Kansas Journal of Medicine
Vol. 13, 2020; Supplement 1

Abstracts from the 2019 Annual Meeting
Kansas Chapter of the American College of Physicians
Wichita, KS

15. **Renal Artery Angioplasty and Stent Placement for Management of Refractory Hypertension Secondary to an Iatrogenic Flap**
    Uzair Mahmood, MBBS, Kamal Gupta, M.D.
16. **Erythema Ab Igne from Heating Pad Use**
    Vijayram Reddy Malladi, M.D., William Salyers, M.D.
17. **Cannabis Coronary Syndrome - Novel Culprit in the Making?**
    Maha Mohamad, M.D., Sinan Khayyat, M.D., Mohinder R. Vindhyal, M.D., M.Ed.
18. **A Differential Conundrum: Severe Rhabdomyolysis in the Setting of Hypothyroidism and Suspected Inborn Error of Metabolism**
    Mark Quinn, M.D., Maharshi Bhakta, M.D.
19. **WATS3D vs. Traditional Forceps Biopsy in Screening of Barrett's Esophagus: A Community Hospital Experience**
    Sachin Srinivasan, M.D., Yasmine Hussein Agha, M.D., Jeff Hyder, M.D., William Kilgore, M.D., Nathan Tofteland, M.D., William Salyers, M.D.
20. **Metastatic Calcification of the Myocardium, Aorta, Mitral Valve, and Lungs: A Rare Presentation of Calciphylaxis Following Acute Kidney Injury**
    Praveen Subramanian, D.O., Nicholas Isom, M.D., Laura Thomas, M.D.
21. **When a Deep Vein Thrombosis becomes an Emergency**
    Michael Sullivan, M.D.
22. **Pancytopenia and Hemolytic Anemia Secondary to Severe Cobalamin Deficiency**
    Andrew Weaver, M.D., Seth Page, M.D.
23. **Shock Teams: The Future of Cardiogenic Shock**
    Robert Weidling, M.D., Mohamed El-Khashab, M.D.
24. **Adherence to Opioid Prescribing Guidelines in an Internal Medicine Clinic**
    Jerrica Werner, M.D., Edward Ellerbeck, M.D., Andrea Allen, M.D., Matthew DaCunha, M.D., Ariel Johnson, M.D., Justin Theleman, M.D.
25. **Palbociclib-Induced Fulminant Hepatic Failure**
    Nathaniel Parker, D.O., Rami Atallah, M.D., Karl Hamouche, M.D.
26. **Neurosarcoid Causing Panhypopituitarism and Diabetes Insipidus**
    John Fritzlen, M.D., Nelli Lakis, M.D., John Leever, M.D., Yasir Jassam, MBChB, MRCP
27. **Cardiopulmonary Arrest in a Type 1 Diabetic Female Secondary to Hypoglycemia Associated Autonomic Failure: Dead in Bed Syndrome**
    Anam Abbasi, M.D., Jeet Mehta, M.D., Lina Huerta-Saenz, M.D.
28. **Immunoglobulin G-4 Related Dacryoadenitis**
    Ethan Alexander, M.D., Branden Comfort, M.D., MPH
29. **A Case of Anti-Jo-1 Antisynthetase Syndrome with Interstitial Lung Disease**
    Daly Al-Hadeethi, M.D., Ammar Al-Obaidi, M.D., Grace Nassim, M.D., Janel Harting, M.D.
30. **Cutaneous Nocardiosis in an AML Patient Treated with Azacitidine (Vidaza®)**
    Mejalli Al-Kofahi, M.D., Laura Thomas, M.D.
31. **Spontaneous, Loculated, and Massive Hemothorax: An Uncommon Complication of Warfarin Therapy**
    Ammar Al-Obaidi, M.D., Daly Al-Hadeethi, M.D., Sinan Khayyat, M.D., Quoc Truong, M.D., Alaeldin Mohammed, M.D.
32. **When Leukemia Changes Colors: A Case of Lineage Switch in Acute Leukemia**
    Joseph Bennett, M.D., Sunil Abhyankar, M.D., Janet Woodroof, M.D., Anurag Singh, M.D.

33. **Empyema due to Colopleural Fistula as a Complication of Metastatic Cholangiocarcinoma**
    Alec Britt, M.D., Brice Zogleman, M.D.
34. **Psoriatic Arthritis: A Diagnosis in the Details**
    Christopher Bryant, D.O., Brent Duran, D.O.
35. **Vitamin B12 Deficiency with Pseudothrombotic Microangiopathy**
    Scott Buess, M.D., Alexander Germann, MS4, Eamon Maloney, M.D., Alaeldin Mohammed, M.D.
36. **Primary Sporadic Gastric Burkitt Lymphoma Presenting with Melena**
    Brandon Calhoun, D.O., Praveen Subramanian, D.O., Daniel Vestal, M.D.
37. **Advance Care Planning Rates in The University of Kansas Health System Cancer Clinics**
    Stacie Carlson, M.D., Megan Haghnegahdar, M.D.
38. **Pulmonary Paragonimiasis**
    Jonathan Chandler, D.O., Lisa Clough, M.D.
39. **A Case of a Group G Beta-Hemolytic Streptococcus Necrotizing Soft Tissue Infection and Early Toxic Shock-Like Syndrome**
    Khalil Choucair, M.D., Pie Pitchetsurnthorn, M.D., Thomas A. Moore, M.D.
40. **Rothia Mucilaginosa Pneumonia in a Patient with Rheumatoid Arthritis**
    Deshanett Clay, M.D., MPH, Mona Brake, M.D., Zubair Hassan, M.D.
41. **Ecthyma Gangrenosum Revealing Pseudomonas Bacteremia in the Immuno-Compromised Patient**
    Connor Grantham, M.D.
42. **Cerebral Nocardiosis in an Immunocompetent Old Adult**
    Sinan Khayyat, M.D., Ammar Al-Obaidi, M.D., Maha Mohamad, M.D., Mohinder R. Vindhyal, M.D., M.Ed.
43. **Disseminated Histoplasmosis in the Setting of Immunosuppression with Infliximab**
    Benton McGivern, M.D., Michael Rouse, D.O.
44. **Citrobacter Koseri CIED Infection in an Immunocompetent Man**
    Nikki Miller, M.D., Jessica Newman, D.O.
45. **Sporotrichosis Bone Presentation in an HIV-Infected Patient**
    Richard Muraga, M.D., Jane Gitau, M.D., Susan Ngunjiri, M.D.
46. **Neurosyphilis with Suspected Tabes Dorsalis, Otic, and Optic Involvement in an AIDS Patient**
    Sonya Parashar, M.D., Ashley Clark, M.D., Branden Comfort, M.D.
47. **Disseminated Mycobacterium Haemophilum Infection Involving Central Nervous System in a Renal Transplant Patient**
    Farhad A. Sami, MBBS, Wissam El Atrouni, M.D., Kathy Newell, M.D., Stephen Waller, M.D., Marie Pacholec, D.O.
48. **Culture Positive Mycobacterium Tuberculosis with Negative T-Spot TB and PCR**
    Stephanie Slimmer, M.D., Albert Eid, M.D.

Delayed Post Hypoxic Leukoencephalopathy

Lorna Nguutu, M.D., Kerby Justin, M.D., Mohammed Hussein, M.D., Brent Duran, D.O.
KU School of Medicine-Wichita

[Judges Award: Best Poster]

Introduction

Delayed post-hypoxic leukoencephalopathy (DPHL) is a syndrome that results after a complete recovery phase from an acute hypoxic brain insult. Patients often present with neurologic and psychiatric decline within days to weeks after the incident. There are no clear pathophysiology, diagnostic approach, or treatment modalities. We present a case of DPHL with progressive neurologic decline.

Case Presentation

A 66-year-old male presented to the hospital for evaluation of waxing and waning neuropsychiatric symptoms, including inattention, paranoia, hand tremors, and confusion. Seventeen days earlier, he had a partial colon resection at an outside hospital secondary to complicated diverticulitis. He was discharged home in stable condition and started having the aforementioned neuropsychiatric symptoms within one week.

Upon initial evaluation, the patient was oriented, appropriately conversant, and walked independently to his room. His neurologic and psychiatric exam was normal. In the subsequent thirty-six days of hospitalization, the patient had a gradual decline in his level of consciousness. This began with bouts of poor mentation and consciousness that waxed and waned. Further neurologic decline ensued as patient was progressively stuporous and eventually obtunded. The change in his mentation was also associated with increased extremity tone. There were no further measurable changes in his neurological exam the remainder of the hospital stay.

A Head/Neck CT/MRA was noncontributory. Initial head MRI was non-diagnostic. Additional workup was negative for infectious, metabolic, inflammatory, toxic, or psychiatric etiology. EEG showed diffuse slowing. Further workup was obtained for rheumatologic, vasculitis, neoplastic etiology, and a complete CSF study including a rare autoimmune panel. A second head MRI was obtained eight days after the initial one and showed extensive periventricular white matter lesions consistent with delayed post hypoxic leukoencephalopathy (DPHL).

Further history gathering was elicited from the prior hospitalization where the patient had surgery. On the second day after the procedure, he had an acute episode of prolonged narcotic induced hypoxia (SpO_2 50s to 60s) with associated hypotension for approximately forty-five minutes. On recognition, he received immediate cardiopulmonary support and later experienced complete recovery with normal progression to his baseline.

With exception of the MRI findings above, all the remainder diagnostic studies were noncontributory.

Conclusions

Initial neuroimaging in DPHL is often nonspecific and leads to a myriad of additional tests. Serial neuroimaging and detailed history taking suggests potential capability to identify DPHL sooner in the appropriate clinical setting. Early identification of the disease would also limit costly and extensive diagnostic studies and hasten the transition to a neuro rehabilitation unit.

Mycoplasma Pneumoniae Encephalitis in an Adult Patient without Preceding Respiratory Symptoms

Maisam Al Patty, M.D., Vivar Cruz, M.D., Maha Assi, M.D.
KU School of Medicine-Wichita

[Chien Liu Award for Best Infectious Disease Poster]

Introduction

Mycoplasma pneumoniae is an atypical bacteria commonly implicated in community-acquired pneumonia. It also causes a wide range of CNS symptoms, such as meningitis, encephalitis, and Guillain-Barré syndrome. These presentations are rare, typically occurring in pediatric populations. We present a case of M. pneumonia associated encephalitis in an adult patient without preceding respiratory disease.

Case Presentation

A 25-year-old male was brought to the emergency room after falling at home within 24 hours of leaving the hospital where he was treated for diabetic ketoacidosis with newly diagnosed diabetes mellitus. Vitals revealed temperature 36.9°C, heart rate of 91, respiration rate of 18, and blood pressure 112/84 mmHg. Exam revealed a lethargic male with intermittent gaze deviation, dysarthria, and right limbs ataxia. The rest of neurological exam was within normal limits including deep tendon reflexes, cranial nerve testing, and motor/sensation testing. CT of brain and chest x-ray were unremarkable. MRI of the brain revealed increased diffusion-weighted and T2 hyperintensity of the corpus callosum, bilateral posterior corona radiata, and bilateral cerebellar peduncles. EEG was unremarkable. A lumbar puncture revealed CSF WBCs 50/mm^3, segmented cells 23%, lymphocyte 70%, monocyte 5%, glucose 142 mg/dl, protein 26 mg/dl, negative gram stain, cultures, and meningitis/encephalitis PCR panel. Upon further investigations, Mycoplasma IgG and IgM serology returned positive. Intravenous Doxycycline was initiated and he was given intravenous immunoglobulin (IVIG) infusions for five days. Four days after starting therapy, follow up MRI brain revealed significant improvement. After 10 days of hospitalization, the patient demonstrated clinical improvement of his neurological symptoms and he was discharged to a rehabilitation center.

Conclusions

This patient presented with Mycoplasma pneumoniae related encephalitis in the absence of preceding respiratory disease. The diagnosis was made based on the patient's clinical symptoms, CSF pleocytosis, and a positive serology of M. pneumoniae. Current literature describes several mechanisms by which this pathogen may cause CNS symptoms including direct invasion, autoimmune response, or vascular occlusion. Mycoplasma associated encephalitis is hypothesized to be driven by immunologic response, thus IVIG is a preferred therapy to ameliorate central nervous system damage. In our case, Doxycycline therapy alone was associated with improvement of MRI findings. IVIG was then administered with gradual improvement of neurological symptoms. Antibiotic therapy, in conjunction with IVIG remains a clinically important part of treatment of Mycoplasma associated neurological disease.

M. pneumoniae is a rare cause of encephalitis that may occur in the absence of respiratory disease. It should be considered in cases where infectious CNS disease is suspected, particularly when CSF pleocytosis is present and CSF microbiological studies are negative. Both antibiotics and IVIG remain the recommended therapy.

Dasatinib Induced Pericarditis

Luke Frankl, M.D., Nicholas Isom, M.D.
KU School of Medicine-Kansas City

Introduction

For patients with CML, the introduction of tyrosine kinase inhibitors (TKIs) in 2000 has decreased the annual mortality from 10-20% to 1-2% and increased ten-year survival rates from 20% to 80-90%. CML is characterized by the fusion of the ABL1 gene on chromosome 9 and the BCR gene on chromosome 22. TKIs specifically target and inhibit the interaction between BCR-ABL1 and ATP thereby blocking cellular proliferation of the malignant clone.

Case Presentation

A 78-year-old female with a history of renal cell carcinoma s/p nephrectomy, hypertension, non-ischemic cardiomyopathy, and diabetes presented to the hospital after outpatient evaluation for workup of severe leukocytosis. She underwent bone marrow biopsy and was found to have CML with blast crisis. She was started on Dasatinib for initial treatment. Eight days after initiation of the treatment, she developed dull, sub-sternal chest pain on deep inspiration. She denied fevers, chills, and shortness of breath. She had a regular rate and rhythm, without murmurs, rubs, jugular venous distention, lower extremity edema, or abnormal breath sounds. Initial EKG showed no ischemic changes. Her troponin was 0.06. CT chest demonstrated a small consolidation in the right lower lobe and mild pericardial thickening and effusion. Over the next two days, the pleuritic chest pain worsened and she became dyspneic. Troponin increased to 6.6, BNP was 964, ESR and CRP were >140 and 31.73, respectively. A repeat EKG showed diffuse ST segment elevation in all leads. She was diagnosed with Dasatinib induced pericarditis, treated with a steroid taper, and transitioned to Imatinib. After which, she made a full recovery.

Conclusions

First line therapy for CML is tyrosine kinase inhibitors. Despite having similar rates of 5-year survival, studies have shown improved responses to therapy with Dasatinib vs. Imatinib. However, Dasatinib has been shown to have higher rates of toxicity. One study reports grade 3 and 4 adverse event rates of 58%. Although most side effects of TKI's are hematologic, Dasatinib has been shown to cause pleural effusions, pericardial effusions, and pulmonary hypertension at rates of 19%, 5%, and 1-2%, respectively. Despite this association with cardiopulmonary toxicity, reports of Dasatinib induced pericarditis are exceedingly rare and appear to be limited to chronic constrictive pericarditis.

There is evidence that the pathogenesis of Dasitinib induced effusions and serositis is immune-mediated. Dasatinib's inhibition of off-target or non BCR-ABL kinases can lead to clonal expansion of large granular lymphocytes of natural killer and cytotoxic T cell lineages, both of which have been implicated in Dasatinib induced pleural effusions and colitis. This mechanism may also explain the development of pericarditis and its response to steroids. Despite the rare association, this case highlights the need for physicians to have acute pericarditis on the differential for chest pain in patients taking Dasatinib.

Rise in Inhalational Lung Injury in the US: The Blunt Truth about Vaping

Kristin Constance, M.D., Samuel Akidiva, M.D., Margaret Hagan, M.D.
KU School of Medicine-Wichita

Introduction

Recent case reports have brought attention to the increasing incidence of severe acute lung injury in patients who have been vaping or "dabbing" CBD oil (process of vaping marijuana oils, extracts, or concentrates). The CDC released a statement in August of this year addressing the rise of reported cases of severe inhalational lung injury associated with vaping. The first reported death attributed to inhalational lung injury from vaping was reported this month.

Case Presentation

An 18-year-old male presented to the Emergency Department for nausea/vomiting and shortness of breath. He reported a chronic dry cough for a few weeks that had worsened after the onset of nausea/vomiting. On arrival he required 2L O_2 by nasal cannula to maintain his oxygenation. Chest x-ray revealed bilateral pulmonary infiltrates concerning for pneumonia.

Investigation revealed frequent marijuana use over the last year, and the working diagnosis shifted to cyclic vomiting syndrome vs. cannabis hyperemesis syndrome with likely aspiration pneumonia. The patient's nausea/vomiting resolved shortly after admission, however, he remained hypoxic. Work-up for bilateral pneumonia revealed negative HIV, streptococcus pneumoniae, and legionella antigens. Sputum culture was collected, however, determined a poor specimen and not finalized. Antibiotics were deescalated from Piperacillin-tazobactam to Amoxicillin-clavulanate.

The patient's oxygen requirements increased from 2L to 10L O_2 by nasal cannula on the third hospital day. Antibiotics were broadened to Piperacillin-Tazobactam and Levofloxacin. Pulmonology consulted, and bronchoscopy with bronchoalveolar lavage revealed mild airway inflammation/edema with hyperemia. The patient was started on IV steroids and moved to the ICU. His oxygen requirements continued to increase despite antibiotics and Infectious disease consulted.

Interview revealed that the patient had been vaping CBD oil daily for the last few months in addition to smoking marijuana. CT chest revealed centrally located diffuse ground glass opacity with peripheral sparing consistent with inhalational injury secondary to CBD oil vaping. Piperacillin-tazobactam was discontinued and Levofloxacin continued along with systemic steroids. Patient's hypoxia resolved and he was weaned from supplemental oxygen prior to discharge. He was sent home with a long steroid taper per pulmonology recommendations for treatment of inhalational lung injury secondary to vaping CBD oil.

Conclusions

This case illustrated the need for a high index of suspicion to appropriately diagnose and treat vaping related inhalational lung disease. Although this patient had a good outcome, his diagnosis was delayed thus delaying appropriate treatment with systemic steroids. The pattern of lung injury can be severe causing significant concern for delayed diagnosis. The long-term effects of CBD oil vaping have yet to be described, but with increasing cases of vaping associated lung injury clinicians are turning their attention towards this topic.

How High Is Too High?

Chisom Eze, M.D., Brent Duran, D.O., Edgard Wehbe, M.D., Patrick McEnulty, M.D., Nolan Fisher, M.D., Reid Eggleston
KU School of Medicine-Wichita

Introduction

Osmotic Demyelination Syndrome (ODS) occurs when there is rapid over-correction of hyponatremia. ODS is characterized by destruction of neuronal myelin sheaths in either the central area of the pons or in other susceptible areas. Although there is no specific treatment, the use of plasmapheresis, IVIG, and methylprednisolone therapy has been successfully implemented. This case report describes an acute onset of ODS with medical history significant for chronic alcoholism and hypertension.

Case Presentation

A 49-year-old male with chronic alcoholism, peptic ulcer disease, and newly diagnosed hypertension presented to the local ED when his wife noticed he was behaving strangely. He was severely hyponatremic which was treated with 3% normal saline (NS) over 5-6 hours, then ½ NS and 4 doses of desmopressin over the next 3 days. At discharge, he had returned to baseline and no longer exhibited weakness or altered level of consciousness with a serum sodium of 128 mEq/L. He was instructed to discontinue taking hydrochlorothiazide previously prescribed by his PCP and to obtain follow-up labs which revealed sodium level increased to 138 mEq/L. His wife noted he began to exhibit stiffness and tremors involving bilateral upper and lower extremities, aphasia, and decreased responsiveness 3 days later. She presumed he was withdrawing from alcohol and later brought him back to the ED where his serum sodium was 141 mEq/L. He was given 1L NS bolus, then ½ NS, desmopressin 2 mcg IV, and started on 5% dextrose in water. Upon admission, his sodium was 140 mEq/L and he exhibited decorticate posturing, aphasia, shaking tremors of the bilateral lower extremities and rigidity. 5% dextrose in water was re-started and MRI of the brain revealed pathologic diffusion restriction within the central pons as well as hyperintense signal in the basal ganglia, confirming suspicion of osmotic demyelination syndrome. During ICU admission, he was given plasmapheresis, IVIG therapy, and IV methylprednisolone to improve prognosis. He required intubation for concerned aspiration and was sedated with concern for alcohol withdrawal. Time will reveal if these therapies were initiated in a timely fashion as to provide our patient a chance at survival.

Conclusions

Risk factors for ODS include chronic alcoholism, history of liver transplant, rapid correction of hyponatremia and malnutrition. Pathophysiology of ODS involves the reduced extracellular osmolality from hyponatremia causing brain cells to release osmotically-active myelin toxic substances to achieve osmotic equilibrium. These osmotic substances cannot be reabsorbed rapidly, and when the sodium levels are increased rapidly, they create osmotic stress that leads to a disruption in the blood-brain barrier, damage to oligodendrocytes, and irreversible myelinolysis. Initial treatment schemes are varied with the immediate goal being re-introduction of hyponatremia, followed by therapeutic plasmapheresis, leading to clinical improvement occurring as early as 1-2 weeks.

What's in the Meat? A Case of Alpha-Gal Syndrome Following Lone Star Tick Bite

John Frey, D.O., Joseph W. Kerley, M.D.
KU School of Medicine-Kansas City

Introduction

Alpha-gal syndrome is a food allergy characterized by the development of IgE antibodies directed at the carbohydrate galactose-alpha-1,3-galactose (alpha-gal). It presents as an allergic reaction after consuming meat from mammals. We present a case of alpha-gal syndrome.

Case Presentation

A 55-year-old female with a past medical history of hypothyroidism presented to our hospital for evaluation of new-onset rash. She reported that the rash originally began on her back, then spread to her chest, arms, and legs. It spared the face, palms of her hands, and soles of her feet. The rash was described as pruritic in nature. She denied shortness of breath. She denied any new medications, supplements, or detergents. She denied recent travel. She reported a history of allergic reactions to latex exposure, but denied any recent exposures. She worked from her home and denied any occupational exposures. She did report being bitten by a Lone Star tick three weeks prior to presentation. She reported that her typical diet was vegan-based, but that over the last two days she had consumed beef products; she stated her symptoms began a few hours after consuming a hamburger.

On initial presentation, the patient's vitals were as follows: temperature 36.4 °C, pulse 97 bpm, RR 20 br/min, BP 92/61 mmHg, O_2 94% on room air. Her exam was remarkable for diffuse erythematous urticarial lesions that were located on the chest, abdomen, back, bilateral arms, and legs. Her CBC was unremarkable with no peripheral eosinophilia on the differential. Her electrolytes, kidney function, and liver function were all normal. Given her rash and hypotension, there was concern for anaphylactic reaction and the patient was given an Epinephrine 0.3 mg IM, Methylprednisolone 25 mg IV, diphenhydramine 50 mg IV, and Famotidine 20 mg IV. Following these medications, the rash began to dissipate and she was admitted for further evaluation.

Additional laboratory testing demonstrated the patient was negative for HIV infections, Rocky Mountain Spotted Fever serologies (IgM+IgG) were negative, and Lyme serologies (IgM and IgG) were also negative. The patient did have an elevated IgE level to galactose-alpha-1,3-galactose at 78.90 kU/L, and was diagnosed with alpha-gal syndrome. She was instructed to avoid meat products from mammalian meat and discharged home.

Conclusions

Alpha-gal syndrome is a food allergy to mammalian meat products. The condition begins when a Lone Star tick bite transmits the alpha-gal sugar molecule into the body. In some people, an immune response occurs and IgE antibodies against alpha-gal are produced. Following antibody development, exposure to mammalian meat can result in mild to severe allergic reactions. There is no specific treatment for alpha-gal syndrome other than avoidance of mammalian meat products. Preventative measures for development of alpha-gal syndrome include avoidance of ticks.

Ski Related Traumatic Brain Injury Resulting in Severe Water Balance Disorder

Kristen E. Funk, M.D., Leigh M. Eck, M.D.
KU School of Medicine-Kansas City

Introduction

We describe a case of a triphasic water balance disorder following a ski related traumatic brain injury (TBI). While the triphasic water balance disorder is well recognized following neurosurgical procedures, it is less frequently described following TBI.

Case Presentation

A 31-year-old male without prior past medical history suffered a TBI following a single person snow skiing incident resulting in loss of consciousness requiring hospital admission. Following discharge from the hospital, he noted polyuria and polydipsia; there was no discussion about these symptoms nor laboratory investigation (such as a water deprivation test) during his initial hospital stay. On reflection, it was assumed that these symptoms were consistent with acute diabetes insipidus (DI) following a TBI. Upon return to his home state, he noted a resolution of polyuria and polydipsia with subsequent development of nausea, vomiting, headache, and abdominal pain. Due to severity of symptoms following his TBI, he sought emergency care.

Laboratory data was significant for hypo-osmolar hyponatremia with a serum sodium of 121 mmol/L (137-147). Urine osmolality was inappropriately concentrated at 871 mos/kg. Upon ruling out adrenal insufficiency and thyroid dysfunction as potential etiologies, a diagnosis of TBI associated syndrome of inappropriate anti-diuretic hormone (SIADH) secretion was made. Due to the acuity of symptoms with serum sodium <130 mmol/L, 3% saline was initiated in addition to a fluid restriction with safe correction of serum sodium. Within four days, SIADH appeared resolved and fluid restriction was stopped. In the next 24 hours, the patient had a urine output of 9 liters with a rising serum sodium and dilute urine output. dDAVP therapy was initiated with resolution of polyuria and normalization of serum sodium. The patient was discharged to home on dDAVP with long term normalization of urine output and serum sodium; with any missed doses of dDAVP following discharge, polyuria ensued. At six months post injury, DI persisted.

Conclusions

This patient presented with the triphasic response of pituitary stalk injury following a TBI. While clinicians watch for this water balance disorder closely following neurosurgical interventions (incidence ~20%), it is less commonly considered following TBI. However, in a prospective study of 50 consecutive patients with severe or moderate TBI, 26% of patients had DI in the acute post-TBI phase with 6% with permanent DI at 12 months post injury. Given the significant possibility of the triphasic response of pituitary stalk injury following TBI, clinicians should be attuned to signs and symptoms and pursue diagnostic workup and treatment when indicated, given the risk for morbidity and mortality of untreated water balance disorders.

ANCA-Associated Vasculitis

Cory Gutovitz, M.D., Erica Howe, M.D.
KU School of Medicine-Kansas City

Introduction

ANCA-associated vasculitis is a rare entity, making a timely and accurate diagnosis challenging. We report a patient presenting with multiple vague symptoms and lab abnormalities, ultimately found to have ANCA-associated vasculitis. The case is a representation of Occam's razor, a belief that multiple symptoms can be explained by a single cause. Thus, it remains pivotal to cast a broad differential diagnosis and conduct a methodical workup.

Case Presentation

A 65-year-old Caucasian female with seasonal allergies and hyperlipidemia presented to the emergency department with five months of progressive generalized weakness, fatigue, and unintentional weight loss. She was following with an outpatient provider, but no clear explanation was found and symptoms persisted. In the ER, labs revealed normocytic anemia (Hgb 7.8), thrombocytosis (Plt 405), leukocytosis (14.6), and mildly elevated creatinine at 1.01. Eighteen months earlier, these were all in the normal range (Hgb 14.1, Plt 263, WBC 7.6, and creatinine 0.68). Chest x-ray and urinalysis showed no infection. She was admitted for further investigation. Subsequently, she had CT chest/abdomen/pelvis, PET/CT, MRI brain, IR-guided biopsy of a chest wall lesion, all without significant diagnostic abnormalities. She was discharged after five days with outpatient follow-up. Her only new medication was vitamin B12, for mild deficiency. Two weeks later, she had a relatively normal bone marrow biopsy. Two months later, she again presented to the ER with worsening of the same symptoms and her lab abnormalities persisted. Her cell count numbers were similar, however, this time her creatinine was now 1.7. She underwent EGD and colonoscopy which showed no GI source for anemia. Nephrology was consulted for progressive decline in renal function and additional lab testing was ordered. Her C-ANCA titer returned positive. Further testing, including renal biopsy, confirmed the diagnosis and she began treatment with IV steroids and rituximab. Her symptoms and lab abnormalities began showing improvement, and she continues to follow with rheumatology for ongoing management.

Conclusions

ANCA autoantibodies, first reported in 1982, bind to neutrophils in the blood and induce inflammatory damage via multiple mechanisms. They manifest pathology in a variety of ways and patients commonly present with non-specific symptoms such as weight loss and general malaise. In this case, closer inspection of UA showed mild microscopic hematuria and mild proteinuria hinting at renal involvement, which occurs in 25-75% of patients with ANCA. Prompt diagnosis is crucial because >50% of treated patients can have disease remission and avoid complications such as renal failure and need for dialysis. This case highlights that sometimes an accurate diagnosis can take weeks-months to ascertain, and it is important to persist and embrace diagnostic challenges.

Measures to Decrease "Door to Balloon" Time
and Incidence of Unnecessary Cardiac Catheterization

Sumaya Hammami, M.D., Niksad Abraham, M.D., Ehsanur Rahman, M.D.,
William S. Weintraub, M.D., Andrew J. Doorey, M.D., Zaher Fanari, M.D.
KU School of Medicine-Wichita

Introduction

Efforts to reduce the door-to-balloon time require a rapid triage decision with faster dispatch to the catheterization lab. However, this may come at the expense of increased risk of incorrect triage decisions and an increased rate of false-positive STEMI (FP-STEMI), whereby a patient is taken emergently to the catheterization laboratory but no STEMI is found.

This study assessed the impact of an aggressive protocol to decrease the time from hospital arrival to onset of reperfusion therapy ("door to balloon [DTB] time") on the incidence of false-positive (FP) diagnosis of ST-segment elevation myocardial infarction (STEMI) and in-hospital mortality.

Methods

The study population included 1,031 consecutive patients with presumed STEMI and confirmed ST-segment elevation who underwent emergent catheterization between July 1, 2008 and December 1, 2012. On July 1, 2009, we instituted an aggressive protocol to reduce DTB time. A quality improvement (QI) initiative was introduced on January 1, 2011 to maintain short DTB while improving outcomes. Outcomes were compared before and after the initiation of the DTB time protocol and similarly before and after the QI initiative. Outcomes were DTB time, the incidence of FP-STEMI, and in-hospital mortality. A review of the emergency catheterization database for the 10-year period from January 1, 2001 through December 31, 2010, was performed for historical comparison.

Results

Of the 1,031 consecutive patients with presumed STEMI who were assessed, 170 were considered to have FP-STEMI. The median DTB time decreased significantly from 76 to 61 minutes with the aggressive DTB time protocol ($P = 0.001$), accompanied by an increase of FP-STEMI (7.7% vs. 16.5%; $P = 0.02$). Although a nonsignificant reduction of in-hospital mortality occurred in patients with true-positive STEMI ($P = 0.60$), a significant increase in in-hospital mortality was seen in patients with FP-STEMI ($P = 0.03$). After the QI initiative, a shorter DTB time (59 minutes) was maintained while decreasing FP-STEMI in-hospital mortality.

Conclusions

Aggressive measures to reduce DTB time were associated with an increased incidence of FP-STEMI and FP-STEMI in-hospital mortality. Efforts to reduce DTB time should be monitored systematically to avoid unnecessary procedures that may delay other appropriate therapies in critically ill patients.

Implications of Mucosal Schwann Cell Hamartomas on Colonoscopy Risk Stratification: A Retrospective Case Series

Jonathan Henke, M.D., Florence Aslinia, M.D., Maura O'Neil, M.D., Becky Lowry, M.D.
KU School of Medicine-Kansas City

Introduction

Mucosal Schwann cell hamartomas (MSCH) are structurally and histologically distinct neuronal polyps. They are distinguished by proliferation of spindle cells in the lamina propria of the colonic mucosa and show diffuse histologic positivity with S100. The concept of a MSCH as a distinct polypoid lesion is relatively novel, first described in 2009. As such, there is a paucity of literature regarding the lesions. They are generally believed to be benign, possessing no malignant potential nor association with polypoid syndromes. However, there is scant evidence regarding appropriate repeat screening interval when a MSCH is identified on colonoscopy. We attempted to further investigate the impact of MSCH on malignant potential and repeat surveillance interval.

Methods

Fifteen colonoscopy reports positive for mucosal Schwann cell hamartomas were identified from KU Medical Center from 2014 to 2019. Patients' medical records were reviewed using electronic health records.

Results

Average age at diagnosis was 58. Average number of MSCH's was 1.4 with an average size of 5.1 mm. The adenoma detection rate (ADR) was 0.53 and adenoma per colonoscopy (APC) was 1.13. One colonoscopy showed evidence of new colonic malignancy. None of the remaining fourteen had dysplastic polyps or adenomas >10 mm. Three colonoscopies had 3-10 adenomas. Three colonoscopies met criteria for genetic workup of hereditary polypoid syndromes, and all were negative for any associated genetic syndrome.

Four patients underwent repeat colonoscopy with an average interval of 23 months. Two repeat colonoscopies had additional hamartomas as well as multiple adenomas. Two had no polyps on repeat. None of the four had evidence of dysplasia. ADR of repeat colonoscopies was 0.50 and APC was 2.0. Of the 11 patients without repeat colonoscopies, none developed gastrointestinal symptoms warranting earlier repeat surveillance colonoscopy.

Conclusions

MSCHs do not appear to possess malignant potential. None of the identified MSCHs showed pathologic evidence of dysplasia, and no subsequent colonoscopies had evidence of dysplastic polyps or malignancy. Additionally, there does not appear to be an association with MSCHs and malignant polypoid syndromes, as none of the 15 patients had other symptoms of syndromes, and 3 patients actually had full negative genetic testing. Of note, ADR and APC rates in our observed colonoscopies were higher than reported norms in the literature, though there is no obvious causal relationship between MSCHs and increased prevalence of adenomas. Overall, our results support traditional repeat colonoscopy intervals based on number and character of adenomatous polyps, regardless of presence of mucosal Schwann cell hamartomas. Our analysis is limited in that only 4 patients have of yet undergone repeat colonoscopy. More follow-up of subsequent colonoscopies may provide additional insight.

Cirrhosis and Barrett's? WATS-3D as a Screening Tool

Yasmine Hussein Agha, M.D., Sachin Srinivasan, M.D., William Salyers, M.D.
KU School of Medicine-Wichita

Introduction

Barrett's esophagus is a pre-malignant condition in patients with long-standing gastroesophageal reflux disease (GERD). The gold standard for diagnosing Barrett's disease is taking 4-quadrant esophageal biopsies during endoscopy followed by demonstration of columnar metaplasia on histopathology. However, in patients with cirrhosis, there is an increased risk of hemorrhagic complications due to esophageal varices and coagulopathy. This case series reports three patients with cirrhosis that were successfully screened for Barrett's using WATS-3D (wide angle trans-epithelial sampling with computer assisted 3-dimensional tissue analysis).

Case Presentation

Case 1: 66-year-old female with a history of cirrhosis secondary to autoimmune hepatitis, portal hypertension, esophageal varices, GERD, and previously diagnosed Barrett's esophagus that had liver and kidney transplants 3 years prior to presentation.
Case 2: 63-year-old male with a history of Barrett's esophagus, Laennec's cirrhosis, esophageal varices, and portal hypertensive gastropathy.
Case 3: 62-year-old male with a history of cirrhosis due to untreated hepatitis C and alcohol abuse, known carrier for HFE gene, GERD, and Barrett's esophagus.

Case 1 and 2 had both WATS-3D brushings as well as traditional biopsies done while Case 3 had WATS-3D brushings alone for concerns of bleeding. There was agreement between the WATS-3D analysis as well as biopsy results in Case 1 and 2. Both tools showed Goblet cell metaplasia in Case 1 and high-grade dysplasia in Case 2. The patient with WATS-3D brushings only had evidence of Goblet cell metaplasia. There were no immediate or post-procedural complications (bleeding, infections) in any of the three patients.

Conclusions

The prevalence of GERD and neoplastic progression in patients with cirrhosis is higher compared to patients without liver disease. However, weighing the benefits and risks of taking biopsies during esophagogastroduodenoscopy, many cases of Barrett's go undetected when physicians refrain from taking biopsies and rely solely on endoscopic findings. Treatment of Barrett's disease is also challenging in the presence of cirrhosis since endoscopic mucosal resection is associated with high risk of bleeding in these individuals. Only one case of Barrett's esophagus in the setting of cirrhosis and varices that was successfully treated has been reported. WATS-3D uses a minimally invasive brush biopsy that resects wide area tissue samples, allowing analysis of the full trans-epithelial thickness of the esophagus. It is less invasive than forceps biopsy, has a higher sampling yield, and would be safer in patients at high risk of bleeding complications such as the patients we presented in this case report.

Post-ERCP Pancreatitis Rates and NSAID Use in a Community Hospital

Jeffrey Hyder, M.D., Yasmine Hussein Agha, M.D., William Salyers, M.D.,
Nathan Tofteland, M.D.
KU School of Medicine-Wichita

Introduction

Endoscopic retrograde cholangiopancreatography (ERCP) has been consistently used for more than 50 years as both a diagnostic and therapeutic procedure in the field of pancreatic and biliary diseases. However, with the relatively invasive nature of the procedure, its use is associated with several side effects including bleeding, cholangitis, pancreatitis, intestinal perforation, and death. Among the cited complications, post-ERCP pancreatitis (PEP) remains the leading cause of morbidity and mortality with an estimated incidence ranging between 1.6 and 15%. Several factors including endoscopic techniques, patient's characteristics and the hospital setting have been described that put patients at a higher risk of developing PEP. Williams et al. reported a significantly increased risk of PEP in a community hospital as opposed to a university hospital. Several pharmacologic agents have been suggested, however, none to date have proven to be consistently effective. Rectal NSAIDs including Diclofenac and Indomethacin have shown promising results in several high-quality clinical trials and meta-analysis, especially in high-risk patients. However, more recent trials failed to show a significant reduction of PEP after NSAID use. There is scant data on the efficacy of rectal NSAIDs in higher risk patients in community hospitals. The aim of this study is to determine if there is an increased risk of PEP in a community hospital and if use of rectal NSAIDs for high-risk patients is beneficial within a community hospital.

Methods

After obtaining IRB approval, the research team reviewed the charts of every adult patient admitted to a community hospital in Wichita for ERCP in a 2-year time period. Primary endpoint was percentage of patients developing PEP in the community hospital setting. Secondary endpoints included NSAID use in high-risk individuals and the change in incidence of PEP in this group with the use of rectal NSAID.

Results

422 patients had ERCP performed during the study time period. Of these, 212 (50.2%) were noted to have at least 1 high risk factor to develop PEP. 24 (5.6%) of the patients received rectal NSAIDs, of which 10 patients were noted to be high-risk for PEP. 9 patients (2.1%) developed PEP during the study time period. Of the nine patients, 3 patients were high risk and did not receive any NSAIDs. The use of NSAIDs did not cause a significant reduction in PEP in the high-risk group (p-value 0.7) or overall (p-value 0.66).

Conclusions

PEP rates in a community hospital setting have not been well established. Our study reports an incidence of 2.1%, comparable to the PEP incidence described in academic trials. Despite the unstandardized use of rectal NSAIDs noted in the study population, there appears to be no significant benefit in their use overall or in the high-risk population.

A Rare Tumor with an Ambiguous Presentation

Monica Khurana, M.D., Georges Elhomsy, M.D.
KU School of Medicine-Wichita

Introduction

Pheochromocytoma is a rare tumor arising from catecholamine producing tumor cells of the adrenal medulla. The annual incidence of pheochromocytoma is approximately 0.8 per 100,000 person-years. The clinical presentation can be variable; however, the "classic triad" consists of headache, diaphoresis, and palpitations. These symptoms can occur in paroxysms and are often seen in the setting of hypertension. Previously, these tumors were considered to typically be sporadic; however, with the recent identification of novel susceptibility genes up to 40% of cases may occur in the context of a hereditary syndrome, such as: multiple endocrine neoplasia, von Hippel-Lindau disease, and neurofibromatosis type 1. We report a case with a nonspecific presentation leading to the diagnosis of pheochromocytoma.

Case Presentation

A 60-year-old female with a medical history of hypertension and primary hyperparathyroidism presented with fatigue, hair loss, palpitations, and cold intolerance. She denied headache, diaphoresis, or paroxysms of symptoms. Vitals revealed hypertension (160/96 mmHg) and physical exam was unrevealing. The patient was referred to a cardiologist and workup for cardiac related causes was negative. Subsequent laboratory workup by endocrinology displayed an unremarkable thyroid panel, elevated plasma normetanephrine: 338 pg/mL (reference range: 0-145 pg/mL) and elevated plasma norepinephrine: 2148 pg/mL (reference range: 0-874 pg/ml). 24-hour urine studies revealed elevated normetanephrine: 1031 ug/24 hr (reference range: 82-500 ug/24 hr). CT abdomen/pelvis displayed a 2.1 cm craniocaudal by 1.8 X 1.5 cm right adrenal gland mass with a mean density of 26 Hounsfield units on noncontrast images. Metaiodobenzylguanidine (MIBG) scan showed intense radiotracer localization involving the right ad renal gland corresponding to the mass seen on CT and consistent with pheochromocytoma. Alpha-adrenergic blockage was initiated prior to intervention and she underwent successful laparoscopic right adrenalectomy. Given our patient's history of hyperparathyroidism and pheochromocytoma, there was concern for multiple endocrine neoplasia type 2A. A medullary thyroid cancer (MTC) workup with calcitonin level and thyroid ultrasound ruled out MTC.

Conclusions

Pheochromocytoma should be considered in patients with ambiguous symptoms possibly related to underlying hypercatecholaminemia, as a missed diagnosis can lead to preventable morbidity or mortality. The diagnosis of pheochromocytoma in our case proved to be a diagnostic challenge as its presentation mimicked other common ailments. This case highlighted the importance of maintaining a high-index of clinical suspicion even in the absence of the classic constellation of symptoms. A discussion with the patient regarding genetic testing should occur, given the high frequency of associated familial disease in at-risk individuals. Positive results can identify those requiring additional surveillance and lead to a better prognosis for patients and their relatives.

Subclavian Steal Syndrome in a Patient Presenting with Hypotension, Dizziness, and Frequent Falls

Cynthia Kibet, M.D., Gilbert Kisang, M.D., Kermit Rust, M.D.
KU School of Medicine-Wichita

Introduction

The subclavian steal syndrome is a phenomenon characterized by a subclavian artery stenosis located proximal to the origin of the vertebral artery. In most cases, subclavian steal is asymptomatic, with incidence/prevalence that is not well defined. Subclavian syndrome implies presence of significant symptoms due to arterial insufficiency in the brain or upper extremity. It is more common on the left upper extremity, more prevalent in men, and atherosclerosis is the most common underlying cause.

Case Presentation

A 67-year-old female with past medical history of chronic heart failure, chronic obstructive pulmonary disease, coronary artery disease status post coronary artery bypass grafting (x3), hypertension, dyslipidemia, lung cancer status post CyberKnife, and stroke presented with complaints of dizziness and frequent falls over a period of four months. She reported symptoms were brought on by activity with an accompanying sensation of light headedness. She denied any history of tinnitus, vertigo, syncope, or presyncope, however, she described sensation of disequilibrium and had several falls during this period. There was history of stroke 1 year prior to presentation with residual left-sided hemiparesis.

Upon evaluation, the patient had significant discrepancies in blood pressure measurements with diastolic in upper extremities of 75 mmHg and systolic in lower extremities of 126 mmHg. Other vital signs were within normal limits. Physical examination was largely unremarkable. The only exception noted on examination was of residual left-sided hemiparesis. Laboratory studies were notable for normal electrolytes, CBC, and procalcitonin. Chest x-ray was notable for right lower lobe atelectasis, which was the location of prior CyberKnife procedure. Carotid ultrasound was notable for retrograde flow in right vertebral artery consistent with subclavian steal. CT angiogram of the neck was notable for right internal carotid occlusion and heavy plaque at all great vessel origins. The patient underwent right brachiocephalic stent placement without complications and with improvement of symptoms. She was discharged home 2 days after the procedure.

Conclusions

Subclavian steal syndrome is a condition characterized by flow reversal in the vertebral artery, ipsilateral to a hemodynamically significant stenosis or occlusion located proximally to the origin of the vertebral artery on the subclavian artery. This condition is mostly asymptomatic, but when present symptoms are mostly due to arm ischemia in the ipsilateral arm. Neurological symptoms are even more infrequent and are due to vertebrobasilar ischemia of the brain. Though rare, vertebrobasilar ischemia should always be kept in mind in patients at risk for subclavian steal presenting with neurological symptoms.

Renal Artery Angioplasty and Stent Placement for Management of Refractory Hypertension Secondary to an Iatrogenic Flap

Uzair Mahmood, MBBS, Kamal Gupta, M.D.
KU School of Medicine-Kansas City

Introduction

New onset, refractory hypertension as a result of an iatrogenic flap following renal artery endarterectomy is a rare complication, with no definitive management guidelines currently reported in the literature.

Case Presentation

We present the case of a 65-year-old female who was referred to the cardiovascular clinic for management of severe hypertension refractory to medical management. She had recently undergone right carotid artery endarterectomy, bilateral trans-aortic renal artery endarterectomy, and bilateral infrarenal aorto-femoral bypass surgery for significant peripheral vascular disease. On a routine one-week follow-up, she reported progressively worsening headaches associated with nausea, vomiting and a transient episode of dysarthria. She was noted to have severe hypertension on initial assessment with systolic blood pressures in the 180s, in contrast to her baseline orthostatic hypotension for which she was on Midodrine and Fludrocortisone. Over the next few weeks, she was managed both in the inpatient and outpatient setting for severe hypertension refractory to maximum medical therapy. A renal artery duplex was obtained that showed right renal artery stenosis, prompting a referral to Interventional Cardiology. A small dissection flap was noted on a subsequent CT angiogram and she underwent angioplasty with bare metal stent placement in the proximal and ostial segments of the right renal artery. She had complete resolution of her previously severe and uncontrolled hypertension on follow-up visit. Post-procedural renal artery duplex did not demonstrate any evidence of stenosis and we were able to discontinue all of her antihypertensive medications.

Conclusions

This case demonstrates that endovascular stenting of the renal artery has a potential for excellent and durable results. It is a safe intervention for iatrogenic renal artery dissection, with subsequent resolution of reno-vascular hypertension.

Erythema Ab Igne from Heating Pad Use

Vijayram Reddy Malladi, M.D., William Salyers, M.D.
KU School of Medicine-Wichita

Introduction

Erythema ab igne (EAI) is a rare condition characterized by reticular, erythematous, and hyper-pigmented patches resulting from chronic exposure to external heat sources. Cases of EAI have been identified with repeated application of heating pads in the treatment of chronic pain that is associated with metastatic malignancy, pancreatitis, or peptic ulcer disease. EAI also has been reported in individuals using car heaters, coal stoves, electric heaters, steam radiators, and, most recently, laptop computers placed on the anterior thighs.

Chronic heat exposure is needed to accumulate the damages, generating an initial pattern of erythema that progresses to reticular hypopigmentation and hyperpigmentation. Sequelae include sub-epidermal bullae, diffuse hyperkeratosis, ulceration, and secondary skin malignancy, such as squamous cell carcinoma (SCC) in situ, SCC, and neuroendocrine carcinoma (Merkel cell carcinoma). Histologically, there is increased elastic tissue in the upper and mid dermis and the presence of squamous cell atypia, which resembles actinic keratoses. This case aims to discuss erythema ab igne related to the use of heating pads and to discuss the correlation of EAI to occult malignancy.

Case Presentation

A 61-year-old white male presented to the gastroenterology clinic with discolored skin over the abdomen which was present for many months. He reported mild tenderness of the skin and denied exposure to heating ducts at home or work. He admitted to using a heating pad/electric blanket off and on for several years with direct contact on the affected skin. He denied any treatment to the affected skin. Physical examination revealed non-tender, reticular, tan-brown patches with faint erythema on the different quadrants of the abdomen. The patient was advised to discontinue use of the heating pad and other sources of external heat on the skin. The patient has history of non-squamous cell lung cancer (previously treated with CyberKnife), Barrett's esophagus, recent biopsy with squamous cell carcinoma involving the bowel, concerning for atypical site of metastatic disease.

Conclusions

EAI has an excellent prognosis. The most important treatment is immediate removal of the source of infrared radiation. Topical treatment with Tretinoin and Hydroquinone has been used for persistent hyperpigmentation, and epithelial atypia may respond to topical therapy with 5-fluorouracil. Biopsy is needed if there is evidence of malignancy, such as unrelenting ulcer, infiltrated borders, or nodules.

EAI may be the first clue to an undiscovered malignancy, such as metastatic adenocarcinoma, adenocarcinoma of the rectum, or pancreatic cancer, as a heating source such as hot water bottles is used repeatedly to relieve chronic pain induced by these undiagnosed cancers. These types of malignancies are especially more suspicious when the lesions of EAI are located on the abdomen, flank, or mid back. Thorough patient history and evaluation is necessary to treat properly and follow patients with EAI.

Cannabis Coronary Syndrome - Novel Culprit in the Making?

Maha Mohamad, M.D., Sinan Khayyat, M.D., Mohinder R. Vindhyal, M.D., M.Ed.
KU School of Medicine-Wichita

Introduction

In Kansas, 8.9% of adults ≥ 26 years old smoked cannabis in the past year, between 2016 and 2017. Cannabis use is typically not regarded as a traditional risk factor for acute coronary syndrome. A few studies have reported that cannabis could promote thrombosis and initiate plaque rupture, but no definitive pathophysiology has been established. We present a case of cannabis-induced acute myocardial infarction in a 45-year-old African American, male patient.

Case Presentation

A 45-year-old, African American, male with no significant past medical history presented to the hospital with typical chest pain that started 30 minutes after he smoked cannabis. The patient's social history revealed a chronic cannabis smoker (5-6 times a day) for the last ten years. His vital signs on admission showed a temperature of 36.2^0 C, heart rate of 85/min, blood pressure of 125/80 mmHg, O_2 saturation of 98% on room air, and a BMI of 27 Kg/m^2. His cardiac exam revealed regular S1 and S2 with no murmurs, rubs, or gallops. EKG in the ER revealed STEMI in the leads V3, V4, V5 and aVL showing anteroseptal involvement. Initial troponin-I on admission was 35.15 ng/ml and gradually increased to 73 ng/ml. Admissions labs were notable for a urine drug screen being positive for cannabis. The patient underwent emergent heart catheterization, which revealed a completely occluded left anterior descending artery, including one of the diagonals. He underwent balloon angioplasty and had a drug-eluting stent placement with established TIMI-3 flow. Twelve hours post-heart catheterization, the patient suddenly developed ventricular tachycardia (VT). The VT required defibrillation to get him in back into normal sinus rhythm. He underwent transthoracic echocardiography (TTE), which revealed mild concentric hypertrophy with an ejection fraction of 35-40%. The TTE also revealed moderate akinesis of the mid anterolateral, apical, and septal segments with a mural apical thrombus. He was started on anticoagulation and remained stable during the hospital stay until discharge.

Conclusions

Cannabis can cause tachycardia, hypertension, and carboxyhemoglobinemia, which can induce myocardial infarction through thrombogenesis, arrhythmogenesis, and plaque-rupture. Even without conventional risk factors, cannabis should be reckoned as a potential cause of acute coronary syndrome. Patient education and awareness about the potentially fatal drug will help in primary and secondary prevention of cardiovascular events.

A Differential Conundrum: Severe Rhabdomyolysis in the Setting of Hypothyroidism and Suspected Inborn Error of Metabolism

Mark Quinn, M.D., Maharshi Bhakta, M.D.
KU School of Medicine-Kansas City

Introduction

Rhabdomyolysis is a severe life-threatening condition that demands focused investigation in situations without obvious insult. Rare causes such as hypothyroidism should be considered in addition to extraneous factors that produce metabolic stress (e.g., fasting and dehydration). Furthermore, molecular etiologies must be differentiated in recurrent cases of rhabdomyolysis. In addition to fluid resuscitation and electrolyte management, careful selection of medical therapy to treat the underlying insult merits thoughtful consideration.

Case Presentation

A 20-year-old male with developmental delay presented to his primary care physician with three days of fatigue, muscle weakness, and myalgias. Initial lab evaluation revealed an elevated creatinine kinase (CK) of 98,539 U/L and he was hospitalized for treatment of rhabdomyolysis. Despite three days of aggressive fluid resuscitation, his clinical picture deteriorated and his CK climbed to 188,160 U/L. He was transferred to our institution where a detailed patient history disclosed a prior episode of rhabdomyolysis as an infant. The occurrence was assumed to be the perturbation of very long-chain acyl-CoA dehydrogenase deficiency, however, a diagnosis was never confirmed and empiric treatment with carnitine supplementation was discontinued. Laboratory investigation at our institution discovered profound hypothyroidism evidenced by TSH of 125 MCU/ML and T4-free 0.5 NG/DL. Treatment with levothyroxine was initiated promptly but his clinic picture and CK did not improve. Additional investigation detected a decreased carnitine level of 20 nmol/mL and an elevated acylcarnitine to free carnitine ratio of 1.3. Levocarnitine was initiated and the following morning a profound drop in CK to 49,000 U/L was observed. His clinical and laboratory picture steadily improved thereafter, and he was discharged in stable condition on Levothyroxine and Levocarnitine. At three-month follow-up, his CK, TSH, and general chemistries were within normal limits.

Conclusions

This case illustrates that rhabdomyolysis can present with multifactorial etiology, and when evaluating for a cause of rhabdomyolysis in a young patient, it is important to consider metabolic disorders as a potential confounding etiology. Hypothyroidism itself is a rare cause of rhabdomyolysis, and levels of CK in our patient far exceeded prior case reports published in the literature. Laboratory findings pertaining to carnitine deficiency do not identify a particular metabolic etiology and are ultimately non-specific. The decision to supplement with Levocarnitine was based on the severe persistent nature of the patient's rhabdomyolysis, laboratory evidence of deficiency, and history of recurrent episodes suggestive of an underlying metabolic etiology. We emphasize that Levocarnitine should be used cautiously in patients with presumed errors of metabolism and preferably preceded by molecular diagnostic testing to guide therapy. Follow-up with a geneticist is the next appropriate step in our patient as diagnostic testing was not conducted as an inpatient.

WATS3D vs. Traditional Forceps Biopsy in Screening of Barrett's Esophagus: A Community Hospital Experience

Sachin Srinivasan, M.D., Yasmine Hussein Agha, M.D., Jeff Hyder, M.D., William Kilgore, M.D., Nathan Tofteland, M.D., William Salyers, M.D.
KU School of Medicine-Wichita

Introduction

Barrett's esophagus is a pre-malignant condition of the esophagus currently diagnosed using random 4-quadrant and targeted cold forceps biopsies during endoscopy. Demonstration of intestinal metaplasia on histopathology and the degree of dysplasia is the cornerstone of Barrett's esophagus (BE) diagnosis and management. This current method of biopsy is fraught with errors due to the randomness of sampling and experience of the operator. Wide-area transepithelial sampling with 3-dimensional computer-assisted analysis (WATS3D) is an emerging technique to collect esophageal samples and improve detection of BE. We compared WATS3D with traditional biopsy as a screening tool in BE.

Methods

The research team reviewed the charts of every adult patient that underwent BE screening with WATS3D and traditional cold forceps biopsy (FB) over the last year across community hospitals in Wichita, KS. The biopsy specimens were processed and analyzed by in-house pathologists while the WATS3D specimens were sent to CDX technology labs, NY. WATS3D specimens were obtained after FB in all instances.

Results

A total of 108 patients were identified to have undergone both WATS3D and FB at the same time for BE screening. FB detected 62 cases (57.4%) while WATS3D detected 83 (76%) cases of BE. This attributed to an absolute increase in 21 cases (18.6%) of incident BE detection by WATS3D. The number needed to test (to detect an additional patient with BE) with WATS3D was 5. We divided the sample into 4 groups: WATS3D+FB+, WATS3D+FB-, WATS3D-FB+, WATS3D-FB- to compare the agreement across the 4 groups. Overall agreement by kappa statistic was 0.74 (good). There were 62 and 23 cases that were identified as positive and negative respectively by both methods. The pathologist read both cases of FB+ that was WATS3D- as intestinal metaplasia with no dysplasia. Of the 21 cases that were FB-, WATS3D identified 15 cases of goblet cell metaplasia, 4 cases of crypt dysplasia, 1 case of low-grade dysplasia, and 1 case of adenocarcinoma. There were no immediate complications reported among the patients studied.

Conclusions

WATS3D identified 20 cases of BE missed by FB, including 1 case of low-grade dysplasia and 1 case of adenocarcinoma. It is possible that the cases missed by WATS3D were due to the presence of an island of BE that was removed during FB. Overall, with no added increase in complications, WATS3D improved incident detection of BE compared to FB alone.

Metastatic Calcification of the Myocardium, Aorta, Mitral Valve, and Lungs: A Rare Presentation of Calciphylaxis Following Acute Kidney Injury

Praveen Subramanian, D.O., Nicholas Isom, M.D., Laura Thomas, M.D.
KU School of Medicine-Kansas City

Introduction

Calciphylaxis, also known as calcific uremic arteriolopathy, can present as areas of skin necrosis in the legs, thighs, and abdomen in end-stage renal disease (ESRD) patients requiring dialysis. The reported incidence of calciphylaxis in ESRD patients is 3.5 cases per 1000 patient years with mortality reaching 50% at 12 months. The pathophysiology is not well described in the literature, but is thought to be secondary to calcification of medial and intimal walls of small arteries and arterioles. Few case reports detailing large vessel, myocardial, valvular, and pulmonary manifestations exist. This case is unique in that the multiorgan calcification along with classic skin necrosis presented in the setting of resolving acute kidney injury, where only short-term dialysis was needed.

Case Presentation

A 27-year-old male with history of methamphetamine use presented to the ED with complaints of ulcerations of his lower extremity and penis. He had a recent two-month hospitalization for severe rhabdomyolysis, which was complicated by progressive acute kidney injury requiring hemodialysis, cardiac arrest, disseminated intravascular coagulation, and empyema. Three days after discontinuation of dialysis, the patient noted ulcerations and intense pain on the legs, as well as blackening of the glans penis. Physical exam was notable for multiple necrotic appearing lesions on the thigh and a necrotic appearing glans and shaft of penis. Admission labs were notable for creatinine of 2.5, calcium 11.3, phosphorus 3.1, parathyroid hormone 16.2, vitamin D (1,25) < 5, vitamin D (25-OH) total 29. A PTH related peptide was within normal limits. CT chest, abdomen, and pelvis revealed bilateral pulmonary infiltrates with high attenuation consistent with pulmonary calcification, high attenuation involving the heart consistent with myocardial calcification, and severe calcific atherosclerosis of the moderate sized arteries of the abdomen. Transthoracic echocardiogram showed normal systolic and diastolic function, heterogeneous appearing myocardium, moderate mitral annular calcification, severe subvalvular calcification involving chordae and papillary muscles, and a sclerotic aortic valve. A punch biopsy of the right posterior thigh lesion was consistent with calciphylaxis. The patient was started on Sodium Thiosulfate for calciphylaxis treatment. Additionally, Pamidronate and Calcitonin were administered for hypercalcemia. At subsequent follow-up, there was resolution of hypercalcemia, kidney injury, and necrotic skin lesions. A cardiac MRI showed stable calcification of myocardium and mitral valve.

Conclusions

Calcification of the ascending aorta, myocardium, mitral valve, large vessels of the abdomen, and lung are rarely described in the literature as a result of calciphylaxis. To date, there is an absence of randomized controlled trials investigating the mortality benefit in evaluation of metastatic calcification in calciphylaxis. Calciphylaxis confers a high mortality even in the absence of extra-cutaneous involvement. It is prudent to evaluate patients who have extra-cutaneous complaints and symptoms with multidisciplinary teams to mitigate the sequela of metastatic calcification.

When a Deep Vein Thrombosis becomes an Emergency

Michael Sullivan, M.D.
KU School of Medicine-Kansas City

Case Presentation

A 36-year-old female presented to the Emergency Department with a one-month history of dyspnea on exertion and a two-day history of worsening left lower extremity swelling, pain, skin color change, and numbness. She has a past medical history of large uterine fibroids with abnormal uterine bleeding who had a pre-term Cesarean section performed 4 months ago. She had started a combined oral contraceptive pill one month prior. She had no prior history of thromboembolism, no family history of thromboembolism, and was not a smoker. Her vitals were stable on presentation. On exam, her left lower extremity was mottled, edematous, and erythematous. Sensation was intact throughout, however, her capillary refill was noted to be delayed in the left lower extremity. The dorsalis pedis and posterior tibialis pulses were palpated bilaterally in addition to being confirmed on the left side with doppler ultrasound. Bilateral lower quadrant masses were palpated in the abdomen. Laboratory results revealed a D-dimer of 2,216 ng/dL and anemia with a hemoglobin of 9.9 g/dL. A CT angiogram of the chest demonstrated bilateral acute segmental and subsegmental pulmonary emboli. A left lower venous ultrasound showed acute occlusion throughout the common femoral, deep femoral, popliteal, posterior tibia, peroneal, and other superficial veins. An arterial doppler ultrasound did not show evidence of hemodynamically significant stenosis. On further review of her records, she had a recent MRI that showed several very large uterine fibroids which were compressing her left iliac vein causing a May-Thurner syndrome. She was started on a heparin drip and taken to Interventional Radiology for catheter directed thrombolysis. The patient subsequently underwent a uterine artery embolization for reduction in size of her fibroids in addition to a left iliac vein stent.

Conclusions

Phlegmasia cerulea dolens is a condition characterized by acute, extensive DVT with associated marked swelling, cyanosis, and pain. It is an important entity to recognize as the condition can very quickly lead to irreversible venous gangrene, massive pulmonary embolism, shock, and death. Due to the high risk of morbidity and mortality despite anticoagulation, the treatment involves either emergent thrombolysis or thrombectomy. The etiology is also important to consider for treatment. In this case, the patient had compression of her external iliac vein from large uterine fibroids causing a May-Thurner syndrome which required an iliac vein stent and reduction in size of her uterine fibroids.

Pancytopenia and Hemolytic Anemia Secondary to Severe Cobalamin Deficiency

Andrew Weaver, M.D., Seth Page, M.D.
KU School of Medicine-Wichita

Introduction

Cobalamin (vitamin B12) deficiency is common and can cause a variety of symptoms and clinical findings. The following case demonstrated severe cobalamin deficiency leading to pancytopenia and hemolytic anemia. These are uncommon but important hematologic abnormalities associated with cobalamin deficiency.

Case Presentation

A 72-year-old white male presented with dyspnea on exertion, chest pain, and unintentional weight loss of 20 pounds over three months. He had a history of benign prostatic hypertrophy, chronic obstructive pulmonary disease, and obstructive sleep apnea. Surgical history consisted of knee and back surgeries as well as a hernia repair. His home medications were budesonide-formoterol, latanoprost, ropinirole, and tamsulosin. He had no allergies. His social history included active tobacco use, active alcohol use, and remote exposure to Agent Orange. He had a family history of heart disease. Review of systems was positive for yellowed skin. Vital signs at the time of admission were unremarkable. Physical examination revealed jaundice and mild bilateral lower extremity edema. Neurologic examination was normal.

Initial laboratory studies showed pancytopenia (hemoglobin 6.7 g/dL with mean corpuscular volume 132 fL, white blood cells 3.2 K/mm3 with absolute neutrophil count 1.7 K/mm3, and platelets 138 K/mm3), hyperbilirubinemia (4.1 mg/dL), and no fecal occult blood. Subsequent testing was notable for a predominance of unconjugated bilirubin, decreased haptoglobin (<8 mg/dL) and vitamin B12 (0 pg/mL), and increased lactate dehydrogenase (1682 U/L) and methylmalonic acid (6,160 nmol/L). Pancytopenia and rare schistocytes were seen on a peripheral blood smear. Ultimately, the patient's presenting symptoms and clinical findings were attributed to severe cobalamin deficiency, and intramuscular cyanocobalamin was initiated. Pernicious anemia was investigated as a potential cause of the deficiency, and both parietal cell and intrinsic factor autoantibodies were positive. The patient continued to receive intramuscular cyanocobalamin after hospital discharge. His blood counts normalized in three months.

Conclusions

Cobalamin deficiency is known to cause macrocytic anemia, leukopenia, and thrombocytopenia. It less commonly causes pancytopenia and hemolytic anemia. Clinicians should consider cobalamin deficiency when exploring these entities.

Shock Teams: The Future of Cardiogenic Shock

Robert Weidling, M.D., Mohamed El-Khashab, M.D.
KU School of Medicine-Kansas City

Introduction

Cardiogenic Shock (CS) remains a disease with a persistently high mortality rate despite years of effort to improve patient outcomes. Early recognition and strong multidisciplinary communication provide the opportunity for rapid institution of therapies that can mitigate mortality. The purpose of this case report is to highlight the emerging multidisciplinary team-based management approach to CS known as "shock teams".

Case Presentation

A 37-year-old African American female with history of stage D CHF secondary to NICM on home Dobutamine infusion presented with a two-day history of fatigue and dyspnea. Vital signs: BP of 80/50, RR of 28, and HR of 130. Physical exam evidenced JVD at 20 cmH_2O, cool UE, and LE and 2+ LEE. Labs showed normal creatinine and minimal elevation of transaminases as well as LA of 1.9. BNP elevated to 1,999.

The patient was admitted to the CCU with Cardiogenic Shock. RHC revealed severely decreased cardiac output with increased filling pressures: CO/CI 2.7/1.3 (fick) and 3.1/1.6 (TD), PA 63/35/34, and PCWP of 40 mmHg. Shock Team activation prompted a real-time conference call between the HF cardiologist, interventional cardiologist, cardiothoracic surgeon, and critical care physician. The joint decision was made for urgent cath lab activation for intra-aortic balloon pump (IABP) placement and escalation to ECMO if needed. Continuous PA catheter monitoring showed successful unloading of both ventricles and increase in CI to 2.6. Next day, a Heartmate 3 BTT LVAD was implanted, then she was discharged with a follow-up in the LVAD clinic.

Conclusions

CS portends an average national mortality rate of 30-40%. Newly emerging data from the National Cardiogenic Shock Initiative points towards early MCS and left ventricular offload as a vital step in curbing CS mortality. With the time sensitive nature of CS and its multimodal management, communication is of paramount importance. A multidisciplinary shock teams' primary goal is to streamline this communication process, rapidly identify CS, and expeditiously intervene with the optimal intervention.

A Shock Team is primarily composed of an advanced HF cardiologist, cardiac interventionalist, CT surgeon, and critical care physician. This multidisciplinary team is activated after a case of suspected CS is identified prompting a real-time conference call with the team members. An initial, secondary, and tertiary treatment plan is formulated jointly by the members and executed. Our case highlights the important role that shock teams play in the prompt diagnosis and treatment of CS.

Adherence to Opioid Prescribing Guidelines in an Internal Medicine Clinic

Jerrica Werner, M.D., Edward Ellerbeck, M.D., Andrea Allen, M.D., Matthew DaCunha, M.D., Ariel Johnson, M.D., Justin Theleman, M.D.
KU School of Medicine-Kansas City

Introduction

Opioid overdose has become a public health crisis with more than 130 people per day in the United States dying of opioid overdose. Prescribing guidelines have been put in place by the Center for Disease Control and Prevention (CDC). However, it is not known how well these guidelines are utilized in our institution's Internal Medicine (IM) clinic.

Methods

A retrospective review of patients from our institution's Internal Medicine clinic was conducted to evaluate the adherence to guidelines during return visits for patients who were prescribed long-term opioid therapy. Eight guidelines were selected from the "When reassessing at Return Visit" section of "Checklist for prescribing opioids for chronic pain" published by the CDC. After excluding patients on Tramadol or Methadone and patients being treated for cancer-related pain, charts of 69 of the 390 patients seen in the IM clinic, between October 2017 and October 2018 who received ≥ 8 opioid prescriptions of ≥ 20 pills from our IM clinic, were randomly reviewed for provider adherence to the 8 CDC guidelines.

Results

Mean age of patients was 61.5 (±14.2) years. Median morphine milliequivalent dose (MME) prescribed by providers was 38.5 (IQR 22.5-60.0). Attending physicians and residents in the IM clinic used a mean of 2.1 and 1.6 of the 8 selected CDC guidelines, respectively ($p = 0.45$). The most adhered to guidelines were the trialing of non-opioid therapies prior to starting opioids (87.0%) and having regular follow-up appointments defined as > 4 per year (65.2%). Other guidelines demonstrated less adherence: yearly urine drug monitoring was conducted for 20.3% of patients, the checking of local prescription drug monitoring programs (PDMP) was documented in 8.7% of visits, total MME for each patient was documented for 7.2% of patients, standardized pain/function assessment was conducted in 1.4% of visits, and assessment of overdose was conducted in 1.4% of visits.

Conclusions

Despite recommended guidelines by the CDC, Internal Medicine physicians within our institution have low adherence to opioid prescribing guidelines for return patients. Efforts to improve CDC guideline adherence could include making standardized tools such as PEG Pain Screening Tool and COMM Screen for current opioid risk easily available, as well as creating an EMR smart-phrase to standardize return visits and document CDC guidelines.

Palbociclib-Induced Fulminant Hepatic Failure

Nathaniel Parker, D.O., Rami Atallah, M.D., Karl Hamouche, M.D.
KU School of Medicine-Wichita

Introduction

In 2019, 300,000 new cases of breast cancer are estimated to be diagnosed, with almost 80% of those likely to be the invasive breast cancer type and hormone receptor-positive. Multiple drug therapies have been developed to target the predominant estrogen receptor (ER) signaling pathway used by these breast cancer types. Termed endocrine therapy, agents include aromatase inhibitors (e.g., letrozole), selective ER modulators (e.g., Tamoxifen), and selective ER down-regulators (e.g., Fulvestrant). However, certain tumor cells are able to use alternative non-estrogen dependent pathways for further growth and survival. Palbociclib has been approved for combination therapy with Letrozole or Fulvestrant to inhibit one known alternate route: cyclin-dependent kinases 4 and 6 (CDK4/6)-retinoblastoma pathway.

Case Presentation

A 79-year-old female presented to the hospital with encephalopathy and jaundice. Past medical history was notable for ER+ / PR- / HER2+ invasive ductal breast carcinoma with known bone metastasis. She was started on Palbociclib and Fulvestrant with second cycle completion date one week prior to presentation. On admission, serum elevations of AST, bilirubin, alkaline phosphatase, INR, and ammonia were noted. Roussel Uclaf Causality Assessment Method (RUCAM) and Drug-Induced Liver Injury Network (DILIN) severity scores for Palbociclib were 8 (probable) and 5+ (fatal), respectively. Sonography showed an enlarged, lobular, and homogeneous liver. There was no evidence of cholelithiasis. Based on sonography and clinical history, acute liver failure likely due to a toxic metabolite was diagnosed. A thorough immunologic and infectious serology workup was non-revealing. Despite standard of care therapies and steroids, her labs worsened and overall condition deteriorated. Supportive treatment was initiated and she expired.

Conclusions

Targeted therapies have revolutionized the landscape of oncologic treatments. The safety and efficacy of Palbociclib in combination with endocrine therapy was primarily evaluated by the PALOMA trials. These trial findings led to the drug's accelerated approval. Among all trials and Palbociclib-treated patient groups, a pooled safety analysis demonstrated that grade 3/4 liver enzyme elevations occurred in 3.3% and 2.3%, respectively. Among all PALOMA trials, the most frequently reported serious adverse event was neutropenia. Among the 1,348 patients enrolled in the three PALOMA trials, none experienced fulminant hepatotoxicity that lead to stopping drug therapy.

The diagnosis of drug-induced liver injury remains challenging. DILIN causality assessment remains a reasonable diagnostic standard. The mechanism of Palbociclib-induced liver enzyme elevation is not well understood. Palbociclib is predominantly metabolized by the hepatic CYP3A4 pathway. Thus, liver injury could be caused by the production of a toxic or immunogenic intermediate. Equally plausible, is coadministration with a strong CYP3A inhibitor, which increases the plasma exposure of Palbociclib. Serum liver enzyme elevations above 3-5 times the upper limit of normal, or any elevations accompanied by jaundice should alarm physicians and lead to temporary or permanent discontinuation of Palbociclib.

Neurosarcoid Causing Panhypopituitarism and Diabetes Insipidus

John Fritzlen, M.D., Nelli Lakis, M.D., John Leever, M.D., Yasir Jassam, MBChB, MRCP
KU School of Medicine-Kansas City

Introduction

Sarcoidosis is a granulomatous inflammatory condition with a prevalence that approximates 100/100,000 but varies with region, race, and sex. It affects the lungs in 90-95% of cases but extrapulmonary sarcoidosis also occurs with neurologic involvement accounting for approximately 3-5%. About half of those cases have neurological changes on presentation. We present a case of neurosarcoidosis that caused panhypopituitarism, diabetes insipidus, and adrenal crisis.

Case Presentation

A 45-year-old Hispanic female with a history of hyperlipidemia presented to an outside hospital for worsening fatigue, progressive visual loss, and polyuria. Initial laboratory revealed a sodium of 166 mEq/L and MRI Head showed a homogeneous mass centered in the hypothalamic region. She was transferred to our hospital where laboratory revealed a TSH 0.060 mIU/L, FSH 2.0 mU/mL, LH < 0.2 mU/mL, urine osmolality 97 mOsm/kg and urine sodium < 10 mmol/L. She was started on Desmopressin, Hydrocortisone, and Levothyroxine. Stereotactic biopsy which showed perivascular and intraparenchymal lymphocytes, forming small aggregates. PET scan showed increased uptake in the hypothalamus, cervical and inguinal lymph nodes, and in the left femur. Lymph node biopsy, bone marrow biopsy, and cytology showed no immunophenotypic evidence of lymphoma. She was scheduled for outpatient excision biopsy of the femur; however, prior to the biopsy, she presented to the emergency room complaining of nausea, vision loss, and hypothermia. She was lethargic, hypotensive, hypothermic, and hypernatremic concerning for adrenal crisis. Neurological examination revealed bitemporal hemianopsia. MRI showed significant increase in size of the hypothalamic mass with extensive edema surrounding the optic tracts. She received stress dose steroids. Neurosurgery performed repeat stereotactic biopsy which revealed perivascular and intraparenchymal lymphocytes with no atypical morphology. A small cluster of macrophages was present suggesting an autoimmune phenomenon or sarcoidosis. Neurology diagnosed her with neurosarcoidosis and started on high dose steroids. A week later, she required admission for increased lethargy, odd behavior, loss of inhibition, and hypernatremia. She was treated with high dose Solumedrol and her symptoms improved. Repeat MRI showed further reduction in size of the hypothalamic lesion with persistent marginal T2 hyperintensity involving the hypothalamus and intracranial optic pathway. A lumbar puncture revealed WBC 5, RBC 1, total protein 84 mg/dL, glucose 120 mg/dL, and oligoclonal bands were negative. A paraneoplastic panel was negative and serum aldolase was normal. She slowly improved before being discharged to rehab.

Conclusions

In this case, neurosarcoidosis preferentially involved the hypothalamus leading to the derangement of the hypothalamic-pituitary-axis and progressive vision loss. The final diagnosis was elusive and delayed due to evaluation of potential hematological malignancy, confounding PET imaging, and non-specific biopsies. The lack of pulmonary findings typically seen in > 90% of patients with sarcoidosis may have also reduced the initial clinical consideration for neurosarcoidosis.

Cardiopulmonary Arrest in a Type 1 Diabetic Female Secondary to Hypoglycemia Associated Autonomic Failure: Dead in Bed Syndrome

Anam Abbasi, M.D., Jeet Mehta, M.D., Lina Huerta-Saenz, M.D.
KU School of Medicine-Wichita

Case Presentation

A 41-year-old Caucasian female with T1D on multiple dose insulin injection therapy (MDI) presented via EMS status-post cardiopulmonary arrest. Her family found her unconscious in an undisturbed bed. Blood sugar was 30 mg/dL and no seizure-like activity was witnessed. EMS found the patient in pulseless electrical activity. Seven rounds of CPR were performed, and one dose of 1 mg intramuscular epinephrine was administered before return of spontaneous circulation. The patient was intubated, sedated, and transported to the emergency department. Her past medical history included peripheral neuropathy and diabetic nephropathy. On arrival, she was tachycardic, tachypneic, and hypotensive. Her body mass index was 36. ECG showed sinus tachycardia with no ST-segment changes or QT prolongation. Labs showed hypoglycemia, severe metabolic acidosis with profound lactic acidosis. A1c was 4.7%. Echocardiogram was unremarkable. CT chest showed right upper lobe infiltrate. She met sepsis criteria. She was started on broad-spectrum antibiotics and intravenous fluid resuscitation including bicarbonate drip. Daily chest x-rays showed the infiltrate improved rapidly suggesting pneumonitis. She was extubated after five days of ventilator support. Continuous electroencephalogram showed no seizure activity. She resumed basal and short-acting insulin with dose adjusted. Further investigation revealed that she had asymptomatic hypoglycemia for the past two years. TSH, free T4, and morning cortisol levels were normal. As other differential diagnoses revealed no etiology of patient's cardiac arrest, her symptoms and presentation were attributed to HAAF. She was discharged after six days with return to normal baseline neurological function.

Conclusions

This case illustrated HAAF as a potential cause of sudden death in patients with type 1 diabetes and hypoglycemic unawareness. Authors have postulated that recurrent hypoglycemia attenuates typical physiologic adrenal response and failure to increase glucagon due to alpha cell failure. There are no guidelines for management and prevention of HAAF. However, the American Diabetes Association recommends that in patients with frequent nocturnal hypoglycemia, recurrent severe hypoglycemia, and/or hypoglycemia unawareness, a sensor-augmented low glucose threshold suspend pump may be beneficial. Patients and their families should recognize that hypoglycemia can occur without sympathetic symptoms and institute timely interventions to prevent sudden death.

Immunoglobulin G-4 Related Dacryoadenitis

Ethan Alexander, M.D., Branden Comfort, M.D., MPH
KU School of Medicine-Kansas City

Introduction

Immunoglobulin G-4 (IgG4) related diseases are a collection of autoimmune inflammatory diseases with varying clinical presentations and clinical courses. Common pathologic manifestations of IgG4 related diseases are autoimmune pancreatitis, sclerosing cholangitis, salivary disease, and inflammatory processes in the orbit. Inflammatory processes in the orbit can easily be misdiagnosed as more common pathology which could lead to a delay in appropriate treatment unknowingly. Here we present a case of suspected IgG4 related dacryoadenitis.

Case Presentation

The patient is a 54-year-old female who presented for an urgent care visit for multiple weeks of left upper eyelid swelling. This was diagnosed as a chalazion and was treated symptomatically. Two weeks later the patient returned to the clinic with worsening swelling and new left eye semi-purulent drainage and pain with all extraocular eye movements. Due to concern for orbital cellulitis, the patient was sent for an emergent maxillary/facial CT. This revealed extensive peri-orbital inflammation around the left lacrimal gland. No fluid collections or intra-orbital changes were seen. The patient was admitted to the hospital and was started on broad spectrum intravenous antibiotics. There was no significant change in her symptoms or physical exam after 24 hours. An autoimmune workup was initiated with serum measurements of the erythrocyte sedimentation rate, complete blood counts, anti-nuclear antibodies, ACE, c-ANCA, p-ANCA, and Ig4/IgG. The only remarkable lab value was an elevated IgG4. The patient was discharged on a high dose steroid taper for likely IgG4 related dacryoadenitis. A biopsy was planned within a week of discharge. Due to underlying psychosocial factors, the patient was lost to follow-up.

Conclusions

IgG4 related autoimmune diseases are varying in their clinical manifestation and presentations. IgG4 dacryoadenitis can present in a similar manner to common outpatient diagnoses like chalazion, hordeolum, or blepharitis. Clinicians should keep IgG4 related diseases on the differential for presentations of pancreatitis, head and neck complaints, and ophthalmologic complaints, especially if the patient does not improve with the standard treatment for the initial diagnosis.

A Case of Anti-Jo-1 Antisynthetase Syndrome with Interstitial Lung Disease

Daly Al-Hadeethi, M.D., Ammar Al-Obaidi, M.D., Grace Nassim, M.D., Janel Harting, M.D.
KU School of Medicine-Wichita

Introduction

Antisynthetase syndrome (AS) is a rare autoimmune disease described in 1990. It is characterized by systemic manifestations such as myositis, interstitial lung disease (ILD), and chronic polyarthritis. It is associated with fever, mechanic hands, and Raynaud's phenomenon. The presence of anti-aminoacyl-tRNA synthetase (anti-ARS) antibodies is highly predictive of ILD. Anti-Jo-1 is the most common anti-ARS antibody.

Case Presentation

A 67-year-old female had an abnormal chest x-ray three years prior, during hip replacement surgery. She had a chest CT at that time which showed mild interstitial abnormalities. She was asymptomatic, so she had no further workup. One year later, she had progressive shortness of breath with exercise along with inflammatory polyarthralgia of the small joints, bilateral palmar erythematous hyperkeratosis, and prolonged fever. Repeated chest x-ray showed progressive interstitial changes. A follow-up CT chest with contrast showed progressive interstitial lung disease. Pulmonary function tests showed a predicted total lung capacity of 85%, but a diffusing capacity that was more impaired at 47% of predicted. Serological evaluation demonstrated that she was ANA and anti-Jo1 positive with a positive anti-SS-A/SS-B and a positive rheumatoid factor. The patient was clinically asymptomatic without any features of an autoimmune process. The pulmonologist elected to pursue VATS biopsy. The lung pathology confirmed that she had usual interstitial pneumonitis due to an autoimmune process with features of abundant small airways bronchiolitis, and more significant parenchymal inflammation. She was started on Prednisone. Despite steroids, the lung function continued to deteriorate. Her predicted total lung capacity declined to 56% and diffusing capacity dropped to 30% of predicted. She was started on Mycophenolate and the Prednisone was increased. The Prednisone bursts helped her increase FVC to 63% predicted. The Mycophenolate was gradually titrated up to the dose of 1,000 milligrams twice daily.

The patient was referred for a second pulmonology opinion. Diagnosis of amyopathic anti-Jo1, SS-A positive antisynthetase syndrome was made. The plan was to start the patient on IVIG and later stop the Mycophenolate, unfortunately her insurance did not cover the IVIG so treatment with Rituximab was started.

Conclusions

More recently described anti-ARS antibodies might confer a phenotype that is distinct from that of anti-Jo-1-positive patients and is characterized by a lower incidence of myositis and a higher incidence of ILD. The concomitance of anti-Jo-1 and anti-Ro/SSA antibodies is linked with a more severe, therapy-resistant form of ILD.

In many patients with AS syndrome-related ILD, the onset of dyspnea is gradual occurring over a matter of months. However, in a subset of patients, the onset of ILD, fever, and respiratory insufficiency is abrupt occurring over a few days or weeks. The coexistence of anti-Jo-1 and anti-Ro/SSA antibodies is associated with a more severe, rather therapy-resistant form of pulmonary involvement.

Cutaneous Nocardiosis in an AML Patient Treated with Azacitidine (Vidaza®)

Mejalli Al-Kofahi, M.D., Laura Thomas, M.D.
KU School of Medicine-Kansas City

Introduction

Nocardia is a gram-positive obligate aerobic filamentous branching bacterium that is a part of the normal soil microflora. It is transmitted via inhalation or direct inoculation, affecting lungs, integument, and brain. We present a case of primary cutaneous nocardiosis in an Acute Myeloid Leukemia (AML) patient on Azacitidine.

Case Presentation

A 69-year-old male presented with medical history of renal disease on hemodialysis, treated testicular cancer, therapy-related AML in complete remission on Azacitidine maintenance therapy, diabetes mellitus. Patient developed abrasions and puncture wounds to his forehead and nasal bridge after a fall. Lesions progressed over 2 months forming painful pustules with purulent discharge despite topical Bacitracin, Cephalexin, and Doxycycline. He was admitted for refractory cellulitis. Pertinent physical exam showed multiple pustules with erythema on forehead and nasal bridge. CBC and CMP were at baseline compared to the last 3 months with resolution of neutropenia. He was started on Cefepime and Daptomycin. A culture swab was obtained and remained negative throughout that one-week hospital stay. Pustules slightly improved and he was discharged on oral Doxycycline. Twelve days after collection, the swab grew Nocardia brasiliensis susceptible to Trimethoprim-Sulfamethoxazole (TMP/SMX). Shave biopsy obtained a day prior to discharge showed branched filaments in the dermis and he was started on TMP/SMX as an outpatient.

Two weeks later, patient suffered another fall prompting admission. Significant improvement of lesions was noted. CT head, chest, abdomen, and pelvis showed multiple left-sided rib fractures, and scattered ground glass and centrilobular opacities, greatest in the right lower lobe (RLL). Bronchoscopy with RLL bronchoalveolar lavage showed within range cell count, negative respiratory viral panel (RVP), fungal culture, acid-fast stain, and PCR for Pneumocystis jiroveci, Mucorales, Aspergillus fumigatus, Aspergillus terreus, and Herpes Simplex 1 and 2. Nocardia PCR from the BAL was inconclusive due to endogenous inhibition. Lesions continued to improve, and he was discharged to finish 6-12 months of TMP/SMX.

Conclusions

Nocardiosis incidence is not well reported, one estimate quoted 500-1,000 cases per year in the U.S. Risk factors include immunosuppression, HIV, cancer, and diabetes. Cutaneous nocardiosis is caused by the less common N. brasiliensis, and is either primary due to direct inoculation, or secondary due to dissemination from a pulmonary or a CNS source. It manifests as indolent cellulitis, abscess, pustules, ulcerations, or mycetoma and mimics common pathogens such as staphylococcus aureus and streptococcus pyrogens. Herein lies the diagnostic difficulty of Nocardia, especially that it requires 2-5 days of inoculation. This is consistent with our case as the patient had AML and was receiving immunosuppression with Azacitidine. Cutaneous Nocardia is often cured with a prolonged course of TMP/SMX. CNS and pulmonary involvements need to be ruled out in most cases due to increased mortality with disseminated disease.

Spontaneous, Loculated, and Massive Hemothorax:
An Uncommon Complication of Warfarin Therapy

Ammar Al-Obaidi, M.D., Daly Al-Hadeethi, M.D., Sinan Khayyat, M.D., Quoc Truong, M.D., Alaeldin Mohammed, M.D.
KU School of Medicine-Wichita

Introduction

A hemothorax is a pleural effusion with a hematocrit of at least 50% of that in peripheral blood. Warfarin is a commonly used oral anticoagulant associated with several adverse drug reactions including bleeding. Important risk factors for major hemorrhage due to warfarin therapy include a history of gastrointestinal bleeding, concurrent use of antiplatelet or nonsteroidal anti-inflammatory drugs, genetic differences in warfarin metabolism, INR variability, comorbid illnesses, and duration of oral anticoagulant therapy. Thoracic hemorrhage accounts for approximately 3% of all hemorrhagic complications associated with warfarin therapy and is usually related to trauma. A small number of spontaneous hemothorax cases that formed due to anticoagulant therapy have been reported.

Case Presentation

A 64-year-old man had a supratherapeutic INR of 13.31 and a prothrombin time of 167.5 seconds during routine checkup. He was asymptomatic but had tachycardia and was hypotensive. Past medical history included chronic atrial fibrillation on warfarin, stroke, hypertension, diabetes, and end stage renal disease on dialysis. Warfarin was held and vitamin K given. The hemoglobin was 6 g/dL and the patient received blood. No visible source of bleeding was identified. Imaging revealed massive left-sided pleural effusion. The patient was breathing comfortably and denied history of trauma. A 4-factor prothrombin complex concentrate was given to reverse the INR. A chest tube drained 650 ml of dark sanguineous output under suction. Subsequent chest x-ray showed persistent opacity over the left lung field and repeat CT scan revealed large loculated hemothorax. tPA was administered through the chest tube with minimal output. Video-assisted thoracoscopic surgery extended to a full thoracotomy with decortication and left lower lobe wedge resection due to thick rind and left lower lobe air leak. Pleural fluid was grossly bloody. Cytology was negative. No diagnostic features of granulomas, dysplasia, neoplastic lesions, or significant acute inflammation were identified with tissue biopsy.

Conclusions

Pulmonary diseases including pleural pathologies are considered significant risk factors for developing nontraumatic hemothorax in the setting of anticoagulation. Our case had no previous pulmonary disease including pleural or pulmonary malignancies and pulmonary embolism. No aortic dissection or other hematologic conditions were diagnosed. Hemothorax is a major indication for tube thoracostomy, particularly in cases with mediastinal shift. However, accelerated drainage may cause significant hypotension and supratherapeutic INR levels should be evaluated carefully. Massive hemothorax can present spontaneously in patients on oral anticoagulant therapy, especially in patients with supratherapeutic INR. Correction of the coagulopathy should be the focus in treating anticoagulation associated hemothorax. Cessation of anticoagulant treatment must be the first step followed by pharmacologic interventions then consideration of tube thoracostomy. Surgical intervention is dependent on individual patient circumstances.

When Leukemia Changes Colors: A Case of Lineage Switch in Acute Leukemia

Joseph Bennett, M.D., Sunil Abhyankar, M.D., Janet Woodroof, M.D., Anurag Singh, M.D.
KU School of Medicine-Kansas City

Introduction

Acute Lymphoblastic Leukemia is one of the most aggressive hematologic malignancies which often carries high relapse rates. Despite aggressive interventions, many patients still develop relapsed disease. The vast majority of relapsed cases show the same morphology, immunophenotype, and genomic expressions as the original leukemia. In rare instances, patients may develop a complete lineage switch from acute lymphoblastic leukemia to acute myeloid leukemia.

Case Presentation

A 61-year-old female presented to the hospital with a chief complaint of malaise and fatigue. Initial labs showed a white blood cell count of 300k. Bone marrow aspiration confirmed a diagnosis of B lymphoblastic leukemia with 100% cellularity and 95% blasts. Concurrent flow cytometry on bone marrow samples showed minor involvement of B-lymphoblastic leukemia/lymphoma. Cytogenetics showed 46,XXt(4;11)(q21;123)[5]/46,XX[15]; 15 metaphases were normal and 5 had a 4;11 translocations with breakpoints at q21 and q23 respectively. She was started on multiagent chemotherapy induction as a part of a clinical trial. After the initial induction chemotherapy, she was found to have 'measurable residual disease' (MRD) on bone marrow evaluation and continued chemotherapy. Months after induction, the patient was found to have relapsed ALL and was started on Inotuzumab. After 2 rounds of Inotuzumab, she developed leukocytosis and a repeat bone marrow examination showed hypercellular bone marrow (40-80%) with 47% blasts with a monocytic differential (55% of total), suggestive of acute myeloid leukemia. No neoplastic lymphoblasts were detected. Cytogeneic analysis at this time showed 46,XX,t(4;11)(q21;q23)[15]/46,XX[5]. Remarkably, this genomic profile had breakpoints at q21 and q23 which was consistent with the genomic profile found at the time of initial diagnosis. Induction was then started with liposomal daunorubicin/cytarabine (CPX-351) to appropriately treat the change from lymphoblastic to myelogenous leukemia. The next bone marrow biopsy on day 14 showed an aplastic bone marrow.

Conclusions

Lineage switch in acute leukemia is a rare and often confounding clinical presentation. When a patient relapses, the importance of repeat immunophenotypic and genomic testing is paramount. Usually, the relapsed blasts adhere to original lineage, often with features of further clonal evolution. With cases of lineage switch, repeat work-up must show the retention of a genetic signature common between the original and the relapsed, secondary leukemia. The mechanism of lineage switch is unclear but current theories involve bipotential progenitor cells or cell reprogramming. In our case, it is also not clear if targeting CD22 by Inotuzumab resulted in the selection of this new clone. In the event of lineage switch, the selected therapeutics will often change and delayed diagnosis with undoubtedly affect clinical outcomes. Our case highlights the importance for clinicians to be aware of this phenomenon so that delays in the patient care can be avoided.

Empyema due to Colopleural Fistula as a Complication of Metastatic Cholangiocarcinoma

Alec Britt, M.D., Brice Zogleman, M.D.
KU School of Medicine-Kansas City

Introduction

Empyema is a condition in which pus collects in the pleural space, the incidence of which is increasing in the United States. It is most often associated with pneumonia or trauma, and necessitates tube thoracostomy for drainage. Organisms associated with development of empyema are those implicated in many pneumoniae, including Staphylococcus aureus Streptococcus pneumoniae. Fistulous formation from the GI tract to the pleural space is a rare complication of malignancy or inflammatory bowel disease, with colopleural fistula being exceedingly rare in the literature. Here we describe a patient who presented with empyema in the setting of a widely metastatic cholangiocarcinoma.

Case Presentation

A 48-year-old African American woman with a past medical history of metastatic cholangiocarcinoma presented to the emergency room for acute onset shortness of breath and pleuritic chest pain. Physical exam yielded absence of right-sided breath sounds. Vital signs were significant for tachycardia and hypoxia, with an oxygen saturation of 88% on room air. Chest x-ray imaging in the emergency room showed a large, right-sided hydropneumothorax, for which a chest tube was placed with drainage of gross pus. Blood cultures were drawn, the empyema was cultured, and the patient was started on broad spectrum antibiotics. The following day, the patient subsequently decompensated with tachycardia and hypoxia when changed to water-seal on her chest tube. Her vitals and clinical status improved after being placed back on continuous suction. The patient continued to have significant output from the chest tube, which appeared and smelled feculent. Blood cultures returned with Escherichia coli, and chest tube output grew Enterococcus faecium, α-hemolytic Streptococcus species, and E coli. Given these findings, a CT scan of the chest, abdomen and pelvis were obtained, revealing a right-sided colopleural fistula arising from a necrotic hepatic flexure. The patient was considered a poor surgical candidate given her widespread metastatic disease and decreased functional status. The patient was maintained on continuous chest tube suction and broad spectrum antibiotic coverage throughout her hospital stay. She eventually developed Candida glabrata fungemia and worsening sepsis despite broad spectrum antibiotics and antifungal treatment, and the patient expired in the hospital.

Conclusions

There is a dearth of data on colopleural fistulas as a complication of gastrointestinal malignancy. Our patient presented with widespread cholangiocarcinoma, including colonic and peritoneal metastases. She likely suffered a colonic perforation as a complication of tissue necrosis, which then developed a fistulous tract with the right hemidiaphragm. The current management for fistulas between the GI tract and pleural space involves surgical evaluation and closure of the fistulous tract, as well as surgical management of the underlying gastrointestinal source. For patients who are not surgical candidates, this leads to significant difficulties in management, and likely poor outcomes.

Psoriatic Arthritis: A Diagnosis in the Details

Christopher Bryant, D.O., Brent Duran, D.O.
KU School of Medicine-Wichita

Introduction

Psoriatic arthritis remains a relatively overlooked diagnosis. Nonspecific features and variable disease severity often lead to delayed or incorrect diagnosis, ultimately delaying appropriate treatment. Delayed treatment of psoriatic arthritis leads to morbidity not only from disease progression, but from associated comorbidities such as cardiovascular disease, diabetes, and psychiatric illness. Judicious analysis of the patient's history, along with appropriate utilization of diagnostic studies and tools may yield early diagnosis and treatment.

Case Presentation

A 62-year-old male presented to the emergency department with one-week duration of right fifth toe erythema, pain, and swelling. History and examination led to a diagnosis of cellulitis; a 10-day course of Doxycycline was prescribed. The patient failed to improve after one week and returned to the ED. History remained negative for constitutional symptoms and vitals were within normal limits. Examination revealed a uniformly swollen, erythematous right fifth toe with a mildly painful proximal interphalangeal (PIP) joint. Range of motion of the right fifth phalanx was limited. Onycholysis and nail pitting was noted on all toes. The right foot dorsum was edematous. An erythematous appearing thin plaque was noted on the dorsal left foot.

Laboratory studies revealed an unremarkable CBC, CMP, and serum uric acid. C-reactive protein was slightly elevated. A three-view x-ray of the right foot showed fifth middle phalanx subluxation at the PIP joint, bony erosions with overhanging margins involving the medial and lateral aspects of the fifth middle phalanx, and soft tissue swelling in the fifth toe and over the dorsal foot.

The ED physician diagnosed the patient with osteomyelitis. Prior to formal admission, evaluation revealed chronic dry, scaly, pruritic lesions on the posterior neck, upper back, and dorsal left foot. The patient reported one similar episode of diffuse digital pain and swelling involving the finger three years prior along with mild pain and morning stiffness involving small joints of the hands, treated with ibuprofen. A working diagnosis of psoriatic arthritis was concluded and the diagnosis was confirmed by rheumatology.

Conclusions

Psoriatic arthritis can be diagnosed in patients with both psoriasis and inflammatory arthritis, after exclusion of mimics such as gout, osteoarthritis, and rheumatoid arthritis. Laboratory analysis should reflect systemic inflammation and exclude other types or arthritis. Plain radiographs should be obtained in suspected psoriatic arthritis to assess for destructive changes; occasionally, MRI may be obtained to assist in the diagnosis. The Toronto Psoriatic Arthritis Screen may be used to determine if further rheumatologic evaluation is warranted. The International Classification of Psoriatic Arthritis may be applied to those with confirmed inflammatory arthritis with 91-100 percent sensitivity and specificity. While screening and classification systems were not employed in our case, utilization of such tools likely would have suggested a rheumatologic versus infectious etiology.

Vitamin B12 Deficiency with Pseudothrombotic Microangiopathy

Scott Buess, M.D., Alexander Germann, MS4, Eamon Maloney, M.D.,
Alaeldin Mohammed, M.D.
KU School of Medicine-Wichita

Introduction

Thrombotic microangiopathy (TMA) encompasses severe conditions caused by microvascular thrombosis including thrombotic thrombocytopenic purpura (TTP) and hemolytic uremic syndrome (HUS). These disorders, associated with high morbidity, share unifying features: microangiopathic hemolytic anemia (MAHA), thrombocytopenia, and organ dysfunction. Cases of metabolism mediated TMA from vitamin B12 deficiency have been reported, termed pseudothrombotic microangiopathy (pseudo-TMA). Differentiating the etiology of TMA is critical as the treatments and prognoses are different. Unnecessary plasma exchange has been reported due to the misdiagnosis of TTP when the actual diagnosis was vitamin B12 deficiency. We report a case of a man presenting with profound pancytopenia in the presence of schistocytes concerning for TTP that was actually secondary to vitamin B12 deficiency.

Case Presentation

A 65-year-old male presented to the emergency room after laboratory evaluation at his primary care provider's office revealed pancytopenia. Labs showed WBC 1.6 K/cmm, Hgb 4.8 g/dl, platelet 34 K/cm, MCV 121.3 fL, reticulocyte count 3.8%, total bilirubin 1.6 mg/dL, LDH 3449 units/L, Cr 0.76 mg/dL, haptoglobin < 8mg/dL, and vitamin B12 66 pg/mL. A peripheral blood smear showed 4% schistocytes, 2+ teardrop cells, and hypersegmented neutrophils. Coomb's test was negative. His iron studies and folate levels were within normal limits. Coagulation studies were within normal limits. The patient was transfused with packed red blood cells. A diagnosis of vitamin B12 deficiency was made and treatment with intramuscular vitamin B12 initiated. His ADAMS 13 activity later returned as 100% activity. He was treated with intramuscular vitamin B12 and follow-up as an outpatient showed complete resolution of his anemia 4 months later.

Conclusions

The incidence of TTP is less than one case per year per million. The most frequent serum vitamin B12 cut-off level to diagnose vitamin B12 deficiency is 150 pmol/L with an estimated prevalence of 5% to 15%. Approximately 2.5% of vitamin B12 deficiency cases present with hemolytic anemia, thrombocytopenia, and schistocytosis. Therefore, even though a small proportion of vitamin B12 presents with MAHA, the incidence is higher than TTP. Thrombocytopenia in the presence of schistocytes on the peripheral blood smear suggest DIC, TTP, and HUS. We propose that vitamin B12 deficiency be added to this list as pseudo-TMA is actually more common than the other aforementioned etiologies.

Obtaining the correct diagnosis is possible if the clinician understands the clinical differences and differing mechanisms. Vitamin B12 deficiency typically presents with an elevated MCV and relatively low reticulocyte count, whereas TTP may or may not present with an elevated MCV and very elevated reticulocyte count. MCV greater than 115 fL is consistent with megaloblastic anemias. Vitamin B12 deficiency typically presents with relatively higher levels of LDH and lower levels of bilirubin compared with TTP.

Primary Sporadic Gastric Burkitt Lymphoma Presenting with Melena

Brandon Calhoun, D.O., Praveen Subramanian, D.O., Daniel Vestal, M.D.
KU School of Medicine-Kansas City

Introduction

Primary gastric lymphoma (PGL) represents a rare cancerous lesion, with an incidence ranging from 4-20% of non-Hodgkin's B cell Lymphoma (NHL) and 1-4% of gastrointestinal related neoplasms. Interestingly, the stomach is the most common extranodal site of NHL, which constitutes about 30-40% of cases. We present a case in which an African American gentlemen presented with a history of melena and subsequently found to have primary sporadic gastric Burkitt's lymphoma (BL). Although, PGL represents a small percentage of gastrointestinal (GI) related neoplasms, it is important to have this particular disease on the differential diagnosis for an upper GI bleed as early detection, and ultimately treatment, can improve survival.

Case Presentation

A 63-year-old African-American male with history of prostatic adenocarcinoma (Stage IIB, GS 8), hypertension, hyperlipidemia, and recent pulmonary embolism on Apixaban presented with a two-week history of melena and a 30-pound weight loss within 6 months. Two weeks prior, the patient started having maroon colored stools that eventually progressed to melena, which prompted him to report to the hospital. At time of presentation, he denied any fever, night sweats, early satiety, abdominal pain, nausea, or emesis. His physical exam was largely unremarkable with the exception of melenic stool on digital rectal exam. Hemoglobin on admission was 13.5 g/dL. Gastroenterology was consulted for possible procedural evaluation for an upper gastrointestinal bleeding source. Esophagogastroduodenoscopy (EGD) was performed which demonstrated a 1.5 cm by 1 cm mass located within the gastric body. Biopsies were taken of the stomach lesion for further histopathological analysis. Immunohistochemical stains of the gastric mucosal biopsy demonstrated CD20+, CD10+, c-Myc+, Bcl-6+, CD3-, CD5-, and Bcl-2-. Ki-67 immunostain showed 100% staining in the lymphoid cells. Additionally, FISH detected c-Myc translocation in 75% of interphase nuclei however did not detect Bcl-6 or IgH/Bcl2 translocation. Given this patient's immunohistochemical findings, Hematology/Oncology was consulted. Positron emission tomography (PET) scan showed a focal increase in activity in the lesser curvature and along gastroduodenal region. Ultimately, the patient was diagnosed with primary sporadic gastric Burkitt Lymphoma. The patient underwent 3 cycles of Rituxan, Etoposide, Cyclophosphamide, Doxorubicin, Vincristine, and Prednisone (R-ECHOP) with a future plan of 2 cycles of high dose Methotrexate. Repeat PET scan at one month from diagnosis was negative for residual hypermetabolic foci. Additionally, his repeat EGD revealed a completely heled mucosal ulcer and biopsy was negative for lymphoma.

Conclusions

Overall, this case illustrates a rare case of Burkitt's Lymphoma presenting as melena. Although, primary gastric lymphoma represents about 1-4% of gastrointestinal related neoplasms, it is important to have this particular disease on the differential diagnosis for an upper GI bleed as early detection, and ultimately treatment, can improve survival.

Advance Care Planning Rates in The University of Kansas Health System Cancer Clinics

Stacie Carlson, M.D., Megan Haghnegahdar, M.D.
KU School of Medicine-Kansas City

Introduction

Advance care planning (ACP) remains under-utilized in late-stage (III and IV) cancer patients, despite improving patients' understanding of prognosis and reducing futile delivery of healthcare services at end-of-life. We sought to characterize rates of ACP at The University of Kansas Health System (TUKHS) outpatient cancer centers to lead to better utilization of ACP. ACP consists of: Advance Directives (AD), Durable Powers of Attorney (DPOA), Transportable Physician Orders for Patient Preferences (T-POPP).

Methods

Retrospective chart review of 630 advanced stage cancer patients seen at TUKHS Outpatient Cancer Center Clinic in 2016 was conducted. Records were examined from initial presentation to a TUKHS oncology provider until death or January 31, 2019. Demographics, primary cancer diagnosis, age of presentation, oncologist, oncology clinic, and ACP documentation were collected. We considered AD, DPOA, and T-POPP when identifying rates of ACP. ACP rates were characterized by clinic, provider, cancer type, and ACP type.

Results

Our sample was composed of 54.1% males and 85.6% white/Caucasians. Average age of first oncology visit at TUKHS was 63.6 years. 4.6% had a T-POPP, 27.3% had DPOA, and 13.6% had an AD. 69% of patients had no form of ACP; 31% had any of the three forms, 17.6% had one, 12% had two, and 1.3% had all three. Clinic A had the highest DPOA rate of 31.23%, Clinic E had the highest AD rate of 19.23%, and Clinic D had the highest T-POPP rate of 6.08%.

Conclusions

ACP rates varied by oncology clinic location and were overall higher than some reported national statistics of 10%; however, there is still a lot of variability in ACP completion by providers, which gives opportunity for process improvement. We recommend interventions to improve ACP documentation including: medical record checks for ACP documents prior to clinic visits, having on-site social work to assist with these documents, and the change in reimbursement for ACP.

Pulmonary Paragonimiasis

Jonathan Chandler, D.O., Lisa Clough, M.D.
KU School of Medicine-Kansas City

Introduction

Hemoptysis is a relatively common hospital presentation that requires an extremely broad differential. We report a case of a recently immigrated young male that presented with hemoptysis and ultimately found to have pulmonary paragonimiasis.

Case Presentation

A 25-year-old Burmese male with no past medical history presented to the emergency department because of an abnormal CT scan of his chest. The patient had been suffering from pleuritic chest pain, shortness of breath, and scant hemoptysis for the past year. Approximately six months prior to presentation, he had immigrated from Burma. He had reportedly been tested for tuberculosis after immigration and was told it was negative. His initial exam was relatively unremarkable for any significant findings. Notable laboratory evaluation revealed a white blood cell count of 9.4 and 8% eosinophils, and an elevated IgE level to 1330. CT scan of the chest with contrast revealed left upper lobe nodular consolidations with surrounding ground glass infiltrates, several cystic/cavitary structures, background diffuse mosaic attenuation, and concern for connection to the central venous pulmonary vasculature consistent with a pulmonary arteriovenous malformation. After 3 acid fast bacilli sputum samples were negative, the patient underwent bronchoscopy with a large infectious workup. The bronchoalveolar lavage (BAL) fluid revealed eggs consistent with paragonimiasis. Treatment was initiated with Praziquantel after discussion with the Center for Disease Control (CDC) and a local parasite expert. The patient tolerated the three-day treatment well and was seen in Infectious Disease clinic 3 weeks after discharge, with complete resolution of his symptoms. Special serologic testing sent to the CDC was ultimately confirmed to be positive for paragonimiasis.

Conclusions

Patients presenting with hemoptysis and peripheral eosinophilia require a broad differential that includes infectious etiologies (helminth, fungal or mycobacterial etiologies), eosinophilic pneumonias, vasculitis, allergic bronchopulmonary aspergillosis, and hypereosinophilic syndromes, among others. Paragonimus is a fluke classically manifesting as a lung infection in individuals that have consumed undercooked shellfish. While most infections occur in immigrants from endemic areas of P. westermanni (primarily in Asian countries), several cases have been reported in individuals consuming undercooked crawfish containing the North American fluke P. kellicotti, during float trips in Missouri. After the parasite is consumed, it travels through the duodenal wall, into the peritoneal cavity, then through the abdominal wall and diaphragm, and into the lungs. Infectious symptoms of the acute phase can include cough, abdominal pain, and low-grade fever with chronic infections manifesting like bronchitis or tuberculosis with blood tinged sputum and cystic or cavitary lesions on lung imaging. In patients presenting with hemoptysis and peripheral eosinophilia, it is important to keep a broad differential.

A Case of a Group G Beta-Hemolytic Streptococcus Necrotizing Soft Tissue Infection and Early Toxic Shock-Like Syndrome

Khalil Choucair, M.D., Pie Pitchetsurnthorn, M.D., Thomas A. Moore, M.D.
KU School of Medicine-Wichita

Introduction

Necrotizing soft tissue infections (NSTIs) are relatively uncommon. They spread rapidly, requiring prompt intervention, and can be complicated by toxic shock syndrome (TSS). TSS-complicated NSTIs usually are caused by beta-hemolytic streptococci; however, group G beta-hemolytic streptococci (GGHS) are an uncommon cause, with only 13 reported cases.

Case Presentation

A 32-year-old male presented to the emergency department with penile pain, swelling, and inability to urinate following a 36-hours entrapment of the penile shaft by a plastic ring. He denied other systemic symptoms. He was afebrile and normotensive. The penis was swollen, erythematous, and tender, with distal shaft skin discoloration. He received amoxicillin-clavulanate 875 mg PO bid with urgent surgical ring removal and exploratory flexible cystoscopy. Following release of obstruction, skin coloration improved and a supra-pubic catheter was inserted. On postoperative day-1, the patient complained of chills and increasing pain involving the entire pelvic area. He was febrile. Manipulation of the shaft elicited excruciating pain and guarding, with disproportionate swelling of its distal 2/3 and the glans, both cold to touch, necrotic, and devoid of sensation. New tense blisters were noted distally. The scrotum was enlarged and tender to touch. A new, erythematous, and tender skin rash overlying the pubic symphysis and both inguinal canals was observed, with well-demarcated, flat borders. Bilateral inguinal lymphadenopathy was present but not prominent; identification of crepitus was limited by tenderness. Labs were remarkable for mild lactic acidosis, hyperazotemia, hyponatremia, and thrombocytopenia. Interval examination two hours later revealed symptoms worsening and rapid rash progression, raising high suspicion for possible NSTI superimposed by early stage streptococcal-induced toxic shock-like syndrome (TSLS). Two sets of peripheral blood, fluid from bullae, and penile skin swabs were cultured. IV immunoglobulin was administered and antibiotic therapy was modified to IV Piperacillin-Tazobactam and Clindamycin. The patient was moved to the ICU. On postoperative day-2, he became afebrile and rash progression halted. He underwent penile and scrotum fasciotomy and debridement. While blood cultures remained negative, fluid from the bullae and penile skin grew Enterbacter cloacae, methicillin-resistant Staphylococcus aureus (MRSA), and GGHS. Surgical tissue cultures were positive for GGHS and Escherichia coli. On postoperative day-3, the rash receded; the patient improved. Antibiotic therapy was discontinued on day-6, and he received a skin graft on day-7.

Conclusions

This is a rare occurrence of NSTI and early onset TSLS caused by GGHS. It highlights the value of thorough examination, recognition of early TSLS signs, and the ensuing prompt management. Early intervention was critical to prevent complete shock and penile amputation. The case highlighted the crucial role of early IVIG therapy and antibiotics with anti-toxin properties, as well as the increasing trend of GGHS infections, and entices a reflection on the emergence of GGHS as a human pathogen.

Rothia Mucilaginosa Pneumonia in a Patient with Rheumatoid Arthritis

Deshanett Clay, M.D., MPH, Mona Brake, M.D., Zubair Hassan, M.D.
KU School of Medicine-Wichita

Introduction

Interstitial lung disease (ILD) and pleural disease are the most common pulmonary complications of rheumatoid arthritis (RA). With a highly variable prevalence, RA-associated pleuropulmonary disease is a major contributor to morbidity and mortality. RA-ILD and infections appear to have the greatest impact on patient outcome. RA-associated pleuropulmonary disease is often asymptomatic, especially early in the disease course, and can be unmasked by infection.

Case Presentation

A 64-year-old man with RA-ILD presented to the emergency department for progressively worsening shortness of breath, dyspnea on exertion, fever and chills, and cough productive of thick, white blood-tinged sputum. He had chronic hypoxic respiratory failure on continuous oxygen, advanced COPD associated with previous tobacco use, history of asbestos exposure, and severe obstructive sleep apnea on CPAP. Medications included Adalimumab, Prednisone, and Hydroxychloroquine.

Approximately two weeks prior, he was hospitalized for six days with acute on chronic hypoxic respiratory failure and treated with antibiotics and steroids. Additional history revealed high titers of anti-cyclic citrullinated peptide antibodies, previous treatment with Methotrexate, and pulmonary function testing demonstrated a restrictive pattern. On re-admission, a clinical diagnosis of sepsis and acute on chronic hypoxic respiratory failure was made. Physical exam revealed mild to moderate respiratory distress and scattered rhonchi. He was started on empiric antibiotics plus high-dose steroids and placed on CPAP. CTA chest showed diffuse ground-glass opacities and fibrosis significantly worse compared to prior study, and stable mediastinal adenopathy. Prior CTA chest also revealed evidence of honeycombing. Viral respiratory nasal swab was positive for rhinovirus. Bronchoalveolar lavage cytology/culture obtained from the right middle lobe on hospital day #1 grew Rothia mucilaginosa and candida albicans. Sputum culture obtained on hospital day #5 grew R. mucilaginosa. All other cultures were negative. Despite antibiotic treatment and steroids, his condition continued to decline. Diffuse interstitial and alveolar opacities persisted on follow-up imaging. Overall prognosis was guarded, and he was not a good candidate for lung transplant. Following a 15-day hospital course, he was discharged home with hospice. He passed away five days after discharge.

Conclusions

This case illustrated R. mucilaginosa pneumonia complicating RA-ILD. R. mucilaginosa, formerly Stomatococcus mucilaginosa, is a gram-positive aerobe, coagulase-negative coccus believed to be of low virulence. Although the organism is part of the normal flora of the oral cavity and upper respiratory tract, it can serve as an opportunistic pathogen in immunocompromised patients and infection can progress despite appropriate therapy. Furthermore, R. mucilaginosa can be easily misidentified as bacteria from the Staphylococcus and Micrococcus genera, potentially resulting in R. mucilaginosa pneumonia being under-reported.

Ecthyma Gangrenosum Revealing Pseudomonas Bacteremia in the Immuno-Compromised Patient

Connor Grantham, M.D.
KU School of Medicine-Kansas City

Introduction

Ecthyma Gangrenosum (EG) is a rare skin manifestation, classically associated with Pseudomonas aeruginosa that can potentially be fatal. P. Aeruginosa is a common cause of gram-negative bacteremia and sepsis in immune-compromised patients that can have devastating results. It is an important organism to consider, as one study of liver transplant patients found that 35% of blood stream infections revealed P. Aeruginosa as the culprit; this was associated with a 30% mortality rate.

Case Presentation

A 60-year-old male with a past medical history of ESRD on hemodialysis and multiple myeloma on chemotherapy was admitted to the ICU due to septic shock. The patient had recently received a pedicure with nail trimming, the subsequent day he noticed a red and painful rash on his right leg that began to spread. He developed a fever and presented to the emergency department. The patient was hypotensive with lab values showing a lactic acidosis and white blood cell count of 9.6 with 30% bands. He was intubated for airway protection, hypoxemic respiratory failure, and progressive septic shock requiring multiple vasopressors. On exam, the right lower extremity demonstrated erythema from his foot to just underneath the knee cap that wrapped around the back of the leg and up through the popliteal fossa. A computed tomography (CT) scan showed moderate subcutaneous soft tissue edema, no fluid collection or osseous involvement. Blood cultures grew P. aeruginosa. He required multiple vasopressors for hemodynamic support. Surgery was consulted and the patient was taken to the OR for surgical exploration and possible debridement. Though, there was no evidence of necrotizing fasciitis. He was treated with antibiotics and his hospital course improved slightly. Unfortunately, the patient continued to deteriorate and developed refractory septic shock and multi-organ failure. He eventually was transitioned to comfort measures and passed away.

Conclusions

This case illustrates the potential for severe infection in the immune-compromised patient and the value of a complete and thorough history and physical evaluation. Ecthyma gangrenosum secondary to P. aeruginosa infection is classically associated with a characteristic skin lesion that is seen in about 1.3 to 3% of P. aeruginosa bacteremia cases. These lesions can occur from breakdown mechanical defense barriers, such as the skin and cause local infection. It results from perivascular bacterial invasion of the media and adventitia of arteries and veins with secondary ischemic necrosis. Although, the patient was treated with volume resuscitation, hemodynamic support, and supportive care this patient expired due to multi-organ failure. Recognition of this is critical to rapid institution of appropriate therapy as there can be increased mortality related to this infection. This syndrome should be considered on the differential diagnosis with immune-compromised patient presenting with P. aeruginosa bacteremia and soft tissue skin changes.

Cerebral Nocardiosis in an Immunocompetent Old Adult

Sinan Khayyat, M.D., Ammar Al-Obaidi, M.D., Maha Mohamad, M.D.,
Mohinder R. Vindhyal, M.D., M.Ed.
KU School of Medicine-Wichita

Introduction

Brain abscess is an intraparenchymal collection of pus. Cerebral nocardiosis is a rare, challenging, opportunistic infectious disease of the central nervous system occurring in both immunocompetent and immunocompromised hosts. It often results in intraparenchymal abscess formation, which represents only 2% of all cerebral abscesses. We present a case of primary multiple brain abscesses due to Nocardia subspecies in an immune-competent 79-year-old male patient.

Case Presentation

A 79-year-old male with past medical history of alcoholism, colon cancer in remission status post colectomy, and chemotherapy ten years ago presented with fatigue and confusion, which has been progressively getting worse for the prior week. Neurological exam revealed no focal neurological deficits and no signs of neck stiffness or meningismus. The patient's mental status also revealed he was not oriented to either time or place. Initial labs were unremarkable and in the normal range. The patient underwent CT brain without contrast, which showed multiple 2-3 cm low-density areas in cerebral hemispheres, brainstem, and the cerebellum suggestive of either multi-focal metastatic brain lesions versus infectious etiology.

Further workup with brain MRI revealed multiple irregular, thick-rimmed ring-enhancing lesions within the supra and infratentorial brain tissue, including the brainstem and cerebellum. CT chest showed an indeterminate thick-walled cavitary nodule in the left upper lung apex with sub-centimeter superior mediastinal and left hilar lymph nodes, which was negative for infection or malignancy. The patient continued to be confused and the decision was made to undergo brain biopsy. The brain biopsy revealed frank abscesses with areas of intense neutrophilic inflammation with tissue necrosis. Further workup revealed filamentary bacteria, morphological suggestive of Nocardia species. The patient started on oral Bactrim DS, Meropenem, and Linezolid, which resulted in an interval decrease in size of lesions on brain MRI and CT chest. The patient clinically improved, being alert and oriented, and discharged to a rehabilitation facility.

Conclusions

Brain abscess in an immunocompetent patient is polymicrobial due to aerobic and anaerobic bacteria. Nocardia abscess is often misdiagnosed or less suspected, often resulting in delayed treatment. Cerebral nocardiosis should be considered in the differential diagnosis of brain abscess, especially with multiple ring-enhancing lesions. Surgical biopsy and visualization of the organism with special stains are necessary for the diagnosis. Specific antimicrobial therapy is essential for a better outcome and decreases complication rate especially with cerebral or severe nocardiosis as it holds a high mortality rate if left untreated.

Disseminated Histoplasmosis in the Setting of Immunosuppression with Infliximab

Benton McGivern, M.D., Michael Rouse, D.O.
KU School of Medicine-Kansas City

Introduction

Disseminated histoplasmosis is a rare fungal infection most commonly seen in immunocompromised individuals living in the Ohio and Mississippi River Valleys. Typical presenting symptoms include fever, fatigue, and weight loss, and a high level of clinical suspicion is required for detection.

Case Presentation

A 68-year-old male with history of sarcoidosis, nocardia, and pulmonary embolism presented for over one month of fatigue, weight loss, lethargy, and fevers. Medications included Valacyclovir, TMP-SMX, Infliximab, Mycophenolate Mofetil, and recent addition of Ciprofloxacin and Metronidazole for gastroenteritis. He was readmitted for fever of 101.5ºF.

On admission, he was febrile at 101ºF, hypotensive at 85/60, and admission labs showed WBC 3.9, Lactate 2.6, AST 154, and ALT 138. Chest x-ray showed focal consolidative infiltrate in the posterior aspect of both lower lobes. CTA chest was negative for embolus and showed stable mediastinal and bilateral hilar adenopathy, but increased diffuse bilateral interstitial infiltrate. Vancomycin, Piperacillin-Tazobactam, and intravenous crystalloids were started. Antigen testing for legionella, streptococcus pneumonia, hepatitis A/B/C, and CMV was negative. EBV capsid antigen was elevated at 524. Viral respiratory PCR was positive for rhinovirus. BAL was non-diagnostic. Fever continued and empiric Fluconazole was started. Galactomannan assay and antigens for cryptococcus, coccidioides, and blastomycosis were negative. CT abdomen/pelvis showed normal liver, mild splenomegaly, and no retroperitoneal adenopathy. Fever continued, and fluconazole was changed to Voriconazole. Antibiotics were tapered from Vancomycin and Piperacillin-Tazobactam to Meropenem. Doxycycline was added for potential vector born disease with workup negative for tularemia, ehrlichia, leptosporidia. Fever increased to 102.7ºF with development of shock requiring pressor support. Repeat bronchoscopy was performed with negative bacterial culture, negative AFB, mycobacterium, and PJP, however, fungal culture showed histoplasma. Histoplasmosis urine antigen was positive with serum antigen elevated at 13.78. Bone marrow biopsy revealed lymphohistiocytic infiltrate and reactive pneumocytes. MNS stain showed budding branching yeast with morphology suggestive of histoplasmosis. CSF studies were negative. Amphotericin B was started and continued for two weeks, follows by Itraconazole continued indefinitely.

Conclusions

Disseminated histoplasmosis is most commonly seen in patients with AIDS, solid organ transplantation, or those being treated with TNF-alpha inhibitors. In those affected with histoplasmosis, only 1 in 2,000 will progress to disseminated infection. The incidence of disseminated histoplasmosis in persons treated with Infliximab is roughly 3 times higher than the normal population. TNF-a is known to participate in the induction and maintenance of protective granulomas, a mechanism used to prevent dissemination of certain infections. Detection is typically with MNS and PAS stain, and treatment is most effective with liposomal Amphotericin B until patients are afebrile and off ventilator or vasopressor support. Itraconazole can then be used for prolonged treatment, which should be continued no less than a year.

Citrobacter Koseri CIED Infection in an Immunocompetent Man

Nikki Miller, M.D., Jessica Newman, D.O.
KU School of Medicine-Kansas City

Introduction

Infective endocarditis and cardiac implantable electronic device (CIED) infections caused by non-HACEK gram-negative aerobic bacilli are rare. Citrobacter koseri is a motile gram-negative bacillus that can be found colonizing the gastrointestinal tract. Though initially considered to be an opportunistic pathogen, it has been infrequently implicated in liver abscess, osteomyelitis, and urinary tract infections. More recently, it has been described as a rare pathogen in infective endocarditis. This is the first known case of Citrobacter koseri CIED infection.

Case Presentation

A 64-year-old man with a past medical history of paroxysmal atrial fibrillation, coronary artery disease, chronic systolic heart failure, hypertension, and chronic obstructive pulmonary disease was initially admitted to an outside institution with a Citrobacter freundii urinary tract infection. One week following discharge, he was hospitalized for leukocytosis and fever. Blood cultures were negative and urine cultures grew coagulase-negative staphylococcus. He completed a 7-day course of Doxycycline prior to discharge. Six days following discharge, he presented with sepsis and lactic acidosis. Two sets of blood cultures grew Citrobacter koseri, which was susceptible to all antibiotics tested. The patient was treated with IV antibiotic therapy initially that was subsequently narrowed to Levofloxacin. One week following discharge, the patient developed recurrent fever and hypoxia and was readmitted. He was diagnosed with a pulmonary embolism and was placed on therapeutic anticoagulation. Repeat blood cultures upon readmission were negative, however, a transesophageal echocardiogram revealed a left ventricular ICD lead vegetation. The patient was transferred to our academic medical center for electrophysiology consultation and device extraction. He was treated with Levofloxacin and Ceftriaxone and the lead was removed without complication. Follow-up blood cultures remained clear. He completed a four-week course of treatment (post-lead removal) with no recurrence of bacteremia.

Conclusions

Only seven cases of infective endocarditis caused by Citrobacter koseri have been reported. This is the first case to our knowledge, in which the patient had a CIED infection. Our patient did not have previously described risk factors such as hemodialysis or intravenous drug use, however, the patient presented following a urinary tract infection. Urinary tract infections with Citrobacter species are a well-described source of bacteremia and this is the most likely contributing factor to our patient's infection. Given the rare occurrence of non-HACEK gram-negative endocarditis infections, evidenced-based treatment recommendations are not well-defined. Current ACC/IDSA guidelines suggest combination therapy with a β-lactam and either an aminoglycoside or a fluoroquinolone are reasonable in these cases.

Sporotrichosis Bone Presentation in an HIV-Infected Patient

Richard Muraga, M.D., Jane Gitau, M.D., Susan Ngunjiri, M.D.
KU School of Medicine-Wichita

Introduction

Sporotrichosis is a fungal infection that is associated with zoonotic transmission, affecting a large at-risk population, which includes HIV-infected individuals. The common human presentation is cutaneous or lymphatic but in rare cases may lead to bone infection as a result of local inoculation or hematogenous spread. We present a unique and diagnostic challenging case of osteoarticular sporotrichosis with positive outcomes post management.

Case Presentation

A 29-year-old man with history of AIDS and non-adherent to antiretrovirals presented with two-month history of right knee pain and locking: one-month history of right wrist pain and swelling. He had been referred to regional academic health center for concerns of it being bone cancer. X-rays of both right wrist and knee showed expansile cystic lesions. Bone biopsy of right distal ulnar confirmed sporotrichosis. The patient was started on Amphotericin B inpatient and later Itraconazole with follow-up with his PCP.

Conclusions

Osteomyelitic sporotrichosis has rarely been presented in humans (about 21 reports) and is more commonly seen in a zoonotic population. In our patient, we did not determine any history of contact with soil, plants, or animals. However, chronic infections may lead to bone affection as a result of local inoculation or hematogenous spread.

Due to its aggressive presentation and common differential diagnosis of malignancy, physicians caring for at-risk patients should add questions to elicit history regarding pet ownership and integrate it in their differential diagnosis of infectious arthritis and/or osteomyelitis for patients that present with similar symptomatology.

Neurosyphilis with Suspected Tabes Dorsalis, Otic, and Optic Involvement in an AIDS Patient

Sonya Parashar, M.D., Ashley Clark, M.D., Branden Comfort, M.D.
KU School of Medicine-Kansas City

Introduction

Between 2016 and 2017, the incidence of syphilis, a sexually transmitted infection caused by Treponema pallidum, increased 10.5% in the United States with more than half of those cases reported in men who have sex with other men (MSM). Amongst MSM who are infected with syphilis, approximately 50% were also co-infected with HIV. Patients with HIV have a higher frequency of progression to neurosyphilis and concomitant uveitis or meningitis.

Case Presentation

A 53-year-old MSM with a past medical history of AIDS and chronic hepatitis B presented to his primary care clinic to establish care with 3-month history of tinnitus, intermittent confusion, and gait ataxia. His last sexual encounter was 4 months prior. His initial physical exam was unremarkable. Given his symptoms, several tests were ordered, including HIV viral load, CD4 count, RPR, chlamydia, gonorrhea, HBeAg/HBeAb, and HBsAg. The following day labs revealed HIV viral load of 55,186, CD4 count of 185, RPR titer of 1:1024, and positive HBeAg and HBsAg. After discussing the patient with Infectious Diseases, the patient was admitted to the hospital for further workup. MRI was obtained and did not show any acute process. Lumbar puncture showed positive VDRL in the CSF and titers of 1:1. Ophthalmology was consulted due to concern for ocular involvement and dilated eye exam revealed possible vitreitis secondary to neurosyphilis. Neurology was consulted and felt that he had tabes dorsalis due to his difficulty with coordination and abnormal sensation on exam. They also recommended a CTA head, ESR, CRP to investigate any possible vasculitis complications of syphilis, which were all negative. Gastroenterology was consulted for his management of hepatitis B and recommended continuing ART as it already contained Tenofovir in addition to hepatitis D testing, right upper quadrant ultrasound, and hepatocellular carcinoma screening every 6 months. He was treated with a 14-day course of Penicillin G 18 million units daily due to pneumocephalus and ocular involvement with close follow-up as an outpatient. As an outpatient, ART resistance testing revealed two mutations, so his therapy was changed to another regimen with subsequent improvement of CD4 count, HIV viral load, and clinical symptoms.

Conclusions

Though studies suggest that the clinical presentation of syphilis is similar for those with or without HIV, other studies have shown that advanced immunosuppression can lead to atypical presentation as well as rapid progression of syphilis. This patient's advanced immunosuppression status from AIDS was a major risk factor in his accelerated progression to neurosyphilis. Current guidelines do not recommend secondary prophylaxis of syphilis.

Disseminated Mycobacterium Haemophilum Infection Involving Central Nervous System in a Renal Transplant Patient

Farhad A. Sami, MBBS, Wissam El Atrouni, M.D., Kathy Newell, M.D.,
Stephen Waller, M.D., Marie Pacholec, D.O.
KU School of Medicine-Kansas City

Introduction

Mycobacterium haemophilum (M. haemophilum) is a slow growing nontuberculous mycobacterium that prefers cooler temperature and requires iron for growth. It usually causes skin and soft tissue infections in immunocompromised hosts and cervical lymphadenitis in healthy children. We present a case of fatal disseminated M. haemophilum infection in an immunocompromised host with central nervous system (CNS) involvement.

Case Presentation

A 65-year-old Hispanic male presented with history of end-stage renal disease status post renal transplantation six years prior (on maintenance Mycophenolate, Tacrolimus, and Prednisone), diabetes mellitus type 2, coronary artery disease, ventricular arrhythmias with implantable cardioverter defibrillator, prior stroke, and cochlear implant. In the four months preceding admission, he had frequent hospitalizations for altered mental status (AMS), sepsis syndromes, and failure to thrive. Two months prior to presentation, he developed progressive swelling and redness of the wrists, right third, and left fifth digits. Computed tomography (CT) showed extensive cellulitis in distal right forearm and hand with chronic osteomyelitis. Serial incision and drainage (I&D) of right wrist yielded Acid Fast Bacilli (AFB) stain and growth on mycobacterial culture. PCR was negative for Mycobacterium tuberculosis. Patient was started on Rifampin, Clarithromycin, and Ethambutol.

Two days later, patient developed AMS and severe septic shock requiring transfer to our facility. CT head revealed indeterminate lesion in the left frontal lobe along with nonspecific hypodensities in the pons and thalamus. Repeat CT upper extremities showed osteomyelitis of distal right radius and small hand bones with adjacent abscesses. I&D revealed bilateral tenosynovitis. Cultures were resent. With suspicion for rapidly growing mycobacterial infection, the regimen was changed to Linezolid, Imipenem, and Azithromycin. Several changes in antimicrobials were necessary throughout hospitalization due to complicated hospital course. Despite aggressive measures, patient developed multiorgan failure culminating in death 10 days after starting anti-mycobacterial drugs. On the day of death, the organism was identified as M. haemophilum. Susceptibilities were not done. On autopsy, the brain had multiple abscesses containing AFB. M. haemophilum grew from the wrists and right finger cultures.

Conclusions

CNS infection with M. haemophilum is rare and has been exclusively reported in HIV or AIDS patients. To our knowledge, this is the first reported case of M. haemophilum involving the CNS in a patient without HIV/AIDS. Because of its fastidious growth requirements, M. haemophilum usually shows on acid fast stains but does not grow on regular AFB cultures. Although it prefers lower temperature for growth and is usually limited to skin and soft tissues, disseminated disease occurs in immunocompromised patients and has high mortality. It is usually treated with a multi-drug regimen including Clarithromycin, Rifampin, Ciprofloxacin, and Amikacin.

Culture Positive Mycobacterium Tuberculosis with Negative T-Spot TB and PCR

Stephanie Slimmer, M.D., Albert Eid, M.D.
KU School of Medicine-Kansas City

Introduction

Mycobacterium tuberculosis is the leading cause of infectious disease mortality worldwide. There were an estimated 1.6 million deaths caused by tuberculosis (TB) in 2017. Though the incidence of TB in the U.S. is only 2.8 per 100,000 persons, diagnosis and treatment is important in order to decrease the worldwide health burden. This case presents a rare diagnostic presentation of TB.

Case Presentation

An 89-year-old female originally from South Korea with a history of latent TB presented to the outpatient oncology clinic after a chest CT at an outside hospital showed innumerable pulmonary nodules concerning for presumed metastatic cancer of unknown primary. She later underwent a PET scan which showed concerns for metastatic pancreatic cancer and a bronchoalveolar lavage (BAL) and biopsy of the dominant lung nodule. The BAL gram stain was negative, and the biopsy showed organizing pneumonia. The patient was later admitted to the ICU with acute hypoxemic respiratory failure, generalized fatigue and worsening dry cough, and she was started on antibiotics for suspected bacterial pneumonia. A T-Spot TB assay at the time of admission was negative, and she underwent a repeat bronchoscopy with biopsy due to worsening clinical status. Mycobacterium tuberculosis polymerase chain reaction (PCR) from the BAL was negative, however, the AFB stain came back positive several days later. Given the negative T-spot TB and M. tuberculosis complex PCR from the BAL, but positive BAL AFB stain, the patient was initiated on nontuberculous mycobacteria therapy. A couple weeks later, the mycobacterial culture from the initial BAL grew M. tuberculosis. Cultures from the repeat bronchoscopy eventually also grew M. tuberculosis. Unfortunately, despite treatment with quadruple TB therapy, the patient had multiple complications and died during the hospitalization. In this case, the diagnosis and treatment of tuberculosis were delayed due to negative PCR and T-spot TB, both of which have rather high sensitivity.

Conclusions

More sensitive and rapid tests have been developed to diagnose TB, providing clear advantage over traditional mycobacterial cultures. These include interferon gamma release assays such as the T-spot TB test and M. tuberculosis nucleic acid amplification tests. Studies estimate the sensitivity of T-Spot TB to be 91%. PCR has been shown to have a 97.4% sensitivity. This case presents an uncommon challenge for TB diagnosis because both T-Spot TB and M tuberculosis PCR were negative. The patient ultimately had multiple cultures positive for M. tuberculosis, which confirmed the diagnosis. The delay of diagnosis and treatment in similar cases has many implications including increased patient morbidity and mortality and exposure of family members and many healthcare workers to TB.