School of COMMUNITY MEDICINE

Clinical Vignette Symposium 2022

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 11th annual Clinical Vignette Symposium (CVS) hosted by the OU 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.

We are excited to bring the Clinical Vignette Symposium back as an in-person event. In addition to poster and podium presentations at the 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. Prizes will be awarded to the top scoring posters and the podium presentations.

I would like to extend a warm thank you to the presenters as well as those who organized and are hosting CVS this year. We hope you enjoy CVS 2022.

Sincerely,

Martina Jelley

Martina Jelley, MD, MSPH, FACP

Professor and Vice Chair for Research, Department of Internal Medicine

Julian Rothbaum Chair in Community Health Research

Assistant Dean for Clinical Research

University of Oklahoma School of Community Medicine

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ORDSA would like to thank all faculty and staff who contributed their time and energy to organizing this event. ORDSA would also like to thank the library staff for their help and services. Finally, ORDSA would like to express our appreciation to faculty who provide financial contributions that allow us to give out monetary prizes.

Clinical Vignette Symposium 2022

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Oral Presenters

Abstract #25 Obesity Medicine Pharmacy Alleviating Comorbidities and Paving the Way for Bariatric Surgery

Mr. Gabriel Thompson - OU-TU School of Community Medicine, Department of Internal Medicine
Dr. Jessie Richards - OU-TU School of Community Medicine, Department of Internal Medicine
Dr. Geoffrey Chow - OU-TU School of Community Medicine, Department of Surgery

Introduction
Bariatric surgery has proven a successful modality for reducing patient morbidity and mortality.1 Sustained loss of excess adiposity dissolves weight-related complications such as metabolic syndrome, dyslipidemia, gastroesophageal reflux disease, diabetes, hypertension, cardiovascular disease, fatty liver disease, obstructive sleep apnea, osteoarthritis, depression, and infertility.2 However, many of these maladies exist as barriers to undergoing bariatric surgery. The arrival of next generation pharmaceutical therapy allows patients to overcome these barriers and achieve prerequisite perioperative weight loss for bariatric surgery.

Case Description
We introduce a case of a 38-year-old male of 606.54 pounds with body-mass-index of 78.16 in-lbs who presented for bariatric surgery evaluation. His past medical history was complicated by gastroesophageal reflux disease, type-2 diabetes mellitus, hypertension, congestive heart failure, lymphedema, sleep apnea, 28 pack-years of smoking and oxygen-dependent COPD. He had successfully lost 50 pounds with diet and exercise in the past but since regained his weight.
At the time of initial evaluation, he required 3 liters of nasal canula oxygen and could walk only 1 city block. His diet exhibited caloric surplus. He was without a regular exercise program. After surgical evaluation, he was referred to an internal medicine bariatrician for pharmaceutical weight loss therapy as well as nutrition, psychiatry, cardiology, and gastrointestinal surgery for an esophagogastroduodenoscopy prior to bariatric surgery. Secondary causes of obesity (e.g. hypothyroidism, Cushing syndrome) were ruled out via laboratory testing. The patient was determined at moderate risk of binge eating. He was started on 25 mg topiramate daily and 0.25 mg weekly semaglutide with dosage increases as indicated. At one month, the patient had been following a diet of 3 protein shakes a day and one small meal. He had lost 54.78 pounds, was no longer requiring home oxygen, and able to exercise at an increased duration. Topirimate was increased to 25 mg BID. semaglutide was increased to 0.5 mg weekly and he was started on 10 mg emapaglifozin daily. At two months, the patient had lost another 32.56 pounds and reported increased wellness and exercise capacity. At the patient's pre-operative bariatric surgery appointment, he was weighed at 513.48 pounds. Over a span of eighty-two days, the patient lost a total of 125 pounds and was deemed a candidate for a staged, sleeve gastrectomy. Surgery was performed following normal cardiac stress testing and esophagogastroduodenoscopy.

Discussion/Conclusion
This case illustrates the overwhelming utility of next generation obesity pharmacotherapy in alleviating adipose-related comorbidities and improving bariatric surgical candidacy.

Abstract #45 Catatonia Associated with Cerebral Venous Thrombosis in a COVID Positive Adolescent

Dr. Sara Verga - OU-TU School of Community Medicine, Department of Psychiatry
Dr. Dean Martin - OU-TU School of Community Medicine, Department of Psychiatry

Introduction

Catatonia is a psychomotor syndrome characterized by abnormal movements and decreased responsiveness. Diagnosis is made by observing or eliciting at least 3 of the following 12 criteria: stupor, catalepsy, waxy flexibility, mutism, negativism, posturing, mannerism, stereotypy, agitation, grimacing, echolalia, echopraxia. It is commonly associated with psychiatric disorders but can also be secondary to a medical condition, more commonly neurological or metabolic conditions.

Case Description

Patient is a 17-year-old female brought in by her guardian for evaluation at a psychiatric assessment center following a month of regressive behavior and concerns of possible hallucinations. While at assessment center, she began hyperventilating and had three seizures. She was transferred to nearby hospital and continued to seize, becoming hypoxic and requiring intubation. CT was unremarkable; UDS was positive for THC and benzodiazepines. Patient was transferred to ICU of a children's hospital. She continued to have poverty of speech, decreased responsiveness, and disorganized behavior after extubation. Child psychiatric was consulted for these concerns, and differential included psychotic disorder and catatonia, either secondary to psychiatric or medical cause. Patient underwent extensive medical evaluation to rule out medical causes, which was overall unremarkable, including EEG, CSF studies, CBC, CRP, and Anti-NMDA antibodies. She did test positive for COVID, which resulted in delay of Brain MRI being obtained. She had partial response to lorazepam challenge, and scheduled doses of lorazepam were started after. Bush Francis Catatonia Scale scores did lower partially with scheduled lorazepam, but full resolution of symptoms was not observed. MRI done on day 10 showed findings suspicious for superior sagittal thrombosis. Brain MRV showed superior sagittal and bilateral transverse venous thrombosis. Patient was started on anticoagulation therapy and discharged from hospital with recommendation of psychiatry and neurology outpatient follow up. She did not follow up with neurology but did have slow resolution of symptoms per outpatient psychiatry records.

Discussion/Conclusion

Catatonia typically results in resolution of symptoms with treatment of underlying cause along with benzodiazepines or electroconvulsive therapy. It is commonly associated with psychiatric disorders, but it is important to evaluate for medical causes as well, especially when there are concerning signs/symptoms. In this patient, there was only a partial response to benzodiazepines, but further improvement with anticoagulation therapy. This along with no previous psychiatric history supports an underlying medical cause. This patient had no history of health conditions associated with hyper-coagulopathies. However, COVID has been associated with risk of arterial and venous thromboembolic complications.

Poster Presentations

Emergency Medicine

Abstract #47 Pulmonary Embolism: A Unique Cause of a Spontaneous Pneumothorax

Dr. Joshua Miller - OU-TU School of Community Medicine, Department of Emergency Medicine
Dr. Eric Lee - OU-TU School of Community Medicine, Department of Emergency Medicine

Introduction

A spontaneous pneumothorax secondary to a pulmonary embolism is a rare complication that has few reported cases in medical literature1. This case highlights the unique sequence of events and the abnormal presentation of a spontaneous pneumothorax secondary to a pulmonary embolism in a young otherwise healthy male.

Case Description

A 25-year-old male presented to the emergency department complaining of constant right sided pleuritic chest pain of sudden onset three days ago. The patient had been treated by a walk-in clinic for a right-sided pneumonia fifteen days prior with an antibiotic and a prednisone taper. The patient had one episode of hemoptysis one day after starting this treatment, but still finished the treatment course and had near resolution of his symptoms. Three days prior to reporting to the emergency department the patient experienced right sided chest pain and worsening shortness of breath. The patient presented with mild tachycardia, but otherwise had unremarkable vital signs with an oxygen saturation of 95% on room air. His physical exam was unremarkable other than markedly decreased right sided breath sounds. Chest x-ray revealed a large right sided pneumothorax. The radiologist suggested a chest CT with IV contrast due to tethering of lung tissue that may affect thoracostomy tube placement. The chest CT showed that the right middle lobe was tethered in areas extending from the right hilum to the lateral pleura. There was an intraluminal thrombus in the right pulmonary artery at the bifurcation of the right middle lobe artery and descending right lower lobe artery with associated pulmonary infarction. A right sided thoracostomy was performed with near full lung expansion. The patient was placed on continuous heparin infusion and admitted to the ICU. The patient was subsequently diagnosed with multiple DVTs, and had laboratory results suggestive of a clotting disorder. He was discharged home five days later with plan to follow up with hematology for further coagulopathy workup.

Discussion/Conclusion

While there are well known causes of a secondary spontaneous pneumothorax, there are few documented cases of a secondary spontaneous pneumothorax caused by a pulmonary embolism. In this case, recent pneumonia led to a sedimentary lifestyle most likely resulting in DVTs and subsequently resulting in a pulmonary embolism and pulmonary infarct. The suggested pathophysiology is infarct of the lung causing peripheral alveolar rupture and leakage of air into the pleural space1. Pulmonary embolism should be considered in hypercoagulable patients with a spontaneous pneumothorax without other common risk factors.

Abstract #61 Necrotizing Fasciitis Presenting as Isolated Hip Pain in a Geriatric Patient

Dr. Nathan Finch - OU-TU School of Community Medicine, Department of Emergency Medicine
Dr. Jeffrey Goodloe - OU-TU School of Community Medicine, Department of Emergency Medicine

Introduction

Necrotizing fasciitis is a life-threatening infection of the deep soft tissues with a 20-30% mortality rate. Classic findings are fever, dermal erythema with edema, crepitus, and pain out of proportion to clinical exam. Physical exam has a sensitivity of 21-46%; however, absence of classic findings cannot rule out this pathology. Computed tomography (CT) has a sensitivity of 88% for necrotizing fasciitis, though imaging should not delay definitive management by surgical debridement if clinically suspected.

Case Description

A 75-year-old female presented to the emergency department with a one-week history of left hip pain. She denied history of recent trauma. She denied fevers or chills. Her medical history was significant for colon cancer with resection and permanent colostomy. Physical exam proved notable for marked pain with both active and passive range of motion of the left hip. Though mild swelling of the entire left leg was appreciated, there were no signs of erythema, crepitus, or fluctuance. The patient was hypertensive and tachycardic at 140 beats per minute. Fluid resuscitation with normal saline was immediately initiated. Laboratory findings revealed leukocytosis of 21,150 and plasma lactate of 3.1 mmol/l. Radiographs of left hip showed possible gas in the soft tissues of the left groin and upper leg but were not definitive per radiologist or emergency physician read. An emergent CT of the pelvis and left femur with contrast showed perirectal abscess with extensive gas in the retroperitoneum and medial compartment of the left thigh. Vancomycin and cefepime were administered for broad-spectrum coverage. Clindamycin was added after CT imaging to cover for additional etiologies. General surgery took the patient emergently to the operating room and found extensive necrotizing fasciitis of the perirectal space and left leg. Wound cultures grew polymicrobial species. Multiple debridements were required and performed. Despite multi-specialty intensive care, the patient died during this inpatient admission.

Discussion/Conclusion

This case demonstrates the need to maintain a broad differential in evaluating common complaints such as isolated hip pain in the elderly. The patient presented with subtle findings of sepsis and pain out of proportion, otherwise lacking the classic physical exam findings of necrotizing fasciitis. The availability of immediate CT imaging was helpful in both diagnosis and rapid surgical intervention. However, despite timely care, this case underscores the high mortality of necrotizing fasciitis and illustrates the necessity for early consideration in presentations involving pain related to joints and soft tissues.

Abstract #69 Acute Hemolytic Anemia Secondary to Brown Recluse Spider Bite

Dr. Gautami Gandham – OU-TU School of Community Medicine, Department of Emergency Medicine
Dr. Christoph Schieche – OU-TU School of Community Medicine, Department of Emergency Medicine

Introduction

Spiders of the genus Loxosceles are known as recluse spiders, violin spiders, fiddleback spiders. The most accurate method of identifying a recluse spider involves counting the eyes. The brown recluse is described as having a violin pattern on its anterior cephalothorax. One of its toxins, phospholipases D cause the majority of the clinical signs, including necrotic wounds, platelet disorders, hemolysis, and acute renal failure. Symptoms may be divided into local signs at the bite site, skin necrosis, and less commonly systemic effects. The site can sometimes be identified by two small cutaneous puncture marks with surrounding erythema. 20% of bites become necrotic. The progression of necrosis from a recluse spider bite typically occurs over several days. The original papule or plaque develops a dusky red or blue color in the center of the lesion, and a dry, depressed center may herald necrosis.

Case Description

A 27-year-old female with no significant medical history presented with fatigue and dyspnea. She was bit by a brown recluse spider on R shoulder one week ago. She saw the spider at that time. She was started on clindamycin at outside ER. Patient reported worsening generalized weakness and shortness of breath for last three days, vomiting last night, pain at wound site and R arm weakness. Endorsed subjective fever. Denied chest pain, diarrhea, abdominal pain. History positive for tobacco use and marijuana use. Physical exam findings include: tachycardia, scleral icterus, jaundiced, and right shoulder necrotic wound. Labs findings include: low hemoglobin, elevated bilirubin, transaminitis. Symptoms and lab work was concerning for acute hemolytic anemia secondary to brown recluse bite. Patient was also started on broad spectrum antibiotics (vancomycin and merrem) due to concern for sepsis. She received blood transfusions. Patient was admitted to ICU. Hematology and Surgery were consulted. Patient was started on prednisone for positive DAT per hematology. She was taken for I&D and wound VAC was placed. After a week of hospitalization, she was discharged on Augmentin and doxycycline.

Discussion/Conclusion

Systemic symptoms are an infrequent complication of recluse bites, and do not correlate with local findings. Rare complications include angioedema, acute hemolytic anemia, DIC, rhabdomyolysis, myonecrosis, renal failure, coma, and death. The risk of these complications is low in the United States. Although hemolytic anemia following a recluse spider bite can be severe, it is typically self-limited. The primary treatment consists of blood transfusions for patients with a rapidly falling hematocrit or uncompensated anemia.

Family Medicine

Abstract #18 Sciwora with Concurrent Concussion: Presentation of Two Cases and Literature Review

Mr. William Maher - OU-Tulsa School of Community Medicine
Mr. Zachariah Fisher - OSU College of Osteopathic Medicine
Mr. William Dudney - OU-Tulsa School of Community Medicine
Dr. Eric Sherburn - OU-Tulsa School of Community Medicine, Department of Family and Community Medicine

Introduction
Spinal cord injury without radiologic abnormality (SCIWORA) is a rare but known entity affecting children more than adults. SCIWORA was first described in 1982 as signs of myelopathy in pediatric patients whose imaging studies revealed no signs of injury. In reviewing the literature, there are conflicting reports as to what constitutes SCIWORA due to advances in MRI imaging in recent years. Our definition includes absence of radiologic abnormality on MRI. To our knowledge, there are few reports linking concussive head injury and SCIWORA. We offer two cases of pediatric females referred to the sports medicine clinic for concussion evaluation.

Case Description
Patient A is a 9-year-old female with a history of anxiety, ADHD, and asthma who fell off a horse and experienced subsequent loss of consciousness, seizure activity, change in mental status, and left hemiparesis. Patient B is a 15-year-old female with no prior medical history who developed headache, severe neck pain, photophobia, numbness and tingling in bilateral upper and lower extremities, and urinary incontinence after a tubing accident. Both patients had obvious concussive injuries; however, both patients also had deficits attributable to a non-concussive etiology, such as spinal cord injury (SCI). Spinal X-ray, CT, and MRI of both patients were negative, suggesting a diagnosis of SCIWORA. Patient A's treatment included rest, held ADHD and anxiety medication, vitamin D3, fish oil, and PT/OT/ST/vestibular therapy. Patient B received IV dexamethasone, prochlorperazine, ondansetron, and diphenhydramine and was discharged with methylprednisolone and diazepam, a soft collar, urodynamic testing, and vestibular therapy. Currently, ~7 months later, Patient A has recovered completely, and Patient B has regained normal motor strength while having only mild concussive symptoms.

Discussion/Conclusion
The actual incidence of SCIWORA is unknown, especially with concurrent concussion. Sports-related injuries are the most common mechanism of SCIWORA in children. Based on the most recent systematic review (2015), since 1982 less than 500 cases of SCIWORA involving pediatric patients have been reported, of which only one occurred with reported concurrent concussion. A 2021 SCI retrospective cohort study seems to be one of the first reports to find a significant association between traumatic brain injury and SCIWORA. We report two cases of SCIWORA discovered during routine concussion evaluation, supporting the possibility that SCIWORA is likely underreported or underrecognized in the setting of sports-related concussion (SRC) and must be considered in the differential diagnosis of all concussion patients.

Abstract #55 Hemophilus Influenza Meningitis with Sinus Thrombosis in a 5-Month-Old Female

Dr. Helga Skaftason - OU-TU School of Community Medicine, Department of Family and Community Medicine
Dr. Jennifer Weakley - OU-TU School of Community Medicine, Department of Family and Community Medicine

Introduction

Haemophilus influenzae (H. flu) is a gram-negative coccobacilli classified as either encapsulated (serotypes a through f) or unencapsulated "non-typable". H. flu, type b (Hib) is the most virulent and concerning with 3 to 6% of all Hib cases in children resulting in death. Vaccinations against Hib began in 1987 for children and 1990 for infants, resulting in Hib meningitis annual incidence in children under five dropping from greater than 10,000 cases to less than 200 cases over the next decade. However, Hib vaccination offers no protection against other H. flu serotypes, including the next most virulent serotype, type A, Hia. Non-b H. flu invasive disease incidence in 2017 was 1.7 cases per 100,000 in children younger than 5. The following is a case of Hia meningitis in a previously healthy 5-month-old female complicated by straight sinus thrombosis.

Case Description

A 5-month-old female presented to an emergency department with her mother reporting fever and bulging fontanelle. The patient's mother had learned about the fontanelle exam during a routine well-child visit. This patient was up to date with two of three Hib vaccines. Lumbar puncture revealed cloudy CSF positive for gram negative diplococci. Patient was started on vancomycin, cephalexin, acyclovir, dexamethasone, and levetiracetam before transfer to another facility for PICU. Straight sinus thrombosis was noted on imaging. Heparin was initiated and later de-escalated to enoxaparin as thrombosis was non-occlusive. The patient transferred to another facility for 21 days of ceftriaxone after H. flu, unknown serotype grew on culture. EEG evaluation returned normal so levetiracetam was discontinued. Outpatient hematology evaluation revealed a negative thrombophilia workup so enoxaprin was ultimately discontinued. The patient had no resultant neurologic or developmental deficits. Outpatient hearing screen is currently pending. Bacterial serotyping later completed and determined to have been Hia.

Discussion/Conclusion

There has been speculation that with improved control of Hib through vaccination, invasive disease caused by other strains of H. flu may increase. In the U.S., this has not been seen. Overall, this case demonstrates that despite vaccination, H. flu as a cause of meningitis should remain on a differential as a cause of severe disease even in vaccinated children. For this 5 month-old, outcomes were good in part due to rapid identification and action by the patient's mother, also highlighting the importance of patient education during routine well-child visits.

Abstract #57 Collapsing Focal Segmental Glomerulosclerosis (cFSGS) – A Rising Cause of Acute Renal Failure

Dr. Jocelyn Gray - OU-TU School of Community Medicine, Department of Family and Community Medicine
Dr. Simone Bigelow - OU-TU School of Community Medicine, Department of Family and Community Medicine
Dr. Morgan Jackson - OU-TU School of Community Medicine, Department of Family and Community Medicine

Introduction

Focal segmental glomerulosclerosis is a histologic finding underlying approximately 25% of nephrotic syndrome cases in adults. The collapsing variant is responsible for an increasing proportion of FSGS, approximately 10-24%. The collapsing variant often presents with more severe nephrotic syndrome and greater renal impairment. It is most often seen in association with HIV infection. This report details a case of rapidly progressive renal failure in a patient with cFSGS not associated with HIV.

Case Description

A 74-year-old female with chronic kidney disease IIIa, hypertension, and diabetes presented to the emergency room with altered mental status. She was found to have sepsis from pneumonia with subsequent bacteremia. She was admitted to the hospital for antibiotics and had rapid improvement in her mental status. She was noted to have an acute kidney injury on admission with a creatinine of 4.12, approximately triple her baseline, which did not improve with intravenous fluids. She also had peripheral edema, hypoalbuminemia, and proteinuria. Her renal failure progressed to a peak creatinine of 6.48. Nephrology was consulted and a renal biopsy was performed. Histology revealed nodular diabetic glomerulopathy with superimposed cFSGS. She was initiated on high dose steroids and her creatinine began to slowly improve. After discharging from the hospital, she continued to follow with nephrology and was tapered off steroids due to little improvement in renal function. She has now progressed to chronic kidney disease IV.

Discussion/Conclusion

The incidence of cFSGS is on the rise. Most cases are associated with HIV, and of the remainder, most are idiopathic. However, cFSGS has been linked with several diseases including lupus, Hepatitis, Parvovirus, Cytomegalovirus, and Covid-19. Diagnosis of cFSGS requires a renal biopsy. Initial treatment usually consists of immunosuppression with high dose corticosteroids. There are few existing studies examining the efficacy of treatment, and most demonstrate poor treatment response. Studies show patients with larger proteinuria and hypoalbuminemia values tend to have worse response to immunosuppression. In this case, the patient's cFSGS was idiopathic. She presented with large proteinuria and hypoalbuminemia and has not responded well to immunosuppression therapy.

In patients with rapidly worsening renal function and signs or symptoms of nephrotic syndrome, collapsing focal segmental glomerulosclerosis should be included in the differential. Incidence of cFSGS is on the rise and is associated with an ever-growing list of disease states. In cFSGS, early detection with intensive treatment may be critical in preserving renal function.

Abstract #59 Onychomadesis, A Rare Side Effect of Hand, Foot and Mouth Disease

Dr. Melissa Gerald - OU-TU School of Community Medicine, Department of Family and Community Medicine
Dr. Simone Bigelow - OU-TU School of Community Medicine, Department of Family and Community Medicine

Introduction

Coxsackie virus A, commonly known as Hand, Foot, and Mouth Disease (HFMD), is a viral illness that most commonly affects infant and children under the age of eight. Symptoms typically include fever and a classic viral exanthem notable for a vesicular rash involving the hands, feet, mouth, and genitalia. Rarely, patients may present 6-8 weeks with a late manifestation involving fingernail and toenail shedding.

Case Description

A 7-year-old previously healthy male presented to clinic with his mother reporting a 3-day history of painful and bloody fingertips after his fingernails started detaching from the nail bed. Physical exam was notable for seven fingernails displaying departure of the nails from the beds in a horizontal fashion with clear lines of demarcation. Additionally, some of the patient's toenails showed early signs of nail shedding. Minimal bleeding was noted, and the patient was tearful on exam. Further investigation revealed the patient had experienced a fever, oral lesions and peeling skin on his hands and feet six weeks prior. Removal of the partially detached nails was performed in clinic. Initially, a dermatology referral was placed but later cancelled after additional research revealed a correlation between nail shedding and HFMD, a condition referred to as onychomadesis. Onychomadesis is self-resolving, and the nails typically grow back without additional incident. The patient's mom was contacted and updated.

Discussion/Conclusion

Onychomadesis is an unusual and late sequelae of HFMD. Fingernail insults in children include fungal infections, vitamin deficiencies, viral illnesses, dermatological conditions, and autoimmune diseases. Thus, gathering a detailed history is essential in arriving at an accurate diagnosis. The mechanism of action behind nail shedding associated with HFMD is not well-understood, though it is postulated that the virus may arrest nail matrix proliferation in these patients.

As family physicians, we frequently see pediatric patients with hand, foot, and mouth disease. While we are familiar with the common signs and symptoms of the viral illness, we also must familiarize ourselves with the less-common and late-presenting ones too. This can help to avoid unnecessary referrals and workups.

Abstract #63 Guillain-Barré Syndrome in the Setting of Ovarian Teratoma, COVID-19 and Lymphocytic Colitis

Dr. Livia Yumi Maruoka Nishi - OU-TU School of Community Medicine, Department of Family and Community Medicine
Dr. Jennifer Weakley - OU-TU School of Community Medicine, Department of Family and Community Medicine

Introduction
Guillain-Barré Syndrome (GBS) is a flaccid symmetric weakness that develops from days to months after the inciting event; 9-17% of the cases can cause severe disability or even death. Its pathogenesis is not completely understood and in some cases the etiology may remain unknown. The incidence of GBS ranges from 0.4 to 2.5 per 100,000 person-years. This case report describes an unknown cause of GBS in the setting of ovarian mature teratoma, COVID-19 infection and lymphocytic colitis.

Case Description
A 29-year-old female with a prolonged history of almost 10 years of chronic diarrhea was seen in a primary care clinic. She was referred to a gastroenterologist who started her on budesonide after a colonoscopy showed lymphocytic colitis. After starting treatment, the patient developed an ascending paralysis and had several admissions to the hospital. Scans included an MRI of spine and head and CT that were all normal. An EMG and lumbar puncture returned consistent with GBS. Additionally due to abnormal uterine bleeding, she was referral to a gynecologist, who ordered a transvaginal ultrasound and pelvic CT. A laparoscopic biopsy confirmed the diagnosis of ovarian mature teratoma. COVID-19 infection was also diagnosed around the same time of developing symptoms of GBS.

Discussion/Conclusion
Several diseases are associated with GBS, with two-thirds of cases preceded by respiratory and gastrointestinal symptoms. The most common infection associated with GBS is Campylobacter jejuni, however it has recently been associated with COVID 19. Rarely GBS is associated with cancer as a paraneoplastic syndrome. Increased neuron-specific enolase serum levels are associated with both GBS and immature teratoma, but an association with mature teratoma has not been identified. Currently there are no studies demonstrating association of steroids and the development of GBS. However, a 2015 study showed that steroids could decrease scavengers responsive for nerve regeneration, delaying its recovery. In our patient, it remains uncertain if the budesonide worsened an already developing GBS or if there was any association with the steroid itself.
GBS is a disheartening disease and can cause severe disability. Our patient experienced several medical issues that could have an association with development of GBS, but no cause and effect can currently be stated. Thus as family physicians, it is important to be aware of possible causes of this debilitating condition to better educate patients to avoid triggers and avoid relapses.

Abstract #68 Recurrent Klebsiella Meningitis in a 69-year-old Female

Dr. John Duke- OU-TU School of Community Medicine, Department of Family and Community Medicine
Dr. Janelle Whitt - OU-TU School of Community Medicine, Department of Family and Community Medicine

Introduction

Meningitis in the geriatric population is uncommon, and Klebsiella meningitis is exceptionally rare, but does still occur. One study reported that, out of over 3,000 cases of meningitis, only 7 were caused by Klebsiella species. We present a case of recurrent Klebsiella meningitis in a 69-year-old Vietnamese female.

Case Description

A 69-year-old Vietnamese female with past medical history of Type 2 diabetes, hypertension, hepatitis C, and anemia presented to the Emergency Department complaining of fever, neck pain, and confusion. Seven weeks prior to this presentation, the patient had been diagnosed with Klebsiella meningitis and treated with 6 weeks of ertapenem. She had follow-up as an outpatient and showed clinical improvement. Initial workup for this presentation showed WBC of 15.12, Hemoglobin of 8.8, lumbar puncture showed WBC of 595/uL with neutrophil predominance, glucose of 28, and protein of 199. CSF culture grew klebsiella, species was not determined. CT head and neck angiogram was negative for hemorrhage or aneurysm. She was treated inpatient with cefepime, and had an ischemic stroke as a complication of the infection. After completing 6 weeks of cefepime she was discharged home with hospice.

Discussion/Conclusion

The initial diagnosis of this patient was quickly reached. It is not possible to say if this recurrence of Klebsiella meningitis was treatment failure or reinfection, however both her previous lumbar puncture and the lumbar puncture on this presentation grew Klebsiella that were sensitive to cephalosporins. She also showed clinical improvement during her first 6-week course of antibiotics. A repeat lumbar puncture to test for cure is not the standard of care and was not obtained.

Geriatric patients are at risk of Klebsiella meningitis, especially because it can be associated with malignancies. Annual preventative care can help reduce the chances of meningitis. Recurrent bacterial meningitis is exceptionally rare, but has a high chance of poor outcome. While rare, recurrence should be considered and treated aggressively to prevent sequelae of the infection.

Internal Medicine

Abstract #15 Actinomyces Radingae in Diabetic Foot Osteomyelitis: A Case Report

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Dr. Danielle Lewis - OU-TU School of Community Medicine, Department of Internal Medicine
Dr. Jabraan Pasha - OU-TU School of Community Medicine, Department of Internal Medicine

Introduction

Actinomyces spp. are Gram-positive rods found in normal human flora, most commonly the oral and gastrointestinal tract.1 Actinomyces radingae is native to the urogenital flora but has been found in cutaneous infections.1-3 To our knowledge, we report the first case of A. radingae as a causative agent in diabetic foot osteomyelitis.

Case Description

A 52-year-old male with a past medical history of CKD3, CHF, DM2, peripheral neuropathy, HTN, gastric sleeve, four left toe amputations, and right hallux amputation presented for a worsening left dorsal foot wound. X-ray of left foot indicated soft tissue swelling and Charcot changes. Follow-up MRI revealed plantar ulceration with fluid collection as well as marrow changes through the mid/forefoot, osseous changes in the talus and calcaneus were also noted. The patient underwent a left metatarsophalangeal joint hardware extraction with incision and drainage as well as open bone sampling of the midfoot. The patient was started on IV vancomycin 1500mg every 12 hours, IV cefepime 2g every 12 hours, and IV metronidazole 500mg every 8 hours. Intraoperative cultures grew Corynebacterium striatum, Staphylococcus aureus, and Actinomyces radingae. A histopathologic diagnosis of acute osteomyelitis was made. The patient was discharged with home health services and an antibiotic regimen consisting of a course of IV vancomycin 1500mg every 24 hours and oral doxycycline 100mg every 12 hours for two weeks. After two weeks, the regimen was adjusted to oral doxycycline 100mg every 12 hours monotherapy for four weeks. Doxycycline was discontinued and oral amoxicillin 1g every 8 hours was initiated for six months.

Discussion/Conclusion

We found 44 cases of confirmed A. radingae infection in the medical literature. All 44 cases were soft tissue infections, located primarily in the upper body, abdomen, groin, or perianal area.1-10 This case of A. radingae is rare not only due to its location on the foot, but also for its involvement in osteomyelitis. Diabetes mellitus can lead to immunosuppression, a known risk factor for actinomycosis.11 In addition to immunosuppression, natural barrier damage from diabetic neuropathy can lead to increased skin and soft tissue infections in diabetic patients.12 When treating patients with diabetic foot infections, an empiric regimen covering Gram-positive cocci should be considered.13 Actinomycosis generally requires prolonged antibiotic therapy, historically 6-12 months, with first-line therapies including amoxicillin and penicillin G.7 This case highlights the importance of considering rare organisms when treating infections in diabetic patients.

Abstract #20 Seeing the Invisible: Nonconvulsive Status Epilepticus Presenting as Acute Mental Status Change

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Introduction

Nonconvulsive status epilepticus (NCSE) is defined as continuous seizure activity lacking gross motor convulsions, lasting longer than 30 minutes[1]. Because of its vague presentation, this condition frequently is underdiagnosed, leading to higher morbidity and mortality compared to the more easily recognized convulsive status epilepticus[1]. Because diagnosis relies heavily on clinical suspicion, early consideration of NCSE in differential diagnoses for acute mental status changes leads to earlier recognition of the condition and improves outcomes.

Case Description

A 30-year-old woman with PMH of unspecified grand mal seizure disorder and hyperlipidemia was brought to the ED for confusion and questionable seizure activity. On arrival, patient was somnolent, slow to respond, and oriented only to self. Prior to hospital arrival, patient had eye flickering and confusion, consistent with prior seizure auras, though without witnessed convulsions. She missed her Lamictal and Keppra doses earlier that day. She was hospitalized for seizures 18 months prior with tonic-clonic convulsions and urinary incontinence. Patient had a history of self-discontinuing antiepileptic drugs during pregnancy. On exam, she had no appreciable motor, sensory, or cranial nerve deficits, and no evidence of urinary incontinence or tongue lacerations. CT head revealed no acute intracranial pathology. The morning after admission, patient's nurse paged the primary team, stating that patient had become unable to follow commands and was no longer able to recognize her mother. STAT EEG was ordered, revealing "continuous generalized spike and wave discharges at 3Hz," suggesting epileptiform activity. Lumbar puncture had not yet been obtained given the recent admission. IV benzodiazepine was trialed. Neurology was emergently consulted and recommended continuous EEG monitoring and loading doses of Keppra for definitive diagnosis and treatment of NCSE. NCSE was aborted that evening. Patient's home Keppra dose was increased on discharge and close follow-up with neurology was arranged.

Discussion/Conclusion

While frank convulsions are definitionally absent in NCSE, symptoms can include ictal cognitive impairment, facial twitching, and eye deviations, as witnessed in this patient[2]. Furthermore, unlike generalized convulsive status epilepticus, NCSE's definitive diagnosis is only achieved on EEG and is estimated to make up 40% of all cases of status epilepticus[1,3]. Lastly, case series from recent years suggest that patients with NCSE with depressed consciousness or obtundation likely have higher morbidity and mortality compared with other presentations of NCSE[1]. Thus, patients like the one in this case likely will prove to have significantly improved outcomes with the early recognition of NCSE via EEG and treatment with a benzodiazepine.

Abstract #23 False Susceptibility: Inducible ESBL in Proteus Mirabilis During Beta-Lactam Therapy

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Introduction

Proteus mirabilis is a Gram-negative bacillus frequently associated with urinary tract infections, especially catheter-associated UTIs (CAUTIs). In recent years, multidrug-resistant P. mirabilis isolates, including those producing extended-spectrum-beta-lactamases (ESBL) are increasing. However, identifying ESBL production on routine sensitivity testing is not always possible, making treating these isolates a challenge.

Case Description

A 65-year-old male with a past medical history of spinal stenosis complicated by paraplegia, neurogenic bladder, chronic indwelling urinary catheter and recurrent UTIs presented from his nursing home with sudden-onset altered mental status and lower abdominal pain. The patient met sepsis criteria with urinalysis indicating a UTI. Urine and blood cultures were collected and vancomycin and Zosyn were initiated. By the next day, his leukocytosis and mentation had improved markedly. Of note, the patient had a similar presentation one month prior and was found to have a P. mirabilis CAUTI with urine sensitivity to amoxicillin/clavulanic acid, which he was prescribed at discharge and subsequently completed at his nursing home. During the present admission, his urine and both blood cultures demonstrated P. mirabilis with a similar resistance profile to the isolate from his previous admission. Infectious Disease was consulted for antimicrobial recommendations given a failure of outpatient therapy involving an antibiotic that was chosen based on sensitivity data. He was subsequently treated with gentamicin and a course of IV ertapenem which was completed at his nursing home.

Discussion/Conclusion

Despite treatment according to sensitivity data at his previous admission, the UTI persisted and extended to the blood with isolates having similar sensitivity profiles to the isolate from his previous admission. P. mirabilis is not commonly associated with AmpC ESBL production, however plasmid exchange of these genes has been noted. These organisms produce low levels of ESBL but higher levels can be “induced” when treated with beta-lactam therapy, resulting in levels that are too high for oral antibiotic therapy to overcome. This inducible ESBL production results in false sensitivities on routine testing prior to the administration of beta-lactams and would result in resistance upon treatment initiation. Treating these ESBL isolates is therefore challenging, since sensitivity results cannot always reliably guide therapy. This highlights the importance of educating prescribers on emerging bacterial resistance patterns and the inability to rely solely on culture sensitivities. In cases such as this patient’s, treatment with IV antibiotics not susceptible to ESBL will likely be required and should be considered with failure of outpatient oral therapy that was based on sensitivity data.

Abstract #27 Acquired Hemophilia a Secondary to Recalcitrant Bullous Pemphigoid

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Introduction

Acquired Hemophilia A (AHA) is a rare bleeding disorder, which occurs due to autoantibodies directed against Factor VIII, resulting in disruption of the coagulation cascade. Underlying causes of AHA include malignancy, autoimmune disease, pregnancy, and idiopathic (1). Bullous Pemphigoid (BP) is an autoimmune subepidermal blistering disorder with few reported cases associated with AHA (2). Management of AHA secondary to BP often involves immunosuppressive therapy (4). This report highlights a unique case of AHA secondary to recalcitrant BP successfully treated with emicizumab.

Case Description

A 72-year-old male with a past medical history of BP, treated with two completed tapers of oral prednisone, hyperlipidemia, type 2 diabetes, prostate cancer status-post radical retropubic prostatectomy and hypertension presents to the emergency department after a syncopal episode. His blood pressure was 98/55 with all other vital signs within normal limits. Physical examination was remarkable for bleeding from BP lesions on his face and oral mucosa. Patient's hemoglobin on admission was 10.4 but was remarkable for a drop to 6.4 g/dL the following day. Coagulation studies showed an elevated PTT of 83.4 seconds and a Factor VIII level of less than three. The patient showed no improvement in Factor VIII levels after treatment with Factor VIII, recombinant Factor VII, and methylprednisolone. Therefore, a factor eight-inhibitor bypass activity (FEIBA) was administered. The patient had minimal improvement in symptoms with a combination of first-line treatments including corticosteroids, cyclophosphamide, and rituximab. Therefore, emicizumab-kxwh was started. Recombinant Factor VIIa was weaned and FEIBA was discontinued in preparation for discharge as the patient's symptoms improved. On discharge, the patient continued prednisone and aminocaproic acid in addition to emicizumab-kxwh and cyclophosphamide infusions.

Discussion/Conclusion

Bullous Pemphigoid is a rare cause of acquired hemophilia A. Potential complications are similar to those with congenital factor deficiencies, but are a diagnostic challenge due to the absence of supporting history (3). The mainstay of treatment focuses on immunosuppression therapy along with the use of hemostatic agents to prevent clinically significant bleeding (4). This case is an example of a patient with BP-induced AHA who failed first-line treatment and underwent treatment with emicizumab. Emicizumab is a recombinant antibody that is designed to act as a mimic to Factor VIII allowing for the eventual activation of Factor X. Currently, emicizumab is approved for prophylaxis of bleeding congenital hemophilia A and hemophilia A with inhibitors. Long-term follow-up is recommended due to potential recurrence despite remission.

Abstract #32 Parakinesia Brachialis Oscitans: An Abnormal Case of Yawning in Stroke Medicine

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Introduction

Parakinesia brachialis oscitans (PBO) describes conditions of hemiplegia where spontaneous yawning causes random movement of the affected upper limb

Case Description

A 57 year old right-handed male presented to the hospital in hypertensive emergency with four and a half hours of right upper extremity weakness and slurred speech. Medical history included atrial fibrillation not on anticoagulation and hypertension. The initial CT head showed chronic microvascular changes and CTA showed no flow limiting stenosis. The MRI showed an acute left posterior capsule ischemic stroke consistent with a left lacunar ischemic infarct likely due to poorly controlled blood pressure. He was outside the thrombolytic window and conservatively managed. Forty-eight hours after presentation it was observed that while yawning he developed spontaneous hand clenching and right arm flexion with 5/5 strength that returned to flaccidity once the yawn was over. This phenomenon continued throughout the remainder of his hospitalization with resolution one month later.

Discussion/Conclusion

This finding was consistent with parakinesia brachialis oscitans which is a poorly understood and rarely documented finding in which a previous plegic limb develops involuntary movement at the onset of yawning and returns to a flaccid state at completion of the yawn. PBO appears to be associated with damage to the internal capsule with subsequent damage to corticospinal inhibitory pathways. This is hypothesized to releases subcortical structures associated with movement and frequent yawning which is a well documented finding in ischemic MCA strokes.
It is important that clinicians recognize PBO as a consequence of cerebrovascular accidents to avoid false assumptions of malingering and a delayed diagnosis of stroke. More importantly, increased awareness of PBO may encourage research into this phenomenon which could influence the development of treatment modalities that take advantage of the pathophysiology to restore motor function in previously plegic limbs following a stroke.

Abstract #41 Obscure Decompensating Neurologic Disease Clarified by Unifying Diagnosis of Leptomeningeal Carcinomatosis

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Introduction

Leptomeningeal carcinomatosis is characterized by infiltration of leptomeninges by metastatic carcinoma, most commonly breast adenocarcinoma.

Case Description

A 45-year-old female with a history of breast cancer status post chemotherapy, radiation, and bilateral mastectomy five years prior, on tamoxifen, was admitted to the general medicine service due to progressively worsening lower extremity weakness and new onset syncope. One year prior to presentation at our facility, the patient developed diplopia and leg weakness which progressively worsened resulting in confinement to a wheelchair and inability to continue her work as an ICU nurse. At that time, she was diagnosed with Chronic Inflammatory Demyelinating Polyneuropathy and underwent IVIG therapy with little improvement. Eight months later a different neurologist diagnosed Myasthenia Gravis and prescribed PLEX therapy, again with minimal improvement. During this period, MRI of the brain and spine were unrevealing and two lumbar punctures only showed mildly elevated protein. Evaluation by the inpatient neurology service at our facility revealed left cranial nerve III and VII palsy, bilateral internuclear ophthalmoplegia, and weakness of bilateral lower extremities with upper motor neuron signs. Neurologic findings were initially attributed to previously diagnosed conditions and syncope was attributed to orthostasis until she developed new onset papilledema and ophthalmoplegia during hospitalization prompting further work up. Extensive neuroimaging including MRI brain and MRI lumbar spine showed papilledema with possible fluid surrounding the optic nerves suspicious for carcinomatosis and diffuse abnormal enhancement of the surface of the spinal cord/conus and cauda equina concerning for carcinomatosis. Ensuing lumbar puncture showed protein greater than 2000 mg/100ml and 23 white blood cells. CSF cytology and flow cytometry were negative for malignancy. Due to clinical suspicion for carcinomatosis the patient underwent craniotomy for biopsy of the cavernous sinus. Pathology showed metastatic breast adenocarcinoma to the leptomeninges. Evaluation by the inpatient oncology team found no evidence of malignancy outside of the CNS on further imaging studies. Additionally, the patient had followed with oncology regularly with her last PET scan four months prior showing no active disease. The patient elected not to pursue treatment and unfortunately succumbed to her disease days after diagnosis.

Discussion/Conclusion

Tumor cells in CSF cytology or characteristic findings on MRI point towards the diagnosis of leptomeningeal carcinomatosis. However, CSF cytology and MRI imaging are only positive in 60% and 70% of patients respectively, resulting in diagnostic challenges.

Abstract #44 Asymptomatic HACEK Endocarditis of a Native Valve: Atypical presentation of a classic etiology

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Introduction

Cardiobacterium hominis is a member of the HACEK (Haemophilus, Aggregatibacter, Cardiobacterium, Eikenella, Kingella) group of microorganisms, which are responsible for only 0.5-6% of infective endocarditis (IE) cases. While the clinical course of C. hominis endocarditis is typically insidious in nature, most patients (up to 95%) are symptomatic. Like more common causes of IE, the majority of reported cases of C. hominis endocarditis involve underlying cardiac abnormalities such as prior valve replacement or known valvular disease; nearly all patients (up to 96%) present with a cardiac murmur.

Case Description

A 64-year-old male with well-controlled Type II Diabetes was admitted to the hospital for acute multi-territory cerebral infarction. Transthoracic echocardiogram showed trace aortic and mitral valve regurgitation; no embolic source was identified. No murmur or arrhythmia were noted. Hospital course was complicated by persistent asymptomatic leukocytosis. After discharge, blood cultures became positive for Penicillin-sensitive Cardiobacterium hominis. After outpatient follow-up and a second set of positive cultures, the patient completed a two-week course of trimethoprim-sulfamethoxazole. At a second follow-up office visit, a new diastolic murmur was noted on physical exam. A third set of cultures were collected, and he was referred for urgent transesophageal echocardiogram. The third set of cultures again were positive for C. hominis, and TEE demonstrated a new 1.4 cm vegetation on the aortic valve. Upon direct inpatient admission, he was found to be in asymptomatic atrial fibrillation via electrocardiogram. Aside from the murmur and atrial fibrillation, IE exam findings were absent. Review of systems was negative. Further history revealed the patient underwent a root canal 3 months prior to his first hospitalization, a potential source of C. hominis. Following initiation of intravenous ceftriaxone, he underwent open replacement of his aortic and mitral valves.

Discussion/Conclusion

HACEK organisms are a classic cause of infective endocarditis. This patient met the two major Duke criteria for diagnosis, but his presentation was unique in multiple ways. Asymptomatic IE, as seen in this patient, is exceedingly rare. Colonization occurred in the setting of a physiologically and anatomically normal valve, as demonstrated by his first echocardiogram. The infection progressed over just several weeks, as demonstrated by his imaging progression and cardiac murmur. It is unclear whether this patient's embolic stroke or development of atrial fibrillation were related to the infection; such associations have been reported in the literature, but no septic embolic phenomena were seen on imaging. Third-generation cephalosporins are the mainstay of treatment, with or without valve replacement.

Abstract #48 HHV8: Anti-Retrovirals You Must Take

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Introduction

Kaposi sarcoma is an AIDS defining illness that presents in patients with a CD4 under 200, and typically manifests in an extensive presentation involving the face, trunk, oral mucosa, and often visceral organs. The following case is a rare presentation with isolated lesions to the foot without diffuse cutaneous or visceral involvement.

Case Description

A 46 yo male with PMH significant for HTN and HIV presented to the hospital for RUE and RLE tingling and numbness, persistent headache for a week, accompanied by nausea, photophobia, sensitivity to sound, and difficulty with gait. Work up revealed an acute left medial ponto-medullary stroke. Of note, patient had been out of his HIV medications (efavirenz, emtricitabine, and tenofovir) for about a year due to loss of insurance. Upon presentation, patient also complained of growths on his RLE, tender to touch, that had been present for two months. They had started as ulcerations but had been gradually growing. These lesions were predominantly flesh colored, 1-3 cm in size, with a wart-like appearance, and present both on the dorsal and ventral surface of the extremity. X-ray imaging was ordered on the foot and showed extensive soft tissue swelling, concerning for cutaneous lesions secondary to neurofibromas. On further questioning, patient denied any other recent skin infections, opportunistic infections and/or family history of neurofibromas. Further labs revealed CRP of 1.1, CD4 count of 95 and HIV viral load of 197,387 copies/mL. Serology testing for Histoplasmosis, Cryptococcus, Blastomycosis, Coccidiomycosis, and Fungitell were negative. An excisional biopsy of patient's RLE lesions was done. Immunohistochemical staining demonstrated positivity with HHV8, diagnostic of Kaposi sarcoma. Pathology confirmed that the lesions were multiple Kaposi sarcomas. A CT chest/abdomen/pelvis was ordered to look for internal signs of sarcoma but only showed descending/proximal wall sigmoid wall thickening concerning for diverticulitis versus colon neoplasm. The patient was started back on antiretroviral therapy, bictegravir, emtricitabine & tenofovir alafenamide. Pt was discharged with close follow up with an HIV specialist, heme/onc, and with instructions to get regular colonoscopies. He was also discharged with trimethoprim/sulfamethoxazole to take for PJP prophylaxis.

Discussion/Conclusion

Kaposi sarcoma should be high on a physician's differential in HIV patients who have been off adequate antiretroviral treatment or have exceptionally low CD4 counts, i.e. under 200. In the lower extremity, Kaposi lesions can present similarly to lesions from diabetic wounds, peripheral vascular disease, bacillary angiomatosis, and even sporotrichosis, however, a biopsy with pathology analysis can reveal the diagnosis.

Abstract #49 Nasty Nivolumab: A Case of a Rare Skin Reaction

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Introduction

Medical providers continue to evaluate evidence of adverse reactions associated with novel immunotherapies that treat malignancies, including dermatologic reactions. Bullous pemphigoid is an uncommon skin condition that presents with thin walled, tense, fluid filled blisters on the surface of the skin. The following is a case report on a patient who developed bullous pemphigoid lesions secondary to his chemotherapy agent, nivolumab.

Case Description

An 82-year-old male with PMH of COPD, hypertension, lung adenocarcinoma with metastasis to the bone actively being treated with nivolumab (Opdivo), and tobacco use disorder presented to the hospital with complaints of a rash. The patient reported symptoms began one year ago, progressed in the past two months, with significant worsening the past week. The rash began on the palms and dorsum of the hands bilaterally, spread to the extremities, oral cavity, anterior/posterior thorax and finally to the abdomen, buttocks, and legs. The lesions were characterized as itchy and painful with friction. The patient denied recent illness, new medications, skin trauma, or exposures to insects or animals. He had recently been evaluated by a dermatologist who prescribed oral steroids which slightly improved the lesions. However, due to concern that their rash might be more serious such as Steven Johnson Syndrome, the patient was transferred to the hospital. Labs indicated WBC 8.5 (L) 3.44; RBC (L) 9.6; HGB (L) 30.3; MCV 88.1; Platelets 214; CRP (H) 4.6; ESR (H) 34. On exam, the lesions were round, erythematous, target-shaped papules as well as bullae ranging from one to five cm. Lesions were located on the palms, axillary region, shoulders, oral right pharyngeal arch mucosa, right upper thorax, buttocks, and right anterior thigh. The skin lesions were cultured and demonstrated normal skin flora. While lesion biopsy results were pending, we initiated Solu-Medrol IV 50mg daily, diphenhydramine 25mg PRN, and Acyclovir due to concern for bullous erythema multiforme secondary to HSV. Biopsy results indicated bullous pemphigoid. Considering medical history and symptom timeline, we suspected the patient's bullous pemphigoid rash was secondary to his chemotherapy agent, nivolumab. The patient was discharged on a tapered prednisone regimen, a high potency topical steroid cream, and plans for an outpatient follow up with his oncologist.

Discussion/Conclusion

PD-1/PD-L1 inhibitor induced bullous pemphigoid is a rare but serious complication of chemotherapy agents like nivolumab, even in patients on long term therapy. Patients with such reactions require quick recognition and treatment with systemic and topical steroids.

Abstract #58 Ketosis-Prone Diabetes in the Setting of COVID-19 Infection

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Introduction

Historically, diabetes has been classified into two categories: type I diabetes mellitus (T1D) or type II diabetes mellitus (T2D), but the emergence of 'atypical' forms of the disease has complicated this previously binary categorization. An increasing incidence of patients presenting in diabetic ketoacidosis (DKA) lacking the typical phenotype of T1D spurred the identification of syndromes of ketosis-prone diabetes (KPD). KPD progresses as an intricate combination of type 1 and type 2 symptomatology where some patients may regain beta cell functionality.
Because of increasing diversity and phenotypic presentations of the disease, research continues to emerge about the pathophysiology of KPD. Cases are classified as either unprovoked or provoked. Provoking factors include clinically significant stressful events such as trauma or infection, including the novel Coronavirus 19 disease (COVID-19).

Case Description

The patient is a 48-year-old male with no significant past medical history who presented to the hospital in DKA three weeks after contracting COVID-19. At presentation, the patient's c-peptide was low and his A1c was 12.5%. Tests for antibodies associated with T1D including Glutamic Acid Decarboxylase 65 (GAD65), Zinc Transporter T8 (ZnT8), and Islet Antigen-2 (IA-2) were negative. After stabilization, the patient was discharged from the hospital on 26 units of insulin. At follow-up one-week later patients home logs reported blood sugars between 113-128, so he was transitioned to 20U insulin daily. By one-month follow-up, the patient was reporting symptoms of hypoglycemia, so insulin was decreased to 10U. Three months later, insulin was discontinued, and his c-peptide measured within normal limits. To date, the patient is not taking any diabetic medications, and his most recent A1c is 5.7%.

Discussion/Conclusion

COVID-19 infection has been identified as a precipitating factor for development of DKA. This patient's presentation in DKA, antibody negative status, and return of beta cell functionality as evidenced by recovery of c-peptide level are suggestive of KPD. This presentation is unique in his near full recovery of beta cell function which more closely mimics an episode of unprovoked KPD. This case presents interesting evidence supporting the spectrum of disease severity and suggesting a lack of information about the pathophysiology of COVID-induced KPD. Further studies are needed to better elucidate the relationship between COVID and new-onset ketosis-prone diabetes.

Abstract #60 Probable Creutzfeldt-Jakob disease? Probably not.

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Introduction

Creutzfeldt-Jakob disease (CJD) is a rare fatal neurodegenerative disease with rapid progression. The definitive diagnosis is made postmortem with tissue sampling showing characteristic findings. However, there are antemortem tests to help determine the likelihood of CJD, including CSF analysis for the presence of protein 14-3-3 and Tau protein, and real-time quaking-induced conversion (RT-QuIC) to assess the presence of the abnormal prion protein. Early diagnosis is crucial, as many treatable diseases can mimic CJD.

Case Description

We present the case of a 46-year-old previously healthy female hospitalized for encephalopathy who rapidly deteriorated from the inability to perform ADLs (admission) to unresponsiveness with significant muscle rigidity and posturing, ultimately requiring intubation. Serial MRIs showed progression of deep white matter T2 signal abnormalities involving the occipital cortex and bilateral cerebral hemispheres, placing panencephalopathic CJD on the differential. Further workup included negative paraneoplastic panel in CSF and serum, normal autoimmune panels, negative malignancy screens, and negative extensive infectious workup. Multiple EEGs were performed, all with non-specific findings of diffuse slowing. Furthermore, CSF was sent to the National Prion Disease Pathology Surveillance Center (NPDPSC) and showed indeterminate RT-QuIC, elevated Tau protein, and positive 14-3-3 protein. The patient was diagnosed with probable CJD according to the CDC's 2018 Diagnostic Criteria for CJD, ultimately expiring eight weeks after admission. The body was sent to NPDPSC for autopsy, including immunohistochemical analysis and Western blot analysis. Immunostaining with 3F4, monoclonal antibody to the prion protein, failed to reveal granular deposits seen in CJD. Immunoblot failed to reveal the presence of abnormal protease-resistant prion protein, PrP 27-30, seen in CJD.

Discussion/Conclusion

Although the patient had positive CSF analysis for protein 14-3-3, definitive testing postmortem ruled out a diagnosis of CJD. Furthermore, antemortem tissue analysis was not pursued as the positive protein 14-3-3 already provided a diagnosis, and the risk of transmission of CJD during brain biopsy would be deemed too high. In conclusion, protein 14-3-3 has a high sensitivity (92%), but moderate specificity (80%). Further development of reliable antemortem testing, in addition to updated diagnostic criteria, needs to be pursued to adequately diagnosis or rule out CJD in patients with an unknown progressive dementia who may benefit from antemortem tissue analysis.

Abstract #62 Gaisbock Syndrome: A Rare Etiology of Secondary Hypertension

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Introduction

Gaisbock syndrome is an unusual clinical disorder first described in 1905 by Dr. Felix Gaisbock as hypertension in males attributed to elevated hematocrit levels without splenomegaly or abnormal leukocyte counts. The underlying pathophysiology for this syndrome was explained as psychiatric disorders that result in chronic stress, extracellular volume depletion due to diuresis, and hypoxemia due to obstructive sleep apnea as these conditions lead to relative and absolute polycythemias. We herein present a rare case of secondary hypertension attributed to Gaisbock syndrome.

Case Description

17-year-old Caucasian male without significant medical history was referred to nephrology clinic for new onset of hypertension with associated headache. He was initially seen by his family physician and evaluation revealed polycythemia with hemoglobin (Hb) of 20 g/dl, hematocrit of 57%, and hypertensive urgency with blood pressure of 160/110 mmHg. Physical exam was within normal limits with normal BMI. Secondary etiologies for hypertension evaluating for renal artery stenosis, Coarctation of the aorta, hyperaldosteronism, and Pheochromocytoma were all unrevealing. Hematological evaluation revealed negative JAK2 mutation and further genetic testing revealed a P50 mutation consistent with familial polycythemia. With other secondary causes ruled out, the patient's hypertension was attributed to Gaisbock syndrome due to familial polycythemia. The patient was initially treated with an Ace-inhibitor and Calcium channel blocker to control hypertension. Eventually one year after starting serial phlebotomy and attaining Hb of 15 -16 g/dl, the patient was able to come off antihypertensive medications and remained normotensive while continuing phlebotomy.

Discussion/Conclusion

While rare, Gaisbock syndrome is a known phenomenon due to increased intravascular pressure from relative and absolute polycythemia. It can be difficult to differentiate, if hypertension is a consequence or its own diagnosis in some cases. It is important to consider other causes of secondary hypertension especially in patients with atypical presentations, such as this patient who was only 17 years old at onset of hypertension. Notably, Gaisbock syndrome does not have one singular etiology and thus treatment strategies may vary from patient to patient. In our patient, phlebotomy has proven to be an effective treatment in controlling hypertension. It is important to control hypertension as hyper viscosity poses an increased risk for thromboembolic complications.

Abstract #67 Novel Use of Dupilumab: Patient with Post-Acute SARS-CoV-2 Sequelae and Severe-Persistent Asthma

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Introduction
Several patients who recover from acute SARS-CoV-2 infection have residual symptoms consistent with Post-Acute Sequelae of SARS-CoV-2 (PASC), including fatigue, shortness of breath, cough, dyspnea on exertion, and cognitive impairment. Dupilumab (Dupixent) is a monoclonal antibody which binds IL-4Ra and inhibits signaling of IL-4 and IL-13 receptors. It is currently FDA approved for treatment of uncontrolled moderate-to-severe atopic dermatitis, asthma, and chronic rhinosinusitis with nasal polyposis. We present a case of a patient with PASC and severe persistent asthma who had significant resolution of her PASC symptoms with Dupilumab.

Case Description
A 51-year-old female with a history of severe persistent asthma previously controlled with Trelegy Ellipta (Fluticasone furoate, Umeclidinium, and Vilanterol) 100 mcg once daily presented to the Allergy clinic with increasing shortness of breath, dyspnea on exertion, and fatigue following a hospital discharge for acute SARS-CoV-2 infection. On physical exam, she was found to be tachypneic with diminished breath sounds at the lung bases with no respiratory distress. EKG showed normal sinus rhythm. CT chest pulmonary embolism with contrast was consistent with COVID-19 pneumonitis and was negative for pulmonary embolism. Since her hospitalization, she had required multiple oral corticosteroid courses over two months with daily rescue albuterol use. Her Trelegy was increased to 200 mcg without significant benefit. She was then started on Dupilumab 300 mg every 2 weeks. At her 3-month follow-up, her fatigue was markedly reduced and had required her rescue albuterol only 4-5 times.

Discussion/Conclusion
Blood samples of COVID-19 patients have revealed elevated levels of several cytokines and interferons, including IL-4, having a direct effect on the inflammation and damage of respiratory tracts, thus aggravating asthma symptoms.
Given the significant clinical improvement shown in this case, further research warrants investigation into Dupilumab as a viable therapy for patients with PASC.

Abstract #70 A Trigger for a Zebra: Pregnancy-Associated Atypical Hemolytic Uremic Syndrome

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Introduction

Atypical hemolytic uremic syndrome is an extremely rare disease with high morbidity and mortality and is commonly triggered by pregnancy.

Case Description

We describe a 27-year-old G4P0212 at 37 weeks and 1 day gestation with a history of severe preeclampsia in a prior pregnancy. She was admitted for delivery due to a diagnosis of preeclampsia with severe features. She underwent a repeat cesarean section complicated by a postpartum hemorrhage necessitating multiple hemostatic modalities and blood transfusions. On post-operative day (POD) 0, urine output noted to be adequate, thus renal labs were not obtained. Between POD 1 and 2, her hypertensive persisted, renal function declined, and the patient became oliguric. Creatinine trended from 0.63 on admission to 3.31. Due to her oliguria and rising creatinine, Medicine and Nephrology teams consulted and hemodialysis was initiated. Additionally, her platelets trended from 148 on admission to 73, LDH was elevated and haptoglobin was low. These lab changes indicated microangiopathic hemolytic anemia. Hematology-Oncology was consulted and initiated steroids and plasmapheresis. Due to her persistent severe hypertension, Critical Care was consulted. While in the intensive care unit (ICU), ADAMTS13 activity (a marker typically decreased in Thrombotic Thrombocytopenic Purpura) was found to be normal, direct Coombs test negative, haptoglobin remained low, peripheral blood smear showed few schistocytes without platelet clumping, and complement was normal. Renal biopsy showed glomerular microangiopathic injury and tubular necrosis. A diagnosis of atypical hemolytic uremic syndrome (aHUS) was made. Her ICU admission was subsequently complicated by multiple cardiac arrests, however complete neurologic recovery was achieved. After discharge, she was started on Ecolizumab and remains hemodialysis dependent.

Discussion/Conclusion

This case signifies how the importance of identifying atypical hemolytic uremic syndrome in a pregnant patient. Most cases of aHUS are associated with mutations in genes that regulate the complement pathway. These mutations are typically not enough to cause aHUS without an environmental trigger. In this case, the trigger was likely pregnancy. Many confounding factors existed in this case leading to delayed diagnosis. For example, she suffered from preeclampsia with severe features and a postpartum hemorrhage which can also result in similar lab and clinical findings, however, our patient failed to improve after typical interventions leading to a search for an alternative diagnosis.

Abstract #71 Hyperphosphatemia, Limb Ischemia, and Hip Pain in a 52-Year-Old Male: Connecting the Dots

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Introduction

Hyperphosphatemic familial tumoral calcinosis (HFTC) is an autosomal recessive disease involving abnormal deposits of calcium in and under skin tissue around joints such as the hips, shoulders, and elbows. Resistance to a phosphate-regulating hormone known as fibroblast growth factor 23 (FGF23) results in hyperphosphatemia. Excess phosphate binds to calcium to form calcifications in soft tissues.

Case Description

A 52 y/o male with a past medical history of hypertension presented with multiple right thigh masses and left leg coolness. Physical exam revealed right thigh lateral and posterior masses and a non-palpable left dorsalis pedis pulse. The patient had hyperphosphatemia and high calcium. CT angiogram findings were consistent with peripheral arterial disease (PAD) of the left lower extremity. CT of the right hip revealed a calcified mass extending into multiple muscles. The work-up ruled out renal failure, hypoparathyroidism, rhabdomyolysis, tumor lysis syndrome, and multiple myeloma. Core biopsy findings revealed tumoral calcinosis. Nephrology was consulted and treated the patient with sevelamer.

Discussion/Conclusion

This case illustrates the importance of considering HFTC as a possible diagnosis for patients with hyperphosphatemia and PAD. Generally, patients with PAD have risk factors such as hypertension, hypercholesterolemia, obesity, diabetes, and a history of smoking. This patient only had one of these risk factors. Therefore, it is possible that HFTC involved vascular calcifications that contributed to his PAD. In addition, a diagnosis of HFTC may prompt patients and family members to undergo genetic testing and measurements of phosphorus levels, which can be used to identify those who would benefit from measures to reduce the chances of developing calcifications. Such measures include implementing a low-phosphate diet, using phosphate binders, and increasing renal excretion of phosphate. Limited clinical research has been performed on HFTC. Clinicians caring for patients with hyperphosphatemia should keep HFTC on the differential list and be vigilant for associated findings.

Abstract #72 Havoc Wreaked by Relentless Immunity: A Case of Neuropathy in a 45-Year-Old Male

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Introduction

Immune checkpoint inhibitors (ICI) such as nivolumab have revolutionized the treatment of metastatic renal cell carcinoma by improving overall survival across various trials. These drugs block certain checkpoints known as CTLA-4 and PDL-1 in an effort to increase the ability of the immune system to destroy malignant cells. Unfortunately, these same mechanisms can rarely cause adverse immune-related events wherein the immune system frequently targets the myelin sheath of motor nerves, causing peripheral neuropathy. Preliminary studies indicate that nivolumab carries a 1-5 % risk of peripheral neuropathy. We present here with a case of a patient with nivolumab-associated neuropathy.

Case Description

A 45-year-old male with a past medical history of metastatic papillary renal cell carcinoma presented as a direct admit to the medicine service for acute numbness in a stocking-glove distribution, bilateral lower extremity weakness with associated pain, and ataxic gait for a month. The patient had been wheelchair-bound for a week. These symptoms began a month after starting nivolumab. Physical exam revealed focal lower extremity hyporeflexia, 4/5 strength in the left lower extremity, diminished sensation to light touch from the knees distally, and absent vibratory sense in the lower extremities. MRI of the brain, thoracic spine, and lumbar spine were negative for acute abnormalities; there were no findings of new metastasis, myelopathy, or compressive radiculopathy. The work-up for peripheral neuropathy ruled out diabetes, hypothyroidism, and deficiencies in copper, vitamin E, and folic acid. Patient was found to be deficient in vitamin B12, so this was replaced. The primary team consulted Medical Oncology. Oncology discontinued nivolumab. The primary team also consulted Neurology. A lumbar puncture by Neurology was negative for malignant cells. Strength, numbness, and gait improved after the patient received prednisone, making Guillain-Barré syndrome less likely. Patient was diagnosed with immune-related peripheral neuropathy attributed to nivolumab.

Discussion/Conclusion

This case illustrates the importance of considering nivolumab as a diagnosis when managing patients with peripheral neuropathy. Symptoms such as numbness and lower extremity weakness are often attributed to causes such as lumbar nerve impingement, which is often treated with anticonvulsants. Conversely, this condition is treated with steroids. Prompt recognition of this condition can lead to earlier use of the appropriate drug, possibly mitigating neurologic toxicity, thereby improving quality of life. Data on the adverse effects of nivolumab have been limited to case reports. This study characterizes the spectrum of neurological adverse reactions associated with the use of nivolumab.

Obstetrics and Gynecology

Abstract #65 A Case of First Trimester Deep Vein Thrombosis Associated with Hyperemesis Gravidarum

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Introduction

Pregnancy infers a 5x increased risk of venous thromboembolism (VTE). Up to 40% of antenatal VTE occur in the first trimester, often prior to initiation of prenatal care. If thrombogenic risk factors are identified early, appropriate thromboprophylaxis can decrease associated complications of VTE.

Case Description

A 30-year-old G5P2022 at 9 weeks gestation presented to the hospital for intractable nausea, vomiting, weight loss, volume depletion, and ketonuria. A clinical diagnosis of hyperemesis gravidarum was made. She was given a bolus of lactated ringers in 5% dextrose and subsequently admitted for further administration of fluid maintenance and antiemetic therapy. She had minimal improvement in symptoms over the following 24 hours and was unable to advance diet from clear liquids. The following morning, she reported new onset tenderness in her left groin and lower extremity upon ambulation. Physical exam was notable for a positive Homan's sign and a difference in calf circumference of 8cm. Although pneumatic compression devices were ordered, they were not utilized overnight secondary to patient discomfort and inability to sleep with use.

Discussion/Conclusion

A STAT compression ultrasound was ordered and therapeutic low-molecular-weight heparin was given for presumed venous thrombus. Imaging showed an acute occlusive venous thrombus of the left common femoral vein, femoral vein, and popliteal vein with a superficial thrombus seen in the proximal portion of the great saphenous vein. Interventional cardiology was consulted. Thrombectomy was performed under ultrasound guidance, and 80-90% of the clot burden was removed. The patient was later discharged home in stable condition on intravenous antiemetics with plan to continue therapeutic low-molecular-weight heparin until six weeks postpartum. Anatomic and physiologic changes of pregnancy begin as early as the fourth week of pregnancy and Result in a prothrombotic state. Thrombotic risk is further increased by early pregnancy complications such as hyperemesis gravidarum. VTEs account for an estimated 10-15% of maternal deaths in high-resource countries. This case highlights the need for routine use of risk assessment tools in early gestation so that adequate thromboprophylaxis may be initiated to prevent maternal morbidity and mortality.

Pediatrics

Abstract #8 Small Bowel Volvulus Without Malrotation in Preterm Neonate

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Introduction

Small bowel volvulus is an emergent condition requiring immediate intervention to prevent necrosis and perforation of the bowel. Volvulus is most often associated with intestinal malrotation and fixation or other anatomic abnormality. There is concern that preterm infants are especially prone to volvulus and present with nonspecific symptoms.

Case Description

A preterm infant was born at 30 weeks and 1180g via repeat lower transverse C section to a G2P1001 mother with severe gestational hypertension. At delivery, the infant had multiple apnea episodes requiring intubation and a dose of surfactant.

On day of life (DOL) 3, abdominal distention and oral-gastric bilious aspirate without bowel sounds developed. Abdominal x-ray indicated mild gaseous distention. The infant was placed NPO and Salem sump was placed to low intermittent suction. Complete blood count, C-reactive protein, and blood culture were unremarkable. Ampicillin, gentamicin, and metronidazole were started for possible infectious ileus. On DOL 4-5, abdominal distention improved with faint bowel sounds and some stool output.

On DOL 6, there was dark green bilious aspirate (20mL) from the Salem-sump with worsening abdominal distention and agitation. Abdominal x-ray showed diffuse bowel distention with no signs of free air. Pediatric Surgery performed an exploratory laparotomy discovering a small bowel obstruction without anatomic abnormality secondary to distal ileal segmental volvulus with necrosis and perforation. The volvulus was reduced, 14 cm of distal small bowel were resected and left in discontinuity, with an abdominal washout, and a temporary abdominal silo was placed.

After exploratory laparotomy, the infant was fluid resuscitated and started on a 14-day course of piperacillin-tazobactam for abdominal necrosis and sepsis. Another surgery was performed on DOL 8 for abdominal washout, total colon insitu with ileocecal valve tied off (Hartmann's pouch) and creation of ileostomy. The infant was extubated on DOL 15 and weaned to nasal cannula on DOL 23. Nasogastric feedings were started on DOL 26 without issue.

Discussion/Conclusion

Most volvulus cases in infants involve malrotation or other anatomic abnormality; however, this case demonstrates a preterm neonate without an anatomic abnormality, highlighting the diverse presentation of volvulus in neonates. Preterm infants with volvulus show nonspecific signs including abdominal distention, bilious aspirate without emesis, and gaseous distention. Physicians should be aware of these signs of obstruction in preterm infants to prevent delay in diagnosis and decrease the risk for intestinal necrosis requiring partial bowel resection.

Abstract #17 A Nearly Fatal Blow: Commotio Cordis and Child Physical Abuse

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Introduction

Commotio cordis is a phenomenon in which sudden cardiac arrest occurs when blunt, non-penetrating trauma to the chest wall leads to cardiac dysrhythmia, particularly ventricular fibrillation. Though the occurrence of commotio cordis is very rare, there are documented cases in children participating in sports, and increasingly in settings of child abuse. This case highlights a child with sudden cardiac arrest secondary to an intentional blow to the chest wall inducing commotio cordis.

Case Description

A 23-month-old male was found unresponsive after a reported asthma exacerbation. Emergency services found him in ventricular fibrillation. After defibrillation and two doses of epinephrine, return of spontaneous circulation was achieved. Upon admission to the Pediatric Intensive Care Unit, extensive bruising was found. Physical exam was notable for bruising to his chest wall over his sternum, on the right side of his chest and mandible, and for abrasions on his ears, lips, right knee, and left axilla, inconsistent with the provided history of asthma exacerbation. Child Abuse Pediatrics was consulted for suspected abusive injury and neglect. Medical history was negative for recent trauma. Social history revealed previous child protective services involvement for human bite marks on the patient and for a reported incident of the father striking and shaking the patient. MRI brain showed hypoxic ischemic injury; additional workup including skeletal survey, echocardiogram, and dilated eye exam was negative. Child physical abuse and medical neglect were diagnosed based on the history and exam findings. The child's father later admitted to striking the child in the chest and stated afterwards the child immediately became unresponsive. After discussion with pediatric cardiology, the diagnosis of commotio cordis leading to pulseless electrical activity, a fatal condition without medical care, was made. He developed seizure-like activity, oral motor deficits, and global developmental delays due to his hypoxic ischemic brain injury, requiring additional medication and gastrostomy tube placement. He was hospitalized 34 days and discharged to a long-term medical rehabilitation facility and placed into kinship foster care. His father was criminally charged and arrested for child abuse.

Discussion/Conclusion

Commotio cordis is a difficult diagnosis to make, requiring a history of chest impact and subsequent cardiac arrest. The literature illustrates pediatric cases primarily with sports involvement. Clinicians must consider commotio cordis in cases of suspected child maltreatment and subsequent cardiac arrest, specifically when no plausible injury mechanism is provided. As no physical or imaging evidence exists to diagnose commotio cordis, a thorough history can be crucial.

Abstract #22 Serotonin Syndrome: A Delayed Diagnosis Due To Multiple Distractors

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Introduction

Serotonin syndrome is a potentially life-threatening syndrome that is most commonly precipitated by the use of serotonergic drugs and results from over-activation of both the peripheral and central postsynaptic 5HT-1A and 5HT-2A receptors. Increased levels of serotonin in the brain lead to symptoms such as abdominal pain, vomiting, hyperreflexia, sustained clonus, muscle twitching, high blood pressure, and agitation. We present a case highlighting the onset of serotonin syndrome symptoms after initiation of ondansetron, a 5-HT3 receptor antagonist in a patient on additional serotonergic medications.

Case Description

A 17-year-old sexually active female presented to the ED with complaints of nausea, vomiting, rash and abdominal pain. History included anxiety and depression treated with citalopram and trazodone, recent UTIs, chronic vaginal discharge, and rash due to promethazine administration at outlying facility despite history of documented allergy. Initial CT imaging showed two kidney stones and an incidental finding of transcolitis of the colon. Supportive care including ondansetron and IV fluids was initiated. Although the larger kidney stone passed spontaneously, she experienced worsening abdominal pain and emesis and developed new onset sustained ankle clonus and hyperreflexia prompting a STAT brain MRI which was normal. Intermittent hypertension was also noted which self-resolved. Extensive labs and imaging were performed due to acute onset of new symptoms which were only remarkable for moderate stool burden on KUB x-ray. Patient's nausea medication was changed to prochlorperazine, famotidine, and as needed calcium carbonate which did not reduce symptoms. Repeat CT showed resolution of transcolitis. Patient's symptoms worsened with increased vomiting and jitteriness. Given her clinical condition, serotonin syndrome was suspected. Per Psychiatry's recommendations, trazodone and citalopram were discontinued resulting in resolution of symptoms. Etiology of symptoms after kidney stone passage is still unclear, but likely due to serotonin syndrome triggered by acute stress of illness and recent introduction of ondansetron. Psychiatry recommended restarting citalopram, and having the patient follow up with PCP to restart the trazodone in a safe manner.

Discussion/Conclusion

Extraneous symptoms at presentation including second kidney stone and incidental finding of transcolitis combined with lack of specific diagnostic testing delayed the diagnosis of serotonin syndrome. Although there may not be vast evidence supporting the claim that 5-HT3 receptor antagonists have the ability to contribute to the development of serotonin syndrome, this case supports the phenomenon. Clinicians should keep this interaction in mind when classic symptoms such as hyperreflexia and hypertension are seen in previously healthy patients on serotonergic medications.

Abstract #34 New Onset Head Swelling in Infant: Harmless or Hazardous?

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Introduction

A subgaleal hemorrhage is a rare accumulation of blood between the galea aponeurotica and periosteum that covers a newborn's skull. It's typically caused during a vaginal delivery when tissue layers separate and small veins that pass through openings of the scalp rupture leading to blood accumulation. Subgaleal hemorrhages can be life-threatening as up to 40% of an infant's blood volume may be contained within the hemorrhage. They typically occur in the immediate newborn period and may take 2 to 3 weeks to resolve.

Case Description

A previously healthy 4-week-old 37-week gestation male presented to the pediatric emergency department (ED) with new onset swelling along the vertex of the skull. He was diagnosed with caput succedaneum and discharged home. Four days later he presented back to the ED with swelling extending to the occiput posteriorly which prompted hospital admission. Head ultrasound revealed posterior calvarial soft tissue swelling however unable to determine type of fluid in the collection. Skeletal survey to rule out potential non-accidental trauma was negative. MRI of the brain was negative for intracranial bleed but could not rule out subgaleal hemorrhage since the fluid collection crossed cranial suture lines. Neurosurgery aspirated the fluid for diagnostics and blood returned, which confirmed the diagnosis of subgaleal hemorrhage. Serial hemoglobin levels obtained to assess for extravascular blood sequestration and showed a downward trend requiring transfusion. The infant's head circumference and vitals remained stable over his 5 day hospitalization. Due to the delayed onset of the hemorrhage, coagulation factors and hemophilia labs were obtained and unremarkable. Further review of the patient's birth history revealed a traumatic birth with non-progressive labor and ultimately a C-section delivery. Newborn records were negative for scalp swelling in the immediate newborn period as would be expected for a subgaleal hemorrhage.

Discussion/Conclusion

Subgaleal hemorrhages are rare but potentially fatal birth complications, particularly in traumatic births and births requiring extraction tools. Signs and symptoms of subgaleal hemorrhages typically present in the immediate newborn period and rarely afterwards. Fatal complications of subgaleal hemorrhage include hypovolemic shock secondary to excess blood accumulation in the subgaleal space in addition to disseminated intravascular coagulation (DIC) in an effort to stop bleeding. This case highlights the rarity of a delayed subgaleal hemorrhage in infancy and the importance of accurate birth history and diagnosis of calvarial soft tissue swelling in order to delay potentially harmful, or even fatal, outcomes.

Abstract #38 Atenolol Use for the Management of Anxiety in a Patient with Asthma

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Introduction

Beta antagonists ("beta blockers") primarily treat cardiovascular diseases, but they may be used off-label for anxiety treatment, for example propranolol for performance-related anxiety. 2 Some beta blockers, like propranolol, are non-selective (binding to both beta-1 receptors in the heart and beta-2 receptors throughout the body), while some like atenolol1 are selective and bind to just beta-1 receptors in the heart. In patients with asthma, non-selective beta blockers have tolerability concerns since they can cause bronchoconstriction. Research shows cardioselective blockers, given for up to a few weeks, did not significantly worsen pulmonary function or respiratory symptoms, nor lead to increased use of inhalers in patients with mild to moderate reactive (reversible) airway disease.3 Furthermore, a study found that 81% of patients diagnosed with anxiety disorders reported positive effects with atenolol treatment.4 This case adds to the limited research that has analyzed the effects of atenolol use for the treatment of anxiety in patients with asthma.

Case Description

A 21-year-old male with a medical history of asthma and cannabis use was admitted to an inpatient psychiatric hospital for psychotic symptoms and was started on risperidone. While in the hospital, the patient experienced episodes of heightened anxiety and elevated blood pressure. Propranolol was avoided to prevent bronchoconstriction and informed consented was obtained to start oral atenolol for anxiety. Burns Anxiety Inventory questionnaires were given prior to atenolol use (score 35) and days after consistent atenolol use (score 37). Patient experienced heightened anxiousness at times and received hydroxyzine to acutely decrease anxiety. Patient verbally expressed decreased anxiety and physically demonstrated decreased restlessness after taking daily atenolol.

Discussion/Conclusion

While studies have shown propranolol's role in treating performance-related anxiety, limited research has examined effects of atenolol for anxiety. Although our patient verbally expressed decreased anxiety after taking atenolol, the Burns Anxiety Inventory results demonstrated mild worsening of anxiety symptoms. Thus, results from this case of atenolol use for anxiety are inconclusive. Limitations to this study include lower dosage used than the positive effects observational study of at least 100mg daily4, use of intermittent hydroxyzine when anxiety symptoms worsened, and limited number of Burns Anxiety Inventory results.
Although inconclusive, this case report demonstrates the potential benefit that atenolol may have in treating anxiety in patients with asthma. Further research should be done to assess the effectiveness of atenolol monotherapy or adjunctive therapy at therapeutic levels in treating anxiety in patients with mild to moderate asthma and COPD.

Abstract #40 Brexipiprazole Toxicity: A Case of Unintentional Ingestion

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Introduction

Brexpiprazole is a new atypical antipsychotic used to relieve symptoms of schizophrenia and depression by partially stimulating serotonin (5-HT1A) and dopamine (D2) receptors and blocking other serotonin (5-HT2A) receptors. It reaches peak serum concentration in 4 hours and has a half-life of 91 hours. It is metabolized by CYP2D6 and CYP3A4 and is ultimately excreted in the feces and urine. Little is known regarding brexpiprazole toxicity secondary to accidental ingestion in pediatric patients making care difficult for emergency care teams, poison control, and inpatient care teams.

Case Description

A 5-year-old male presented to the OU Health Fostering Hope Pediatric Clinic of Tulsa after developing difficulty walking and speaking, drooling, and an altered mental status. The previous day, he had been taken to the emergency department after ingesting 5 to 8 tablets of his father's 2mg brexpiprazole. He reportedly had spent the weekend at his father's residence where he took the medication. His mother took him to the local emergency department where poison control was consulted and was observed for 6 hours where he remained asymptomatic with normal CBC, BMP, and vital signs before being discharged. Upon physical exam in clinic, he had facial flushing, was crying, drooling, and holding out his tongue. The treating doctor consulted the clinic pharmacist and direct admission to the hospital for observation was recommended due to possibility of delayed symptoms of toxic ingestion.

Upon admission, he was afebrile with stable vital signs, but notably somnolent with slow speech and drooling. He had increased tone and a diffuse maculopapular rash on his face, chest, and extremities. Creatine kinase, glucose, BMP, liver transaminases, and CBC were all normal, but EKG showed QTc prolongation (>480 msec). He was given a fluid bolus but required no other intervention. Forty-eight hours after admission he returned to neurological baseline and was discharged.

Discussion/Conclusion

This case illustrates three important takeaway points about brexpiprazole toxicity. First, symptoms may have a delayed onset and a 6-hour observation time after ingestion might be inadequate to rule out toxicity. Patients should be monitored for possible toxic effects of atypical antipsychotics, including altered mental status, QTc prolongation, seizures, hypoglycemia, and rhabdomyolysis for 3-5 days post ingestion. Symptoms will typically resolve with only supportive care.

Abstract #42 Seizure-Like Activity as an Initial Presentation of Suspected Hypopituitarism

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Introduction

Hypopituitarism is defined as a deficiency of one or more of the hormones secreted by the pituitary gland. An underactive pituitary gland can result in metabolic imbalances leading to a lack of cortisol. A severe cortisol-deficiency may lead to severely low glucose levels, presenting as seizure-like activity. This can be exacerbated in the presence of underlying of underlying viral or bacterial infections.

Case Description

4-year-old with known septo-optic nerve hypoplasia, congenital hypothyroidism, infantile spasms, global developmental delay, deafness, and epilepsy presented to the emergency department. Medications prior to admission included valproic acid, topiramate, and levothyroxine. Her initial presentation consisted of waxing and waning levels of consciousness with a glucose of 26mg/dL on arrival. On admission, she was febrile with a Tmax of 39.2C. The primary concern was for an infectious etiology; prompting a viral respiratory panel and lumbar puncture which were both unremarkable. Urine and blood workup were also unremarkable. Inflammatory markers, COVID-Ab, urine drug screen, CT head, and thyroid studies were all unremarkable. An EEG showed no seizure-activity. Valproic acid and topiramate levels were within normal limits. MRI brain was unremarkable with exception of an absent septum pellucidum. We further expanded our differential to include concerns of hypopituitarism in a child with septo-optic dysplasia (optic nerve hypoplasia, absent septum pellucidum, and pituitary gland dysfunction). Notably, the child was 5th percentile for height and 0.5th percentile for weight. A morning cortisol (<1) and adrenocorticotropic hormone, or ACTH (<1), were obtained and concerning for secondary or tertiary adrenal insufficiency. Pediatric endocrinology recommended starting hydrocortisone with significant clinical improvement. Additionally, her IGF-1 was <10. Growth hormone deficiency is also suspected but an outpatient stimulation test is indicated to confirm the diagnosis.

Discussion/Conclusion

Hypopituitarism is commonly seen in children with mid-line defects with wide-ranging manifestations. Additionally, the differential of encephalopathy with seizure activity elicits a wide differential diagnosis. Decreased ACTH secretion is a crucial component of the hypothalamic-pituitary-adrenal axis, which can lead to imbalances in cortisol and ultimately disruption of homeostasis in times of physiologic stress. Seizure-activity and altered mental status are not classically thought of being involved in the initial presentation of adrenal insufficiency, but should be considered in the differential, especially in patients with midline defects.

Abstract #50 Polymicrogyria

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Introduction

Polymicrogyria (PMG) is described as a malformation of the cortex secondary to abnormal development in utero. It is characterized as an excessive number of small, irregularly shaped, abnormally fused gyri with shallow sulci. The etiology is not fully understood- possible hypotheses include multiple genetic and chromosomal abnormalities, intrauterine cytomegalovirus, neurological focal calcifications, infarction and/or viral inclusion. The incidence of polymicrogyria has not been identified. However, through the advancements of imaging techniques, there has been an increase in proper diagnosing which has led to a deeper understanding of the condition.
Clinical manifestation and severity of polymicrogyria is dependent on the location and number of abnormally folded gyri. It can be described as either focal, diffuse, bilateral, or unilateral. Thus, symptoms can range from mild cognitive impairment (speech and motor delay) with little to no epileptic activity to severe encephalopathies and drug-resistant intractable epilepsy.

Case Description

The patient is a 3-year-old female with intractable drug resistant epilepsy who returns to her primary care office for follow up. During the 15-minute encounter she has seven episodes of absence seizures followed by a postictal state. She has moderate cognitive dysfunction with a developmental age of approximately 18 months. She is currently on Keppra 1000 mg daily and Clonazepam 1 mg daily. Prior MRI which was completed at 18 months of age was significant for polymicrogyria. She was currently awaiting pediatric neurology to determine if she is a candidate for lobar resection.

Discussion/Conclusion

A diagnosis of polymicrogyria must be made by brain imaging. It is preferred that a T1 weighted MRI be used which will show an abnormally thick cortex with multiple small and abnormally fused gyri. Early recognition is imperative as it leads to early interventional treatment such as antiepileptics, physical therapy, occupational therapy, speech therapy, and surgical interventions such as vagal nerve simulation or lobar resection. Furthermore, due to the high risk of developing intractable epileptic seizures, close monitoring at an early age is warranted. While there is no cure for polymicrogyria, providers with adequate knowledge on the disease process and treatment plan can improve patient's quality of life.

Abstract #51 Severe Childhood Obesity Due to Neglect in Prader-Willi Syndrome

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Introduction

Prader-Willi Syndrome (PWS) is characterized by hypotonia and hyperphagia. Patients with PWS are prone to failure to thrive diagnoses as infants and obesity as they age. Neglect occurs when the needs of a child are not adequately met. Neglect is considered in cases of failure to thrive and malnutrition if children are not receiving appropriate nutrition. Neglect is also considered when patients with complex medical diagnoses do not receive adequate medical treatment. A population in which nutritional and medical neglect should be considered is pediatric patients with severe obesity given the medical complexity of obesity-related comorbidities and increased risk of mortality.

Case Description

An 11-year-old male with PWS weighing 165 kg was admitted to the pediatric intensive care unit with hypoxia, chest pain, edema, and severe obesity. Subsequent evaluation and consultation with multiple pediatric subspecialists led to diagnoses of heart failure, obstructive sleep apnea, pulmonary hypertension, and fatty liver disease. Respiratory support was required. Child Abuse Pediatrics was consulted due to neglect concerns. His parents reported a restricted calorie diet at home and described him as “lazy” and unable to get his own food due to becoming tired. He previously received lisdexamfetamine for appetite suppression, which had not been refilled for several months. Extensive medical record review indicated obesity for a significant portion of his life. Referrals to endocrinology had been made 4 years and 1 year prior to presentation, with no follow-up. Hospital documentation included parents providing extra food despite being advised otherwise. Based on the history and exam, nutritional and medical neglect were diagnosed. Weight management through a multidisciplinary approach was recommended, and a referral to child protective services was made. Unfortunately, his clinical status worsened over the following two weeks; he ultimately expired from multi-organ failure.

Discussion/Conclusion

The most frequent cause of death for patients with PWS is respiratory failure. The diagnosis of neglect is typically multifactorial. With severe obesity, this includes the presence of comorbid conditions and the risk they pose for serious impending harm. If caregivers are provided appropriate education, a reasonable treatment plan, and resources to address severe obesity, but fail to meet the nutritional and medical needs of the child, this constitutes neglect. This case highlights the need for further monitoring of children with medical complexity related to severe obesity to make sure appropriate follow-up is completed with specialists. Early intervention may reduce morbidity and mortality of these patients.

Abstract #52 Baylisacaris Procyonis: A Rare Cause of Eosinophilic Meningoencephalitis in Children

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Introduction

Baylisascaris procyonis is a nematode that colonizes raccoons resulting in human infection via accidental ingestion of egg infested feces. Larvae migrate from the intestine to the CNS potentially causing significant inflammation. Infections in humans are exceedingly rare, with most occurring in children due to their propensity for geophagia. This case describes a toddler who developed severe meningoencephalitis secondary to neural larva migrans from Baylisasaris procyonis.

Case Description

A previously healthy 17-month-old male from rural Oklahoma presented to the ER with concerns of decreased activity, irritability, and anorexia. He was found to be positive for rhino/enterovirus and a second virus by PCR. His initial CBC was most notable for leukocytosis (30k) with an elevated eosinophil count (23%, 6.9). Given the irritability, fever, and leukocytosis a lumbar puncture was performed showing CSF pleocytosis (6) with 16% eosinophils as well as an opening pressure of 37 cm H2O. Clinically, he developed encephalopathy and diffuse hypotonia with intact reflexes. MRI of the brain and total spine, CSF culture, and BioFire PCR testing were unremarkable. EEG showed diffuse cerebral dysfunction, but no seizure-like activity. A dilated ophthalmological exam was performed and revealed mild papilledema with retinal pallor but no larvae. Serial CBCs showed persistent leukocytosis and eosinophilia (up to 12.4k absolute). Due to his persistently altered state, neurologic findings, and lab abnormalities, the evaluation then focused on oncologic, autoimmune, and infectious causes including uncommon viral, fungal, parasitic, and bacterial etiologies. He was transferred to a different facility where he developed marked diffuse hypertonia and expanded testing ultimately confirmed baylisascaris infection. Prior to confirmation of the parasitic infection, empiric albendazole and corticosteroid therapy were started yielding lab improvement without dramatic clinical change. Once the patient was stabilized, he was transferred to an inpatient rehabilitation facility for intensive therapy to help improve his neurologic outcome.

Discussion/Conclusion

Baylisascaris procyonis is a rare cause of eosinophilic meningoencephalitis and can be devastating or fatal in children. Treatment of the infection is typically an anti-helminthic and corticosteroids. Early diagnosis is crucial as prompt treatment is thought to be associated with better prognosis. However, given the scarcity of infection it is unclear if treatment improves outcomes. This case highlights the potential diagnostic complexity when a child presents with altered mental status and hypertonia.

Abstract #53 Osteogenesis Imperfecta: A Case Study

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Introduction

Osteogenesis imperfecta is a rare connective tissue disorder with an incidence of around 1 in 15,000-20,000. This disease affects type 1 collagen leading to bone fragility and deformity. Common physical exam findings include excess fractures, short stature, scoliosis or kyphosis, blue sclera, hearing loss, abnormal teeth development with discoloration, ligamentous laxity, hypermobility, and bone abnormalities. Due to presentation, child abuse, osteomalacia, and rickets are common conditions often considered in the differential diagnosis for osteogenesis imperfecta.

Case Description

A fussy 2-month-old female was brought to the emergency room by her parents after they discovered lower extremity swelling since picking her up from daycare. Imaging showed a femur fracture, 5th and 6th rib fractures and bilateral chronic subdural hematomas. After consulting a child abuse physician, these injuries were diagnosed as non-accidental trauma. The Department of Human Services took custody of the child and placed her into a foster home. At 10 months old the child experienced another femur fracture and was brought back to the hospital for more testing. Similarly to the last visit, non-accidental trauma was suspected since the patient had previously suffered these types of injuries after returning from daycare. Further imaging indicated severe osteopenia in addition to the fracture. A metabolic workup was started in the inpatient setting and follow-up with genetics was scheduled. Initial labs showed a normal calcium, parathyroid hormone and vitamin D level. Social work and DHS were consulted and would continue to follow outpatient. Given that the status of her bones and the severe osteopenia was the likely cause of fracture, the patient was discharged with her parents with close outpatient follow-up with the child's PCP, orthopedics, child abuse and genetics. At the age of 18 months, she was diagnosed with osteogenesis imperfecta. She is currently receiving pamidronate and her care is managed by a multidisciplinary team.

Discussion/Conclusion

Due to the increased incidence of atypical fractures in children with osteogenesis imperfecta, children with the disease can be misdiagnosed with abuse. This case study demonstrates the importance of timely recognition of clinical features and appropriate workup for osteogenesis imperfecta.

Abstract #54 Hemodynamically Stable Fascicular Ventricular Tachycardia in a 2-week-old Infant

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Introduction

Fascicular Ventricular Tachycardia (also known as verapamil sensitive ventricular tachycardia) is a rare tachyarrhythmia in children characterized by a wide complex tachycardia with associated right bundle branch block and superior or right axis deviation. This tachyarrhythmia is important to distinguish from other forms of tachycardia as the treatment is vastly different and uncommon in the pediatric population.

Case Description

A previously healthy 2-week-old male, born at 39 weeks gestation without complication, presented to the OU-Tulsa Pediatric Clinic for his 2-week-old well child visit. He was found to be feeding well without tachypnea, diaphoresis, or cyanosis and had surpassed his birth weight. Review of his vital signs revealed tachycardia into the 180s. Physical exam revealed a persistent tachycardia that did not vary with respiration or with calming of the infant and a prominent S2 was also heard. An urgent echocardiogram was obtained given the concern for structural heart disease. Results revealed a structurally normal heart with hyperdynamic function. Due to concerns of abnormal heart rhythm demonstrated on echocardiogram, an ECG was obtained that revealed a wide complex tachycardia concerning for ventricular tachycardia. The infant was then referred to pediatric electrophysiology for definitive management of ventricular tachycardia. Upon evaluation in the PICU, the ventricular tachycardia was deemed to be fascicular ventricular tachycardia and verapamil infusion was started with resolution of the tachycardia. Once the infant stabilized with intravenous verapamil, he was transitioned to oral daily verapamil with continued control of the tachycardia.

Discussion/Conclusion

Fascicular ventricular tachycardia in infants and children is hard to identify as it is quite rare in this population. Verapamil is contraindicated in infants due to the risk of cardiovascular collapse and death in infants when given via intravenous push. However, in this subset of ventricular tachycardia, verapamil is the only therapy that will treat the arrhythmia. Correct identification and treatment of this arrhythmia is imperative as cardiac failure will occur if left untreated. This case highlights correct identification and prompt treatment in a neonate with fascicular ventricular tachycardia and emphasizes the importance of close attention to the cardiovascular exam in all well child visits.

Psychiatry

Abstract #26 Citalopram Induced REM Sleep Behavior Disorder

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Introduction

Rapid eye movement (REM) sleep behavior disorder (RBD) is a parasomnia characterized by the loss of skeletal muscle paralysis that normally occurs during REM sleep. RBD ranges in severity from subtle hand movements to violent thrashing, punching and kicking. Patients typically seek medical attention with concerns for injury or possible injury to themselves and/or their bed partner. While there are many possible etiologies for RBD, it is commonly associated with selective serotonin reuptake inhibitors (SSRIs), the mainstay treatment for mood and anxiety disorders. Here we examine a clinical case of RBD that manifested from citalopram use.

Case Description

A 71 year-old female with a history of generalized anxiety disorder initially presented to primary care with abnormal sleep behaviors. During the night, patient reported episodes including shouting, thrashing and jumping out of bed. Her episodes became so severe her husband was physically harmed, and they needed to sleep separately. A sleep specialist reviewed her symptomatology, and a polysomnography was performed. The sleep study displayed elevation of motor tone during REM sleep by electromyography, thus confirming RBD. It was determined she was suffering from RBD caused by citalopram, which had been managing her anxiety symptoms for the past several years. After discontinuing citalopram and over the course of the next few months, her symptoms began to slowly diminish. Though patient's RBD improved, her anxiety symptoms soon returned, and so, she was referred to our psychiatry clinic for alternative treatment recommendations.

Discussion/Conclusion

A significant portion of medications utilized to treat mood and anxiety disorders, including SSRIs, SNRIs and TCAS, have serotonergic activity, which is thought to precipitate RBD. This is plausible considering serotonin is well known to promote wakefulness and reduce REM sleep. Although RBD is rare side effect amongst patients utilizing SSRIs (prevalence of 0.48%), clinicians must consider RBD as an adverse effect considering SSRIs are known to be common culprits of RBD.

Abstract #35 Treatment of Forced Normalization Psychosis in Lennox–Gastaut Syndrome

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Introduction

Lennox-Gastaut syndrome (LGS) is a severe form of epilepsy that begins in early childhood. LGS is characterized by epileptic seizures, psychomotor delay, behavior disorders and characteristic EEG findings. It's most common in males with onset before 8 years old. Language and motor regression frequently occur as well as severe behavioral disorders. Long-term prognosis is poor with 85-90% of patients experiencing seizures in adulthood despite adequate treatment. Forced Normalization (FN) describes the emergence of psychiatric symptoms following seizure control or reduction in epileptiform activity on EEG in a patient with uncontrolled epilepsy. Most literature supports a trial with an antipsychotic but information regarding the treatment of psychosis secondary to forced normalization is sparse with mixed outcomes. Here we discuss a complex treatment case of LGS in which FN psychosis developed after vagus nerve stimulation was decreased.

Case Description

Ms. L is an 18-year-old female with LGS who developed delusions, hallucinations, and disorganized behavior in 2020. She began having intractable epileptic seizures in 2006, at age 3. Her seizures progressed, resulting in functional impairment despite medication management. In 2014, a vagal nerve stimulator (VNS) placed that normalized her EEG and provided a few months of seizure control. In 2018, her VNS was replaced after causing shoulder pain. Due to symptoms returning three months later, her VNS frequency was reduced in January 2020. In February, patient presented to ER with delusions, hallucinations, and disorganized behavior. She has since trialed several antipsychotic medications (Risperidone, Aripiprazole, Olanzapine, Lumateperone and Haloperidol) at max dose as well as in combination. Regardless of treatment, patient had no change in symptoms. Clozapine was considered but her history of LGS was viewed as a contraindication due to lowering seizure threshold.

Discussion/Conclusion

Lennox-Gastaut's syndrome is a rare pediatric seizure disorder that can be associated with forced normalization. There are no established guidelines to treating the associated psychosis. Of the available research, forced normalization psychosis typically occurs after starting, compared to lowering or discontinuing, an anti-epileptic treatment. Furthermore, forced normalization is an umbrella term for any psychiatric concern after stabilization including anxiety, mood, and behavioral concerns. These discrepancies further complicate treatment research as effective treatments for psychosis can differ from mood or behavioral concerns. This case demonstrates the complexity and heterogeneity in the development and treatment of psychotic disorders.

Surgery

Abstract #12 Popliteal Artery Aneurysm Repaired with RGSV Bypass and Exclusion of Popliteal Artery

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Introduction

Popliteal artery aneurysms are known as “silent killers” as they can rupture causing uncontrolled bleeding or they can form blood clots that can thrombose or embolize, causing limb loss. Even after open surgical intervention with GSV bypass, graft aneurysm or occlusion may occur. We present a case of a patient treated with right great saphenous vein (rGSV) bypass, presenting with a popliteal aneurysm.

Case Description

A 51-year-old male with hypertension, hyperlipidemia, hypothyroidism, and a never-smoker with a family history of ruptured abdominal aortic aneurysm (mother) presents to the emergency department with right leg/calf pain that began the day prior while driving his truck. Physical examination was unremarkable and pedal pulses were palpable. Computed tomography angiogram (CTA) was performed and noted multifocal aneurysmal dilation of the right popliteal artery, measuring up to 1.7 cm. The popliteal artery aneurysm was subsequently repaired with right great saphenous vein bypass and exclusion of the popliteal artery.

Discussion/Conclusion

Popliteal artery aneurysms are the most common form of peripheral arterial aneurysms. Anatomically, the popliteal artery is a branch of the femoral artery and is located in the region of the knee and lower leg. Popliteal artery aneurysms can cause significant morbidity and mortality by causing complications, such as severe aneurysmal rupture or blood clots (thrombosis) that lead to ischemia of the lower limb, and potentially loss of the limb. There are multiple types of popliteal artery aneurysm management procedures, that include an open medial approach, an open posterior approach, or an endovascular approach; however, with longer duration of vascular patency and lower recurrence of aneurysm, open surgical approach continues to be the gold standard for popliteal artery aneurysm repair. In terms of an open surgical approach, the great saphenous vein can be used as a graft to bypass the popliteal artery aneurysm. Bypass and exclusion of an aneurysmal popliteal artery can prevent life threatening, uncontrolled bleeding from aneurysmal rupture, thus improving patient morbidity and mortality.

Abstract #14 Thoracic Aortic Dissection Repaired with Tevar and Retrograde Laser Fenestration

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Introduction

Aortic disease (dissection and aneurysm) can extend from thoracic to abdominal aorta which may require treatment. Even after open surgical intervention, aortic degeneration and propagation of dissection and aneurysm may occur. We present a case of patient treated with thoracic endovascular aortic aneurysm repair (TEVAR) for dissection and aneurysmal disease, presenting with chronic intermittent back/chest pain.

Case Description

A 70 year-old male with hypertension, hyperlipidemia, previous tobacco smoking, history of ascending aortic dissection status post replacement with Dacron graft presents with intermittent chest tightness, back/chest pain, and chronic fatigue and was evaluated for complicated chronic aortic dissection with aneurysmal degeneration. Upon examination, no postprandial pain or GI bleed was noted and there were palpable radial & femoral arteries bilaterally, Right DP/PT pulses were palpable, and left DP/PT pulses was biphasic. Computed tomography angiogram (CTA) was performed and noted redemonstration of chronic type A aortic dissection status post repair with aneurysmal dilation of the aortic isthmus up to 5cm. The thoracic aortic dissection was subsequently repaired with Thoracic Endovascular Aortic Aneurysm Repair (TEVAR) with retrograde laser fenestration and the left subclavian artery was stented.

Discussion/Conclusion

There are two common types of classifications of aortic dissections: Stanford Type A and Stanford Type B aortic dissections. Stanford Type A aortic dissections are any aortic dissection that involves the ascending aorta and urgent open surgical repair of the ascending aorta is recommended. Stanford Type B aortic dissection is confined to the descending aorta. Medical management with blood pressure control is the mainstay treatment for Stanford Type B and surgical intervention is reserved for complicated aortic dissections, including aneurysmal degeneration or malperfusion. In addition to open surgical procedures, thoracic endovascular aortic aneurysm repair (TEVAR) can be carried out and it carries the advantage of the absence of a thoracotomy scar, reduced need for mechanical circulatory support, and no requirement of aortic cross clamping, thereby offering a resultant reduction in overall hospital morbidity, mortality, and length of stay. In-situ retrograde laser fenestration can be used in conjunction with the TEVAR procedure and it uses light energy to put holes in a graft to allow for dilation and stenting.

In conclusion, in-situ retrograde laser fenestration is a quick and effective method for revascularizing aortic arch branches after thoracic endovascular aneurysmal repair (TEVAR). Initial morbidity and mortality is decreased with TEVAR, and thus makes it an important method for dissection and aneurysmal repair.

Abstract #28 The Challenges of Identifying Pyoderma Gangrenosum

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Introduction

Pyoderma Gangrenosum (PG) is a rare disease that affects an estimated three to ten cases per million. The etiology and pathogenesis of PG remain poorly understood, causing it to be commonly misdiagnosed. The clinical presentation has overlapping features with many other diseases, and there is currently no gold standard criteria for diagnosis. Therefore, PG is typically a diagnosis of exclusion, often leading to prolonged hospitalizations, treatment, and recovery.

Case Description

A 23-year-old male with a history of multiple rectal abscesses requiring hospitalization for incision and drainage presented with tachycardia and severe pain in his rectal area. On physical exam, the patient had perianal erythema and induration, a right gluteal wound, purulent drainage from the perianal wound, and severe tenderness. The patient was diagnosed with Crohn's disease and admitted for incision and drainage of recurrent perirectal abscesses. From January 2021 through January 2022, he underwent multiple surgeries for complications from uncontrolled Crohn's disease. He developed complicated perianal and ischiorectal wounds, posterior rectocutaneous fistulas, and coccygeal osteomyelitis. He was treated with broad-spectrum antibiotics and incision and drainage that was complicated by repeated ostomy and wound breakdown, including a stage 4 sacral decubitus ulcer. PG was identified as a diagnosis of exclusion after one year of hospitalization. Cyclosporine was administered and wound healing began. Unfortunately, this patient routinely refused wound care, physical therapy, and physical examinations from his providers. Upon discharge, he was set up with home health, but his prognosis is poor compared to the average PG patient given his secondary disorders, poor treatment compliance, socioeconomic factors, and the severity of his disease.

Discussion/Conclusion

The 2018 diagnostic criteria developed via a Delphi consensus study have enhanced identification of PG. The criteria include one major criterion and eight minor criteria. The major criterion requires a biopsy of the ulcer edge demonstrating a neutrophilic infiltrate. The eight minor criteria include (1) exclusion of infection, (2) pathergy, (3) personal history of inflammatory bowel disease or inflammatory arthritis, (4) history of papule, pustule, or vesicle that rapidly ulcerated, (5) peripheral erythema, undermining border, and tenderness at the site of ulceration, (6) multiple ulcerations (at least on occurring on an anterior lower leg), (7) cribriform or "wrinkled" paper scar(s) at sites of healed ulcers, and (8) decrease in ulcer size within one month of initiating immunosuppressive medications. If the major criterion and four minor criteria are met, PG can be diagnosed with a sensitivity of 86% and specificity of 90%.

Abstract #29 Superior Mesenteric Artery Syndrome: The Face Behind the Mask

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Introduction

Superior Mesenteric Artery Syndrome (SMAS) is a rare condition with an estimated incidence of 0.013-0.3% in the general population. SMAS is caused by a reduced aortomesenteric angle leading to duodenal obstruction. This causes vague gastrointestinal symptoms such as post-prandial abdominal pain, nausea, vomiting, dyspepsia or reflux, early satiety, anorexia, and weight loss.
We present a case of SMA syndrome in a young female with unexplained weight loss and abdominal pain for 2 years.

Case Description

The patient is a 19-year-old female who presented with a 2-year history of post-prandial abdominal pain, bloating, nausea, intolerance to liquids and solids, fatigue, anorexia, and unintentional 30-pound weight loss. Physical exam was normal, including a BMI of 18.82. From the onset of symptoms, the patient had a normal EGD, a gastric emptying study showing mild delay at 4 hours, a normal colonoscopy with negative biopsies, and CT with contrast. An upper GI series with small bowel follow through showed narrowing of the third portion of the duodenum with mild proximal dilatation consistent with SMAS. Subsequent CT angiogram revealed an aortomesenteric angle of 15 degrees. All preoperative labs were within normal limits. After much counseling and preoperative workup, the patient underwent duodenojejunostomy with resection of the duodenal stump. Postoperative course was uneventful.

Discussion/Conclusion

SMAS often mimics many other common gastrointestinal conditions. Prompt diagnosis of SMAS is crucial to prevent potential complications. Complications include dehydration, electrolyte imbalance, gastric perforation, and cachexia. Mortality of patients with SMAS is reported to be near 33%. Awareness of this disease by providers is necessary to increase chances of diagnosis and timely treatment.

Abstract #36 Jejunal Free Flap for Reconstruction of Radical Neck Dissection

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Introduction

A jejunal free flap is the gold standard for radical reconstruction of the upper gastrointestinal tract. Most commonly, this procedure is performed for reconstruction of the oropharynx, pharynx, and esophagus after radical dissection of head and neck cancer.

Case Description

The patient is a 74-year-old man with PMH significant for squamous cell carcinoma of the neck s/p radical resection and chemoradiation, hypertension, hypothyroidism, and GERD who presented for recurrent laryngeal squamous cell carcinoma with malignant tracheoesophageal fistula. Other PSH is notable for gastrostomy tube for severe malnutrition, tracheostomy, and repair of an abdominal aortic aneurysm. He received a repeat resection of the carcinoma with laparoscopic harvest of the jejunum and reconstruction of the upper gastrointestinal system with an associated jejunal free flap with microvascular anastomosis. Surgical resection with negative margins was achieved. Postoperatively, he was admitted to the ICU for close monitoring. The patient progressed well and was transferred to a general med/surg floor on POD 3. On POD 7, the patient underwent internalization of jejunostomy from the donor site. On discharge, doppler of the jejunal anastomoses remained audible. He was discharged home on POD 8 and instructed to remain NPO for 1 week until clinic follow up. At one week clinic follow up, he had advanced to clear liquid diet and advised to advance as tolerated.

Discussion/Conclusion

Jejunal free flaps are a feasible mechanism for reconstructing the upper gastrointestinal system. For pathology limited to the upper esophagus, this has less morbidity than a total esophagectomy. Jejunal necrosis due to inadequate vascular anastomosis remains a potentially fatal sequela of flap failure and can cause sepsis and carotid artery hemorrhage.

Abstract #37 Colon Carcinoma Perforating into the Duodenum: An Unusual Indication for the Whipple Procedure

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Introduction

Pancreaticoduodenectomy, also referred to as the Whipple procedure, is a radical resection and reconstruction surgery in which the pancreas duodenum and bile duct are excised, due to shared vascular supply. The most common indication for this procedure is pancreatic adenocarcinoma. We present an interesting case of a patient who underwent an en bloc right hemicolotomy with the Whipple procedure for a complicated hepatic flexure colon cancer.

Case Description

The patient is a 68-year-old woman who initially presented with several months of periumbilical abdominal pain, unexplained weight loss, nausea, vomiting, diarrhea, and melena. Initial Workup included a CT which was significant for a circumferential colonic mass near the hepatic flexure with mesenteric lymphadenopathy and most concerning - a large fistulous connection in the second and third portion of the duodenum. Patient was clinically staged as cT4N1M0. Patient underwent an en bloc right hemicolectomy and Whipple procedure. Postoperative course was complicated by a pelvic abscess, resolved with radiographically guided drainage, and a superficial skin infection. Final path revealed a pT4bN2aM0 poorly differentiated adenocarcinoma of the ascending colon with tumor extension into the duodenum. Margins were negative.

Discussion/Conclusion

This case highlights a very unusual indication for the Whipple procedure. Colon cancer with perforation comprises 3-10% of initial presentation of colon cancer. Usually the perforation is intraperitoneal and free, necessitating urgent surgical intervention. It is extremely rare to have a colon cancer fistulize to the duodenum. Complete oncologic resection with negative margins and adjuvant therapy gives the patient the best chance at prolonged survival.

Abstract #39 Renal Cell Carcinoma Induced Vein Thrombosis to the Right Atria: A Case of Snake Thrombus

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Introduction

In 2021, renal cancer was the 6th most common malignancy in men and 9th most common in women. Of these cases, the majority are renal cell carcinoma (RCC). Renal cell carcinoma classically presents as the triad: hematuria, flank pain, and a palpable abdominal mass. Radical nephrectomy is the primary treatment for RCC.

Case Description

Our case presents a 45-year-old male with acute chest and back pain due to a pulmonary embolism. CT and subsequent MRI also showed a 12cm left renal mass with a large vena cava thrombus extending to the right atrium, which had been diagnosed 1 week prior. Symptoms included a 1-month history of weakness and weight loss, back pain, and hematuria. The patient underwent a left nephrectomy, thrombectomy, and sternotomy with right atrial and ventricular thrombectomy. During the procedure, the patient had an estimated blood loss of 12L. Despite multiple transfusions and aggressive CVICU care, the patient's condition worsened until passing.

Discussion/Conclusion

This case presented a unique thrombus associated with RCC extending through the IVC and into the heart. Few previous case reports illustrate occurrence of similar thrombi in the presence of RCC. These associated thrombi present increased procedural risk and difficulty. Despite these risks, radical nephrectomy serves as the mainstay of treatment for patients with RCC.