending colon, sigmoid colon, and rectum. LDH was found to be low, and no schistocytes were found on peripheral smear. Flexible sigmoidoscopy was completed, and the patient was found to have diffuse severe inflammation of the rectum and sigmoid colon. Biopsy revealed HSCT-TMA. IV steroids and infliximab were initiated, and his home lenalidomide was held. His condition improved, and he was transitioned to oral steroids with a taper regimen at discharge. Follow up appointments were scheduled for patient to receive remaining loading doses of infliximab.

Discussion

Among patients with a history of HSCT, HSCT-TMA should be considered as a potential diagnosis when ≥ 3 of the Jodele criteria are present. In the absence of laboratory evidence confirming the diagnosis, biopsy of the impacted organ systems can provide definitive diagnosis. Among patients with HSCT-TMA, a minority will experience gastrointestinal symptoms. These symptoms typically manifest as abdominal pain, diarrhea, nausea, vomiting, and bleeding, but severe cases can lead to ischemic colitis which should be recognized promptly to improve clinical outcomes. In the absence of universally accepted criteria, the Jodele criteria is often used to diagnose HSCT-TMA. Diagnosis requires ≥ 4 of the following within 14 days: anemia, thrombocytopenia, elevated LDH, elevated spot random urine protein/creatinine ratio, hypertension, presence of schistocytes on peripheral smear and evidence of compliment activation. After a diagnosis is made, treatment is largely supportive and focuses on managing hypertension and initiating dialysis if necessary. Discontinuation of calcineurin inhibitors (tacrolimus or cyclosporine) and sirolimus, and the addition of rituximab, eculizumab or defibrotide can be considered when general supportive measures fail to improve symptoms.

Abstract #44: Waldenström Macroglobulinemia-induced Type I Cryoglobulinemia Presenting as Spurious Thrombocytosis and Limb Ischemia

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Introduction

Cryoglobulinemia is a rare condition characterized by precipitating immunoglobulins causing temperature-dependent hyperviscosity. Type I Cryoglobulinemia, which is due to a monoclonal gammopathy such as Waldenström's Macroglobulinemia, may result in severe ischemic complications and false hematologic lab results. We introduce a case of Waldenström-associated Type I Cryoglobulinemia presenting as extreme spurious thrombocytosis.

Case Description

An 81-year-old male with coronary artery disease, previous STEMI with PCI, and hypertension presented with three days of progressive left lower extremity pain and discoloration. Computed tomographic angiography showed bilateral moderate stenosis of the superficial femoral artery with the obstruction of the left popliteal and posterior tibial arteries. Vascular surgery was consulted for evaluation of acute limb ischemia.

The initial platelet count was 403k/uL on admission. A repeat evaluation four hours later was reported at 2.4 million/uL with a subsequent value of 2.1 million/uL. Hematology was consulted for severe thrombocytosis. On the peripheral blood smear, red blood cell agglutination was observed, and amorphous basophilic extracellular material was seen, however there was a notable absence of thrombocytosis. Multiple repeat platelet counts were in the normal range. This raised the possibility of cryoglobulin precipitation causing spurious thrombocytosis as a result of cryogelation. Additional testing revealed significantly low complement (C3 37 mg/dL, C4 2 mg/dL), elevated serum viscosity (2.8), and an IgM monoclonal spike of 1.5 g/dL on SPEP.

Plasma exchange was urgently initiated, however circuit clotting presumably, due to cryoglobulin precipitation, caused frequent interruption of the procedure. The patient received intravenous high-dose methylprednisolone and rituximab. While vascular perfusion improved with these interventions, the continued irreversible critical limb ischemia ultimately resulted in left below-knee amputation. Cryoglobulin tests subsequently returned positive at 17%, consistent with a diagnosis of Type I Cryoglobulinemia. Bone marrow biopsy ultimately demonstrated lymphoplasmacytic lymphoma with MYD88 mutation, confirming the diagnosis of Type I Cryoglobulinemia due to Waldenström's. Acalabrutinib was initiated.

Discussion

This case demonstrates one of the rare yet serious manifestations of the Waldenström-associated type I cryoglobulinemia presenting as spurious thrombocytosis. Cryoglobulin disease may artificially increase reported platelet counts. Apheresis can be complicated by the precipitation of cryoglobulin, slowing down the definitive therapy. It is imperative that spurious thrombocytosis be confirmed on peripheral blood smear. Additionally, warming protocols, plasma exchange, and lymphoma- directed therapy must be instituted promptly. This case highlights the significance of timely identification of spurious laboratory artifacts, which may delay correct diagnosis and emergent treatment.

Abstract #46: Name That Rash: A Case of Eczema Herpeticum

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Introduction

Eczema Herpeticum is an invasive cutaneous infection of eczematous areas caused by herpes simplex-1, usually presenting as vesicles and erosions with hemorrhagic crust. The condition is caused by infection of compromised skin barriers in atopic dermatitis or reactivation of HSV that then spreads to normal skin. It can be disfiguring and potentially fatal due to systemic viremia and superimposed bacterial or fungal infection if not properly identified and treated.

Case Description

The patient is an 18-year-old female with past medical history of asthma, allergies, and eczema who presented due to a rash on her chest and arms that began to spread up to her face and down her torso. She reported that the rash started the day after she had been swimming in a pool one week prior to presentation. She noted she has had eczema on her abdomen, back, and arms before, never her lips or breasts. On initial presentation, the patient was afebrile, normotensive, and tachycardic. On physical exam, the patient had a rash that was heterogeneous in appearance. In areas, the rash appeared as vesicles and in some areas as pustules, and there were some erosions all on an erythematous base, mainly around the patient's breast and torso, on the arms, on the face, as well as two lesions on the tongue. The patient was seen by dermatology virtually in the ER, who recommended a prednisone taper, triamcinolone ointment, hydrocortisone for the lips, and doxycycline for an atopic dermatitis flare, but the rash continued to spread. Due to the worsening rash and multiple stages with vesicles and crusted erosions, HSV was suspected. Acyclovir 10 mg/kg q8hr was started, and the patient's rash improved significantly with treatment. She was discharged with a course of oral valacyclovir. Later, HSV IgG and an HSV PCR swab of one of the lesions returned positive.

Discussion

The most important diagnostic tool used to identify eczema herpeticum is physical examination. HSV IgG is not always indicative of active infection, IgM is not reliable, and PCR takes time to result. The main clues were the simultaneous stages of the rash with vesicles and "punched out" erosions. Additionally, the rash started on her existing eczematous patches and spread to include the mucosal surfaces of the lips and tongue. Early identification and initiating antiviral therapy are vital for reducing mortality. Moderate/severe cases require hospitalization for IV acyclovir; mild cases can be managed with PO valacyclovir.

Abstract #48: Pondering Upon Protozoa: A Case of Amebic Encephalitis

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Introduction

Amoebic encephalitis can be classified as primary amebic meningoencephalitis (PAM) caused by Naegleria fowleri or granulomatous amebic encephalitis (GAE) caused by Acanthamoeba species. Naegleria is found in fresh water, while Acanthamoeba is found in soil. These infections have a mortality rate of >90% despite antimicrobial treatment.

Case Description

The patient is a 21-year-old male with no past medical history who presented with headache, neck pain, fever, chills, photophobia, nausea, vomiting, and confusion. He reported having taken a foot-first dive into stagnant water in a river, with water forced up his nose. On physical exam, the patient had painful eye movement and nuchal rigidity. CT head was normal, the only abnormal lab value was a WBC count of 15.7k. Lumbar puncture was done due to the worsening headache and meningeal signs, which showed the presence of many WBCs, primarily lymphocytes. Due to the history of forceful river water up the nose, there was immediate concern for primary amebic encephalitis. Liposomal amphotericin B was initiated immediately along with azithromycin, fluconazole, and rifampin, and miltefosine. Upon examination of the CSF under a microscope, organisms “consistent with ameba” were seen. The case was discussed with the state department of health and the CDC. The samples were sent to the CDC for amebic PCR testing. Photos and videos of organisms were also forwarded for telediagnosis. The CDC report for free-living ameba PCR for Naegleria fowleri and Acanthamoeba species was negative. The CDC telediagnosis team identified the organism seen on the patient’s CSF as “Protozoa”, a class that includes amebic organisms. The patient’s symptoms improved significantly with treatment. Despite the negative PCR testing, the visualized organisms and the patient’s history were most consistent Naegleria fowleri infection so treatment was continued. A 14-day course of liposomal amphotericin B and a 28-day course of azithromycin, fluconazole, rifampin, and miltefosine were completed.

Discussion

The two most likely clinical entities this condition could be are primary amebic meningoencephalitis caused by Naegleria fowleri or granulomatous amebic encephalitis caused by Acanthamoeba species, given the presence of protozoa in the CSF. The shape of the organism found on the CSF and the clinical history of exposure to freshwater up the nose are more consistent with Naegleria fowleri. Considering that PAM is an almost universally fatal condition, even with proper antimicrobial treatment, its prompt recognition and immediate initiation of treatment are critical for having even a small chance of recovery.

Abstract #61: Seronegative Autoimmune Hepatitis Presenting With Cholestatic Features and Extensive Lymphadenopathy

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Introduction

Autoimmune hepatitis (AIH) is a chronic inflammatory liver disease characterized by immune mediated hepatocellular injury. Diagnosis is often supported by positive autoimmune serologies; however, seronegative presentations can occur and pose a diagnostic challenge, particularly when accompanied by atypical imaging findings such as lymphadenopathy or cholestatic features.

Case Description

A 23 year old male presented with one month of pruritus, mild jaundice, and intermittent postprandial abdominal pain. Laboratory evaluation revealed a mixed hepatocellular and cholestatic pattern with markedly elevated AST up to 589 U/L, ALT up to 768 U/L, alkaline phosphatase up to 297 U/L, and total bilirubin up to 3.3 mg/dL. Coagulation studies showed a mildly elevated PTT with preserved INR and albumin. The patient also had an elevated protein gap and hypergammaglobulinemia with elevated IgG and IgM levels.

Extensive infectious, metabolic, and genetic workup including viral hepatitis panels, EBV and CMV testing, iron studies, and Wilson disease evaluation was unrevealing. Autoimmune serologies were notable for negative ANA, anti-smooth muscle antibody, and antimitochondrial antibody, with mildly elevated F-actin IgG and positive PR3 ANCA. Imaging demonstrated splenomegaly and extensive mesenteric in the right lower quadrant and porta hepatis lymphadenopathy without biliary obstruction. MRCP showed no intrahepatic or extrahepatic ductal dilation.

Given persistent transaminase elevation and concern for inflammatory or infiltrative disease, a core liver biopsy was performed. Histopathology demonstrated marked interface hepatitis with confluent periportal necrosis, prominent plasma cell infiltrates, and some periductal fibrosis without bile duct loss. These findings are compatible with active chronic hepatitis consistent with AIH Grade 3 Stage 2. Although focal periductal fibrosis raised consideration for primary sclerosing cholangitis, imaging correlation did not support this diagnosis.

The patient was initiated on high dose prednisone with plans for close outpatient gastroenterology and hepatology follow up, including endoscopic evaluation and further assessment of lymphadenopathy.

Discussion

This case highlights the importance of liver biopsy in diagnosing seronegative autoimmune hepatitis, particularly in patients with atypical cholestatic features and extensive lymphadenopathy. Early recognition and treatment are essential to prevent disease progression despite negative conventional autoimmune markers.

Abstract #66: Ras-Associated Autoimmune Leukoproliferative Disorder Mimicking Lupus and CMML

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Introduction

Ras-associated autoimmune leukoproliferative disorder (RALD) is a rare clonal hematologic condition characterized by autoimmune manifestations and myeloproliferative features driven by activating RAS pathway mutations. RALD is most commonly described in pediatric populations and can clinically resemble systemic lupus erythematosus (SLE) or myeloid neoplasms such as juvenile or chronic myelomonocytic leukemia (JMML/CMML). Adult presentations are uncommon and may pose significant diagnostic challenges. We describe an adult patient whose lupus-like presentation and hematopathologic findings initially suggested SLE and CMML, but whose clinical course and molecular features supported a diagnosis of RALD.

Case Description

A 30-year-old woman presented with Raynaud phenomenon, oral ulcers, rashes, lymphadenopathy, serositis, inflammatory arthritis, and a positive antinuclear antibody, leading to an initial diagnosis of SLE. Over time, she developed leukocytosis, splenomegaly, and thrombocytopenia. Skin biopsy demonstrated a cell-poor interface dermatitis. Due to progressive splenomegaly, splenectomy was performed and revealed effacement of red and white pulp by a myelomonocytic infiltrate. Bone marrow biopsy showed hypercellularity with myeloid and erythroid dysplasia, and peripheral blood smear demonstrated dysplastic granulocytes and monocytes. Next-generation sequencing of peripheral blood identified a pathogenic KRAS mutation (p.G12A) with a variant allele frequency of 44.4%, without additional somatic mutations. Although the patient met diagnostic criteria for CMML, her age, indolent clinical course, and isolated KRAS mutation raised concern for RALD rather than a primary myeloid malignancy.

Discussion

RALD remains poorly characterized in adults and is not formally classified within current WHO or ICC frameworks. This case illustrates how RALD may closely mimic both autoimmune disease and CMML, creating risk for misdiagnosis and inappropriate treatment. Unlike previously reported RALD cases that demonstrate monocytic infiltrates in the skin, this patient's dermatopathology was more consistent with cutaneous lupus, further complicating diagnosis. Recognition of RALD is essential, as misclassification as CMML may lead to unnecessary cytotoxic therapy, while treatment directed solely at autoimmune disease may be ineffective. Increased awareness of adult-onset RALD and integration of molecular testing are critical for accurate diagnosis and appropriate management.

Abstract #77: Gastrointestinal Involvement of Disseminated Histoplasmosis

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Introduction

Duodenal ulcers are most commonly associated with *Helicobacter pylori* infection or the use of nonsteroidal anti-inflammatory drugs. However, in disseminated histoplasmosis, gastrointestinal involvement occurs in up to 70% of cases, with ulcerative lesions reported in approximately 40–50%. Recognition of gastrointestinal histoplasmosis is critical, as it frequently mimics inflammatory bowel disease, peptic ulcer disease, or gastrointestinal malignancy, leading to diagnostic delay and inappropriate therapy. This case illustrates an atypical presentation of disseminated histoplasmosis manifesting as a refractory duodenal ulcer. Highlighting the diagnostic challenges posed by overlap with inflammatory bowel disease and emphasizes he importance of targeted antifungal therapy.

Case Description

A 70-year-old male presented for melena and fatigue. His medical history notable for ileocolonic Crohn's disease s/p total proctocolectomy with end ileostomy on adalimumab, and recurrent admissions up to five times in a six-month period for gastrointestinal bleeding. Five months prior to presentation to our facility patient had an EGD that showed clotted blood with an estimated blood loss approximately 3 liters. Gastric mucosal examination at that time was impaired due to large amount of blood, hemostasis was pursued and no biopsies were obtained. One month later, repeat EGD revealed a Forrest class III duodenal ulcer, with biopsies interpreted as consistent with Crohn's disease involvement. A subsequent EGD three months later showed a persistent duodenal sweep ulcer requiring thermal ablation. Upon presentation to our facility hemoglobin was initially 7.9 g/dL and decreased to 6.4 g/dL . CT abdomen pelvis with contrast showing gastric wall thickening. Patient underwent another EGD with histopathological findings consistent with histoplasmosis. The patient was discharged with close infectious disease follow-up and continued adalimumab. Unfortunately, the patient presented with recurrent bleed and succumbed to hemorrhagic shock.

Discussion

Duodenal ulcers from histoplasmosis are exceedingly rare, given that disseminated histoplasmosis occurs in 1/100,00 cases and is even less common in immunocompetent individuals. The rarity is compounded by the fact that even when GI histoplasmosis occurs, the duodenum is not the most commonly affected site. Even in cases of disseminated histoplasmosis, GI involvement commonly affects the ileum and colon. This diagnosis should be considered particularly in immunocompromised patients, especially those with AIDS or undergoing anti-TNF therapy in endemic areas. Progressive disseminated histoplasmosis is lethal but with the gold standard of amphotericin B with transition to itraconazole treatment, 80% of cases have good prognosis. However inappropriate therapies including immunosuppressive agents worsen the fungal infection.

Abstract #78: A Rare Case of Gastric Erosion by Diaphragmatic Mesh

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Introduction

Thoracotomy is a commonly performed surgical procedure but nonetheless carries a potential range of complications involving adjacent structures including the gastrointestinal tract. Intraoperatively it can be due to position induced gastroesophageal reflux or direct surgical injury to the diaphragm or stomach. Postoperatively the anatomic changes resulting from this procedure can lead to gastric distension and/or vagal disruption affecting gastric motility. Although most reported complications are pulmonary or cardiovascular in nature, gastric injury remains an underrecognized and potentially life-threatening consequence of thoracotomy. This case highlights an unusual gastric complication following thoracotomy with diaphragmatic plication, emphasizing the importance of maintaining a broad differential diagnosis in postoperative patients presenting with gastrointestinal bleeding.

Case Description

A 67-year-old man with history of left thoracotomy with diaphragm plication presented following a syncopal episode preceded by 2-3 weeks of melena. On admission his hemoglobin was 6.9 g/dL from a baseline approximately at 11 g/dL. In the emergency department he was given prothrombin complex concentrate for apixaban reversal and started on 2 units pRBC and a pantoprazole drip. Given the concern for upper gastrointestinal bleeding, EGD was performed. Endoscopic examination revealed a rectangular, solid, mesh object appearing in the gastric fundus adjacent to a Forrest Class IIa ulcer. Trauma surgery was consulted and an exploratory laparotomy was performed. The exploratory laparotomy noted that the fundus of stomach was adherent to left diaphragm with prior pledget eroding into lumen of stomach. The foreign body was removed, and a partial gastrectomy was performed. In the following months patient had resolution of his melena and his hemoglobin continued to rise.

Discussion

Gastropleural fistulas are rare but serious complications of thoracotomies. While specific incidence rates are not well-defined in literature, these occur primarily in the setting of combined diaphragmatic resection, acute gastric distension with volvulus, or iatrogenic injury. If left unrecognized, such erosion may progress to full-thickness perforation or gastropleural fistula formation, potentially leading to empyema, sepsis, and increased mortality. Prompt surgical intervention is essential once such complications are identified, as conservative management is typically insufficient. While meticulous surgical technique remains the cornerstone of prevention, this case raises consideration for heightened postoperative surveillance. In select high-risk patients, postoperative surveillance with imaging or endoscopic evaluation may be beneficial for early detection of gastric involvement, although standardized guidelines are currently lacking.

Obstetrics and Gynecology

Abstract #4: Poorly Controlled Type 2 Diabetes Complicating Early Pregnancy of Uncertain Location

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Introduction

Pregnancy in patients with poorly controlled type 2 diabetes mellitus (T2DM) is associated with significant risks in the first trimester, including pregnancy loss, congenital anomalies, and abnormal implantation. Early recognition and aggressive glycemic management are critical, particularly in underserved populations with limited access to care. Pregnancies of uncertain location present additional challenges requiring close monitoring. This case highlights the intersection of uncontrolled T2DM, early pregnancy, and diagnostic uncertainty in a free clinic setting.

Case Description

A 24-year-old woman presented to a free clinic in December 2025 for type 2 diabetes follow-up and pregnancy confirmation. She was gravida 3 para 1011, with one prior term vaginal delivery and one second-trimester loss. Based on her last menstrual period (LMP), she presented at around 8 weeks' gestation. She had been diagnosed with T2DM a few weeks prior to her LMP with a hemoglobin A1c of 13.3%. She was started on metformin, dapagliflozin, and insulin. Home blood glucose values remained elevated in the 200–300 mg/dL range.

A urine pregnancy test in clinic was positive, prompting discontinuation of metformin and dapagliflozin. She received education on sliding scale insulin use and was scheduled for next-day follow-up in a family medicine clinic for coordination of care and to reinforce insulin management. Diabetes education and insulin titration were complicated by a language barrier, requiring interpreter services to ensure accurate understanding. In clinic, her random blood glucose remained >200 mg/dL, prompting referral to the obstetrics emergency department for intravenous insulin infusions for expedited glycemic control.

Transvaginal ultrasound initially failed to clearly identify an intrauterine pregnancy. Repeat imaging demonstrated a gestational sac located in the right cornua of the uterus. Given concern for pregnancy with cornual implantation, the patient was admitted for close monitoring with serial beta-human chorionic gonadotropin measurements and repeat imaging.

Discussion

This case underscores the complexities of managing early pregnancy in the setting of severe hyperglycemia. Poorly controlled T2DM may contribute to early pregnancy complications, including abnormal implantation, macrosomia, teratogenic effects, and pregnancy loss. Cornual or interstitial pregnancies carry increased risk of rupture and hemorrhage, necessitating early recognition and close surveillance. Additionally, this case highlights the importance of medication reconciliation in pregnancy, patient education regarding insulin use, and the critical role of coordinated care for uninsured patients. Language barriers further complicated diabetes education, emphasizing the need for culturally and linguistically appropriate resources. Early multidisciplinary involvement is essential to optimize outcomes in high-risk pregnancies complicated by metabolic disease and diagnostic uncertainty.

Abstract #31: Fetus Papyraceus in a Multifetal Pregnancy Complicated by Gestational Immune Thrombocytopenia

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Introduction

Fetus papyraceus (FP) is characterized by intrauterine demise of one fetus early in a multifetal gestation with subsequent fetal compression and desiccation between the membranes of the surviving twin. FP is a rare complication occurring in 1 in 12,500 twin pregnancies and can be associated with increased maternal and fetal complications. Early detection and diagnosis of intrauterine FP by serial ultrasound (US) examinations and placental evaluations is crucial for mitigating adverse events in these pregnancies.

Case Description

A 39-year-old Gravida 5, Para 4, Living 4 at 39 weeks gestation, with a history of three prior cesarean deliveries and Influenza A infection, presented to the labor and delivery unit as a transfer from an outside clinic for evaluation of gestational thrombocytopenia. She had limited prenatal care, establishing care at 36 weeks gestation with an US demonstrating a single live intrauterine pregnancy with an estimated gestational age of 36 weeks and 5 days and estimated fetal weight of 3270g. Repeat US on admission demonstrated a single intrauterine pregnancy with dating consistent with prior US; advanced gestational age limited anatomic evaluation, but no obvious abnormalities identified. Platelet count was 53,000 prior to transfer and 58,000 on admission. Peripheral blood smear revealed no schistocytes with the likely diagnosis being Immune thrombocytopenia (ITP) due to Influenza A infection. She received 8 units of platelets, intravenous immunoglobulin, and dexamethasone with a resulting platelet count of 93,000. Delivery via c-section was performed with delivery of a healthy female baby weighing 3720g. The term placenta was delivered along with a firm white membranous structure. On gross examination, the compressed structure measured 16cm x 7cm x 0.5cm and was noted to have anatomical features such as rib notches, upper and lower extremity, and bony structures confirming the diagnosis of fetus papyraceus. The patient was counseled on the fetal demise and reported no prior knowledge of a twin gestation. She was discharged with normalizing platelets.

Discussion

There is no established association between FP and gestational ITP, suggesting their concurrence in this case is likely coincidental. However, ITP has been associated with increased risk of maternal, fetal, and neonatal complications in multifetal gestations. Early initiation of prenatal care and consistent follow up is critical for timely identification of multifetal gestations and detection of associated complications, including FP and ITP. Thorough ultrasonographic evaluation in late pregnancy, particularly in patients with limited prenatal care, is essential for determining fetal plurality and optimizing maternal and fetal outcomes.

Abstract #42: Cesarean Anesthesia in Achondroplasia Parturient with Scoliosis and Heart Failure

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Introduction

Achondroplasia is the most prevalent skeletal dysplasia encountered in pregnancy and presents unique anesthetic challenges during cesarean deliveries. This population frequently requires cesarean delivery due to cephalopelvic disproportion. Historically, general anesthesia was preferred due to the technical difficulties, unpredictable anesthetic spread, and increased risk of high spinal blockade with neuraxial techniques in this population. Recent evidence, however, indicates that neuraxial anesthesia, particularly combined spinal–epidural techniques with individualized dosing and vigilant hemodynamic monitoring, can be safely and effectively used in achondroplasia patients.

Case Description

A 28-year-old gravida 3, para 0-1-1-1 woman at 33 weeks' gestation with achondroplasia presented for repeat cesarean delivery after evaluation of preterm contractions. Her medical history included peripartum cardiomyopathy with an ejection fraction of 45% during a previous pregnancy, intermittent supraventricular tachycardia, asthma, chronic hypertension, polyhydramnios, scoliosis with prior T3–L2 spinal fusion, Arnold–Chiari malformation status post decompression, class III obesity (BMI 53), obstructive sleep apnea, and a previous cesarean delivery performed under GA complicated by postoperative respiratory failure and prolonged intensive care unit (ICU) hospitalization.
Multidisciplinary planning involving anesthesiology, maternal-fetal medicine, and neonatology made a joint decision to proceed with repeat cesarean delivery at 34 weeks of gestation. An arterial line was placed in the operating room. Anesthesia was administered via a low-dose combined spinal-epidural block placed at approximately L4-L5, with special consideration for existing spinal hardware. Intrathecal medications included 100 mcg morphine, 15 mcg fentanyl, and 7.5 mg hyperbaric 0.75% bupivacaine. The patient's blood pressure was maintained by careful titration of a norepinephrine infusion. Adequate surgical analgesia was established, and the cesarean delivery proceeded without intraoperative complications.
Postoperatively, the patient was transferred to a cardiovascular ICU for 24-hour monitoring of potential cardiovascular decompensation. She was discharged home in stable condition on postoperative day four.

Discussion

This case highlights the complexities of anesthetic management in patients with multiple high-risk factors, including peripartum cardiomyopathy, supraventricular tachycardia, spinal fusion, Arnold–Chiari malformation, and class III obesity. General anesthesia carries increased cardiopulmonary risk in individuals with a history of cardiomyopathy and arrhythmias. In contrast, neuraxial anesthesia presents technical and hemodynamic challenges in those with altered spinal anatomy. Current literature supports the use of carefully titrated neuraxial techniques, particularly combined spinal-epidural, in selected high-risk achondroplastic parturients. However, reports describing patients with multiple overlapping high-risk conditions remain limited, underscoring the need for further investigation to guide anesthetic decision-making in medically complex obstetric patients.

Abstract #45: Back Pain Leads to New Diagnosis of Acute Myeloid Leukemia in Pregnancy

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Introduction

Leukemia is a complication found in approximately 1 in 75,000 to 100,000 pregnancies with acute myeloid leukemia constituting approximately two thirds of all cases (Fracchiolla). The overlap between common pregnancy symptoms and presenting features of hematologic malignancies often complicate timely diagnosis. Symptoms of leukemia include fatigue, night sweats, and body aches, which are frequently attributed to pregnancy. Concerns about fetal risk complicate diagnosis and treatment in pregnancy. Most cases are diagnosed in the second and third trimesters, but evidence may be skewed by those that were diagnosed in the first trimester and chose termination to proceed with timely treatment of their disease (Brenner).

Case Description

A 31 year old G4P1202 female presented to the emergency department at 29 weeks gestation with acute on chronic low back pain. Presentation included maternal tachycardia, bilateral lower extremity weakness, numbness, lumbar back pain, and intermittent urinary incontinence. MRI revealed abnormal T1 hypointense marrow throughout the lumbosacral spine with multiple lesions to bone, measuring up to 2.9cm. Laboratory evaluation demonstrated borderline leukopenia, anemia, thrombocytopenia, elevated PT/INR, elevated fibrinogen, transaminitis, and elevated lactate dehydrogenase. She was admitted and given betamethasone for fetal lung maturity, to improve thrombocytopenia, and for pain management. Bone marrow biopsy was performed, confirming Acute Myeloid Leukemia (AML). She began treatment with Cytarabine and Daunorubicin. Revumenib, a teratogen, was subsequently started. She underwent repeat cesarean delivery at 31 weeks gestation for maternal benefit to expedite treatment. She demonstrated good response to treatment and recently underwent a stem cell transplant.

Discussion

Malignancy in pregnancy is rare and directly impacts maternal and fetal outcomes. Diagnosis is challenging due to hallmark symptoms overlapping with common pregnancy complaints. Once considered as a differential, leukemia evaluation is similar in pregnant and non-pregnant states including imaging and bone marrow biopsy for diagnosis. Subsequent treatment involves chemotherapy agents. The greatest chemotherapy risks in pregnancy occur during organogenesis. Pregnancy termination is often advised at this gestational age to avoid treatment delays with grave maternal consequences (Cardonick; Loren). If pregnancy is continued, treatment should be delayed until the 2nd trimester (Loren). Treatment carries risks for intrauterine growth restriction and fetal demise. (Cardonick). Prompt treatment with chemotherapy increases remission rates from 67% for delayed treatment, to 80% for immediate treatment (Zhu). Pregnancy is physiologically demanding, but we must consider a broad differential when concerns arise.

Abstract #70: Vulvar Lichen Sclerosus Malignant Transformation to Squamous Cell Carcinoma

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Introduction

Vulvar lichen sclerosus (LS) is a benign, chronic inflammatory dermatologic disease characterized by white, atrophic plaques. Management consists of close surveillance and symptomatic treatment aimed at preventing tissue architectural changes. Progression to vulvar squamous cell carcinoma is rare, occurring in 2-5% of women with vulvar LS despite adequate treatment and follow-up. This case highlights the malignant evolution of LS and emphasizes the need for continued vigilance and early diagnostic evaluation of new lesions.

Case Description

An 86-year-old female, with a past medical history of mixed urinary incontinence status post InterStim Implant in 2021 and vulvar lichen sclerosus, presented to the gynecology clinic for evaluation of pelvic pain and bladder incontinence. She reported being diagnosed with LS in 2021 and has been treated by an outside provider with Clobetasol and Tacrolimus. Flares are characterized as persistent pruritus, pain, dysuria, bleeding, and soreness of the vulvar and vaginal area. Symptoms are worsened by urinary leakage, bowel movements, and mechanical stimulation of the area. Patient reported no history of abnormal pap smears. On physical exam of the genital area, there were two raised white nodules (medial nodule 1cm x 0.5cm; lateral mass 3mm x 4mm) and an ulcerated nodule on the lateral aspect of the left labia majora. Moderate vulvovaginal atrophy, smooth introitus, and minimal vaginal rugae were noted. SureSwab Vaginosis/Vaginitis and HSV DNA PCR were negative. Conservative management failed to improve the patient's symptoms, therefore, Gynecology Oncology was consulted and recommended vulvar biopsy of the new lesions. Surgical biopsy was preformed alongside urethral bulking to treat her urinary incontinence. Pathology from the vulvar biopsy revealed invasive well-differentiated squamous cell carcinoma of the vulva. PET-CT was obtained to assess staging and prognosis revealing no evidence of metastatic disease. Based on the imaging findings, surgical resection of the tumor along with sentinel inguinal lymph node biopsy was the recommended management. Additional treatment with adjuvant chemotherapy and radiation may be warranted if full resection is not possible.

Discussion

Although high-potency topical steroids are associated with reduced symptom burden and malignancy risk in LS, progression to squamous cell carcinoma is still possible. This case is notable for malignant transformation despite appropriate symptom management and emphasizes the importance of routine pelvic examinations every 6-12 months in those with LS to assess for lesions or masses. A low threshold for biopsy and further workup of new or persistent vulvar LS lesions is critical for timely identification and treatment of potential carcinoma.

Pediatrics

Abstract #9: Symptomatic Closed Spinal Dysraphism in an Adolescent

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Introduction

Closed spinal dysraphism is a congenital anomaly generally classified as a type of neural tube defect that often presents in infancy or early childhood but may less commonly present in adolescence or early adulthood. We present a case of closed spinal dysraphism in an adolescent who became symptomatic several months after a blunt traumatic injury to his lumbar spine.

Case Description

A 15-year-old male presented to the emergency department with chronic bilateral testicular pain, lumbar and leg pain, weakness, and numbness, dysuria, and constipation after blunt trauma to lumbar spine six months prior. These symptoms had been intermittent since the injury but acutely worsening in the last two months. He endorsed suicidal ideation attributed to severe ongoing pain. Pain was initially attributed to growing pain, urinary tract infections, and infectious enteritis, and due to severity was managed with hydrocodone-acetaminophen and gabapentin which he took continually for two months without improvement of pain. He was admitted for a workup where cerebral and full spine MRI and angiogram were non-diagnostic. Neurological exams varied greatly- initially completely normal, then asymmetric hyperreflexia, asymmetric clonus with variable laterality, and asymmetric finger-to-nose testing. He did not have any lumbar cutaneous findings. All basic labs were normal in addition to normal paraneoplastic profile and cerebrospinal studies. Urinalysis and CT scans of chest, abdomen, and pelvis were all normal. Lumbar spine MRI was initially read as normal but upon requested reevaluation showed fatty filum terminale concerning for a tethered cord. Pediatric neurosurgery recommended surgical evaluation and urodynamic studies. Urodynamic studies were normal and he was taken to the operating room for partial L5-S1 laminectomies with microscopic sectioning of the filum. Post-operatively he had incision site pain but resolution of neurological symptoms. At outpatient follow-up two weeks post operatively he reported significant resolution of his pain.

Discussion

This case represents an older presentation of closed spinal dysraphism in which differential diagnoses included growing pains, testicular torsion, urinary tract infection, sexually transmitted infection, Guillain-Barré syndrome, and acute inflammatory demyelinating polyneuropathy, all of which are more common than adolescent closed spinal dysraphism. It is imperative to diagnose and treat closed spinal dysraphism as early as possible to prevent irreversible and worsening neurological and urological symptoms.

Abstract #18: Reaction to Dubai Chocolate in Adolescent with Pre-existing Nut Allergy

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Introduction

Tree nuts and peanut allergens are among the most common causes of anaphylaxis related fatalities. Dubai Chocolate consists of pistachio and tahini paste, mixed with gluten and encased in chocolate. These ingredients are often not included on product labels, including chocolates imported from other countries, potentially leading to uninformed allergen ingestions such as this one.

Case Description

A 13-year-old presented to the ER after eating Dubai chocolate that evening and immediately experiencing throat pain. This pain quickly worsened, and EMS noted the adolescent was stridulous and vomited three times. The adolescent had a previous reaction to mixed tree nuts with lip swelling and emesis that resolved with diphenhydramine. Past medical history also includes eczema, asthma, and seasonal allergies. Cetirizine was given at home, and due to anaphylaxis, EMS gave two doses of intramuscular epinephrine, along with one dose of diphenhydramine, methylprednisolone, and one nebulized albuterol treatment. Home medications include daily cetirizine and albuterol as needed. In the emergency room, vital signs were stable. Pertinent physical exam findings included periorbital edema with breath sounds diminished on auscultation bilaterally with reported pruritis. The adolescent was mildly tachypneic without retractions, wheezing, or stridor. Additional medications included famotidine and albuterol after which respirations decreased from 24 to 20 breaths per minute. The adolescent was admitted to the hospital for observation overnight. With ongoing symptomatic improvement, the adolescent was discharged home with anaphylaxis and epinephrine education, an epinephrine prescription, and instructions to continue daily cetirizine and as needed albuterol. Due to anaphylaxis, the patient's primary care provider was recommended to place a referral to a Pediatric Allergy and Immunology.

Discussion

The pistachio paste and sesame seeds in Dubai chocolate can cause pharyngeal edema and anaphylaxis in those with tree nut and lupin allergies. It is important for people to know that Dubai chocolate may cause anaphylaxis for anyone with a tree nut or peanut allergy, so US governmental oversight of proper labeling is imperative. There is currently limited literature surrounding this area, but it highlights the importance of those with top allergens to refrain from consuming foods without proper labeling of ingredients. To prevent deaths, the standard of care for treatment of anaphylaxis remains intramuscular epinephrine as a first-line agent as well as an allergy specialist follow up to identify the allergen and prevent reactions.

Abstract #28: Beyond Common Etiologies: Infantile Pyknocytosis in Neonatal Anemia

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Introduction

Infantile pyknocytosis is a rare, transient neonatal disorder characterized by hemolytic anemia, jaundice, and pyknocytes on peripheral blood smear. We report a preterm twin presenting with late-onset jaundice and anemia, highlighting the need to consider this condition in unexplained neonatal hemolysis.

Case Description

A 3-week-old identical dichorionic–diamniotic twin born at 34 weeks and 6 days gestation with history of an 11-day NICU stay and hyperbilirubinemia requiring phototherapy, presented with late indirect hyperbilirubinemia and anemia requiring transfusion. Due to clinical instability, the infant initially required transfer to the pediatric intensive care unit, where a sepsis workup including blood culture was negative. Pediatric hematology was consulted, and labs obtained revealed severe normocytic anemia with evidence of hemolysis, including elevated LDH and reticulocytosis. Direct antiglobulin test and delayed hemolytic transfusion reaction screening were negative. TSH was within normal limits. Urinalysis and fecal occult blood testing were also negative. Hereditary Hemolytic Anemia Cascade did not identify a hemoglobinopathy or enzymopathy, and findings were not consistent with hereditary spherocytosis. Review of the peripheral blood smear demonstrated findings of pyknocytes which, in the setting of hyperbilirubinemia and significant hemolysis, are consistent with the diagnosis of infantile pyknocytosis (IP). The initial hyperbilirubinemia resolved with phototherapy and remained down-trending following discontinuation. Hemolysis was managed with continued packed red blood cell transfusions, totaling six transfusions over a two-month period. The clinical course followed the expected transient and self-resolving nature of infantile pyknocytosis, with subsequent resolution of hemolysis and stabilization of hemoglobin on outpatient follow-up.

Discussion

In this infant, late-onset jaundice, significant anemia, the presence of pyknocytes on peripheral blood smear, and the subsequent resolution of hemolysis followed the characteristic course of infantile pyknocytosis. Phototherapy and red blood cell transfusions were sufficient to address clinical symptoms until spontaneous resolution. Diagnosis requires careful exclusion of other more common causes of neonatal hemolytic anemia, such as hemoglobinopathies, enzymopathies, and immune-mediated conditions. Because the condition resolves spontaneously and is identified only after ruling out other causes, reports of infantile pyknocytosis in the literature are limited. These findings underscore the importance of considering this uncommon and likely underrecognized condition in neonates with hemolytic anemia when more well-known etiologies have been excluded.

Abstract #29: Beyond the Crepitus: When Esophageal Tears Warrant Considering Child Abuse

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Introduction

Subcutaneous emphysema in pediatrics has been associated with conditions that disrupt the oropharyngeal and esophageal mucosa or increase intraabdominal or intrathoracic pressure. Examples of this include foreign body ingestion, upper respiratory illness, increased Valsalva, and obstructive lung processes. Child physical abuse has been increasingly documented as an additional mechanism to consider in cases of subcutaneous emphysema. We present a case of subcutaneous emphysema with esophageal tear due to child abuse to further support child physical abuse as a can't-miss consideration in subcutaneous emphysema.

Case Description

A six-month-old infant with GERD presented to the emergency center with fever, poor oral intake, and neck swelling. Labs showed elevated CRP and procalcitonin. On exam, there was palpable crepitus in the anterior neck and submandibular region with scattered bruising. CT soft tissue neck showed extensive subcutaneous emphysema of the submandibular, retropharyngeal, and upper chest extending to the anterior mediastinum. Nasolaryngoscopy was performed and showed no evidence of injury. With no trauma history provided and no mechanism explaining the findings identified, concerns for child physical abuse were raised. CT head and skeletal survey were negative. Child abuse pediatrics, DHS, and law enforcement became involved. The infant developed fever and intermittent bradycardia, concerning anterior mediastinitis. IV ampicillin/sulbactam was started, however the infant continued to have poor feeding with concern for aspiration. An esophageal tear was later identified on dysphagiagram. With no clear injury mechanism to explain the exam findings, child physical abuse was diagnosed. An NG tube was placed for enteral nutrition. Antibiotics were continued until CRP and procalcitonin returned to normal. Repeat CT chest and neck showed resolution of subcutaneous emphysema and pneumomediastinum, and a direct laryngobronchoscopy performed later showed no evidence of residual mucosal injury. Antibiotics were discontinued, and the infant was able to tolerate oral feeds before being discharged home.

Discussion

Subcutaneous emphysema without a clear injury mechanism should raise concern for non-accidental trauma, especially in the presence of bruising in a non-mobile child. Other literature has outlined the importance of direct visualization of the tissues in cases of subcutaneous emphysema without a clear mechanism. Previously healthy infants with subcutaneous emphysema without a clear injury mechanism require extensive work-up with direct visualization via scope to evaluate the integrity of the tissues.

Abstract #36: Congenital Autoimmune Third-Degree Heart Block

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Introduction

Congenital atrioventricular (AV) block is a disturbance in conduction through the AV node which occurs spontaneously in utero or in the neonatal period. Fetal bradycardia, defined as a pulse of 40 to 80 beats per minute, is commonly the initial sign of this disorder. In utero exposure to maternal anti-Ro or anti-La autoantibodies, produced in both Systemic Lupus Erythematosus and Sjogren's syndrome, account for 60-90% of all congenital heart block cases. While rare in the general population, congenital complete heart block (CHB) occurs in approximately 2 percent of pregnancies among women with anti-Ro or anti-La autoantibodies, with a recurrence rate of approximately 16 percent. Infants diagnosed with congenital CHB are typically treated with observation until symptoms warrant permanent pacemaker implantation.

Case Description

A neonate was delivered at 36w+5d via emergency c-section due to non-reassuring fetal heart tones. Mother's history included uncontrolled chronic hypertension and chlamydia 4 months prior to delivery. Upon arrival to labor and delivery, mother had non-reactive syphilis Ab and negative Ab screen. The neonate was delivered with Apgar scores of 8 and 9 at 1 and 5 minutes of life, respectively. On initial assessment, they were bradycardic with heart rates in the 50-60s. Oxygen desaturations to 86% prompted use of 40% flow-by and transfer to NICU, where they were placed on continuous telemetry. STAT EKG confirmed complete heart block, and pediatric cardiology was consulted. Despite CHB, the patient did not display signs or symptoms of low cardiac output. The initial echocardiogram revealed a small patent foramen ovale with bidirectional shunt, moderate tricuspid insufficiency, usual marked discoordination of cardiac wall motion due to complete heart block, and normal biventricular systolic function. A high suspicion of maternal lupus causing congenital lupus prompted obtaining anti-Ro and anti-La antibodies, which were elevated. Repeat echocardiogram was unchanged. The neonate was discharged with a plan to monitor heart function closely. Cardiac function has remained stable through follow-up, with plans for eventual pacemaker placement when symptoms or dysfunction occur.

Discussion

This case demonstrates the importance of incorporating clinical context in medical decision making. Neonatal bradycardia without evidence of poor cardiac output (e.g. hypotension, mottled skin, poor tone, etc.) warrants further cardiac workup as opposed to resuscitative efforts, which in this case revealed complete heart block. Additionally, this case supports the association between congenital third-degree heart block and maternal systemic lupus erythematosus, despite a previously asymptomatic mother.

Abstract #53: Salmonella Osteomyelitis In An Adolescent Male Golfer

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Introduction

The incidence of osteomyelitis is 1.2-13 per 100,000 children, with approximately half of the cases occurring in children under the age of five. Most cases involve long bones, but 10-25% affect other bones. Fever and pain are the most common presenting symptoms, with most kids presenting within one week of symptom onset. The most common organisms are *Staphylococcus aureus* and *Streptococcus pneumoniae*, whereas osteomyelitis due to *Salmonella* species is rare in otherwise healthy children.

Case Description

A fourteen-year-old male, an avid golfer, presented with a six-week history of left knee pain and a three-week history of progressive left knee swelling. The pain was described as achy and was more prominent with physical activity. He had played in many golf tournaments throughout the summer. Despite increasing pain and swelling, he denied having any systemic symptoms, including fevers, cough, congestion, emesis, diarrhea, ear pain, or rashes.

Physical examination revealed a 5 x 5 cm area of swelling over the proximal tibia without erythema or warmth. There was mild tenderness to palpation over the proximal tibia, with full range of motion and no neurovascular deficits. No pain was elicited with varus or valgus stress testing. Firm endpoint felt with the anterior drawer and Lachman test.

X-ray exhibited a lucency of the proximal tibial metaphysis with possible extension into the proximal epiphysis. Labs significant for an elevated ESR (26 mm/hr), elevated CRP (1.49 mg/L), and CBC showed a slight leukocytosis (9.4 cells/uL). MRI significant for an intraosseous abscess of the proximal tibial metaphysis measuring 2 x 4.6 x 1.7 cm with surrounding osteomyelitis of the tibial metaphysis, which extends into the epiphysis of the tibia. The patient was taken to surgery for incision and drainage. Cultures during this procedure grew Salmonella. Final diagnosis was a left proximal tibia Brodie's abscess secondary to *Salmonella* infection.

Discussion

This is a rare example of osteomyelitis secondary to Salmonella in a healthy child without predisposing factors. *Salmonella* accounts for only 0.45% of all osteomyelitis cases. Furthermore, this patient did not have hemoglobinopathies and was previously healthy, which are predisposing factors. The subacute clinical course observed in this case is consistent with the common presentation of aBrodie's Abscess. This case highlights the importance of avoiding diagnostic anchoring and considering early radiography evaluation when the clinical presentation does not align with a more typical diagnosis.

Abstract #56: Hyperinsulinemic Hypoglycemia from 15q26 Deletion Disrupting IGF-1 Receptor Signaling: Genetic Case Report

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Introduction

15q26 deletions are relatively rare chromosomal abnormalities that commonly affect the IGF-1 receptor resulting in growth-related abnormalities. We present a case of 15q26 deletion involving the IGF-1 receptor identified through the workup of hyperinsulinemic hypoglycemia induced seizure episodes.

Case Description

A 4-year-old child was admitted to the hospital after presenting to the emergency room due to a seizure with documented hypoglycemia. Their past medical history included alpha-1 antitrypsin deficiency with zz phenotype, subglottic stenosis requiring dilations, dysphagia with G-Tube dependency, a 46,xx,der(15)t(4;15)(q28.3;26.2) chromosomal abnormality, and multiple occurrences of seizure-like episodes. The physical exam upon admission was normal, as the child had returned to baseline in the emergency room after receiving interventions that included correction of hypoglycemia. The child had no recent fevers nor were there signs/symptoms of illness. After admission and discussion with the parents, dextrose containing fluids were held to allow for critical and diagnostic blood samplings to be obtained during a hypoglycemic event. When the child's blood sugar was less than 54 mg/dl, sampling was collected and then IV dextrose was administered to reverse the hypoglycemia. Relevant lab results included elevated insulin level of 43µIU/mL (reference range 3.0-19.0 µIU/mL). Insulinoma concern was ruled out via an unremarkable PET scan. Inborn errors of metabolism were not identified on thorough work-up. The patient was able to fully wean off dextrose-containing IV fluids when transitioned from bolus feeds to continuous G-tube feedings from 0900-1800 and 2100-0600. Acetazolamide was also initiated for the treatment of hyperinsulinemia at the recommendation of the pediatric endocrinologist. There was no ongoing hypoglycemia or seizures, and the child was discharged home with close follow-up with the primary care provider and pediatric endocrinologist.

Discussion

After further research and conversation with the geneticist, it was determined that the 15q26.2 deletion involved the IGF-1 receptor which can lead to insulin resistance and compensatory hyperinsulinemia. Based on this information, the patient's recurrent seizures are likely associated with hypoglycemia. Transient hyperinsulinemic hypoglycemia, such as the kind explored in this case, often improve with time. However, acetazolamide has been shown to be effective in preventing hypoglycemic episodes before sustained neurologic injury. Clinicians should consider chromosomal deletions in unexplained hyperinsulinemia.

Abstract #62: Look Again: Pediatric Moyamoya Disease

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Introduction

Moyamoya disease is a progressive cerebrovascular disorder caused by narrowing or occlusion of the internal carotid arteries and the formation of fragile collateral vessels with unclear pathophysiology, though genetic predisposition has been described in the literature. Moyamoya disease is known to have a female predominance in the United States with bimodal age distribution. Early diagnosis paired with prompt surgical intervention is essential to prevent disease progression.

Case Description

A previously healthy 3-year-old female initially presented to the emergency department with 1 day of left upper extremity paralysis, left lower extremity weakness, headache and non-bloody diarrhea. A stroke protocol was initiated, with resultant negative CT head, as well as normal anticoagulation labs. CT angiogram (CTA) was negative for large vessel obstruction; however, it was limited which prompted a repeat CTA. Stool was positive for sapovirus. Given persistent focal neurological deficits, a routine EEG was obtained ruling out epileptiform activity but showed mild encephalopathy with mild slowing in the right posterior region. MRI brain was significant for right MCA territory infarct with a normal spine MRI. Repeat CT angiogram showed “small caliber of intracranial arteries” with differential diagnosis of severe stenosis, atresia or occlusion. Images were reviewed with Neurointerventional Radiology and were found to be concerning for Moyamoya disease. Daily aspirin was started, and IV fluids were given due to concerns of dehydration as a trigger for her cerebral infarct. She was referred to Pediatric Neurovascular Surgery with plans for catheter angiogram and surgical repair, as well as continued intensive outpatient occupational therapy for functional and adaptive mobility.

Discussion

This case highlights the diagnostic challenges of Moyamoya disease in young children as they often present with nonspecific symptoms of headache, weakness, or seizure-like activity until a major ischemic infarct. While brain MRI and angiography are diagnostic, expert review is often necessary as changes can initially be subtle. Dehydration is a major precipitating factor for ischemic events in Moyamoya disease as it lowers blood pressure and increases blood viscosity. Early neuroimaging is critical for prompt referral to the appropriate specialist for definitive diagnostic angiography and surgical management to optimize neurological outcomes. This case demonstrates the need for a multidisciplinary team and appropriate reassessment in pediatric stroke presentations.

Abstract #63: Todd's Paralysis in Adolescent with New Onset Seizure-Disorder

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Introduction

Todd's paralysis is a neurological disorder in which a seizure is followed by a period of temporary weakness or paralysis. This paralysis can be partial or complete and lasting anywhere from 30 minutes to 36 hours. Approximately 13% of all seizures show signs of Todd's paralysis and there is no predilection related to gender or race. Although the etiology is unknown, the most accepted theory involves exhaustion of the primary motor cortex (or any other part of the brain) after neuronal hyperexcitation in the setting of a seizure. Todd's paralysis corresponds to the ictal topography, and the symptoms presented will localize to the area of the brain where the seizure occurred.

Case Description

A previously healthy 15-year-old admitted with altered mental status, aphasia and right sided hemiparesis/neglect. The adolescent reported right sided jaw pain fever and URI symptoms weeks prior to admission. Submental swelling worsened to needing amoxicillin treatment. On the day of arrival, the adolescent was found face down in the bathroom covered in emesis and unresponsive secondary to a presumed seizure. Due to concerns for possible stroke, imaging was performed. CT of the head showed a right submandibular soft tissue mass. Additional imaging negative. EEG showed notable left hemispheric slowing suggestive of lateral cerebral dysfunction. Pediatric Neurology felt the adolescent likely had a left hemispheric seizure resulting in postictal aphasia and Todd's paralysis. Oxcarbazepine was prescribed for anti-seizure prophylaxis. The adolescent showed substantial neurologic improvement, returning to baseline on day two of hospitalization. Neurologic symptoms were estimated to have lasted greater than 24 hours. Post-discharged instructions included continued antibiotic treatment for sialadenitis and follow up with outpatient neurology.

Discussion

This case demonstrates that Todd's paralysis can mimic a stroke, and the importance of differentiation of the two. Careful work up is needed to determine the cause of symptoms in relation to one diagnosis or the other. It also illustrates symptoms not typically seen in Todd's paralysis including post-ictal aphasia and prolonged symptoms. Interestingly in this case, there was no known history of a seizure disorder prior to presentation, but genetic predilection and a febrile illness likely lowered the seizure threshold.

Abstract #64: Air Leak Syndromes in Status Asthmaticus

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Introduction

Asthma is a chronic inflammatory disorder characterized by reversible air flow obstruction and bronchial hyper responsiveness. While most asthma exacerbations are resolved with standard bronchodilator and corticosteroid therapy, severe attacks can occasionally lead to rare but serious complications such as pneumothorax, pneumomediastinum, and subcutaneous emphysema.

Case Description

A 10-year-old with moderate persistent asthma presented to ER with fever, cough, and dyspnea which was unresponsive to home albuterol treatment. On admission, oxygen saturation was in the 80s and improved to 90s with 4 liters of oxygen. Physical exam revealed bilateral coarse rhonchi and subcutaneous crepitus. Initial chest X-ray showed a small left apical pneumothorax, pneumomediastinum, and subcutaneous emphysema. Viral studies were negative. The child initially received bronchodilators and IV corticosteroids. Despite stable imaging, the child had transient hypoxic episodes on hospital days 5 and 7 requiring brief oxygen support. Pulmonology was consulted. Mycoplasma testing was negative, and controller therapy was changed from mometasone to mometasone/formoterol. On hospital day 8, the child had a new onset of low-grade fever, tachycardia, lower lung lobe decreased breath sounds, and leukocytosis of 19,000 concerning underlying pneumonia for which antibiotics were started. The repeat chest x-ray was stable. An echocardiogram was done to rule out myocarditis, with normal results. The child improved with no oxygen desaturation for 48 hours and was discharged on day 10 with extensive asthma education.

Discussion

This case highlights that although air leaks are rare complications of acute asthma, they should be considered in the setting of forceful coughing and severe bronchospasm. Awareness of these entities is essential as a clinical presentation may mimic worsening asthma or more critical thoracic emergencies. Early recognition through careful physical examination and imaging allows for timely conservative management in most cases and helps prevent unnecessary invasive interventions.

Abstract #65: Adolescent with Chronic Inflammatory Demyelinating Polyneuropathy and Genetic Variants: A Case Report

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Introduction

Chronic Inflammatory Demyelinating Polyneuropathy (CIDP) is an autoimmune condition that affects the myelin sheath around peripheral nerves. Symptoms of CIDP include progressing muscle weakness, numbness, difficulty with balance and coordination, loss or weakening of deep tendon reflexes, and neuropathic pain. CIDP currently has no known cause.

Case Description

A 13-year-old with a history of classic congenital adrenal hyperplasia (CAH), autism spectrum disorder (ASD), avoidant restrictive food intake disorder (ARFID), and iron deficiency anemia was hospitalized for bilateral lower extremity weakness, fatigue, and gait instability. Although the adolescent was able to go from lying to a standing position, there was required assistance with ataxic ambulation, symmetric lower extremity weakness, absent deep tendon reflexes, no clonus and Babinski down going. An extensive work up included MRI thoracic spine that showed previously noted chronic central compression deformities of the T6 and T7 vertebral bodies, MRI cervical spine with possible early developing syrinx, and MRI brain with no abnormalities. A lumbar puncture revealed elevated protein of 79 mg/dL with monoclonal gammaglobinopathy but without cells. Although Guillain Barre Syndrome (GBS) was considered, it was deemed less likely than CIDP due to the long onset, presence of monoclonal gammaglobinopathy in CSF which is uncommon in pediatric GBS, no preceding infection before symptoms, and no respiratory concerns. Whole Exome Sequencing (WES) later revealed: CYP21A2, heterozygous for multiple pathogenic variants, confirming adrenal insufficiency due to CAH; GIGYF1, a likely pathogenic variant associated with autism spectrum disorder and neurodevelopmental features; and DNMY1H, a variant of uncertain significance, with a potential association with neurologic symptoms but requiring additional investigation for potential association with CIDP. IVIG was started and provided partial improvement. Upon discharge home, adolescent continued IVIG therapy, syrinx monitoring, physical and occupational therapy.

Discussion

CIDP is a rare condition for adolescents. Chronic steroid replacement, as needed for CAH, has not been associated with CIDP; however, high-dose steroid use as first-line treatment for CIDP has been documented to exacerbate symptoms. Therefore, Pulse oral corticosteroids in pediatric patients have been used. This case may show a genetic link to the patient's weakness. Although variants in DYNC1HI genes have known associates with neurological disorders, like Charcot-Marie-Tooth disease, spinal muscular atrophy, and seizure disorders, further research into the DNMY1H variant is needed to see if associations may contribute to the neurologic symptoms seen in CIDP. Clinicians should consider how genetic predisposition modifies immune-related neuropathies and integrate genetic findings into a broad diagnostic and therapeutic framework.

Abstract #67: Retinopathy After Rapid Blood Glucose Control in CFRD: A Case Report

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Introduction

Cystic fibrosis, a multisystem autosomal recessive disorder, affects the salt and water conductance via CFTR channels. This disrupts many organ systems such as sweat glands, lungs, gastrointestinal, and pancreatic secretions. Cystic-Fibrosis Related Diabetes (CFRD) is the manifestation of the abnormal glucose metabolism, insulin resistance, and beta cell dysfunction through the defect in CFTR. This report investigates an adult with CFRD who, after rapid reduction in A1c, presented with asymmetric bilateral painless decreased visual acuity and papilledema with hemorrhages in the nerve fiber layer of the retina

Case Description

A 35-year-old with cystic fibrosis and cystic-fibrosis related diabetes for 5 years rapidly reduced their blood glucose. Their A1c went from 14.6 to 8.7 over 3 months. Two months after the rapid reduction in A1c, the patient presented to their routine optometrist after 45 days of gradual painless right eye vision loss. On exam papilledema was noted and urgent MRI and spinal tap were completed. The MRI demonstrated bilateral optic disc fluid distention consistent with papilledema without intracranial findings. The spinal tap demonstrated normal cerebrospinal fluid and opening pressure. After further serological, ophthalmological, and neurological examinations, there were no definitive answers of the etiology. Diagnoses of non-arteritic anterior ischemic optic neuropathy, infectious causes, and side effects of cystic fibrosis medications were discussed between the teams. The patient's vision continued to progressively worsen despite acetazolamide and glucocorticoids. Six weeks after the initial onset in their right eye, they began developing similar symptoms in the left eye.

Discussion

The interplay between cystic fibrosis' secretory dysfunction and the microvascular complications involved with diabetes indicate an etiology of vision loss in cystic fibrosis. These rapid decreases in vision specifically after A1c control have been documented in patients with Type 1 and Type 2 Diabetes but not in CFRD. Describing the course of treatment prior to control of A1c and the onset and gradual resolution of vision changes can help providers best carefully manage patient's diabetes and decrease microvascular side effects such as non-arteritic anterior ischemic optic neuropathy.

Abstract #68: Anaphylactic Reaction Due to Previously Unknown Allergy in Dubai Chocolate Brownie

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Introduction

Anaphylaxis is a life-threatening allergic reaction commonly seen in children. We present an adolescent without any known food allergies who developed anaphylaxis after eating a Dubai chocolate brownie containing pistachios.

Case Description

A 13-year-old previously healthy adolescent, presented to the emergency department with swelling of the lips, tongue, and throat. The adolescent previously ate a Dubai chocolate brownie containing pistachios, and the mother reported that within 5 minutes they developed the described symptoms, as well as dizziness, chest tightness, some shortness of breath, and redness of the eyes with periorbital swelling. There was no known previous food allergy. Upon arrival to the ED, within 15 minutes of ingestion, vital signs included elevated heart rate at 100 and blood pressure 141/89. IM epinephrine was administered for anaphylaxis along with IV diphenhydramine, famotidine, and methylprednisolone. The adolescent was admitted to the hospital for observation, and there was denial of any ongoing chest pain, shortness of breath, tongue or lip swelling, nausea, or vomiting. Mild uvular swelling remained the only finding on physical exam. Following ongoing clinical improvement, anaphylaxis education, demonstration of epinephrine pen administration, and providing an epinephrine pen prescription, the adolescent was discharged with a diagnosis of anaphylaxis likely secondary to pistachio nut allergy. Recommendations were given to follow-up with primary care provider and an Allergy and Immunology specialist.

Discussion

This case highlights a relatively rare initial presentation for an adolescent onset allergy. Pistachio is an uncommon initially presenting tree nut allergy, and a less frequent cause of tree nut induced anaphylaxis. Initial presentation of tree nut allergy may occur in adolescence; however, a significant majority first present in childhood, with an initial reaction at median age of 36 months (mean 77 months). Though the Dubai chocolate brownie was advertised as containing pistachios on the vendor's website, the patient was unaware of their allergy. In addition, pistachio has a lower allergy eliciting dose in comparison with peanuts, which highlights pistachio as a potent allergen in susceptible individuals. While peanut anaphylaxis is widely recognized, there is less awareness of pistachio anaphylaxis. This highlights the uniqueness of this case, as well as the importance of future research in the incidence of pistachio-related anaphylaxis and the first-time incidence of tree nut allergy in adolescence.

Abstract #69: Incidental Thoracic Neuroblastoma in a Child with Cystic Fibrosis: A Case Report

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Introduction

Thoracic Neuroblastomas are malignant tumors made of neural crest-derived sympathetic nervous tissue found in the chest of primarily pediatric patients. We present a case of an incidental finding of a thoracic neuroblastoma on abdominal x-ray (KUB) in a child with cystic fibrosis (CF) that was overlooked on previous imaging, emphasizing the importance of scrutinizing imaging beyond the primary reason it was obtained.

Case Description

An 11-year-old with a history of CF with GI and pulmonary manifestations presented to pediatric gastroenterology with acute on chronic constipation manifesting as bloody stools. Physical exam findings were unremarkable. A KUB was obtained, showing a soft tissue density over the left lower chest. Further evaluation with CT revealed a posterior mediastinal mass most suggestive of a neuroblastic tumor with multiple image-defined risk factors, including encasement of the descending thoracic aorta and left main pulmonary artery, flattening of the left inferior pulmonary vein and compression and narrowing of the left mainstem and lower lobe bronchus. Further studies including MIBG and biopsy were consistent with neuroblastoma, while MRI ruled out spinal cord involvement. The International Neuroblastoma Staging System defines this as stage 2B, meaning ipsilateral non-adherent lymph nodes were positive for tumor cells. Per the International Neuroblastoma Risk Group classification, vital structure involvement in multiple body compartments stratifies this into the L2 group. N-MYC oncogene amplification, a critical prognostic factor, was negative. In conjunction with other factors, this assigned the child to an intermediate-risk group. Pre-treatment tumor measurements were 5.3 x 5.2 x 9.2 cm. They underwent two cycles of chemotherapy with carboplatin, etoposide, cyclophosphamide, dexrazoxane, and doxorubicin. Post-treatment tumor measurements were 5.9 x 5.4 x 9.6 cm, demonstrating no significant response to chemotherapy. A tumor debulking procedure successfully decreased tumor size to 2.5 x 2.1 x 7.3 cm. Current monitoring plans will include imaging every three months.

Discussion

In independent image review, this tumor was visible on prior KUBs. The patient remained asymptomatic throughout the entire diagnostic course, as do most pediatric patients. If not for this incidental finding, the child's prognosis could have been compromised. This case is a reminder to carefully review imaging without bias related to reason for imaging. No correlative relationship exists between CF and neuroblastomas, thus making this diagnosis one of vigilance rather than expectation.

Abstract #71: Prolidase Deficiency Diagnosed in Infant with Neutropenic Fever with Right Foot Osteomyelitis

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Introduction

Prolidase Deficiency (PD) is an exceedingly rare autosomal recessive (AR) genetic disorder caused by mutations of the peptidase D (PEPD) gene. This leads to deficiencies in the prolidase D enzyme resulting in abnormal metabolism of proline-containing proteins such as collagen. PD is categorized as a metabolic disorder and an inborn error of immunity with dermatologic symptoms most common, typically non-healing ulcers on the hands or feet. Other clinical manifestations include dysmorphic features, intellectual disabilities, recurrent infections, anemia, autoimmunity, and splenomegaly. This case describes an infant diagnosed with PD with atypical immunologic findings.

Case Description

A 9-month-old with history of eczema, impetigo, and cellulitis initially presented to clinic with fever, fussiness, and persistent right foot cellulitis. Outpatient labs were concerning for neutropenia requiring hospital admission. Labs were significant for severe neutropenia with Absolute neutrophil count (ANC) of 100 cells/microliter, mild anemia, elevated CRP, and ferritin. MRI confirmed right foot osteomyelitis, and the infant was started on IV antibiotics. Palpable splenomegaly on exam was confirmed by US and CT imaging. Preliminary infectious workup, including GPP, RPP, and blood cultures, was negative. Pediatric Hematology Oncology was consulted, and the infant underwent bone marrow (BM) evaluation which revealed severe myeloid hypoplasia. ANC remained persistently low despite treatment with G-CSF but showed mild response when switched to GM-CSF. Pediatric Infectious Disease (ID) and Pediatric Immunology were consulted. Extensive infectious disease workup was negative for HIV, Hepatitis, and fungal infections with evidence of prior EBV and CMV infections. The infant was found to have elevated IgE at 4892 kU/L with abnormal lymphocyte antigen and mitogen testing. Despite only mild improvement in neutropenia, the infant's symptoms resolved, and they were deemed stable for discharge on antibiotics and fungal prophylaxis. Genetic testing via Invitae IEM and cytopenia panel resulted positive for AR PD as well as 2 other variants of unknown significance (VUS) possibly associated with Riddle Syndrome and Cohen Syndrome.

Discussion

PD has a worldwide incidence of 1-2 cases per million live births, and due to its rarity, there are no clinical practice guidelines or standard therapies. Patients are typically managed symptomatically. PD is classically associated with skin manifestations, elevated IgE, splenomegaly, and recurrent infections as seen in this infant. However, severe persistent neutropenia despite treatment, as also seen in this infant, is an atypical finding. Management of neutropenia in the setting of PD requires further investigation and novel approaches.

Abstract #72: Congenital Hyperinsulinism Requiring Total Pancreatectomy

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Introduction

Congenital hyperinsulinism (CH) leads to severe and persistent hypoglycemia in infancy and childhood. Inappropriate insulin secretion results in severe hypoglycemia and suppresses ketogenesis which increases the risk of neurological sequelae, ultimately necessitating medical or surgical treatment. Several known genetic mutations and syndromes are associated with CH. We present a severe case of CH in which genetic evaluation revealed no previously associated monogenic variants but did reveal a variant in the UBE2A gene.

Case Description

An infant born at 37 weeks weighing 3510 grams had severe hypoglycemia at birth requiring TPN that was slowly weaned over one month in the NICU. He had persistent episodes of hypoglycemia and failure to thrive during the first few months of life. At 5 months of age, he was admitted for lethargy and hypoglycemia requiring dextrose containing fluids. At that time he was noted to have increased insulin levels and low beta hydroxybutyrate, consistent with hyperinsulinism. The infant continued to have hypoglycemia despite diazoxide treatment and dextrose-containing fluids were unable to be weaned. He was then transferred for specialty treatment. Repeat labs confirmed hyperinsulinism while receiving glucose at a high infusion rate. Additionally, glucagon infusion was required. He had persistent hypoglycemia with octreotide therapy and was unable to tolerate weaning of dextrose fluids. He underwent pancreatic head removal; however no focal lesion was observed and pathologic review indicated increased islet cells and nuclear enlargement. Post operatively, the infant continued to require high glucose infusion rates, so he underwent a total pancreatectomy for diffuse disease. Following pancreatectomy, the infant was started on hybrid closed loop insulin pump therapy and pancreatic enzyme replacement. Genetic testing throughout his workup was negative for any known variants in hyperinsulinism related genes; however, he was found to have a UBE2A variant which was reported to be a possible pathogenic variant.

Discussion

There are many known genetic causes of congenital hyperinsulinism based on monogenic variants or genetic syndromes. In this case, no variants were noted in the genes typically associated with CH; however, a variant in the UBE2A gene was discovered and could be pathogenic for this infant's hyperinsulinism. No previous cases of UBE2A leading to hyperinsulinism have been reported. Typically, UBE2A deficiency causes intellectual delay, distinct facies, and developmental delays. However, UBE2A deficiency could potentially be the cause of this infant's CH. More research into the relationship between this gene and hyperinsulinism is necessary to determine definitive correlation.

Abstract #75: Atypical Lumbar and Unilateral Knee Involvement in Pediatric IgA Vasculitis

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Introduction

Immunoglobulin A (IgA) vasculitis is the most common systemic vasculitis in children. It is a small-vessel disorder caused by the perivascular deposition of IgA-containing immune complexes. The condition occurs more frequently in the fall and winter months, coinciding with an increase in upper respiratory tract infections. IgA vasculitis often follows an antecedent bacterial or viral illness, most commonly caused by *Streptococcus pneumoniae*, *Streptococcus pyogenes*, or rhinovirus/enterovirus. Clinical manifestations typically involve the skin, joints, gastrointestinal tract, and kidneys. In approximately 94% of pediatric cases, the disease is self-limiting; in the remaining cases, renal involvement remains the primary determinant of a long-term prognosis.

Case Description

A previously healthy, immunized 7 year-old presented to the emergency department with acute onset left knee and lumbar pain. Physical exam revealed significant left knee swelling, lumbar midline swelling, and palpable purpura on bilateral lower extremities. There was no history of trauma but the child had an upper respiratory infection one week prior. A complete blood count revealed leukocytosis and thrombocytosis. Urinalysis showed elevated urobilinogen 1.0 mg/dL and trace blood without proteinuria. Urine microscopy was unremarkable with no RBC or WBCs. Inflammatory markers were mildly elevated. Imaging revealed prepatellar fluid on the left knee x-ray and crescentic fluid superficial to the posterior paraspinous musculature on a lumbar spine CT with contrast. Based on the clinical history, examination, and laboratory findings, a diagnosis of IgA vasculitis was made. The child was treated with naproxen and discharged with instructions for close follow-up with primary care for urinalysis every two weeks.

Discussion

The presence of acute back pain with swelling and rash prompted a broader diagnostic workup. The joints most commonly affected in IgA vasculitis are the knees and ankles. However, the diagnosis in this case was supported by the combination of palpable purpura and arthralgia following a recent viral infection. Diagnostic testing is useful for excluding other causes of purpura, as platelet counts and coagulation studies typically remain normal in IgA vasculitis. Laboratory evaluation also helps assess renal involvement, which is the primary determinant of long-term prognosis. Although IgA vasculitis is typically self-limiting, urinary monitoring is recommended for at least six months in patients without renal involvement and for twelve months in those with renal involvement. Recognizing that renal involvement may appear as a later finding is critical for long-term renal outcomes in children with recent IgA vasculitis.

Psychiatry

Abstract #33: Treatment Resistant Bipolar Disorder with Psychotic Symptoms in Comorbid Autism Spectrum Disorder

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Introduction

Autism spectrum disorder (ASD) is a major focus in psychiatry as prevalence rises and clinicians are challenged to distinguish core autistic traits from co-occurring, treatable psychiatric conditions like bipolar disorder and psychosis. In 2022 approximately 3.2% of 8-year-olds had a current ASD diagnosis. Diagnosing mood and psychotic disorders in individuals with ASD is difficult due to symptom overlap, atypical presentations, and communication barriers. About 7% of individuals with ASD have co-occurring bipolar disorder, which is challenging to diagnose given its fluctuating depressive and manic episodes, inherent diagnostic complexity, and misuse of the term “bipolar” in everyday language.

Case Description

A 17-year-old transgender male with Bipolar I disorder and Autism Spectrum Disorder presented with severe mania and psychosis refractory to two weeks of inpatient treatment with high-dose antipsychotics, mood stabilizers, and benzodiazepines. He was admitted to SFCH general pediatric service for psychiatric consultation and ECT evaluation. At admission, medications included chlorpromazine 100 mg TID, lithium 300 mg daily / 600 mg nightly, olanzapine 5 mg daily / 15 mg nightly, diphenhydramine 50 mg nightly, and lorazepam 3 mg nightly. He remained acutely manic with paranoid delusions involving the FBI and was responding to internal stimuli. Laboratory and neuroimaging studies were unremarkable except for subtherapeutic lithium level of 0.3. Although ECT was initially planned, lithium dosing was increased to 900 mg daily / 1200 mg nightly, achieving a therapeutic level of 0.7; resulting in resolution of manic symptoms. Within three months of discharge, he developed significant metabolic side effects (weight gain, hyperlipidemia, hypertriglyceridemia, rising HbA1c). Attempts to reduce medication burden were complicated by symptom monitoring challenges related to his ASD-related fixed interests in government agencies. Ultimately, stabilization on therapeutic lurasidone allowed tapering and discontinuation of lithium, chlorpromazine, lorazepam, and olanzapine.

Discussion

Psychiatric medication regimens must be individualized and often involve trial and error due to absence of tests predicting treatment response. When multiple trials of high-dose antipsychotics and mood stabilizers fail, electroconvulsive therapy (ECT) is often considered, despite potential risks and side effects. This highlights the importance of ensuring medications are trialed at appropriate and patient-specific dosages. This case emphasizes need for longitudinal assessment and careful evaluation of symptom timing, laboratory values, and changes from baseline when diagnosing bipolar disorder in individuals with ASD. Psychotic symptoms may resemble ASD-related fixed interests, complicating diagnosis. Awareness of these challenges is critical to improving diagnostic accuracy, guiding effective treatment, and reducing misdiagnosis and ineffective interventions.

Abstract #43: Diagnostic Complexity in Severe Bipolar Disorder: A Case of Dissociative Amnesia

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Introduction

Dissociative amnesia is the inability to recall autobiographical information and has three levels of severity: localized, selective, and generalized which is the rare type. Generalized amnesia is typically associated with those exposed to severe emotional stress or conflict.

Case Description

A 39-year-old female was brought to the hospital after being found driving erratically. Patient presented with mania, psychosis, catatonia, and generalized retrograde amnesia. Thorough public records and outside medical record search, we discovered that patient had a history of bipolar 1 disorder, previously treated with paliperidone palmitate, as well as recent history of catatonia and generalized retrograde amnesia with fugue. Psychological testing completed at previous psychiatric facility noted concern for organic causes of amnesia in addition to stress.

Patient was started on olanzapine, but was switched to risperidone due to availability of long-acting injectable formulation. Patient was briefly on lorazepam (for catatonia symptoms) and lithium, which was discontinued due to side effects and patient refusal. After receiving risperidone 100 mg subcutaneous injection, patient was discharged to a shelter. However, she re-admitted within 12 hours due to poor mental status. She was eventually transferred to a general medical hospital for further encephalopathy work up. MRI brain and EEG were unremarkable. Urine drug screen, TSH, ANA, rheumatoid factor, C3/C4 complements, ammonia, HIV, syphilis, cancer markers, ceruloplasmin, urine copper, and NMDA antibodies were all negative. Cognitive testing performed was within normal limits. Lumbar puncture did reveal HHV-6 found by qualitative PCR. Infectious disease suspected this was secondary to previous infection and did not warrant treatment.

Patient spontaneously improved and began regaining memories. Her risperidone dose was maximized to 125 mg subcutaneous and patient discharged on court committed outpatient status to be followed by an assertive community treatment team.

Discussion

Dissociative amnesia is a complex and challenging diagnosis due to patient limitations in providing a history and the importance of ruling out other potential psychiatric, neurological and medical causes. This patient does meet criteria for this diagnosis due to her abnormal retrograde amnesia and significant functional impairment. No underlying neurological, medical, or substance induced cause was identified. She does have a cooccurring Bipolar 1 disorder diagnosis, but this would not explain her symptoms.

These patients are highly vulnerable and at times may require hospitalization for safety concerns. Currently, there are no evidence-based treatments for dissociative amnesia. However, treatment of any cooccurring disorders and psychotherapy is often pursued.

Surgery

Abstract #23: Subacute Internal Herniation Following Roux-en-Y Gastric Bypass Presenting as Bilateral Lower Extremity Edema

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Introduction
Internal hernia is a known complication of Roux-en-Y gastric bypass (RYGB), but presentations involving venous outflow obstruction are rare. We report a case in which subacute bilateral lower extremity edema and non-specific abdominal bloating for several weeks presented in a 38-year-old woman with a history of RYGB performed two years prior.

Case Description
Our patient is a 38-year-old woman with history of RYGB presenting with complaints of lower extremity edema and postprandial abdominal bloating for weeks. She reported to the emergency department after worsening abdominal pain and imaging was suggestive of an internal herniation with mesenteric swirling. Retrospective review of images showed compression of the inferior vena cava (IVC) due to the internal hernia and likely the mechanism for the lower extremity edema due to impaired venous return. The patient underwent surgical exploration which revealed herniation of her jejunojejunostomy through Petersen's defect, a potential space created as a result of a normal gastric bypass procedure. This was reduced and the Petersen's defect was closed. After surgery, both abdominal bloating and lower extremity edema resolved at 2- and 4-week follow-up.

Discussion
Internal herniations through Petersen's defect following gastric bypass have been previously reported, but IVC compression mimicking peripheral venous congestion is scarcely cited. This case highlights the importance of high clinical suspicion for internal hernia in post-gastric bypass patients, even with atypical subacute symptoms. Early recognition of internal hernias can prevent progression to bowel ischemia allowing for early surgical intervention.

Abstract #32: Omental Free Flap Approach for Cerebral Revascularization Following Moyamoya Related Ischemia

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Introduction

Moyamoya is a rare vascular condition characterized by the narrowing of cerebral arteries resulting in debilitating cerebrovascular ischemic events. The underlying etiology of arterial narrowing from moyamoya is still not fully understood, and its prevalence follows a bimodal distribution, with peaks in childhood and early adulthood. Primary treatments for Moyamoya include direct revascularization, with direct extracranial-to-intracranial bypass, and indirect revascularization, including omental flap transposition. The human omental microvascular endothelial (HOME) cells of the omentum have demonstrated the ability to produce vascular endothelial growth factor (VEGF) and fibroblast growth factor-2 (FGF-2), required for proper neovascularization. Pedicled flaps have predominantly been performed in pediatric cases with increased efficacy stemming from age-dependent cerebrovascular plasticity. However, it has been conducted in various groups and shown to produce upwards of 98% improvement in cerebral revascularization. There are limited salvage options after direct bypass in adults. In this case, we present a novel omental free flap to the brain in the treatment of complicated Moyamoya.

Case Description

37-year-old female presenting with longstanding and progressive left upper and lower extremity weakness. Previous medical history of ischemic stroke secondary to Moyamoya. Middle cerebral artery stenosis was suspected from clinical findings consistent with damage to the right prefrontal cortex. Angiography was performed, confirming cerebral arterial stenosis and anatomy not suitable for a direct bypass. Revascularization using an omental free flap was performed with a multidisciplinary team. The omentum was harvested laparoscopically with preservation of the right gastroepiploic pedicle. The flap was implanted to the superficial temporal artery. On 90-day follow up cerebral angiography showed improved cerebral blood flow, and the patient had improved strength in both the left upper and lower extremities.

Discussion

Moyamoya is primarily treated through less invasive direct revascularization; however, when this approach fails or in instances of complex and extensive ischemia, there are limited options. Omental free flap as a multidisciplinary salvage option provides rich vascular supply and facilitates neoangiogenesis due to properties of the omentum. This treatment resulted in improved cerebral perfusion and improved patient function and quality of life. Current advancements enable laparoscopic omental harvesting, decreasing invasiveness. This case is unique due to the combined rarity of the condition, age at the time of treatment, and the complex mechanism underlying the treatment modality. This case demonstrates the feasibility and proof of concept that omental free flaps can be used as a salvage option and may have a role in earlier treatment of Moyamoya.

Abstract #38: Finding of Clear Cell Hidradenoma During Abdominal Cyst Excision

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Introduction

Clear cell hidradenoma (CCH) is a rare benign tumor originating from apical sweat glands [1]. It can display either apocrine or eccrine differentiation [1]. General presentation is that of cystic nodules, 5-30 mm in size, most commonly found on the scalp or trunk in women, though they may also present on the limbs and can occur in all ages [1,2]. Lesions are solitary and characterized by slow growth with potential serous discharge [2]. Although benign, CCH have a 10% rate of recurrence following surgical incision [2]. Here we report the case of CCH found in a cyst excised from the abdomen of a 60-year-old male.

Case Description

The patient is a 60-year-old male who underwent abdominal cyst excision. The cyst was examined during his clinic visit and measured about 1.5 cm long. Past medical history was notable for diabetes and prosthetic right leg.

Excision of the cyst was performed in clinic. Following injection of local anesthetic at the incision site, an elliptical incision was made and the cyst was removed, after which skin was closed.

The patient tolerated the procedure well, and postoperative course was uneventful. The incision was well approximated and well healing at the one-week follow-up, and the patient reported minimal pain.

Pathologic evaluation of the specimen revealed skin with cystic structure, elliptical in shape and yellow-white in color. Cyst contents included granular material consisting of squamous neoplasm with clear cell features, and cells showed no significant atypia or necrosis. Cytology specimen was negative for malignancy. Additionally, the tumor was found to stain positive with cytokeratin and negative with vimentin. Staining for cytokeratin and vimentin was done to rule out the remote possibility of metastases from clear cell renal cell carcinoma.

Discussion

The differential diagnosis for abdominal subcutaneous masses is broad, including abscesses, sebaceous cysts, lipomas and epidermal inclusion cysts [3]. Within this spectrum lies CCH, a benign adnexal tumor with rare transformation to its malignant counterpart, hidradenocarcinoma.

Imaging studies such as MRI can be used to detect CCH [3,4]. Treatment of choice is wide surgical excision with examination of pathology following excision to confirm the diagnosis, as done in our case. Prognosis is thought to be excellent with surgical excision, though long-term follow-up may be needed to track potential benign metastases or transformation to hidradenocarcinoma. Should such transformation occur, prognosis is poorer due to high rates of metastasis and recurrence, requiring wide and deep excision.

Abstract #73: Ventriculoperitoneal Shunting: Salvage After Complicated Aneurysmal Subarachnoid Hemorrhage

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Introduction

Subarachnoid hemorrhage (SAH) secondary to ruptured intracranial aneurysm is a neurologic emergency associated with significant morbidity and mortality. In certain cases, cerebrospinal fluid (CSF) circulation can be obstructed secondary to blood products causing obstructive hydrocephalus. External ventricular drains (EVDs) are frequently used for acute CSF diversion and intracranial pressure monitoring in the neurocritical care setting. However, patients with persistent or communicating hydrocephalus require long-term CSF diversion. Timing of chronic shunting depends on worsening ventricular enlargement, neurological symptoms, and cerebral vasospasm. This clinical vignette highlights the course of a patient with aneurysmal SAH who required prolonged external CSF diversion and ultimately a VP shunt placement.

Case Description

A 34-year-old man presented with four days of sudden-onset severe headache, diplopia, nausea, vomiting, and recent methamphetamine use. Computed tomography of the head demonstrated diffuse subarachnoid hemorrhage. The patient was classified as Fisher grade 1 and Hunt-Hess grade 3. An etiology was not initially identified for his SAH, and an external ventricular drain was placed. The EVD was kept open at 20cm H_2O and progress was monitored with transcranial Doppler ultrasound. Computed tomography of the head continued to show ventricular enlargement despite EVD placement. The Nimodipine 60mg q4H was scheduled for increasingly severe vasospasm. After two diagnostic cerebral angiograms a right fetal aneurysm in the PCA was discovered. This was treated with craniotomy and clipping of the aneurysm.

The months-long postoperative course was complicated by a growing pseudomeningocele, with *Staphylococcal epidermidis* infection. Antibiotics, washout, and placement of a second EVD were required. The EVD was converted to a ventriculoperitoneal shunt placement. The patient subsequently developed gram-negative bacteremia requiring removal of the shunt.

After completion of a course of antibiotics, there was improvement in symptoms. The patient was discharged after eight weeks with improved functional status with plan to return to work despite continued headaches at decreased severity.

Discussion

This case illustrates the common progression from acute hydrocephalus managed with EVD placement to chronic shunt-dependent hydrocephalus following aneurysmal SAH. Prolonged EVD use increases risks of infection and limits mobilization, making the transition to VP shunting critical in these patients. Persistent failure of EVD weaning, high CSF output, and radiographic ventricular enlargement are key indicators for long-term shunt placement. Early recognition of shunt dependency may reduce hospital length of stay and improve neurologic recovery. This vignette underscores the importance of individualized decision-making in the management of post-SAH hydrocephalus and highlights VP shunting as an essential intervention in selected patients.

Abstract #76: Calming the Storm: Managing Paroxysmal Sympathetic Hyperactivity Following Traumatic Brain Injury

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Introduction

Paroxysmal Sympathetic Hyperactivity (PSH) is a complex syndrome of inappropriate sympathetic activation that can occur following traumatic brain injuries. Episodes vary in length and triggers, and may include tachycardia, tachypnea, hypertension, diaphoresis, hyperthermia, dystonia, and posturing.

Case Description

A 19-year-old male was brought to the trauma bay following a high-speed MVC. His Glasgow Coma Score was 6 on assessment and he was subsequently intubated. Following imaging evaluation, the patient's neurologic and cranial pathologies included multiple intracranial hemorrhages, bitemporal skull fractures, a sphenoid fracture, and possible seizure activity causing reactive leukocytosis. He was transferred to the neurotrauma ICU for further management. His care team initiated levetiracetam for seizure prophylaxis, propofol sedation, and multimodal pain control of acetaminophen, oxycodone, and fentanyl. EEG demonstrated no evidence of seizures, but MRI demonstrated restricted diffusion consistent with diffuse axonal injury (DIA). Over the next several days, the patient experienced numerous episodes of hypertension, tachycardia >200 BPM, and significant agitation concerning for PSH. We concluded that these episodes were triggered by minor stimuli, such as tracheostomy care, bowel movement cleanup, and heart and lung exams. The patient was given PRN lorazepam to treat his PSH episodes. This was effective but required frequent dosing about 1.5 hours apart. Due to patient's age, propofol was changed to dexmedetomidine, which provided deeper but more stable sedation. The frequency and depth of analgesia and sedation required to control the patient's PSH created concern that his care team could not appropriately assess and track neurological status. Per the recommendations of his neurocritical care team, the patient was started on propranolol, bromocriptine, clonazepam, and gabapentin to wean his heavy sedation, prevent PSH episodes, and minimize need for abortive lorazepam doses. A few days after initiating this new PHS regimen, the patient tolerated nursing care tasks and stimuli while requiring minimal doses of lorazepam. Dexmedetomidine sedation was eventually discontinued, after which the patient began to localize to pain and open his eyes to stimuli during neurologic exams. He was eventually stable enough to be transferred to long-term acute care, and episodes of PSH had completely subsided.

Discussion

This case illustrates the variability in PSH treatment and prevention. While diagnostic criteria for PSH were determined in 2014, no formal treatment guidelines currently exist, and frequent medication changes and dose modifications are usually necessary. TBI patients experiencing PSH have worse neurological and overall outcomes, so frequent reassessment and clinician flexibility is required for optimal patient care.