NEW JERSEY CHAPTER

AMERICAN COLLEGE OF PHYSICIANS

REGIONAL SCIENTIFIC MEETING

ASSOCIATES ABSTRACT COMPETITION

MARCH 6, 2015

PARTICIPATING INSTITUTIONS

Thank you to all the programs who submitted abstracts for this year's abstract competition. Abstracts were received from the following programs:

- Atlanticare Regional Medical Center (Dominik Zampino, MD, FACP)
- Capital Health Regional Medical Center (Saba Hasan, MD, FACP)
- Cooper University Hospital (Brian Gable, MD, FACP)
- Englewood Hospital And Medical Center (Jonathon Shammash,MD)
- HUMC Mountainside (Bijal Mehta, MD)
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DISCLAIMER

It is assumed that all participants adhered to the rules as stated in the original abstract submission form. It is also assumed that the abstracts submitted were original works, represented by the true authors. The abstracts appear in no particular order. Judging was performed in an attempt to minimize bias. Judges were unaware of the authors or institutions the competitor unless they were directly involved with the associate. Although there were many excellent abstracts those selected to be presented as poster or oral presentation were chosen on the basis of content. This content was felt to be intriguing from a clinical education standpoint, thought provoking, or could stimulate debate regarding our current practice of medicine.

ORAL PRESENTATIONS

THE USE OF A STANDARD VISIT TEMPLATE TO MONITOR PATIENTS ON OPIOID PRESCRIPTIONS IN THE PRIMARY CARE CLINIC: A METHOD FOR A SAFE PRACTICE

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Englewood Hospital and Medical Center (Jonathon Shammash, MD)

BACKGROUND: For healthcare professionals, medical errors relating to opioid medications are at the heart of many lawsuits, with most stemming from monitoring-related errors. While there has been improvement in awareness of prescription medications in the US in the past few years, the abuse of prescribed pain relievers continues to rise. In our community primary care clinic, failure of appropriate documentation and monitoring was identified as a major area for improvement to reduce opioid abuse.In this paper, we present an implementation of a template for the electronic medical record system as an intervention to improve our practice in the outpatient primary care clinic.

METHODS: This is a prospective study on the implementation of a template in an electronic medical record system (EMR) to monitor patients receiving opioid prescriptions from our providers.The template was designed for internal medical residents using eClinicalWorks. It was to be used during any patient visit for refills of prescription. We also encouraged physicians to obtain access to the NJ prescription-monitoring program. All patients who used more than two opioid prescriptions in the last year were included. A total of 36 patients were identified. Data were collected after 3 months. Outcome measures included the fraction of patients who signed the opioid contract, the fraction of medical residents who used the template for documentation, the fraction of preceptor physicians who used New Jersey's Prescription Monitoring Program and the number of patients who were denied further prescriptions for detected aberrant behavior.

RESULTS: All patients receiving chronic pain management had signed opioid contracts, up from 38% before template adoption. 92% of residents used the template during each follow-up visit. Patient compliance with urine toxicology screens increased from 58% to 65% after implementation of the template. 61% of physicians verified the patient's medication list during pain management visits with New Jersey' prescription monitoring program. By the end of the study, a total of 21 patients were no longer prescribed opioids for violation of contract, aberrant behaviors or referral to a pain specialist.

CONCLUSION: We developed a template for an electronic medical record system for monitoring pain regimens that include opioids in the primary care clinic. Physicians find the template easy to use. It helps bring non-adherence to opioid regimens to provider's attention earlier, which improves patient's safety and physician's liability.

ARE WE BEING AN OXY-MORON: THE OVERUSE OF OXYGEN IN A COMMUNITY HOSPITAL SETTING

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Monmouth Medical Center (Margaret Eng, MD, FACP)

INTRODUCTION: Oxygen is one of the most important and yet the most misused therapy in an in-patient hospital setting. In spite of having clear indications physicians often tend inadvertently order oxygen on every patient being admitted. This leads to wastage of resources and increases the cost of healthcare. More importantly, oxygen therapy is not benign and has deleterious adverse effects. Our objective was to study the overuse of oxygen and institute an intervention to prevent the wastage of resources and prevention of such potential adverse events.

METHODS: We designed a pilot project and implemented on one of our telemetry units. We first assessed the patients on a single inpatient floor at bedside and looked into their electronic health records for indication of oxygen, co morbidities, orders for oxygen therapy, orders for titration, actual implementation of the physician orders by nurses and respiratory therapist. Our intervention was to educate the residents, nurses on the targeted floor and respiratory therapists about the potential adverse effects of overuse of oxygen, importance of titration of oxygen to a set goal, indications and expenses involved in the usage and wastage of oxygen therapy. After 4 weeks of intervention, we collected post-intervention data using the same parameters on the same floor.

RESULTS: The total number of patient's in the pre-intervention and post intervention arms were 40 each. In the pre-intervention arm, 28 patients had active orders of oxygen of which 24 had indicationsto be on oxygen therapy. 18 patients were using oxygen. 12 patients were using oxygen without being titrated to the goal of saturation >92%. In the post-interventiom arm 22 patients were on oxygen of which all the patient's had an indication to use oxygen (p-value - 0.6825). 20 of these 22 patients were using NC thus with a significant P-value of 0.0447 after an intervention. Only 4 of these patients were on therapy without titration (p value – 0.0761). Based on our calculations, the total annual saving after 1 intervention on a floor having 40 patients was $2441.12.

CONCLUSION: Based on our results, we concluded that oxygen is often used a placebo because of lack of awareness of its potential hazards as mentioned above and its expenses involved. This involves: (1) Oxygen therapy being initiated without an appropriate indication. (2) Wastage of oxygen and oxygen delivery devices in patients who are off the floor or are doing well without oxygen therapy (due to lack of titration). (3) Lack of awareness about adverse effects of oxygen. By the means of education of the physicians, trainee physicians, nurses and ancillary staff, we calculated that we can save a significant amount of expense and also avoid the preventable adverse effects of overuse of oxygen therapy.

IMPROVED EFFICIENCY AND COST SAVINGS FROM TRIAGING SELECTED TRANSIENT ISCHEMIC ATTACK PATIENTS TO OUTPATIENT URGENT CARE

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Monmouth Medical Center (Margaret Eng, MD)

BACKGROUND: Although most patients presenting with suspected transient ischemic attack (TIA) in the United States are currently hospitalized for urgent evaluation, many are later found to have alternate diagnoses, and it is not clear that hospital admission is either necessary or cost effective in all cases.

OBJECTIVE: To report the outcomes, diagnostic efficiency and cost savings from triaging selected transient ischemic attack (TIA) patients with lower expected stroke risks into an outpatient TIA rapid evaluation center (TREC) to avoid hospitalization.

METHODS: We started an open-access ABCD2 score-based outpatient TIA Rapid Evaluation Center (TREC). Patients referred to the TREC are seen on the next weekday and undergo a diagnostic evaluation then consultation with a stroke neurologist. We collected prospective data from all TREC patients seen during its first year, and compared them to the patients who were still admitted to the hospital with a primary diagnosis of TIA during the same period.

RESULTS: We saw 74 TREC patients within an average of 1.25 days of referral during its first year of operation (56 from the emergency room and 18 from physician offices). Only 2 TREC patients needed admission to the hospital, the remainder completed their evaluation as out-patients. Only 1 TREC patient had a follow-up cerebrovascular event. Patients referred to the TREC had lower ABCD2 scores (1.8 vs. 3.8, $p < 0.001$) and were less likely to have a final diagnosis of TIA (19% vs. 77%, $p<0.001$). Nearly all patients underwent CT scan, lipid panel and EKG. However, TREC patients were more likely to undergo carotid ultrasound (99% vs. 84%, $p=0.001$) and MRI of the brain (89% vs. 68%, $p=0.001$). Based from our financial analysis, TREC patients were evaluated at significant cost savings. Both average hospital charges ($2,270 vs. $6,232, $p=0.03$) and average hospital costs ($666 vs. $6,523, $p<0.00001$) were significantly lower in TREC patients compared to hospitalized patients. In its first year, institution of the TREC resulted in average cost savings of $340,000 at our community medical center.

CONCLUSION: Our TREC program allowed us to avoid hospitalization for selected TIA patients, and still offer timely and efficient diagnostic evaluations at significant cost savings.

IMPROVING TELEMETRY UTILIZATION IN MEDICINE TEACHING SERVICE

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Saint Peter's University Hospital (Nayan Kothari, MD)

INTRODUCTION: Telemetry is a commonly used tool to monitor patients at high risk for arrhythmia or sudden death. While the American Heart Association (AHA) has issued guidelines for appropriate use of telemetry, unnecessary use continues to lead to wastage of resources. The goal of our study was to develop and assess the efficacy of an intervention to reduce inappropriate utilization of telemetry.

METHODS: We performed chart audits of patients (N = 122) admitted to Cardiac Progressive Care Unit (CPCU) or Med/Surg unit with cardiac monitor for the month of April 2014. Residents and faculty were educated regarding the AHA classification (Classes 1,2 and 3) and indications for telemetry. Using pre-designed forms, data were again collected for patients (N = 77) admitted to Cardiac Progressive Care Unit (CPCU) or Med/Surg unit with cardiac monitor for the month of September 2014. The residents were required to complete the forms on daily basis. Data were compared across intervention (pre vs post), based on location (CPCU vs Med/Surg). Primary endpoints included number of patients on telemetry and length of stay (LOS). Data were compared using two-sample t-test or chi-square.

RESULTS: In the Med/Surg unit, there was a significant decrease in the total number of patients (61 vs 32, $p<0.002$), driven mostly by a reduction in the number of patients admitted with Class 3 indications (34 vs 8, $p<0.001$). LOS, however, was not significantly decreased [4.29(0.35) vs 3.65(0.48), $p=0.27$]. In the CPCU there was no significant difference in number of patients (61 vs 45, $p=0.12$) or LOS [3.68(0.30) vs 4.47(0.43), $p=0.14$] following intervention.

DISCUSSION: The effects in this study were modest, however they highlight the importance and efficacy of our intervention. Reduction in the number of patients on the Med/Surg unit prevents unnecessary testing and downstream costs that the hospital would otherwise incur from continuous telemetry monitoring. We observed a trend towards decrease in number of patients admitted to CPCU during the post-intervention period, reducing hospital cost by approximately $200,000. Additionally, the increase in LOS on CPCU, though not statistically significant, could be due to patients with appropriate diagnoses being admitted to CPCU.

CONCLUSION: Simple intervention like educating physicians can play a crucial role in increasing awareness of the telemetry guidelines. Together with frequent monitoring it has the potential to significantly improve hospital resource utilization.

POSTER PRESENTATIONS

CLARKSON'S DISEASE: A RARE POTENTIALLY FATAL CLINICAL SYNDROME WITH UNDETERMINED PATHOPHYSIOLOGY

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Idiopathic Systemic Capillary Leak syndrome (SCLS), also known as Clarkson's disease is a rare and potentially fatal disorder of unknown etiology. It is characterized by episodes of profound hypotension, generalized edema, hemoconcentration and hypoalbuminemia without albuminuria. Fewer than 150 cases have been described in the literature, since it was first reported in 1960, with a preponderance for middle aged white males. We illustrate a case of an acute SCLS episode in an otherwise healthy young Hispanic male. A 24 year old male presented to the emergency department with chest pain and shortness of breath. The patient was diagnosed with SCLS two years ago and was maintained on Intravenous Immunoglobulin (IVIG) infusions every two weeks. On presentation, the patient was tachycardic, tachypneic, hypotensive, and afebrile. He had dry mucus membranes, delayed capillary refill, cool extremities and diminished peripheral pulses. Electrocardiogram showed sinus tachycardia without ischemic changes. He received three liters of 0.9% normal saline fluid boluses, and was continued on aggressive fluid resuscitation, with concurrent albumin and IVIG infusions. A Transthoracic Echocardiogram obtained to evaluate his cardiac function showed preserved left ventricular ejection fraction (LVEF of 60%). Although the patients' blood pressure improved with crystalloid and colloid resusitation, he remained tachycardic and tachypneic, eventually becoming hypoxic. A Chest X-ray revealed pulmonary vascular congestion. Pulmonary embolism was ruled out with a normal ventilation-perfusion scan. Intravenous fluids were discontinued and diuretic therapy was initiated with additional support of Bi-level positive airway pressure. Despite these measures, the patient's respiratory status continued to deteriorate requiring intubation, mechanical ventilation and transfer to the intensive care unit. There he recieved Levophed and aggressive diuretic therapy. The patient was successfully extubated on day 12 and subsequently discharged home on day 13. SCLS is a challenging entity owing to its rarity, uncertain pathophysiology, and lack of validated diagnostic criteria and therapeutic modalities. The syndrome has been described as having several distinct phases. The initial leak phase, consisting of hemoconcentration and hypovolemia and the post leak phase consisting of restoration of capillary barrier function with fluid mobilization from the tissues into the circulation and auto-diuresis. As our case demonstrates, cardiopulmonary failure can occur due to overzealous fluid resuscitation during the post-capillary leak phase. Early central venous pressure monitoring to guide fluid and cathecholamine therapy through each phase may have been beneficial. Other documented complications include compartment syndrome, venous and arterial thrombosis and renal failure from hypoperfusion-induced acute tubular necrosis or myoglobinuria from rhabdomyolysis. The literature shows that steroids, terbutaline, aminophyline and immunoglobulins have been used with varying degrees of success. Further investigation to determine the underlying pathophysiology and perhaps target more adequate therapeutic approaches may prove beneficial in understanding the mechanisms behind this rare and potentially fatal condition.

HEMOLYTIC UREMIC SYNDROME ASSOCIATED WITH CLOPIDOGREL USE – A CASE REPORT

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INTRODUCTION: The antiplatelet drug clopidogrel has largely replaced ticlopidine, due to an association between ticlopidine and thrombotic thrombocytopenic purpura–hemolytic uremic syndrome (TTP-HUS). Clopidogrel has been reported to be safe and effective in reducing vascular events. Nevertheless, there is growing evidence that Clopidogrel may also be associated with TTP-HUS.

CASE REPORT: A 43 year old male with history of chronic kidney disease, hypertension, and heart failure with preserved ejection fraction presented with progressively worsening abdominal pain, nausea, vomiting, dizziness, weakness, constipation and anuria for several days. The patient underwent cardiac catheterization one month before presentation and was taking Clopidogrel since then. Vital signs included blood pressure 240/148 mmHg; heart rate 92 bpm; respiration rate 20/min; temperature 98 F; oxygen saturation 100% on ambient air. Physical examination was significant for moderate discomfort due to abdominal pain, icteric sclerae, right upper quadrant and epigastric tenderness with a normal neurologic exam. Initial laboratory data revealed creatinine 9.94 mg/dL, BUN 87 mg/dl, WBC 10,900/L, platelets 30,000/L, LDH 6375 U/L, total bilirubin 4.2 mg/dL, direct bilirubin 0.7 mg/dL, INR 1.0 and schistocytes on peripheral smear. The patient underwent emergent plasmapheresis given the high suspicion for thrombotic thrombocytopenic purpura (TTP). Clinical improvement was achieved within the first 24 hours. Platelet count steadily increased until normalized. ADAMTS13 activity sent on admission was normal; therefore the diagnosis of TTP was excluded and treatment for hemolytic uremic syndrome (HUS) was started with Eculizumab. Patient was discharged stable to home carrying a diagnosis of end stage kidney disease requiring dialysis. Clopidogrel was not reintroduced and on follow up patient was clinically stable with normal platelet count.

DISCUSSION: Clopidogrel has an improved safety profile compared with that of ticlopidine; however, recent reports have implicated clopidogrel as a possible causative agent of thrombotic thrombocytopenic purpura-hemolytic uremic syndrome (TTP-HUS), which is the most severe adverse reaction associated with ticlopidine. Phase III trials involving 20,000 patients treated with clopidogrel yielded no reports of TTP-HUS. However, with the increasing number of coronary stent procedures performed annually, the potential exists for recognition of clopidogrel-associated TTP- HUS. Several case reports have implicated it as a cause of TTP-HUS. Although there are only a few documented cases of clopidogrel-induced TTP-HUS, growing awareness of the possible problem may result in increased reporting. The index of suspicion for TTP-HUS should be high when we encounter patients taking clopidogrel who present with unexplained fever, renal failure, neurologic symptoms, bleeding, purpura or thrombocytopenia. Recognition of a drug-associated etiology in a patient with TTP-HUS is critical to avoid re-exposure and recurrent illness.

ANTI-COAGULATION IMPROVED HEADACHE ASSOCIATED WITH LEVONORGESTREL-RELEASING INTRAUTERINE DEVICE

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Cerebral venous sinus thrombosis has an estimated annual incidence of 3-4 cases per 1 million people, with about 75% of cases occurring in women. Worsening and gradually progressive headache is the most common clinical presentation, however stroke-like symptoms including aphasia or hemiparesis, seizures, or signs of increased intracranial pressure may also be presenting symptoms. Cerebral venous thrombosis is diagnosed with abnormal signal on brain MRI or absence of flow on a MRV. Risk factors for developing cerebral venous thrombosis include prothrombotic conditions and hormonal contraceptives. A 34-year-old woman without preexisting medical problems presented with a weeklong severe right-sided headache associated with nausea and photophobia. She had a levonorgestrel-releasing intrauterine device insertion a month before her presentation. She had normal vitals and normal physical examination, including an unremarkable neurological examination. She was treated for migraine headache with sumatriptan and ibuprofen. Her symptoms improved minimally but she returned after two days with worsening headaches and nausea. A non-contrast magnetic resonance imaging (MRI) brain showed a filling defect and a magnetic resonance venogram (MRV) of head demonstrated occlusive thrombus in the sagittal sinus and bilateral transverse sinuses, confirming a diagnosis of cerebral venous thrombosis. She was treated with intravenous heparin and continued on warfarin. The levonorgestrel-releasing intrauterine device was removed. Her headaches improved in two weeks, and a repeat MRV of head during follow up five-months later showed significant improvement in the previously described thrombi. Raising awareness of cerebral venous thrombosis may improve the diagnosis of this condition. It should be considered in a young or middle-aged patient with recent or unusual headache, those who are pregnant, have existing thrombophilia, or those on hormonal contraceptive therapy. Anticoagulation is the mainstay treatment and more than 80% of patients treated for this condition have a good neurologic outcome.

UNCONTROLLED DIABETES AND PERFECT GLYCOSYLATED HEMOGLOBIN: ARE WE MISSING SOMETHING?

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INTRODUCTION: Hemoglobin A1c (HbA1c) may be an unreliable measure of glycemic control in patients with inherited hemoglobinopathies.

CASE PRESENTATION: A 36-year-old African-American woman established care with history of type-2 diabetes mellitus (T2DM) for 2 years. She was asymptomatic. She had a strong family history of T2DM. Although she was taking metformin 500 mg orally twice-a-day, her fasting blood glucose ranged between 200 and 350 mg/dL. She admitted having difficulty in limiting high calorie food intake. She denied use of alcohol, cigarette, or drugs. Her BMI was 36.5 Kg/M2. Her vital signs were normal. Rest of the physical examination was unremarkable. Relevant diagnostic studies showed fasting blood glucose 286 mg/dL, total cholesterol 232 mg/dL, LDL 115 mg/dL, triglycerides 410 mg/dL, hemoglobin 13.8 g/dL and urine microalbumin 13.2 mg/24-hour. Her HbA1c was 4.1%. Considering her longstanding history of elevated fasting glucose levels the value of HbA1c was deemed possible lab error. A repeat HbA1c was 4.0%. An underlying associated hemoglobinopathy was suspected. Her hemoglobin electrophoresis showed 60% HbA (less than 3.5% HbA2) and 40% HbS confirming a diagnosis of sickle cell trait. Metformin dosage was increased and she was counseled for low-calorie diet and exercise, in addition to genetic counseling. Her long-term glycemic control was subsequently monitored by periodic measurement of serum fructosamine levels.

DISCUSSION: HbA1c measures the percentage of glycalated hemoglobin A, and is usually an excellent measure of glycemic control over the preceding 2-3 months. However, it can be unreliable in monitoring diabetes in people with inherited hemoglobin variants. The two most common variants, HbS and HbC, exist alongside normal HbA in the asymptomatic heterozygote forms known respectively as sickle cell trait and HbC trait. The prevalence of sickle cell trait in the African American community is 8-10% and may be as high as 25-30% in West Africa. Furthermore, the prevalence of T2DM in African-Americans over 20 years of age is about 20%, with a rising disease burden in West Africa as well. Given this epidemiology, clinicians must be aware of potential limitations of the HbA1c test, as it can either over- or underestimate average blood glucose. A hemoglobinopathy should be suspected when blood glucose measurements do not correlate with HbA1c. In such cases, an alternative test, serum fructosamine, a measure of glycated protein, should be considered. However, fructosamine has its own limitations. It measures blood glucose over a shorter period of only 2-3 weeks and is a less standardized assay. Furthermore, there is a lack of evidence validating its use as a marker for glycemic control and microvascular risk. Conclusion: Inherited hemoglobin variants should be considered in patients with discrepancies between HbA1c and blood glucose measurement. In such cases, serum fructosamine may be considered as an alternative test.

HASHIMOTO'S LEGACY: MORE THAN JUST THE THYROID

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BACKGROUND: Steroid-responsive encephalopathy associated with autoimmune thyroiditis (Hashimoto's encephalopathy) is an exceedingly rare syndrome associated with anti-thyroid antibodies that is characterized by a range of neuropsychiatric symptoms. Given its variable presentation, no clear diagnostic criteria have been established. The diagnosis may be overlooked when the evaluation for other etiologies of encephalopathy has been unrevealing.

CASE PRESENTATION:A 44 year-old male with a medical history significant for Hashimoto's thyroiditis with resulting hypothyroidism, vitiligo, and rheumatoid arthritis presented to the emergency department with 3-4 months of chronic and progressive behavioral change that included increased irritability, introversion, insomnia, and the recent development of hallucinations, myoclonus, and tremors. Physical exam revealed frequent episodes of myoclonus, clonus, and symmetric hyperreflexia of the upper and lower extremities. Imaging studies of the brain revealed no significant abnormality. Laboratory studies showed an elevated thyroid stimulating hormone of 156.5 uIU/ml (normal 0.27-4.20 uIU/ml), elevated anti-thyroglobulin antibody: 27 IU/ml (normal ≤ 1 IU/ml), and elevated antithyroperoxidase antibody: 49 IU/ml (normal < 9 IU/mL). Serum vitamin B12, ammonia, cosyntropin stimulation test, lumbar puncture and cerebrospinal fluid analysis for an infectious etiology were normal. Electroencephalography revealed epileptiform activity with generalized sharp and slow waves associated with episodes of bilateral myoclonic jerks. Given the patient's presenting neurological symptoms in the setting of significant hypothyroidism and elevated anti-thyroid antibodies, a clinical diagnosis of Hashimoto's encephalopathy was established. The patient was started on intravenous methylprednisolone (1 mg/kg) and within three days of therapy there was complete resolution of both the clinical abnormalities and the epileptiform activity on repeat electroencephalography. The patient was subsequently discharged on oral prednisone (1 mg/kg/day) with plans to taper as an outpatient.

DISCUSSION: Steroid-responsive encephalopathy associated with autoimmune thyroiditis (Hashimoto's encephalopathy) is a diagnosis of exclusion after other causes of encephalopathy have been excluded. It is most commonly encountered in women and is often associated with elevated serum levels of anti-thyroperoxidase and anti-thyroglobulin antibodies, although the exact pathophysiologic relationship between these antibodies and the encephalopathy remains unknown. Common clinical findings include cognitive impairment, tremor, myoclonus, ataxia, seizures, sleep disturbance, hyperreflexia, and psychosis, which were encountered in our patient. Treatment is high-dose corticosteroid therapy generally tapered to clinical improvement.

CONCLUSIONS: Due to its potential to be effectively treated with immunosuppressive therapy with resolution of many life-altering symptoms, Hashimoto's encephalopathy should be considered in the differential diagnosis of encephalopathy of unknown etiology.

HYPERAMMONEMIA: IT'S NOT ALWAYS LIVER

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INTRODUCTION: Altered mental status (AMS) is one of the leading causes of elderly patients presenting to the emergency department (ED). Hyperammonemia is often missed as a cause of AMS in patients with no prior history of liver dysfunction. We are reporting a case of a patient with AMS that was found to have with a normal liver.

CASE PRESENTATION: This is the case of a 68-year-old Hispanic woman with a history of hypertension, non-insulin dependent diabetes mellitus and dyslipidemia who presented to the ED with AMS for 1 day. Collateral history from the patient's daughter revealed that the patient had been having chronic constipation for some time with intermittent episodes of diarrhea. Her home medications included losartan/hydrochlorothiazide, simvastatin, glimepiride, and sitagliptin/metformin. She had no history of alcohol consumption or liver dysfunction. In the ED, her vitals were stable. Her Glasgow Coma Score was determined to be 5 (E1V1M3) and physical examination was significant for generalized abdominal tenderness to palpation and hyperactive bowel sounds. Initial lab values were significant for a white blood cell count of 22.3, BUN of 30.0 and creatinine of 1.5. The patient's aspartate aminotransferase was 66 IU/L while her alanine aminotransferase was 38 IU/L. The alkaline phosphatase was 135 IU/L, total protein 7.6, and albumin 3.3. Her lactic acid was 5.1 and ammonia was 102 UMOL/L. A stool sample was positive for Clostridium difficile antigen but negative for the toxin. The patient was admitted to the intensive care unit for further evaluation. A CT of the abdomen with PO and IV contrast was done and demonstrated splenic and gastric varices with a splenorenal shunt and a diminutive but patent portal vein. This finding suggested that venous blood draining from the intestines, and thus rich in ammonia from gut flora (especially during chronic constipation), would bypass liver detoxification because of the splenorenal shunt and enter systemic circulation through the renal veins into the inferior vena cava. The ammonia could then enter the cerebrospinal fluid and produce an AMS. Ultrasonography of liver did not show any evidence of parenchymal abnormality of the liver or increased portal vein pressure. The patient's hyperammonemia was managed with lactulose and C. difficile was treated with metronidazole and vancomycin. Her mental status improved, and she was discharged home on lactulose.

DISCUSSION: The presence of hyperammonemia with no liver dysfunction can go unnoticed. There have been case reports of congenital splenorenal shunts, which can bypass the excess ammonia from the liver directly to the systemic circulation. Enzyme deficiency in the urea cycle can also produce hyperammonemia in a normal liver.

CONCLUSION: The presence of splenorenal shunts should be considered in patients with elevated ammonia level in the absence of significant liver dysfunction.

UNCONTROLLED DIABETES AND AS PRESENTING SYMPTOM OF MINIMAL CHANGE DISEASE

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OBJECTIVES: 1) Recognize that diarrhea due to bowel wall edema can be a presenting symptom of nephrotic syndrome. 2) Recognize the differential diagnosis of bowel wall thickening on CT scan in a patient with nephritic syndrome.

CASE: 24 year old woman presented to the emergency department with 1 day of watery diarrhea associated with right upper and lower quadrant abdominal pain. ROS: 20 pound weight gain in one month. CT Abdomen/Pelvis with PO/IV contrast: small amount of ascites; circumferential wall thickening of the cecum and entire ascending colon. Her history and imaging findings led to a working diagnosis of right sided colitis, and she was discharged home on ciprofloxacin and metronidazole. She was called back two days later when 1/2 blood cultures grew gram negative bacilli. EXAM: BP: 130/64 Periorbital swelling, +1 sacral/+2 pretibial pitting edema. LABS: Albumin <1g/dL, total cholesterol 329mg/dL, triglycerides 158 mg/dL, LDL 221mg/dL, HDL of 52mg/dL, 24-hour urine protein 8.39 grams. C3 190 mg/dL(85-170), C4 30.2mg/dL(16-40), ANA negative, ASO Screening negative (153iU/mL). Urine creatinine 222.65mg/dL,urine protein 987mg/dL (Protein/Creatine Ratio 4.43). HIV not detected by enzyme immunoassay. Urinalysis >300mg/dL protein, WBC's 8-10/HPF, +nitrites. Normal kidneys on CT imaging. Antibiotics were continued for the bacteremia, the diarrhea improved during the hospitalization, possibly due to the use of low doses of a loop diuretic to palliate the symptoms of the diffuse peripheral edema. Renal biopsy revealed Minimal Change Disease. The patient is followed as an outpatient and was started on oral prednisone 60mg/day.

DISCUSSION: Nephrotic Syndrome is diagnosed in the presence of proteinuria greater than 3.5 grams per day, weight gain, edema, hypoalbuminemia and hyperlipidemia. Gastrointestinal symptoms are not commonly described and a pubmed.org search (terms: nephrotic, bowel, diarrhea, edema) only revealed a case of chylous ascites theoretically due to lymphatic impairment due to bowel wall edema, but no mention was made that bowel wall thickening was documented on imaging studies [PMID 17044480]. The failure of the diarrhea to improve until the admission where low-dose diuretics were given in addition to the antibiotics, and the presence of severe peripheral edema strongly suggest that the CT findings were edema and not the less likely simultaneous presence of concurrent inflammatory colitis in a young woman. It is likely that the bacteremia was transmigration across edematous bowel wall. To our knowledge, this is the first reported case of nephrotic syndrome presenting with diarrhea due to hypoalbuminemia related colon edema leading to decreased absorption of water. Clinicians should be aware that in the presence of nephrotic syndrome with anasarca, the differential of bowel thickening on CT scan includes edema as well as other more common conditions.

AORTICO-LEFT ATRIAL FISTULA: A RARE COMPLICATION OF INFECTIVE ENDOCARDITIS

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Monmouth Medical Center (Margaret Eng, FACP)

INTRODUCTION: Paravalvular aortic root abscess with intracardiac fistula formation is an exceedingly rare complication of infective endocarditis. This complication places patients at increased risk for congestive heart failure, heart block and death.

CASE DESCRIPTION: A 68-year-old Bosnian female with prior aortic valve replacement with a bioprosthetic valve for aortic regurgitation was admitted for worsening shortness of breath, fever and lethargy. She was recently on a vacation in Bosnia, where she fell ill and was hospitalized for 1 month for sepsis and renal failure. She had an extensive past medical history, pertinent for coronary artery disease, s/p PCI and stenting, diastolic congestive heart failure, atrial fibrillation, chronic kidney disease, systemic hypertension, multiple prior cerebrovascular accidents and chronic UTI. In the ED, she became markedly hypotensive and hypoxic. She was intubated and started on dopamine infusion. A bedside transthoracic echocardiogram revealed a paravalvular leak around the bioprosthetic valve, raising concern for an aortic root abscess. EKG revealed atrial fibrillation with low voltage QRS, without evidence of bundle branch blocks or conduction delays. The patient developed septic shock and was started on broad spectrum antibiotic therapy and pressor support. A transesophageal echocardiogram revealed an extensive aortic root abscess. The abscess had ruptured into the left atrium, with a fistula connecting the aortic root to the left atrial cavity. The abscess was located around the bioprosthetic aortic valve which had a large vegetation and severe paravalvular aortic regurgitation. She became hemodynamically unstable during the procedure and was brought to the operating room for emergent surgery. She underwent homograft aortic root replacement, VSD repair and ligation of the aortico-left atrial fistula. Two sets of blood cultures grew Enterococcus faecalis. Postoperatively, she developed worsening septic shock, requiring multiple pressors, disseminated intravascular coagulation, and anuria, requiring CVVHD. She eventually expired 5 days later.

DISCUSSION: Intracardiac fistula formation is a rare and particularly problematic complication of periannular spread of infective endocarditis, with high mortality despite adequate therapy. In prosthetic valves, this process usually begins on the prosthesis cuff, and often extends outside the valvular apparatus, resulting in valvular dehiscence, abscess formation, and myocardial involvement. Operative treatment remains the cornerstone of management. Surgical treatment involves removal of all infected tissue including annular elements, followed by reconstruction of the annulus for safe anchoring of a valve conduit. Early rather than delayed surgical intervention has been shown to improve survival. In our patient, a delay in the diagnosis, which in turn led to a delay in surgical intervention, contributed to the poor outcome. This case illustrates that a high index of suspicion, prompt diagnosis by echocardiography, and early rather than delayed surgical intervention, are crucial to improving treatment outcomes for this rare condition.

ACUTE PANCREATITIS WITH NORMAL LIPASE AND AMYLASE: A CASE SERIES

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INTRODUCTION: Lipase and/or amylase elevation greater than 3 times upper limit of normal (ULN) is one of the three diagnostic criteria for the diagnosis of AP as per the American College of Gastroenterology practice guidelines. Both lipase and amylase are extremely sensitive for the diagnosis of AP with sensitivity in the range of 85-100% for lipase and 81-95% for amylase. AP with either lipase or amylase being normal is a rare entity but has been reported before. AP with both lipase and amylase normal is an extremely rare event with only isolated case reports in literature. We report a series of 9 cases with imaging proven AP who presented with normal lipase and amylase

CASE DESCRIPTION AND DISCUSSION: All the 9 cases were from our institution, admitted with abdominal pain consistent with AP. All the cases had CT abdomen findings consistent with AP. None of the patients had imaging or clinical features of chronic pancreatitis and one patient had recurrent pancreatitis with 3 episodes in the past, the last episode being several months ago. At the time of admission lipase and amylase levels were normal in all the 9 patients. The enzyme levels were repeated within 24 hours in 8 out of the 9 cases and were found to be normal. In 2 cases, the normal enzyme levels could be explained by late presentation, however in majority of the cases, the reason for lack of enzyme elevation was not clear. The mean (±SD) time from the onset of symptoms to the presentation in the Emergency department was 69.33 hours (±1.4). In 6 out of the 9 cases, the etiology of AP was unclear and 1 case was due to gall stones. All the cases were mild as per Modified Atlanta Classification and Determinant based classification.

CONCLUSIONS: Imaging confirmed AP can occur in patient in the absence of pancreatic enzyme elevation. Specific scenarios where enzyme levels are more likely to be normal include: a. Severe necrotizing pancreatitis b. Very early or late in the course of the disease In any patient having risk factors for AP, with abdominal pain consistent with AP, the diagnosis of AP should be considered even if the pancreatic enzyme levels are normal at the time of presentation.

EXCHANGE TRANSFUSION IN SICKLE CELL INTRAHEPATIC CHOLESTASIS

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Hepatic dysfunction in sickle cell disease may be caused by various clinicopathologic entities such as acute hepatic sequestration, sickle cell intrahepatic cholestasis (SCIC), benign hyperbilirubinemia, gallstones, iron overload and viral hepatitis. SCIC happens to carry a high case fatality rate. A 31 year old African American male known to have sickle cell anemia (homozygous - HbSS) presented to the emergency room with bilateral lower extremity and back pain. His past history included recurrent admissions for acute painful crises, multiple blood transfusions leading to iron overload and cholecystectomy. On exam, he was noted to have scleral icterus with all vital signs being within normal limits; a left sided chest wall port was noted to be intact. His laboratory studies revealed the following values: Hemoglobin 6.2, reticulocyte count 16, total bilirubin 4.0 (direct component 1.8), ALT 78, AST 132 and alkaline phosphatase 274. With a presumptive diagnosis of acute painful vaso-occlusive crisis the patient was admitted to the medical floor and prescribed intravenous fluids, hydromorphone for pain relief and one unit of packed red cells. Over the following 48 hours, he developed severe right upper quadrant abdominal pain and his bilirubin rose to 50 mg/dl with direct component measured at 37mg/dl. He was also noted to have renal failure at this time with serum creatinine measuring 3.5 mg/dl. A flat plate and ultrasound of his abdomen were unrevealing and an MRCP done only showed diffusely decreased signal intensity throughout the liver (suggestive of hemosiderosis) and periportal edema. Viral serologies excluded acute viral hepatitis. With the differential of SCIC in mind, the patient was placed on exchange transfusions which dramatically led to an improvement in his overall clinical status and serum bilirubin downtrended to 10mg/dl during his ensuing hospital course. An improvement in his renal function was also recorded. This case brings to our attention the infrequently encountered complication of Sickle Cell Disease, namely SCIC. The pathophysiology is believed to be sickling of the red cells within the hepatic sinusoids which leads to congestion of the vascular bed; this is followed by ballooning and necrosis of the surrounding hepatocytes thereby causing conjugated hyperbilirubinemia. Accompanying renal failure prohibits excretion of bilirubin, compounding the existing problem. The management includes RBC exchange, correction of coagulopathy and encephalopathy if any; and in warranted cases, Liver transplantation. Maintaining an index of suspicion, timely diagnosis and institution of therapy are paramount given the mortality attributed to SCIC in previously reported literature.

ATYPICAL PRESENTATION OF ROCKY MOUNTAIN SPOTTED FEVER IN A YOUNG ADULT

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Rocky Mountain spotted fever (RMSF) is a curable tick-borne disease that can be potentially lethal. Its causative agent Rickettsia rickettsii is a gram negative intracellular bacterium with a tropism for vascular endothelial cells. Classic symptoms of RMSF include fever; which is almost always present, headaches and rash in the appropriate setting. However all of these diagnostic clues may not be present which can lead to delay in diagnosis and appropriate antibiotic therapy leading to poor outcomes in certain cases. RMSF very rarely may involve the myocardium but solely presenting with cardiac signs and symptoms without any of the typical features- fever, rash or headaches is even rarer and may pose a diagnostic challenge. We report a case of an unusual presentation of RMSF in a 20 years old male patient with no past medical history who presented with severe retrosternal chest pain, electrocardiographic changes and troponin elevation without any fever, rash or headaches. The chest pain was associated with diaphoresis, 5 episodes of vomiting but no shortness of breath, abdominal pain. He is a non-smoker, non-alcoholic and denies use of recreational drug. He went camping a month earlier.On admission his Cardiac enzymes showed a CK level of 939IU/L (normal 40-300IU/L), CKMB 58.62ng/mL (normal 1.0-5.0ng/mL). Initial troponin I was 17.50ng/mL (normal <0.30) and 20 hours later was 54.33ng/mL. An electrocardiogram showed a 2mm ST elevation in inferior leads and V6. It also showed mild ST depression in leads V1-V3. Patient was given aspirin, clopidogrel and heparin. A Left heart cardiac catheterization was done, result of which revealed normal coronaries and normal left ventricular ejection fraction. At this point presumptive diagnosis of Myocarditis due to RMSF was made and diagnostic testing was further pursued. Results of RMSF IgG titre was elevated at 1:128 indicating recent infection with Rickettsia rickettsii which is the etiologic agent of RMSF. The patient was subsequently treated with Doxycycline. This case demonstrates the fact that RMSF can present solely with signs and symptoms of Myocarditis without any of the typical features of RMSF. It also strongly emphasizes the need for physicians to have a high index of suspicion in the appropriate setting and recognize the complication of Myocarditis secondary to RMSF and prevent unnecessary invasive cardiac procedures especially in young patients with no significant personal or family history.

STATIN AND ACQUIRED VITAMIN B12 DEFICIENCY

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INTRODUCTION: Vitamin B12 deficiency is estimated to affect 10%-15% of people over the age of 60. Besides malabsorption and pernicious anemia, prolonged usage of certain medications have been linked with Vitamin B12 deficiency.

CASE PRESENTATION: 65 year old female patient with PMHx of HTN and hyperlipidemia presented to ER for one syncope episode. She denied head trauma, no headache, no vision or hearing changes, no palpitation, no weakness or numbness, no abdominal discomfort, no diarrhea or weight loss. She eats balanced diet all her life and she was on HCTZ-candesartan and atenolol for hypertension, and pravastatin was started for hyperlipidemia 10 months ago. Upon admission, her vitals were HR 70, BP 106/53, RR 16, O2 saturation 100% on room air, temperature 99.0. Physical examination including neurological examination was within normal limits. Lab values: Hb 13 g/dl, and MCV 103 fl, compared to Hb 14.2 g/dl and MCV 90.2 fl 10 months ago, Vitamin B12 88 pg/ml, and folic acid was 17.1 ng/ml, homocystine was 27.9 µmol/L, and methylmalonic acid was 1541 nmol/l. Further investigation including MRI, carotid ultrasound, electrolytes and EEG were re-assuring. Vitamin B12 deficiency was diagnosed and pt was prescribed with PO Vitamin B12 and discharged home.

DISCUSSION: Prevalence of vitamin deficiencies increases with age. Prolonged usage of medications such as metformin and H2 blocker have been shown to associated with Vitamin B12 deficiency. One previous study has shown a correlation of simvastatin with vitamin B12 deficiency. In the past, screening for vitamin B-12 deficiency was indicated only for the evaluation of those with relevant symptoms and signs, such as anemia, neuropathy, or cognitive impairment. However, elderly people who have Vitamin B12 deficiency frequently lack the classical signs and symptoms. Statins are widely prescribe as a long term therapy for hyperlipidemia and our case and other studies have shown possible correlations of acquired Vitamin B12 deficiency with chronic statins' usage. Therefore, it seems reasonable to draw baseline CBC and/or Vitamin B12 level and monitor vitamin B12 levels periodically in patients taking stains.

HIV VASCULOPATHY AND VARICELLA VASCULITIS IN A PATIENT WITH HIV/AIDS

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INTRODUCTION: HIV vasculopathy and varicella vasculitis are two infectious causes of strokes in HIV/AIDS patients. Early identification would help treat and prevent recurrence.

CASE PRESENTATION: A 52 year-old woman with HIV/AIDS (CD4 count of 4) presented with altered mental status and being non-verbal for seven hours. Family denied any seizure activity, tongue biting, or bowel or bladder incontinence. Two months prior to admission she had developed an erythematous, vesicular rash on her extremities. Over the past month she had been losing weight with no apparent hematemesis, abdominal pain, hematochezia, melena, fevers, or chills. She appeared cachectic with hyper-reflexia and increased muscle tone in both the upper and lower extremities. Initial labs showed leukopenia with bands of 20%. CT scan of the head showed multiple low-density foci in left frontal lobe and right internal capsule. An MRI showed multiple foci of acute and sub-acute infarction along multiple vascular territories in both cerebral hemispheres. She was started on empiric treatment for meningitis with Ceftriaxone, Vancomycin, Ampicillin, and Acyclovir. An electroencephalogram was obtained that showed moderate slowing and no epileptiform activity. An echocardiogram showed no vegetation. A lumbar puncture was performed which showed clear fluid with an opening pressure of 18 cm H2O, 5 white blood cells (79% lymphocytes), glucose 34, and protein 88. Further cerebrospinal fluid studies detected both Varicella and HIV DNA. She was also seen by Ophthalmology for retinitis which was initially misidentified as CMV. A CT angiogram showed irregularity of left anterior cerebral and right middle cerebral arteries consistent with vasculitis. She was thus diagnosed with HIV vasculopathy and Varicella vasculitis along with vesicular rash and retinitis.

DISCUSSION: Cerebral infarction may be present in 4% to 29% of patients with AIDS based on autopsy results. An HIV-associated vasculopathy has been described in AIDS patients upon autopsy showing small vessel thickening, pigment deposition, and perivascular inflammatory infiltrates in the absence of an identifiable cause (lymphoma, embolism, or non-HIV central nervous system infection). Varicella zoster vasculopathy is often chronic with lesions occurring at the grey-white matter junction and mostly involving both large and small arteries. CSF mononuclear pleocytosis and a shingles rash may not be present in a third of the cases hence a high index of suspicion is necessary, and early and aggressive antiviral therapy may improve the outcome in such patients.

AUTOIMMUNE VESTIBULOPATHY IMPROVED WITH HYDROXYCHLOROQUINE AND STEROIDS

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The most common causes of vestibulopathy are ototoxic aminoglycosides, Meniere's disease and meningitis. Reports on autoimmune vestibulopathy are rare. We present a case of autoimmune vestibulopathy in a 32 year old African-American woman with severe intermittent chronic vertigo and dizziness for fourteen months that improved with steroids and hydroxychloroquine. The dizziness and vertigo affected her daily life and work, sometimes associated with headache and nausea but not with hearing loss, weakness, numbness or tingling. There was no history of antibiotic use or head trauma. Past medical history included Vitamin D deficiency and iron deficient anemia. Physical exam and routine labs such as complete blood count with differential and comprehensive metabolic panel were unrevealing. Treatment was initiated with meclizine and vestibular physical therapy without improvement. Dizziness improved briefly but recurred one week after she was started on topiramate 15 mg bid. The patient was then referred to neurology and rheumatology. Rheumatoid factor, cyclic citrullinated peptides, anti-neutrophil cytoplasmic antibodies, antiphospholipid antibodies, beta 2-glycoprotein-antibody, anti-jo-1 antibody, complement C3 and C4, anti-ds DNA antibodies, antiscleroderma-70 antibodies, CPK, TSH and RPR were within normal limits; erythrocyte sedimentation rate was elevated at 48 mm/hr, C-reactive protein was elevated at 6.5 mg/L, antinuclear antibodies was 1:80; anti-SSA was positive and anti-SSB was negative. Sialogram was unremarkable. MRI of the brain with and without intravenous contrast revealed non-specific nodular hyperintensities in bifrontal and left frontal parietal subcortical white matter and normal bilateral seventh and eighth intracranial nerve complex. Electronystagmogram and rotator chair test confirmed bilateral central vestibular dysfunction and left peripheral vestibular dysfunction. Autoimmune vestibulopathy was diagnosed and she was started on hydroxychlooquine 200mg daily. Methylprednisolone 64 mg per day was added tapering the dose 8 mg every 5 days until 16 mg daily within 4 weeks. She was also placed on calcium and vitamin D. Four weeks later the patient reported significant improvement of dizziness and complete resolution of vertigo, remaining stable on hydroxychlooquine for twelve months now. Autoimmune vestibulopathy is a relatively rare condition. The diagnosis can be difficult as there are no universally accepted diagnostic criteria or diagnostic tests. Our patient had only isolated signs and symptoms of bilateral vestibulopathy. An auto-immunological etiology was likely based on the fact that other causes had been excluded and the good response to steroids and hydroxychloroquine. The treatment trials on autoimmune inner ear disorders that have been published have focused on hearing loss. Very few cases on isolated autoimmune vestibulopathy were reported in the past. From the clinical course and response to treatment of our patient, we conclude that a short term treatment trial of steroids can be diagnostic for autoimmune vestibulopathy and should be started early on to preserve or even improve vestibular function.

BACLOFEN INDUCED BRAIN DEATH?

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INTRODUCTION: Baclofen, a widely used central acting muscle relaxant, can mimic brain death in overdose. While cases published are rare, here we describe a patient who overdosed on Baclofen, met brain death criteria, who subsequently was discharged fully functional. Introduction: Brain death implies the permanent absence of cerebral and brainstem functions—usually described as a persistent vegetative state. In adult patients, trauma and subarachnoid hemorrhage are the most common etiologies of brain death. Before the clinical exam for brain death can be done, a few pre-requisites have to be cleared: core temperature > 36 C, SBP >100, no drug or poisoning which confound the clinical assessment, and exclusion of medical conditions which may confound the exam. All the following must be present to clinically diagnose brain death: absent motor response, pupillary light reflexes, dilated fixed pupils, absent gag reflex, sucking reflex, caloric responses. Furthermore, apnea as demonstrated by the apnea test must be done, as well.

CASE REPORT AND DISCUSSION: Patient is a 30 year Hispanic male with past medical history of paraplegia status post traumatic injury one year prior causing fracture of the T7 vertebrae arrived to the Emergency Department when a friend found him unconscious, nonreactive to verbal and painful stimuli. He was last witnessed the night before carrying out his normal activities of daily living. Status post field intubation, ED examination found him unresponsive to touch, pain and vibration. Reflexes were 2+ bilateral patellar, brachial, bracioradialis and Babinski was down-going. Cough, gag, and corneal reflexes were not able to be elicited but pupils 1mm were fixed, constricted and midpoint unreactive to light and accommodation. Furthermore, caloric testing was done and found to be absent reflex. Tone of upper and extremity limbs was normal. Otherwise, patient's exam was within normal limits. Head CT without contrast displayed mild sinusitis, otherwise normal. Glascow Coma Scale Score was E1V1M1. EKG was also found to be normal sinus rhythm. Home medications were reviewed: Lyrica 3 mg BID, Neurontin 800 mg TID, Omeprazole 20 mg daily, baclofen 10 mg daily and duloxetine 30 mg daily. The first brain death exam was performed the following day which fulfilled brain death criteria. The second exam was scheduled to be done 24 hours later. 12 hours after the first Head CT without contrast was done, imaging was repeated—results were within normal limits. The night of Admission Day 2, the patient self-extubated and was immediately able to speak, and function at his baseline. After further discussion with the patient, he stated to have attempted suicide with baclofen. Any patient on Baclofen meeting brain death criteria should be re-evaluated frequently to rule out reversible metabolic cause secondary to drug overdose.

GLOBAL AMNESIA: RARE CLINICAL PRESENTATION OF POSTERIOR CIRCULATION STROKE

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INTRODUCTION: Beauty of cerebrovascular disease in neurological sciences is ability to localize anatomical lesion based on clinical characteristics. Ability to deduct the anatomical correlation of posterior infarcts is challenging because majority of the time the clinical presentation in these cases is a symptom salad with a syndrome like picture rather than clear cut clinical deficits. Posterior circulation strokes contribute one fifth of all ischemic stroke cases. One of the rarest forms of presentation is by amnesia.

CASE REPORT: An 88 year old Hispanic female was brought to the hospital due to confusion of unknown duration. She was last seen normal 2 days ago. Past medical history is significant for hypertension, hyperlipidemia, chronic systolic heart failure, bio prosthetic mitral valve replacement, atrial fibrillation currently on anticoagulation. History could not be obtained from patient however family reports no recent history of fever, fall or similar experiences in past. Patient was living independently prior coming to the hospital. On physical examination, patient was found to have profound global amnesia with predominant retrograde memory loss. Neuropsychological testing revealed normal executive functioning, intelligence, attention, language, and mood. Her short term working memory was also intact. Her confusion was attributed solely because of her memory loss to the extent where she was unable to identify her family. She had no other complaints. No gross motor, sensory, cerebellar deficits were noted. Initial lab tests revealed patient was sub therapeutic despite being on Coumadin for anticoagulation. Computed tomography of brain without contrast was unremarkable. Magnetic resonance imaging of brain revealed multiple tiny infarcts in bilateral occipital lobes, right cerebellum and posterior pons. The multiplicity of infarcts, regionalization of vascular supply, prominent cardiac risk factors, state of sub therapeutic anti-coagulation point towards etiology of thromboembolism to distribution of posterior circulation. Patient was treated for Ischemic stroke and given higher doses of Coumadin to achieve therapeutic range of INR. At 6 weeks of follow-up patient completely regained her memory and currently has no neurological deficits. One very important differential to consider in this case would be transient global amnesia (TGA), condition whose etiology is still questioned.

CONCLUSIONS: The clinical presentation of vertebra-basilar cerebrovascular ischemic events is radically different from our usual encounters. Since the presentations are very different from each other and have a whole spectrum of symptoms to present with, we should be familiar with these cases for effective diagnosis and management. One step further from stroke would be basilar artery occlusion itself with the similar etiology and pathophysiology and has very high rate of fatality and mortality. This case once again underlines the unique nature of posterior circulation strokes thus helping us in future practice.

A SUSCEPTIBLE BOWEL: THE NATURAL COURSE AFTER DISSEMINATED HISTOPLASMOSIS

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INTRODUCTION: Histoplasmosis is the most prevalent endemic mycosis in the United States entering the human body by inhaling the microconidia, with gastrointestinal (GI) tract involvement reported in about 70-80% of cases. Plenty has been said in regards to isolated episodes of progressive disseminated histoplasmosis (PDH) affecting the intestine, yet to be determined is the natural course of a disrupted bowel rendered susceptible to either a possible structural intestinal complication, or a superimposed infection.

CASE DESCRIPTION: A 49 year-old heterosexual male from Honduras with AIDS (CD4 count of 87 and viral load of >10 E6 copies/ml off HAART) presented with fever, abdominal pain, and bloody mucoid diarrhea. He appeared toxic, wasted, but hemodynamically stable. There was no rash, peripheral lymphadenopathy, with hyperactive bowel sounds, borborygmi, and periumbilical tenderness radiating to left lower quadrant associated with guarding. He required multiple blood transfusions due to significant anemia. Colonoscopy revealed a mass-like ulcer in the ascending colon, initially identified as bowel wall thickening accompanied by fat stranding on CT abdomen. Biopsy was consistent with chronic granulomatous fungal colitis. Cosyntropin stimulation test ruled out adrenal insufficiency. Induction Amphotericin B was started followed by oral itraconazole with significant clinical improvement. Urinary antigen for Histoplasma was positive. Patient returned one week later with worsening abdominal distension and recurrence of diarrhea secondary to noncompliance to medications. C. difficile colitis complicated the course but improved after oral metronidazole. Currently, patient is at his 4 month of itraconazole with a plan to repeat colonoscopy to assess colonic mass.

DISCUSSION: Histoplasmosis spectrum of disease varies according to the immunological status of the host with 59% of cases occurring in immunocompromised, and surprisingly 41% in immunocompetent patients. PDH has been previously described as GI bleeding, malignancy-like masses, perforation, peritonitis to a malabsorpton syndrome depending on the involvement of the intestinal layer mainly from the ileo-cecal region to colon due to an abundance in gastrointestinal-associated lymphoid tissue (GALT). The risk of relapse or complications are significantly reduced by maintenance therapy after 12 to 24 months. Nevertheless, PDH sometimes takes an unexpected turn when co-infections such as parasitic, viral or bacterial agents are present leading to an increased Histoplasma pathogenicity. As seen in our case, C difficile could have potentially precipitated a fulminant presentation based on markers of intestinal inflammation and the emergence of strains such as the virulent C difficile NAP1/027. Should this occur, be alert to manage differently as it could affect the long-term outcome.

DTAP-VACCINE INDUCED MYOPERICARDITIS MIMICKING ST ELEVATION MYOCARDIAL INFARCTION

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INTRODUCTION: Vaccine related myocarditis is one rare cause of myocarditis. Smallpox vaccination-induced myocarditis has been well-reported in literature however only two cases of myocarditis related to DTaP vaccination have been reported. Both of these cases were documented in the pediatric population.

CASE DESCRIPTION: We present a 37 year old Portuguese male with past medical history only significant for drug abuse that came to the emergency room with complaints of pressure like chest pains, pleuritic in nature associated with fevers and chills for five days. The patient had a DTAP vaccine placed five days prior to the admission after which his symptoms began. Upon arrival to the emergency room an electrocardiogram was performed which showed ST elevations in leads II, III, AVF, v4 and v5 . An emergency echocardiogram done at bedside showed an ejection fraction 40% with moderate global hypokinesis but no evidence of pericardial fluid. Immediate cardiac catheterization revealed normal coronary arteries. First Troponin I level was elevated at 30. The patient was transferred to CCU with a presumed differential diagnosis of Myocarditis vs Coronary Vasospasm. Rheumatologic screening and serum viral antibody titers for suspected acute infectious causes were all negative. This included Coxsackie virus group B, Human immunodeficiency virus (HIV), Cytomegalovirus, Ebstein-Barr virus, Hepatitis virus family, and Influenza viruses. In addition, urine and hair samples were sent for drug screen; they were negative for recent cocaine or amphetamine use. Cardiac MRI with and without gadolinium was performed for definitive diagnosis and showed delayed myocardial enhancement involving the myocardium of the mid-inferior and mid-inferolateral wall of the left ventricle. These findings were consistent with edema, inflammation, and myocarditis. Troponin I values were trended to monitor extent of myocardial damage and serial values reached 30, 46.4, 17.8, 0.19, respectively. The patient was treated with Colchicine and NSAIDs and his symptoms improved significantly over the following 3 days.

DISCUSSION: Myocarditis has multiple etiologies however vaccine related causes are rare. In a thorough review of literature, we found only two cases of myocarditis induced by tetanus vaccine. Both were reported in the juvenile population: one in a 3 month old after a DTAP vaccine and the other in a 13 year old male after tetanus vaccination. Our case may be the first one seen in an adult. DTAP induced myocarditis should be suspected in patients with chest pains and fevers with an antecedent of the vaccine and promptly evaluated as it can cause detrimental repercussions.

MEDICAL STUDENT RESEARCH POSTER

INFLAMMATORY MARKERS ASSOCIATED WITH DISEASE PROGRESSION OF HIV, HEPATITIS B, AND HEPATITIS C: AN ANALYSIS OF NATIONAL HEALTH AND NUTRITIONAL EXAMINATION SURVEY BETWEEN THE YEARS 1999-2010

Srikanta Banerjee, Patrick Ogueji Ofor, MD; William Anthony, MD, MBA; Raxit Patel, MD, MPH

Introduction: Chronic viruses like Hepatitis B, Hepatitis C, and HIV are known to be associated with cardiovascular disease (CVD) due to medication side effects and pathophysiological burden of the virus. However, in this retrospective cross-sectional analysis the hypothesis was tested if inflammatory biomark are associated with cardiovascular disease (CVD) in those with chronic viral infections. High sensitivity C-reactive protein (hs-CRP) is a tangible and under-recognized biomarker that can signal health care practitioners about the eminent risk of CVD.

Methods: The National Health and Nutrition Examination Survey (NHANES) is a cross sectional survey done on the non-institutionalized population of the United States by the Center for Disease Control and Prevention and National Center for Health Statistics. All patients from the nationally representative NHANES study, 20 years and older with HIV, Hepatitis B, or Hepatitis C between the years 1999-2010 were included in the analysis. Due to the complex sampling design, sample weights were utilized in order to analyze the data. Comparisons were analyzed using Pearson's Chi Square, simple, and multivariable logistic regression to determine the relationship of hs-CRP and chronic viruses. All missing variables were excluded.

Results: Data on at least one viral infection was available for 470 individuals (64.7% males & 35.3% females) who met the criteria of having chronic viruses and was representative of 2,737,489 individuals. Out of those with known viral status, 19.3% had HIV mono-infection, 23.6% had Hepatitis B mono-infection, 55.3% had Hepatitis C mono-infection, and 1.9% had co-infection. Elevated hs-CRP (>2 mg/dL vs. <1 mg/dL) was associated with CVD in unadjusted analyses (OR = 7.44, 95% CI: 2.42, 22.87, $p < .001$) among individuals with chronic virus; the association remained strong (OR = 9.04, 95% CI: 2.50, 32.66, $p < .001$) after the results were controlled for current infection and CVD risk factors (e.g. diabetes status and hypertension).

Conclusion: There is a significant relationship between high hs-CRP levels and patients with CVD, among patients with chronic viruses. Consequently, hs-CRP may be used as an effective prognostic biomarker to predict the occurrence of CVD before it proves fatal for the patient, in this subpopulation. More longitudinal studies need to be done to understand the role of other inflammatory markers and how anti-inflammatory agents may prevent CVD.