Abstract Title: Use Clean Needles, Boil you Cotton: Advice for the Modern Drug User

Abstract Information:

Introduction:
Fever in an intravenous drug abuser results in a wide differential diagnosis for the physician to consider, ranging from simple soft tissue infections to endocarditis or epidural abscess. This large breadth of possibilities often leads to dilemmas on which studies to order first, usually resulting in an expensive evaluation. We present one more option to add to the differential diagnosis in an IV drug user who presents with fever, with the hopes of increasing awareness of this condition to the medical community.

Case Description:
A 31 year old female with a history significant for IV drug abuse presented with dyspnea, chest pain, severe abdominal pain, right arm swelling, and generalized weakness for several days. She worked for a home health company and admitted to recently injecting hydromorphone and other opiates into her veins. Physical exam on admission was notable for a high fever, tachycardia, and right forearm edema, erythema, and induration, with a benign chest and abdominal exam. Ancillary studies revealed a urine toxicology positive for opiates, methadone, and cocaine, as well as a white blood cell count of 13. Blood cultures were positive for gram negative rods within 24 hours, and after about 5 days they were speciated to Pantoea agglomerans, diagnostic of “cotton fever.” TTE was negative for valvular vegetations. She was ultimately treated with a 14 day course of levofloxacin.

Discussion:
“Cotton fever” is a common affliction known to injection drug users but less frequently recognized among the medical community. Cotton fever is an often benign, self-limited febrile syndrome that presents with fevers, myalgias, nausea, vomiting, and abdominal pain in injection drug users after filtering their drugs through cotton balls. Cotton and cotton plants are heavily colonized with Pantoea (formerly Enterobacter) agglomerans, a gram negative bacillus, but rarely does Pantoea bacteremia occur.

Cotton fever is a diagnosis of exclusion and typically occurs about 30 minutes after injection drug use. The syndrome is classically associated with heroin use, though has been described after injection of hydromorphone or methylphenidate.

The pathophysiology of cotton fever is unclear, though two main theories attempt to explain the syndrome. First, there is the possibility that cotton fibers have pyrogenic activity causing a
release of cytokines, resulting in sepsis. The second theory proposes that an endotoxin is released from a gram negative bacillus such as \textit{P. agglomerans}. Cotton fever typically self-resolves in 24-48 hours, though symptoms may last longer when associated with bacteremia.

There has only been one other case of \textit{Pantoea} bacteremia reported, though there have been case reports of the bacterium’s association with septic arthritis and spondylodiscitis, as well as osteomyelitis and various eye infections. Our case is the second example in the literature that the actual bacterium can be translocated from cotton, rather than just the toxin. Treatment of cotton fever alone is supportive care, though treatment of \textit{Pantoea} bacteremia typically requires 14 days of a fluoroquinolone or trimethoprim-sulfamethoxazole. Cotton fever can be prevented by boiling cotton before use.
Abstract Title: A Rare Cause of Unresponsive Patient After Hip Arthroplasty: Cerebral Fat Emboli Syndrome

Abstract Information:

Case

A previously healthy 58-year-old male presented for revision of left hip arthroplasty. Surgery was prolonged with significant bone manipulation. Patient received spinal anesthesia only, without paralytics agents. Intra-operatively, he developed atrial fibrillation. Rate was controlled with esmolol and he converted back to sinus rhythm. Blood pressure remained stable throughout procedure. Intra-operatively, he received, fentanyl, propofol, lidocaine, tranexamic acid, ketamine and 2.5 liters of LR solution. Post-operatively the patient was unresponsive but protecting his airway. CT scan was without acute abnormality. MRI showed scattered punctate foci in watershed type distribution consistent with fat emboli. Foci were also seen in the thalamus and basal ganglia. Chest X-ray showed evidence of significant bilateral interstitial prominence consistent with fat emboli associated ARDS. Transthoracic echocardiogram was negative for any significant valvular disease; bubble study was negative for patent foramen ovale. Given continued unresponsiveness, there was concern for seizure activity. Electroencephalogram showed seizure activity and intermittent status. He was started on Keppra and Ativan, without improvement in mental status. EEG showed resolution of seizure activity. The patient showed minimal improvement, but continued to be non-verbal. He was able to open his eyes and make some vocalizations. After discussion, family decided to transition to comfort care given patient previously expressed wishes about quality of life.

Discussion

Fat embolism Syndrome (FES) is a rare clinical entity, classically developing 12 – 72 hours after pelvic and long bone fracture. FES has been reported rarely after pancreatitis, bone marrow transplant, liposuction and more commonly after arthroplastic surgery with significant bone manipulation. Incidence of FES after long bone fracture is reported at <1%, though other studies have cited up to 30%. FES is primarily a clinical diagnosis characterized by the triad of petechial rash, respiratory distress and neurological dysfunction. There is no diagnostic gold standard, but Guard’s criteria are frequently cited to assist in diagnosis. Two theories for how FES develops have gained acceptance: The Mechanical Theory and The Biochemical Theory. The former suggests that physical obstruction of microcirculation in critical vascular beds by fat particles released into damaged venous sinusoids of the long bones or pelvis. The latter suggests hormonal changes
secondary to trauma or sepsis trigger release of free fatty acids (FFA) that cause cascading damage to vulnerable populations of cells in critical vascular beds.

Lungs are the most common organ system affected in fat emboli syndrome, with nervous system involvement less frequently observed. Cases where neurologic symptoms are the predominant finding are referred to cerebral fat embolism syndrome (CFES). Neurological symptoms have a broad range including headache, lethargy, delirium, stupor, convulsions, and coma. MRI is the most helpful imaging modality in diagnosing cerebral FES as CT is usually without acute abnormality. Diffusion weighted MRI will present with a “starfield” pattern where bright spots representing punctate foci ischemia are seen in a watershed distribution. These imaging changes are thought to represent cytotoxic edema which develops immediately after the injury. T2 weighted image finding develop later and likely represent vasogenic edema.

Mortality for FES is downtrending, likely due to advances in resuscitative care strategies, but is still upwards of 10%. Prior studies have not shown a benefit of steroids or heparin after development, though steroids may provide benefit in prevention. Management continues to be supportive care and early surgical fixation of fractures. Delayed recovery is also a common characteristic associated with cerebral FES, with >50% of cases returning to baseline functional status (need citation). Prior case reports document complete neurologic recovery at 6 or more months.

References


2014;48(1):100-103.
Abstract Title: Peritonitis: Acute Coronary Syndrome, Pneumonia or Gastroesophageal Reflux Disease? An Interesting Presentation of High Altitude Pulmonary Edema in a Young, Healthy Male.

Abstract Information:

Introduction
As internists, it is routine for us to admit patients who present with chest pain, productive cough, or systemic inflammatory response syndrome (SIRS). Typically, we end up making the diagnosis of myocardial infarction, pneumonia, congestive heart failure or sepsis, and managing it accordingly. Less commonplace, is a patient found to have high altitude pulmonary edema (HAPE). At > 8,000 ft, hypobaric hypoxia induces low oxygen pressure associated symptoms, including hyperventilation, tachycardia and fluid loss due to decreased thirst. At greater than 9800 ft., however, HAPE is a much more concerning, potentially fatal condition that has no respect for age or health status.

Case Report
A 37 year old, otherwise healthy male presented to outlying facility at approximately 7,884 ft., complaining of new onset cough, shortness of breath and fatigue. Usual heartburn pain, which was no longer responding to Tums. Forty-eight hours before, travel started in his Kansas hometown at just greater than 2,000 ft, to his present location. Initially evaluated in local clinic, he was found to be tachycardia and had O2 saturation in the 70’s, thus sent to the emergency department. Upon arrival, he had troponin of 0.498, leukocytosis, and a BNP of 5,798. Electrocardiogram showed inverted T waves in anterior precordial leads and biphasic T-waves in inferior and lateral leads. On chest xray (CXR), there were increased interstitial markings in bilateral mid and lower lungs with a patchy opacity at left lung base. He was given lovenox, aspirin, lasix, flown to our facility and admitted for a non-ST elevation myocardial infarction.

Upon arrival troponin was still elevated at 0.226. Leukocytosis had trended up and ECG findings were still present. Cardiology was consulted and was started on 325mg ASA, statin and lovenox. Wells score was 1.5. A left heart catheterization, showed elevated left ventricular and diastolic pressures, suggestive of sleep apnea. Triglycerides were 244 and patient had a low HDL. Repeat CXR had only minor, questionable pulmonary edema. Patient improved and was able to be discharged home with no new medications.

Discussion
With increasing lifespan, transportation and improved infrastructure, people of all ages are trekking up into the vast and mountainous lands at both moderate (6500 ft to just over 13,000 ft) and very high altitudes (>18,000 ft). In Colorado, with even our average altitude been 6,800 ft, Even slight additional elevations can be high enough for complications. Symptoms include: Difficulty walking, inability to keep up, congestion, cough, poor judgement, tachycardia, cyanosis, fever up to 101.3, perfuse perspiration and tight chest, keeping in mind these will be worse at night. Both primary care physicians and hospital admitting physicians should be aware of these signs and symptoms and be able to treat accordingly.

Resources (Additional resources will likely be used)


3. M. Iwase, MD. PhD, Y. Ito, MD, K, Takada, MD, K Shiino, MD, Y. Kato, MD, PhD, Y. Ozaki, MD, PhD, FESC. Altitude-induced Pulmonary Hypertension on One-Day Rapid Ascent of Mount Fuji: Incidence and Therapeutic Effects of Sildenafil.

Case: A 66-year-old male with chief complaint of dyspnea on exertion, lower extremity swelling, and decreased urine output is found to have renal failure and bilateral deep vein thrombosis (DVT). He denies chest pain. Vitals: initial tachycardia resolves with 2L oxygen, normotensive, respiratory rate of 32. IV heparin is started. Labs: Pro-BNP 24,993; troponin 0.09 and lactate 5.5. Echocardiogram reveals large mobile thrombus in the right atrium. Pulmonology, nephrology, interventional radiology, and cardiology discuss treatment options including systemic tissue plasminogen activator (TPA), catheter directed TPA, and inferior vena cava filter. Given patient's clinical stability, anticoagulation is continued and a lasix drip started for hypervolemia with plans for thrombolysis should patient become hemodynamically unstable.

The following morning patient has a pulseless electrical activity arrest. Bedside ultrasound shows no pericardial effusion, pneumothorax, or right atrial thrombus. Patient is declared dead after 50 minutes of resuscitation efforts, including TPA.

Discussion: There are no treatment guidelines for right atrial thrombus (RAT) in hemodynamically stable patients. UpToDate recommends considering thrombolysis on a case by case basis. Consideration is particularly recommended when "patients develop signs of deterioration, including increasing tachycardia, clinical signs of shock, worsening right heart dysfunction, worsening blood pressure, significant hypoxemia," which were not present in our patient. [9]

Torbicki et al analyzed an international pulmonary embolism (PE) registry, including 1,011 patients with PE and echocardiogram evaluation. 37 patients had RAT with PE. Despite treatment, fourteen-day mortality was 23.5%, 20.8%, 25% among those with RAT with PE treated with heparin, thrombolysis and embolectomy, respectively, vs 11% with isolated PE.[6] Rose et al analyzed 177 cases of RAT and showed mortality rates: 100% no therapy (n=16), 29.6% heparin alone (n=35), 23.8% surgical embolectomy (n=63), and 11.3% thrombolysis (n=62) suggesting lower mortality with thrombolysis.[3] Unfortunately,
2016 CHEST guidelines regarding antithrombotic therapy in venous thromboembolism fail to address RATs.[8]

Several studies demonstrate success with novel interventions.[1,4,5] The AngioVac is a percutaneous device used with venovenous bypass to remove thrombi. Donaldson et al evaluated the AngioVac in 14 consecutive cases between April 2010 and July 2013 at Massachusetts General Hospital. Intracardiac thrombi were present in 11 of the 14 cases; all thrombi were completely removed. Two patients did not survive to discharge.[1] Resnick and Salsamendi both reviewed 7 cases where AngioVac was used for thrombectomy and also found promising results.[4][5]

Despite numerous therapeutic options, there are no guidelines for optimal management of RAT. In her editorial in Insights in Chest Disease, Bushra et al highlights the challenging situation posed by RAT. They recommend multidisciplinary evaluation of treatment options based on clinical experience and available resources.[7] Unfortunately, this strategy was ineffective in our case. Further literature review, randomized control trials, and ultimately evidence based guidelines would aide management of RAT.


A 41-year-old gentleman with history of spinal trauma status post several spinal surgeries and chronic arachnoiditis presented to the emergency department reporting vertigo, generalized weakness, headache, and fever developing over the past two days. On exam he was alert and oriented, with irregularly irregular tachycardia, tachypnea, and a temperature of 39.2°C. Additionally, he was noted to have pronounced truncal ataxia and multidirectional saccadic eye movements. There were no changes in sensation or strength, and no meningismus or rash. Initial labs were unremarkable. ECG indicated new onset of atrial fibrillation. MRI of the brain and spine was notable for spinal fluid collections consistent with his previous studies without new abnormality. Based on this presentation, the diagnosis of opsoclonus myoclonus syndrome was made, and levetiracetam was initiated for symptom management. The investigation for an underlying etiology was initially complicated by inability to obtain CSF; multiple attempts at lumbar puncture under imaging guidance were unsuccessful, and the patient declined cervical puncture. As a result, he was treated empirically for meningitis with a ten day course of ceftriaxone and acyclovir. A serum paraneoplastic panel and PET CT was unrevealing for malignancy. Viral studies ultimately demonstrated West Nile Virus (WNV) infection, with positive serum WNV IgG and IgM. The patient underwent successful cardioversion for his new atrial fibrillation, and was treated with 2g/kg IVIG, which was administered over the course of three days; steroids were withheld due to concern for ongoing infection. The patient experienced significant improvement in his disabling symptoms which were documented on video.

Discussion:

There have been nearly 44,000 cases of West Nile Virus (WNV) reported in the United States since 1991. Only a quarter of those infected develop symptoms, typically presenting with fever, headache, myalgias, and rash. About 1% develop neuroinvasive disease, characterized by encephalitis, meningitis, and flaccid paralysis. WNV infection is rarely associated with myocarditis; this has only been described in a few case and autopsy reports. Opsoclonus myoclonus syndrome (OMS) is a rare condition with an estimated 1 case per 10 million population. The characteristic ataxia (dancing feet) and random horizontal and vertical saccades (dancing eyes) are thought to be due to autoimmune attack on omnipause neurons in the cerebellum. OMS most commonly presents in children with neuroblastoma but can also occur as a paraneoplastic or parainfectious syndrome in adults. Treatment typically includes IVIG or plasmapheresis with high dose steroids. There have been only 4 reported cases of OMS occurring in patients with WNV. Our patient represents an extremely rare case of WNV-associated OMS as well as cardiac involvement from WNV. As the incidence of WNV rises, it will be increasingly important to consider this in the differential for patients presenting with sepsis and unusual neurologic and cardiac findings.
References:


Abstract Title: The Exceptionally Rare Coumadin Associated Side Effect of Cholesterol Embolization

Abstract Information:

An exceptionally rare, but potentially fatal side effect of Coumadin therapy is cholesterol embolization, incidence as low as 6.2 cases per million and as high as 1.4% in pt's undergoing cardiac catheterization (Varis 2010). We present the case of an elderly male who presented with acute renal failure and was subsequently found to have renal atheroemboli bilaterally without evidence of embolization elsewhere. The patient had been started on Warfarin within the 5 months prior.

The patient was initially started on Coumadin after a deep vein thrombosis (DVT) within peroneal and posterior tibial veins of the right lower extremity in September of 2015. In January of 2016, the patient presented again with abdominal pain, weakness, nausea and vomiting for several weeks. This was associated with a petechial rash and easy bruising. Labs revealed 35% eosinophils, platelets of 2 x 10^3/uL, and a creatinine of 15.4 mg/dL. Previous labs in September had been normal.

Considering the rash, acute renal failure, and severe thrombocytopenia; initial consideration was hemolytic uremic syndrome (HUS), Acute Tubular Interstitial Nephritis (ATIN), and rheumatologic or autoimmune pathologies. The patient was trialed on steroids with no improvement. Immunologic testing and urine eosinophils were negative. Anti Streptolysin-O was additionally negative. After attempting plasma exchange, which was not beneficial, the patient was started on hemodialysis day 7 of hospitalization.

On hospital day 12, due to persistent renal impairment, the patient underwent a kidney biopsy showing arteriosclerosis with occlusive cholesterol embolism of the left kidney, nephrosclerosis affecting 85% of the sampled glomeruli, and interstitial fibrosis. The patient was discharged and continued on hemodialysis.

Our case is unique for several reasons. First, the patient suffering from atheroemboli after initiation of Coumadin therapy is quite rare. The patient demonstrated a diffuse petechial rash, which was quite different from the livedo reticularis often found. Also, the pt did not have evidence of embolization to other areas. Though isolated renal involvement is rare, it should remain in the differential.

In patients beginning anticoagulation therapy, atheroemboli are a serious, but often less considered complication due to their rarity. Despite recognition as a distinct clinical entity, cholesterol embolization...
syndrome has no specific therapy. Treatment goals consist of supportive care for end organ dysfunction and secondary prophylaxis against future embolizations. As cholesterol embolization are manifestations of atherosclerotic disease, traditional modifications such as cessation of smoking, lipid control and control of hypertension apply. (Kronzon 2010)

Bibliography:


Abstract Title: A Serious Pain in the Back: Epidural Abscess and Osteomyelitis in Patient Admitted for Alcohol Withdrawal

Abstract Information:

Case description: A 50-year-old gentleman was admitted for acute on chronic back pain and was thought to be in alcohol withdrawal. History obtained from the patient’s wife revealed that he had chronic back pain for years which had worsened recently. He had been undergoing an outpatient workup, and a recent thoracic x-ray noted a compression fracture. Despite outpatient treatment, the pain worsened acutely over the last ten days. Notably, he had a long history of alcohol dependence and a well-documented history of alcohol withdrawal.

On presentation, the patient was oriented only to self. He denied any back pain. On exam he had kyphosis of the thoracic spine, without any palpable masses or erythema in the area. His neurologic exam was pertinent for slightly decreased strength in the right lower extremity that was attributed to poor effort. His initial labs were remarkable for an elevated WBC count and a CRP of 217. He was started on lorazepam by CIWA protocol for presumed alcohol withdrawal. He continued to have an altered mental status despite high doses of lorazepam, and was transferred to the ICU for more aggressive management. Approximately 12 hours later, he had a sudden increase in oxygen requirement and required intubation. CXR showed an unexplained right sided mediastinal elliptical opacity. Follow up CT Chest showed osteomyelitis with destruction of the T8 vertebral body and associated epidural abscess and spinal cord compression. The patient required emergent neurosurgical intervention. Subsequent cultures were positive for MSSA and he was started on oxacillin and rifampin. Postoperatively, the patient was paraplegic and required a prolonged ICU stay.

Discussion: This case presents a number of opportunities to examine cognitive error in clinical decision-making, especially when treating patients with known alcohol or drug use. Despite the mounting evidence that there was an alternative process at work in this patient, the diagnosis of alcohol withdrawal was consistently at the forefront of the medical management plan. Imaging of the patient’s back was suggested but not performed expeditiously as the team waited for medical stability from alcohol withdrawal. Additionally, during the initial assessment, the patient’s neurologic exam was noted to be unremarkable despite there being documented decreased strength in the right lower extremity. This asymmetric neurological finding was attributed to “poor effort”.

Patients with known alcohol and drug dependence are frequent in the hospital setting. Often, these cases are straightforward. As in this case, however, it is important to assess the whole patient and to further evaluate physical exam findings and lab values that do not add up. A confused patient is not always a withdrawing patient, even in the setting of known alcohol dependence.
Abstract Title: Meat Cleaver Fever

Abstract Information:

Case Presentation
A 61-year-old man presented with four days of generalized weakness, fatigue, and fevers. Symptoms began with increasing fatigue over two days, followed by subjective fevers, and finally generalized weakness. He also reported myalgias and bilateral retro-orbital headache without neck stiffness. He had no significant medical history and was not taking medications. He migrated from Mexico to Colorado twenty years ago and had not traveled outside of the country for the past year. He worked for the last seven years in a slaughterhouse production line performing sheep evisceration. Vital signs revealed a temperature of 38.1 degrees Celsius, heart rate of 94, blood pressure of 108/75, and no tachypnea nor hypoxemia. The neurological, cardiac, and pulmonary exams were normal. Aspartate and alanine transaminase levels were elevated at 58 and 93 respectively. Cerebrospinal fluid analysis revealed normal glucose, 54 mg/dL total protein, 12 nucleated cells, and a gram stain without visualized microorganisms. Final cerebrospinal fluid studies were negative for Herpes simplex, Varicella, and Enterovirus species. Both Brucella and Leptospira antibodies were negative. Coxiella burnetii serology resulted a week later with phase 1 and phase 2 IgG titers of 1:32 and 1:128 respectively, supporting the diagnosis of acute Q fever.

Discussion
Q fever is a zoonotic disease caused by Coxiella burnetii that can be challenging for hospitalists to diagnose. Cattle, sheep, and goats are the primary reservoirs for this highly virulent organism making slaughterhouse workers particularly vulnerable for this infection. Colorado is one of seven states that accounts for more than half of all cases of Q fever in the United States due to the high prevalence of slaughterhouses. The acute phase of Q fever is characterized by a nonspecific febrile illness that includes fever, fatigue, myalgias, sometimes accompanied by retro-orbital headache and pneumonia. The laboratory abnormality most commonly observed is increased transaminase levels, seen in up to 85% of cases. Due to delay in seroconversion required to confirm acute Q fever, empiric treatment should be started based on clinical suspicion. Doxycycline is the most effective treatment for acute Q fever, especially if started within the first three days of symptoms. The gold standard for confirming the diagnosis of acute Q fever is demonstration of at least a fourfold rise in phase 2 IgG antibody titer over three to six weeks. The differential diagnosis for a fever in a slaughterhouse worker should also include brucellosis and leptospirosis, both of which can present similarly to Q fever.

Conclusion
Empiric treatment with doxycycline should be started early in slaughterhouse workers who present with fever, retro-orbital headache, and elevated liver enzymes while awaiting serologic diagnosis confirmation. This case exemplifies the importance of taking a thorough social history in order to generate an inclusive differential diagnosis.
Abstract Title: Anorexia and Elevated Alkaline Phosphatase in a Woman with Crohn’s Disease

Abstract Information:

Introduction:

Case Description

A 68-year-old woman with Crohn’s Disease presented with 2 months of nausea, vomiting, and anorexia. One month prior to presentation, she had a self-limited episode of chalk-colored stools lasting 1 week. Three weeks prior to presentation, she noted onset of daily fevers occurring every evening, measured at 101-102°F. Two weeks prior to presentation, her chronic abdominal pain worsened and became unresponsive to conservative therapy. Oral budesonide was trialed 2 weeks prior to presentation without benefit.

Medical history was significant only for stable Crohn’s Disease on chronic azathioprine. She had received an ileocecectomy in 1974 at the time of diagnosis. She reported no recent changes in medications. She had received a 10-day course of augmentin 3.5 months prior to presentation. She reported drinking 12 glasses of wine weekly until 2 months prior to presentation. She had not used alcohol in the last 2 months. She had never smoked. She was retired, but previously had worked as a research nurse.

She appeared chronically ill and fatigued. There was no jaundice. Cardiopulmonary exam was unremarkable. Abdomen was soft but tender in the right lower quadrant without rebound or guarding. She had no rash, palmar erythema, or telangiectasias.

Labs on admission showed an alkaline phosphatase of 1050, AST of 290, ALT of 110, and total bilirubin of 1.3. ANA, anti-smooth muscle, and anti-mitochondrial antibodies were negative. Workup for acute or chronic viral hepatitis A, B, and C was negative. Contrasted CT of the abdomen and pelvis did not reveal small bowel or colonic inflammation. MRCP revealed an unremarkable intrahepatic and extrahepatic biliary tree.

Upper endoscopy showed diffuse inflammation and atrophic mucosa of the 2nd and 3rd portions of the duodenum without ulcerations. Biopsies showed severe acute-on-chronic inflammation with surface ulceration without lymphocytosis. Colonoscopy was unable to be completed due to inability to tolerate bowel prep. Liver biopsy was pursued which revealed viral cytopathic changes consistent with CMV, and CMV immunostain was positive. Review of duodenal biopsy showed a CMV-positive immunostain.
**Discussion and Conclusions**

We present a case of concurrent CMV hepatitis and duodenitis in a woman with Crohn’s Disease on chronic azathioprine. Thiopurines have been implicated as placing individuals with inflammatory bowel disease at higher risk of CMV hepatitis. This case was complicated by the possibility of azathioprine-induced hepatotoxicity and augmentin-associated cholestatic liver injury. In review of the literature, CMV duodenitis appears rare with a single reported case in a series of upper gastrointestinal CMV infections. Reported cases of concurrent bowel and liver infection are limited.
Abstract Title: Spontaneous Intercostal Lung Herniation: A Case Report

Abstract Information:

Introduction: Spinal cord infarction is a rare and devastating disorder often resulting in severe disability including paraplegia or quadriplegia. Diagnosis is often delayed or missed as it frequently mimics other disease processes including AIDP, transverse myelitis, or compressive lesions on the spinal cord.

Case: A 60 y/o female with RA, CAD, and lumbar disc disease presented with bilateral lower extremity weakness. One week prior to admission, while standing in her kitchen she experienced the acute onset of weakness and lower extremity tingling requiring her to lie down. Symptoms spontaneously resolved after 20 minutes. The morning of admission while using the toilet she noticed the same tingling and weakness and could not get up. Initially able to lift her left leg and move her feet bilaterally, the weakness progressed and she was completely unable to move her lower extremities by hospital arrival. She also could not urinate despite having the urge. Her neurologic exam demonstrated: normal cranial nerves, normal upper extremities, 0/5 bilateral lower extremity strength with areflexia, downgoing toes, diminished sensation across all modalities and no defined sensory level over her torso and normal rectal tone. MRI showed changes consistent with known degenerative disc disease with a disc extrusion at L1-L2, and bulges at L2-L3/L3-L4/L4-L5. An LP showed a normal cell count and elevated protein of 133 concerning for AIDP. IVIG was started, but unfortunately there was no response so other etiologies including spinal cord infarct were reconsidered, prompting repeat MRI with contrast and DWI. This demonstrated a cord infarction at T9 with edema but no evidence of hemorrhage. She was placed on clopidogrel in addition to ASA and received a short course of pulse steroids for spinal cord edema. There was mild improvement in lower limb motor function and she was discharged to an acute rehab facility.

Discussion: Workup of suspected spinal cord infarct includes MRI, however, sensitivity is limited in the first several hours from symptom onset. As such if the clinical suspicion is high and the initial MRI is normal, follow up imaging is recommended with DWI sequencing as was performed in this case, revealing the infarct. While the CSF is typically normal in patients who have a spinal cord infarct, it can show pleocytosis or an elevated protein as was noted in this case. Unfortunately, treatment options are limited. There are a few case reports of thrombolytic use; however delay in diagnosis presents a barrier. Use of steroids is also controversial. In patients who carry underlying vascular risk factors it is reasonable to start antiplatelet agents. Prognosis is mixed but generally worse in patients with cervical spine lesions, advanced age, female gender and severe deficits at presentation. Often intensive acute rehab is required.
Abstract Title: Cardiac Tamponade Causing PEA Arrest: Initial Presentation of Postpericardiotomy Syndrome

Abstract Information:

Case

69 year old male with history of severe aortic stenosis, with valve replacement about 6 weeks prior, presented for progressive dyspnea on exertion. He was seen day prior at another ED for similar symptoms, but left before receiving care. On presentation, he was hypotensive and in respiratory distress, requiring BiPAP. CXR showed bilateral infiltrates concerning for pulmonary edema. He then became acutely anxious, followed by bradycardia and cardiac arrest. Bedside echocardiogram showed large pericardial effusion with echocardiographic evidence of tamponade. Emergent pericardiocentesis was performed. After 2 L of serosanguinous fluid removal, he had return of spontaneous circulation. Pericardial drain was placed and he was started on colchicine. Fluid studies were consistent with inflammatory etiology. His drain was removed 4 days after presentation without reaccumulation of pericardial fluid. He was discharged on colchicine and ibuprofen.

Discussion

Postpericardiotomy syndrome (PPS) is a type of post-cardiac injury syndrome that can cause pericarditis and pericardial effusion. Incidence of PPS is reported at 15-20%, with most cases (80%) occurring within the first month after procedure. 90% of patients have a pericardial effusion at the time of diagnosis of PPS, but cardiac tamponade occurs in less than 2% of patients with PPS. There are some studies to suggest an immunologic response to cardiac antigens released during surgery as the mechanism, though children after heart transplantation still develop PPS, even while on high dose immunosuppression.

Prior studies have shown a benefit in prevention with colchicine. In both the COPS and COPS 2 trials, colchicine started prior to surgery and immediately postoperatively, showed a significant reduction in incidence of PPS. Steroids have not shown to provide benefit. After development of PPS, use of non-steroidal anti-inflammatory medications increases resolution rates and reduces recurrence. In a small single center trial, ibuprofen and colchicine together were shown to decrease intervention rates (both pericardiocentesis and pericardial window).

There are no clear guidelines on the management of PPS. Given prior studies, the use of colchicine and NSAIDs after development of PPS is reasonable. These agents may increase resolution rates and decrease recurrence.
Abstract Title: Choreiform Movements in an Elderly Diabetic Female Associated with Hyperglycemia

Abstract Information:

Introduction

Non-ketotic hyperglycemia has been described as a rare cause of choreoathetoid movements. We present the case of an 87 y/o diabetic female who was found to have hyperglycemia and hyperosmolarity in the clinical setting of left upper and lower extremity chorea.

Case Description

87 year old female, with a history of diabetes and prior stroke, presented with abnormal and uncontrollable left arm and left foot movement for 3 days. She reported running out of her anti-hyperglycemic agents 2 months prior. Physical exam was remarkable for choreiform and ballistic movements of the left upper extremity and choreiform movements of the left foot. No other focal neurologic abnormalities were elicited. Labs were significant for a blood glucose of 647 and calculated serum osmolality of 321. The patient underwent CT of the head which was significant for increased density in the right caudate and putamen. No other abnormalities were found to explain her chorea. The patient’s blood glucose was corrected with complete resolution of her choreiform movements.

Discussion

Non-ketotic hyperglycemic associated hemichorea is a rare cause of hemichorea/hemiballism. This syndrome is more common in elderly females of Asian descent. It can be the presenting symptom for diabetes or present after periods of poor glucose control, as was the case with our patient. Prior studies report glucose of 170-1260 mg/dl and serum osmolality of 290-335 mOsm/l. CT imaging may show hyperdense putamen or caudate nucleus on the contralateral side to the patient's symptoms. T1 -weighted MRI images show high density signal intensity in the basal ganglia on the contralateral side. T2- weighted images have more variability in findings. The changes seen on imaging usually resolve after resolution of symptoms. There are reports of imaging changes in the absence of hemichorea-hemiballism, but with hyperglycemia.

The mechanism for this syndrome is unknown, though several theories exist. Hyperviscosity leading to neuron and neurotransmitter dysfunction, specifically GABA, may result in uncontrolled movements. One SPECT study showed decreased perfusion of the striatum contralateral to the affected side. Some have proposed petechial hemorrhages or myelinolysis in the affected areas, resulting in movement disorder.
Management involved normalizing blood glucose. In the majority of cases, treatment of the hyperglycemia results in complete resolution. In refractory cases, postsynaptic dopamine antagonists, such as atypical antipsychotics may ameliorate the symptoms. Although not well studied, medications with GABA antagonism, such as benzodiazepines have been used. Repeated episodes may result in permanent movement disorders.
Abstract Title: Impact of Cannabis use on Concurrent Alcohol Dependence and Phosphatidylethanol Levels

Abstract Information:

Purpose: Recent legislation in Colorado has resulted in legalized cannabis for recreational use. This has likely contributed to increasing use compared to other states across all age groups. Alcohol consumption is also prevalent in Colorado; 66% of adults report alcohol use in the past month and 8% report alcohol abuse or dependence in the past year, also known as alcohol use disorders (AUDs). Current literature has suggested that combined use of alcohol and cannabis is common and among people with cannabis use disorders, AUDs are increased six-fold. The prevalence of cannabis use in Colorado among people with AUDs is not established. We sought to determine if cannabis use among people with AUDs differed from controls, and further, if cannabis use among those with AUDs was associated with more severe alcohol dependency defined by Alcohol Use Disorders Identification Test (AUDIT) scores and AUDIT-C (abbreviated AUDIT focusing on consumption). Whole blood phosphatidylethanol (PEth) levels were used to confirm recent, heavy alcohol intake.

Methods: Subjects with AUDs, defined as an AUDIT score of >=8 in men, and >=5 in women, were enrolled from an alcohol detoxification center in Denver, Colorado. Age-, sex-, and cigarette smoking-matched controls with AUDIT scores below these thresholds from the Denver metro area were also enrolled. Neither group had significant comorbid conditions; those with substance use other than alcohol or cannabis were excluded. Cannabis use was verified with a urine toxicology screen. Measurements of PEth from all subjects were performed using whole blood.

Results: Forty-six subjects with AUDs and thirty-three controls were enrolled. There were no baseline differences between the groups in terms of age (41.7 versus 40.5 years), sex (17% versus 24% female), or cannabis use (39% versus 45%). Average AUDIT scores were 27.6 among AUD subjects and 2.1 among controls; AUDIT-C were 10.3 and 2.0, respectively. PEth values were higher among AUD subjects (1177.1 versus 211.4 ng/ml). PEth was significantly correlated with both AUDIT scores (Spearman’s rho = 0.70, p<0.0001) and AUDIT-C (Spearman’s rho = 0.72, p<0.0001). Among AUD subjects, those with concomitant cannabis use had significantly higher AUDIT-C (11.1 vs 9.89, p = 0.046); a similar trend was observed with AUDIT (30.0 vs 26.6, p=0.26). Among subjects with AUDs, PEth values did not differ between cannabis users and non-users (1031.9 vs 1293.6 ng/ml, p=0.38).

Conclusion: Higher AUDIT scores suggest more severe alcohol dependency. Higher AUDIT-C and AUDIT scores observed among cannabis users may therefore indicate a possible cannabis and alcohol
codependency dilemma in Colorado. Based on PEth values, quantity of alcohol consumed did not vary based on cannabis use. Our observations indicate the potential utility in targeting substance abuse resources towards individuals with AUDs to promote the highest yield in reducing physical and mental health-related consequences.
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**Abstract Title:** Fecal Microbiota Transplantation for Clostridium Difficile Colitis with Toxic Megacolon

**Abstract Information:**

Introduction: Clostridium difficile infection (CDI) causes a toxin mediated colitis, is the most frequent cause of nosocomial infection in the United States, and has a prevalence of 15 cases per 1,000 discharges. Toxic megacolon is a less common but more severe complication of CDI with 24% mortality in 2010. Intravenous metronidazole and oral vancomycin are the first-line antibiotics for severe CDI, but disease relapses in 15 - 35% of cases. Fidaxomicin is an alternative agent. Fecal microbiota transplantation (FMT) has emerged as a highly successful option in recurrent CDI, effective in 85 - 90% of patients who are not critically ill. The role of FMT in patients with severe CDI complicated by toxic megacolon is unknown.

Case Description: A 52-year-old woman presented with diarrhea, abdominal distention, worsening mental status, and reported fever to 102°F. Medical history was significant for cirrhosis complicated by ascites requiring weekly paracentesis. Two months prior to admission, she had been hospitalized for severe CDI associated with hypotension, leukocytosis, and lactic acidosis, and recovered after treatment with oral vancomycin. In the week prior to admission CDI relapsed and was again treated with vancomycin. On presentation, her temperature was 99°F, her pulse was 100 beats per minute, blood pressure was 90/50 mmHg, and respiratory rate was 30 breaths per minute. She was somnolent with decreased concentration and difficulty following commands, but was oriented. Her abdomen was distended, tender, hyper tympanic, and had a fluid wave. White blood cell count was 66.5 x 10^9 /L with 91.2% neutrophils, hemoglobin was 10.3 g/dL, bicarbonate was 12 mmol/L, creatinine was 3.40 mg/dL, and lactate was 2.0 mmol/L. Abdominal x-ray showed colonic ileus with thumbprinting and distension to 7.4 cm. Computed tomography of the abdomen revealed colonic wall thickening, ascites, and a cirrhotic liver. Stool Clostridium difficile toxin was positive, confirming the diagnosis of CDI associated toxic megacolon. She was intubated for respiratory failure. She was deemed inoperable due to high risk of surgical mortality, and underwent FMT with 150 cc of stool administered through a post-pyloric nasogastric tube and 100 cc by enema. Six hours after FMT, she was initiated on a 5-day course of oral vancomycin 125 mg four times daily. She improved markedly, was successfully extubated on the 7th hospital day, and was discharged in stable condition.

Discussion: FMT is infrequently reported in cases of toxic megacolon, but should be considered as a critical adjunctive option due to its low risk compared with surgical colectomy. In this patient, recurrent CDI was complicated by systemic inflammatory response syndrome, toxic megacolon, and decompensation of underlying cirrhosis,
increasing her risk of surgical mortality to over 50%. While several modalities exist, combined administration of FMT via nasogastric tube and enema were successful in our patient.
Abstract Title: Shortness of Breath in an Immunosuppressed Patient

Abstract Information:

CASE PRESENTATION

A 65-year-old male with history of HTN, COPD, and recurrent stage IV lung adenocarcinoma presented with one week of worsening shortness of breath and productive cough. He denied fevers, chills, chest pain, or any other localizing symptoms. Vitals were significant for temperature of 38.1, HR of 109, RR 26, and O2 saturation of 71% on RA which improved to 93% on 6L NC. Crackles were noted throughout the R lung only and he appeared to be in respiratory distress. Notable labs included leukocytosis to 12.2 and a negative troponin. CXR revealed left upper lobe, right middle lobe and right lower lobe opacities. CTPE showed small right middle lobe segmental PE (non obstructive), ground glass opacities worse in the right lung, and consolidations in right middle lobe and right lower lobe with air bronchograms.

A presumptive diagnosis of hospital acquired pneumonia and PE was made and the patient was started on vancomycin, cefepime, and a heparin infusion. It was noted that the patient was on nivolumab monotherapy for recurrent lung adenocarcinoma which was started 6 months prior to presentation. A drug reaction related to nivolumab was considered but the asymmetry of infiltrates on CT argued against this. On hospital day 2, nasal swab viral studies and influenza PCR returned negative. The patient continued to worsen, requiring 10L heated hi-flow oxygen. A bronchoscopy was performed and the bronchoalveolar lavage studies were significant for lymphocytes at 47%, a finding suggestive of drug induced pneumonitis. Steroid therapy was initiated and nivolumab was discontinued. His respiratory status improved and he was later discharged without supplemental oxygen.

DISCUSSION

Nivolumab is a humanized IgG4 anti-PD-1 monoclonal antibody. The use of antibodies against PD-1, which blocks inhibitory T-cell checkpoints, has been a great aid for the treatment of advanced cancers. Adverse drug reactions (ADRs) due to antineoplastic agents are a common form of iatrogenic injury. 10-20% of all patients treated with an antineoplastic agent have some form of lung toxicity. Pneumonitis is a rare side effect to these medications and is thought to be immune-mediated.

Pneumonia is a common cause of pulmonary infiltrates and respiratory compromise in patients with cancer and should always be ruled out. Imaging is an important diagnostic tool, but as highlighted in this case, it is not always enough to make a diagnosis. Typically, on CT, pneumonitis presents as homogenous ground glass opacities that are symmetric and bilateral. This case highlights the importance of keeping a broad differential, re-evaluating the initial diagnosis made, and always considering an iatrogenic etiology.
Abstract Title: Pharming: A Case of Refractory Seizures Due to Chronic Diphenhydramine Overdose

Abstract Information:

Introduction:
Diphenhydramine abuse is common, especially as treatment for insomnia and for intoxicant effect, and symptoms associated with anticholinergic overdose are dose-dependent. With ingestion of greater than 300-1000 mg daily, symptoms can include visual hallucinations, seizures, coma and life threatening QRS or QT prolongation.

Case Description:
PT is a 19 year old female with intractable seizures who presented to the epilepsy-monitoring unit (EMU) for further evaluation after failing to respond to antiepileptic drug (AED) therapy. PT’s neurological issues began at 15 years old, which included tremor, difficulty concentrating and episodic involuntary limb/torso movements often triggered by studying, focusing, reading or playing piano. At age 18, PT experienced worsening insomnia and depression, and her father began noticing focal seizures that would progress into generalized bilateral convulsions with loss of consciousness. PT’s father noted that the seizures often occurred around the 20th of every month despite increasing doses of Levetiracetam. At age 19, PT’s seizures became more frequent and she was experiencing colorful visual hallucinations. Her medications were changed to Lamotrigine and Topiramate, but her seizures continued to be refractory despite increased AED doses.

On the first day in the EMU PT suffered numerous brief seizures and periods of hemodynamic instability. On hospital day 2 she had a prolonged seizure aborted with ativan. A CNA then discovered numerous diphenhydramine pills underneath the recliner chair. The neurologist reviewed the recorded video, which revealed PT taking several handfuls, 300-500 mg at one time, of diphenhydramine pills overnight and, notably, one hour before her prolonged seizure. She was intubated and sedated with propofol for seizure control. Her QRS remained stable, her symptoms eventually subsided and she was successfully extubated.

Discussion:
With advancements in biotechnology and imaging the ability to determine a patient’s specific epilepsy etiology has improved, often identifying genetic, structural or metabolic causes. However, the etiology of up to one third of all cases remains unknown. PT’s epilepsy was associated with strange features that included visual hallucinations, altered mental status, urinary retention, hyperkinetic movements and hemodynamic instability, and was refractory to several antiepileptic drugs. When it was discovered that her seizures were from chronic daily diphenhydramine overdose the picture came together and she eventually endorsed motivations for use that included insomnia, depression, intoxication and suicidal ideations.

“Pharming,” or the use of prescription or OTC drugs for intoxication purposes, among teens has been trending up in recent years. Recreational use of diphenhydramine for
hallucinogenic effect is reported in the literature at doses of 300-700 mg. Furthermore, with chronic use, addiction ensues and withdrawal symptoms can be very distressing. Through the efforts of diligent physicians, assisted by advances in technology, the cause of PT’s seizures was finally determined and she was ultimately treated for her diphenhydramine addiction.
Abstract Title: Which came First? Sepsis or the Stent?

Abstract Information:

INTRODUCTION: Sepsis is one of the most common reasons for admission to a hospital, but the etiology is not always obvious. Careful consideration of the source should include potential complications of recent endovascular manipulation.

CASE PRESENTATION: A 77-year-old man presented with chills, malaise, and abdominal pain. Two weeks prior, the patient was admitted for a partial small bowel obstruction and urinary tract infection, and was incidentally found to have a thrombosed saccular pseudoaneurysm of the descending thoracic aorta. The pseudoaneurysm was noted to expand rapidly, prompting endovascular repair prior to discharge. His other past medical history was significant for a colostomy due to anal atresia, hypertension, coronary artery disease, and diabetes mellitus type 2. He was not an IV drug user; his only recent surgery was the endovascular repair. He was afebrile, but rigoring with a respiratory rate of 26. The rest of his vital signs were within normal limits. His abdomen was mildly distended and diffusely tender. His white blood cell count was 10.6 x 10^9/L with absolute neutrophil count of 9.4 x 10^9/L. Lactate was 9.1 mmol/L. CT angiogram was only notable for extensive vascular plaque. There was no leak around the stent. He was stabilized with fluids and antibiotics. Ultimately, blood cultures collected on admission grew non-typhoid salmonella, suggesting bacteremia from a mycotic aneurysm as the etiology of this patient’s sepsis.

DISCUSSION: Bacteremia from a mycotic aneurysm is not a common cause of sepsis, but should be considered in those with known or new aneurysms. Mycotic aneurysms can develop from direct inoculation with arterial manipulation or seeding from prior bacteremia. Risk factors for seeding of the vessel wall include immunosuppression, atherosclerosis, and pre-existing aneurysm. Atherosclerotic plaques provide a location for invasion. Diagnosis of a mycotic aneurysm is based upon imaging and confirmed with blood or biopsy cultures. Salmonella is the causative organism in 15-24% of all cases, with staphylococcus aureus being the most common pathogen overall. Some risk factors for nontyphoidal salmonella bacteremia include extremes of age, immunosuppression, hemoglobinopathies, and alteration of the gastrointestinal tract. Treatment of mycotic aneurysms typically involves both antibiotics and surgery. Resection is preferred, but endovascular repair is becoming more common for high-risk locations. Duration of antibiotics depends on the ability to fully remove infected tissue. Sometimes lifelong antibiotic suppression is required. In this case, it is suspected that the patient was infected with salmonella months prior to his presentation and seeded an atherosclerotic plaque resulting in the aneurysm. Manipulation with endovascular stent placement caused bacteremia. He was not septic immediately after the procedure because of antibiotic coverage for a UTI as well as coating of the stent with antibiotics.
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Abstract Title: Clostridium Difficile: A Common Infection with a Rare, Fatal Complication

Abstract Information:

Case Description

A 34 year old female with recurrent small bowel obstructions, pancreatitis, recent Clostridium difficile colitis, and extensive abdominal surgical history beginning in childhood presented with abdominal pain, distension, anorexia and altered mental status. On a recent admission she was treated conservatively for small bowel obstruction. Since that time, she had slowly declined. CT scan on presentation demonstrated fluid-filled small bowel, stool-filled colon, and hyperenhancing mucosa. She was taken by surgery for exploratory laparotomy given continued elevated lactate and concerning abdominal exam. Intra-operatively, the bowel appeared healthy. After surgery, she was admitted to the ICU where she developed signs of septic shock and was started on broad-spectrum antibiotics. Three days after admission, blood cultures grew Clostridium difficile and the patient was switched to oral and rectal vancomycin with intravenous metronidazole. In the next two days, she developed recurrent hypotension requiring vasopressor therapy. She was taken back to surgery due to concern for bowel ischemia where they found a small perforation, which was repaired. Given continued fevers, Tigecycline and fungal coverage were added to her antibiotic regimen. The patient was taken again back to the operating room two days later due to concerning exam. More areas of the colon were perforated, but these were unable to be repaired due to friability of the tissue. The following day, given the patient’s poor prognosis and continued decline, the family decided to withdrawal care and the patient passed.

Discussion

Clostridium difficile colitis is a commonly encountered infection in the hospital. Extraintestinal manifestations of C. difficile colitis are rare and few cases of C. difficile bacteremia have been reported in the literature. Most prior cases describe C. difficile bacteremia in the setting of a polymicrobial bacteremia, unlike this case. Risk factors based on previous reported cases include malignancy, recent antibiotic use, alcohol abuse and abdominal surgery. Several mechanisms to understand the pathogenesis of C. difficile bacteremia have been postulated. One hypothesizes bacterial transfer through injured intestinal mucosa. Bacterial translocation to blood, lymph nodes, and peritoneum through an inflamed or immunosuppressed gut has also been suggested. In our patient’s case both mechanisms could have been possible given her small bowel obstruction and ileus after recent C. difficile colitis in the setting of chronic illness.
In patients with history of *C. difficile* colitis, abdominal surgery and recent antibiotic use, it is important to keep *C. difficile* bacteremia on the differential. From review of other cases, presenting symptoms including fever and abdominal pain are nonspecific creating further diagnostic dilemma. Given the high degree of fatality, early recognition of *C. difficile* bacteremia is imperative. Additionally, further research is needed to determine appropriate antibiotic therapy in cases where traditional treatment is not adequate to prevent mortality.
Abstract Title: Bradycardia and Mobitz Type I Heart Block in a Case of Intraorbital Foreign Body

Abstract Information:

Abstract: A 25-year-old male was brought to the emergency department by ambulance with a wound to his right eye and facial abrasions. On examination, the patient had a complete hyphema and globe rupture of the right eye. Vision in the left eye was 20/20, with normal pupil and intraocular pressure. He was also noted to be intermittently bradycardic to as low as 19 bpm, and an EKG showed a ventricular rate of 42 with Mobitz Type I atrioventricular block. Toxicology screening was positive for marijuana and cocaine. CT of the head without contrast showed a right globe rupture and a ballistic fragment embedded in the inferior left orbit. The fragment appeared to have traversed the right orbit before penetrating the ethmoid sinuses into the left orbit. The left globe appeared uninjured. After successful emergent repair of the ruptured globe, he required ICU admission due to persistent bradycardia necessitating IV atropine and glycopyrrolate. The patient continued to require pharmacologic intervention for several days. After left exploratory orbitotomy for removal of a retained foreign body, his bradycardia promptly resolved.

Discussion: The oculocardiac reflex (OCR) is a well-described variant of the trigeminocardiac reflex (TCR), in which afferent stimulation of any trigeminal nerve branch can result in increased parasympathetic tone of the vagus nerve. This can result in bradycardia, asystole, nausea, apnea, and hemodynamic instability. Refractory bradycardia requiring intensive care management is rarely seen. Muscle entrapment associated with orbital fractures is a commonly described mechanism for sustained bradycardia, but association with an intraorbital foreign body is very uncommon. Association with second degree heart block is not described in the literature.

The TCR is usually a self-limited phenomena. However, sustained periods of bradycardia have been observed in cases of extraocular muscle entrapment in orbital fractures in children, and also due to intraorbital foreign bodies requiring surgical intervention. Recurrent OCR due to intraorbital foreign body in a patient forty years after the initial trauma has also been reported. Treatment with competitive acetylcholine antagonists, such as atropine and glycopyrrolate, are commonly used and usually effective means of temporarily correcting the bradycardia and asystole, and were used effectively in this case until definitive surgical intervention was performed.

In summary, this case demonstrates the successful treatment of heart block and sustained bradycardia resulting from an intraorbital foreign body. One must consider the possibility of a retained foreign body in cases of orbital trauma and arrhythmia. There are few reported cases of persistent bradycardia secondary to intraorbital foreign bodies, and we believe this to be the first case describing a Mobitz Type I atrioventricular block.
Abstract Title: A 34-Year Old Female with Acute Hypoxemic Respiratory Failure and Proximal Muscle Weakness

Abstract Information:

Case Description: A patient with hypoxemic respiratory failure

Discussion:
Wound associated botulism is an unusual presentation. Early detection of this potentially life threatening illness can significantly shorten length of hospital stay and improve prognosis. We present a case of a 34-year-old female with a history of heroin abuse who presented to the ED with acute respiratory failure, diplopia, and proximal muscle weakness. There was early concern for wound botulism as the instigating process. After discussion with the CDC, she was given equine serum heptavalent botulism antitoxin. Laboratory analysis later confirmed our suspicion. Symptoms improved and the patient was liberated from mechanical ventilation on day 14 and discharged from the hospital on day 23.
Abstract Title: Atypical Presentation and Recovery of Central Pontine Myelinolysis in a Post-Liver Transplant Patient

Abstract Information:

Introduction:
Central Pontine Myelinolysis (CPM) is one of the most debilitating neurological condition that can occur following liver transplantation. This generally occurs in the setting of rapidly corrected hyponatremia and has a poor prognosis.

Case Description:
A 46 y/o M with PMH significant for remote alcohol use, seizure disorder, and cirrhosis developed ESLD over the year prior to liver transplantation despite clearance of Hepatitis C Virus with Sofosbuvir/Ledipasvir therapy. Portal Vein Thrombosis and severe ascites were treated with Transjugular Intrahepatic Portosystemic Shunt (TIPS). He presented for liver transplantation without evidence of bleeding or infection. TIPS was confirmed to be patent via US and he had been treated with lactulose therapy for Hepatic Encephalopathy. He underwent liver transplantation on hospital day #4; Model for End Stage Liver Disease (MELD) at that time was 16. Approximately, 10 days post-transplantation the patient became lethargic, with full body motor weakness progressing to paralysis requiring mechanical intubation for over 2 weeks. He exhibited “locked in symptoms” prior to sedation accompanying mechanical ventilation. Non-contrasted MRI of brain showed hyper-intense signal within the central p ons. Spinal fluid obtained via lumbar puncture lacked acute abnormalities. EEG was performed with no signs of seizure activity. Of note, the patient’s serum sodium levels prior to, during and after surgery varied only between 129-135 mg/dl. Neurological consultation supported a diagnosis of CPM. Tacrolimus induced neuro-toxicity was additionally considered. Tacrolimus therapy was replaced with Basiliximab therapy. The patient recovered motor function slowly over more than 4 months without any discernible residual motor- nerve deficits.

Discussion:
Post-transplant CPM has an estimated incidence ranging from of 5-29% depending on the study evaluated. This condition typically appears 5-7 days post-transplant but cases have been reported up to 12 days after liver transplant, however, this is rare. There is sufficient data to suggest that pre-transplantation baseline sodium plays a predictive indicator for incidence of CPM. However, recent literature suggests patients may be more prone to this condition secondary to the immunosuppresants, which they receive post–transplant; most notably, Cyclosporine. There have been reports of Tacrolimus induced PRESS syndrome and this is a noted complication through the manufacturer. However, there have been far less associated case reports associated with CPM, but it has been reported.

This case suggests that CPM presenting post-hepatic transplant may be associated with immunosuppressive neurotoxicity rather than the more common osmotic risk of hyponatremia. Additionally in this case, it seems that these neurological deficits may improve, possibly correlated with early diagnosis and immunosuppressive agent change.